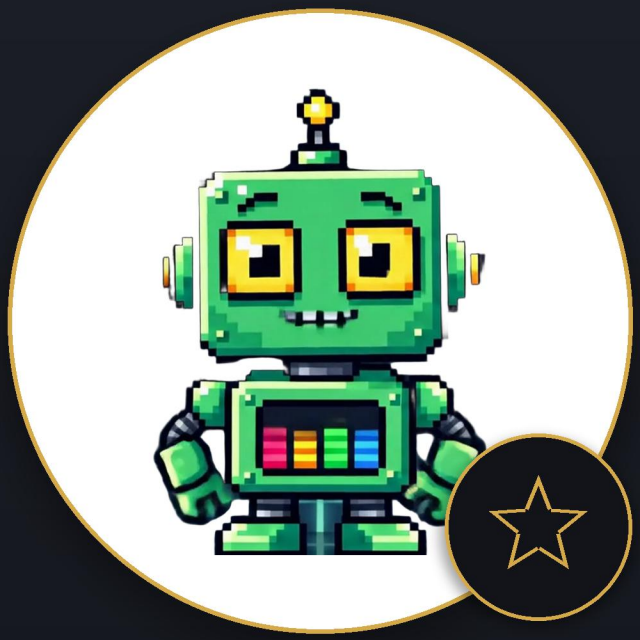
BIT BY BIT MD

THE C-FACTOR SERIES

BOOK 7

THE FINAL REVIEW

All-Subject High-Yield



Mechanism-First · MCAT + USMLE Step 1 Pre-Prep

2026 EDITION

Bit by Bit MD · The C-Factor Series

Chapter 0 · The Final Review: Complete High-Yield Compression for MCAT and Premed

Six books of mechanism, distilled to one volume of rapid review

Books 1 through 6 built the mechanistic scaffolding of every premedical subject. This volume is the rapid-retrieval companion. It compresses the highest-yield content from biology, general chemistry, organic chemistry, physics, psychology and sociology, and biochemistry into one bullet-and-table-dense reference. The goal is not to teach the material from scratch but to make every concept retrievable in seconds. Use this book after the others, in the final weeks before the exam, for spaced retrieval and last-pass consolidation. Every page is built for speed.

Learning Objectives

- Retrieve every high-yield premedical concept in seconds, not minutes
- Master the discriminator pairs that account for most question-bank errors
- Consolidate six books of mechanism into one workable last-pass tool
- Anchor MCAT test-day reasoning to the smallest possible set of governing principles
- Identify weak spots quickly using the master discriminator index
- Walk into test day with the entire premedical canon at fingertip speed

Six books, one cheat sheet. This is not the place you learn the material. This is the place you retrieve it. If a concept here feels unfamiliar, that is a signal to return to the parent chapter in Books 1 through 6. If it feels almost familiar, this is the page that locks it in. Mechanism first, always. Then the bullet, then the table, then the discriminator, then the next item.

PART I · HOW TO USE THIS REVIEW

This volume is the rapid-review companion to The C-Factor Series. It assumes you have already worked through the parent books at least once and now need a single source of compressed truth for spaced retrieval and pre-exam consolidation. It is deliberately bullet-heavy, table-heavy, and discriminator-heavy. Prose has been stripped to the minimum needed to make a point land.

HIGH-YIELD

- Read this book in passes of one part per day in the final two weeks before the exam
- Cover the right column of every table; force recall from the left column
- Star anything that does not retrieve in under five seconds; revisit in 24 hours
- Treat every discriminator as a flashcard: name the distinction without reading the box
- Use the master discriminator index in Part VIII as a final-week diagnostic
- On test day, glance at Part IX (Test-Day Anchors) only; everything else should already be inside you

DISCRIMINATOR | THIS BOOK vs THE PARENT VOLUMES

The parent volumes (Books 1-6) teach mechanism in depth, with passages, clinical bridges, and full explanatory prose. This book strips that to the bone for retrieval. If you are encountering a concept for the first time here, you are using the book wrong. Go back to the parent chapter, learn the mechanism, then return here to compress and consolidate. Reading this book in isolation produces recognition without understanding, which is exactly what the MCAT does not reward.

WHY DENSITY WORKS IN THE FINAL WEEKS

In the consolidation phase of exam prep, the limiting factor is not exposure to new ideas but rapid retrieval of ideas you already half-know. Dense, mechanically-organized review material maximizes the number of retrieval cues per minute of study and lets you find the weak spots fast. A bullet you cannot complete from memory is more diagnostic than a chapter you can read passively. This entire volume is built around that principle.

PART II · BIOLOGY HIGH-YIELD

II.1 The Cell

- Prokaryotes: no nucleus, no membrane-bound organelles, circular DNA in nucleoid, 70S ribosomes, peptidoglycan cell wall (bacteria) or pseudopeptidoglycan (archaea)
- Eukaryotes: nucleus, membrane-bound organelles, linear DNA in chromosomes, 80S ribosomes, no cell wall in animals
- Endosymbiotic theory: mitochondria and chloroplasts have their own circular DNA, 70S ribosomes, double membrane, divide by binary fission
- Cytoskeleton: microfilaments (actin, 7 nm), intermediate filaments (10 nm), microtubules (tubulin, 25 nm)

Organelle	Function	Distinguishing feature
Nucleus	DNA storage, transcription	Double membrane with pores; nucleolus inside makes rRNA
Rough ER	Protein synthesis for secretion or membrane	Studded with ribosomes; continuous with nuclear envelope
Smooth ER	Lipid synthesis, detoxification, Ca^{2+} storage	No ribosomes; prominent in liver and gonads

Organelle	Function	Distinguishing feature
Golgi apparatus	Modifies, sorts, packages proteins	Cis face receives, trans face ships; glycosylation site
Lysosome	Hydrolytic digestion	Acidic pH (~4.5); contains acid hydrolases
Peroxisome	Fatty acid β-oxidation (very long chain), H_2O_2 metabolism	Contains catalase
Mitochondrion	Oxidative phosphorylation, TCA, β-oxidation	Double membrane, own DNA, cristae
Ribosome	Protein synthesis	80S (euk) or 70S (prok); rRNA + protein
Centrosome	Microtubule organizing center	Two centrioles at right angles in animal cells
Proteasome	Ubiquitin-tagged protein degradation	Barrel-shaped, ATP-dependent

II.2 Membrane Transport

Type	Down/Against gradient	ATP required	Carrier?
Simple diffusion	Down	No	No (small, nonpolar)
Facilitated diffusion	Down	No	Yes (channel or carrier)
Primary active transport	Against	Yes (direct)	Yes (e.g., Na^+/K^+ ATPase)
Secondary active transport	Against (one solute)	No direct ATP; uses gradient	Yes (symport or antiport)
Osmosis	Down (water)	No	Aquaporins (facilitated)
Endo/exocytosis	Bulk	Yes	Vesicle-mediated

- Na^+/K^+ ATPase: 3 Na^+ out, 2 K^+ in per ATP; maintains resting membrane potential
- SGLT (Na^+-glucose cotransport): secondary active in intestine and kidney
- GLUT transporters: facilitated diffusion of glucose; GLUT4 is insulin-responsive in muscle and adipose

II.3 Cell Division and Cancer

Phase	Key event
G1	Cell growth; R (restriction) point checkpoint
S	DNA replication; sister chromatids form
G2	Growth; G2/M checkpoint (DNA damage check)
Prophase	Chromosomes condense; nuclear envelope breaks down; spindle forms
Metaphase	Chromosomes align at metaphase plate; M checkpoint
Anaphase	Sister chromatids separate to opposite poles
Telophase	Nuclear envelopes reform; chromosomes decondense

Phase	Key event
Cytokinesis	Cytoplasm divides (cleavage furrow in animals, cell plate in plants)

- Mitosis: 1 diploid parent → 2 diploid identical daughters; somatic cells
- Meiosis: 1 diploid parent → 4 haploid genetically unique daughters; gametes
- Meiosis I: homologous chromosomes separate (reductional); crossing over in prophase I
- Meiosis II: sister chromatids separate (equational); resembles mitosis
- Sources of variation: crossing over (prophase I), independent assortment (metaphase I), random fertilization

DISCRIMINATOR | MITOSIS vs MEIOSIS I vs MEIOSIS II

MITOSIS: sister chromatids separate, daughters are diploid clones. MEIOSIS I: HOMOLOGOUS chromosomes separate (the reductional division); chromosome number halves. MEIOSIS II: sister chromatids separate (looks like mitosis but on haploid cells). If chromosome number halves, it is meiosis I. If chromatids separate, it is mitosis or meiosis II. If both occur in haploid cells, it is meiosis II.

- Hallmarks of cancer: sustained proliferative signaling, evasion of growth suppressors, resisting cell death, replicative immortality, angiogenesis, invasion/metastasis, altered metabolism, immune evasion, genomic instability, inflammation
- Proto-oncogenes (gain-of-function → oncogene): Ras, Myc, HER2, Bcr-Abl
- Tumor suppressors (loss-of-function): p53 (the guardian), Rb, BRCA1/2, APC
- p53: activated by DNA damage; induces p21 → CDK inhibition → G1 arrest; if damage severe, apoptosis

II.4 Genetics

Pattern	Key features
Autosomal dominant	Affects both sexes, no skipping generations, ~50% offspring of affected
Autosomal recessive	Can skip generations, two carriers can have affected child
X-linked recessive	Mostly males affected; carrier mother to son; no male-to-male transmission
X-linked dominant	No male-to-male; affected fathers pass to all daughters
Mitochondrial	Maternal only; all children of affected mother affected
Y-linked	Father to all sons only

- Codominance: both alleles expressed (ABO blood: AB type)
- Incomplete dominance: blended phenotype (red × white → pink)
- Penetrance: % with genotype that express phenotype
- Expressivity: degree of expression in those who do
- Pleiotropy: one gene, multiple phenotypes (sickle cell)

- Epistasis: one gene masks expression of another
- Linkage: genes on same chromosome inherited together unless crossing over separates them

HIGH-YIELD

- Hardy-Weinberg equilibrium: $p^2 + 2pq + q^2 = 1$; $p + q = 1$
- Assumptions: no mutation, no selection, no migration, random mating, large population
- If q^2 = disease frequency, q = sqrt(disease freq), p = 1 - q, carrier freq = 2pq
- Punnett squares: monohybrid 3:1 (dominant phenotype), dihybrid 9:3:3:1
- Test cross: cross with homozygous recessive to determine unknown genotype

II.5 Molecular Biology

- DNA: double helix, antiparallel, 5' to 3', A-T (2 H-bonds), G-C (3 H-bonds), sugar = deoxyribose
- RNA: single-stranded, U replaces T, sugar = ribose, 2' OH
- Histones: positively charged (lysine, arginine); wrap DNA; acetylation loosens, methylation often condenses
- Replication: semiconservative; origin → bidirectional forks; helicase unwinds; SSB stabilizes; topoisomerase relieves supercoils
- Leading strand: continuous, 5'→3' toward fork
- Lagging strand: discontinuous, Okazaki fragments, primase lays RNA primer, DNA pol I removes primer, ligase seals
- Telomeres: TTAGGG repeats; telomerase extends in germ cells/stem cells/cancer

Process	Enzyme	Direction	Template
DNA replication	DNA polymerase III (prok), δ/ε (euk)	5' → 3'	DNA
Transcription	RNA polymerase (I=rRNA, II=mRNA, III=tRNA)	5' → 3'	DNA (antisense strand)
Translation	Ribosome (peptidyl transferase = rRNA, a ribozyme)	N → C terminus	mRNA (5' → 3')
Reverse transcription	Reverse transcriptase	5' → 3' DNA	RNA

- mRNA processing (eukaryotes): 5' cap (7-methylguanosine), 3' poly-A tail, splicing removes introns
- Splicing: spliceosome (snRNPs) recognizes GU at 5' splice site and AG at 3'; forms lariat
- Alternative splicing: one gene, multiple proteins
- Genetic code: triplet, degenerate (multiple codons per AA), nearly universal, no overlap, no punctuation; start = AUG (Met); stop = UAA, UAG, UGA
- Mutations: silent (same AA), missense (different AA), nonsense (premature stop), frameshift (insertion/deletion not multiple of 3)

DISCRIMINATOR | OPERONS (prokaryotes) vs EUKARYOTIC GENE REGULATION

OPERONS: polycistronic mRNA, regulated as one unit (lac, trp); lac operon induced by allolactose (removes lac repressor) AND glucose absent (cAMP-CAP binds). EUKARYOTES: monocistronic mRNA; regulation at chromatin (histone acetylation opens; methylation often closes), transcription factor binding to enhancers/promoters, alternative splicing, mRNA stability, translation rate, and post-translational modification. Operons toggle whole pathways; eukaryotic regulation is combinatorial and layered.

II.6 Embryology

Germ layer	Forms
Ectoderm	Epidermis, hair, nails, nervous system, neural crest (PNS, melanocytes, adrenal medulla), lens, inner ear
Mesoderm	Muscle, bone, cartilage, blood, heart, kidneys, gonads, dermis, connective tissue
Endoderm	Gut epithelium, liver, pancreas, lungs, thyroid, bladder lining

- Cleavage: rapid mitosis without growth → morula → blastula (blastocyst in mammals)
- Gastrulation: forms three germ layers; primitive streak in birds/mammals
- Neurulation: notochord induces ectoderm → neural plate → neural tube (CNS) + neural crest
- Determination: cell fate fixed; Differentiation: cell expresses phenotype
- Induction: one tissue influences another's fate (notochord → neural tube)
- Apoptosis: programmed cell death; sculpts fingers, removes excess neurons

II.7 Endocrine System

Hormone class	Examples	Receptor location	Mechanism
Peptide	Insulin, glucagon, GH, ADH, oxytocin, PTH, calcitonin	Cell surface	Second messenger (cAMP, IP3, DAG)
Steroid	Cortisol, aldosterone, estrogen, testosterone, progesterone, vitamin D	Cytoplasmic or nuclear	Direct gene transcription
Amine (catecholamine)	Epinephrine, norepinephrine	Cell surface (α, β)	cAMP (β) or IP3 (α1) or ↓cAMP (α2)
Amine (thyroid)	T3, T4	Nuclear	Gene transcription (acts like steroid)

- HPA axis: hypothalamus CRH → ant pituitary ACTH → adrenal cortex cortisol
- HPG axis: hypothalamus GnRH → ant pituitary LH/FSH → gonads sex steroids
- HPT axis: hypothalamus TRH → ant pituitary TSH → thyroid T3/T4
- Posterior pituitary: stores ADH and oxytocin (made in hypothalamus)

- Anterior pituitary hormones (FLAT PEG): FSH, LH, ACTH, TSH, Prolactin, Endorphins, GH
- Insulin: β cells, fed state, anabolic, glucose into cells via GLUT4 in muscle/fat
- Glucagon: α cells, fasting, catabolic, glycogenolysis and gluconeogenesis
- Calcium: PTH ↑ Ca^{2+} (bone resorption, renal reabsorption, activates vit D); Calcitonin ↓ Ca^{2+}; Vit D ↑ intestinal Ca^{2+} absorption

II.8 Nervous System

- Neuron: dendrites receive, soma integrates, axon transmits, axon terminal releases neurotransmitter
- Resting potential: ~ -70 mV; maintained by Na^+/K^+ ATPase and K^+ leak channels
- Action potential: depolarization (Na^+ in) → repolarization (K^+ out) → hyperpolarization (K^+ still out) → return to rest
- All-or-none; refractory periods: absolute (no AP possible) then relative (stronger stimulus needed)
- Myelin (oligodendrocytes in CNS, Schwann in PNS) → saltatory conduction at nodes of Ranvier → faster
- Synapse: AP → Ca^{2+} in presynaptic terminal → vesicle fusion → NT release → postsynaptic receptor

Neurotransmitter	Source	Effect
Acetylcholine (ACh)	NMJ, parasympathetic, preganglionic sympathetic, CNS	Muscle contraction; ↓ in Alzheimer
Dopamine	Substantia nigra, VTA	Movement (↓ Parkinson), reward, motivation; ↑ in schizophrenia
Norepinephrine	Locus coeruleus, sympathetic post	Arousal, fight or flight
Serotonin (5-HT)	Raphe nuclei	Mood, appetite, sleep; ↓ in depression
GABA	CNS interneurons	Main inhibitory; opens Cl^- channels
Glutamate	CNS	Main excitatory; learning (NMDA)
Glycine	Spinal cord	Inhibitory in spinal cord
Endorphins	Pituitary, CNS	Pain modulation

- CNS = brain + spinal cord; PNS = everything else
- Somatic: voluntary, skeletal muscle; Autonomic: involuntary, smooth/cardiac/glands
- Sympathetic (fight or flight): thoracolumbar, short pre/long post, NE (most), epi (adrenal medulla)
- Parasympathetic (rest and digest): craniosacral, long pre/short post, ACh throughout
- Reflex arc: sensory neuron → spinal cord (interneuron) → motor neuron; monosynaptic = stretch reflex

II.9 Cardiovascular System

- Path of blood: RA → tricuspid → RV → pulmonary valve → pulm artery → lungs → pulm veins → LA → mitral (bicuspid) → LV → aortic valve → aorta → body → vena cava → RA
- Pulmonary artery carries deoxygenated blood; pulmonary vein carries oxygenated

- SA node: pacemaker (~70 bpm) → AV node (delay) → bundle of His → Purkinje fibers
- Cardiac muscle: striated, single nucleus, intercalated discs with gap junctions
- Cardiac cycle: systole (ventricular contraction) and diastole (ventricular relaxation, filling)
- Stroke volume = EDV - ESV; CO = SV × HR; MAP = CO × TPR
- Frank-Starling: ↑ EDV → ↑ stretch → ↑ contraction force

ECG wave	Represents
P wave	Atrial depolarization
PR interval	AV node delay
QRS complex	Ventricular depolarization (atrial repol hidden)
ST segment	Ventricular plateau
T wave	Ventricular repolarization

- Starling forces (capillary): hydrostatic pressure pushes OUT; oncotic (colloid osmotic) pressure pulls IN
- Net filtration favors out at arteriolar end, in at venular end; lymphatics return excess
- Edema causes: ↑ hydrostatic (heart failure), ↓ oncotic (nephrotic, liver failure), ↑ permeability (inflammation), lymph blockage
- Blood pressure regulation: baroreceptors (carotid, aortic arch), RAAS, ADH, ANP
- Hematocrit: ~45% (% RBCs); plasma ~55%

CLINICAL BRIDGE | HOW BLOOD PRESSURE IS DEFENDED

When BP drops, baroreceptors fire less → ↑ sympathetic → ↑ HR, ↑ contractility, vasoconstriction. Simultaneously, juxtaglomerular cells release renin → angiotensin I → ACE in lungs → angiotensin II (potent vasoconstrictor, stimulates aldosterone → Na^+ and water retention, stimulates ADH from posterior pituitary → water retention). Net effect: ↑ volume, ↑ TPR, ↑ MAP. ACE inhibitors and ARBs target this axis.

II.10 Respiratory System

- Path: nose → pharynx → larynx → trachea → bronchi → bronchioles → alveoli (gas exchange)
- Diaphragm contracts → thoracic volume ↑ → P ↓ → air in (inspiration is active); expiration is passive at rest
- Surfactant (type II pneumocytes): ↓ surface tension; deficient in premature infants → RDS

Lung volume/capacity	Definition
Tidal volume (TV)	Volume per normal breath (~500 mL)
Inspiratory reserve (IRV)	Max additional inhalation above TV
Expiratory reserve (ERV)	Max additional exhalation below TV
Residual volume (RV)	Air remaining after max exhalation (cannot be measured by spirometry)
Vital capacity (VC)	TV + IRV + ERV (max exhale after max inhale)

Lung volume/capacity	Definition
Total lung capacity (TLC)	VC + RV
Functional residual capacity (FRC)	ERV + RV (after normal exhale)

- O_2 carried 98% on hemoglobin (Hb), 2% dissolved; CO_2 ~70% as HCO_3^-, ~20% on Hb (carbamino), ~10% dissolved
- Oxygen-hemoglobin dissociation curve: sigmoid (cooperative binding); P50 ~27 mmHg
- Right shift (↓ affinity, releases O_2): ↑ CO_2, ↓ pH, ↑ temperature, ↑ 2,3-BPG (Bohr effect)
- Left shift (↑ affinity, holds O_2): ↓ CO_2, ↑ pH, ↓ temperature, ↓ 2,3-BPG, fetal Hb, CO poisoning
- Chloride shift: in RBC at tissues, HCO_3^- out, Cl^- in

II.11 Renal System

Nephron segment	What it does
Glomerulus	Filtration (size and charge selective)
Proximal convoluted tubule (PCT)	Bulk reabsorption: 65% Na^+/H_2O, all glucose/AAs, most HCO_3^-
Descending loop of Henle	Water reabsorption only (permeable to H_2O, not solutes)
Ascending loop of Henle	Solute reabsorption only ($Na^+/K^+/2Cl^-$ symporter); impermeable to H_2O
Distal convoluted tubule (DCT)	Na^+/Cl^- reabsorption; Ca^{2+} reabsorption (PTH-regulated)
Collecting duct	ADH-regulated water reabsorption; aldosterone-regulated Na^+/K^+

- RAAS: low BP/Na^+/perfusion → renin from JG cells → angiotensin I → ACE → angiotensin II → vasoconstriction + aldosterone (↑ Na^+ reabsorption) + ADH
- ADH (vasopressin): inserts aquaporins in collecting duct → water reabsorbed → concentrated urine
- Aldosterone: ↑ Na^+ in, ↑ K^+ out, ↑ H^+ out (principal cells)
- ANP: opposes RAAS; released by atrial stretch; ↑ Na^+ excretion, vasodilation
- Counter-current multiplier: loop of Henle creates medullary gradient; vasa recta preserves it

Acid-base disorder	Primary change	Compensation
Metabolic acidosis	↓ HCO_3^-	Hyperventilation (↓ PCO_2)
Metabolic alkalosis	↑ HCO_3^-	Hypoventilation (↑ PCO_2)
Respiratory acidosis	↑ PCO_2	Renal ↑ HCO_3^- reabsorption (slow)
Respiratory alkalosis	↓ PCO_2	Renal ↓ HCO_3^- reabsorption (slow)

- Anion gap = Na^+ - (Cl^- + HCO_3^-); normal ~8-12
- High anion gap acidosis (MUDPILES): Methanol, Uremia, DKA, Propylene glycol, Iron/INH, Lactic acidosis, Ethylene glycol, Salicylates

- Henderson-Hasselbalch: $pH = 6.1 + \log([HCO_3^-]/(0.03 \times PCO_2))$

II.12 Digestive System

Enzyme	Source	Substrate	Optimal pH
Salivary amylase	Salivary glands	Starch	~7 (neutral)
Pepsin	Chief cells (stomach)	Proteins	~2 (acidic)
Pancreatic amylase	Pancreas	Starch	~8 (alkaline)
Trypsin/chymotrypsin	Pancreas (zymogens)	Proteins	~8
Lipase	Pancreas	Triglycerides	~8
Lactase, sucrase, maltase	Small intestine brush border	Disaccharides	~7
Enteropeptidase (enterokinase)	Duodenum	Activates trypsinogen	~8

- Stomach: HCl from parietal cells (proton pump), intrinsic factor (B12 absorption) also from parietal
- Gastrin: G cells $\rightarrow \uparrow$ HCl secretion
- Secretin: duodenum $\rightarrow \uparrow$ pancreatic HCO_3^- (neutralizes acid chyme)
- CCK: duodenum (response to fat) $\rightarrow \uparrow$ bile release from gallbladder + $\uparrow$ pancreatic enzymes
- Absorption sites: duodenum (iron, Ca), jejunum (most nutrients), ileum (B12, bile salts), colon (water, electrolytes)
- Bile: emulsifies fat; made in liver, stored in gallbladder, secreted into duodenum
- Liver: detox, plasma proteins, urea cycle, glucose homeostasis, bile, vitamin storage (A, D, E, K, B12)

II.13 Immune System

Feature	Innate	Adaptive
Specificity	Pattern-based (PAMPs)	Antigen-specific
Memory	No	Yes
Speed	Immediate	Days to weeks (first exposure)
Cells	Macrophages, neutrophils, NK, dendritic, mast, eosinophils, basophils	B cells, T cells
Molecules	Complement, cytokines, defensins	Antibodies, TCR

- T-cells: mature in thymus; cell-mediated; need MHC presentation
- CD4 helper T: recognize MHC II (on APCs); coordinate response via cytokines
- CD8 cytotoxic T: recognize MHC I (on all nucleated cells); kill infected/tumor cells
- B-cells: mature in bone marrow; humoral; produce antibodies; can be APCs
- Plasma cells: antibody factories (differentiated B-cells)

- MHC I: on all nucleated cells; presents endogenous (viral) peptides
- MHC II: on APCs only (macrophages, dendritic, B-cells); presents exogenous peptides

Antibody	Role
IgG	Most abundant; secondary response; crosses placenta
IgM	First produced in primary response; pentamer; agglutination
IgA	Mucosal (saliva, tears, milk, GI); dimer
IgE	Allergy and parasites; binds mast cells/basophils
IgD	B-cell receptor; function less clear

- Complement: classical (Ab-triggered), alternative (microbial surface), lectin (mannose)
- All pathways converge on C3 → C3a (anaphylatoxin) + C3b (opsonin) → MAC (C5-C9, lyses cell)
- Hypersensitivity: I = IgE/mast (allergy, anaphylaxis); II = Ab against cell surface; III = immune complexes; IV = T-cell delayed (TB skin test)

CLINICAL BRIDGE | VACCINATION AND HERD IMMUNITY

Vaccines expose the adaptive immune system to antigen without causing disease, generating memory B and T cells. On re-exposure, the secondary response is faster (days), stronger (IgG dominant), and longer-lasting than the primary (IgM-led) response. When enough of a population is immune, transmission chains break (herd immunity); the threshold depends on the basic reproduction number R_0. Higher R_0 requires higher vaccination coverage.

II.14 Microbiology

- Bacteria: prokaryotic; classified by shape (cocci, bacilli, spirochetes), Gram stain, O_2 requirement, metabolism
- Gram positive: thick peptidoglycan, retains crystal violet (purple); examples: Staph, Strep, Bacillus, Clostridium, Listeria
- Gram negative: thin peptidoglycan + outer membrane with LPS (endotoxin); examples: E. coli, Salmonella, Neisseria, Pseudomonas
- Aerobic, anaerobic (obligate, facultative, aerotolerant)
- Viruses: not cells; obligate intracellular; DNA or RNA, single or double-stranded; enveloped or naked
- Retroviruses: RNA → DNA via reverse transcriptase (HIV)
- Fungi: eukaryotic; chitin cell wall, ergosterol membrane; yeasts (unicellular) and molds (hyphae)
- Parasites: protozoa (Plasmodium, Giardia, Trypanosoma) and helminths (worms)
- Prions: misfolded proteins; no nucleic acid; cause CJD, mad cow

II.15 Ecology and Evolution

- Natural selection: heritable variation + differential reproduction → change in allele frequency
- Fitness: reproductive success (not survival per se)

- Selection types: directional (one extreme favored), stabilizing (mean favored), disruptive (extremes favored)
- Sexual selection: mate choice or competition; can drive traits that ↓ survival
- Genetic drift: random allele frequency change; strongest in small populations (founder effect, bottleneck)
- Gene flow: migration changes allele frequencies
- Speciation: allopatric (geographic isolation), sympatric (same area, e.g., polyploidy in plants)
- Reproductive isolation: prezygotic (temporal, behavioral, mechanical, gametic) and postzygotic (hybrid inviability, sterility)

Ecological interaction	Effect on species A	Effect on species B
Mutualism	+	+
Commensalism	+	0
Parasitism	+	-
Predation	+	-
Competition	-	-

- Food web: producers (autotrophs) → primary consumers → secondary → tertiary; ~10% energy transfer per trophic level
- r-selected: many offspring, little care, unstable environments (insects, frogs)
- K-selected: few offspring, much care, stable environments (mammals, large birds)
- Population growth: exponential $dN/dt = rN$; logistic $dN/dt = rN(1 - N/K)$

DISCRIMINATOR | DIRECTIONAL vs STABILIZING vs DISRUPTIVE SELECTION

DIRECTIONAL: one tail favored → distribution shifts (peppered moth darkening). STABILIZING: mean favored → distribution narrows (human birth weight). DISRUPTIVE: both extremes favored → bimodal distribution → can drive speciation. Match the curve, not the words.

HIGH-YIELD

- Hardy-Weinberg: $p^2 + 2pq + q^2 = 1$; assumes no selection, mutation, migration, drift, mating bias
- DNA replication: semiconservative, 5' to 3' only, leading continuous and lagging discontinuous
- Action potential: Na^+ in (depol) → K^+ out (repol); absolute then relative refractory
- Sympathetic = NE (mostly); Parasympathetic = ACh
- Right shift = release O_2 at tissues; Left shift = hold O_2 (fetal Hb, CO)
- Loop of Henle: descending water out, ascending solute out
- RAAS axis triggers: ↓ BP, ↓ Na^+, ↓ renal perfusion
- MHC I on all nucleated cells (endogenous); MHC II on APCs (exogenous)

PART III · GENERAL CHEMISTRY HIGH-YIELD

III.1 Atomic Structure and Periodic Trends

Quantum number	Symbol	Meaning	Values
Principal	n	Shell, energy level	1, 2, 3, ...
Azimuthal (angular)	l	Subshell, shape	0 to n-1 (s, p, d, f)
Magnetic	ml	Orbital orientation	-l to +l
Spin	ms	Spin direction	+1/2 or -1/2

- Pauli exclusion: no two electrons in an atom share all four quantum numbers
- Hund's rule: fill degenerate orbitals singly with parallel spins before pairing
- Aufbau: fill from lowest energy up; 1s 2s 2p 3s 3p 4s 3d 4p 5s 4d 5p ...
- Anomalous configurations: Cr [Ar] $4s^1$ $3d^5$ and Cu [Ar] $4s^1$ $3d^{10}$ (half/full d-shell stability)
- Cations: remove from highest n first (4s before 3d for transition metals)

Trend	Across period (L→R)	Down group	Why
Atomic radius	Decreases	Increases	More protons pull in (across); more shells (down)
Ionization energy	Increases	Decreases	Tighter hold (across); farther from nucleus (down)
Electron affinity	More negative (across)	Less negative (down)	Want to gain (right); harder to hold (down)
Electronegativity	Increases	Decreases	F most EN; Cs/Fr least
Metallic character	Decreases	Increases	Opposite of EN

- Effective nuclear charge: Zeff = Z - S (shielding); ↑ across period (S nearly constant, Z ↑)
- Isoelectronic species: same electron count; smaller cation, larger anion
- Exceptions in IE: small drop at group 13 (lose first p) and group 16 (lose paired p e^-)

III.2 Bonding and Intermolecular Forces

Bond type	Atoms	ΔEN
Nonpolar covalent	Two nonmetals, similar EN	<0.5
Polar covalent	Two nonmetals, different EN	0.5 - 1.7
Ionic	Metal + nonmetal	>1.7
Metallic	Metal + metal	delocalized electron sea

- Lewis structures: count valence e^-, draw skeleton, complete octets (H = 2), check formal charges
- Formal charge = valence e^- - lone pair e^- - (1/2) bonding e^-
- VSEPR: electron pairs repel; geometry by # of e^- domains
- Linear (2), trigonal planar (3), tetrahedral (4), trigonal bipyramidal (5), octahedral (6)
- Bond polarity vs molecular polarity: symmetric molecules with polar bonds can be nonpolar (CO_2, CCl_4)

Intermolecular force	Strength	Found in
Ion-dipole	Strongest IMF	Salt in water
Hydrogen bond	Strong dipole	N-H, O-H, F-H to lone pair on N, O, F
Dipole-dipole	Moderate	Polar molecules
London dispersion (van der Waals)	Weakest (but ↑ with size)	All molecules

- Stronger IMFs: ↑ boiling point, ↑ melting point, ↑ viscosity, ↑ surface tension, ↓ vapor pressure
- Water's anomalies (high BP, ice less dense than liquid, high heat capacity) all from H-bonding

III.3 Stoichiometry

- 1 mole = 6.022×10^{23} particles (Avogadro)
- Molar mass: sum of atomic masses (g/mol)
- Moles = mass / molar mass = volume × molarity (for solutions) = PV/RT (for gases at non-STP)
- Limiting reagent: the one that produces less product (run the stoichiometry from both)
- Percent yield = (actual / theoretical) × 100
- Empirical formula: simplest whole number ratio; molecular formula = (empirical) × n
- Balanced equation: same number of each atom on both sides; balance one element at a time

III.4 States of Matter and Gases

- Ideal gas law: PV = nRT (R = 0.0821 L·atm/mol·K = 8.314 J/mol·K)
- STP: 0 °C (273 K) and 1 atm; 22.4 L/mol at STP
- Boyle: $P_1V_1 = P_2V_2$ (constant T, n)
- Charles: $V_1/T_1 = V_2/T_2$ (constant P, n)
- Avogadro: $V_1/n_1 = V_2/n_2$ (constant P, T)
- Combined: $P_1V_1/T_1 = P_2V_2/T_2$
- Dalton's: P_total = ΣP_i; partial pressure P_i = X_i × P_total
- Graham's law of effusion: $rate_1/rate_2 = sqrt(M_2/M_1)$; lighter gas effuses faster

Real gas deviation	When
High pressure	Molecules close → finite volume matters → measured V > ideal V
Low temperature	Attractions matter → measured P < ideal P
Ideal behavior	Low P, high T, small nonpolar molecules

- van der Waals: $(P + a(n/V)^2)(V - nb) = nRT$; a corrects for attractions, b for volume
- Phase diagram: solid/liquid/gas regions, triple point, critical point

- Above critical point: supercritical fluid (no liquid-gas distinction)
- Water: solid-liquid line has NEGATIVE slope (ice less dense)
- Sublimation: solid → gas (e.g., dry ice, snow on cold day)

III.5 Thermodynamics

Quantity	Symbol	Sign convention
Enthalpy	ΔH	+ endothermic (absorbs heat); - exothermic (releases)
Entropy	ΔS	+ disorder ↑; - disorder ↓
Gibbs free energy	ΔG	- spontaneous; + nonspontaneous; 0 equilibrium
Heat	q	+ into system; - out of system
Work	w	+ on system; - by system (chem convention)

- ΔG = ΔH - TΔS (Gibbs equation)
- Spontaneity: ΔG < 0 always at high T if ΔH<0, ΔS>0; never if ΔH>0, ΔS<0; depends on T otherwise
- ΔG° = -RT ln K = -nFE° (links ΔG, K, and cell potential)
- Hess's law: ΔH overall = sum of ΔH steps (state function)
- 1st law: ΔU = q + w (energy conserved)
- 2nd law: ΔS_universe > 0 for spontaneous process
- 3rd law: S of perfect crystal at 0 K = 0
- Constant P: q = ΔH; Constant V: q = ΔU
- Calorimetry: q = mcΔT (sensible heat); q = mL (phase change at constant T)

III.6 Kinetics

- Rate = -Δ[reactant]/Δt = +Δ[product]/Δt (divide by coefficient)
- Rate law: rate = k[A]^x[B]^y; orders x, y determined experimentally (not from coefficients)
- Zero order: $[A] = [A]_0 - kt$; half-life depends on $[A]_0$
- First order: $\ln[A] = \ln[A]_0 - kt$; $t½ = 0.693/k$ (independent of $[A]_0$)
- Second order: $1/[A] = 1/[A]_0 + kt$; $t½ = 1/(k[A]_0)$
- Arrhenius: k = A·exp(-Ea/RT); higher T or lower Ea → faster
- Catalyst: lowers Ea (both forward and reverse equally); does NOT change ΔG or K
- Transition state: highest-energy point on reaction coordinate; activated complex; cannot be isolated

DISCRIMINATOR | RATE-DETERMINING STEP vs OVERALL REACTION

The rate law reflects only steps up to and including the slowest (rate-determining) step. Fast steps before the RDS contribute via their equilibrium constants. Fast steps after the RDS do not affect the rate law. The molecularity of the elementary RDS gives the order with respect to its reactants, but only after substituting equilibria from earlier fast steps.

III.7 Equilibrium

- K_eq = [products]^c / [reactants]^a (pure solids and liquids excluded)
- Kp = Kc(RT)^Δn (Δn = moles gas products - moles gas reactants)
- Q vs K: Q < K → forward; Q > K → reverse; Q = K → equilibrium
- Le Chatelier: stress shifts equilibrium to relieve stress
- Add reactant → forward; remove product → forward; ↑ T → endothermic direction; ↑ P → fewer moles gas
- Catalyst: does NOT shift equilibrium; only speeds approach
- Solubility product Ksp: for sparingly soluble salts; e.g., $AgCl(s) \leftrightarrow Ag^+ + Cl^-$, $Ksp = [Ag^+][Cl^-]$
- Common ion effect: adding a common ion → ↓ solubility (Q > Ksp temporarily)
- Will precipitate when Q > Ksp

III.8 Acids and Bases

Definition	Acid	Base
Arrhenius	Releases H^+ in water	Releases OH^- in water
Brønsted-Lowry	Proton donor	Proton acceptor
Lewis	Electron pair acceptor	Electron pair donor

- $pH = -\log[H^+]$; $pOH = -\log[OH^-]$; pH + pOH = 14 at 25 °C
- $K_w = [H^+][OH^-] = 10^{-14}$ at 25 °C
- Strong acids (fully dissociate): HCl, HBr, HI, HNO_3, H_2SO_4, $HClO_4$, $HClO_3$
- Strong bases: group 1 hydroxides, $Ca(OH)_2$, $Sr(OH)_2$, $Ba(OH)_2$
- Conjugate pair: differ by one H^+; stronger acid → weaker conjugate base
- $K_a \times K_b = K_w$ for conjugate pair; pKa + pKb = 14
- Henderson-Hasselbalch: $pH = pK_a + \log([A^-]/[HA])$
- Buffer: weak acid + conjugate base (or weak base + conjugate acid); resists pH change
- Best buffering: pH within ±1 of pKa; $[A^-] = [HA]$ when pH = pKa
- Titration: equivalence point = moles acid = moles base added
- Strong-strong titration: equivalence at pH 7
- Weak acid + strong base: equivalence at pH > 7; half-equivalence at pH = pKa

- Indicators: change color near their pKa

III.9 Electrochemistry

- Oxidation: loss of e^- (OIL); reduction: gain of e^- (RIG)
- Oxidizing agent gets reduced; reducing agent gets oxidized
- Oxidation states: assign with H=+1 (except metal hydrides, -1), O=-2 (except peroxides, -1)
- Galvanic (voltaic) cell: spontaneous, $\Delta G<0$, $E° > 0$; produces electricity
- Electrolytic cell: nonspontaneous, $\Delta G>0$, $E° < 0$; requires electricity
- Anode: oxidation (Anode-Oxidation, vowels match); cathode: reduction
- Salt bridge: maintains charge balance in galvanic; ions migrate to balance
- Electrons flow from anode to cathode through external wire (both cell types)
- $E°_{cell} = E°_{cathode} - E°_{anode}$ (both as reduction potentials)
- $\Delta G° = -nFE°_{cell}$; F = 96485 C/mol
- Nernst: $E = E° - (RT/nF) \ln Q = E° - (0.0592/n) \log Q$ at 25 °C
- At equilibrium: E = 0, Q = K
- Faraday's laws: moles e^- = It/F; mass deposited proportional to charge

DISCRIMINATOR | GALVANIC vs ELECTROLYTIC CELL

BOTH: anode = oxidation, cathode = reduction, electrons flow anode → cathode in wire. GALVANIC: spontaneous, generates current, anode is the NEGATIVE terminal (electrons leaving), cathode positive. ELECTROLYTIC: requires external power, anode is the POSITIVE terminal (connected to + terminal of battery), cathode negative. Signs flip; oxidation-at-anode rule does not.

CLINICAL BRIDGE | ACID-BASE IN THE BODY

Blood pH is held at 7.35-7.45 by the bicarbonate buffer (HCO_3^- / H_2CO_3 / CO_2). The lungs regulate CO_2 (fast, minutes); the kidneys regulate HCO_3^- (slow, hours to days). Hyperventilation blows off CO_2 → respiratory alkalosis. Diabetic ketoacidosis produces ketoacids → metabolic acidosis with elevated anion gap. The body compensates by hyperventilating (Kussmaul breathing). Henderson-Hasselbalch for blood: $pH = 6.1 + \log([HCO_3^-]/0.03 \cdot PCO_2)$.

HIGH-YIELD

- PV = nRT and the gas laws; remember T in Kelvin
- $\Delta G = \Delta H - T\Delta S$; $\Delta G = -RT \ln K = -nFE$
- Arrhenius: higher T or lower Ea → faster
- Le Chatelier: shift opposes the stress
- Henderson-Hasselbalch: pH = pKa + log(base/acid)
- Galvanic spontaneous ($E° > 0$); electrolytic needs power ($E° < 0$)

PART IV · ORGANIC CHEMISTRY HIGH-YIELD

IV.1 Structure and Stereochemistry

Hybridization	Geometry	Bond angle	Bonds
sp	Linear	180°	2 σ, 2 π (triple bond or 2 doubles)
sp^2	Trigonal planar	120°	3 σ, 1 π (double bond)
sp^3	Tetrahedral	109.5°	4 σ

- Sigma bond: head-on overlap; can be single, present in all bonds
- Pi bond: side-on overlap; only in double or triple bonds; prevents rotation
- Conjugation: alternating single/double bonds; allows delocalization; ↓ energy
- Aromaticity (Hückel): cyclic, planar, fully conjugated, 4n+2 π electrons
- Antiaromatic: 4n π electrons → unstable
- Resonance: delocalized π or lone pair electrons; structures contribute to weighted average
- Chirality: nonsuperimposable mirror image; usually has stereocenter (carbon with 4 different groups)
- Enantiomers: nonsuperimposable mirror images; identical physical properties except optical rotation and chiral environment behavior
- Diastereomers: stereoisomers that are NOT mirror images; different physical properties
- Meso compound: has stereocenters but internal mirror plane → achiral overall
- Number of stereoisomers ≤ 2^n where n = stereocenters
- R/S assignment: rank substituents by atomic number (Cahn-Ingold-Prelog); lowest priority back; clockwise = R, counterclockwise = S
- E/Z: higher priority on same side = Z; opposite side = E

DISCRIMINATOR | ENANTIOMERS vs DIASTEREOMERS

ENANTIOMERS: mirror images that cannot be superimposed; ALL stereocenters inverted; identical everything except chiral interactions (optical rotation opposite, chiral chromatography, drug receptor binding). DIASTEREOMERS: not mirror images; AT LEAST ONE but not ALL stereocenters differ; different melting points, boiling points, solubility, retention times. Cis/trans isomers are a special case of diastereomers.

IV.2 Nomenclature (IUPAC priorities)

1. Carboxylic acid (-oic acid) > anhydride > ester > amide > nitrile > aldehyde (-al) > ketone (-one) > alcohol (-ol) > amine (-amine) > ether > alkene/alkyne > alkane
2. Longest carbon chain that contains highest-priority group
3. Number to give lowest locant to the principal group
4. Substituents listed alphabetically

IV.3 Functional Groups and Reactivity

Group	Structure	Key reactivity
Alkane	C-C, C-H	Combustion, radical halogenation only
Alkene	C=C	Electrophilic addition (Markovnikov)
Alkyne	C≡C	Addition; terminal alkyne acidic (pKa ~25)
Alcohol	R-OH	H-bond donor; can oxidize to carbonyl
Ether	R-O-R'	Mostly inert; good solvent
Amine	$R\text{-}NH_2$	Basic, nucleophilic
Aldehyde	R-CHO	Carbonyl C electrophilic; oxidize to COOH
Ketone	R-CO-R'	Less reactive than aldehyde; alpha-H acidic
Carboxylic acid	R-COOH	Acidic (pKa ~4-5); H-bond donor and acceptor
Ester	R-COO-R'	Hydrolyzes (acid or base/saponification)
Amide	$R\text{-}CO\text{-}NR'_2$	Most stable carbonyl derivative; resonance
Acid halide	R-COCl	Most reactive carbonyl derivative

- Reactivity of carbonyl derivatives (most to least): acid halide > anhydride > ester ≈ COOH > amide
- Reason: leaving group stability; chloride best leaving group; amide N has worst leaving group + resonance
- Oxidation ladder: 1° alcohol → aldehyde → carboxylic acid; 2° alcohol → ketone; 3° alcohol no oxidation

IV.4 Reaction Mechanisms (SN1, SN2, E1, E2)

Factor	SN1	SN2	E1	E2
Substrate	3° > 2°	1° > 2°	3° > 2°	3° > 2° > 1°
Nucleophile/base	Weak nucleophile	Strong nucleophile	Weak base	Strong, bulky base
Solvent	Polar protic	Polar aprotic	Polar protic	Either
Kinetics	1st order (substrate)	2nd order	1st order	2nd order
Stereochem	Racemization	Inversion (Walden)	No specific	Anti-periplanar
Carbocation?	Yes (can rearrange)	No	Yes (can rearrange)	No
Temperature	Either	Either	Higher	Higher

- Stabilities: carbocation 3° > 2° > 1° > methyl; carbanion reverse
- Hyperconjugation and induction stabilize carbocations; resonance is strongest stabilizer (benzylic, allylic)

- Bulky base (LDA, KOtBu) favors E2 over SN2 and Hofmann product (less substituted alkene)
- Small base (NaOEt) favors Zaitsev (more substituted alkene)
- Polar protic solvents (water, ROH) stabilize carbocations and ions → favor SN1/E1
- Polar aprotic solvents (DMSO, DMF, acetone) leave nucleophile naked → favor SN2

IV.5 Carbonyl Chemistry

- Nucleophilic addition: Nu attacks carbonyl C → tetrahedral alkoxide → protonate to alcohol
- Hydrate (gem-diol): water adds; unstable except formaldehyde
- Hemiacetal: one ROH adds; unstable except in sugars (cyclic)
- Acetal: two ROH (or diol); stable (good protecting group); requires acid catalyst, removes water
- Imine: $R_2C{=}NR'$; primary amine + carbonyl, lose water
- Enamine: $R_2C{=}CR'\text{-}NR''_2$; secondary amine + carbonyl, lose water
- Cyanohydrin: HCN adds → $R_2C(OH)(CN)$
- Alpha-hydrogen acidity: pKa ~20 for ketones, ~25 for esters; due to enolate resonance
- Tautomerization: keto ↔ enol; keto usually favored
- Enolate formation: strong base (LDA) removes alpha-H
- Aldol condensation: enolate of one carbonyl + electrophile carbon of another → β-hydroxy carbonyl; can dehydrate to α,β-unsaturated
- Claisen condensation: enolate of ester attacks another ester → β-ketoester (e.g., acetoacetate)
- Dieckmann: intramolecular Claisen → cyclic β-ketoester
- Decarboxylation: β-keto acids and malonic acids lose CO_2 on heating

IV.6 Aromatic Chemistry

- Benzene resists addition (loses aromaticity); does electrophilic aromatic substitution (EAS) instead
- EAS steps: electrophile generated → attacks ring (arenium ion) → deprotonation restores aromaticity
- EAS reactions: halogenation, nitration (HNO_3/H_2SO_4), sulfonation, Friedel-Crafts alkylation/acylation

Substituent	Effect	Directs
-OH, -OR, $-NH_2$, $-NR_2$	Strong activator (donate via lone pair)	ortho/para
Alkyl, -Ar	Weak activator (induction, hyperconjugation)	ortho/para
-F, -Cl, -Br, -I	Weak deactivator BUT ortho/para director	ortho/para
$-NO_2$, -CN, $-SO_3H$, -COR, -COOH	Strong deactivator	meta
$-NR_3^+$	Strong deactivator	meta

DISCRIMINATOR | ORTHO/PARA vs META DIRECTORS

Ortho/para directors stabilize the arenium ion intermediate at ortho or para positions through resonance (donating groups put a lone pair in conjugation with the ring) or by hyperconjugation (alkyl groups). Halogens are unique: their inductive withdrawal makes them deactivators, but their lone pair donation directs ortho/para. Meta directors destabilize the ortho/para arenium ions more than the meta (positive charge ends up next to an electron-poor center).

IV.7 Spectroscopy

IR absorption (cm^{-1})	Bond
~3300	O-H (broad if H-bonded) or N-H (sharper)
~3000	C-H (sp^3 slightly below, sp^2 slightly above 3000)
~2200	C≡C, C≡N (often weak)
~1700-1750	C=O (carbonyl); esters ~1735, ketones ~1715, COOH broad with OH
~1600	C=C (aromatic or alkene)
~1000-1300	C-O

- ^1H NMR shifts (ppm): TMS = 0; sp^3 CH ~0.5-2; alpha to carbonyl ~2-3; alkene CH ~5-7; aromatic CH ~6.5-8; aldehyde H ~9-10; COOH H ~10-12
- Splitting (n+1 rule): n equivalent neighbors give n+1 peaks (doublet, triplet, quartet)
- Integration: relative number of protons under each peak
- Chemical shift = deshielding; electronegative groups and π systems pull electrons away → ↑ shift
- ^{13}C NMR: each unique carbon = one peak; no splitting if proton-decoupled
- Mass spec: M^+ = molecular ion (mass = MW); fragments give structural clues

IV.8 Biological Molecules

- 20 amino acids: alpha-amino acid with R group; all chiral (L-config) except glycine
- Peptide bond: amide linkage between COOH and amine; trans (planar, restricted rotation)
- Isoelectric point (pI): pH where net charge = 0; for nonionizable R: pI = (pKa1 + pKa2)/2
- Carbohydrates: aldoses (CHO) or ketoses (C=O); D/L by Fischer; alpha/beta anomers differ at anomeric C
- Glycosidic bond: from anomeric C-OH + alcohol → loss of water
- Reducing sugar: has free anomeric carbon (open-chain aldehyde or ketone); glucose, fructose, lactose, maltose are reducing; sucrose is NOT
- Lipids: triglycerides (glycerol + 3 FA), phospholipids (amphipathic, membranes), steroids (4 fused rings), sphingolipids
- Saturated FA: no C=C, solid (animal fat); Unsaturated: C=C, liquid (oils); trans-FA from partial hydrogenation
- Nucleic acids: nucleotide = base + sugar + phosphate; phosphodiester bond 3'-5'

DISCRIMINATOR | ALPHA vs BETA ANOMERS (sugars)

Anomers differ only at the anomeric carbon (the former carbonyl C). In D-sugars drawn in standard Haworth projection, ALPHA has the anomeric -OH down (trans to the CH_2OH at C5 in glucose), BETA has it up. Beta-glucose is the building block of cellulose (digestion requires cellulase, which humans lack); alpha-glucose forms starch and glycogen (digestible by amylase).

HIGH-YIELD

- SN1/E1: 3° substrate, weak nucleophile/base, polar protic, carbocation intermediate
- SN2: 1° or 2°, strong nucleophile, polar aprotic, inversion
- E2: strong bulky base, anti-periplanar
- Carbonyl reactivity: acid halide > anhydride > ester > amide
- Activators (donors) direct ortho/para; deactivators direct meta; halogens are weak deactivators that direct ortho/para
- Aromaticity = cyclic + planar + fully conjugated + 4n+2 π electrons
- C=O IR ~1700 cm^{-1} is the most diagnostic single IR peak

PART V · PHYSICS HIGH-YIELD

V.1 Kinematics

- Displacement = Δx (vector); distance = total path (scalar)
- Velocity = Δx/Δt (vector); speed = |velocity| (scalar)
- Acceleration = Δv/Δt
- Equations (constant a): $v = v_0 + at$; $x = x_0 + v_0t + (1/2)at^2$; $v^2 = v_0^2 + 2a\Delta x$; $x = x_0 + (v + v_0)t/2$
- Free fall: $a = -g = -9.8\ m/s^2$ downward; ignore air resistance
- Projectile motion: x and y independent; $a_x = 0$, $a_\gamma = -g$; same $v_{0\gamma}$ at launch and landing height
- Max range at 45° launch angle (level ground); time to apex = $v_0 \sin\theta/g$

V.2 Forces and Newton's Laws

- Newton's 1st: F_net = 0 → constant velocity (inertia)
- Newton's 2nd: F_net = ma
- Newton's 3rd: action-reaction pairs equal and opposite on DIFFERENT objects
- Free body diagram: draw all forces on object as vectors; resolve into x and y; sum = ma
- Weight W = mg
- Normal force N perpendicular to surface; on incline N = mg·cosθ; gravity component along incline = mg·sinθ
- Friction: f = μN; static (μs, up to max) ≥ kinetic (μk)
- Tension: same magnitude throughout massless rope

V.3 Energy and Work

- Work $W = F \cdot d \cdot \cos\theta$ (J); only force along displacement counts
- Kinetic energy $KE = (1/2)mv^2$
- Gravitational PE = mgh
- Spring $PE = (1/2)kx^2$; force = -kx (Hooke)
- Work-energy theorem: $W_{net} = \Delta KE$
- Conservation of mechanical energy (no friction): KE + PE = constant
- Power $P = W/t = F \cdot v$ (W = J/s)
- Efficiency η = useful output / total input × 100%

V.4 Momentum

- Momentum p = mv (vector); conserved in isolated system
- Impulse $J = F \cdot \Delta t = \Delta p$
- Elastic collision: KE and p conserved (ideal)
- Inelastic collision: only p conserved; some KE → heat, sound, deformation
- Perfectly inelastic: objects stick; max KE loss
- Center of mass: $\Sigma m_i x_i COM / \Sigma m_i$; $F_{ext} = M \cdot a_{COM}$

V.5 Rotation

- Angular displacement θ, velocity ω, acceleration α; analogues to linear
- $v = r\omega$; $a_{tangential} = r\alpha$; centripetal $a_c = v^2/r = \omega^2 r$
- Centripetal force $F_c = mv^2/r$ (directed toward center)
- Torque $\tau = r \times F = rF \cdot \sin\theta$; rotational analog of force
- Moment of inertia $I = \Sigma m_i r_i^2$ (depends on axis and mass distribution)
- Angular momentum $L = I\omega$; conserved if no net external torque (ice skater pulls in arms → ω ↑)
- Rotational $KE = (1/2)I\omega^2$
- Equilibrium: $\Sigma F = 0$ AND $\Sigma\tau = 0$

V.6 Fluids

- Density $\rho = m/V$ (kg/m^3); water = 1000 kg/m^3 = 1 g/cm^3
- Specific gravity $SG = \rho/\rho_{water}$
- Pressure $P = F/A$ ($Pa = N/m^2$); 1 atm ≈ 101 kPa ≈ 760 mmHg
- Hydrostatic pressure: $P = P_0 + \rho gh$ (gauge if P_0=0)
- Pascal's principle: pressure applied to enclosed fluid transmitted equally; hydraulic press $F_1/A_1 = F_2/A_2$
- Archimedes: buoyant force = weight of fluid displaced; $F_b = \rho_{fluid} \cdot V_{submerged} \cdot g$

- Floating: ρ_object < ρ_fluid; fraction submerged = ρ_object/ρ_fluid
- Continuity (incompressible): $A_1v_1 = A_2v_2$ (smaller area → faster flow)
- Bernoulli: $P + (1/2)\rho v^2 + \rho gh$ = constant; faster flow → lower pressure
- Poiseuille (viscous flow in pipe): $Q = \pi r^4 \Delta P/(8\eta L)$; flow $\propto r^4$ (huge sensitivity to radius)
- Viscosity η: internal friction; water ~1 cP, blood ~3-4 cP
- Reynolds number: Re = ρvD/η; high Re → turbulent
- Surface tension: cohesive forces at liquid-gas interface; capillary rise in narrow tubes

V.7 Waves and Sound

- v = fλ; frequency f (Hz), wavelength λ (m)
- Period T = 1/f
- Transverse: displacement perpendicular to propagation (light, string)
- Longitudinal: displacement || propagation (sound)
- Sound speed: ↑ in denser, less compressible media; faster in solids than gases
- Intensity I = P/A; decibels $\beta = 10 \cdot \log(I/I_0)$, $I_0 = 10^{-12}$ W/m^2
- Doppler: f' = f·(v ± v_observer)/(v -/+ v_source); approaching → higher f, receding → lower
- Standing waves on string (both ends fixed): λ_n = 2L/n, f_n = nv/(2L)
- Open pipe (both ends): f_n = nv/(2L); Closed-open: f_n = nv/(4L), odd n only
- Beats: $|f_1 - f_2|$
- Constructive interference: in phase (Δpath = nλ); destructive: out of phase (Δpath = (n+1/2)λ)

V.8 Optics

- Reflection: angle of incidence = angle of reflection (both from normal)
- Refraction: $n_1\sin\theta_1 = n_2\sin\theta_2$ (Snell); n = c/v
- Total internal reflection: from denser to less dense beyond critical angle $\theta_c = \sin^{-1}(n_2/n_1)$
- Thin lens / mirror: 1/f = 1/d_o + 1/d_i; m = -d_i/d_o = h_i/h_o
- Sign conventions: f > 0 converging; d_i > 0 same side as image; m < 0 inverted
- Converging lens (convex): can form real or virtual; real if object beyond f
- Diverging lens (concave): always virtual, upright, smaller
- Concave mirror: converging; convex mirror: diverging

Configuration	Image
Converging lens, object beyond 2f	Real, inverted, smaller
Converging lens, object at 2f	Real, inverted, same size
Converging lens, between f and 2f	Real, inverted, larger
Converging lens, object inside f	Virtual, upright, larger (magnifier)
Diverging lens, any object distance	Virtual, upright, smaller

- Power of lens $P = 1/f$ (diopters, m^{-1})
- Diffraction: bending around obstacles; greatest when slit $\approx \lambda$
- Double-slit interference: $d \cdot \sin\theta = m\lambda$ (bright); single slit: $d \cdot \sin\theta = m\lambda$ (dark, $\neq 0$)
- Polarization: confines E-field to one plane; partial reflection at Brewster's angle

V.9 Electricity and Magnetism

- Coulomb's law: $F = kq_1q_2/r^2$ ($k = 8.99 \times 10^9$ N·m^2/C^2); like repel, unlike attract
- Electric field $E = F/q = kq/r^2$; units N/C or V/m
- Electric potential $V = kq/r$; $PE = qV$; $W = q\Delta V$
- Field points from + to -; positive test charge accelerates toward lower potential
- Parallel plate capacitor: $E = V/d$ uniform; $C = \varepsilon A/d$; $Q = CV$; $U = (1/2)CV^2$
- Capacitors in series: $1/C_{eq} = \Sigma 1/C_i$; in parallel: $C_{eq} = \Sigma C_i$
- Current $I = \Delta Q/\Delta t$ (A = C/s); conventional current = + charge flow direction
- Ohm's law: $V = IR$
- Resistance $R = \rho L/A$ (ρ = resistivity)
- Power $P = IV = I^2R = V^2/R$
- Resistors in series: $R_{eq} = \Sigma R_i$; in parallel: $1/R_{eq} = \Sigma 1/R_i$
- Kirchhoff: $\Sigma I_{in} = \Sigma I_{out}$ (node); ΣV around loop = 0
- Magnetic force on moving charge: $F = qv \times B$; $F = qvB \cdot \sin\theta$ (perpendicular to both v and B)
- Right-hand rule for positive charges; left-hand for negative (or flip)
- Force on current-carrying wire: $F = IL \times B$
- Magnetic flux $\Phi = B \cdot A \cdot \cos\theta$
- Faraday's law: $EMF = -d\Phi/dt$; Lenz: induced current opposes change
- Transformer: $V_p/V_s = N_p/N_s$; $I_p/I_s = N_s/N_p$ (power conserved ideally)
- RC circuit charging: $V_C = V_0(1 - \exp(-t/RC))$; time constant $\tau = RC$
- RC discharging: $V_C = V_0 \cdot \exp(-t/RC)$
- Capacitor blocks DC steady state (acts as open); passes high-frequency AC

- Inductor passes DC (acts as wire steady state); blocks high-frequency AC

V.10 Modern Physics

- Photon energy $E = hf = hc/\lambda$ ($h = 6.626 \times 10^{-34}$ J·s)
- Photoelectric effect: photons eject electrons only if E > work function ϕ; $KE_{max} = hf - \phi$
- Intensity (more photons) ↑ electron count but NOT energy per electron
- de Broglie: $\lambda = h/p = h/(mv)$; matter waves; significant only for very small mass
- Heisenberg uncertainty: $\Delta x \cdot \Delta p \geq \hbar/2$
- Alpha decay: emits ${}^{4}_{2}He$; mass -4, atomic number -2
- Beta minus decay: $n \rightarrow p + e^{-}$ + antineutrino; atomic number +1, mass same
- Beta plus (positron): $p \rightarrow n + e^{+}$ + neutrino; atomic number -1
- Gamma decay: photon emission only; no change in Z or A
- Electron capture: $p + e^{-} \rightarrow n$; atomic number -1
- Half-life: t = t½·n where n = number of half-lives; $N = N_0 \cdot (1/2)^n$
- Fission: heavy nucleus splits (U-235); fusion: light nuclei combine (sun)
- Mass defect: $\Delta m \cdot c^2$ = binding energy

DISCRIMINATOR | INTENSITY vs FREQUENCY (photoelectric effect)

Classical physics predicted that brighter light (higher intensity) should give higher-energy electrons. The photoelectric effect showed otherwise: FREQUENCY (not intensity) determines whether and how energetically electrons are ejected; INTENSITY determines how MANY electrons are ejected (once above threshold frequency). This established light as quantized (photons).

CLINICAL BRIDGE | DOPPLER ULTRASOUND

When ultrasound reflects off moving blood cells, the returned frequency differs from the transmitted (Doppler shift). Approaching flow shifts up; receding flow shifts down. The magnitude is proportional to velocity. This is how vascular ultrasound measures blood flow velocity, identifies stenoses (high-velocity jets), and characterizes cardiac valve disease.

HIGH-YIELD

- Kinematics: $v^2 = v_0^2 + 2a\Delta x$; projectile motion: x and y independent
- F = ma; weight = mg; friction $f = \mu N$
- Energy: $W = \Delta KE$; conserve KE+PE without friction
- Bernoulli: faster → lower pressure; Poiseuille: $Q \propto r^4$
- $v = f\lambda$; Doppler approach raises pitch
- Snell: $n_1 \sin\theta_1 = n_2 \sin\theta_2$; lens: $1/f = 1/d_o + 1/d_i$
- Ohm: V = IR; capacitors parallel add C; resistors series add R
- Photon $E = hf$; half-life $N = N_0 \cdot (1/2)^n$

PART VI · PSYCHOLOGY & SOCIOLOGY HIGH-YIELD

VI.1 Sensation and Perception

- Sensation: detection by sensory receptors (transduction of stimulus to neural signal)
- Perception: interpretation in CNS
- Absolute threshold: minimum stimulus detectable 50% of time
- Difference threshold (JND, just noticeable difference): smallest detectable change
- Weber's law: $\Delta I/I$ = constant (proportional, not absolute)
- Signal detection theory: hits, misses, false alarms, correct rejections; influenced by motivation, expectation
- Sensory adaptation: ↓ response to constant stimulus
- Vision: rods (low light, peripheral, no color) vs cones (color, central, fovea); transduction in retina
- Hearing: cochlea → hair cells; high freq at base, low freq at apex (place theory)
- Taste: sweet, salty, sour, bitter, umami; tongue + palate
- Smell: olfactory bulb; one of few senses NOT routed through thalamus
- Touch, proprioception, vestibular (balance, semicircular canals)
- Top-down processing: concept-driven (expectations shape perception)
- Bottom-up: data-driven (start from raw sensation)
- Gestalt principles: figure-ground, proximity, similarity, continuity, closure

VI.2 Learning

Type	Mechanism	Example
Classical conditioning	Pair neutral with US → CS elicits CR	Pavlov: bell + food → bell elicits salivation
Operant conditioning	Behavior shaped by consequences	Skinner box: lever press for food
Observational learning	Watching others (Bandura)	Bobo doll experiment
Habituation	↓ response to repeated stimulus	Tuning out clock ticking
Sensitization	↑ response after intense stimulus	Startle after loud noise

Operant consequence	Effect on behavior	Adds or removes
Positive reinforcement	↑	Adds desirable (treat)
Negative reinforcement	↑	Removes aversive (seatbelt beep stops)
Positive punishment	↓	Adds aversive (scolding)
Negative punishment	↓	Removes desirable (timeout)

- Reinforcement schedules:

- Fixed ratio (FR): reinforce every n responses; high rate with brief pause after reinforcement
- Variable ratio (VR): reinforce after unpredictable # responses; highest, most persistent rate (gambling)
- Fixed interval (FI): reinforce first response after fixed time; scalloped pattern
- Variable interval (VI): reinforce after unpredictable time; steady moderate response
- Extinction: stop reinforcing → behavior decreases; VR most resistant to extinction

VI.3 Memory

- Sensory memory: brief, modality-specific (iconic = visual ~250 ms; echoic = auditory ~3 s)
- Short-term (working) memory: ~7 ± 2 items, ~20-30 s (Miller's number); maintained by rehearsal
- Long-term: essentially unlimited capacity and duration
- Encoding: getting info in (semantic > phonemic > visual)
- Storage: maintaining info
- Retrieval: accessing info (recall > recognition test difficulty)
- Long-term divisions: explicit (declarative: episodic = events, semantic = facts) and implicit (procedural = skills; priming)
- Hippocampus: forms new explicit memories (damage → anterograde amnesia)
- Cerebellum and basal ganglia: implicit (procedural)
- Amygdala: emotional memory
- LTP (long-term potentiation): NMDA receptors in hippocampus; cellular basis of memory
- Forgetting: decay, interference (proactive = old blocks new; retroactive = new blocks old), retrieval failure
- Mnemonics: method of loci, chunking, hierarchies

VI.4 Cognition

- Piaget stages: sensorimotor (0-2, object permanence), preoperational (2-7, egocentrism, no conservation), concrete operational (7-11, conservation, logic on concrete), formal operational (12+, abstract, hypothetical)
- Vygotsky: zone of proximal development; learning is social
- Problem solving: algorithm (systematic, guaranteed), heuristic (shortcut, usually fast)
- Heuristics and biases: availability (judge by ease of recall), representativeness (judge by similarity to prototype), anchoring, confirmation bias, hindsight bias
- Functional fixedness: cannot see novel uses of objects
- Language: phoneme → morpheme → syntax → semantics → pragmatics
- Whorf hypothesis (linguistic relativity): language shapes thought (controversial in strong form)
- Critical period for language: ~birth to puberty

VI.5 Motivation and Emotion

- Drive-reduction: physiological need → drive → reduce via behavior

- Arousal theory (Yerkes-Dodson): optimal performance at moderate arousal; lower for hard tasks
- Maslow's hierarchy: physiological → safety → love/belonging → esteem → self-actualization (lower must be met first, in classical form)
- Incentive theory: external rewards motivate; overjustification: extrinsic ↓ intrinsic motivation

Emotion theory	Sequence
James-Lange	Stimulus → bodily response → emotion (see bear → run → fear)
Cannon-Bard	Stimulus → simultaneous bodily and emotion (see bear → run AND feel fear at same time)
Schachter-Singer (two-factor)	Stimulus → arousal + cognitive label → emotion (see bear → arousal + 'this is danger' → fear)
Lazarus	Stimulus → cognitive appraisal → emotion → bodily

VI.6 Personality

- Trait: enduring characteristics; Big Five (OCEAN): Openness, Conscientiousness, Extraversion, Agreeableness, Neuroticism
- Psychoanalytic (Freud): id (pleasure, unconscious), ego (reality), superego (moral); defense mechanisms (repression, projection, displacement, sublimation, rationalization, regression)
- Humanistic (Rogers, Maslow): unconditional positive regard, self-actualization
- Social-cognitive (Bandura): reciprocal determinism between behavior, environment, personal factors; self-efficacy
- Behaviorist: personality = learned behaviors

VI.7 Psychological Disorders

Category	Examples
Mood	Major depressive, bipolar, persistent depressive
Anxiety	Generalized anxiety, panic, phobias, social anxiety
OCD and related	OCD, body dysmorphic, hoarding
Trauma	PTSD, acute stress
Dissociative	Dissociative identity, amnesia, depersonalization
Psychotic	Schizophrenia (positive: hallucinations, delusions; negative: flat affect, avolition)
Personality	Cluster A odd, B dramatic (antisocial, borderline, histrionic, narcissistic), C anxious
Neurodevelopmental	ADHD, autism, intellectual disability
Eating	Anorexia, bulimia, binge eating
Somatic symptom and dissociative	

- Biological correlates: depression ↓ 5-HT, NE; schizophrenia ↑ dopamine; Alzheimer ↓ ACh; Parkinson ↓ dopamine
- DSM-5: standard diagnostic manual
- Biopsychosocial model: biological + psychological + social contributions

VI.8 Therapy

- Psychodynamic: insight into unconscious
- Behavioral: classical/operant techniques (systematic desensitization, exposure)
- Cognitive: identify and change maladaptive thoughts
- CBT: combines cognitive and behavioral; most evidence-based
- Humanistic: client-centered, empathy, unconditional positive regard
- Biomedical: drugs (SSRIs for depression, antipsychotics for schizophrenia, lithium for bipolar, benzodiazepines for anxiety)
- ECT for severe depression refractory to drugs

VI.9 Social Psychology

- Attribution: dispositional (internal) vs situational (external)
- Fundamental attribution error: overestimate dispositional in others
- Self-serving bias: attribute own success to dispositional, failure to situational
- Actor-observer bias: self situational; others dispositional
- Conformity (Asch): match group; ↑ with group size to ~5, unanimity
- Obedience (Milgram): obey authority even at personal moral cost
- Bystander effect: more bystanders → less likely any individual helps (diffusion of responsibility)
- Groupthink: pressure for consensus suppresses dissent
- Group polarization: discussion strengthens initial inclinations
- Social facilitation: ↑ performance on easy/familiar tasks in presence of others; ↓ on difficult
- Cognitive dissonance: discomfort from conflicting beliefs/actions → change one
- Prejudice: attitude; discrimination: behavior; stereotype: belief
- Stereotype threat: anxiety from fear of confirming stereotype impairs performance

VI.10 Sociology

Theory	Lens
Functionalism (Durkheim)	Society as system of interrelated parts maintaining stability
Conflict theory (Marx)	Society as competition over scarce resources; power and inequality
Symbolic interactionism (Mead)	Micro-level meaning-making through symbols and interactions
Social constructionism	Reality is constructed through social processes
Feminist theory	Gender as a key axis of stratification
Rational choice	Individuals as rational actors maximizing benefit

- Socialization: agents = family, school, peers, media, workplace
- Status: ascribed (born into) vs achieved (earned); master status dominates identity
- Role conflict: incompatible roles; role strain: tension within one role
- Institutions: family, education, religion, economy, government, healthcare
- Stratification: class (economic), caste (rigid, ascribed), estate (medieval)
- Social mobility: vertical (up/down class), horizontal (lateral), intergenerational
- Demographic transition: high birth + high death → high birth + low death → low both → low birth, low death
- Demography: fertility rate, mortality rate, migration; age pyramids
- Health disparities: by SES, race, gender, geography
- Sick role (Parsons): exempt from normal roles, not responsible for illness, expected to seek care, expected to want to get well
- Medicalization: redefining problems as medical (e.g., ADHD, addiction)
- Iron triangle of healthcare: cost, access, quality

DISCRIMINATOR | CLASSICAL vs OPERANT CONDITIONING

CLASSICAL pairs two stimuli to elicit an automatic response (Pavlov: bell + food → bell alone causes salivation). The behavior is REFLEXIVE and elicited. OPERANT pairs a behavior with a consequence to change its frequency (Skinner: rat presses lever → gets food → presses more). The behavior is VOLUNTARY and emitted. Classical: organism reacts; operant: organism acts.

DISCRIMINATOR | JAMES-LANGE vs CANNON-BARD vs SCHACHTER-SINGER

JAMES-LANGE: body comes first, emotion is the perception of bodily change ('I run, therefore I fear'). CANNON-BARD: body and emotion happen simultaneously and independently. SCHACHTER-SINGER (two-factor): you need both arousal and a cognitive label to have a specific emotion. If you see arousal as the cause and emotion as the effect, it is James-Lange. If they are simultaneous, Cannon-Bard. If a cognitive label is needed, Schachter-Singer.

CLINICAL BRIDGE | THE BIOPSYCHOSOCIAL MODEL

Modern medicine treats illness as the intersection of biological (genes, anatomy, biochemistry), psychological (cognition, emotion, behavior), and social (family, culture, SES, access) factors. A patient with chronic back pain has tissue damage (bio), pain catastrophizing or depression (psych), and stressors or supports at work and home (social). Effective care addresses all three. This framework is tested heavily on the MCAT psych/soc section.

HIGH-YIELD

- Weber's law: ΔI/I is constant; perceived change is relative not absolute
- Classical = reflexive (stimulus-stimulus); operant = voluntary (behavior-consequence)
- Variable ratio = most persistent; gambling-like
- Hippocampus stores new explicit; cerebellum stores procedural
- Schachter-Singer two-factor: arousal + cognitive label
- Big Five = OCEAN
- Fundamental attribution error: others dispositional; self situational
- Stratification axes: class, race, gender, age

PART VII · BIOCHEMISTRY HIGH-YIELD

VII.1 Amino Acids and Proteins

- 20 standard amino acids; all alpha-amino acids; all L-configuration; all chiral except glycine (R = H)
- Nonpolar: G, A, V, L, I, M, F, W, P (proline is unique imino acid, kinks helices)
- Polar uncharged: S, T, C, N, Q, Y (Y is aromatic)
- Acidic (negative at physiological pH): D (Asp), E (Glu)
- Basic (positive): K (Lys), R (Arg), H (His; pKa ~6, partial protonation)
- Aromatic: F, W, Y (absorb at 280 nm; W strongest)
- Sulfur-containing: C (forms disulfide bonds), M

Protein structure	Forces	Description
Primary	Peptide (covalent)	Sequence of AAs
Secondary	H-bonds backbone	Alpha helix, beta sheet, turns
Tertiary	All non-covalent + disulfide	3D fold of one polypeptide
Quaternary	All non-covalent + disulfide	Multiple polypeptide subunits

- Alpha helix: H-bond between i and i+4; R groups outside; proline disrupts
- Beta sheet: H-bonds between strands; parallel or antiparallel; R groups alternate sides
- Tertiary forces: hydrophobic effect (often dominant), H-bonds, salt bridges, van der Waals, disulfide (only covalent)
- Disulfide: cysteine + cysteine; -2H; only in oxidizing environment (extracellular, ER)

- Denaturation: loss of higher structure; primary preserved; heat, pH, detergents, urea
- Hemoglobin: 4 subunits ($2\alpha 2\beta$); cooperative O_2 binding (sigmoidal curve); allosteric (Bohr, 2,3-BPG)
- Myoglobin: 1 subunit; non-cooperative (hyperbolic curve); muscle O_2 storage; higher affinity than Hb

VII.2 Enzyme Kinetics

- Enzymes lower Ea; do NOT change ΔG, K, or equilibrium position
- Michaelis-Menten: $V = V_{max}[S]/(K_m + [S])$
- Km: [S] at $V = V_{max}/2$; inversely related to enzyme affinity; lower Km = higher affinity
- $V_{max} = k_{cat} \cdot [E]_{total}$
- k_{cat} / K_m = catalytic efficiency; diffusion-limited maximum ~10^8-10^9 $M^{-1}s^{-1}$
- Lineweaver-Burk: $1/V = (K_m/V_{max})(1/[S]) + 1/V_{max}$; double reciprocal linearizes MM

Inhibition	Vmax	Km	Lineweaver-Burk
Competitive	Unchanged	↑	Same y-intercept, ↑ slope and x-intercept (closer to origin in 1/Km)
Noncompetitive (pure)	↓	Unchanged	Same x-intercept, ↑ y-intercept
Uncompetitive	↓	↓	Parallel lines (same slope, ↑ y-intercept)
Mixed	↓	↑ or ↓	Intersect off axes

- Allosteric enzymes: cooperative (sigmoidal V vs [S]); regulated by effectors at sites distinct from active site
- Feedback inhibition: end product inhibits early enzyme
- Regulation: covalent modification (phosphorylation), allosteric, zymogen activation, gene expression, compartmentalization

VII.3 Glycolysis and TCA

- Glycolysis: cytoplasm; 1 glucose → 2 pyruvate; net 2 ATP, 2 NADH; anaerobic-compatible
- Rate-limiting enzyme: phosphofructokinase-1 (PFK-1); activated by AMP, F-2,6-BP; inhibited by ATP, citrate
- Hexokinase (most tissues, low Km, inhibited by G6P) vs glucokinase (liver/β cells, high Km, NOT inhibited by G6P, induced by insulin)
- Pyruvate kinase: last step; activated by F-1,6-BP (feed-forward); inhibited by ATP, alanine
- Anaerobic: pyruvate → lactate (regenerates NAD^+); aerobic: pyruvate → acetyl-CoA
- PDH (pyruvate dehydrogenase): pyruvate + NAD^+ + CoA → acetyl-CoA + NADH + CO_2
- PDH cofactors: TPP (B1), lipoate, CoA (B5), FAD (B2), NAD (B3) - 'Tender Loving Care For Nancy'
- Activated by ADP, NAD^+, Ca^{2+}; inhibited by ATP, NADH, acetyl-CoA
- TCA cycle: mitochondrial matrix; per acetyl-CoA: 3 NADH, 1 $FADH_2$, 1 GTP, 2 CO_2

- Per glucose (2 acetyl-CoA): 6 NADH, 2 $FADH_2$, 2 GTP, 4 CO_2
- Rate-limiting: isocitrate dehydrogenase (also citrate synthase, α-ketoglutarate DH key)
- Inhibited by: ATP, NADH; activated by: ADP, NAD^+, Ca^{2+}

VII.4 Oxidative Phosphorylation

ETC complex	Function	Inhibitor
I (NADH dehydrogenase)	NADH → CoQ; pumps H^+	Rotenone
II (succinate dehydrogenase)	$FADH_2$ → CoQ; no H^+ pump	Malonate
III (cytochrome bc1)	CoQ → cyt c; pumps H^+	Antimycin A
IV (cytochrome c oxidase)	Cyt c → O_2 → H_2O; pumps H^+	Cyanide, CO, azide
V (ATP synthase)	H^+ flow → ATP	Oligomycin

- Electron path: NADH → I → CoQ → III → cyt c → IV → O_2; $FADH_2$ enters at CoQ (bypasses I)
- Proton gradient (chemiosmosis): pumped into intermembrane space; flows back through ATP synthase
- P:O ratio: NADH ~2.5 ATP; $FADH_2$ ~1.5 ATP
- Net ATP per glucose: ~30-32 (depends on shuttle)
- Uncouplers (2,4-DNP, thermogenin/UCP1 in brown fat): dissipate gradient as heat; ETC runs without ATP synthesis
- Brown fat: thermogenesis in infants and hibernators via UCP1

DISCRIMINATOR | ETC INHIBITORS vs UNCOUPLERS vs ATP SYNTHASE INHIBITORS

ETC INHIBITORS (rotenone, cyanide): stop electron flow; gradient collapses; no ATP. UNCOUPLERS (2,4-DNP, thermogenin): electrons flow but H^+ leaks back without making ATP → all energy as heat; O_2 consumption stays high. ATP SYNTHASE INHIBITORS (oligomycin): gradient builds up until ETC stalls; both ATP synthesis and O_2 consumption drop. Watch the O_2 consumption to tell them apart.

VII.5 Gluconeogenesis, Glycogen, PPP

- Gluconeogenesis: liver (also kidney); makes glucose during fasting; mostly reversed glycolysis
- Four unique bypass enzymes:
- 1. Pyruvate carboxylase (mito): pyruvate + CO_2 → OAA; uses ATP, biotin (B7); activated by acetyl-CoA
- 2. PEP carboxykinase (PEPCK, cytosol): OAA → PEP; uses GTP
- 3. Fructose-1,6-bisphosphatase: F-1,6-BP → F-6-P; rate-limiting; inhibited by AMP, F-2,6-BP
- 4. Glucose-6-phosphatase (ER, liver only): G-6-P → glucose; muscle lacks this
- Substrates: lactate (Cori cycle), glycerol, glucogenic AAs (alanine, glutamine, etc.); even-chain FA CANNOT (acetyl-CoA cannot net make glucose)
- Glycogenesis: glucose → G-6-P → G-1-P → UDP-glucose → glycogen (glycogen synthase: α-1,4 bonds; branching enzyme: α-1,6)

- Glycogenolysis: glycogen phosphorylase (rate-limiting); makes G-1-P → G-6-P
- Insulin: ↑ glycogen synthase (dephosphorylates); ↓ glycogen phosphorylase
- Glucagon/epinephrine: cAMP → PKA → phosphorylates both (active phosphorylase, inactive synthase)
- Glycogen storage diseases:
- Type I (von Gierke): G-6-Pase deficiency; severe fasting hypoglycemia, hepatomegaly
- Type II (Pompe): lysosomal α-1,4-glucosidase; cardiomegaly, hypotonia (only one affecting heart)
- Type III (Cori): debranching enzyme; milder hypoglycemia, normal lactate
- Type V (McArdle): muscle glycogen phosphorylase; exercise intolerance, no rise in lactate with exercise
- Pentose phosphate pathway (PPP/HMP shunt): cytoplasm; makes NADPH and ribose-5-P
- Rate-limiting: G6PD (glucose-6-phosphate dehydrogenase); produces NADPH
- NADPH uses: reductive biosynthesis (FA, cholesterol), glutathione regeneration (oxidative defense), respiratory burst in phagocytes
- G6PD deficiency: hemolytic anemia with oxidative stress (fava beans, sulfa drugs, primaquine); Heinz bodies, bite cells

VII.6 Lipid Metabolism

- Beta-oxidation (mitochondria): FA + ATP → acyl-CoA (cytoplasm) → carnitine shuttle → mito → cycle of dehydrogenation, hydration, oxidation, thiolysis
- Each round: 1 acetyl-CoA + 1 NADH + 1 $FADH_2$; chain shortened by 2 C
- Carnitine shuttle: rate-limiting; CPT-I (outer mito) inhibited by malonyl-CoA
- Odd-chain FA: ends with propionyl-CoA → methylmalonyl-CoA → succinyl-CoA (B12, biotin required)
- Unsaturated FA: needs isomerase and reductase
- Very long chain and branched: peroxisomes
- Fatty acid synthesis (cytoplasm): rate-limiting acetyl-CoA carboxylase (ACC); makes malonyl-CoA
- ACC activated by citrate, insulin; inhibited by palmitoyl-CoA, glucagon
- FA synthase: multi-domain enzyme; adds 2 C at a time using malonyl-CoA + NADPH; makes palmitate (C16)
- Acetyl-CoA from mito to cytoplasm via citrate shuttle
- Ketone bodies: liver makes during fasting/starvation/DKA; acetoacetate, β-hydroxybutyrate, acetone
- Substrates: acetyl-CoA from β-oxidation
- Liver cannot use ketones (no thiophorase); brain and muscle do
- Rate-limiting: HMG-CoA synthase (mitochondrial; ≠ cytosolic for cholesterol)
- Cholesterol synthesis: cytoplasm and ER; rate-limiting HMG-CoA reductase (target of statins)
- Cholesterol → bile acids, steroid hormones, vitamin D, membrane component

Lipoprotein	Carries	Source	Notes
Chylomicron	Dietary TG (mostly)	Intestine	Largest, lowest density
VLDL	Endogenous TG	Liver	Becomes IDL → LDL
IDL	Intermediate	VLDL remnants	
LDL	Cholesterol to tissues	From IDL	'Bad' cholesterol
HDL	Cholesterol from tissues to liver	Liver and intestine	'Good' cholesterol; reverse transport

VII.7 Amino Acid Catabolism and Urea Cycle

- Transamination: AA + α-KG → α-keto acid + glutamate (ALT for alanine, AST for aspartate); uses PLP (B6)
- Oxidative deamination of glutamate: glutamate dehydrogenase → α-KG + NH_3 + NADH
- NH_3 is toxic, especially to brain; converted to urea in liver
- Urea cycle (liver, partly mitochondrial, partly cytosolic): 5 enzymes
- 1. CPS-I (mito): NH_3 + CO_2 → carbamoyl phosphate; rate-limiting; activated by N-acetylglutamate
- 2. OTC (mito): carbamoyl-P + ornithine → citrulline
- 3. Argininosuccinate synthase (cytosol): citrulline + aspartate → argininosuccinate
- 4. Argininosuccinase: → arginine + fumarate
- 5. Arginase: arginine → urea + ornithine
- Net: 2 N (one from NH_3, one from aspartate) + 1 CO_2 + 3 ATP → 1 urea
- Hyperammonemia: defects in cycle → encephalopathy; OTC deficiency is X-linked, most common
- Ketogenic only AAs: Leucine, Lysine (can only make acetyl-CoA / acetoacetyl-CoA; cannot net make glucose)
- Glucogenic only AAs: most others
- Both: Phe, Tyr, Trp, Ile, Thr

VII.8 Nucleotide Metabolism

- De novo purine synthesis: from PRPP; ring built ON the sugar; uses glycine, aspartate, glutamine, CO_2, formyl-THF
- Rate-limiting: glutamine PRPP amidotransferase; inhibited by IMP, AMP, GMP
- De novo pyrimidine: ring built first, THEN attached to PRPP; uses aspartate, glutamine, CO_2
- Rate-limiting: CPS-II (cytoplasmic; different from urea cycle CPS-I)
- Salvage: HGPRT (purines) and APRT; saves bases that would otherwise be degraded
- Lesch-Nyhan: HGPRT deficiency; hyperuricemia, gout, self-mutilation, intellectual disability
- Purine degradation → uric acid (rate-limited by xanthine oxidase)
- Gout: ↑ uric acid; deposits in joints; treated with allopurinol (xanthine oxidase inhibitor)
- Key drugs:

- Methotrexate: inhibits dihydrofolate reductase → ↓ THF → ↓ thymidylate synthesis
- 5-FU: inhibits thymidylate synthase
- 6-MP: inhibits purine synthesis (must be activated by HGPRT)
- Hydroxyurea: inhibits ribonucleotide reductase (dNTP synthesis)
- Allopurinol: xanthine oxidase inhibitor (gout)

VII.9 Vitamins and Cofactors

Vitamin	Cofactor name	Key role	Deficiency
B1 (thiamine)	TPP	PDH, α-KG DH, transketolase, branched-chain	Beriberi, Wernicke-Korsakoff
B2 (riboflavin)	FAD, FMN	Redox	Cheilosis, glossitis
B3 (niacin)	NAD^+, $NADP^+$	Redox; from tryptophan	Pellagra (3D: dermatitis, diarrhea, dementia)
B5 (pantothenate)	CoA	Acyl carrier	Rare
B6 (pyridoxine)	PLP	Transamination, decarboxylation	Sideroblastic anemia, convulsions
B7 (biotin)	Biotin	Carboxylation (pyruvate carboxylase, ACC, propionyl-CoA carboxylase)	Rare; raw egg whites (avidin)
B9 (folate)	THF	1-C transfer, DNA synthesis	Megaloblastic anemia, neural tube defects
B12 (cobalamin)	Methylcobalamin, adenosylcobalamin	Methylmalonyl-CoA mutase, methionine synthase	Megaloblastic anemia + neurologic (SCD)
C (ascorbate)	-	Collagen hydroxylation, iron absorption, antioxidant	Scurvy
A (retinol)	-	Vision (retinal), gene expression	Night blindness, dry skin
D (cholecalciferol)	-	Ca^{2+} absorption	Rickets (kids), osteomalacia (adults)
E (tocopherol)	-	Antioxidant	Hemolysis, neurologic
K (phylloquinone)	-	γ-carboxylation of clotting factors	Bleeding (warfarin blocks)

- Fat-soluble: A, D, E, K (stored, toxic in excess)
- Water-soluble: B complex, C (excreted, B12 stored in liver)
- B12 absorption: requires intrinsic factor from parietal cells; absorbed in terminal ileum

VII.10 Hormonal Integration

State	Dominant hormone	Key fluxes
Fed	Insulin	Glycogenesis, lipogenesis, protein synthesis; glucose into muscle/fat (GLUT4)
Early fasting (post-absorptive)	Glucagon	Glycogenolysis dominates (~12 h supply)
Prolonged fasting (~1-3 d)	Glucagon, cortisol	Gluconeogenesis dominates; protein breakdown
Starvation (>3 d)	Cortisol, glucagon; ↓ T3	Ketones become brain fuel; protein conservation

- Insulin secretion: glucose → β cell → glucokinase → ↑ ATP → close K_ATP → depolarize → Ca^{2+} in → insulin release
- Glucagon: α cell; response to low glucose, AAs
- Cortisol: stress; gluconeogenesis, protein catabolism, immunosuppression, lipolysis
- Epinephrine: acute stress; glycogenolysis, lipolysis (β receptors)
- Type 1 diabetes: autoimmune β cell destruction; no insulin; ketosis-prone
- Type 2 diabetes: insulin resistance; eventual β cell exhaustion; not usually ketotic
- DKA: T1D; high glucose, ketones (β-hydroxybutyrate), metabolic acidosis, Kussmaul breathing
- HHS: T2D; very high glucose, NO significant ketosis, severe dehydration

VII.11 DNA, RNA, Translation, and Drugs

- DNA replication enzymes: helicase, topoisomerase, SSB, primase, DNA pol III (main, prok) or δ/ε (euk), DNA pol I (removes primer), ligase
- Proofreading: DNA pol 3'→5' exonuclease activity
- Telomerase: extends 3' end with TTAGGG; active in stem cells, germ cells, cancer
- Transcription: RNA pol II makes mRNA (eukaryotes); RNA pol does not need primer
- Eukaryotic mRNA: 5' cap (7-mG), splicing (snRNPs), 3' polyA tail
- Translation: ribosome (80S euk = 60S + 40S; 70S prok = 50S + 30S)
- Initiation, elongation (A site → P site → E site), termination (release factor at stop)

Antibiotic class	Target
Aminoglycosides (gentamicin)	30S; misreading
Tetracyclines	30S; block A site
Macrolides (erythromycin)	50S; block translocation
Chloramphenicol	50S; peptidyl transferase
Linezolid	50S; initiation
Rifampin	Bacterial RNA pol

Antibiotic class	Target
Fluoroquinolones	DNA gyrase (topoisomerase II)
β-lactams (penicillin)	Cell wall (transpeptidase)
Vancomycin	Cell wall (D-Ala-D-Ala)

DISCRIMINATOR | HEXOKINASE vs GLUCOKINASE

BOTH phosphorylate glucose → G-6-P. HEXOKINASE: most tissues, LOW Km (high affinity, works at low glucose), low Vmax, INHIBITED by G-6-P (its own product). GLUCOKINASE: liver and pancreatic β cells, HIGH Km (low affinity, scales with glucose), high Vmax, NOT inhibited by G-6-P, INDUCED by insulin. Glucokinase is a glucose sensor in β cells; the high Km is why insulin secretion scales with blood glucose.

DISCRIMINATOR | DE NOVO vs SALVAGE NUCLEOTIDE SYNTHESIS

DE NOVO: builds nucleotides from scratch (small precursors: amino acids, CO_2, formate); energy-expensive, used in dividing cells. SALVAGE: recycles free bases via HGPRT (purines) or APRT; uses much less energy. Lesch-Nyhan = no HGPRT = no salvage → de novo overdrive → uric acid overproduction → gout and neuropsychiatric features.

DISCRIMINATOR | KETOGENIC vs GLUCOGENIC AMINO ACIDS

KETOGENIC ONLY (Leu, Lys): produce only acetyl-CoA or acetoacetyl-CoA; CANNOT net produce glucose because acetyl-CoA → 2 CO_2 in TCA without net carbon gain. GLUCOGENIC: produce pyruvate or TCA intermediates (other than acetyl-CoA), which can enter gluconeogenesis. BOTH: Phe, Tyr, Trp, Ile, Thr.

CLINICAL BRIDGE | DIABETIC KETOACIDOSIS (DKA)

T1D patient stops insulin → unrestrained lipolysis → flood of FFA to liver → β-oxidation overruns TCA capacity → acetyl-CoA piles up → ketogenesis (acetoacetate, β-hydroxybutyrate) → metabolic acidosis with high anion gap. Hyperglycemia → osmotic diuresis → dehydration. Kussmaul respiration is the respiratory compensation. Treatment: insulin, IV fluids, K^+ replacement (insulin shifts K^+ into cells).

CLINICAL BRIDGE | G6PD DEFICIENCY

X-linked deficiency of glucose-6-phosphate dehydrogenase → less NADPH → reduced glutathione regeneration → oxidative damage on RBCs. Triggered by oxidative stress (fava beans, sulfa drugs, primaquine, infection). Smear: Heinz bodies (denatured Hb) and bite cells. Most common human enzyme deficiency; provides resistance to malaria, hence high frequency in Mediterranean and African populations.

HIGH-YIELD

- Michaelis-Menten: V = Vmax[S]/(Km+[S]); Km is [S] at half Vmax
- Competitive ↑ Km only; noncompetitive ↓ Vmax only; uncompetitive ↓ both
- Glycolysis RLE: PFK-1; gluconeogenesis RLE: F-1,6-BPase
- PDH cofactors: TPP, lipoate, CoA, FAD, NAD (1, lipoate, 5, 2, 3)
- ETC inhibitors stop flow; uncouplers leak protons (heat); oligomycin blocks ATP synthase
- Urea cycle RLE: CPS-I (activated by N-acetylglutamate)
- Ketogenic only: Leu, Lys
- Insulin: fed, anabolic; Glucagon: fasted, catabolic
- B12 + folate deficiency both cause megaloblastic anemia; only B12 causes neuro

PART VIII · THE ULTIMATE DISCRIMINATOR INDEX

One row per look-alike pair across all subjects. The single distinguishing feature is what to commit to memory. If you can complete this table from the left column alone, you have the discriminator layer of the test mastered.

Pair (A vs B)	The single distinction
Mitosis vs Meiosis I	Mitosis: sister chromatids separate; Meiosis I: homologs separate (reductional)
Meiosis I vs Meiosis II	Meiosis I halves chromosome number; Meiosis II separates sister chromatids on haploid cells
Prokaryote vs Eukaryote	Eukaryote has membrane-bound nucleus and organelles; prokaryote does not
Rough ER vs Smooth ER	Rough has ribosomes (secreted/membrane proteins); smooth makes lipids, detoxifies
Mitochondrion vs Chloroplast	Both endosymbionts; chloroplast does photosynthesis (plants only)
Active vs Passive transport	Active uses ATP (or gradient); passive goes down gradient, no ATP
Primary vs Secondary active transport	Primary uses ATP directly; secondary uses ion gradient established by primary
MHC I vs MHC II	I on all nucleated cells (endogenous peptides); II on APCs only (exogenous)
Helper T (CD4) vs Cytotoxic T (CD8)	CD4 binds MHC II, coordinates; CD8 binds MHC I, kills
Innate vs Adaptive immunity	Innate: fast, no memory, pattern-based; Adaptive: slow first, memory, specific
IgG vs IgM	IgG: secondary response, crosses placenta; IgM: primary response, pentamer
Gram positive vs Gram negative	G+ thick peptidoglycan retains crystal violet; G- has outer membrane + LPS
Sympathetic vs Parasympathetic	Sympathetic = NE, fight or flight; Parasympathetic = ACh, rest and digest

Pair (A vs B)	The single distinction
Rods vs Cones	Rods: low light, no color, peripheral; Cones: color, central, fovea
Right shift vs Left shift (Hb-O_2)	Right releases O_2 at tissue (Bohr); Left holds O_2 (fetal Hb, CO)
Loop of Henle descending vs ascending	Descending: water out only; Ascending: solute out only
Aldosterone vs ADH	Aldosterone: Na^+ in (water follows), K^+ out; ADH: water reabsorption via aquaporins
Insulin vs Glucagon	Insulin: fed/anabolic; Glucagon: fasting/catabolic
Hexokinase vs Glucokinase	Hexokinase low Km, inhibited by G6P; Glucokinase high Km, not inhibited, in liver/β cells
Glycolysis vs Gluconeogenesis RLE	Glycolysis: PFK-1; Gluconeogenesis: F-1,6-bisphosphatase
Glycogenesis vs Glycogenolysis	Glycogenesis: glycogen synthase (insulin); Glycogenolysis: phosphorylase (glucagon)
ETC inhibitor vs Uncoupler	Inhibitor stops electron flow; uncoupler dissipates gradient as heat (O_2 use stays high)
Ketogenic vs Glucogenic AA	Ketogenic only (Leu, Lys) → no net glucose; glucogenic enters gluconeogenesis
De novo vs Salvage nucleotide	De novo from scratch; salvage recycles bases (HGPRT, APRT)
DNA vs RNA	DNA: deoxyribose, T, double-stranded; RNA: ribose, U, single-stranded
Replication vs Transcription	Replication: DNA → DNA (entire genome); Transcription: DNA → RNA (one gene)
Leading vs Lagging strand	Leading: continuous 5'→3' toward fork; Lagging: discontinuous Okazaki fragments
Operon vs Eukaryotic gene regulation	Operon: polycistronic, one switch; Eukaryote: monocistronic, multilayer regulation
Endotoxin vs Exotoxin	Endo: LPS in G- outer membrane, heat stable; Exo: secreted proteins, often heat-labile
Alpha helix vs Beta sheet	Helix: H-bonds i to i+4 within one strand; Sheet: H-bonds between strands
Enantiomers vs Diastereomers	Enantiomers: mirror images, ALL stereocenters opposite; Diastereomers: some but not all differ
SN1 vs SN2	SN1: 3° substrate, polar protic, racemize, carbocation; SN2: 1°, polar aprotic, inversion, concerted
E1 vs E2	E1: like SN1 (carbocation, weak base); E2: strong base, anti-periplanar, concerted
Ortho/para vs Meta directors	Activators direct ortho/para (donate); deactivators direct meta (withdraw); halogens are exception
Aldehyde vs Ketone	Aldehyde: CHO (more reactive, oxidizes to acid); Ketone: C between two carbons

Pair (A vs B)	The single distinction
Carboxylic acid vs Ester	COOH: H-bond donor, acidic; Ester: no H on O, fruity smell, hydrolyzes to acid + alcohol
Reducing vs Non-reducing sugar	Reducing has free anomeric C (open chain); sucrose is non-reducing
Alpha vs Beta glucose	Alpha: anomeric OH down (Haworth) = starch/glycogen; Beta: OH up = cellulose
Ionic vs Covalent vs Metallic bond	Ionic: $\Delta EN > 1.7$; Covalent: < 1.7; Metallic: electron sea
H-bond vs Dipole-dipole	H-bond: specifically N-H, O-H, F-H to N, O, F; subset of dipole-dipole
Endothermic vs Exothermic	Endo absorbs heat ($\Delta H > 0$); Exo releases heat ($\Delta H < 0$)
Spontaneous vs Nonspontaneous	Spontaneous: $\Delta G < 0$; equilibrium $\Delta G = 0$
Galvanic vs Electrolytic	Galvanic: spontaneous, $E > 0$; Electrolytic: forced, $E < 0$
Strong vs Weak acid	Strong: fully dissociates (Ka large); Weak: partial dissociation (has buffering)
Buffer vs Equivalence point	Buffer region: pH near pKa; Equivalence: moles acid = moles base
Le Chatelier shift	Stress is opposed: add reactant $\rightarrow$ forward; $\uparrow T \rightarrow$ endothermic direction
Elastic vs Inelastic collision	Elastic: KE and p conserved; Inelastic: only p conserved (KE lost to heat)
Series vs Parallel resistors	Series: $R_{eq} = \Sigma R$ (current same); Parallel: $1/R_{eq} = \Sigma 1/R$ (voltage same)
Converging vs Diverging lens	Converging: $f > 0$, can be real or virtual; Diverging: $f < 0$, always virtual upright smaller
Reflection vs Refraction	Reflection: bounce off (angles equal); Refraction: bend through (Snell)
Doppler approach vs recede	Approach: higher frequency; Recede: lower frequency
Photon intensity vs frequency	Intensity: # photons; Frequency: energy per photon ($E = hf$)
Classical vs Operant conditioning	Classical: pair stimuli, reflexive response; Operant: pair behavior with consequence, voluntary
Positive vs Negative reinforcement	Both $\uparrow$ behavior; positive adds something, negative removes something
Reinforcement vs Punishment	Reinforcement $\uparrow$ behavior; Punishment $\downarrow$ behavior
Encoding vs Retrieval failure	Encoding: never stored; Retrieval: stored but cannot access (tip of tongue)
James-Lange vs Cannon-Bard	James-Lange: body first, then emotion; Cannon-Bard: simultaneous
Schachter-Singer vs others	Two-factor: requires cognitive label PLUS arousal
Proactive vs Retroactive interference	Proactive: old blocks new; Retroactive: new blocks old

Pair (A vs B)	The single distinction
Fundamental attribution error vs Self-serving bias	FAE: others' behavior = dispositional; Self-serving: own successes dispositional, failures situational
Functionalism vs Conflict theory	Functionalism: society = cooperating parts; Conflict: society = competition over resources
Symbolic interactionism	Micro-level focus on meanings derived from interaction
Ascribed vs Achieved status	Ascribed: born into (race, sex assigned at birth); Achieved: earned (profession)
Race vs Ethnicity	Race: physical/biological emphasis (socially constructed); Ethnicity: shared culture/heritage
Type I vs Type II error	Type I: false positive (reject true null); Type II: false negative (fail to reject false null)

PART IX · TEST-DAY ANCHORS

IX.1 Section-by-Section Approach

- Chem/Phys (C/P): 59 questions, 95 min; mostly physics + gen chem + some biochem; passages quantitative; estimate aggressively, watch units
- CARS (Critical Analysis and Reasoning Skills): 53 questions, 90 min; no outside knowledge; main idea + author tone + inference; do not bring real-world knowledge into answers
- Bio/Biochem (B/B): 59 questions, 95 min; bio + biochem + some orgo; experimental passages dominate; reason from mechanism
- Psych/Soc (P/S): 59 questions, 95 min; terminology-heavy but mechanism-light; learn the vocabulary cold

IX.2 Pacing Strategy

- Sciences: ~1.5 min/question, 7-8 min/passage on average; discretes go faster, passages slower
- CARS: ~10 min per passage including questions; do NOT save them for end of an answer block
- Skip and flag rule: if you cannot get traction in 30 seconds, mark and move on; come back at end
- Last 5 minutes: confirm every question has an answer (no blanks; no penalty for guessing)
- Discrete questions (no passage): often appear in clusters; treat as pure recall

IX.3 Mechanism-First Reasoning Template

1. Identify what kind of question it is: recall, calculation, experimental interpretation, reasoning from passage
2. If experimental: read graph/table FIRST; identify dependent and independent variables; note units
3. Restate the mechanism in your own words: what is the molecule/system doing, why
4. Predict the answer BEFORE looking at choices
5. Cross out clearly wrong choices (often two are easy eliminations)
6. If stuck between two: which one matches the MECHANISM exactly, not just the vocabulary

IX.4 If You Only Memorize One Thing Per Topic

Topic	The one thing
Cell biology	Organelle functions table
Genetics	Hardy-Weinberg ($p^2 + 2pq + q^2 = 1$) and patterns of inheritance
Molecular biology	Central dogma: replication (semiconservative), transcription, translation
Endocrine	Insulin/glucagon counter-regulation
Nervous system	Action potential phases (Na^+ in, K^+ out)
Cardiovascular	Cardiac cycle and Starling forces
Respiratory	O_2-Hb curve and Bohr effect
Renal	Nephron segments and what each does
Digestive	Pancreatic/intestinal enzymes table
Immune	MHC I vs II; innate vs adaptive
Atomic structure	Periodic trends across and down
Bonding	IMF ranking: ion-dipole > H-bond > dipole-dipole > LDF
Thermo	$\Delta G = \Delta H - T\Delta S$
Kinetics	Rate-determining step controls rate law; catalysts ↓ Ea only
Equilibrium	Le Chatelier: shift opposes stress
Acid-base	Henderson-Hasselbalch: pH = pKa + log(base/acid)
Electrochem	E°_cell = E°_cathode - E°_anode; $\Delta G = -nFE$
Orgo structure	Hybridization and chirality (R/S, E/Z)
Orgo mechanisms	SN1/SN2/E1/E2 4-factor table (substrate, nucleophile, solvent, temperature)
Carbonyl	Reactivity ranking: acid halide > anhydride > ester > amide
Aromatic	Activators ortho/para, deactivators meta; halogens are weak deactivators that direct o/p
Spectroscopy	IR ~1700 = C=O; NMR n+1 splitting; aromatic H at 6.5-8 ppm
Kinematics	$v^2 = v_0^{\ 2} + 2a\Delta x$ and projectile x/y independence
Newton	F = ma; weight = mg
Energy	W_net = ΔKE; conserve mechanical energy without friction
Fluids	$A_1v_1 = A_2v_2$ (continuity); Bernoulli faster = lower pressure
Waves	$v = f\lambda$; Doppler approach raises pitch

Topic	The one thing
Optics	Snell: $n_1 \sin\theta_1 = n_2 \sin\theta_2$; lens equation 1/f = 1/d_o + 1/d_i
Circuits	V = IR; capacitors and resistors in series vs parallel
Modern	Photon E = hf; half-life: $N = N_0(1/2)^n$
Psychology	Classical vs operant; reinforcement schedules
Memory	Sensory → short-term (7±2, ~20 s) → long-term
Personality	Big Five = OCEAN
Social	Fundamental attribution error and bystander effect
Sociology	Three theoretical lenses: functionalism, conflict, symbolic interactionism
Amino acids	20 AAs grouped by polarity, charge, aromaticity
Enzyme kinetics	Michaelis-Menten and 3 inhibition types on Lineweaver-Burk
Glycolysis	PFK-1 is the gatekeeper; activated by AMP, inhibited by ATP/citrate
TCA	3 NADH + 1 $FADH_2$ + 1 GTP per acetyl-CoA
ETC	Complexes I-IV pump H^+ except II; ATP synthase makes ATP from gradient
Gluconeogenesis	4 unique enzymes (PC, PEPCK, F-1,6-BPase, G-6-Pase); only in liver/kidney
β-oxidation	Acyl-CoA → 1 acetyl-CoA + 1 NADH + 1 $FADH_2$ per round
Urea cycle	5 enzymes; CPS-I is RLE in mitochondria; activated by N-acetylglutamate
Vitamins	Fat-soluble = ADEK; B12 needs intrinsic factor

IX.5 Day-Before and Day-Of Anchors

- Day before: light review of cheat sheets only; no new material; sleep early
- Morning of: eat protein + complex carbs; coffee if customary, not a new variable
- Bring: ID, snacks for breaks, water (allowed in locker), layered clothing
- Between sections: stand, stretch, hydrate, brief carbs; do NOT review missed material
- If anxious mid-test: 4 breaths in / 6 breaths out; reset to the current question only
- Last question of section: commit to your best answer; no question is worth more than the next

IX.6 Common Trap Patterns

- Extreme language (always, never, all, none): almost always wrong in CARS and Psych/Soc
- Choices that mix correct vocabulary with the wrong relationship: read carefully
- Calculation choices that include common unit errors (×10 off): always check magnitude

- Mechanism trap: the answer that 'sounds right' but contradicts the underlying chemistry
- Reverse causality: confusing what regulates what
- Out-of-scope answers in CARS: must be derivable from the passage
- Premise mismatch: when the question stem flips a normal scenario (e.g., inhibitor instead of activator)

WHY MECHANISM BEATS MEMORIZATION ON TEST DAY

The MCAT writes questions to discriminate students who memorized facts from students who understood mechanisms. The fastest way to do this is to put a familiar mechanism in an unfamiliar context (a new enzyme name, a novel drug, a passage about a non-standard organism). A memorized fact transfers poorly. A mechanism transfers automatically. If you can explain WHY an enzyme behaves the way it does, you can predict its behavior under any modification the test writer dreams up.

THE BOTTOM LINE

Book 7 compresses Books 1-6 into a final-pass reference: biology, general chemistry, organic chemistry, physics, psychology and sociology, and biochemistry, each reduced to its highest-yield bullets, tables, and discriminators. Use it for spaced retrieval in the final weeks, not as a first-pass teacher. The Master Discriminator Index in Part VIII is the single best diagnostic tool for what you still confuse. Test-day performance follows mechanism, not memorization. The C-Factor Series ends where it began: every concept earned mechanistically, retrievable in seconds, transferable to any context the exam puts in front of you.

→ See: Book 1 (Biology), Book 2 (General Chemistry), Book 3 (Organic Chemistry), Book 4 (Physics), Book 5 (Psychology and Sociology), Book 6 (Biochemistry). This review consolidates all six. If anything in Part II through Part VII felt unfamiliar on first read, return to the parent chapter for the full mechanistic treatment, then use this volume to lock it in.