Anatomy image atlas | 2027

Full source pages, original labels, and mechanism review

156 source pages from the five requested documents are preserved after this guide. PDF text and vector artwork retain their original resolution. The companion Atlas.html contains 300-dpi PNG views, zoom controls, concept explanations and online textbook alternatives.

How to use an image

1. Identify the structure or axes. 2. Trace every arrow and describe what moves. 3. Connect each label to its function. 4. Hide the explanation and redraw the mechanism. 5. Predict a change caused by a named disease.

Image quality

300 dpi describes the page render, not the resolution of every embedded picture. Blurry source photographs cannot acquire missing detail by enlargement. Use the original PDF for vector labels and the linked textbook alternatives for key diagrams. No anatomical labels have been invented or altered.

Scope

AP_RULE_2027 remains the scope authority. The older slides and wiki snapshots include extra topics and occasional errors. Read the corrections below before memorizing source wording.

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Old tournament keys can contain outdated drug indications.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.

Mechanism companion

Air pathway and respiratory functions

R-AIR | Core | S27 pp.5; S23 pp.5-9; YR3 pp.8-12; RESP pp.1-2; supplementary reading WR pp.1-4

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

Check your understanding: Where does the conducting zone end, and why does that boundary matter?

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

Lungs, pleura and respiratory histology

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

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

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

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

Pressure, volume, airflow and ventilation calculations

R-VENT | Core | S27 p.6; S23 pp.10,13; YR3 pp.13,16; RESP p.2; supplementary reading WR pp.4-5

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.

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

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

Patterns of breathing

R-PAT | Core | S27 p.7; S23 p.11; YR3 p.14; supplementary reading WR pp.5-6

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

Check your understanding: Can rapid breathing occur without hyperventilation?

Avoid: Hyperpnea during exercise is not automatically pathological hyperventilation.

Spirograms, lung volumes and obstructive/restrictive patterns

R-SPIRO | Core | S27 pp.6,8; S23 pp.12-14; YR3 pp.15-17; supplementary reading WR p.5

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

Check your understanding: FEV1 = 1.8 L and FVC = 3.6 L. Calculate and interpret.

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

Gas exchange, diffusion and ventilation-perfusion matching

R-GAS | Core | S27 p.9; S23 pp.16-17; YR3 pp.19-20; supplementary reading WR pp.7-8

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

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

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

Oxygen transport, dissociation curves and carbon dioxide

R-HB | Core | S27 p.10; S23 pp.16-18; YR3 pp.19-21; supplementary reading WR pp.7-8

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

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

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

Exercise and high altitude

R-EX | Core | S27 p.11; YR3 p.22; supplementary reading WR pp.6-7

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

Check your understanding: Why is there less usable oxygen at altitude despite a similar percentage of oxygen?

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

Breathing control and acid-base chemistry

R-CTRL | State & National | S27 p.14; S23 pp.15,18; YR3 pp.18,21; supplementary reading WR pp.6,12

CO2 rises and pH falls: respiratory acidosis.

Check your understanding: A person hypoventilates. Predict CO2 and pH before renal compensation.

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

Food pathway, organs and functions

D-ANAT | Core | S27 pp.17-18; YR3 pp.25-37; DIG pp.1-3; supplementary reading WD pp.1-6

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

Check your understanding: Which structures receive food: pancreas, duodenum, gallbladder or jejunum?

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

Four gut-wall layers and enteric nerves

D-WALL | Core | S27 p.19; S23 digestive anatomy; YR3 digestive section; supplementary reading WD pp.2-5

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

Check your understanding: Where is the myenteric plexus relative to the circular and longitudinal muscle?

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

Compare esophagus, stomach, small intestine and colon

D-HIST | Core | S27 p.20; YR3 pp.31-37; histology resources listed in RES p.1; supplementary reading WD pp.4-6

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

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

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

Mechanical digestion, propulsion and chemical digestion

D-MECH | Core | S27 p.21-22; YR3 pp.26,38-39; DIG pp.1-2; supplementary reading WD pp.2-7

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

Check your understanding: Why does bile increase the effectiveness of lipase without being an enzyme?

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

Carbohydrate digestion and absorption

D-CARB | Core | S27 p.22; YR3 p.39; DIG p.2; supplementary reading WD pp.6-8

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

Check your understanding: What products result from lactase acting on lactose?

Avoid: Amylase does not digest protein, fat or cellulose.

Protein digestion, acid and gastric protection

D-PROT | Core | S27 pp.20,22; YR3 pp.33,39; DIG p.2; supplementary reading WD pp.6-8

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

Check your understanding: Why does the pancreas release trypsinogen rather than active trypsin?

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

Fat digestion, micelles and lymphatic absorption

D-FAT | Core | S27 p.22; YR3 pp.35-39; DIG p.2; supplementary reading WD pp.6-8

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

Check your understanding: Distinguish a micelle from a chylomicron.

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

Liver, pancreas, Kupffer cells and digestive regulation

D-LIVER | State & National | S27 p.26; YR3 pp.35-36; DIG pp.2-3; supplementary reading WD pp.6-8

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

Check your understanding: What does a Kupffer cell do that a hepatocyte does not primarily do?

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

Exercise, obesity and digestive function

D-EX | Core | S27 p.23; YR3 pp.41-44; supplementary reading WD pp.8-9

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

Check your understanding: Why can strenuous exercise cause GI symptoms while moderate activity helps motility?

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

Immune organs and histology

I-ORG | Core | S27 p.29; YR3 pp.46-50; IMM pp.9-11; supplementary reading WI p.7

Spleen filters blood; lymph nodes filter lymph.

Check your understanding: Which organ filters blood and which filters lymph?

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

Innate barriers, receptors and immune cells

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

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

Check your understanding: Why is a natural killer cell classified as innate even though it is a lymphocyte?

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

Complement and inflammation

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

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

Check your understanding: Name one complement function each for C3b and C5b-C9.

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

Adaptive immunity, antigen presentation and tolerance

I-ADAPT | Core | S27 p.31; YR3 pp.57-65; IMM p.4; supplementary reading WI pp.4-6

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

Check your understanding: Pair MHC I and MHC II with the responding T-cell populations.

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

Antibodies and sources of immunity

I-AB | Core | S27 pp.31-32; YR3 pp.60-68; supplementary reading WI pp.5-6

Secretory IgA protects mucosal surfaces; IgG crosses the placenta.

Check your understanding: Which antibody protects mucosal surfaces, and which crosses the placenta?

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

Response sequence, memory and allergy

I-RESP | Core | S27 p.32; YR3 pp.56-68,71; supplementary reading WI pp.4-6

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

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

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

Four hypersensitivity mechanisms

I-HYP | Core | S27 p.35; YR3 p.71; supplementary reading WI pp.7-8

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

Check your understanding: Compare serum sickness with contact dermatitis.

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

Transplants and prevention of rejection

I-GRAFT | State & National | S27 p.36; supplementary reading WI p.16

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

Check your understanding: Why is matching alone insufficient to prevent every allograft rejection?

Avoid: Autograft means same individual, not merely the same species.

Cancer immunotherapy: antibodies and CAR-T

I-THER | State & National | S27 p.37; YR3 p.70

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

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

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

Starter Pack

Anatomy & Physiology

2026 – 2027 Season

Respiratory System • Digestive System • Immune System

Pack Contents

- **This pack contains Note Slides to help you prepare for this event**
  - Explanations beyond the on-slide bullets are included in the Notes section of each slide — open Presenter View or Notes Page view in PowerPoint to read them
  - Diagrams are included throughout wherever a visual aids understanding (anatomy, gas exchange, spirograms, immune mechanisms, etc.)
- **Content follows the official 2026–2027 (“2027”) Science Olympiad Anatomy & Physiology B/C event rules**
  - Topics marked National-level topic (italicized in the official rules) are only assessed at the National Tournament
- **Example Regional / State / National exams for this event are provided as a separate companion document**

Topic Areas

The event covers three major systems, each with additional depth assessed only at the State and National levels:

Respiratory System

- Anatomy & ventilation mechanics
- Spirometry & gas exchange
- O_2 transport & regulation
- COPD, asthma, pneumonia + more

Digestive System

- Alimentary canal anatomy
- Mechanical & chemical digestion
- Liver / pancreas function
- Ulcers, IBD, hepatitis + more

Immune System

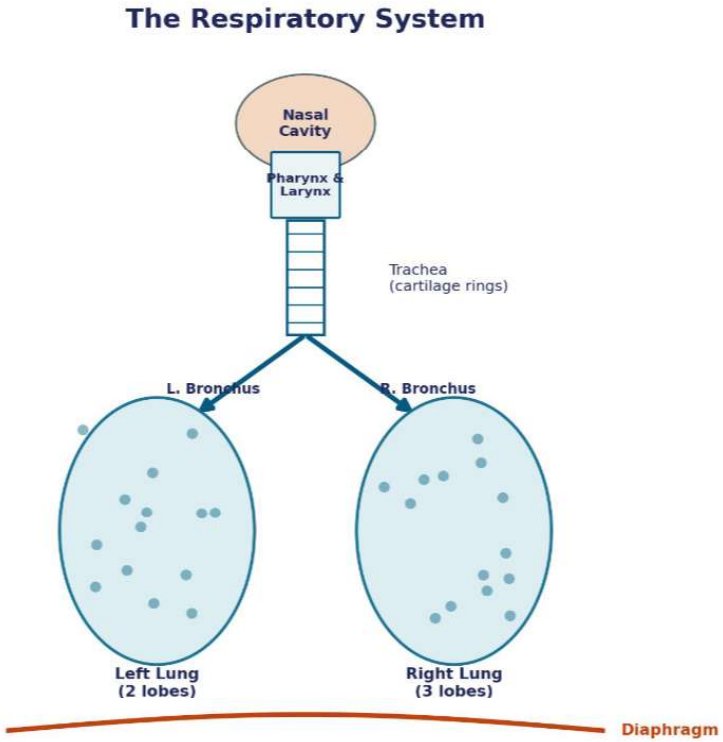
- Innate vs. adaptive immunity
- Hypersensitivity types I–IV
- Autoimmune & immunodeficiency
- Transplants & immunotherapy

Respiratory System

Anatomy • Ventilation • Gas Exchange • Disorders • Regulation

Anatomy & Major Functions

- Conducting zone: nasal cavity → pharynx → larynx → trachea → bronchi → bronchioles — warms, filters, and humidifies air; no gas exchange occurs here
- Respiratory zone: respiratory bronchioles, alveolar ducts, alveoli — the actual site of gas exchange
- Right lung has 3 lobes; left lung has 2 lobes (leaves room for the heart's cardiac notch)
- Pleura: visceral (covers lung) and parietal (lines cavity) membranes separated by pleural fluid, which reduces friction during breathing
- Core functions: pulmonary ventilation, gas exchange, blood pH regulation (via CO_2), phonation, olfaction, and protection (mucociliary escalator, cough reflex)



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

Mechanics & Measures of Pulmonary Ventilation

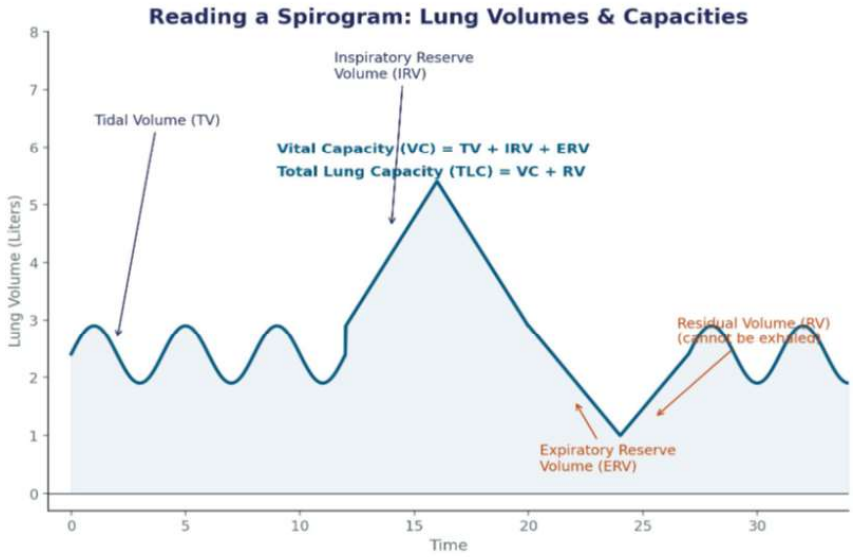
- Ventilation = bulk airflow into/out of the lungs, driven by pressure gradients (Boyle's Law: pressure and volume are inversely related)
- Inspiration: diaphragm + external intercostals contract → thoracic volume increases → intrapulmonary pressure drops below atmospheric → air flows in
- Quiet expiration is passive (elastic recoil of lungs/chest wall); forced expiration recruits internal intercostals and abdominal muscles
- Key measures: tidal volume (TV), minute ventilation (TV × respiratory rate), FEV1, FVC, and the FEV1/FVC ratio — used to distinguish obstructive vs. restrictive disease
- Compliance = ease of lung expansion; elastance = tendency to recoil back — both affect the work of breathing

Patterns of Breathing

- Eupnea — normal, quiet breathing at rest
- Hyperpnea (increased depth to meet metabolic demand, e.g., exercise) vs. tachypnea (increased rate) vs. hyperventilation (excess ventilation relative to metabolic need, drops CO_2)
- Apnea (cessation of breathing), dyspnea (difficulty/labored breathing), orthopnea (dyspnea when lying flat)
- Kussmaul breathing: deep, labored breathing seen in diabetic ketoacidosis (blowing off CO_2 to compensate for metabolic acidosis)
- Cheyne-Stokes breathing: cyclical waxing/waning depth with pauses, associated with brain injury or heart failure; Biot's breathing: irregular groups of breaths separated by apnea

Reading a Spirogram

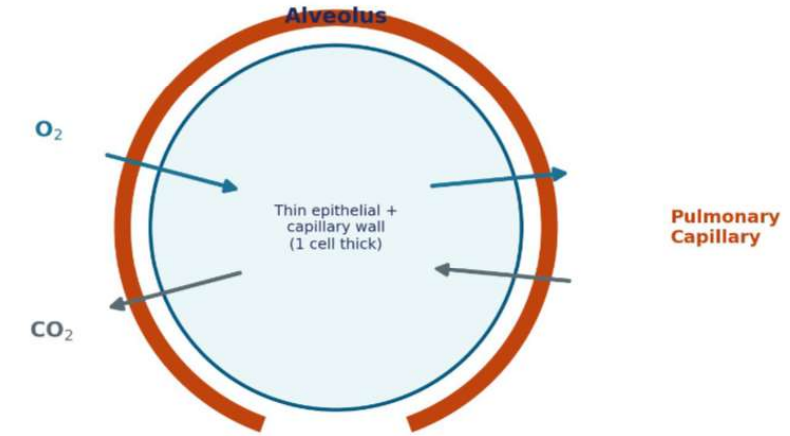
- Tidal Volume (TV): air moved in a normal quiet breath (~500 mL)
- Inspiratory Reserve Volume (IRV) / Expiratory Reserve Volume (ERV): extra air that can be forcefully inhaled/exhaled beyond tidal breathing
- Residual Volume (RV): air remaining in the lungs after maximal exhalation — cannot be measured directly by spirometry (keeps alveoli open)
- Vital Capacity (VC) = TV + IRV + ERV; Total Lung Capacity (TLC) = VC + RV
- Be able to read volumes directly off a spirogram trace and calculate any capacity from the component volumes



Lung volumes/capacities plotted on a spirometer trace

Gas Exchange in the Alveoli

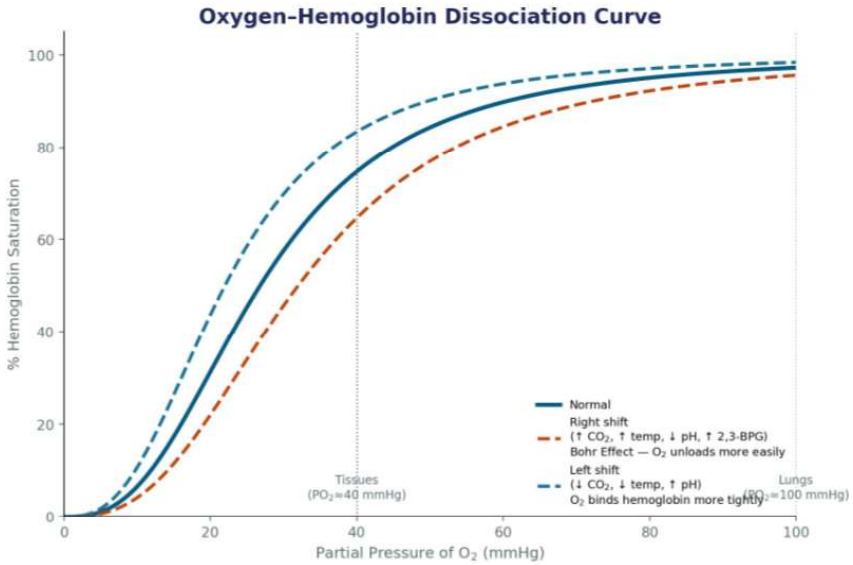
- Gas exchange occurs by simple diffusion across the thin alveolar-capillary membrane (about one cell thick)
- Gases move down partial-pressure gradients: O_2 diffuses from alveolar air (high PO_2) into blood (low PO_2); CO_2 diffuses from blood into alveolar air
- Diffusion rate depends on surface area, membrane thickness, partial-pressure gradient, and diffusion distance (Fick's Law of Diffusion)
- Ventilation-perfusion (V/Q) matching: efficient gas exchange requires airflow (ventilation) and blood flow (perfusion) to be well matched within each region of the lung



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

Oxygen Transport & the Dissociation Curve

- ~98% of O_2 in blood is carried bound to hemoglobin (Hb); the rest is dissolved in plasma
- The O_2-Hb dissociation curve is sigmoidal due to cooperative binding — each O_2 bound makes the next easier to bind
- Bohr effect: increased CO_2, decreased pH, increased temperature, and increased 2,3-BPG shift the curve right — hemoglobin releases O_2 more readily (helpful in exercising tissue)
- A left shift (opposite conditions) increases Hb's affinity for O_2, favoring loading in the lungs
- CO_2 is transported mainly as bicarbonate (HCO_3^-) in plasma (~70%), plus carbaminohemoglobin (~23%) and dissolved CO_2 (~7%)



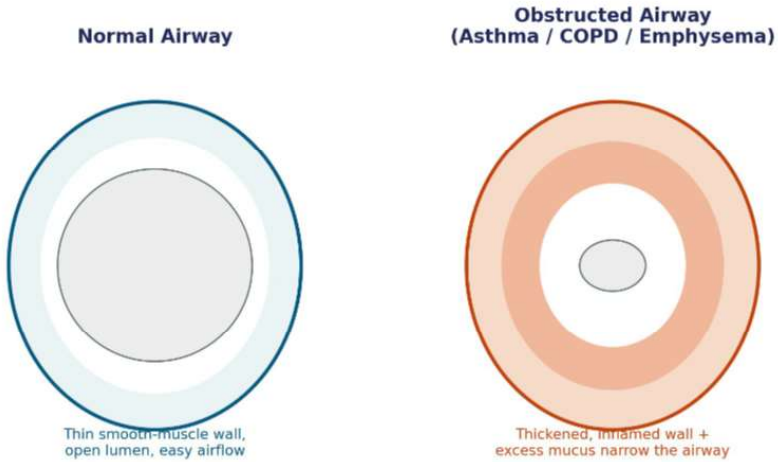
Sigmoidal O_2-hemoglobin dissociation curve, with Bohr-effect shifts

Effects of Exercise & High Altitude

- Exercise increases minute ventilation and O_2 demand; working muscles generate more CO_2/heat/acid, right-shifting the dissociation curve to unload O_2 faster
- High altitude: lower atmospheric PO_2 triggers hyperventilation and, over days/weeks, increased erythropoietin (EPO) and red blood cell production (acclimatization)
- Short-term altitude exposure without acclimatization risks altitude sickness, including high-altitude pulmonary edema (HAPE) and high-altitude cerebral edema (HACE)
- Trained athletes may show increased vital capacity, more efficient gas exchange, and greater capillary density in muscle over time

Respiratory Disorders I

- COPD (chronic obstructive pulmonary disease): umbrella term including chronic bronchitis and emphysema; progressive airflow limitation, often from smoking
- Asthma: reversible airway inflammation/bronchoconstriction triggered by allergens, exercise, or irritants
- Emphysema: destruction of alveolar walls reduces surface area for gas exchange and elastic recoil
- Pneumonia: infection (bacterial, viral, or fungal) causing alveolar inflammation and fluid/pus accumulation
- Sleep apnea: repeated pauses in breathing during sleep (obstructive — airway collapse, or central — brain fails to signal breathing)
- Cystic fibrosis: genetic disorder causing thick, sticky mucus that obstructs airways and promotes chronic infection



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

Additional Respiratory Diseases

National-level topic

- Tuberculosis (TB): bacterial infection (Mycobacterium tuberculosis), often forming granulomas in the lungs
- Pulmonary edema: fluid accumulation in alveoli, often from heart failure, impairing gas exchange
- Pleurisy: inflammation of the pleural membranes, causing sharp pain with breathing
- Lung cancer: small cell vs. non-small cell lung cancer — differ in cell origin, growth rate, and treatment approach
- Pulmonary fibrosis: scarring/thickening of lung tissue that reduces compliance and impairs diffusion
- Pneumoconiosis: occupational lung diseases from inhaled particles — silicosis (silica dust), coal worker's pneumoconiosis (“black lung”), asbestosis (asbestos fibers)

Regulation of Breathing & Blood Chemistry

- Breathing is controlled by respiratory centers in the medulla oblongata and pons, which set the basic rhythm
- Central chemoreceptors (in the brainstem) sense CSF pH, which reflects blood CO_2 — the primary driver of ventilation rate
- Peripheral chemoreceptors (carotid and aortic bodies) sense blood O_2, CO_2, and pH, and become more important when O_2 drops significantly
- $CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$ (carbonic acid equilibrium) links breathing directly to blood pH
- Respiratory acidosis/alkalosis result from hypo-/hyperventilation; the kidneys and lungs work together (metabolic vs. respiratory compensation) to maintain pH near 7.4

Treatments & Prevention

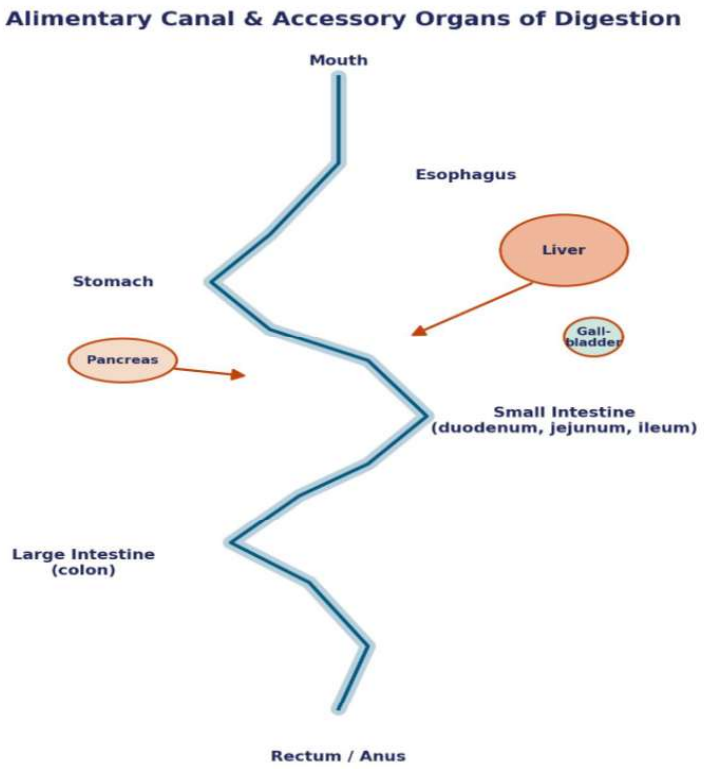
- Bronchodilators (beta-agonists, anticholinergics) relax airway smooth muscle — used in asthma and COPD
- Inhaled or systemic corticosteroids reduce airway inflammation
- Supplemental oxygen therapy and CPAP/BiPAP support gas exchange and airway patency (e.g., in sleep apnea)
- Antibiotics/antivirals for infectious causes (pneumonia, TB); airway clearance therapy and CFTR modulators for cystic fibrosis
- Vaccination (influenza, pneumococcal, TB in some countries), smoking cessation, pulmonary rehabilitation, and surgery (e.g., lobectomy) as prevention/treatment strategies across these conditions

Digestive System

Anatomy • Mechanical & Chemical Digestion • Liver/Pancreas • Disorders

Anatomy & Functions of the Digestive System

- Four basic functions: ingestion, digestion (mechanical + chemical), absorption, and elimination (defecation)
- The alimentary canal (GI tract) is a continuous tube from mouth to anus, roughly 9 meters long in life
- Accessory organs (salivary glands, liver, gallbladder, pancreas) contribute secretions but food never passes through them
- Digestion breaks macromolecules into absorbable units; absorption occurs mainly across the small intestine's lining into blood/lymph



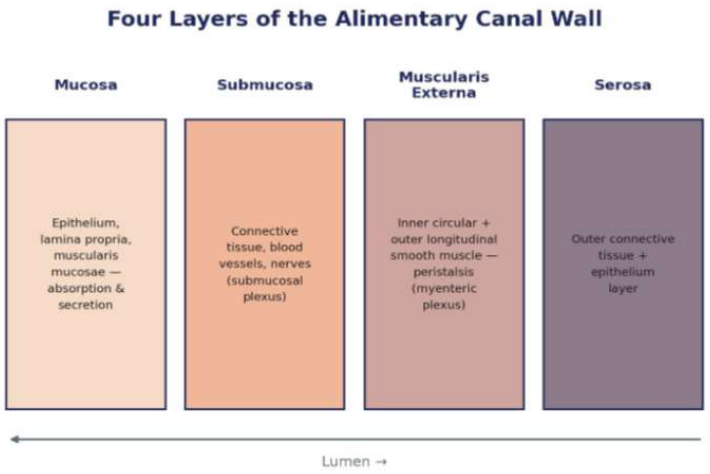
The ~9-meter alimentary canal, plus accessory organs

Parts of the Alimentary Canal & Accessory Organs

- Mouth: mechanical digestion (teeth, tongue) + chemical digestion begins (salivary amylase breaks down starch)
- Pharynx & esophagus: passageway; peristalsis moves the bolus to the stomach
- Stomach: mixes food with acid and enzymes into chyme; regions include cardia, fundus, body, pylorus
- Small intestine: duodenum (receives bile + pancreatic secretions), jejunum, ileum — primary site of digestion and absorption
- Large intestine: cecum, colon, rectum, anus — water/electrolyte absorption and waste storage
- Accessory organs: salivary glands, liver, gallbladder (stores/concentrates bile), pancreas (enzymes + bicarbonate)

Four Layers of the Alimentary Canal Wall

- Mucosa (innermost): epithelium + lamina propria + muscularis mucosae — secretion and absorption
- Submucosa: dense connective tissue with blood vessels, lymphatics, and the submucosal (Meissner's) nerve plexus
- Muscularis externa: inner circular + outer longitudinal smooth muscle layers — generates peristalsis; contains the myenteric (Auerbach's) plexus between the layers
- Serosa (or adventitia in some regions): outer connective tissue ± epithelium, anchors/protects the organ



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

Comparing the Lining Along the Canal

- Esophagus: stratified squamous epithelium — built for protection against abrasion from swallowed food
- Stomach: simple columnar epithelium with gastric pits leading to gastric glands (mucous neck cells, parietal cells — HCl, chief cells — pepsinogen, G cells — gastrin)
- Small intestine: villi and microvilli (brush border) dramatically increase surface area for absorption; contains crypts of Lieberkühn
- Large intestine: no villi; abundant goblet cells for mucus secretion; wall has pouches called haustra; specializes in water/electrolyte reabsorption

Mechanical vs. Chemical Digestion

- Mechanical digestion: physically breaks food into smaller pieces without altering its chemical structure — mastication, churning in the stomach, segmentation, and peristalsis
- Chemical digestion: enzymes (and bile, for fats) break covalent bonds in macromolecules to yield absorbable subunits
- Mechanical digestion increases surface area, which speeds up chemical digestion — the two work together throughout the canal

Chemical Digestion: Proteins, Fats & Carbohydrates

- Carbohydrates: salivary and pancreatic amylase break starch into disaccharides; brush border enzymes (e.g., maltase, sucrase, lactase) finish breaking these into monosaccharides for absorption
- Proteins: pepsin (stomach, activated from pepsinogen by HCl) begins protein digestion; pancreatic trypsin and chymotrypsin continue it; brush border peptidases yield absorbable amino acids/dipeptides
- Fats: bile salts (from the liver, stored in the gallbladder) emulsify fat into smaller droplets (not a chemical reaction); pancreatic lipase then chemically digests fats into fatty acids and monoglycerides
- Fat digestion products form micelles for transport to the intestinal wall, are reassembled into triglycerides inside intestinal cells, and packaged into chylomicrons that enter lymphatic lacteals rather than blood capillaries

Effects of Exercise & Obesity

- Moderate exercise can support healthy GI motility; intense exercise diverts blood flow away from the gut toward muscles, which can cause GI distress or, in extreme cases, ischemia
- Obesity is linked to increased risk of GERD, gallstones, non-alcoholic fatty liver disease, altered gut motility, and changes in the gut microbiome
- Both obesity and inactivity are associated with increased risk of several digestive system cancers

Core Digestive Diseases

- Stomach & duodenal (peptic) ulcers: erosion of the mucosal lining, often from H. pylori infection or NSAID use, with acid contributing to damage
- Cancers of the digestive system: can arise anywhere along the canal or in accessory organs (e.g., colorectal, gastric, pancreatic, liver cancer)
- Diarrhea: increased frequency/liquidity of stool from infection, malabsorption, or motility disorders; risk of dehydration and electrolyte imbalance
- Lactose intolerance: insufficient lactase enzyme leads to undigested lactose fermenting in the colon, causing bloating, gas, and diarrhea
- Hepatitis: inflammation of the liver, from viral infection (Hepatitis A/B/C, etc.), toxins, or autoimmune causes
- Appendicitis: inflammation/infection of the appendix, risk of rupture and peritonitis if untreated

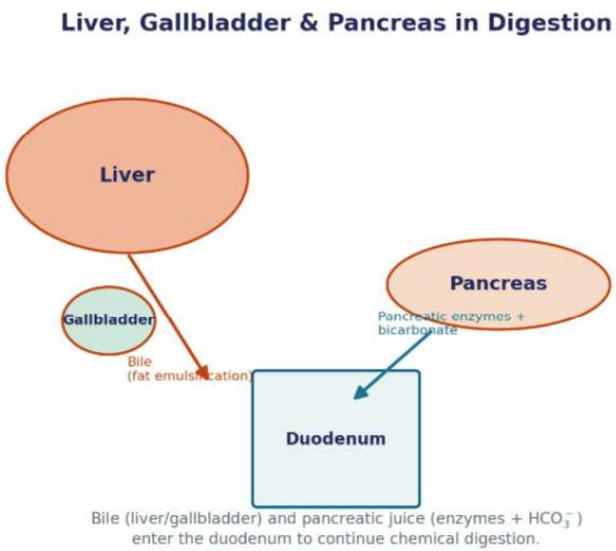
Additional Digestive Diseases

National-level topic

- Diverticular disease: small pouches (diverticula) form in the colon wall and can become inflamed or infected (diverticulitis)
- GERD (gastroesophageal reflux disease): stomach acid backs up into the esophagus, damaging its lining over time
- Peptic ulcer disease (PUD): chronic/recurrent ulceration of the stomach or duodenum
- Ulcerative colitis vs. Crohn's disease (inflammatory bowel diseases): ulcerative colitis is continuous inflammation limited to the colon/rectum mucosa; Crohn's can affect any part of the GI tract, is often patchy ("skip lesions"), and involves the full thickness of the wall
- Celiac disease: autoimmune reaction to gluten damages small intestine villi, impairing absorption

The Liver & Pancreas in Digestion

- Liver: produces bile (for fat emulsification), detoxifies blood, stores glycogen, synthesizes plasma proteins; receives blood from the GI tract via the hepatic portal vein
- Kupffer cells: specialized resident macrophages lining liver sinusoids that phagocytose bacteria, debris, and old blood cells arriving from the gut
- Gallbladder: stores and concentrates bile between meals, releases it into the duodenum in response to a fatty meal
- Pancreas has both functions: exocrine (digestive enzymes — amylase, lipase, proteases — plus bicarbonate to neutralize stomach acid, delivered via the pancreatic duct) and endocrine (insulin/glucagon from the islets of Langerhans, released into blood)



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

Treatments & Prevention

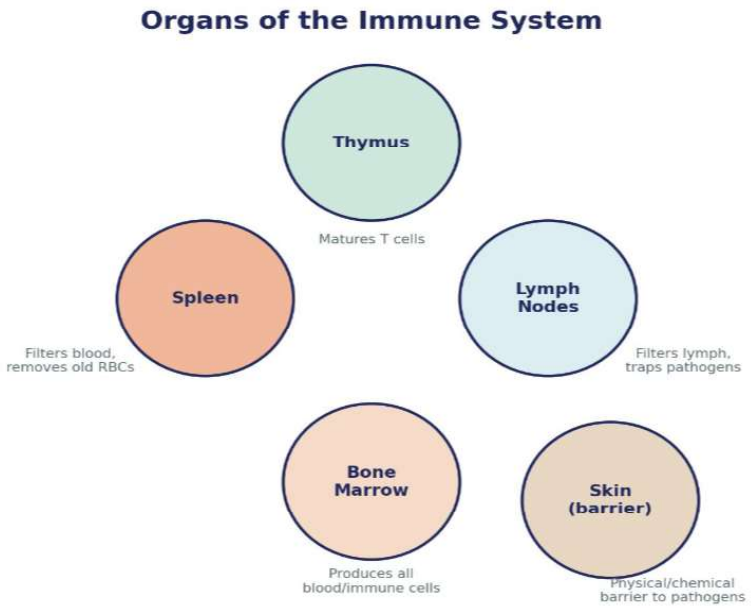
- Antacids, H2 blockers, and proton pump inhibitors (PPIs) reduce stomach acid for ulcers/GERD
- Antibiotics to eradicate H. pylori (a common cause of peptic ulcers)
- Surgery: appendectomy, cholecystectomy (gallbladder removal), bowel resection for severe IBD
- Dietary management: gluten-free diet (celiac), lactose-free diet (lactose intolerance), fiber for diverticular disease
- Biologic medications (e.g., anti-inflammatory antibody therapies) for inflammatory bowel disease; vaccination for Hepatitis A/B

Immune System

Innate & Adaptive Immunity • Hypersensitivity • Disorders • Immunotherapy

Anatomy of the Immune System

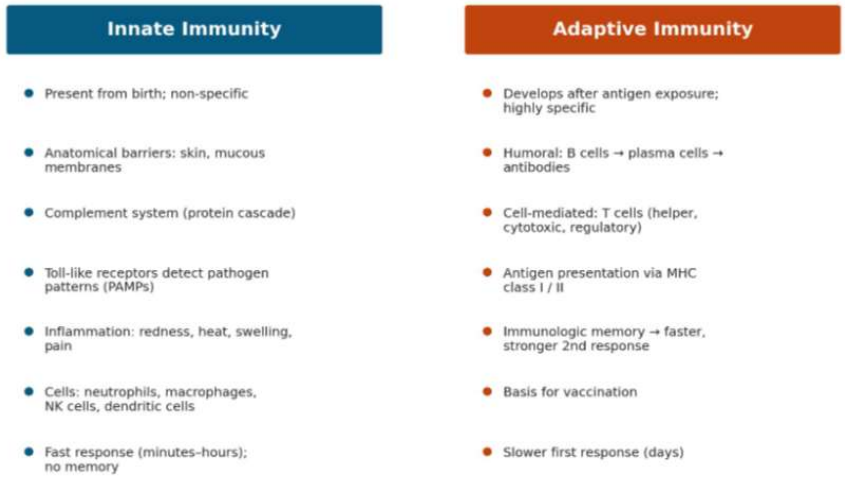
- Bone marrow: origin of all blood and immune cells (hematopoiesis), including B cells (which mature here)
- Thymus: site where T cells mature and learn to distinguish self from non-self
- Spleen: filters blood, removes aged red blood cells, and provides a site for immune surveillance/response to blood-borne antigens
- Lymph nodes: filter lymph fluid, trap pathogens/antigens, and are sites where adaptive immune responses are initiated
- Skin: the body's largest physical barrier, with chemical defenses (low pH, antimicrobial secretions) and immune cells (e.g., Langerhans cells)



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

Innate Immunity

- Present from birth, non-specific, and fast-acting (minutes to hours) — the body's first line of defense
- Anatomical barriers: skin and mucous membranes physically block pathogen entry
- Complement system: a cascade of blood proteins that can directly lyse pathogens (membrane attack complex), tag them for destruction (opsonization), and attract immune cells (chemotaxis)
- Toll-like receptors (TLRs) on innate immune cells recognize conserved pathogen patterns (PAMPs) and trigger a response
- Inflammation: redness, heat, swelling, and pain result from increased blood flow and vessel permeability at a site of infection/injury
- Key cells: neutrophils, macrophages, natural killer (NK) cells, and dendritic cells



Innate vs. adaptive immunity, side by side

Adaptive Immunity

- Develops after exposure to a specific antigen; highly specific and slower to activate initially (days), but improves with repeated exposure
- Humoral immunity: B cells recognize antigen and differentiate into plasma cells, which secrete antibodies specific to that antigen
- Cell-mediated immunity: T cells (helper T cells coordinate the response; cytotoxic T cells kill infected/abnormal cells; regulatory T cells limit the response)
- Antigen presentation via MHC class I (all nucleated cells, presents to cytotoxic T cells) and MHC class II (antigen-presenting cells, presents to helper T cells)
- Immunologic memory: memory B and T cells persist after an infection, enabling a faster, stronger secondary response — the basis for vaccination

The Immune Response & Allergic Reactions

- General immune response sequence: pathogen recognition → innate response → antigen presentation → adaptive activation (clonal selection/expansion) → effector response (pathogen clearance) → memory cell formation
- Primary immune response (first exposure) is slower and weaker than the secondary response (later exposure), due to pre-existing memory cells
- Allergic reactions occur when the immune system mounts an inappropriate IgE antibody response against a normally harmless antigen (allergen)
- On re-exposure, allergen binds IgE on mast cells/basophils, triggering degranulation and release of histamine and other mediators, producing allergy symptoms

Immunodeficiency Disorders

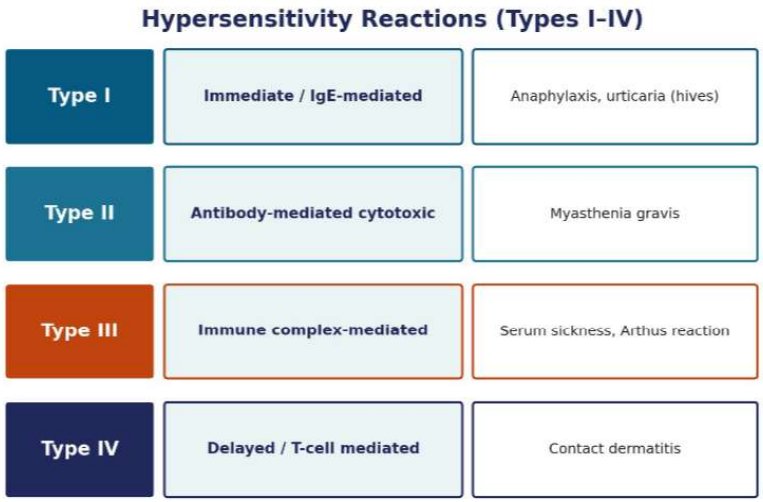
- HIV/AIDS: HIV infects and destroys CD4+ helper T cells, progressively crippling the adaptive immune response; AIDS is the advanced stage marked by opportunistic infections
- Common Variable Immunodeficiency (CVID): impaired antibody (immunoglobulin) production, leading to recurrent infections
- Severe Combined Immunodeficiency (SCID): a group of genetic disorders causing defects in both B and T cell function ("bubble boy disease") — life-threatening without treatment such as bone marrow transplant

Autoimmune Diseases

- Multiple sclerosis (MS): immune system attacks myelin in the central nervous system, disrupting nerve signal conduction
- Rheumatoid arthritis (RA): immune attack on the synovium (joint lining) causes chronic joint inflammation and damage
- Hashimoto thyroiditis: autoimmune attack on the thyroid gland leads to hypothyroidism
- Graves' disease: antibodies stimulate the TSH receptor, causing hyperthyroidism

Hypersensitivity Reactions (Types I–IV)

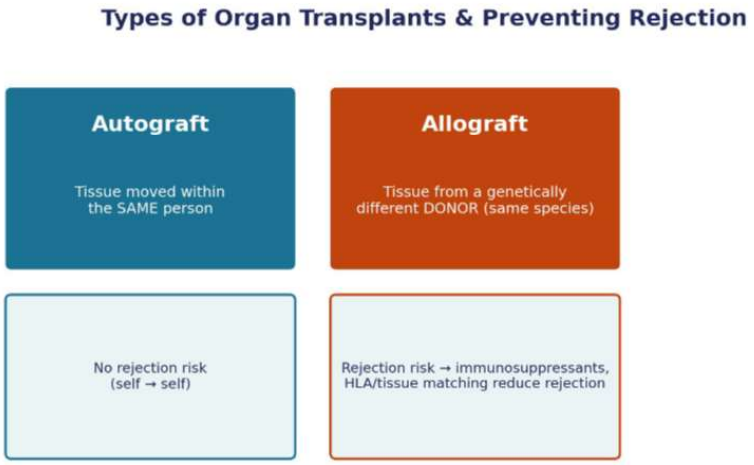
- Type I (immediate, IgE-mediated): mast cell degranulation on re-exposure to allergen — anaphylaxis, urticaria (hives)
- Type II (antibody-mediated cytotoxic): antibodies bind antigens on the body's own cells, marking them for destruction — myasthenia gravis (antibodies block acetylcholine receptors)
- Type III (immune complex-mediated): antigen-antibody complexes deposit in tissues and trigger inflammation — serum sickness, Arthus reaction
- Type IV (delayed-type, T-cell mediated): no antibodies involved; sensitized T cells cause a reaction 24–72 hours after exposure — contact dermatitis



The four hypersensitivity types, mechanism and a classic example

Organ Transplants & Preventing Rejection

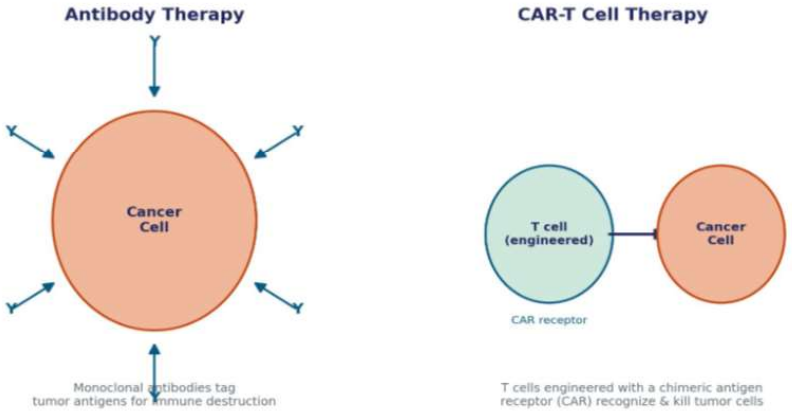
- Autograft: tissue moved from one site to another within the same person — no rejection risk since it is immunologically self
- Allograft: tissue from a genetically different donor of the same species — carries rejection risk since the recipient's immune system may recognize donor antigens (via MHC/HLA molecules) as foreign
- Rejection prevention: HLA/tissue typing to find the closest donor match, plus lifelong immunosuppressant medications to dampen the recipient's immune response
- Rejection can be hyperacute (minutes-hours), acute (days-weeks), or chronic (months-years), depending on the immune mechanism involved



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

Cancer Immunotherapies

- Antibody therapies: monoclonal antibodies engineered to bind specific tumor antigens, marking cancer cells for immune destruction or blocking growth signals
- Checkpoint inhibitor antibodies release the 'brakes' the immune system normally uses to avoid over-activation, unleashing T cells against tumors
- CAR-T cell therapy: a patient's own T cells are engineered to express a chimeric antigen receptor (CAR) that recognizes a specific tumor antigen, then reinfused to seek and destroy cancer cells



Antibody therapy and CAR-T cell therapy, simplified

Additional Immune Disorders

National-level topic

- Systemic lupus erythematosus (SLE): systemic autoimmune disease that can affect skin, joints, kidneys, and other organs; associated with immune complex deposition (Type III mechanism)
- Dermatomyositis: autoimmune inflammation of skin and muscle, causing rash and muscle weakness
- Scleroderma: autoimmune disease causing abnormal connective tissue/collagen buildup, hardening skin and sometimes internal organs
- Psoriasis and psoriatic arthritis: chronic autoimmune skin condition (thick, scaly plaques) that can also cause joint inflammation (psoriatic arthritis)

Treatments & Prevention

- Immunosuppressant drugs (e.g., corticosteroids, calcineurin inhibitors) dampen an overactive or misdirected immune response — used in autoimmune disease and transplant rejection
- Biologic drugs (e.g., TNF inhibitors) target specific immune signaling molecules in autoimmune/inflammatory disease
- Antihistamines and epinephrine (EpiPen) treat allergic reactions; epinephrine is first-line for anaphylaxis
- Vaccination trains adaptive immunity (memory cells) against a pathogen before natural exposure occurs
- IVIG (intravenous immunoglobulin) can supplement antibody levels in some immunodeficiency disorders

Anatomy and Physiology B/C

Starter Pack



Pack Contents

- How to use this Pack.
- Note Slides on Event Topics
- Sample Tests
- Answer Keys for Sample Tests

How to use this Pack

- Event Starter Packs are a collection of notes and past exams created by top event supervisors and rule committee members
- Use the notes to jump start your collection of resources and creation of your own notes.
    - Don't simply rely on the information presented here; dig deeper, learn more
- Use terms and topics listed as a starting point for own research
- Use the notes to create your own exams to test your knowledge
- Use the different exams provided to test your knowledge and identify areas to study more in depth

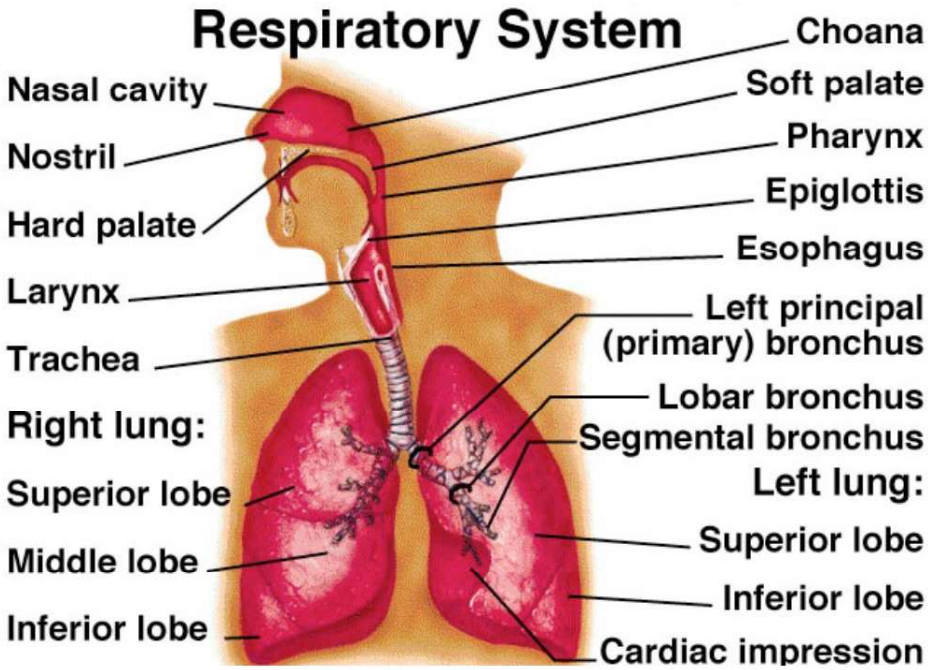
RESPIRATORY SYSTEM

Respiratory System – Functions

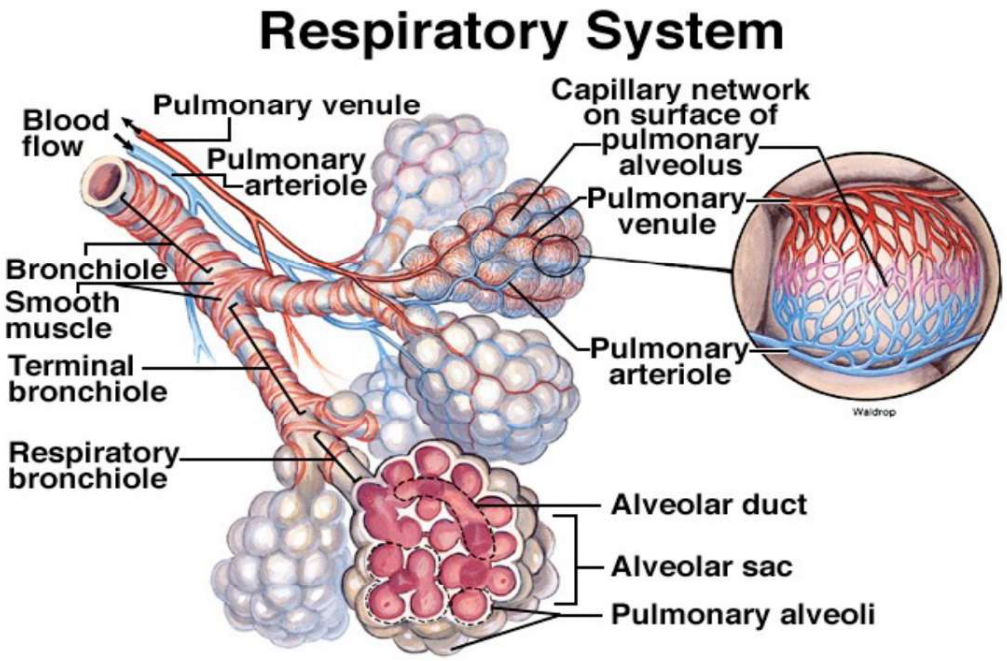
Basic functions of the respiratory system are:

1. provides oxygen to the blood stream and removes carbon dioxide
2. enables sound production or vocalization as expired air passes over the vocal chords
3. enables protective and reflexive non-breathing air movements such as coughing and sneezing, to keep the air passages clear
4. control of Acid-Base balance
5. control of blood pH

Respiratory System - Principal Organs



Respiratory System – Lungs



Non-respiratory Air Movements

Table 17.5 Nonrespiratory Air Movements

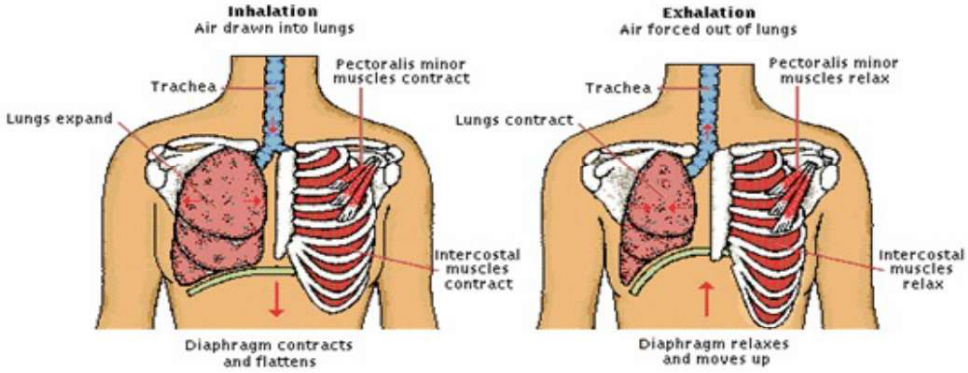
Air Movement	Mechanism	Comments
Coughing	Deep inspiration followed by a closure of of the glottis. The forceful expiration that results abruptly opens the glottis, sending a blast of air through the upper respiratory tract.	Reflexive or voluntary. Stimulus may be foreign material irritating the larynx 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.

Respiration Process

A collective term for the following processes:

- Pulmonary Ventilation
  - Movement of air into the lungs (inspiration)
  - Movement of air out of the lungs (expiration)
- External Respiration
  - Movement of oxygen from the lungs to the blood
  - Movement of carbon dioxide from the blood to the lungs
- Transport of Respiratory Gases
  - Transport of oxygen from the lungs to the tissues
  - Transport of carbon dioxide from the tissues to the lungs
- Internal Respiration
  - Movement of oxygen from blood to the tissue cells
  - Movement of carbon dioxide from tissue cells to blood

Pulmonary Ventilation



The intercostal muscles and the diaphragm work together

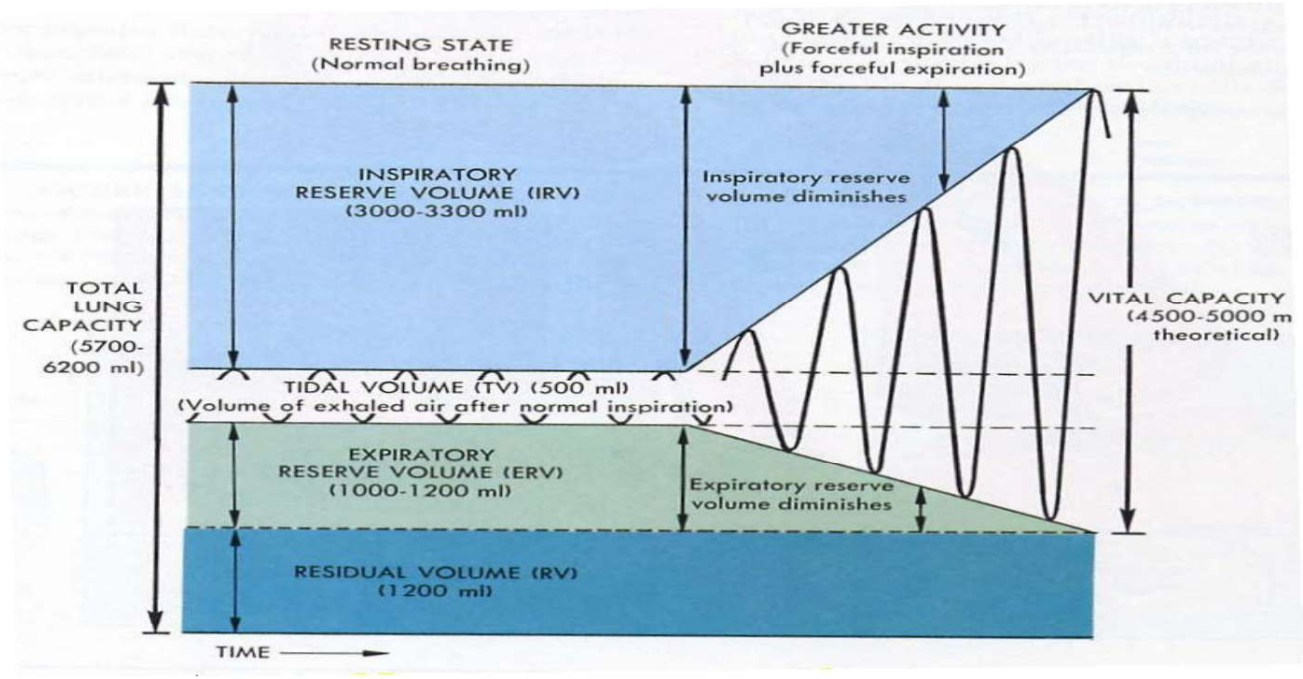
Inspiration, or inhalation – a very active process that requires input of energy
Air flows into the lungs when the thoracic pressure falls below atmospheric pressure. The diaphragm moves downward and flattens while the intercostal muscles contract.

Expiration, or exhalation – a passive process that takes advantage of the recoil properties of elastic fibers Air is forced out of the lungs when the thoracic pressure rises above atmospheric pressure. The diaphragm and expiratory muscles relax.

Patterns of Breathing

- Apnea – temporary cessation of breathing (one or more skipped breaths)
- Dyspnea – labored, gasping breathing; shortness of breath
- Eupnea – normal, relaxed, quiet breathing
- Hyperpnea – increased rate and depth of breathing in response to exercise, pain, or other conditions
- Hyperventilation – increased pulmonary ventilation in excess of metabolic demand
- Hypoventilation – reduced pulmonary ventilation
- Orthopnea – Dyspnea that occurs when a person is lying down
- Respiratory arrest – permanent cessation of breathing
- Tachypnea – accelerated respiration

Pulmonary Ventilation - Volumes



Measures of Pulmonary Ventilation

Respiratory volumes – values determined by using a spirometer

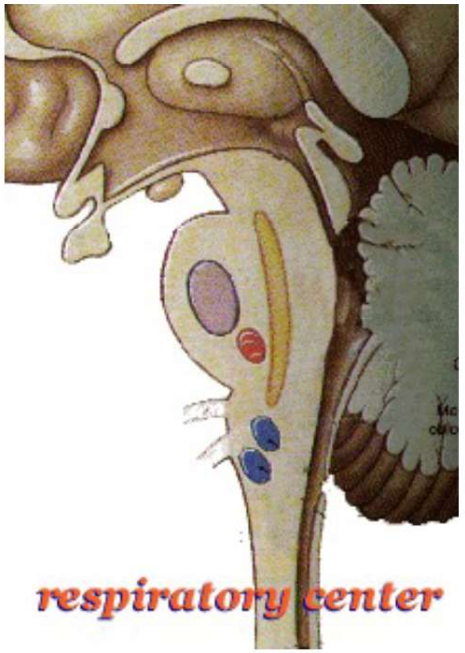
- **Tidal Volume (TV) – amount of air inhaled or exhaled with each breath under resting conditions**
- **Inspiratory Reserve Volume (IRV) – amount of air that can be inhaled during forced breathing in addition to resting tidal volume**
- **Expiratory Reserve Volume (ERV) – amount of air that can be exhaled during forced breathing in addition to tidal volume**
- **Residual Volume (RV) – Amount of air remaining in the lungs after a forced exhalation.**

Formulas – Capacities

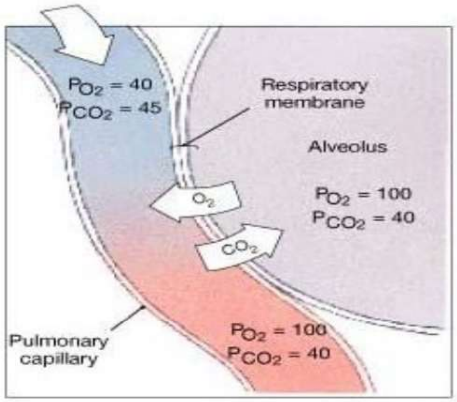
- Vital Capacity – maximum amount of air that can be expired after taking the deepest breath possible (VC = TV + IRV + ERV)
- Inspiratory Capacity – maximum volume of air that can be inhaled following exhalation of resting tidal volume (IC = TV + IRV)
- Functional Residual Capacity – volume of air remaining in the lungs following exhalation of resting volume (FRC = ERV + RV)
- Total Lung Capacity – total volume of air that the lungs can hold (TLC = VC + RV)

Control of Respiratory System

- Respiratory control centers – found in the pons and the medulla oblongata
- Control breathing
- Adjusts the rate and depth of breathing according to oxygen and carbon dioxide levels
- Afferent connections to the brainstem
- Hypothalmus and limbic system send signals to respiratory control centers

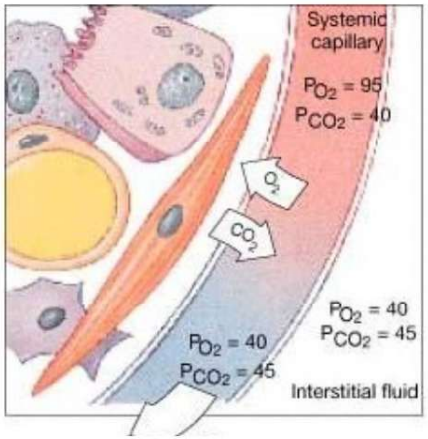


Gas Exchange and Transport



- Alveolar Gas Exchange – the loading of oxygen and the unloading of carbon dioxide in the lungs
- Oxygen is carried in the blood bound to hemoglobin (98.5%) and dissolved in plasma (1.5%)
- Carbon dioxide is transported in three forms
  - Carbonic acid – 90% of carbon dioxide reacts with water to form carbonic acid
  - Carboamino compounds – 5% binds to plasma proteins and hemoglobin
  - Dissolved gas – 5% carried in the blood as dissolved gas

Systemic Gas Exchange



- **Carbon dioxide loading -The Haldane Effect – the lower the partial pressure of oxygen and saturation of it in hemoglobin, the more carbon dioxide can be carried in the blood**
- **Oxygen unloading from hemoglobin molecules**

Blood Chemistry & Respiratory Rhythm

Hydrogen ion concentrations -strongly influence respiration

Carbon dioxide concentrations -strongly influence respiration

Oxygen concentrations - have little effect on respiration

Effects of Exercise on Respiratory System

During exercise

- The muscle cells use up more oxygen and produce increased amounts of carbon dioxide.
- The lungs and heart have to work harder to supply the extra oxygen and remove the carbon dioxide.
- Your breathing rate increases and you breathe more deeply. Heart rate also increases in order to transport the oxygenated blood to the muscles.
- Muscle cell respiration increases - more oxygen is used up and levels of carbon dioxide rise.
- The brain detects increasing levels of carbon dioxide - a signal is sent to the lungs to increase breathing.
- Breathing rate and the volume of air in each breath increase - This means that more gaseous exchange takes place.
- The brain also tells the heart to beat faster so that more blood is pumped to the lungs for gaseous exchange.
- More oxygenated blood is gets to the muscles and more carbon dioxide is removed.

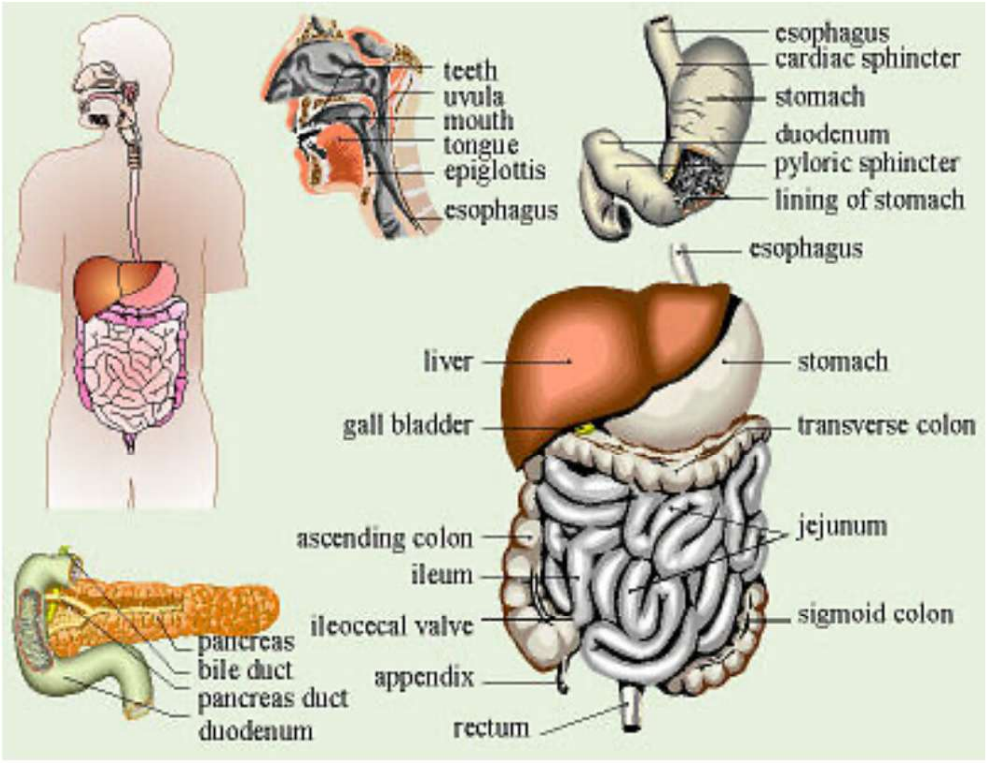
Disorders of the Respiratory System

- Clinical Disorders and Diseases of the Respiratory System
  - Hypoxia – deficiency of oxygen in a tissue or the inability to use oxygen
  - Oxygen Toxicity – excess oxygen, causing the build up of peroxides and free radicals
- Chronic obstructive pulmonary diseases – long-term obstruction of airflow and a substantial reduction in pulmonary ventilation
  - Chronic bronchitis – cilia are immobilized and reduced in number; goblet cells increase their production of mucus → mucus clogs the airways and breeds infection
  - Emphysema – alveolar walls break down and the surface area of the lungs is reduced
  - Asthma – allergens trigger the release of histamine and other inflammatory chemicals that cause intense bronchoconstriction
- Lung Cancer – cancer of the lung
- Acute Rhinitis – the common cold
- Laryngitis – inflammation of the vocal folds
- Pneumonia – lower respiratory infection that causes fluid build up in the lungs
- Sleep Apnea – Cessation of breathing for 10 seconds or longer during sleep
- Tuberculosis – pulmonary infection with Mycobacterium tuberculosis; reduces lung compliance

DIGESTIVE SYSTEM

DIGESTIVE SYSTEM – digest foods

extracellular (outside of cell) in digestive canal



BASIC PROCESSES OF THE DIGESTIVE SYSTEM

- **INGESTION -- intake of food**
- **DIGESTION – breakdown of food**
  - **Mechanical Digestion – physical breakdown**
  - **Chemical Digestion – chemical breakdown of macromolecules to monomers**
- **Absorption -- Transport of the products of digestion into the blood**
- **Defecation -- Elimination of undigested waste**

ORGANS OF DIGESTIVE TRACT (mouth to anus)

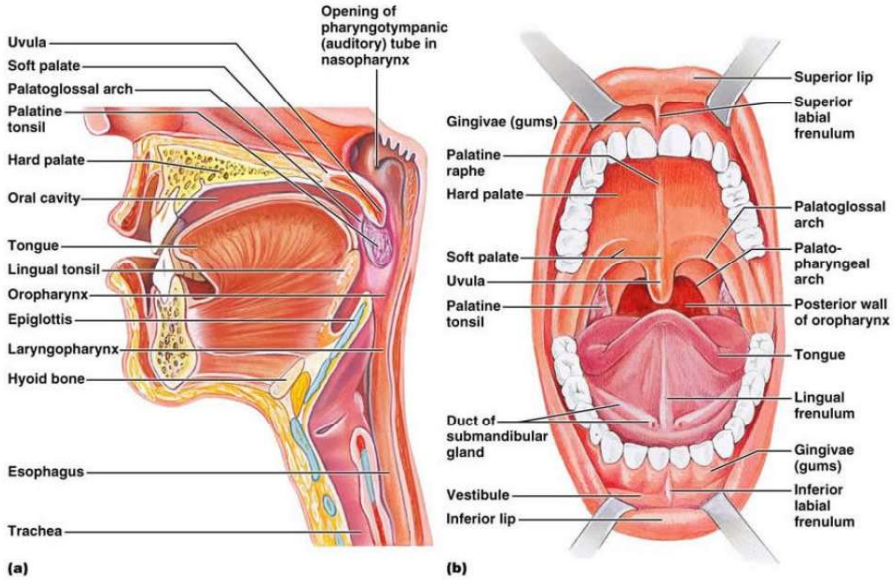
- Mouth - Chewing, Digestion begins
- Pharynx - Swallowing
- Esophagus - Transports food to stomach
- Stomach - Storage of food, Digestion of protein
- Small Intestine - Majority of digestion and absorption of food
- Large Intestines - Absorption of water, Waste storage
- Anus - Elimination of waste

ASCESSORY ORGANS

SECRETE FLUIDS INTO DIGESTIVE TRACT

- Salivary Glands - Secrete salivary amylase
- Liver - Produces bile
- Gallbladder - Storage of bile
- Pancreas - Secretes pancreatic amylase and other digestive enzymes

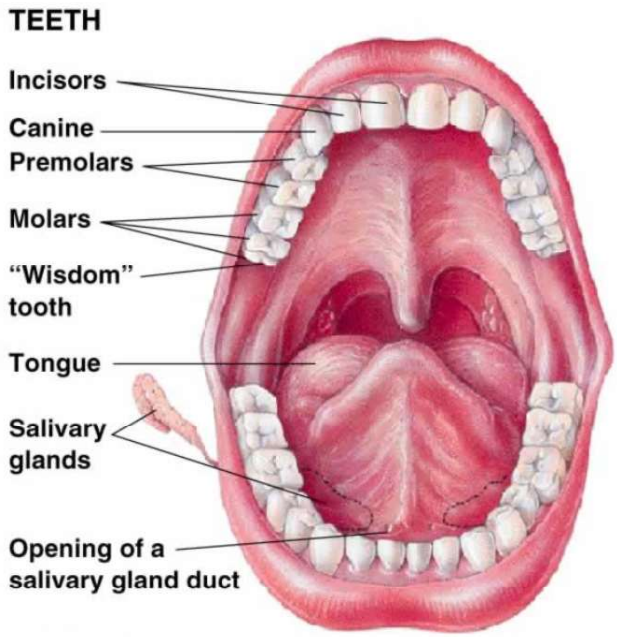
MOUTH



- Opens to outside to facilitate feeding
- Aids in preparation of food for digestion
- Foods are broken down mechanically by chewing
- Saliva is added as a lubricant from the auxiliary saliva glands
- Saliva contains amylase, an enzyme that digests starch
- Serves as an organ for speech and pleasure
- Includes cheeks, lips, tongue, palate, teeth – primary & secondary

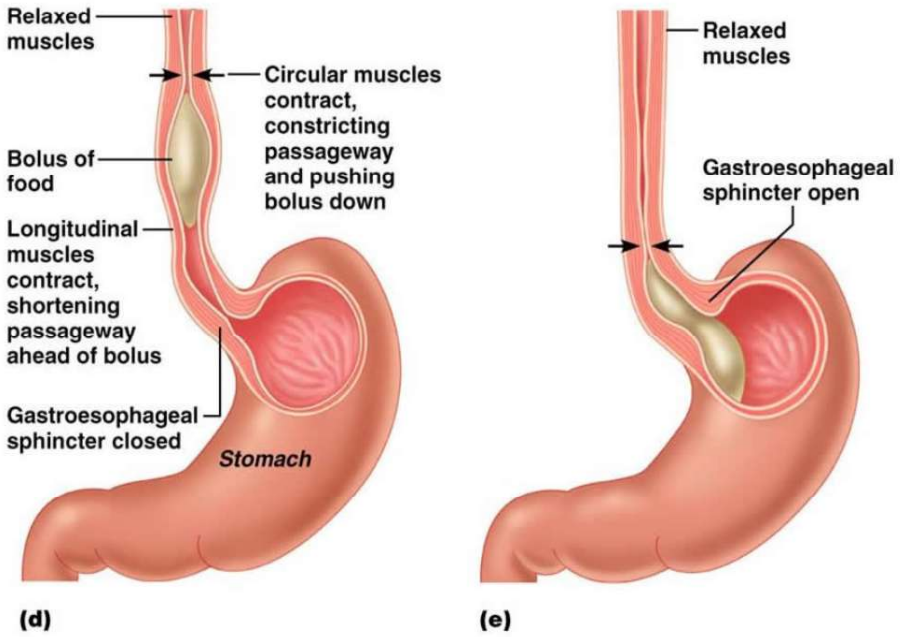
TEETH

- *Incisors (8)* – for biting food
- *Canines (4)* - for grasping and tearing food
- *Bicuspids (8)* – for grinding and crushing food
- *Molars (12)* – for grinding food

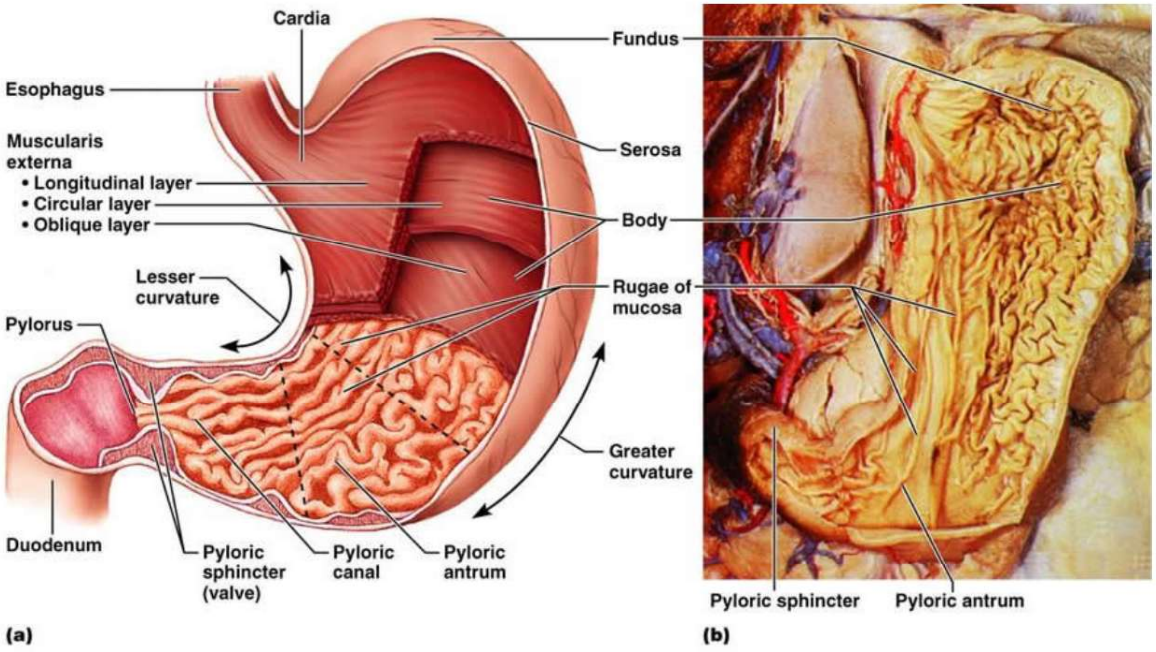


ESOPHAGUS

a simple tube between the mouth and stomach – peristalsis aides in swallowing



STOMACH

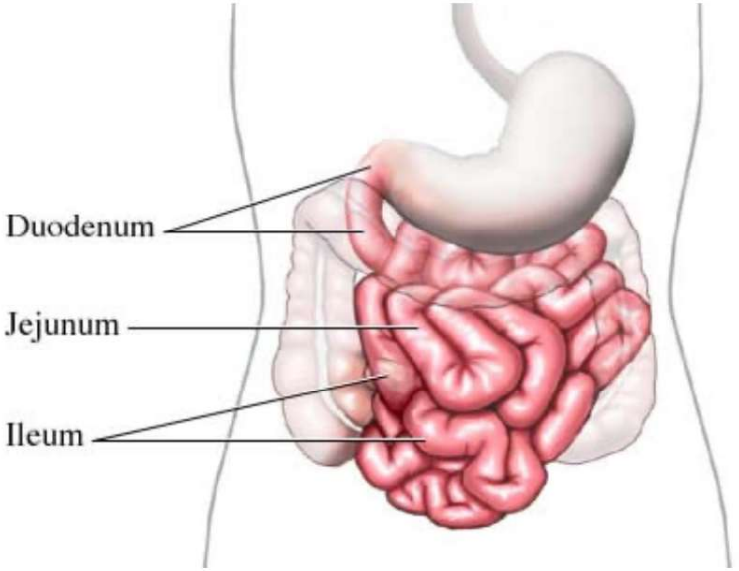

Cardia
Fundus
Esophagus
Muscularis externa
• Longitudinal layer
• Circular layer
• Oblique layer
Serosa
Body
Lesser curvature
Rugae of mucosa
Pylorus
Greater curvature
Duodenum
Pyloric sphincter (valve)
Pyloric canal
Pyloric antrum
(a)
Pyloric sphincter
Pyloric antrum
(b)

STOMACH

- Enzyme digestion of proteins initiated
- Foods reduced to a liquid form
- Walls lined with millions of gastric glands
- Several kinds of cells in gastric glands
- Very little absorption from stomach – some water, ethanol, drugs as aspirin, and certain ions

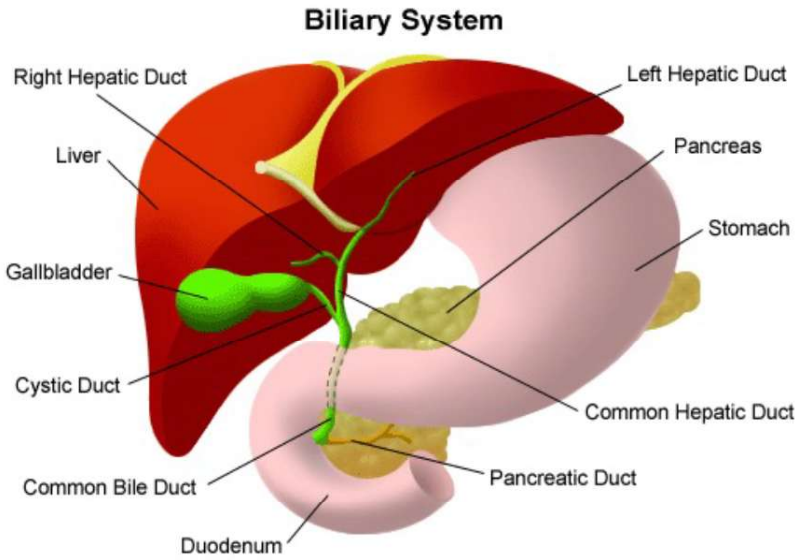
SMALL INTESTINE

- most of chemical enzymatic digestion occur
- almost all nutrients are absorbed
- Accessory glands – liver, gall bladder, and pancreas provide secretions to assist with chemical enzymatic digestion



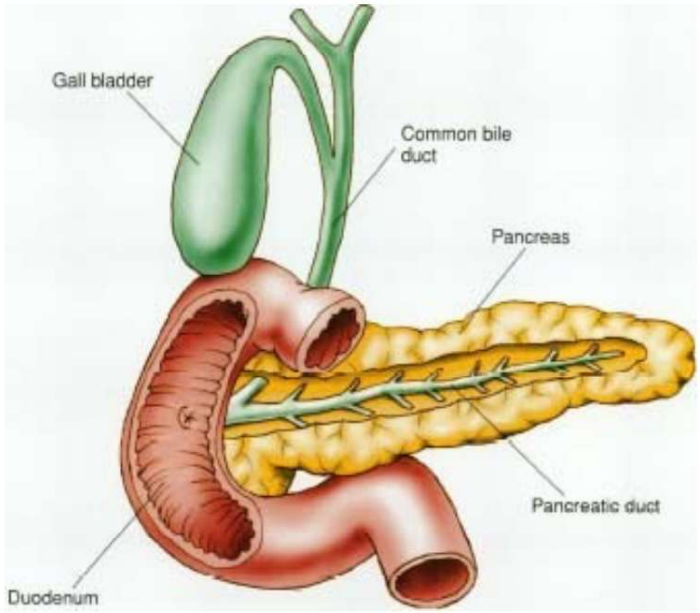
LIVER and GALL BLADDER

- **Liver**: - provides bile salts to the small intestine, which are critical for digestion and absorption of fats.
- **Gallbladder** – stores bile



PANCREAS

- **Pancreas: - provides digestive enzymes to the small intestine which are critical for digestion of fats, carbohydrates and protein.**



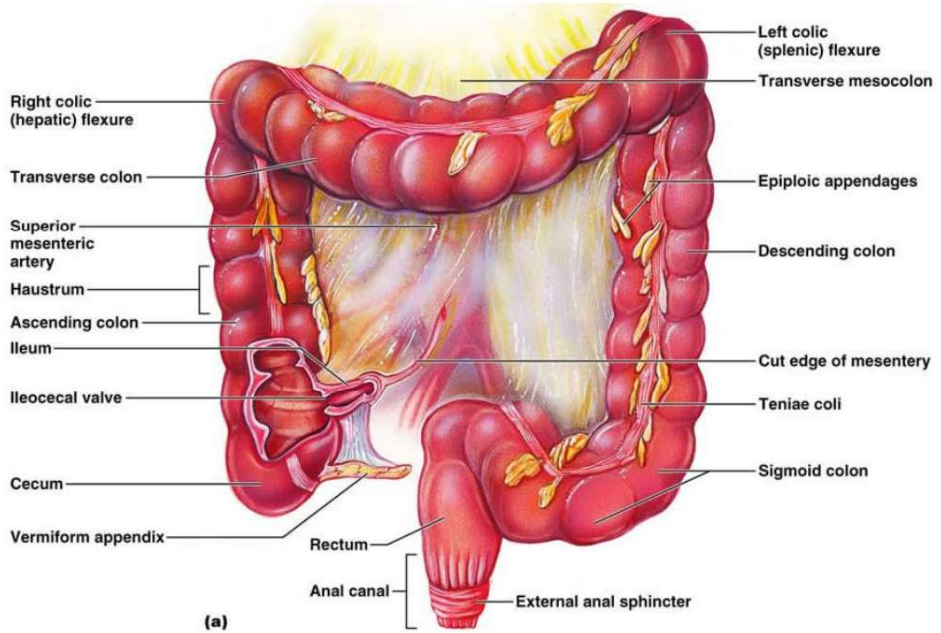
LARGE INTESTINES

Colon:

- liquid residue – mainly water with undigested materal
- water is absorbed,
- bacterial fermentation takes place
- feces are formed.

Rectum: collects undigested waste

Anus: expels undigested waste – muscles to control exit and prevent leakage.

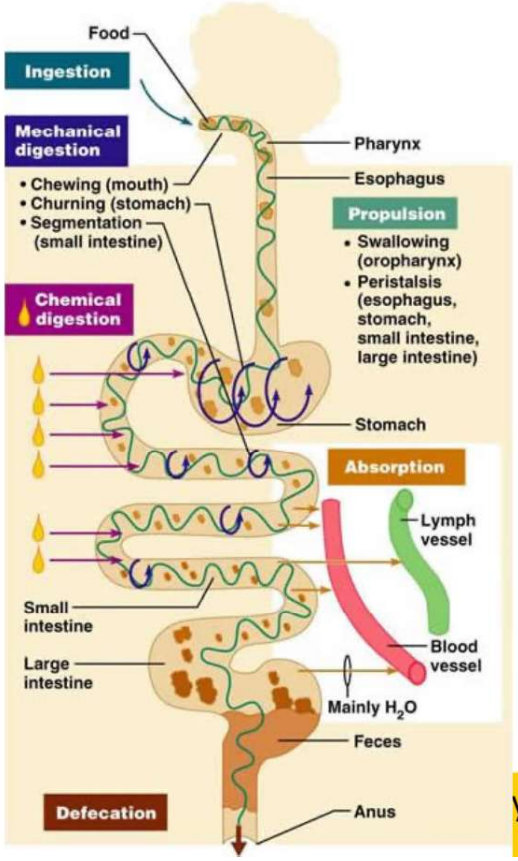


DIGESTIVE PROCESS

- Ingestion – intake of food
- Digestion – breakdown of food bit by bit into molecules small enough to be absorbed

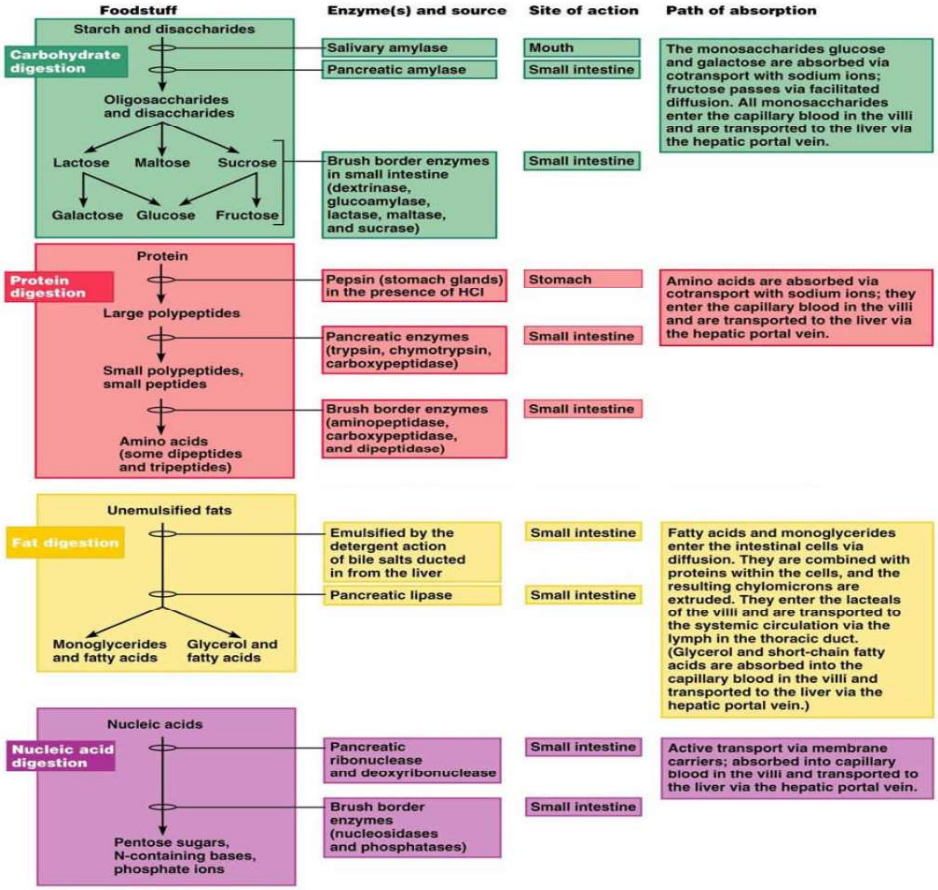
  Mechanical Digestion – physical breakdown of food

  Chemical Digestion – chemical breakdown of macromolecules to monomers
- Absorption – transport of productions into the blood
- Elimination (Defecation) - elimination of undigested waste



CHEMICAL DIGESTION

- CARBOHYDRATES
- PROTEIN
- FATS
- NUCLEIC ACIDS



Common Disorders of Digestive System

- Stomach and duodenal ulcers
- Cancers of the digestive system
- Diarrhea
- Lactose Intolerance
- Hepatitis
- Crohn's Disease, GERD, Diverticular Disease, Celiac Disease (National)

Role of Fiber in Digestion

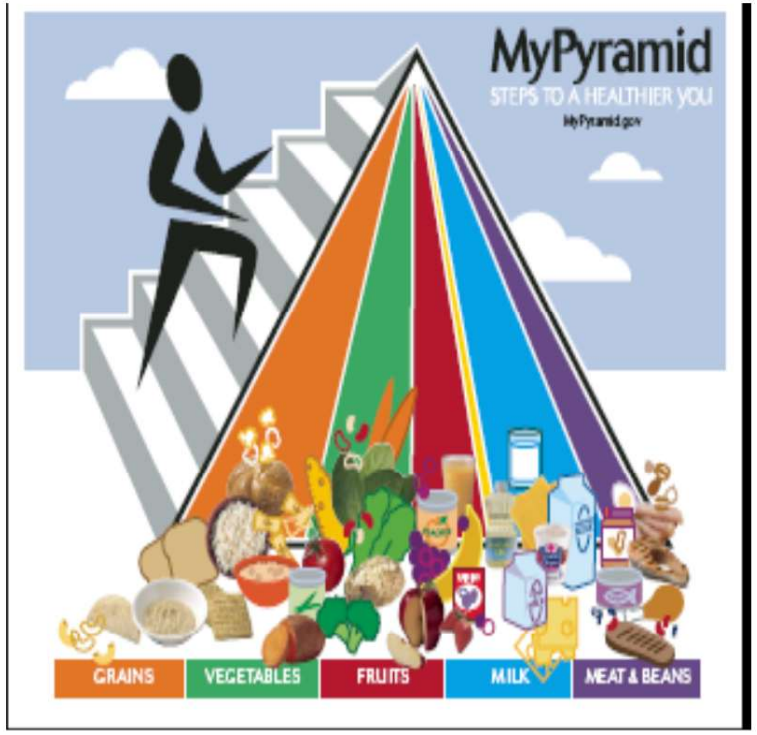
- Fiber is found mostly in plant
- There are two types – insoluble fiber and soluble fiber
- Insoluble fiber is a type of fiber which cannot be dissolved in water
- Insoluble fiber draws water to the intestine, increasing the bulk and softness of waste products
- Soluble fiber which can be dissolved in water
- Soluble fiber can be digested slowly and it slows the digestive process and keeps the stomach fuller longer leaving the body feeling full for a longer period of time
- Digestion and absorption of carbohydrates are slower so that glucose (sugar) in food enters the bloodstream more slowly, which keeps blood sugar on a more even level
- The slow absorption of sugar gives the body an opportunity to regulate blood sugar levels

New Food Group Pyramid

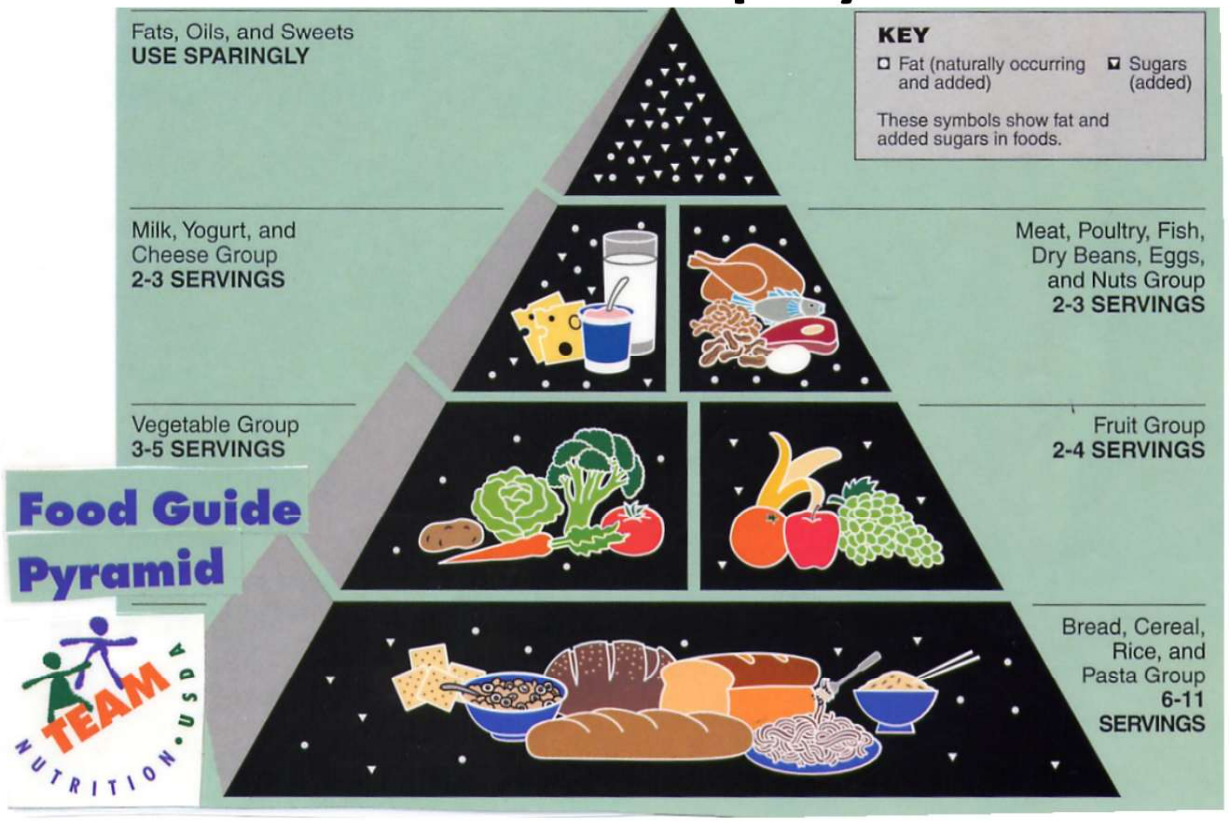
2000 calorie diet

- Grains – 6 oz daily
- Vegetables – 2 ½ cups daily
- Fruits – 2 cups daily
- Milk – 3 cups daily
- Meats and Beans –

  5 ½ oz daily



Old Food Group Pyramid



Food Labels

- Serving Size
- Serving per container
- Calories
- Nutrients – g and % Daily values
- Vitamins and Minerals-

  % Daily Values
- 2000 calorie diet

Sample label for
Macaroni & Cheese

Nutrition Facts

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

Amount Per Serving	
Calories 250	Calories from Fat 110
	% 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 diet. Your Daily Values may be higher or lower depending on 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	2,400mg
Total Carbohydrate		300g	375g
Dietary Fiber		25g	30g

Nutritional Disorders

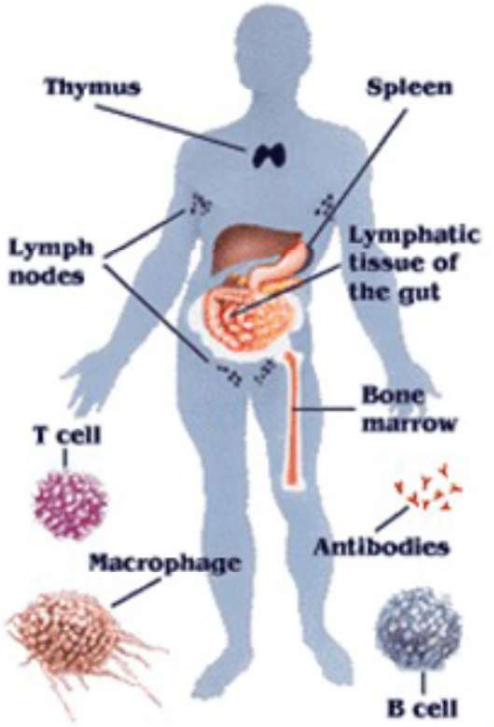
- Suggested in the rules
  - Xerophthalmia
  - Pernicious Anemia
  - Scurvy
  - Rickets
  - Vitamin-poisoning
- Other common disorders or conditions
  - Osteoporosis
  - Obesity
  - Anorexia
  - Bulimia

IMMUNE SYSTEM

Immune System

The body's defense against:

- **disease causing organisms**
- **malfunctioning cells**
- **foreign particles**

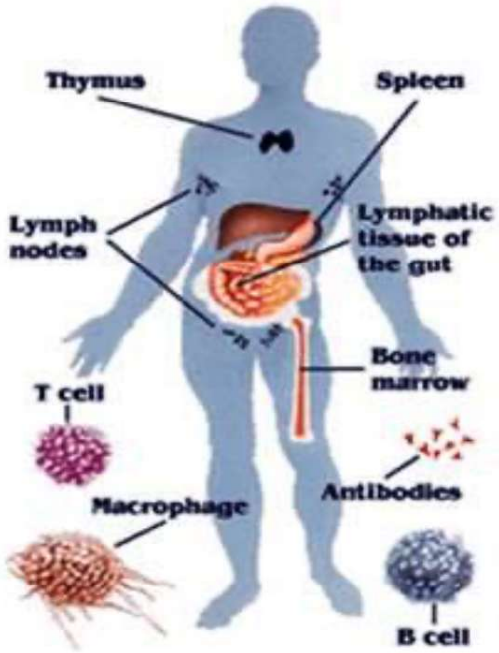


Basic Immunology

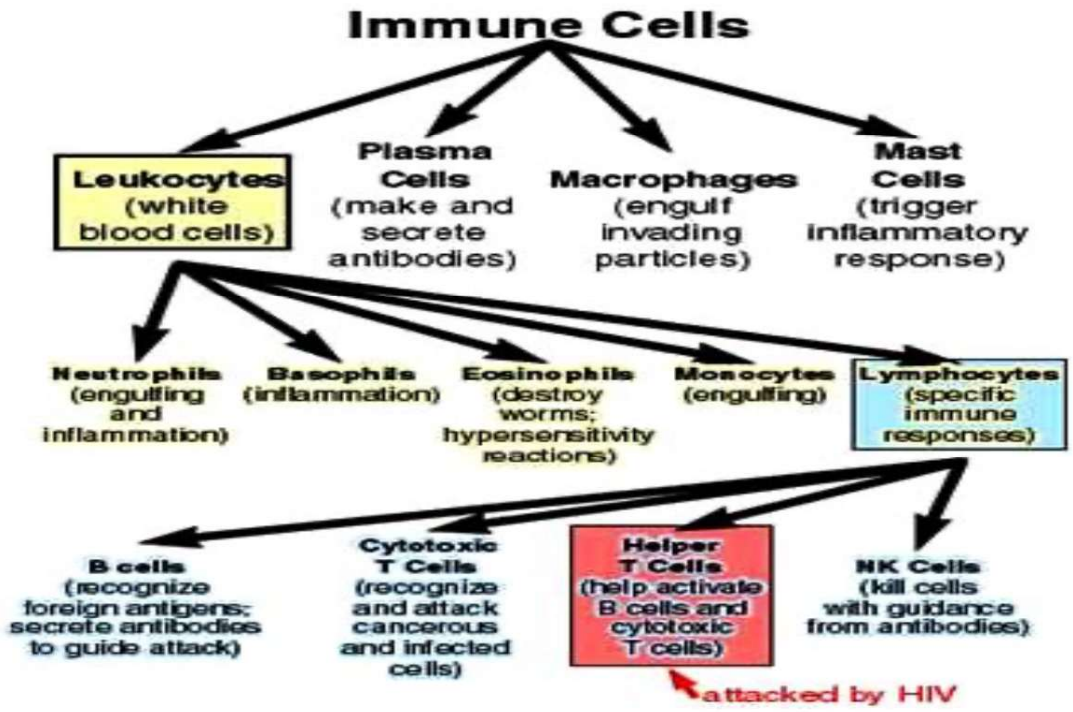
- Depends on the ability of the immune system to distinguish between ***self*** and ***non-self*** molecules
- ***Self*** molecules are those components of an organism's body that can be distinguished from foreign substances by the immune system
  - ***Autoimmunity*** is an immune reaction against self molecules (causes various diseases)
- ***Non-self*** molecules are those recognized as foreign molecules
  - One class of non-self molecules are called **antigens** (short **for *anti*body *gen*erators**) and are defined as substances that bind to specific immune receptors and elicit an immune response

Immune System Components

- specific cells - lymphocytes, macrophages, etc., originate from precursor cells in the bone marrow and patrol tissues by circulating in either the blood or lymphatics, migrating into connective tissue or collecting in immune organs
- lymphatic organs- thymus, spleen, tonsils, lymph nodes
- diffuse lymphatic tissue -collections of lymphocytes and other immune cells dispersed in the lining of the digestive and respiratory tracts and in the skin

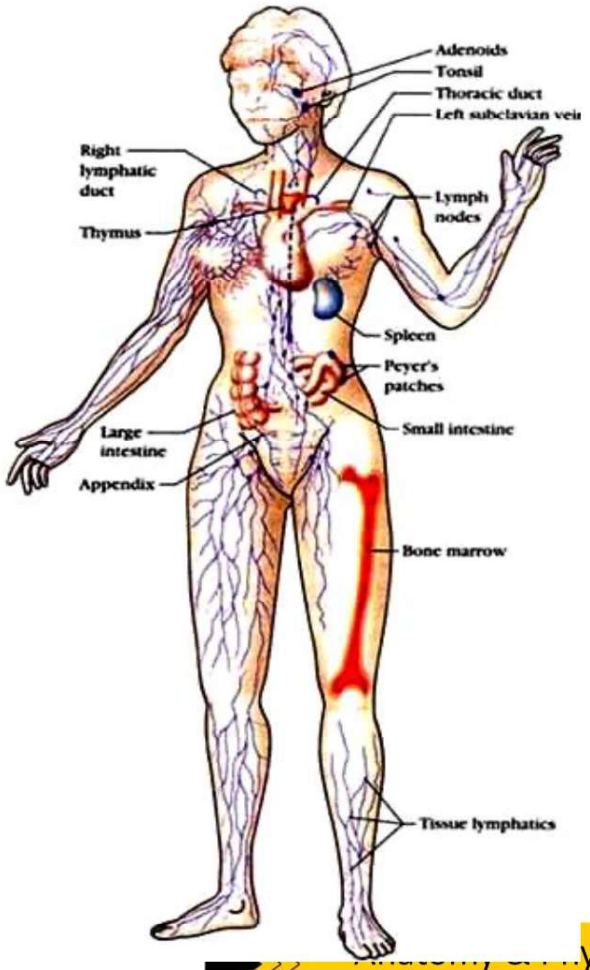


Types of Cells



Lymphatic Organs

- Lymph Nodes
- Spleen
- Thymus
- Red Bone Marrow
- Immune Tissue in Organs – GALT, MALT, SALT



Plan of Protection

- *Immunity* is the ability to defend against infectious agents, foreign cells and abnormal cells eg. cancerous cells
  - 1st Line of defense – Block entry
  - 2nd Line of Defense – Fight Local Infections
  - 3rd Line of Defense – Combat Major Infections

Nonspecific Response

- Responds quickly, fights all invaders and consists of:
- *First line of defense* – intact skin and mucosae and secretions of skin and mucous membranes prevent entry of microorganisms
- *Second line of defense* – phagocytic white blood cells, antimicrobial proteins, and other cells
- Inflammatory response process is key
- Inhibit invaders from spreading throughout the body

First line of Defense

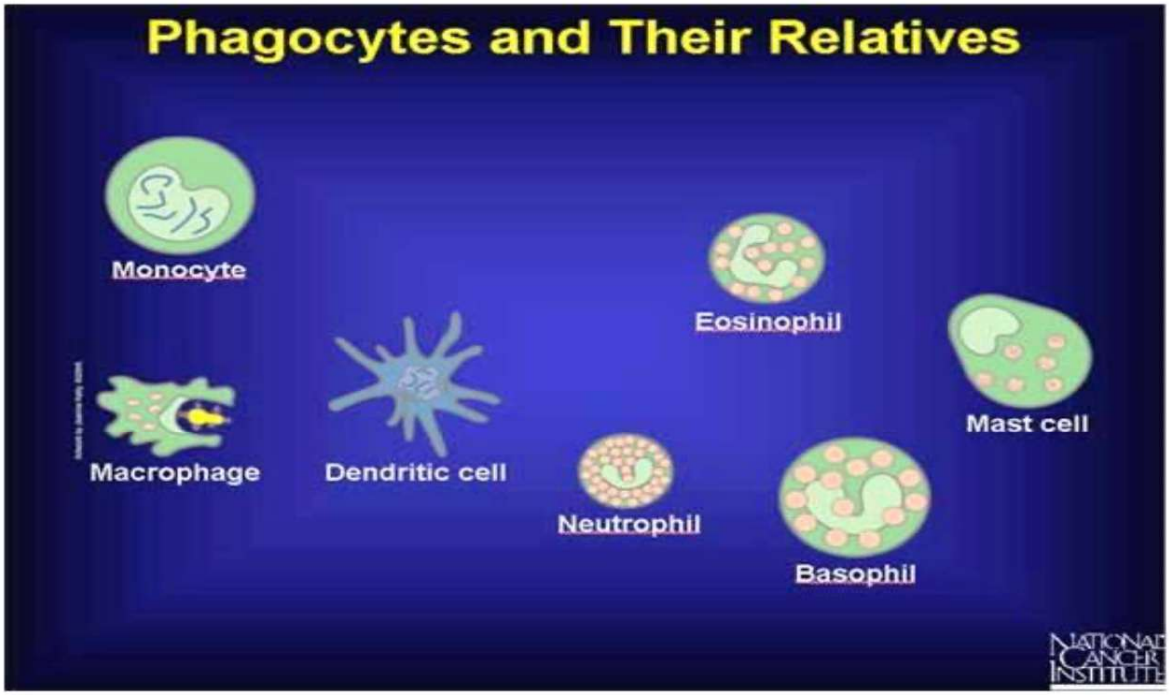
- Non specific barriers to block entry
- Skin – physical & chemical barrier
- Mucous membranes
- Nasal hairs and microscopic cilia
- Gastric juice, vaginal secretions & urine
- Natural flora
- Tears, saliva and sweat glands
- Cerumen or Ear Wax

Second Line of Defense

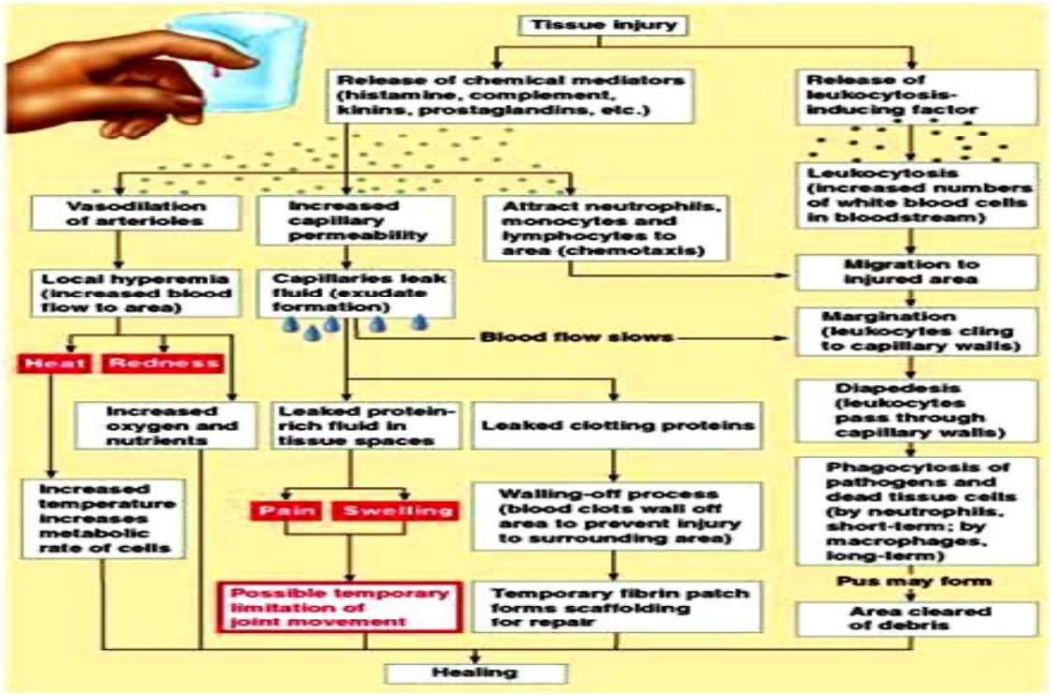
- Fight local infection with Inflammation Process
- Response is a non-specific, immediate, maximal response
- Consists of phagocytosis, complement protein response
- Involve the Inflammation Process



Phagocytes and Their Relatives



Inflammation Process



Third Line of Defense

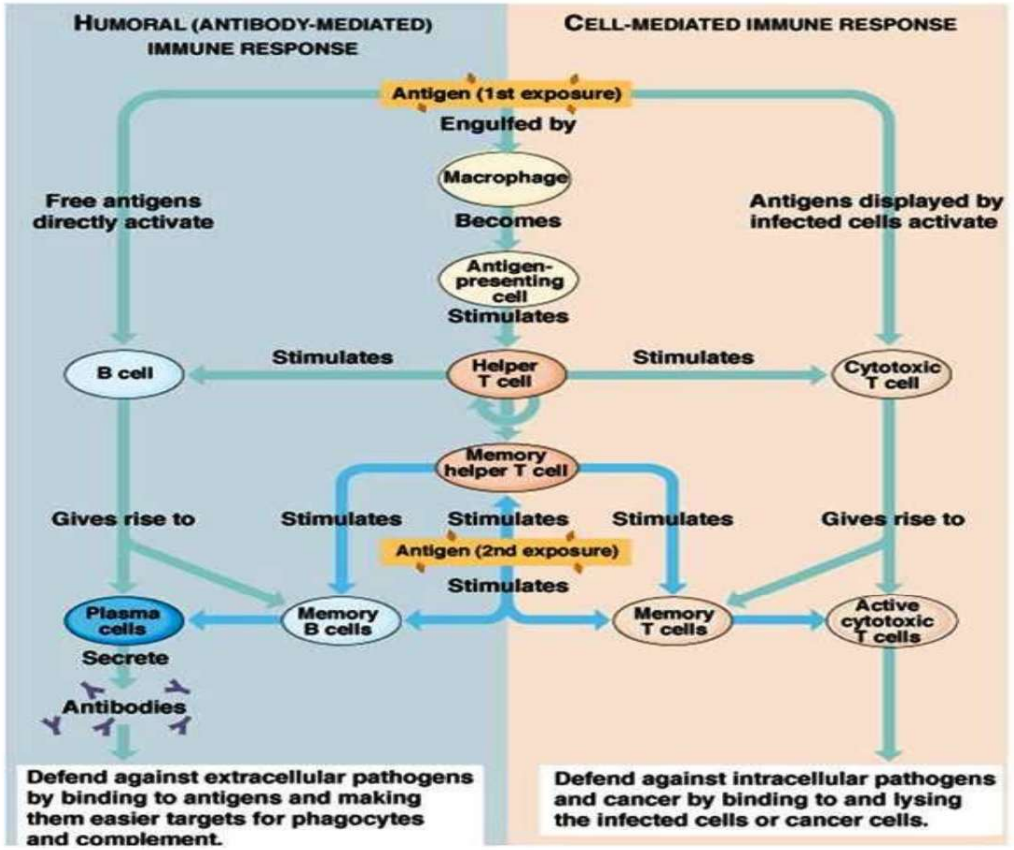
Specific Response

- takes longer to react
- work on specific types of invaders which it identifies and targets for destruction
- not restricted to initial site of invasion/infection – whole body protection
- a stronger immune response as well as immunological memory

Antigens

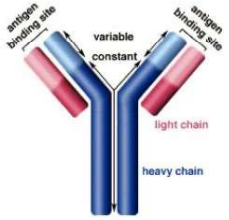
- Antigens are proteins or carbohydrate chain of a glycoprotein within a plasma membrane which the body recognizes as “nonself”
- antigen presentation - specific immune response is antigen-specific and requires the recognition of specific “non-self” antigens

Specific Defense



Humorial – Antibody (Extracellular Response)

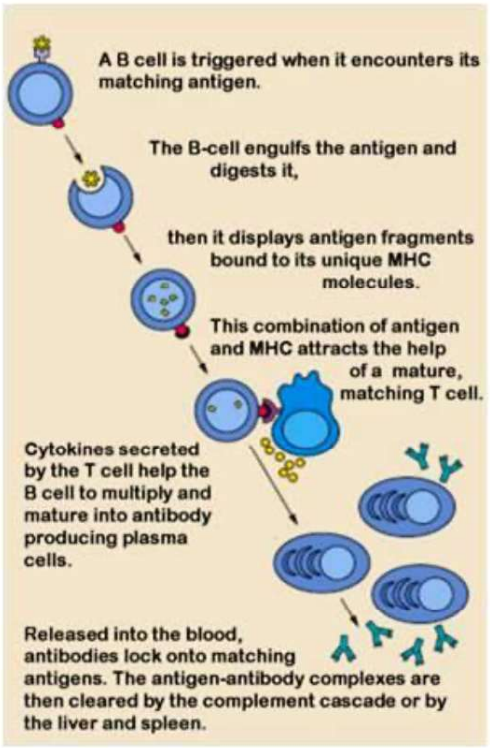
- B cells
- Plasma Cells -produce antibodies



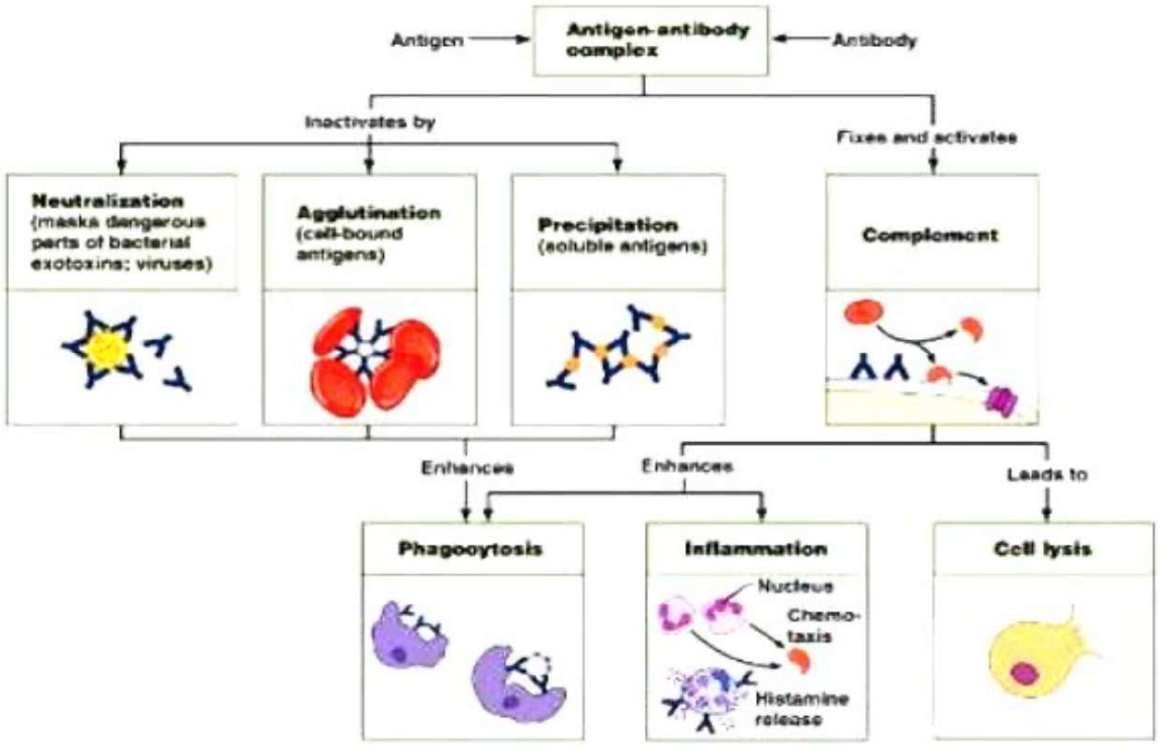


Antibo
Complex

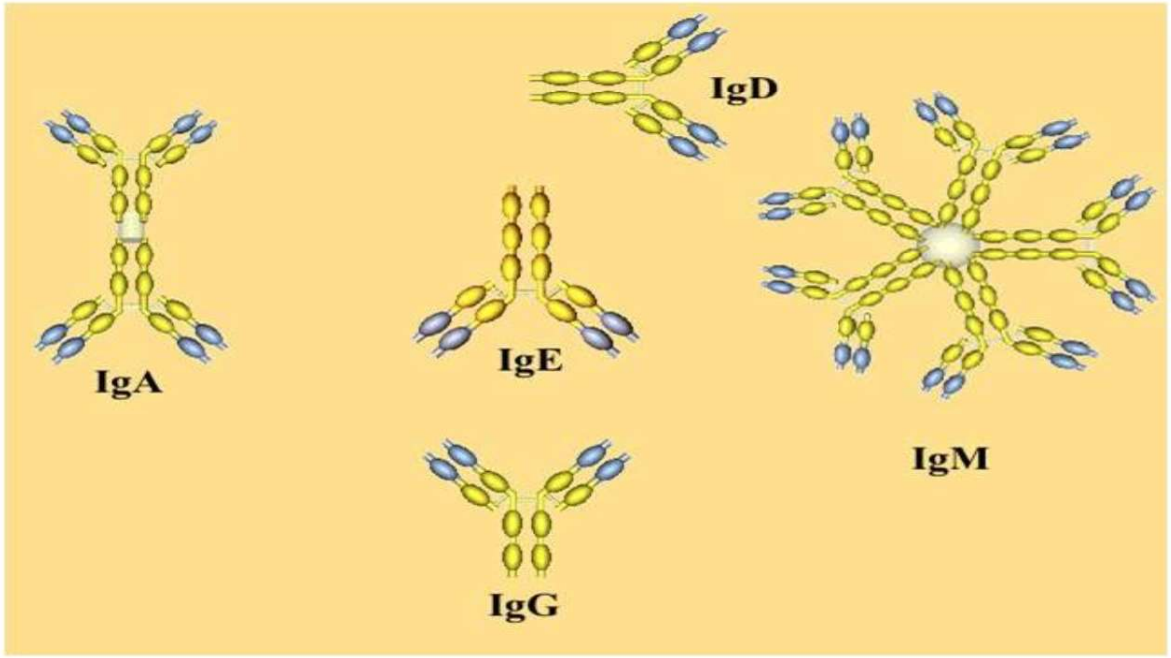
- Helper T Cells
- Memory Cells



Antigen-Antibody Complex Functions



Classes of Antibodies

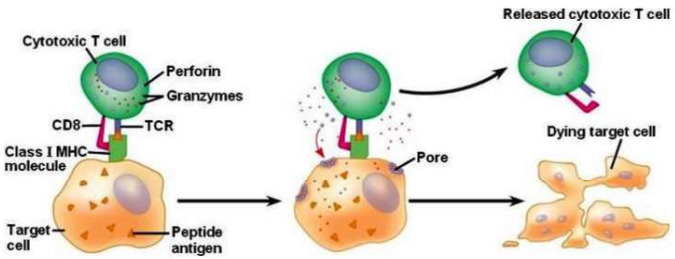


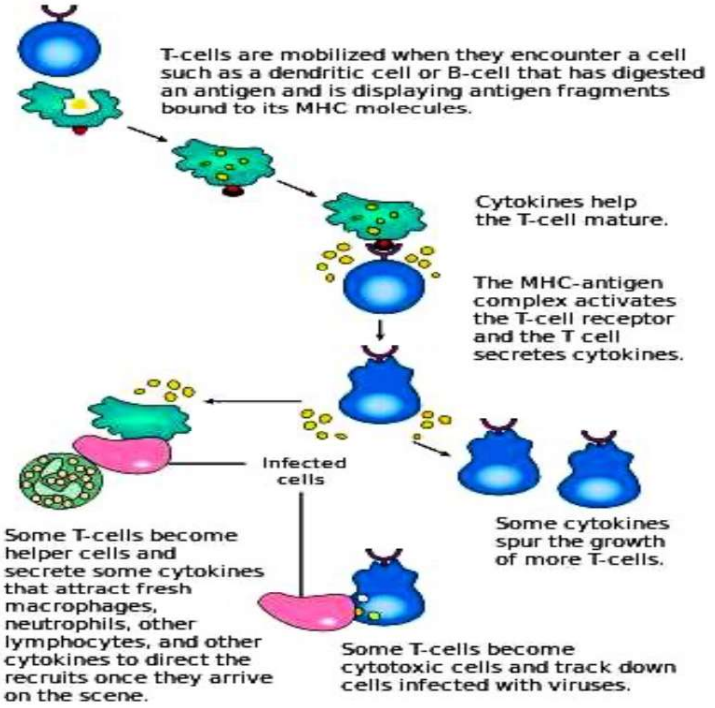
Classes of Antibodies

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.**

Cell-mediated immune response

Within the cell
involves the activation of phagocytes, antigen-specific cytotoxic T-lymphocytes, and the release of various cytokines in response to an antigen

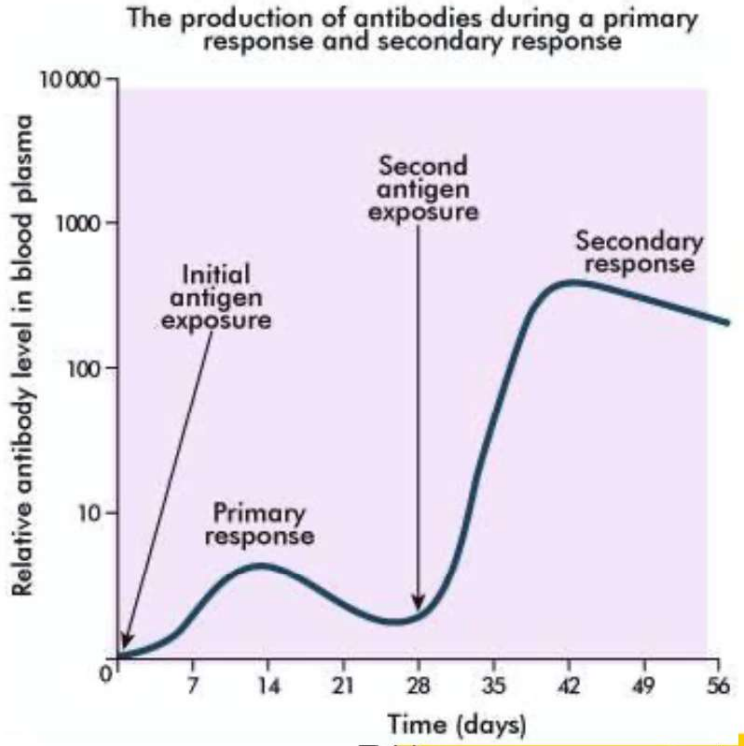




Memory B & T Cells

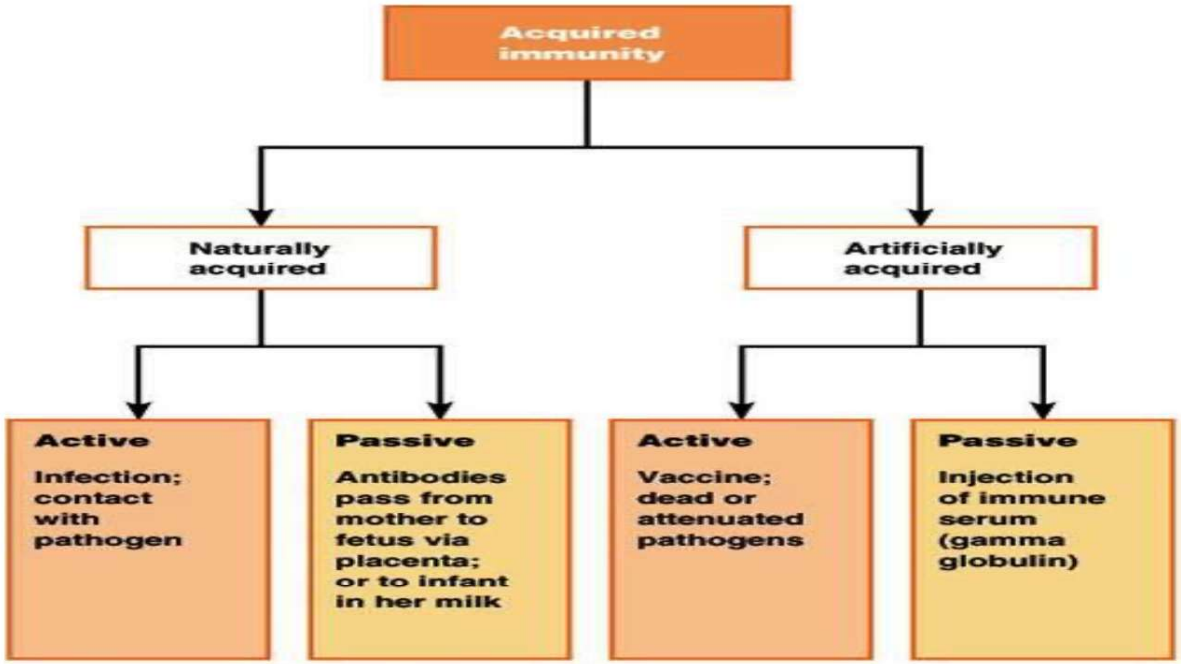
Should a pathogen infect the body more than once, these specific memory cells are used to *quickly* eliminate

Primary & Secondary Immunity

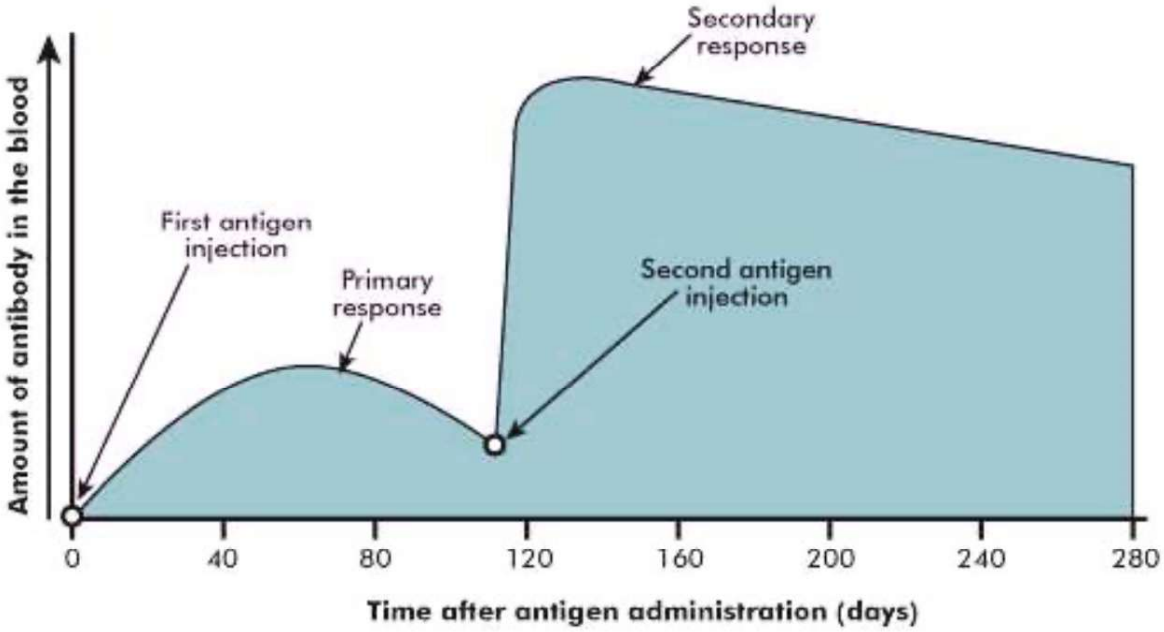


Sources of Specific Immunity
Inborn & Acquired

- Inborn Immunity – immunity for certain diseases is inherited
- Acquired Immunity – immunity can be acquired through infection or artificially by medical intervention



Immunization



Antibiotics and Antivirals

- Antibiotics or antibacterials – group of medications used to kill bacteria by preventing them from dividing
- There is concern about the extensive use of antibiotics resulting in resistant forms of bacteria and "superbugs"
- Antivirals – group of medications used to treat viral infections but they cannot destroy the virus. Rather they inhibit the virus from reproducing and developing.

Cultured Antibodies

- **Monoclonal antibodies – cloning of many copies of the same antibody which can be useful in fighting diseases because they can be designed specifically to only target a certain antigen, such as one that is found on cancer cells.**

Allergies

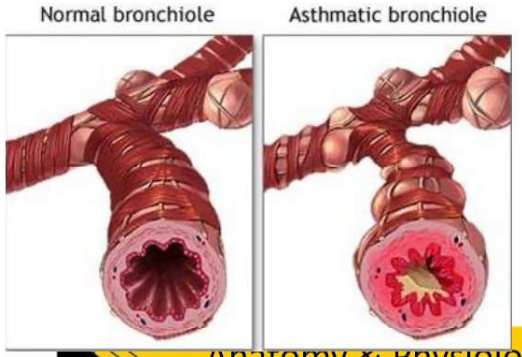


- Hypersensitivity of the immune system to relatively harmless environmental antigens - the immune system reacts to an outside substance that it normally would ignore
- Allergy types (food, dust, mold, seasonal), symptoms and signs (skin rash, itching, red bumps, sneezing).

Asthma

- an obstructive pulmonary disorder characterized by recurring spasms of muscles in bronchial walls accompanied by edema and mucus production which make breathing difficult
- it causes the airways of the lungs to swell and narrow, leading to wheezing, shortness of breath, chest tightness, and coughing





AIDS -HIV

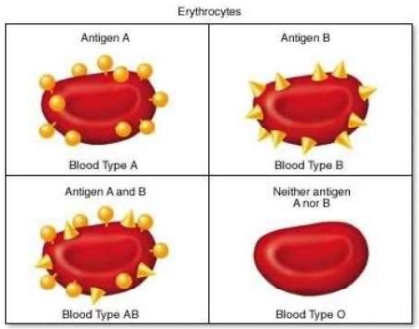
- AIDS - (acquired immune deficiency syndrome) is the final stage of HIV disease, which causes severe damage to the immune system-caused by infection with human immunodeficiency virus (HIV)- HIV infects vital cells in the human immune system such as helper T cells, macrophages, and dendrite cells

Autoimmune Disorders

- Condition that occurs when the immune system mistakenly attacks and destroys healthy body tissue
- The immune system can't tell the difference between healthy body tissue and antigens. The result is an immune response that destroys normal body tissues
- More than 80 different types – Multiple sclerosis, Rheumatoid arthritis, Systemic lupus erythematosus

ABO Antigens

- The surface membranes of RBCs carry proteins that act as antigens in some recipients
- Type A blood has A antigens only.
- Type B blood has B antigens only.
- Type AB blood has both A and B antigens present
- Type O blood lacks both A and B antigens
- Blood plasma contains antibodies to the blood types not present.
- Exposure to foreign blood antigens results in agglutination or clumping of RBCs, prevents circulation of blood, and the RBCs burst



RH Factor

- Another important antigen used in matching blood types
- Persons with Rh factor on RBC membrane are Rh positive; Rh negative lack the Rh factor protein.
- Rh negative individuals do not automatically have antibodies to Rh factor but develop immunity when exposed to it.
- Hemolytic disease of the newborn (HDN) can occur when mother is Rh negative and baby is Rh positive

Wiki - Scioly.org

Anatomy/Digestive System

The **Digestive System** is a topic of the event Anatomy and Physiology. It is part of the event during the 2027, 2023, and 2018 seasons, along with the Respiratory and Immune Systems.

For a list of secretions in the digestive system, see the Digestive Secretion List.

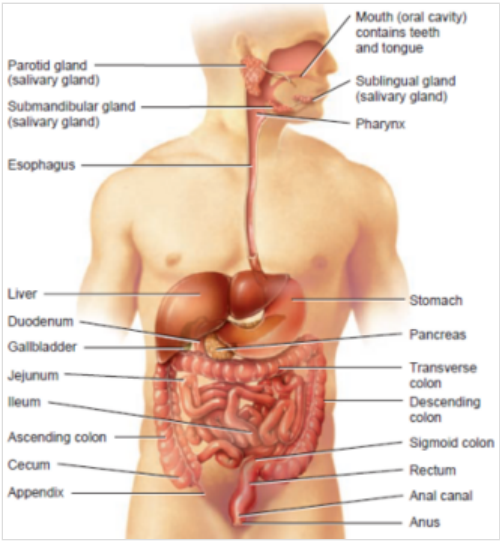
Overview and Functions

The goal of the digestive system is to process or digest food in the body. Food is any material consumed for nutrition. It provides the body with essential molecules and elements that can be utilized elsewhere in the body. There are four main processes that occur in this system - ingestion, digestion, absorption, and elimination. Ingestion is the taking in of food, and digestion involves breaking it down. Absorption moves the food into the bloodstream, though it does not involve actually taking those absorbed nutrients and making them a part of the body. The usage of the absorbed molecules is known as assimilation. Finally, waste is removed from the body through elimination.

The digestive system is made up of two groups of organs - those in the **alimentary canal** and **accessory organs**. Members of the alimentary canal include the esophagus and the intestines. These organs make up the continuous tube that passes from the mouth to the anus. Accessory organs such as the salivary glands and the pancreas provide aid to those organs in the alimentary canal by producing enzymes or performing other actions.

Summary of the Digestive Tract

The digestive tract is about 8 meters, or 28 feet, long. It can take 15 to 72 hours for food to pass from the mouth to the anus.



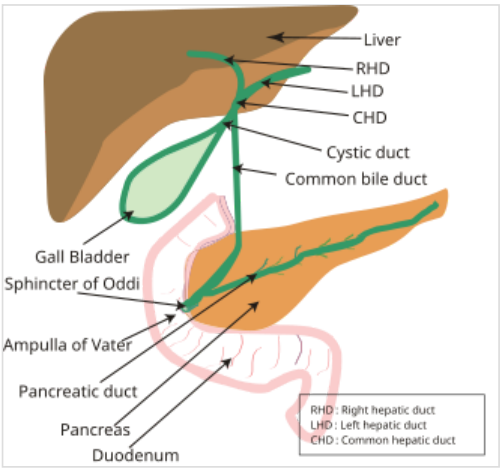
The digestive system

The mouth is where the digestion of food starts. The mouth is a good example of mechanical digestion and chemical digestion at work. The teeth chomp, chew, and tear food down into smaller pieces (mechanical digestion). The molecular structure of the food does not change in mechanical digestion. Then, the salivary glands secrete saliva into the mouth, breaking down starches into dextrin and maltose (chemical digestion). Saliva is a form of chemical digestion because it changes the molecular structure of the food. These digestive processes create food boluses.

After food boluses are developed in the mouth, they go down the **pharynx**. The pharynx is a shared connection between the nasal cavities, the mouth, the air tract, and the esophagus. Therefore, there must be a structure to prevent the boluses from entering the trachea (passage to the lungs). This is why we have an epiglottis. The epiglottis is a small flap which can open and close the entrance to the trachea. When food boluses travel through the pharynx, the epiglottis folds down to protect the airway. However, in instances such as talking or eating, the epiglottis can get confused. In order to talk, the epiglottis must be open. In order to swallow, the epiglottis must be closed. Therefore when attempting both tasks, the epiglottis might malfunction and this could result in choking.

After the pharynx, food bolus is transported to the stomach via the **esophagus**/oesophagus. The esophagus is a muscle-walled tube that goes from the throat to the lower digestive tract. To help food get down, muscular contractions called peristalsis push food down towards the stomach. To demonstrate peristalsis motions, imagine a sphere in a rubber tube. The diameter of the sphere is a little more than the diameter of the tube. To move the ball out, one would squeeze the tube from

one end, and slowly move his/her hand towards the ball while squeezing to push it out. The reason peristalsis is important is when digestion is working against gravity. For example, when eating upside down, the food bolus is still able to move to the stomach because of peristalsis contractions.



A more detailed look at the biliary system

Next in the digestive tract comes the **stomach**. When we think of digestion, the first thing that comes to mind is the stomach. It acts like a waiting room for food bolus. While the food boluses are waiting, the stomach breaks the food down into a liquid-like mixture by churning the food (mechanical digestion) and secreting gastric acid (chemical digestion). Gastric acid also kills bacteria that may be in the food.

When food boluses are ready to be further digested, they transfer to the **duodenum** by contractions of the stomach walls. The duodenum is the first part of the small intestine and is about 25 cm in length. The duodenum is stationary and is fixed behind sheets of connective tissue called peritoneum. Glands in the duodenum secrete a thick alkaline fluid that counteracts the acidic nature of the chemicals the food bile has absorbed. The gallbladder also secretes pancreatic juice and bile into the duodenum.

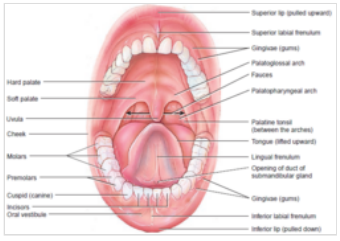
The **liver** is a very important part of the digestive system, even though food does not directly pass through it. The liver produces bile (a greenish fluid which aids in the digestion of fats), which is stored in the gallbladder. When bile is needed, it joins with secretions from the pancreas and goes through the common bile duct into the duodenum.

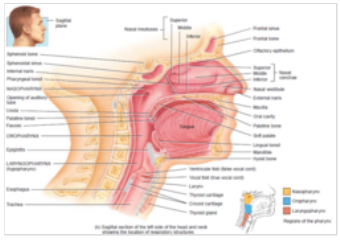
After the duodenum comes the two other parts of the small intestine, the **jejunum** and the **ileum**. These are what make up the bulk of the small intestine. The jejunum is the first part while the ileum is the second part. Here, food boluses are digested even further, and some nutrients are absorbed through the walls. The ileum is the longest portion of the small intestine.

After passing through the duodenum, jejunum, and the ileum (the small intestine), food bolus makes its way through the **large intestine**. The large intestine is actually shorter than the small intestine, but larger in diameter. Water and electrolytes are removed in the large intestine. Also, microbes such as bacteria aid in further digestion. Finally, food bolus comes out of the anus as feces.

In-Depth Descriptions

The Oral Cavity


Front view of the oral cavity

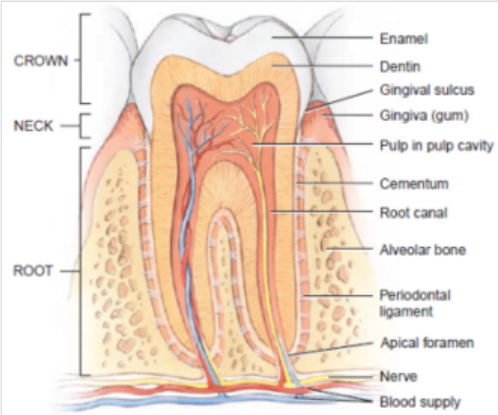

Side view of the oral cavity

The **oral cavity** is another name for the mouth. It is the first part of the digestive tract.

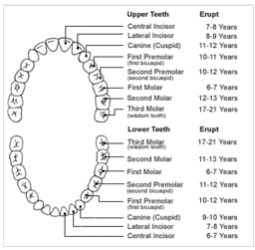
- The **lips**, formed by the orbicularis oris muscles, help in food intake and determining temperature of food.
- The **buccinator muscles** assist in the process of **mastication**, or chewing. Mastication is the first process of digestion.
- The **tongue**, a large muscle, helps move the food around the oral cavity. It also is necessary in swallowing. Swallowing is also called deglutition.
- The surface that forms the top of the oral cavity is called the palate. It is split into two parts.

- The **hard palate** is the most anterior section. It contains bone. This is often referred to as the "roof of the mouth".
- The **soft palate** is more posterior. It separates the oral cavity from the nasal cavity. It helps by preventing food from entering the nasal cavity.

- The **tonsils** are part of the lymphatic system. They are tissues to the right and left of the mouth.
- The **uvula** is also part of the lymphatic system. It is an extension of the soft palate, and is hanging down from it. The uvula is often wrongly referred to as the tonsil.



Cross section of a tooth



A labeled diagram of all teeth

Teeth

The **teeth** chew and break down the food mechanically. A child has 20 deciduous teeth while an adult has 32 permanent teeth. There are four types of teeth: Incisors, canines, premolars, and molars.

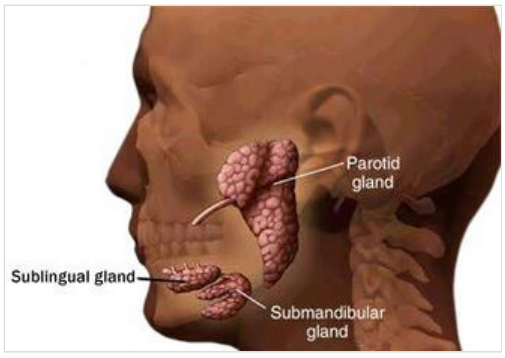
Teeth consist of the **crown**, the **neck**, and the **root**.

- The crown is the upper surface of the tooth. Bumps on the crown are called **cusps**.
- The neck is the region under the crown, but above the alveolar bone.
- The root is the region inside the alveolar bone.

Salivary Glands

The **salivary glands** produce saliva, which is secreted into the oral cavity. There are two types of salivary glands: **serous** and **mucous**. Serous saliva is enzymatic, while mucus acts as a lubricant.

There are three pairs of salivary glands: the parotid, submandibular, and sublingual.



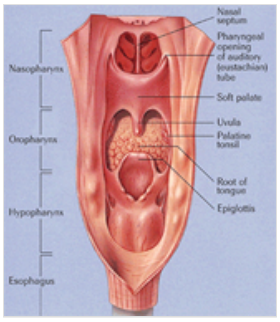
Salivary glands

- The **parotid glands** are the largest. They are located slightly posterior to the cheeks and make 25% of the saliva, though this amount rises to 50% when eating. They are serous.
- The **submandibular glands** are below the mandible bone and make up 70% of salivary production. They produce more serous saliva.
- The **sublingual glands** are the smallest and make 5% of the saliva. They are below the floor of the oral cavity. They produce mostly mucus.

The Pharynx

The **pharynx** is often referred to as the back of the throat. Food pieces, now called bolus, travel from the oral cavity to the pharynx in the process of **deglutition**, or swallowing. The pharynx is also a shared passageway, connecting the nasal cavity, oral cavity, air passageway, and epiglottis. There are certain structures inside the pharynx to conduct the movement of air

and food.



Pharynx

- During deglutition, the soft palate elevates to prevent food from entering the nasal cavity.
- Another structure, the **epiglottis**, bends down to prevent food from entering the air passageway.
- If the epiglottis fails to function, food bolus may be lodged in the air passageway (choking).

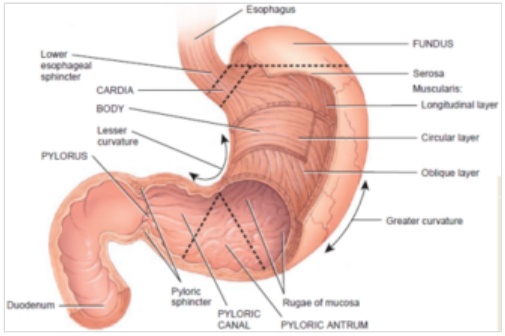
The Esophagus

After food bolus passes through the pharynx, it goes through the **esophagus**, or oesophagus. The esophagus is directly under the pharynx. There is no defining structure that separates the pharynx from the esophagus. The esophagus is about 25 cm long in adults.

Muscular contractions called **peristaltic waves** propel food down the esophagus. This is why food can still travel through the esophagus while the body is lying down or even upside-down.

The Stomach

The stomach is the first part of the gastrointestinal tract. Its main purpose is to store and churn food.



The stomach

- The layers, from innermost to outermost, are the **mucosa** (made of simple columnar epithelium), **submucosa**, **oblique** muscle (helps churn food), **circular** muscle (helps churn food), **longitudinal** muscle (helps churn food), and **serosa** (reduces friction between stomach and neighboring body parts).
- There are three main sections to the stomach: the fundus, the body, and the pylorus. The **fundus** is the top part of the stomach, or the roof. The **body** is the bulk of the stomach. The leftmost region is the **pylorus**.
- The texture of the inner surface of the stomach largely depends on how much food bolus it contains. If it does not contain much food bolus, the stomach is small and the inner surface has lots of folds. These folds are called **rugae**. If the stomach does contain lots of food bolus (like after a meal), the stomach is bigger and rugae are not present.

The Small Intestine

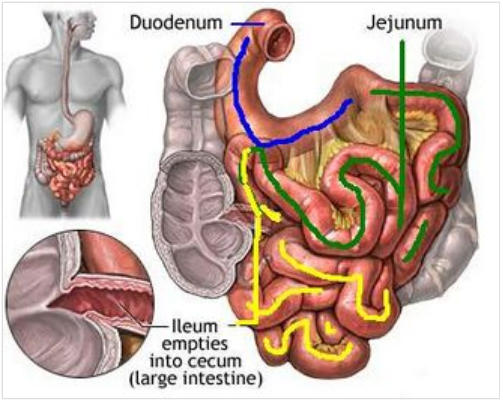
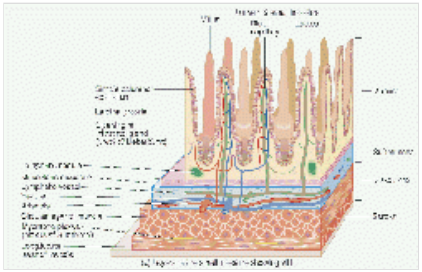
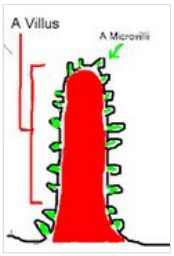


Diagram of the small intestine


Layers of the small intestine

The small intestine is the next part of the digestive tract. The small intestine is about 6 meters long, and so it is the longest part of the digestive tract. Its main purpose is the digestion of food and absorption of nutrients. The small intestine consists of three sections: The **duodenum** (25 cm), the **jejunum** (2.5 m), and the **ileum** (3.5 m).

The layers are, from innermost to outermost, the **mucosa**, **submucosa**, **muscularis - circular**, **muscularis- longitudinal**, and **serosa**.



Villi and microvilli

The mucosa has four major cell types:

- **Absorptive cells** digest and absorb food
- **Goblet cells** produce mucus
- **Granular cells** protect from bacteria
- **Endocrine cells** produce regulatory hormones

Since digestion only occurs at the surface, there must be a lot of surface area for effective digestion. To increase surface area, the small intestine has circular folds, **villi**, and **microvilli**. Villi are tiny finger-like projections. Microvilli are even smaller finger-like projections that are on the villi.

Chyme moves through the small intestine with two types of contractions.

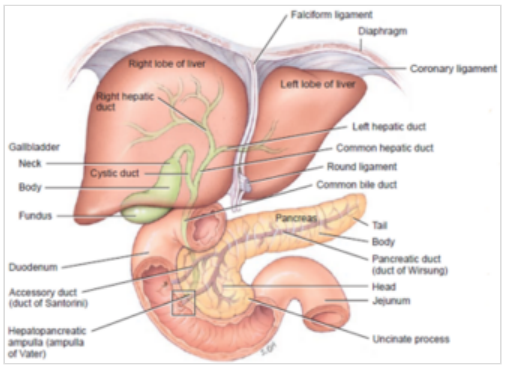
- **Peristaltic contractions** propel chyme forward. They are similar to the contractions in the esophagus.
- **Segmental contractions** mix chyme by squeezing in outward motions. NOTE: Segmental Contractions DO NOT help move food through the GI tract.

The differences between the duodenum, jejunum, and ileum are as follows:

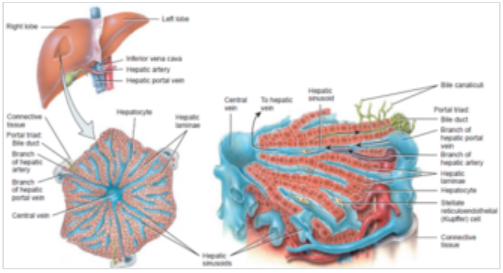
- A gradual decrease in diameter (from duodenum to ileum)
- a gradual decrease in thickness of intestinal wall
- a gradual decrease in number of circular folds
- a gradual decrease in number of villi.

Only the ileum contains *Peyer's patches*, which are clusters of lymph nodes.

Accessory Organs



A diagram of the accessory organs


The hepatic portal triad

The liver and gallbladder, along with the pancreas, are classified as **accessory organs**. However, they are not simply fashion garments. The accessory organs of the digestive tract are vital to the processes of the digestive system.

The Liver and Gallbladder

The liver weighs about three pounds and is on the right-hand side of the body, right beneath the diaphragm. It is divided by the **falciform ligament**. The liver is attached to the underside of the diaphragm by the **coronary ligament**. The liver contains 4 lobes, right, left, caudate, and quadrate.

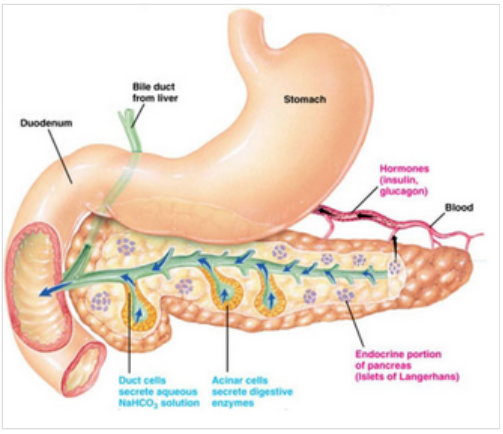
The liver is a part of more than one anatomical system. As well as functions associated with the digestive system, the liver also stores nutrients, cleans blood, and makes blood proteins. However, because this is the digestive page, we will be focusing on the digestive functions. The liver can also regenerate itself. This is useful because if one part of someone's liver is

surgically removed (if they have cancer), the liver can regrow itself and there would be no need of having a liver donor. The ability for it to regenerate is very important because as it filters out blood, it comes into contact with many different chemicals which can damage liver cells.

The main function of the liver is the production of bile. The liver produces and secretes about 700 mL of bile every day. The groups of cells that produce bile are called **hepatocytes**. **Bile** helps make fat digestion easier by breaking fat particles into smaller, more manageable ones. This is called emulsification. Bile is stored in the gallbladder until it is needed. The gallbladder also concentrates the bile by removing the water from it. Then, it is transported to the duodenum. Note that bile is NOT a digestive enzyme, it emulsifies fat. Bile also contains bile salts, these are critical for digestion of fats.

The Pancreas

The pancreas is an accessory organ that secretes enzymes into the duodenum. It is located underneath and a little to the left to the liver. It is made up of endocrine and exocrine tissue. The **endocrine tissue** produces hormones, and the **exocrine tissue** produces digestive enzymes.



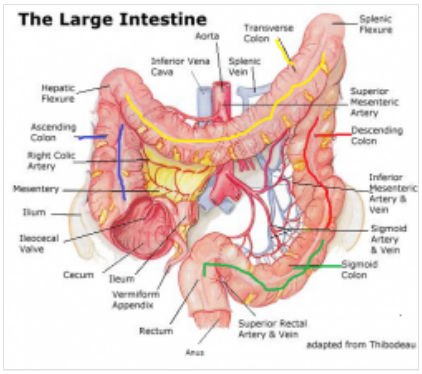
The pancreas

The digestive enzymes are created by **acinar cells** in the pancreas. Secretions of the pancreas travel through the **pancreatic duct** and join the common bile duct. They enter the duodenum through the duodenal papilla. (Check the Digestive Secretion List for a list of pancreatic secretions.)

The pancreas also produces **hydrogen carbonate** (HCO3-) which helps counter the acidic quality of chyme that enters the duodenum from the stomach. This stops pepsin digestion but makes the other secretions of the pancreas more effective.

The Large Intestine

The large intestine is the final part of the digestive tract. It is about 5 feet long, and consists of the **cecum**, **colon**, **rectum**, and **anal canal**. Note that it is shorter than the small intestine. The reason it is called the large intestine is that it's wider than the small intestine. The large intestine is mobile, and undergoes several muscular contractions called **mass movements**. Mass movements propel the colon towards the anus to help move food along with peristalsis. Note that the large intestine generally does not absorb nutrients. Although some nutrients such as vitamin K are absorbed, the main site of nutrient absorption is the small intestine. The large intestine mainly only absorbs water and salt.



The large intestine

The Cecum

The cecum, or caecum, is the first part of the large intestine. Food from the small intestine enters the cecum through the ileocecal valve.

A small pouch-like attachment to the cecum is the **appendix**. The appendix has no real function, as it was probably lost through human evolution. Organs that have no real function like the appendix are referred to as **vestigial organs**. Scientists believe the appendix was used to digest a diet with a lot of cellulose (plant fiber). Today, the appendix may store helpful bacteria. This is important because if an infection infects the large intestine, and all of the helpful bacteria were gone, the appendix could move some of the good bacteria from itself to the large intestine.

The Colon

The colon makes up the bulk of the large intestine. There are four parts to the colon- the ascending colon, the transverse colon, the descending colon, and the sigmoid colon. The **ascending colon** is the first part, being right after the cecum. Food travels upwards through it. Next is the **transverse colon**. Food travels from left to right. Then, food goes down through the **descending colon** and **sigmoid colon**.

The colon is where chyme is converted to feces. Water and salts are absorbed in the large intestine, and microorganisms such as helpful bacteria help break down components of the feces. The colon stores the feces until **defecation**, where the feces is transported out of the digestive tract through the anus.

The inner wall of the large intestine is lined with mucus. It also has straight, tubular glands called **crypts** that have goblet cells to produce the mucus. Longitudinal, smooth muscle does not have its own layer in the large intestine. Instead, it exists in the form of three strips called **teniae coli**. Attached to the outer wall are small fatty sacs called **appendices epiploicae**.

The Rectum and Anal Canal

After food travels through the sigmoid colon, it enters the rectum. The **rectum** is a straight, muscular tube. The muscle layers are very thick to push food into the anal canal. The **anal canal** is the last 2-3 centimeters of the digestive tract. The anal canal has an even thicker muscle layer.

The **peritoneum** is a serous membrane that surrounds the abdominal cavity and organs. The inner layer is called the **visceral peritoneum**, and the outer layer is called the **parietal peritoneum**. A double layer of peritoneum is called **mesentery**.

Regulation of Digestive Secretions and Processes

Digestive secretions are regulated by the parasympathetic nervous system and exocrine and endocrine hormones.

Regulation of Stomach

Initiation of digestion is mostly driven by the vagus nerve (cranial nerve X). When the vagus nerve is stimulated, gastric secretions begin.

Stomach Secretions

The stomach secretes a few very important chemicals that are vital in order for the process of digestion to occur. The stomach is made up of a few specialized cells that produce these enzymes. *For a list of secretions in the digestive system, see the Digestive Secretion List*. In this list, you will find a few cells named "Chief" and "Parietal" cells and these two cells are very important in the digestion of proteins. As you may know, proteins are primarily digested in the stomach and you can find that the prime reason for this is due to an enzyme called pepsin. Pepsin is formed when pepsinogen - an enzyme produced by Chief Cells) and HCl (hydrochloric acid) - a chemical produced by parietal cells - are combined, you get pepsin, whose functions are to break down these proteins. *Another type of cell you may see in the stomach are mucous cells, which produce mucus. The function of this mucus is to prevent the stomach from self-digesting, as the stomach has chemicals that have low pH levels.*

Digestion, Absorption, and Transport of Nutrients

Digested molecules of food and water, as well as minerals from the diet, are absorbed throughout the small intestine. These molecules pass through the mucosa via absorptive cells. Lipids are absorbed last of the three main food groups, since they must first be broken down by lipase and bile salts. (See also: Mineral Absorption (https://vivo.colostate.edu/hbooks/pathphys/digestion/smallgut/absorb_minerals.html))

Diseases of the Digestive System

There are many different diseases and disorders that affect the digestive system. In the subcategories below describe how the disease or disorder affects the body, from the cellular level up to the entire organism. The first six diseases/disorders are required for all levels, with the remaining 4 being necessary for the national level competition only.

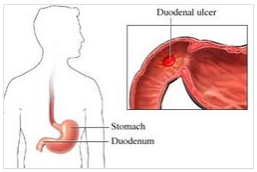
Stomach and Duodenal Ulcers

Ulcers occur when the mucus in the stomach or the intestines deteriorates, leaving the flesh directly exposed to the acid contained in these organs. Ulcers can also occur in the esophagus, but the focus is on the stomach and intestines.


A Gastric Ulcer

These ulcers can occur for several reasons:

1. (60% of stomach, 90% of duodenal) A bacteria, *Helicobacter pylori*, inflames the tissue by living in the mucus.
2. An extreme intake of either sour or spicy foods with a high acid content.
3. The use of NSAIDs may also play a role in this condition. NSAIDs block the body's ability to make more mucus, thus the mucous layer breaks down, giving the gastric acid an exposed portion of the skin.



A Duodenal Ulcer

Ulcers can be either benign or harmful, with most duodenal ulcers being benign.

Ulcers can be treated by using common drugs. Avoid smoking and drinking, as both inhibit healing of the ulcers.

Cancers of the Digestive System

If a tumor affects the digestive system, it is called a **neoplasm**. These neoplasms can occur in a variety of places in the system, including the esophagus, stomach, small intestines, and colon. There are several variations of cancer in each of these sub-groups, listed in their appropriate section below.

Esophageal (Oesophageal) Cancers

There are two major types of cancer occurring in the esophagus, as the other types are exceptionally rare. There are:

1. Squamous cell cancer (approximately 90%-95% of all cases, worldwide)
2. Adenocarcinoma (fastest growing in the United States, responsible for most of the rest of the approximately 5%-10%)

There are several major symptoms of esophageal cancers. They include vomiting blood, regurgitation of food, heartburn, difficulty swallowing, and having chest pain that has nothing to do with eating.

Tests include:

- Barium swallow (must swallow a drink containing barium, then x-rays are taken to look for any abnormalities in the travel of the liquid)
- chest MRI (used for determining the progression of the cancer)
- endoscopic ultrasound (also used for the progression of the disease)
- esophagogastroduodenoscopy (use of a small camera inserted through the mouth to view the tissue in the throat, stomach, duodenum)
- biopsy
- PET scan (used to determine stage of disease/whether surgery is possible to remove cancerous growth).

For these cancers, surgery, chemotherapy, or radiation are the most common treatments. Chemotherapy and radiation are most often used to help reduce the cancer to a point where surgery can be used, or to help reduce the symptoms (the latter case is generally when the cancer is considered incurable).

Stomach Cancers

Stomach, or gastric, cancer is defined as a cancer that develops in the lining of the stomach. There are a number of factors that influence a person's vulnerability and likelihood to develop this form of cancer, but some of the most important are:

- The presence of *Helicobacter pylori* in the stomach, or ulcers caused by this bacteria, and surgery performed on any such ulcer (accounts for approximately 60% of all cases)
- Smoking
- Anemia
- Long-term gastritis
- Obesity
- A diet high in salted and/or pickled foods

- Heavy drinking (severe alcohol abuse usually results in gastric lining damage)
- Genetics (hereditary forms account for 10 to 13% of all cases)
- Long-term use of NSAIDs (Nonsteroidal anti-inflammatory drugs)

When the cancer is in early stages, the symptoms seem rather innocuous, appearing as indigestion, nausea, heartburn, bloating, and/or a loss of appetite. As the cancer progresses, the symptoms often become far more severe, including, but not limited to, moderate to severe stomach pain, blood in stool, weight loss, vomiting, continued bloating and heartburn, and yellowing of the eyes and skin.

An overwhelming majority of cases of gastric cancer are classified as carcinomas, meaning the cancer develops in the epithelial tissue inside the stomach. However, other forms of cancer, such as lymphomas, may arise in other portions of the stomach, but are considered far rarer. Because the initial symptoms are similar to that of many lesser diseases, the warning signs are usually passed off as the ill effects of a food or a virus. Stomach cancers tend to develop slowly over a period of several years as a result.

There are several tests or examinations performed to determine if a patient has a form of gastric cancer. The most common is a gastroscopic examination (upper endoscopy), using a camera attached to a wire to view the inside of the stomach. Other common diagnosis methods are an Upper GI series and various CT (computed tomography) scans of the abdominal region. Biopsies are also commonly used to determine the malignancy of the tumor. A new method, utilizing a breathalyzer-like device, is being developed by Chinese and Israeli scientists, with a successful clinical study performed in 2013-2014.

Treatment methods remain very few and ineffective for gastric cancers. Surgical removal of the tumor is the most effective manner of treatment, with chemotherapy, radiation, and targeted therapy mostly being used to reduce the size of the tumor and provide palliative care. Survival rates for stomach cancer are extremely low (five-year survival rate is below 10%) for several reasons. First, this particular form of cancer is primarily found in geriatric patients (median age of presentation is between 70 and 75 years of age). Also, by the time symptoms are attributed to something serious, the cancer has generally metastasized.

Other interesting facts about stomach cancer:

- Occurs twice as often in males as in females
- 5th leading cause of cancer
- 3rd leading cause of death from cancer
- Before 1930's, since most food was pickled or salted before refrigeration was developed, it was the most common cause of cancer-related death.
- Most commonly found in East Asia and Eastern Europe. (Much less common elsewhere)

Colorectal Cancers

Colorectal cancer (CRC), also known by various other names (such as colon cancer, bowel cancer, and rectal cancer), refers to the neoplasms affecting the lower portion of the large intestine, which is known as the colon, and the rectum. A majority of colorectal cancer cases are a result of the lifestyle of the patient. Factors include, but are not limited to, the following:

- Age (geriatric patients are at higher risk)
- Race (people of African descent are at higher risk)
- Obesity
- Diabetes
- Smoking
- Sedentary lifestyle (exceptionally low levels of exercise)
- Excessive consumption of red or processed meats
- Excessive alcohol consumption
- Crohn's disease, ulcerative colitis, and IBS (irritable bowel syndrome)
- Genetic disorders such as familial adenomatous polyposis

Symptoms for this form of cancer include rectal bleeding, blood in stool, unexplained weight loss, a change in bowel movements (either constipation or diarrhea) that lasts for four or more weeks, and weakness or fatigue that is unexplained. A large portion of patients reported no early signs or symptoms for the disease, so regular screening past 50 years of age is recommended. Because more than 80% of colorectal cancer cases arise from a certain form of polyp called "adenomatous polyps," regular screening can pick up these early indicators up to 2-3 years before symptoms even begin to appear.

There are several testing methods, but the most common include:

- Fecal Occult Blood Testing (FOBT)
- Flexible sigmoidoscopy
- Colonoscopy
- Multitarget stool DNA screening test

In some countries, such as the United Kingdom, testing is organized for all persons older than 50. In the United States, it is recommended that those at high risk start regular screening at 40, and those with normal risk begin at 50, but no organized testing is performed nationwide.

The treatment methods for colon and rectal cancer vary slightly from each other, in part due to the difference in physiology between the two areas, and also how they react to the treatment types. If caught early on, the cancer is removed by laparotomy (a cut in the abdomen) to remove the cancerous section, and the colon is reattached. If the tumor takes up, or has spread to, a large portion of the colon, a colostomy may be necessary. If the tumor is larger, chemotherapy is used to shrink the tumor before surgery. Radiation therapy is generally not used in any stage of colon cancer due to the sensitivity of the lower intestine to radiation. The major difference in treatment between rectal and colon cancer is the use of radiation in second stage (and higher) tumors for rectal cancer, since the sensitivity is much lower in that region. Palliative care is usually the only course of treatment if the cancer has progressed to an advanced stage, especially for colon cancer. After successful removal of a tumor, the patient is encouraged to exercise quite regularly, as there is some evidence that indicates that exercise reduces the risk of re-emergence of cancer.

Interesting facts about colorectal cancer:

- Drinking 5 or more glasses of water a day is linked to lower risk for these cancers
- The five-year survival rate is below 60% but the rate is approximately 100% if caught in the M0 stage or earlier, and approximately 90% if caught in the T1 or T2 stages.
- Metastasized colorectal cancer usually spreads to the liver and then the lungs
- In 2012, 1.4 million people were diagnosed with new cases
- 65% of all cases are found in developed countries

Diarrhea

Also spelled diarrhoea, this condition occurs when an individual has three or more loose or liquid bowel movements in a day. If this condition is prolonged, it can lead to severe dehydration, and even death in some instances. For this reason, a patient with diarrhea should be given fluids, especially in the form of clean water, on a regular basis. Very young and elderly patients should be monitored closely with this condition, as diarrhea affects them more severely.

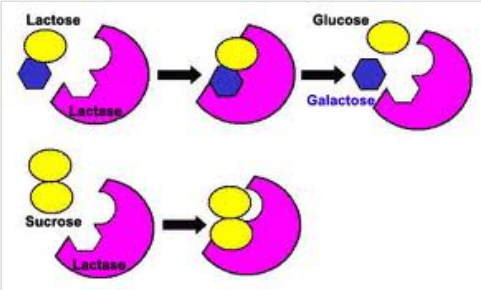
Diarrhea can be utilized by two major parties: the host's own body in attempting to fight off infection, and also by the pathogen attempting to harm the host. Because of this, it is often important to determine the cause of the issue before prescribing or self-medicating with antidiarrheals.

Symptoms associated with diarrhea include, but are not limited to:

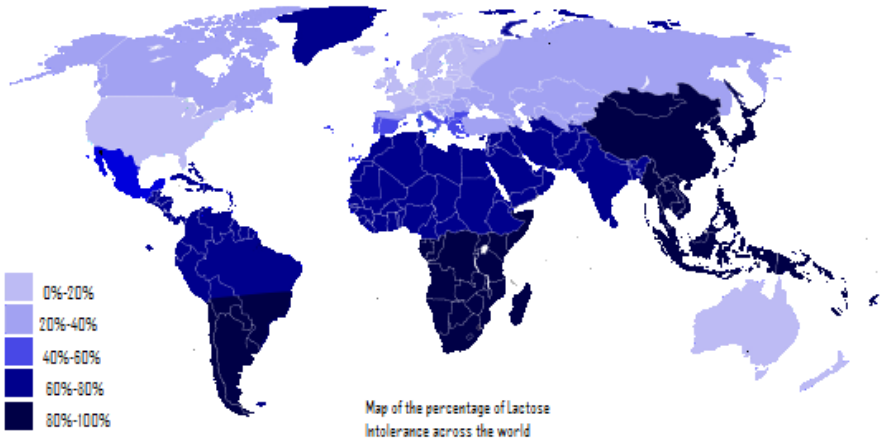
- Loose and/or watery stools
- Pain or cramps in the abdomen
- Bloating

- Nausea

Lactose Intolerance



A chart on how lactase works



Lactose intolerance is the digestive system's inability to break down a sugar called lactose, hence the name. This most commonly occurs due to the body's lack or insufficient quantities of an enzyme, called lactase, used to break the lactose for energy. As a result, the person with this condition will experience pain because of their body's inability to digest the sugar. Most infants have a proper amount of lactase enzymes inside their bodies in order to break down the lactose found in their mother's milk. However, as they age, the amount of active lactase dramatically decreases in most cases. Lactose intolerance is the human normal, with lactose "tolerance" being a rather new human evolution, especially in those of Northern European descent. People with lactose intolerance should avoid consuming dairy products. A lactase pill can be ingested in order to receive the enzyme lactase needed to digest dairy products.

Hepatitis

Hepatitis is an inflammation of the liver caused by a virus or high alcohol consumption. There are multiple forms of hepatovirus, including: A, B, C, D, E, and F. Hepatitis can cause many complications such as liver cirrhosis, cancer, and death.

Hepatitis A: This is a very contagious viral disease caused by the hepatitis A virus. It is very rare but it can be spread by contaminated food and water. The virus spreads by the fecal-oral route. It can also be spread by contact with someone who has the virus. Only humans can transmit the virus. Some symptoms include flu-like symptoms and jaundice. This disease is preventable by vaccine. It can also be prevented by good hygiene and sanitation. It can clear up in about 1-2 months with rest and hydration.

Hepatitis B: It is a dangerous liver infection. In the early stages, it is inflamed. In the late stages, cirrhosis occurs. Cirrhosis is when liver tissue is replaced by scar tissue. It spreads by sexual contact and it can easily be preventable by vaccine. The condition can clear up on its own. However, chronic cases may require medication and possibly a liver transplant. Some symptoms include jaundice and dark urine. It is easily preventable by absences or no sexual activity.

Hepatitis C: It is an infection of the liver that is caused by the hepatitis C virus. It is spread by contaminated blood. This can occur by unsterile tattoo needles or dirty needles used by a hospital. Some symptoms include loss of appetite and jaundice. It is diagnosed by blood testing for antibodies or viral RNA. It is easily preventable by using sterile blood equipment. There is no vaccine against this virus. It can be treated by using medications such as antivirals.

Hepatitis D: It is a liver disease that is caused by the hepatitis D virus. It occurs only among people who are infected with the Hepatitis B virus. It can be prevented by sexual absence. Essentially, the same technique as preventing the hepatitis b virus (above). Symptoms include abdominal pain, nausea, and fatigue. There are few treatments for it.

Hepatitis E: It is a liver disease caused by the hepatitis E virus. The liver gets inflamed. It is usually spread by contaminated water (contaminated by fecal matter). Symptoms include but are not limited to jaundice and lack of appetite. Hepatitis E usually goes away in about 4-6 weeks.

Hepatitis F: This is a hypothetical virus. Several Hepatitis F cases came in the 90's but none of them had substantial evidence.

Appendicitis

Sometimes, the appendix may become infected and inflamed, a condition called appendicitis. Appendicitis is tested by touching the McBurney's point on the patient. This is located about 1/3 away from the left pelvic bone to the belly button. If the McBurney's point is extremely sensitive to the pressure, there is a good chance the patient has appendicitis. Appendicitis can be treated in most cases with an appendectomy, where the appendix is removed.

Diverticular Disease

Diverticular disease is a general term used to describe the experience of symptoms related to diverticula (small bulges or pockets that develop in the intestine). Diverticular disease is related to two other conditions called diverticulosis and diverticulitis. All of these conditions affect the large intestine, especially the portion closest to the rectum which is called the *sigmoid colon*.

Diverticula are considered a normal part of aging, as most people develop them later in life. Doctors and health institutions estimate that approximately 5% of the population have developed diverticula by age 40, and over 50% by age 80. The exact cause of these bulges has not been determined yet, but there is a strong correlation between low fiber, high red meat diets and the development of the pockets (but not solid evidence). Other factors include:

- Smoking
- Obesity (and even just being overweight)
- History of constipation
- Long-term use of NSAIDs (i.e. ibuprofen, etc.)
- Family history of developing diverticula before age 50

The symptoms most often associated with diverticular disease, before it progresses to diverticulitis, include:

- Abdominal pain, usually in the lower quadrants
- Bloating
- Long-term changes in bowel movements (constipation and/or diarrhea)
- Episodic constipation followed by diarrhea (a pattern often described as going to the bathroom repeatedly but only passing *rabbit pellet* stools)

Because these symptoms are generally minor and often not very painful, this disease is usually diagnosed during other tests. Only approximately one in every four people who develop diverticula experience symptoms such as the ones listed above. However, if the symptoms do appear to persist, and the doctor deems it necessary, typically a non-invasive test will be ordered to look for a source. These exams include CT scans, X-rays, and an ultrasound.

Treatment for this disease is generally not necessary, as the symptoms are not harmful, unless a complication is found during testing. A diet high in fiber and low in red meats is recommended to reduce the risk of more diverticula forming. Also, it is necessary to avoid the use of laxatives to treat the condition, and use of enemas should be reduced.

Diverticulosis

The overwhelming majority of people that have diverticula have no noticeable symptoms whatsoever, and the diverticula do not affect the function of their intestine. This condition is referred to as diverticulosis. In the long run, this condition can lead to the discomfort and other symptoms associated with diverticular disease, and eventually (in some, but not all, cases) diverticulitis. In rare cases, the diverticula can lead to perforation or stricture of the large intestine, fistulas, and rectal bleeding. For a more in-depth discussion of these symptoms, see the diverticulitis symptoms and complications section below.

Because diverticulosis does not normally present with any symptoms, there is no standard testing. Usually, the diverticula are discovered during any number of other routine tests for cancer or other diseases, such as a colonoscopy or sigmoidoscopy. There is no treatment necessary unless a complication is found during a test, though a diet high in fiber and

low in red meats is recommended. It is also necessary to avoid intake of laxatives if diagnosed with this condition, and enemas are to be used infrequently.

Diverticulitis

As the name of the condition indicates, diverticulitis is when the diverticula become inflamed. This inflammation is thought to be caused by either a small perforation of one of the diverticula that the body is able to heal, or an infection of bacteria inside the bulge in the colon, although an exact cause is not currently known. Diverticulitis usually presents after the minor, more generalized, symptoms of diverticular disease. Risk factors that correlate to a likelihood to develop diverticulitis are essentially the same as diverticular disease. These include a diet low in fiber and high in red meat, smoking, being overweight or obese, a history of constipation, long-term use of NSAIDS (i.e. ibuprofen, naproxen, etc.), and a family history of developing diverticula before age 50.

The symptoms of diverticulitis are often far more severe than those of either diverticular disease or diverticulosis, and a number of secondary issues can occur, leading to *complicated diverticulitis* (referring to the complications). Some symptoms and potential complications include:

- Severe abdominal pain, usually more noticeable and painful on the left side
- A fever of 100.4 degrees Fahrenheit (approximately 38 degrees Celsius) or higher
- Severe diarrhea
- Rectal bleeding
- Formation of a **stricture** (when a section of the colon narrows that prevents stool from passing through easily)
- Formation of an **abscess** (basically a pocket of pus sealed off within the colon)
- Formation of a **fistula** (this occurs when the diverticulum forms a connection with another organ or the skin). Commonly occurs with the fistula tract leading to the bladder. Other potential sites include the uterus, vagina, skin, or another portion of the intestine.
- **Peritonitis** (literally the inflammation of the peritoneum, which is the membrane tissue covering and surrounding the organs in the abdomen)

GERD

GERD (**G**astro**e**sophageal **R**eflux **D**isease) is a disease in which the contents of the stomach travel back up the esophagus, irritating the lower esophageal area (and sometimes the upper area too) and causing a sensation described as "heartburn" which is worsened by eating or lying down. GERD is caused by a dysfunctional lower esophageal sphincter (LES) which would typically function to close off the esophagus from the lumen of the stomach. Some of the atypical symptoms of GERD include difficulty swallowing, sore throat, and regurgitation of food.

Usually, a diagnosis can be made by a physician with a description of the symptoms which may be followed up by an endoscopy (more specifically, an esophagogastroduodenoscopy, or EGD) in which the physician searches for tissue erosions, thickening of the mucosal layer, and possibly ulcers in the esophagus to help confirm the diagnosis. For a more definite diagnosis, a pH monitor is placed over the LES, and the results (characterized by timely decreases in pH, indicating acid reflux) are compared with the patient's description of symptoms according to the time they occur. This is called "ambulatory pH monitoring."

There is no actual cure for GERD, but the symptoms of GERD can be treated.

1. For heartburn, antacids can be taken after meals but relief will not last very long. For longer relief, proton pump inhibitors (PPIs) can be used to reduce the amount of HCl (hydrochloric acid) produced in the stomach.
2. Acetaminophen can help relieve pain associated with GERD.
3. Problematic foods should be avoided.

Crohn's Disease

Crohn's Disease is a form of IBD (Inflammatory Bowel Disease) which is characterized by chronic inflammation of the GI tract. Although it is usually associated with the large intestine, Crohn's disease can affect any part of the GI tract, as opposed to **ulcerative colitis** (which only affects the large intestine). It is categorized as an autoimmune disorder. This means that the body's immune system overreacts and starts attacking healthy body cells in the affected areas of the GI tract.

Individuals with Crohn's disease experience chronic pain due to the inflammation which results from the hyperimmune response. Other symptoms include: pain with defecating, fever, diarrhea or constipation, abdominal pain, fatigue, bloody stool, and ulcers anywhere in the GI tract (including the mouth).

The exact cause of Crohn's disease is unknown. Heredity and environment factors have been shown to affect development of the disease. Risk factors include smoking and a family history of Crohn's disease. Crohn's disease can occur in individuals of any age or sex, but it has been observed to be most frequent in men ages 15-35.

There are several methods for diagnosis:

- Colonoscopy, sigmoidoscopy, endoscopy, and enteroscopy to check for physical signs such as strictures and inflammation
- MRI (Magnetic Resonance Imaging) or CT (Computed Tomography) scan
- Upper GI series (x-rays or the stomach and esophagus) mainly to look for ulcers
- Physical examination may reveal abdominal masses/lumps and tenderness when certain areas are palpated

There is no specific cure for Crohn's disease, although the symptoms can be controlled to some degree by medication and changes in diet. In severe cases, surgery (bowel resection) may be required to remove certain affected areas of the GI tract. Since the cause of Crohn's disease is unknown, there really aren't any known methods of prevention.

Celiac Disease

Also spelled as Coeliac Disease, Celiac Disease is when the body has an immune reaction to gluten and can experience symptoms such as diarrhea or bloating. Also, the villi and microvilli are flat reducing the amount of absorption. It is a reaction to gliadins and glutenins (gluten proteins). The body's immune response attacks the villi in the small intestine, making nutrient absorption harder. Celiac disease is also hereditary. For treatment, people should avoid eating anything with gluten, such as wheat, barley, rye, or any other grains with gluten. This will help with the symptoms and let the intestine heal.

Most people that have Celiac disease have the HLA-DQ2 allele. For the most part, the most effective treatment is not eating gluten (having a gluten-free diet).

Outside References

American Society of Colon and Rectal Surgeons: Diverticular Disease (https://www.fascrs.org/patients/disease-condition/diverticular-disease)

Mayo Clinic: Colon Cancer (http://www.mayoclinic.org/diseases-conditions/colon-cancer/symptoms-causes/dxc-20188239)

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Anatomy/Immune System

The **Immune System** is a topic of Anatomy and Physiology in the 2027 season, alongside the Digestive and Respiratory Systems. The immune system is composed of cells, tissues, and organs that work together to defend the body against attacks by "foreign" invaders.

Overview

The immune system protects the body from disease and potentially harmful foreign invaders called **pathogens**. There are five types of pathogens which include **bacteria**, **viruses**, **fungi**, **protozoa**, and **helminths** (worms). The first 3 are often referred to as **microorganisms**, and the latter 2 are also known as **parasites**. The process through which pathogens cause disease is called **pathogenesis**.

The immune system defends against pathogens two ways: preventing foreign organisms from entering the body and destroying harmful organisms that do enter the body. It detects pathogens and differentiates between potentially harmful agents and the body's own healthy tissues. When the immune system cannot differentiate harmful foreign invaders and its own cells, it is an autoimmune disease. It creates an immune response to itself.

The Immune System

There are 3 levels of the immune system. The first two are **nonspecific** (or **innate**), meaning they defend against all kinds of pathogens. The third is **specific** (also **adaptive** or **acquired**), because it identifies and targets certain pathogens. As people age, their specific immune systems adapt to the pathogens they encounter, which decreases the likelihood of getting sick from previously-encountered pathogens.

First Line of Defense

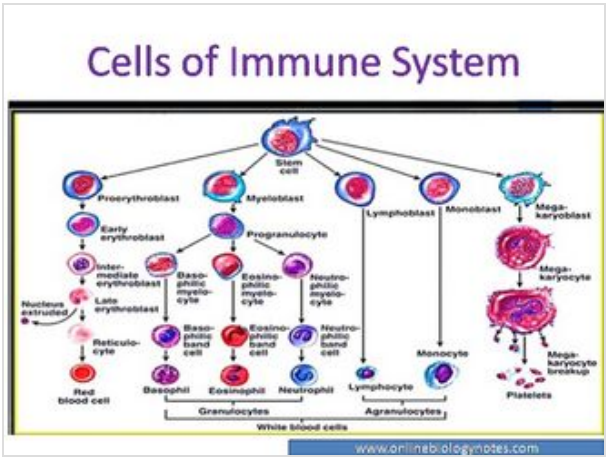
The first line of defense is made up primarily of the skin, mucous membranes, and their secretions. They protect against external pathogens from entering the body. Here are some examples:

- The **skin** is a physical barrier made of dead cells. It secretes oily and acidic secretions from sweat glands, which inhibit bacterial growth.
- **Sebum** (unsaturated fatty acids) provides a protective film on the skin and inhibits growth
- Secretions from **mucous membranes** (saliva, tears, sweat, etc.) contain antimicrobial proteins such as **lysozyme**, which breaks down bacterial cell walls.
- **Vibrissae** (nose hair) filters microbes, dust, and pollutants within air.

- **Cilia** that line the lungs traps and moves foreign substances out from the lungs towards the throat with a sweeping motion.
- **Gastric juice**, which is highly acidic, kills microbes in the stomach.
- **Symbiotic bacteria** in the digestive tract and vagina out-compete other potentially harmful organisms.
- **Urine** flushes microbes from the urethra.
- **Defecation** and **vomiting** also expel microorganisms.

Second Line of Defense

The second line of defense deals with pathogens that have entered the body. It involves several nonspecific mechanisms.



Cells of the Immune System

- **Phagocytes** are white blood cells (leukocytes) which engulf pathogens by extending **pseudopods** to surround it. This process is called **phagocytosis**. Leukocytes are formed in stem cells in bone marrow through the process of **hematopoiesis** (this term refers to all blood cells).
  - **Macrophages** are larger, long-living versions of **monocytes**. Monocytes circulate through the blood stream, and are known as macrophages when they mature. Macrophages move throughout blood, lymph, and body tissues. They are specialized in the removal of dying or dead cells and cellular debris as well as pathogens. Macrophages also play an important role in chronic inflammation.
  - **Neutrophils** are the most abundant type of white blood cell. They are normally present in the blood stream, but quickly enter tissues to phagocytize pathogens, primarily bacteria, in acute inflammation. They respond within minutes to the site of injury.
  - **Mast Cells** also secrete histamine, as well as serotonin. They help cause inflammation and respond to wound injuries.
  - **Dendritic Cells** are the messengers between the innate and adaptive immune systems. Dendritic cells acquire and present antigens to lymphocytes to activate them. These can differentiate from **monocytes** like Macrophage.
- **Basophils** and **Eosinophils** are also white blood cells, but are not phagocytes. Basophils secrete chemicals such as **histamine**. Histamine triggers vasodilation, causing more phagocytes to be brought into the area. Histamine is also known as being responsible for the symptoms of allergies and the common cold. Eosinophils are short-lived and have a wide range of functions, including attacking parasites and helping with allergic responses. The proteins they make can be harmful to the body's own tissues as well as pathogens.
- **Natural Killer Cells** (NK cells) attack abnormal or pathogen-infected body cells, such as tumors, by releasing toxic granules to kill the cells.

- The **Complement System** is a group of about 30 proteins which assist defense reactions. They help by enhancing the process of phagocytosis, attracting phagocytes to foreign cells, and promoting cell lysis. They are generally synthesized by the liver.
- **Interferons** are secreted by cells invaded by a virus. They stimulate neighboring cells to produce proteins that will help defend against the viruses.
- **Chemokines** are a type of cytokine that guide the movement of cells. Cells respond to certain chemokines by moving towards areas of higher concentrations of chemokines. In the immune system, they create a chemical gradient to attract neutrophils and other leukocytes to the wound site.
- **Fevers** are an increase in body temperature. Substances that induce fevers are called **pyrogens**. The increased temperature inhibits bacterial growth and increases the rate of tissue repair during an infection. It may also help certain types of immune cells function more efficiently.

The Inflammatory Response

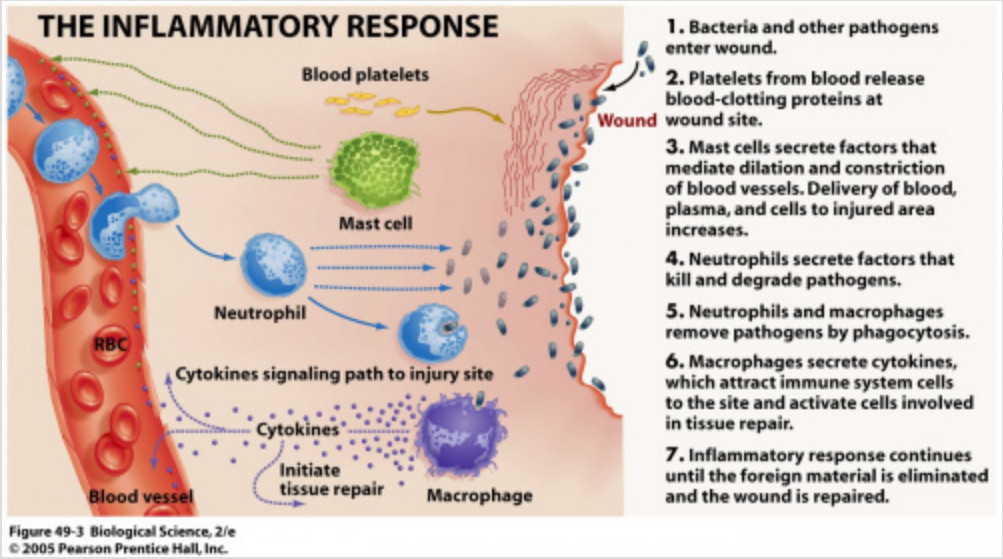
The **inflammatory response** is the biological response to harmful stimuli, such as burns, chemical irritants, frostbite, physical injury, or pathogen infection. (Given this is the Immune System page, this section will focus on the inflammatory response to pathogen infection.) It is characterized by **swelling**, **pain**, **redness**, **warmth**, and **dysfunction** of organs involved (tumor, dolor, rubor, calor, and functio laesa, respectively). Its purpose is to increase blood supply to the area to bring nutrients, proteins, and white blood cells to the affected tissues. Note that the epithelium and the capillaries are separated by interstitial fluid rather than being located right next to each other. In the example a splinter, the following events occur as part of the inflammatory response:

- **1.** Damaged cells release **chemokines**.
- **2. Mast cells**, responding to chemokines, direct contact with the splinter, or bacteria, release **histamine**.
- **3.** Histamine causes capillary endothelial cells to enlarge and move outwards, creating a swell in the capillary which encourages fluid collection. The capillary walls also become more porous. This process is called **vasodilation**. Vasodilation causes redness, temperature increase and swelling. The increase in temperature causes an increased metabolic rate in cells. Activated capillary endothelial cells also display adhesion molecules called **selectins** on the inner capillary surface.
- **4.** Phagocytes, namely neutrophils, are attracted to, slowed down by, and roll along the wall due to the selectins. Chemicals in the bloodstream activate **integrins**, adhesion receptors on neutrophils. The integrins then bind to adhesion receptor molecules on the capillary endothelial cell surfaces. The accumulation and adhesion of leukocytes to the blood vessel walls is called **margination** or **pavementing**.
- **5.** Leukocytes squeeze through openings in the capillary walls (**extravasation**, **emigration**, or more specifically **diapedesis**). **Exudation**, or the entering of fluid from the bloodstream into the interstitial fluid, also occurs. This fluid, mainly plasma, contains proteins and antibodies. Fluid buildup due to exudation is called an **edema** and is visible as a swelling (tumor). The plasma also help activate the complement, **kinin** (generates proteins that sustain physical inflammatory

effects, namely vasodilation), **coagulation** (clotting), and **fibrinolysis** (counter-balances the coagulation effect) systems.

- **6.** Neutrophils and other phagocytes attack invading bacteria that have entered due to the splinter. **Complement** helps phagocytes engulf foreign cells, and stimulates additional histamine secretion by basophils.

The inflammatory response (and the immune system in general) often consists of reactions to certain stimuli which in turn cause more reactions. Signaling pathways like this can be referred to as **biochemical cascades**.



The Inflammatory Response

The Immune Response

The Immune response is the third line of defense. It is a **specific defense system**, meaning it targets specific antigens. **Antigens** are molecules, usually proteins or polysaccharides, that are identified as foreign to the body. These molecules could be toxins, part of a viral protein cote, or molecules unique to certain bacteria, protozoa, pollen, or other foreign cells.

MHC

The **major histocompatibility complex**, or **MHC**, is the mechanism the immune system uses to differentiate between self and foreign cells. It is a collection of glycoproteins that are displayed on the cell membranes of every body cell. MHC molecules display protein fragments from inside the cell. These molecular protein fragments are known as epitopes. Each individual has a highly unique (but not always, especially in the case of identical twins) set of MHC molecules. T-cells interact with MHC molecules and "read" the epitopes to ensure that foreign invaders are not present in the cells. There are three classes of MHC, but only MHC I and MHC II play a role in immunity:

MHC Class I

All nucleic cells in the body, which includes all cells except for erythrocytes, display MHC Class I protein fragments. Cytotoxic T-cells will dock their CD8 receptors to the MHC molecules, which allows the T-cell receptors to connect with the epitope. If the T-cell receptors fit the epitope, the cytotoxic T-cell will trigger apoptosis, which is programmed cell death. Otherwise, the cytotoxic T-cell will move on to another cell. This is how cytotoxic T-cells play a role in adaptive immunity, because the shape of the T-cell receptors varies for each antigen that T-cells encounter.

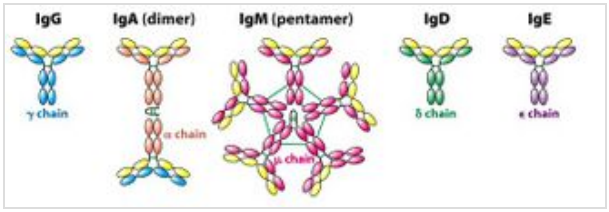
MHC Class II

MHC Class II is typically displayed by antigen-presenting cells, or APCs: B-cells, macrophages, and dendritic cells. After an APC consumes an antigen, the proteins in the antigen are broken up and displayed as epitopes on the Class II MHC molecules of the APC. Helper T-cells will dock their CD4 receptors to MHC Class II molecules. If the helper T-cell is "naive" and has not been exposed to any antigens, the T-cell receptors will bind to the epitope and learn to recognize that particular antigen. This activates the helper T-cell, which can now start secreting cytokines and multiplying itself. The cytokines tell nearby B-cells and cytotoxic T-cells to look for that antigen and kill it via apoptosis or antibody production.

Lymphocytes and their Components

Lymphocytes are white blood cells that concentrate in lymphatic tissues such as the lymph nodes, thymus gland, and the spleen. They are the primary agents of the immune response.

- **B cells** originate and mature in bone marrow (think: "B" for bone). They respond to antigens using specialized proteins called antibodies on their plasma membrane surface.
  - **Plasma cells**: release speciic antibodies which circulate through the body, binding to antigens
  - **Memory cells**: long-living B cells that do not release antibodies but circulate through the body and respond to subsequent invasions. Memory B cells provide immunity, as their response is much more quick and powerful than the first occurrence.
- The **antibodies**, or **immunoglobulins**(Ig) of B cells are antigen receptors only to a particular antigen. Antibodies inactivate antigens by binding to them, stimulating complement proteins to promote macrophage phagocytosis. There are 5 classes of antibodies, each a variation of the basic Y-shaped protein with variable regions which give them specificity to antigens. The 5 classes are as follows:



Immunoglobulin Structures

  - **IgA**: second most common, major class of Ig in secretions like mucus
  - **IgD**: not very common, mainly found on B cell surfaces
  - **IgE**: least common, involved in allergic reactions, helpful in diagnosing parasites
  - **IgG**: most common and versatile, only class transferred across placenta, is an **opsonin** (enhances phagocytosis)

    - **IgM**: third most common, first made by fetus, good at clumping microorganisms in preparation for excretions
- **T cells** originate in the bone marrow but mature in the thymus gland (think: "T" for thymus). They have antigen receptors which are not antibodies but recognition sites for molecules displayed by non-self cells. Non-self cells, such as invaded body cells, cancer cells and tissue transplant cells, display different markers than self cells. When T cells encounter a non-self cell, they divide and produce two kinds of cells:
    - **Cytotoxic T cells** or **killer T cells** destroys non-self cells by causing them to lyse.
    - **Helper T cells** stimulate proliferation of B cells and cytotoxic T cells.

Clonal Selection

When an antigen binds to a B cell or a non-self cell binds to a T cell, the B or T cell divides to produce many identical copies. This results in an increased number of the B or T cell that can respond to the specific antigen.

Cell-Mediated Response

The cell-mediated response occurs as a response to non-self cells, and involves mainly T cells.

- **1**. In a cellular infection, antigens are broken down by the cell and presented at the cell surface by class I MHC proteins.
- **2**. T cells bind to the MHC detect antigens and undergo clonal selection, initiating the production of cytotoxic T cells.
- **3.** Helper T cells bind to macrophages which are displaying marker combinations which signal with marks that they have engulfed a pathogen.
- **4.** Helper T cells produce **interleukins**, communication chemicals, to stimulate T cell and B cell proliferation. This initiates a **positive-feedback cycle**, increasing the concentration of leukocytes in the area.

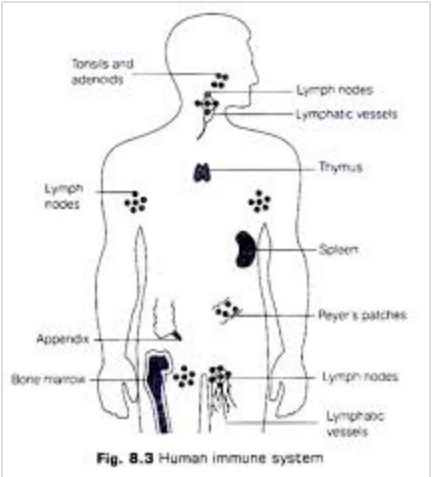
Humoral Response

The humoral response, or **antibody-mediated response** occurs as a response to antigens or pathogens circulating in the blood or lymph.

- **1**. An antigen is engulfed by a phagocyte. It displays the antigen on its surface using a class II MHC protein.
- **2.** B cells recognize the antigen and produce plasma cells, which release antibodies that bind with antigens or antigen-carrying pathogens.
- **3.** B cells produce memory cells, providing future immunity.
- **4.** Macrophages and helper T cells stimulate B cell production through cell-mediated response.

The Lymph System

The lymph system assists the immune system and may be also be considered part of the circulatory system, particularly because of its work with lymphocytes. Its main purpose is to transport white blood cells and remove interstitial fluid. It consists of a network of lymphatic vessels, nodes and organs.

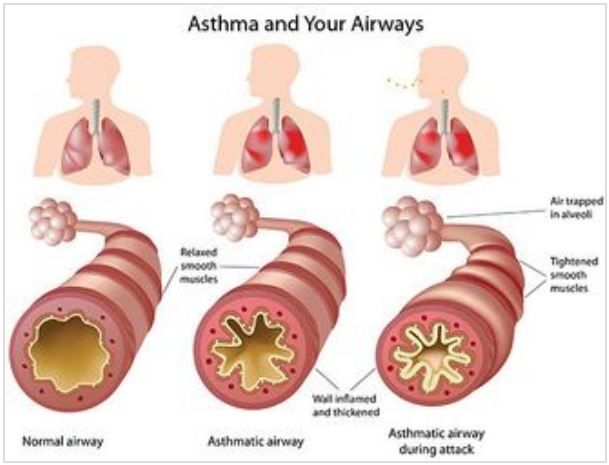


Organs of the Lymphatic System

- The **spleen** filters and stores blood. It is located above the left kidney and is commonly purple and fist-shaped. The spleen also stores white blood cells and platelets, recycles red blood cells, and fights some types of bacteria, including meningitis and pneumonia.
- The **thymus** stores immature lymphocytes and is the site of T cell maturation. It is located at the sternum.
- The **tonsils** are part of the body's first line of defense, and also help produce T cells. However, they do not have a significant function and are often removed due to inflammation. They are located in the pharynx.
- **Lymph Nodes** are oval-shaped filters placed throughout the lymphatic vessels.
- A fluid called **lymph** circulates the lymphatic system. It is derived from interstitial fluid and carries bacteria to the lymph nodes, where they are destroyed by leukocytes. The lymph also transports fats coming from the organs in the digestive system. Its name comes from the Roman deity of freshwater, Lympha.

Diseases and Disorders

Asthma



Comparing Normal and Asthma Affected Bronchioles

- **General Overview**: Asthma can cause wheezing, chest tightness, shortness of breath, or coughing. When the airways are swollen, there is less space for air to pass through, causing moderate to extreme discomfort when breathing during an asthma attack. Usually the inflammation of the airways is triggered by the inhalation of certain substances, such as dust or smoke. When the airways react to the substance, the muscles tighten. Cells in the airways may produce more mucus than usual. Asthma symptoms may be mild and treatable with an inhaler or other asthma medicines. These symptoms should be treated immediately to prevent them from worsening and becoming an asthma attack. These symptoms will go away after using asthma medicine. Sometimes extreme inflammation of the airways may happen. This is called an Asthma attack or a flareup or an exacerbation. Asthma attacks may require medical attention and support. The disease has no cure and you live with it. Thanks to today's medical technology, including rescue

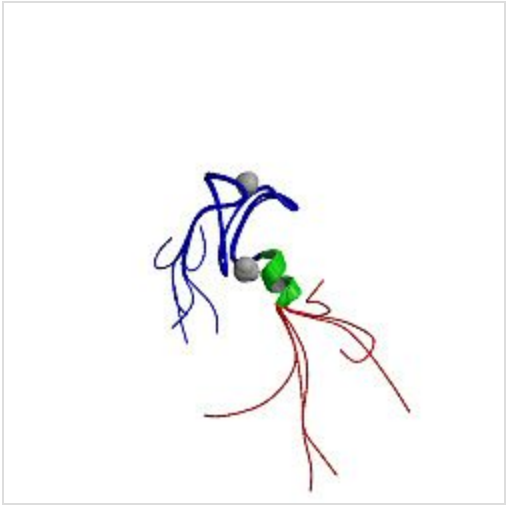
inhalers that treat symptoms and controller inhalers that prevent them, people can live a happy, active, and normal life even if they have asthma.

- **Causes**: Asthma attacks may be triggered by inhalation of certain substances, such as dust or smoke, which cause the muscles to tighten. Asthma is most likely acquired through environmental factors and inheritance, and thus it isn't caused by any particular pathogen
- **Symptoms**: Wheezing, chest tightness, shortness of breath, coughing. Moderate to extreme discomfort while breathing.
- **Treatment**: Treatment for asthma involves using bronchodilators to expand the diameter of the bronchi. Rescue inhalers during an asthma attack have albuterol in them. Long-term use inhalers generally have formoterol, salmeterol, or tiotropium. There is no cure to asthma, and someone born with asthma will most likely have it for the rest of their lives.

Ataxia Telangiectasia

- **General Overview**: Ataxia Telangiectasia is also referred to as AT, Cerebello-Oculocutaneous Telangiectasia, Immunodeficiency with Ataxia Telangiectasia, and Louis-Bar Syndrome, and is a rare disease targeting the immune system. It is a genetic disorder characterized by impaired coordination of voluntary movements (hence the name ataxia), the development of reddish lesions of the skin and mucous membranes due to permanent widening of groups of blood vessels (hence the name telangiectasia), and impaired functioning of the immune system, resulting in increased susceptibility to sinopulmonary infections. Individuals with AT also have an increased risk of developing certain malignancies, such as lymphoma, leukemia, and malignancies in the brain.
- **Causes**: Ataxia Telangiectasia is an autosomal recessive genetic disorder, which means that one gene is received from the mother and the other is received from the father. The gene that causes is this the ATM (stands for “A-T mutated”) gene, which is located on the long arm of chromosome 11 (11q22.3). This gene encodes a protein that plays a role in cell division after DNA damage has been done. The mutation causing AT causes an absence or a defect in this protein. Since DNA isn’t being repaired in any mutations that occur during cell division, the likelihood for getting cancer is much higher.
- **Symptoms**: Symptoms are present when an affected person is in their infant years. They have diminished control of skeletal muscle and lack of coordination. Dilation of blood vessels in the eye is usually visible starting from ages 3-6. Lymph nodes, adenoids, tonsils, and other lymphatic organs. Because of their impaired immune system, people with Ataxia Telangiectasia are much more susceptible to infections, particularly ones in the upper and lower respiratory system. They also deal with premature aging and many hormone deficiencies.
- **Treatment**: Since there is no cure for Ataxia Telangiectasia, treatment focuses more on symptoms and psychological growth. For respiratory infections, patients may take an antibiotic and get postural drainage of the bronchial tubes and lungs. To make their immune systems stronger, they may get gammaglobulins (antibodies) injected. For some, Vitamin E therapy helps with providing temporary relief of symptoms. Diazepam (Valium) is a drug that may help with controlling slurred speech and involuntary muscle contractions. Genetic counseling for patients and their families may help to provide emotional support.

Autoimmune Polyglandular Syndromes



AIRE Protein, the protein which is affected by the mutation that causes Autoimmune Polyglandular Syndrome Type 1

- **General Overview**: Autoimmune Polyglandular Syndromes, also known as Autoimmune polyendocrine syndromes (APSs), polyglandular autoimmune syndromes (PGASs) or polyendocrine autoimmune syndromes, are a group of disorders that are genetically inherited and characterized by autoimmune activity against more than one endocrine organ. There are three autoimmune polyglandular syndromes. There is APS Type 1, APS Type 2, and IPEX (immunodysregulation polyendocrinopathy enteropathy X-linked syndrome). Of these, APS Type 2 is the most common and is also known as Schmidt's Syndrome. Diagnosis is through looking at family history and doing genetic testing, a CT scan, endoscopy, MRI, Ultrasound, and screening of hormone levels.
- **Causes**:
  - *Autoimmune Polyglandular Syndrome Type 1*: APS Type 1 is an autosomal recessive genetic disorder. This means that one recessive allele has to come from the mother and one has to come from the father. The gene that causes this is the AIRE gene (makes a protein that is an autoimmune regulator), which is located on the long arm of chromosome number 11 (21q22.3). When this gene is mutated, then the autoimmune regulator protein is deformed or absent, and thus its function is not fulfilled. This causes an autoimmune reaction to be mounted against the endocrine organs, and thus many hormone deficiencies result from this condition.
  - *Autoimmune Polyglandular Syndrome Type 2*: It is known that the cause of APS Type 2 is genetic, but the actual cause hasn't been linked to one single gene, but instead a few. It has an autosomal recessive or an autosomal dominant inheritance pattern. Individuals are at higher risk of this disorder when they carry a certain human leukocyte antigen (HLA-DQ2(DR3 (DQB*0201)) and HLA-DQ8(DR4 (DQB1*0302)))
  - *Immunodysregulation Polyendocrinopathy Enteropathy X-linked Syndrome (IPEX)*: IPEX is an x-linked recessive genetic disorder. This means that in males, the recessive gene has to be inherited from the mother, and in females, the recessive gene has to be inherited by both the mother and the father. Because of this, the disorder is more common in males. The gene involved is the FOXP3 gene, which is located on the short arm of the X chromosome (Xp11.23). This gene codes for the FOXP3 protein, which regulates and controls development and function of regulatory T cells. Because of the mutation in this gene, the regulatory T cells don't serve their function in the immune response, and leads to an autoimmune response against many endocrine glands, as well as organs of the integumentary system.
- **Symptoms**:

- *Autoimmune Polyglandular Syndrome Type 1*: Symptoms of APS Type 1 include the following conditions: hypoparathyroidism, hypogonadism, vitiligo, alopecia, malabsorption, anemia, cataract, and adrenal hyperplasia. In general, there is a lack of hormones in the body due to an autoimmune response being mounted against more than one endocrine gland.
- *Autoimmune Polyglandular Syndrome Type 2*: A sign of this disorder is if any affected organs show chronic inflammatory infiltrate. Symptoms of APS Type 2 include the following conditions or may have some of these disorders: Nausea, frequent urination, palpitations, weight loss, anorexia, low blood pressure, hypoparathyroidism, myalgias, Hashimoto thyroiditis, Graves' disease, anemia, hypogonadism, and diabetes mellitus
- *Immunodysregulation Polyendocrinopathy Enteropathy X-linked Syndrome (IPEX)*: Symptoms of IPEX include the following conditions: eczema, diarrhea, hypothyroidism, and lymphadenopathy.

- **Treatment**:
  - *Autoimmune Polyglandular Syndrome Type 1*: Because there is no cure for this syndrome, treatment is targeted towards the symptoms. Examples of treatment include hormone replacement, antifungal treatment, and immunosuppressive treatment.
  - *Autoimmune Polyglandular Syndrome Type 2*: Because there is no cure for this genetic disorder, treatment for management is available, and includes: Cyclosporin A, Isohormonal therapy, supplements of Thyroid-stimulating hormone, glucocorticoids, and dietary guidlines (for nutrition and if the patient has diabetes).
  - *Immunodysregulation Polyendocrinopathy Enteropathy X-linked Syndrome (IPEX)*: Because there is no cure for this disorder, the most common treatment is ingestion of corticosteroids. Other treatments include: TPN (for nutritional purposes), Cyclosporin A, FK506, Sirolimus only if FK506y proves to be ineffective), granulocyte colony stimulating factor, bone marrow transplant, and Rituximab.

Burkitt Lymphoma

- **General Overview**: Burkitt lymphoma is a type of Non-Hodgkin Lymphoma, in which cancer starts in the B cells. This type of lymphoma is recognized as the fastest growing human tumor, making it fatal very quickly if left untreated. This lymphoma is named after a British surgeon named Denis Burkitt, who first discovered this type of cancer in Africa. This cancer is commonly found in young children who also have malaria and Epstein-Burr, which is the virus that causes Infectious Mononucleosis. Burkitt lymphoma is most commonly found in Africa for this reason, and is rare outside of Africa. This malignancy used to commonly be associated with people who had HIV until HAART (highly active antiretroviral therapy) became used to treat HIV/AIDS. This conditions is diagnosed through a biopsy of an enlarged lymph node. After this, additional tests to diagnose this condition include: CT scans of the chest, abdomen, and pelvis, X-ray of the chest, PET scan, bone marrow biopsy, spinal fluid exams, and blood tests to measure for kidney and liver function.
- **Causes**: All variants of Burkitt's lymphoma are characterized by dysregulation of the c-myc gene on the long arm of the 8th chromosome (8q24). People with malaria, HIV/AIDS, and Epstein-Burr

are more likely to get Burkitt's Lymphoma than those who don't have these disorders.

- **Symptoms**: A bulky tumor mass usually forms in the abdomen and from there spreads to the liver, spleen, and bone marrow. If left undiagnosed for a while, then the malignancy can also spread to the spinal cord, affecting the central nervous system. This can cause nerve damage, weakness, and paralysis. In other variants of this lymphoma, tumors usually start around facial bones or the jaw, and spread to the rest of the body, particularly the gastrointestinal system and reproductive organs. Common symptoms for all variants of Burkitt's Lymphoma include: loss of appetite, weight loss, fever, fatigue, and night sweats.
- **Treatment**: Treatment for Burkitt Lymphoma includes intensive intravenous chemotherapy. The different drugs that can be used for chemotherapy include: cyclophosphamide, doxorubicin, vincristine, methotrexate, cytarabine, ifosfamide, etoposide, and rituximab. If the cancer is also affecting the central nervous system, then drugs may be injected into the cerebrospinal fluid, known as intrathecal chemotherapy.

Diabetes, type 1

- **General Overview**: Diabetes mellitus (or simply diabetes) is a metabolic disorder which can arise for a multitude of reasons. Type 1 diabetes (also known as insulin-dependent diabetes or IDDM) arises typically in children or adolescents, while type 2 diabetes (non insulin-dependent diabetes or NIDDM) is much more frequent in adults. Type 1 diabetes is much less frequent, causing only about ten percent of cases in the United States. In both types, insulin cannot regulate the transport of glucose to other areas of the body which results in various complications.
- **Causes**: In type 1 diabetes, the insulin producing beta cells of the pancreas are attacked by T cells, which results in the pancreas being unable to produce insulin. The specific cause of this autoimmune response is unknown, however. Over 50 genes are associated with type 1 diabetes, though one of the most notable is HLA-DQB1 (or IDDM1) which is located on the p arm of chromosome 6. The protein coded by this gene plays a heavy role in the human leukocyte antigen (HLA) response similar to one involved in type 2 autoimmune polyglandular syndrome. Various alleles of HLA-DQB1 also plays a role in several other disorders, such as celiac disease and multiple sclerosis. While the risk for type 1 diabetes is thought to be inherited, simply inheriting risk factors does not mean that an affected individual will develop type 1 diabetes. Development of the disorder is typically triggered by an environmental factor (such as a viral infection), though research on this is still ongoing.
- **Symptoms**: Diabetes is characterized by sustained high blood sugar levels, with symptoms of untreated diabetes consisting of frequent urination or increased thirst and appetite. In type 1 diabetes, symptoms typically develop more rapidly than in type 2 diabetes. Type 1 diabetes can also result in a complication known as diabetic ketoacidosis (DKA), a metabolic reaction resulting from a shortage of insulin. Symptoms include nausea or vomiting, abdominal pain, deep or gasping breathing, and a particular smell on the breath described as fruity or acetone-like. In severe cases, confusion or a decreased level of consciousness can develop. Diabetic retinopathy can also cause damage to the eyes as a result of complications in blood vessels. Many complications as a result of diabetes occur in the heart, eyes, kidneys, or feet.

- **Treatment**: Type 1 diabetes is managed by insulin injections, as the body cannot produce its own insulin. Insulin injections may take the form of syringes or pens, while some diabetics may prefer an insulin pump. Treatments typically focus on lowering and normalizing the blood glucose content, which avoids complications.

DiGeorge syndrome

- **General Overview**: DiGeorge syndrome (also known as 22q11.2 deletion syndrome or many other names (https://ghr.nlm.nih.gov/condition/22q112-deletion-syndrome#synonyms)) is a genetic syndrome affecting approximately 1 in 4,000 people. It is named after Angelo DiGeorge, who discovered this condition in 1968. Issues in the immune system often arise as a result of problems in the thymus, though the severity differs from person to person. Detecting this syndrome through typical karyotyping can be difficult, so the preferred method is fluorescence *in situ* hybridization or FISH.
- **Causes**: DiGeorge syndrome is caused by the deletion of a specific area of chromosome 22 (the 22q11.2 location). The syndrome is typically caused by a mutation during early development and is only inherited in about 10% of cases. However, the syndrome is autosomal dominant, meaning that only one affected chromosome will cause this syndrome.
- **Symptoms**: Symptoms can vary wildly, even within members of the same family with this syndrome. Some of the most common symptoms are congenital heart problems, cleft palate, and recurring immune system problems. Issues with the immune system typically stem from a T cell-mediated response due to issues with the thymus. Many individuals with DiGeorge syndrome either lack a thymus or have a hypoplastic (underdeveloped) thymus, resulting in more frequent or more severe infections. Other common symptoms include differences in cognitive development, low blood calcium levels (hypocalcemia), and issues with the kidneys. Affected individuals may also have blue-ish skin, as a result of poor circulation.
- **Treatment**: Treatment for DiGeorge syndrome depends on the symptoms the affected individual is experiencing. Many affected individuals undergo cardiac surgery or thymus transplants depending on what is necessary for treatment. Hypocalcemia may also be treated with supplements in order to ensure proper levels of calcium in the blood.

Familial Mediterranean Fever

- **General Overview**: Familial Mediterranean Fever (FMF or many other names (https://ghr.nlm.nih.gov/condition/familial-mediterranean-fever#synonyms)) is an inherited disorder that occurs most frequently in people of Mediterranean descent. However, this disorder can occur in anyone regardless of background. It is an autoinflammatory disease, meaning that affected individuals do not produce autoantibodies or antigen-specific cells. Autoimmune disorders are caused by issues in the innate immune system rather than the adaptive immune system.
- **Causes**: FMF is caused by a mutation in the MEFV gene, located at 16p13.3 (the p arm of chromosome 16). MEFV normally codes for a protein called pyrin (or marenostrin) that is found in some white blood cells. Pyrin plays a role in fighting infections, as well as regulating inflammation. As a result, many symptoms of FMF have to do with excess inflammation.

- **Symptoms**: FMF is a periodic fever disorder, characterized by frequent episodes of inflammation (both systemic and organ-specific). There are seven types of attacks that can occur, mostly in conjunction with fever. Attacks typically happen in the abdomen, joints and chest, but can also occur elsewhere. Kidney failure is a potential complication of this disorder, as well as vasculitis (inflammation of blood vessels) and arthritis.
- **Treatment**: FMF attacks are typically treated using painkillers and anti-inflammatory drugs. Attack frequency can be decreased using Colchicine, commonly prescribed for gout. Anakinra may be used if the affected individual does not respond to colchicine alone.

Graves' Disease

- **General Overview**: Overproduction of thyroxine, which is a form of hyperthyroidism. It is an autoimmune disorder.
- **Causes**: Exact cause unknown. Can often be genetic: it is more common in people whose families have a history of thyroid or autoimmune diseases. In genetically predisposed individuals, environmental factors can also set off the disease. More common in females than males.
- **Symptoms**: Swelling around eyes, bulging eyes, rapid/irregular heartbeats, heat intolerance, hand tremors, fatigue, frequent bowel movements.
- **Treatment**: Reduce thyroxine with drugs, radioactive iodine, or surgery

Immunodeficiency with hyper-IgM

- **General Overview**: Immunodeficiency with hyper-IgM is when the body cannot switch from IgM antibody production to production of other types of antibodies.
- **Causes**: *HIGM1:* The most common form, accounting for ~70% of all cases. This is an x-linked syndrome, so it affects males more than females. It is caused by a mutation in the *CD40LG* gene which creates the CD40 ligand protein on T cell surfaces. With a dysfunctional CD40 ligand to bind to the CD40 on B lymphocytes, they never get a signal to switch antibodies. *HIGM2:* Caused by mutations in the *AICDA* gene. The *AICDA* gene produces the *HIGM3:* Affects males and females equally. This form The AID enzyme induces mutation and can change the chains of antibodies to make them IgG, IgM, IgE, IgA, or IgD. People with HIGM2 lack this enzyme. is caused by mutation of *CD40* gene itself. In this case, the B cell simply just lacks a CD40 receptor. This way, it cannot receive a signal to switch antibodies. *UNG Deficiency*: UNG deficiency is caused by a lack of uracil-DNA glycosylase enzyme due to mutations in the *UNG* gene. This enzyme forces antibody diversity, and without it, it is harder to switch to other antibody types.
- **Symptoms**: Frequent, severe bacterial and viral respiratory infections, fluctuating levels of immunoglobulins, chronic diarrhea, neutropenia leading to mouth ulcers.
- **Treatment**:

Chronic myeloid leukemia

- **General Overview**: Chronic myeloid leukemia (CML) is a slow-growing cancer in bone marrow

that leads to overproduction of abnormal WBCs.

- **Causes**: Caused by a translocation of chromosomes 9 and 22 (creating a Philadelphia chromosome), forming the *BCR-ABL* gene. The proteins produced by this gene tell cancer cells to grow uncontrollably. There is no known genetic predisposition, but radiation is a known risk factor
- **Symptoms**: There are 3 phases of CML. The first, the chronic phase, shows mild or no symptoms. Most are diagnosed here, and the disease is highly responsive to treatment. The second, the accelerated phase, is a transition phase. Symptoms include fever, unintentional weight loss, poor appetite, night sweats, bone pain, an enlarged spleen causing left-sided abdominal discomfort, anemia, and easy bruising. This phase is also marked by abnormal platelet counts, high levels of basophils, and new, abnormal chromosomal changes in the leukemia cells, beyond the initial Philadelphia chromosome. The third phase, the blast phase, acts like acute leukemia and requires urgent intervention. Symptoms include anemia, neutropenia, thrombocytopenia, an enlarged spleen, bone/joint pain, night sweats, weight loss, hyperviscosity, skin lesions, swollen lymph nodes, and leukostasis.
- **Treatment**: Treatment usually involves Tyrosine Kinase Inhibitors (TKIs), which inhibit the protein made by the *BCR-ABL* gene. Common TKIs include imatinib, dasatinib, nilotinib, bosutinib, and asciminib.

Multiple Sclerosis

- **General Overview**: Within the CNS, the immune system attacks the myelin as well as the nerve fibers. The antigen is unknown and damaged myelin leads scarred tissue (sclerosis). During the immune response, there are increased helper T cells and increased killer T cells attacking myelin.
- **Causes**: The exact antigen is unknown, but it consists of an autoimmune reaction to the myelin sheaths of neurons.
- **Symptoms**: Common multiple sclerosis symptoms include fatigue, numbness, tingling, muscle weakness, muscle spasms, stiffness, loss of balance, dizziness, vertigo. Additional symptoms often involve blurred vision, double vision, partial or complete vision loss, eye pain, bladder problems, bowel dysfunction, cognitive changes like brain fog or memory issues, depression, anxiety, emotional mood swings, sexual difficulties, slurred speech, tremors, trouble swallowing, chronic pain, and a unique electric-shock sensation down the spine called Lhermitte's sign.
- **Treatment**: Treatment consists of antibodies against specific T cells. It is currently experimental, and meds are for reducing symptoms.

Rheumatoid Arthritis

- **General Overview**: A chronic inflammatory disorder affecting many joints, including those in the hands and feet. The body’s immune system attacks its own tissue, including joints. Severe cases it attack internal organs. Systems: affects joints linings, causing painful swelling. Over long periods of time, the inflammation can cause bone erosion and joint deformity. Treatment: no cure. Physiotherapy and medication (anti-rheumatic drugs).

- **Causes**: Caused by your immune system attacking the healthy tissue in your body, which causes rheumatoid arthritis to be an autoimmune disorder. Your immune system sends antibodies to the lining of your joints, where they attack the healthy tissue surrounding the joint. This causes the thin layer of cells (synovium) covering your joints to become sore and inflamed, and this releases chemicals that damage nearby bones, tendons, cartilage, and ligaments.
- **Symptoms**: Symptoms include stiffness lasting for 45 minutes or longer, especially after walking or sitting for long periods of time. Joints may be tender to the touch, feel warm, and be visibly swollen. Symptoms usually appear first in smaller joints, such as small joints of the hands and feet before larger joints like the elbow. Also, symptoms are similar on both sides of the body. Tiredness, fever, lack of appetite. For more, go here (https://www.mayoclinic.org/diseases-conditions/rheumatoid-arthritis/symptoms-causes/syc-20353648)
- **Treatment**:

Severe combined immunodeficiency

- **General Overview**:
- **Causes**:
- **Symptoms**:
- **Treatment**:

Systemic Lupus Erythematosus

- **General Overview**: Autoimmune disorder 1) Severe fatigue and joint pain, headaches, rash on cheeks, nos “butterfly rash”, hair loss, anemia, kidney failure. Treatment: anti-inflammatory meds
- **Causes**:
- **Symptoms**:
- **Treatment**:

Hypersensitivity

Hypersensitivity is an excessive or abnormal sensitivity to a substance. **Type I:** triggered by an innocuous foreign substance (dust, pollen, animal dander) the antigen simultaneously binds more than one membrane bound IgE on a mast cell (white blood cell contains many granules rich in histamine and heparin) (basophil). This is called cross-linking, triggers the release of mast cell granules full of histamine. Histamine then goes on to signal the various changes associated with allergies. This allergic reaction happens almost instantly and the symptoms can become evident within minutes. From histamine granule to histamine degranulation during crosslinking. **Type II:** process, which IgG or IgM binds to cell to cause injury or death. Targeted at the body’s own cells. The antibody binds to the host cell. **Type III:** tissue damage created by immune complexes (aggregations of antigen and antibodies). A single antibody can bind multiple antigens, which are bound to multiple antibody. This “clump” of antigens and antibody deposits in tissues (vessel, kidneys, or joints) and

causes inflammation by activating complement and attracting neutrophils. **Type IV:** delayed hypersensitivity and involves Th1 T-Cells attracting and activating macrophages. It takes a few days to kick in and this is the only type of hypersensitivity that doesn't involve antibodies.

Organ Transplants and Rejection

The immune system may reject an organ from a donor if the organ does not match your body exactly. Antirejection medicines (immunosuppressants) may be used, but they can weaken the immune system.

There are three types of organ rejection; **hyperacute**, **acute**, and **chronic**.

- **Hyperacute** rejection occurs a few minutes after the transplant when the antigens are completely unmatched. The tissue must be removed right away so the recipient does not die. This type of rejection is seen when a recipient is given the wrong type of blood. For example, a person given type A blood when he or she is type B.
- **Acute** rejection may occur any time from the first week after the transplant to 3 months afterward. All recipients have some amount of acute rejection.
- **Chronic** rejection can take place over many years. The body's constant immune response against the new organ slowly damages the transplanted tissues or organ.

Surgeries

Allograft vs. Autograft

Allograft: Surgically transplanted tissue. Not from the same person Can be used in bone, tendons, ligaments, skin, heart valves. Donor parts are not genetic matches. Increased rejection. One surgery, less healing time, less expensive.

Autograft: Transplant one place to another on same person. Can be used in bone and ligament grafts. Less rejection. Two surgeries, therefore there is more time that is needed to heal and is also more expensive.

Resources

Official soinc.org Training Handout 2013 (http://www.soinc.org/sites/default/files/ANATOMY_HANDOUT_2013.doc)
Official soinc.org Training PowerPoint 2014 (http://www.soinc.org/sites/default/files/uploaded_files/A%26P-2014.ppt)
University of Illinois Biological Science: Immune System Lecture (http://www.uic.edu/classes/bios/bios100/lecturesf04am/lect23.htm)
Garland Science Excerpt: Innate Immunity (http://www.garlandscience.com/res/pdf/9780815342434_ch02.pdf)
University of Washington: Phagocytosis (http://courses.washington.edu/conj/bloodcells/phagocytosis.htm)

McGraw-Hill: Inflammatory Response Video (http://highered.mcgraw-hill.com/olcweb/cgi/pluginpop.cgi?it=swf::600::480::/sites/dl/free/0073532215/788107/InflammatoryRes.swf::The%20Inflammatory%20Response)

Khan Academy: Inflammatory Response Video (https://www.khanacademy.org/science/biology/immunology/v/inflammatory-response)

Boston University: the Immune System (http://sphweb.bumc.bu.edu/otlt/mph-modules/eh/eh_immunity_b/eh_immunity_b_print.html)

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Anatomy/Respiratory System

The **Respiratory System** is a topic of Anatomy and Physiology for the 2027 season, along with the Immune System and Digestive System.

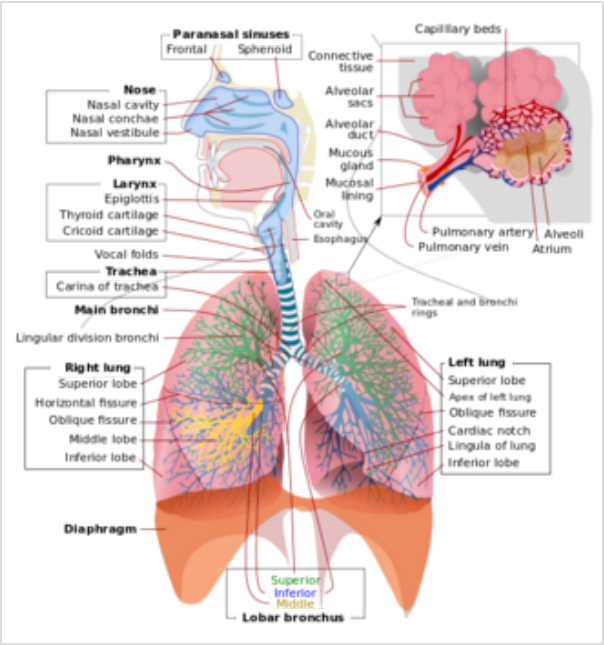
Functions

- **Protection** - enables protective and reflective non-breathing air movements such as coughing and sneezing
- **Ventilation** - the physical motion of breathing
- **Respiration** - supplies oxygen and removes carbon dioxide from blood as it circulates through the body (external respiration, not cellular respiration); the exchange of gases
- **Regulating blood pH** - the more carbon dioxide in the bloodstream, the lower the pH of the blood
- **Speech** and other vocal sounds

Mechanics

The Respiratory system is controlled in mammals by the **diaphragm muscle**.

When the diaphragm relaxes, the thoracic cavity decreases in size. However, when the muscle contracts, it pulls downward, adding size to the thoracic cavity. While this happens, the **intercostal muscles** pull upwards on the ribcage, adding more space to the thoracic cavity. When the diaphragm relaxes, it pushes upward on the lungs, along with the intercostal muscles pushing downward on the ribcage. These muscles decrease the volume while increasing their internal pressure. When the diaphragm contracts, the lungs gain volume and lose pressure, causing a partial vacuum. Having no choice at this point but to follow the laws of nature, air follows the pressure gradient and rushes into the lungs.



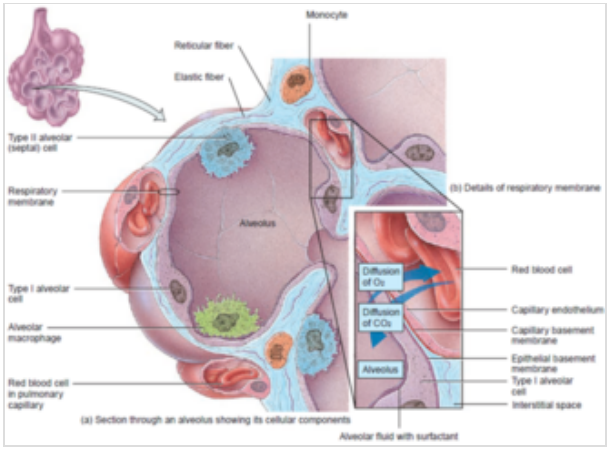
There are two main breathing patterns: **quiet breathing** and **forced breathing**.

In **quiet breathing**, muscles contract involuntarily to let air in the lungs (an active process), but muscles relax when you exhale (passive process).

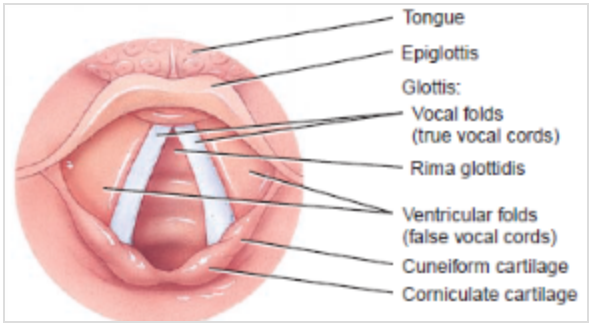
In **forced breathing**, muscles contract voluntarily in both inspiration and expiration.

Parts of the Respiratory System

The **upper respiratory system** consists of the organs from the nostrils to the pharynx, in order of where the air passes. The **lower respiratory system** stretches from the top of the trachea to the diaphragm.



Alveoli



Vocal cords

- Air is taken in through the **nose**.
  - The **nostrils** are one of two places where air enters and exits respiratory system. It has hair to trap dirt, dust particles, and bacteria.
  - The **nasal septum** separates the nasal cavities.
    - Three bones of the **nasal conchae** provide more surface area inside the nose, as they are rolled up like conch shells.
    - The **respiratory mucosa** lines the nasal cavity and has tiny cilia that move dirty mucus toward the outside of the nostrils.
  - **Lacrimal glands** secrete tears that flow across the eye's surface and drain through the openings in the lacrimal puncta (more commonly known as the corner of the eye) into nasolacrimal ducts and nasal cavities. This is why your nose runs when you cry.
  - **Sinuses** are air spaces in the skull that lighten the weight of the head. They open into nasal cavities to receive air and are lined with mucous membranes.
- Air passes through the **pharynx** on its way to the lungs.
  - The area between the nasal cavities and the pharynx is called the *choana*
  - The **nasopharynx** is the top part of the throat where the nasal cavities drain.
    - The **hard palate** can be felt at the roof of the mouth. It is a bony plate that separates the mouth (oral cavity) from the nose (nasal cavities).
    - The soft part on the roof of the mouth, nearer the back of the throat, is the **soft palate**.
    - Beyond the soft palate itself is the nasopharynx. The soft palate moves backward when you swallow so that the nasopharynx is blocked, ensuring you don't literally inhale your food. (When you laugh while eating, the soft palate begins to move back but is thrust forward suddenly, sending the contents of your mouth to your nasal cavities to fly out of your nose.)

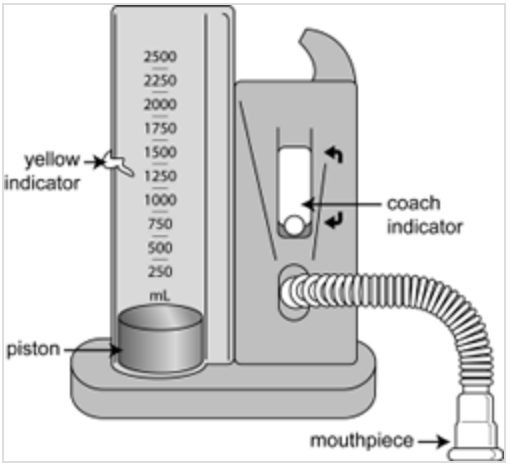
    - The **oropharynx** is in the back of the mouth.
        - It extends from the uvula to the level of the hyoid bone.
        - It is also the location of the **epiglottis**, a cartilage structure that guides materials passing through to either the trachea or esophagus, depending on what the material is. It covers the glottis and blocks food from getting into the larynx when swallowing.
    - The **laryngopharynx** is the lower part of the throat adjacent to the larynx.
        - The **larynx**, more commonly known as the voice box, is a triangular structure that produces your voice. When you aren't speaking, your voice box **opens**, and when you are speaking, your voice box **closes**. This is why you have limited breath to talk, and you have to breathe consistently, especially when you are singing or playing an instrument.
- At the apex of the triangle is the **thyroid cartilage**, better known as the Adam's apple.
    - At the top of the larynx is the **glottis**, which is the opening through which air passes.
    - The **vocal chords** are inside the glottis. They gather mucous membranes that cover ligaments. Pushing more air over the vocal chords makes the sound (singing, speaking, etc.) louder, while tightening the vocal chords narrows the glottis, making a higher-pitched sound.
- The **trachea** (more commonly known as the windpipe) is a tube that runs from the larynx to just above the lungs.
    - It divides into two large branches behind the sternum called **primary bronchi** that enter each lung.
    - Both the trachea and the bronchi are made of smooth muscle and cartilage, allowing airways to contract and expand.
- The **lungs** are large paired organs within the chest cavity on either side of the heart. They are protected by the rib cage.
    - The lungs sit atop the **diaphragm**, a powerful muscle fixed to the lower ribs, sternum, and lumbar vertebrae.
    - The **heart** sits in a cavity between the two lungs called the **mediastinum**.
    - The lungs are separated into lobes (three for the right lung, two for the left), then segments, then lobules.
    - The lungs are sealed off from the inside surface of the thoracic (chest) cavity by two layers of **pleurae**. The **visceral pleurae** cover the outer surface of the lungs and the **parietal pleurae** cover the inside surface of the thoracic cavity. The **pleural cavity** is the potential space between the two pleurae and contains a lubricating fluid called **intrapleural fluid**.
    - Because the pressure between the two pleurae is about 4 mmHg (milimetres of mercury) lower than that of the atmosphere and the inside of the lungs, the outer surfaces of the visceral pleurae are always "stuck" to the internal surface of the parietal pleurae, and the lungs are kept inflated in this way.
    - After the primary bronchus enters the lung on each side, it splits into secondary and tertiary branches called **bronchi**.

        - Tertiary bronchi divide into smaller branches called **bronchioles**. At the end of the smallest bronchioles (the terminal bronchioles) are grape cluster-like structures called **alveolar sacs**, and each sac contains **alveoli** (the individual grapes). The alveoli's walls are composed of a simple squamous epithelium designed to facilitate rapid diffusion and elastic tissue. Each alveolus is wrapped with capillaries.
    - The interface of simple squamous epithelium of the alveolus and pulmonary capillary is called the **respiratory membrane** (the single, fused boundary between the alveolar and pulmonary capillary lumens), and it is here where gas exchange takes place. Blood vessels criss cross each alveolus, providing a large surface area for gas exchange between the alveoli and pulmonary capillaries.
        - The respiratory membrane is also where carbon dioxide is eliminated. It flows into the air in your lungs and you breathe it out.
- The **diaphragm** is a dome-shaped muscle separating the thoracic (chest) cavity from the abdominal (belly area) cavity. It contracts downward (flattens) during inspiration, increasing the volume of the thoracic cavity, and inflating the lungs. Consequently, air rushes into the lungs following the pressure gradient (since air pressure in the lungs is now lower than atmospheric air pressure). When expiring, it simply relaxes, and the elasticity of the lungs pushes air out again.
    - The **motor phrenic nerves** contract and relax the diaphragm.
    - The diaphragm can also exert pressure on the abdominal cavity, helping expel vomit, feces, and urine.

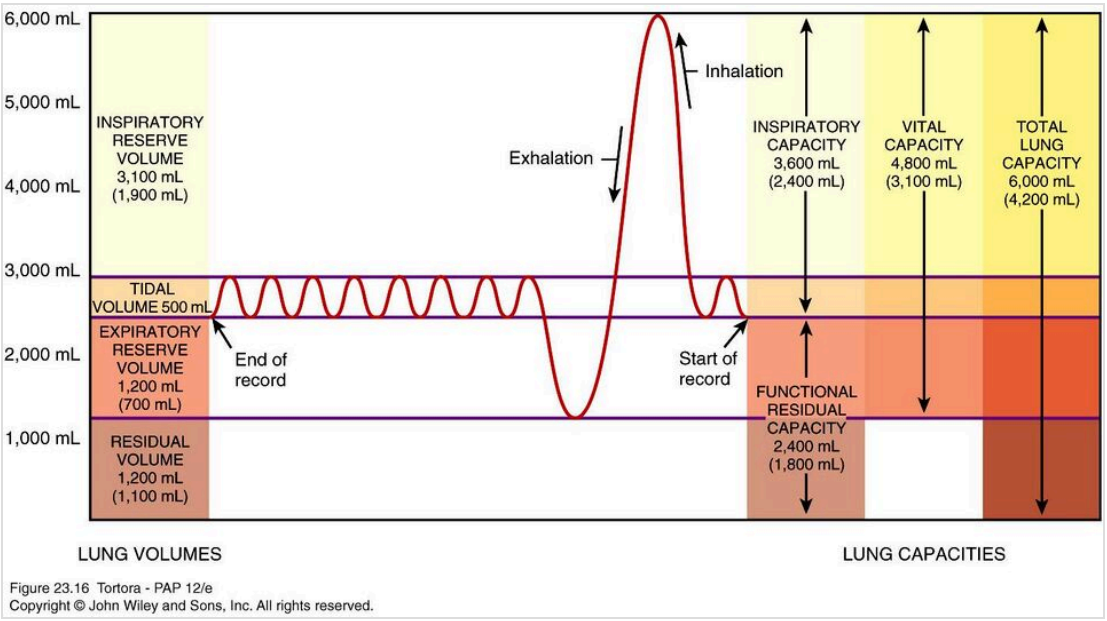
Pulmonary Volumes and Capacities

Pulmonary volumes are measured by a **spirometer**. The **normal breathing rate** is 12-20 cycles per minute, which totals to 17000+ breaths per day.

A spirogram is a graphical representation of the respiratory volumes captured by the spirometer. Spirograms can be indicative of certain respiratory disorders or of healthy lung function.



Spirometer



The numbers in the parentheses are for women.

Pulmonary Volumes

- **Tidal Volume** (TV) – the volume of air inspired or expired in a regular breath (500 mL)
- **Inspiratory Reserve Volume** (IRV) – the maximum amount of air that can be inspired *after* breathing in normally (3000 mL)
- **Expiratory Reserve Volume** (ERV) – the maximum amount of air that can be expired *after* breathing out normally (1100 mL)
- **Residual Volume** (RV) – the amount of air still remaining in the respiratory passages and lungs after a maximum expiration (1200 mL)

Pulmonary Capacities

- **Functional Residual Capacity** (FRC) – ERV + RV
- **Inspiratory Capacity** (IC) – TV + IRV
- **Vital Capacity** (VC) – IRV + TV + ERV
- **Total Lung Capacity** (TLC) – IRV + ERV + TV + RV (normally 6000mL in a healthy male adult)
- **Pulmonary Ventilation** – TV * respiratory rate (the total volume of gas entering the lungs per minute)
- **Alveolar ventilation** = (TV - dead space) * respiratory rate (the volume of gas per unit time that reaches the alveoli)

- **Dead space ventilation** = dead space * respiratory rate (the volume of gas per unit time that does not reach these respiratory portions, but instead remains in the airways [trachea, bronchi, etc.])

Control of Breathing

Unlike digestion, pupil contraction, and the beating of the heart, breathing can be controlled both unconsciously and consciously. Few other actions are like this (blinking and coughing are two other examples). When an individual is not actively thinking about breathing, such as when the body is at rest, the body begins to unconsciously regulate air intake. When not being actively controlled, breathing is controlled by the brain stem, specifically the medulla oblongata and pons. The brain stem will automatically regulate breathing as needed by the body. For instance, when exercising, the body needs ATP for muscle contraction to occur. When strength training, the body enters the zone called **anaerobic exercise**. This triggers anaerobic metabolism, which causes a buildup of lactic acid in the body. This lowers blood pH, which can be fatal. The brain stem then increases the rate of breathing.

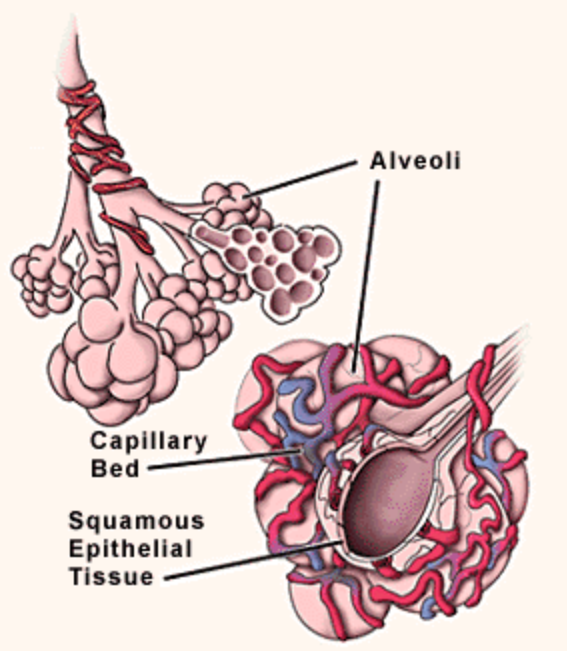
However, when performing activities such as pranayama yoga, the individual is consciously deciding when to breathe. Conscious breathing control comes from the cerebral cortex, though this control can be overridden by chemoreceptor reflexes.

High altitude

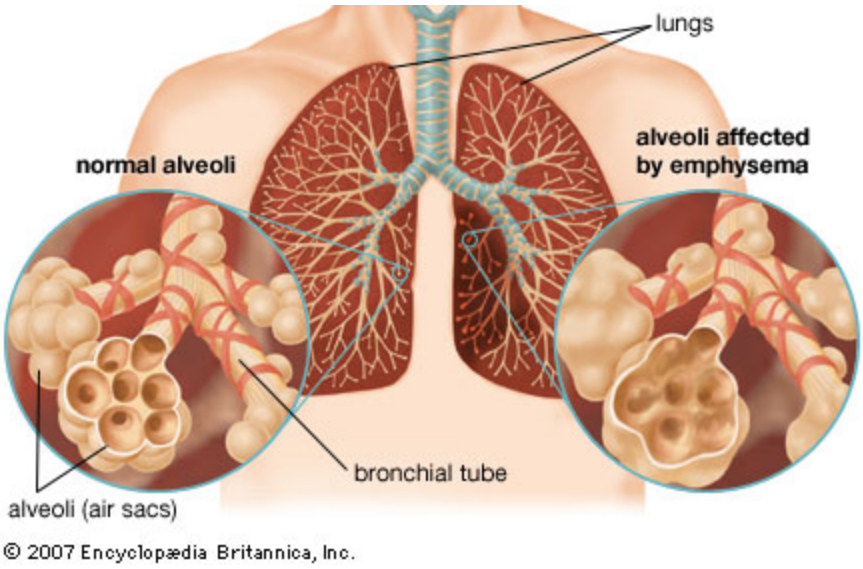
High altitude causes decrease in the amount of oxygen. With high altitude it can lead to hypoxia. Hypoxia has effects on body weight, muscle structure and exercise capacity, mental functioning, and sleep quality. The most important is hyperventilation which is achieved by hypoxic ventilatory response of the peripheral chemoreceptors. Hyperventilation results in increase in arterial carbondioxide concentration. Altitude also affects sleep and cardiac output, which is the other determinant of oxygen delivery. When exposed to high altitude, the resting pulse rate increases rapidly, but with acclimatization, heart rate and cardiac output tend to fall. Another important component that leads to decrease in cardiac output is the reduction in the stroke volume with

acclimatization. The levels of CO_2 in the blood can drop and this can switch off the drive to breathe. After the body senses a further drop in O_2 levels breathing is started again. Periodic breathing is a result from instability in the control system through the hypoxic drive or the response to CO_2.

Gas Exchange and Transport



Gas exchange



1. Oxygen in air dissolves into fluid lining the alveolus and diffuses through alveolar wall and capillary wall.
2. Oxygen enters blood plasma inside capillary. Gas exchange occurs in the capillary beds.
3. Oxygen binds to hemoglobin in red blood cells.
4. Carbon dioxide diffuses out of blood plasma and into air in alveolus.
5. Oxygenated blood leaves heart and circulates through capillaries to tissue.
6. Red blood cells arrive at tissue, rich in oxygen that binds to the hemoglobin in the tissue.
7. Oxygen leaves hemoglobin in red blood cells and diffuses across blood capillary walls and into tissue cells.
8. Carbon dioxide diffuses out of tissue into blood plasma, which carries red blood cells into lungs. Completely dissociated hemoglobin enters the lung in red blood cells ready to be oxygenated.

Transport of gases in the blood is essential to life. The gases are carried in the blood in one of two ways: in the blood plasma or combined with hemoglobin.

Chloride shift

The chloride shift is a process that brings safe carbon dioxide back to the lungs, using bicarbonate ions. In a red blood cell, carbon dioxide needs to go back into the lungs to be exhaled out. The bicarbonate ions are the one to carry carbon dioxide, so bicarbonate also needs to go into the lungs. Because bicarbonate would create an imbalance in the red blood cell if it leaves, something takes its place, chloride. If bicarbonate leaves, chloride comes in. This happens because they are on the opposite sides of the pH scale and one is pumped out to let other one in. This creates an electrical balance but is safe to carry out for the lungs.

Diseases of the Respiratory System

Acute Rhinitis

Also known as the common cold. The common symptoms are a runny nose, wheezing, teary eyes, and impairment of smell. It is caused by bacterial/viral infections of the nose as well as inhaled irritants.

Asthma

Asthma is caused by inflammation in the airways. When an asthma attack occurs, the muscles surrounding the airways become tight and the lining of the air passages swells. Childhood asthma occurs more frequently in boys than in girls (the young male's airway size is smaller when compared to female's airway, until around age 20, when ratio of asthma between men and women is roughly the same). It is also caused by a genetic predisposition and general sensitivity (40-50% of children with eczema develop asthma). The common triggers are generally the same as those of allergies: Animals, dust, changes in weather (most often cold weather), chemicals in the air or in food, exercise, mold, pollen, respiratory infections, stress, tobacco smoke, aspirin & other nonsteroidal anti-inflammatory drugs (NSAIDs).

Symptoms include a cough with or without sputum production, pulling in of the skin between the ribs when breathing, shortness of breath that worsens with exercise or activity, wheezing, abnormal breathing pattern (expiration is 2x as long as inspiration), apnea, and chest pain or tightness.

The goal of treatment is to avoid the substances that trigger symptoms and control airway inflammation. There are two basic kinds of medication: Control drugs to prevent attacks (take them daily even if you feel fine) and quick-relief “rescue” drugs for use during attacks (work fast to control symptoms; take them when you are coughing, wheezing, having trouble breathing, just before exercise, or during an attack; usually an inhaler).

Chronic Obstructive Pulmonary Diseases (COPD)

COPDs are very common lung diseases. The 2 primary forms of COPD are chronic bronchitis and emphysema; most sufferers have a combination of both conditions. COPDs are characterized by a gradual (and irreversible) decrease in the ability to push air out of the lungs due to decreased lung

compliance and elasticity. Affected individuals also experience labored breathing, called dyspnea. COPDs can be caused by smoking (the best form of prevention is not smoking), but can also be caused by rare genetic mutations (alpha-1 antitrypsin deficiency) or exposure to airborne irritants.

In **chronic bronchitis**, the bronchi become inflamed, and goblet cells produce unusually high amounts of mucus. This increases airway resistance and therefore gas exchange and pulmonary ventilation are impaired.

Emphysema

Emphysema is a disease in which lung tissue and alveolar walls are destroyed, decreasing the surface area available for gas exchange in the alveoli. The enlarged alveoli also damage pulmonary capillaries, increasing resistance to blood flow and forcing the right ventricle of the heart to work harder and enlarge due to the strain. The lungs eventually lose their elasticity, forcing the affected individual to use accessory respiratory muscles (intercostals and abdominal muscles) to breathe. As a result, emphysema patients have labored breathing more so than chronic bronchitis patients. Also, bronchial airways open during inspiration but collapse during expiration, trapping air in the alveoli (creating alveolar dead spaces) and hyperinflating the lungs. This condition causes the affected individual's chest to seem "inflated," hence the appearance is called "barrel chest." The condition is caused by long-term exposure to airborne irritants.

Cystic fibrosis

A life-threatening disease passed down through families that causes thick, sticky mucus to build up in the lungs, digestive tract (especially the pancreas), and other areas of the body. It is one of the most common chronic lung diseases in children & young adults. The average life span of someone afflicted with cystic fibrosis is 66 years.

Caused by a defective gene, it results in life-threatening lung infections and serious digestion problems, and may also affect the sweat glands. Millions of Americans carry the defective CF gene, but do not have any symptoms, because a person with CF must inherit two defective CF genes - one from each parent (1 in 29 white Americans have the CF gene). CF is the most common, deadly inherited disorder affecting Caucasians in the US and is more common among those of Northern or Central European descent. Most children with CF are diagnosed by age 2.

Symptoms include fever, increased coughing and shortness of breath, loss of appetite, increased sputum, and sinus pain/pressure caused by infection or polyps.

There is no cure, but treatment includes antibiotics to prevent and treat lung and sinus infections, inhaled medicines to help open the airways, DNase enzyme therapy to thin mucus & make it easier to cough up, lung transplants in some cases, and oxygen therapy. It can also be relieved by avoiding smoke, dust, dirt, fumes, household chemicals, fireplace smoke, & mold or mildew, clearing or bringing up mucus or secretions from the airways (1-4x/day), drinking plenty of fluids, and exercising 2-3 times a week. There is no prevention, but the gene may be detected with screening.

Hypoxia

Hypoxia is a condition where the whole body or a certain region of the body is deprived of oxygen. The classic symptom of hypoxia is cyanosis (skin turning blue), but it is not characteristic of all cases of hypoxia and is usually visible only in fair-skinned individuals. Treatment differs by type of hypoxia, of which there are several:

1. Anemic hypoxia: characterized by an abnormally low number of red blood cells (RBCs) in the blood or RBCs with too little hemoglobin (Hb).
2. Ischemic hypoxia: characterized by an abnormal blood flow due to partial or complete blood vessel blockage.
3. Histotoxic hypoxia: characterized by the inability of body cells to metabolize oxygen even though adequate amounts of it are delivered. This type of hypoxia is caused by metabolic poisons (those that mess with cell metabolism), a common one being cyanide.
4. Hypoxemic hypoxia: characterized by abnormally low partial pressures of oxygen in the blood. Most commonly, it is caused by breathing in air which contains too little oxygen. Carbon monoxide poisoning is a special type of hypoxemic hypoxia. Carbon monoxide (CO) displaces oxygen in RBCs very easily since the affinity of hemoglobin for carbon monoxide is about 200 times greater than that for oxygen. CO poisoning does not produce the usual symptoms of hypoxia. Instead, the affected individual experiences headache and confusion. This condition is treated using hyperbaric therapy or by breathing pure oxygen until the CO has cleared the the body.

Oxygen Toxicity

Oxygen Toxicity is a result from breathing molecular (pure) oxygen at high pressures (such as from oxygen tanks in scuba diving) for a considerable period of time. Oxygen toxicity can affect the central nervous system and pulmonary functions.

Lung Cancer

Cancer of the lungs is the leading cause of cancer deaths. Symptoms of lung cancer include a constant cough, shortness of breath, and infections such as bronchitis and pneumonia. Treatments (not cures) include chemotherapy, surgery, and radiation therapy.

Laryngitis

Laryngitis is inflammation of the vocal folds. This is usually caused by viruses. Symptoms include dysphonia (loss or hoarseness of voice). Can be acute (<3 weeks) or chronic.

Pneumonia

Pneumonia is an infection of the lung. Community-acquired pneumonia is ordinary pneumonia; hospital-acquired pneumonia is acquired in a hospital. It is a common illness that affects millions of people each year in the United States.

It can have multiple causes: Bacteria (the most serious and common cause – common kinds are *Streptococcus pneumoniae* (pneumococcus), *Legionella pneumophila*, *Mycoplasma pneumoniae*, *Chlamydophila pneumoniae* (atypical/walking pneumonia)), viruses (esp. in infants & young children), and fungi (*Pneumocystis jirovecii* (immune-compromised)). Symptoms include a cough (with some pneumonias you may cough up greenish or yellow mucus, or even bloody mucus), fever (mild or high), shaking chills, shortness of breath, confusion (esp. in older people), excess sweating and clammy skin, headache, loss of appetite, low energy, fatigue, and sharp or stabbing chest pains.

It can be treated with IV fluids and antibiotics, oxygen therapy, regular antibiotics, humid air, and deep breathing. Precautions one can take are hand-washing, not smoking, and vaccinations.

Sleep apnea

Sleep apnea is when breathing repeatedly stops and starts while sleeping.

There are three main types: **obstructive sleep apnea** (the more common form, which occurs when throat muscles relax), **central sleep apnea** (which occurs when the brain doesn't send proper signals to the breathing muscles), and **complex sleep apnea syndrome** (a combination of the other two).

Obstructive sleep apnea is caused by the muscles in the back of the throat supporting the soft palate, uvula, tonsils, and tongue relax, narrowing or closing the airway during inspiration. Many people with this type of sleep apnea think they sleep well all night.

Central sleep apnea occurs when the brain fails to transmit signals to breathing muscles and is most commonly caused by heart disease (it can also be, less commonly, caused by a stroke). Patients with central sleep apnea are more likely to remember waking up than those with obstructive.

Complex sleep apnea syndrome is caused by an upper airway obstruction like obstructive, but patients also have problems with the rhythm of breathing and occasional lapses of breathing effort.

Symptoms include daytime hypersomnia, loud snoring (more common in obstructive), observed episodes of breathing cessation during sleep, abrupt awakenings accompanied by shortness of breath (more common in obstructive), awakening with a dry mouth or sore throat, morning headache, insomnia.

For obstructive sleep apnea, treatment includes a continuous positive airway pressure (CPAP - machine that delivers air pressure through a mask placed over your nose while you sleep), adjustable airway pressure devices, oral appliances, and surgery (removes excess tissue from nose/throat that may be vibrating - examples include uvulopalatopharyngoplasty (UPPP), maxillomandibular advancement, tracheostomy, nasal surgery to remove polyps or correct a deviated nasal septum, surgery to remove enlarged tonsils or adenoids).

For central and complex sleep apnea, the patient may be treated for the medical problem at the root of the cause, or may be given supplemental oxygen, a CPAP, bilevel positive airway pressure (BPAP - a device that builds to a higher pressure when you inhale and decreases to a lower pressure when you exhale), adaptive servo-ventilation (ASV - a device that learns your normal breathing pattern and pressure to normalize breathing pattern and prevent pauses in breathing).

Tuberculosis

Tuberculosis is a contagious bacterial infection of the lungs (but can spread to other organs). Tuberculosis is shown by weight loss, excessive sweating, and coughing up blood. It can be treated with drugs such as Isoniazid and Rifampin.

Mycobacterium tuberculosis has a cell wall with a lot of mycolic acid, making it highly resistant to common antibiotics and the immune system. It is also an obligate aerobic, which is why it infects the lungs.

Alveolar macrophages engulf the TB bacteria, but the bacteria survive the insides of the macrophage. Therefore, the immune system builds a cellular wall around infected macrophages, called **tubercules** or **granulomas**. Over time, these calcified granulomas and nearby lymph nodes form what is known as a **Ghon Complex**.

TB causes structural destruction and fibrosis and exhibits a restrictive lung disease pattern (decrease in VC and TV).

Nationals Topics

Blood Chemistry, Respiratory Rhythm, and Regulation of the Respiratory System

Respiration is affected by blood pH and concentrations of carbon dioxide and oxygen, which are detected by chemoreceptors. Peripheral chemoreceptors in the arteries and aorta detect partial pressures of oxygen (PaO_2), but only at extremely low levels, so oxygen concentration does not have a large impact on ventilation.

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