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HIGHLIGHTED HANDOUT

MB501T

Cell Biology (Theory)

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COMPREHENSIVE STUDY HANDOUT

MB501T

Cell Biology (Theory)

Lecture-wise study notes prepared for Virtual University of Pakistan students.

Covers all major topics with objectives, key definitions, summaries and exam pointers.



13 study units

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Unit 1: The Cell: Discovery, Theory and Organization

Learning Objectives

- Objective 1:** Trace the discovery of cells and the cell theory
- Objective 2:** Compare prokaryotic and eukaryotic cells
- Objective 3:** Explain microscopy principles underlying cell biology

Cell biology began with the microscope: Hooke's cork 'cells' (1665), Leeuwenhoek's living microbes, Brown's nucleus, and Schleiden and Schwann's cell theory (1838-39): all living things are made of cells, the cell is life's basic unit, and all cells arise from pre-existing cells (Virchow's later addition). The theory unified biology: despite life's diversity, one cellular organization underlies it.

Prokaryotes (bacteria, archaea) lack membrane-bound organelles and a true nucleus; their circular DNA lies in the nucleoid, and they are small (0.5-5 μm) with 70S ribosomes. Eukaryotes (protists, fungi, plants, animals) compartmentalize functions in organelles, carry linear chromosomes in a nucleus, and are typically 10-100 μm . The endosymbiotic theory explains mitochondria and chloroplasts as engulfed bacteria, supported by their own DNA and double membranes.

Seeing cells requires microscopy. Light microscopes resolve ~ 200 nm, enough for cells and nuclei with staining; fluorescence and confocal add molecular specificity and optical sectioning. Electron microscopy (TEM for ultrastructure, SEM for surfaces) resolves nanometers but kills the specimen.

Very important: match the tool to the question; exams often ask which microscope reveals a given structure. 

Key Definitions

Cell theory is defined as all organisms consist of cells; the cell is life's basic unit; cells arise only from cells.

Endosymbiotic theory is defined as mitochondria and chloroplasts originated as engulfed free-living bacteria.

Resolution is defined as the minimum distinguishable distance; ~ 200 nm for light microscopy.

Key Points to Remember

- Cell theory: Schleiden, Schwann (1838-39); Virchow added 'from pre-existing cells'.
- Prokaryote: no organelles, nucleoid, 70S ribosomes, small. Eukaryote: organelles, nucleus, larger.
- Endosymbiosis evidence: own DNA, double membranes, bacterial-like ribosomes.
- Light (~ 200 nm, live, stained) vs EM (nm, dead, ultrastructure).

Unit Summary

Cell theory unified biology around the cell; prokaryotes and eukaryotes differ in compartmentalization; microscopy choice follows the structure studied.

Exam Pointer

Very important: state cell theory's three tenets and evidence for endosymbiosis. Microscope-selection questions are standard.

Unit 2: The Plasma Membrane: Structure and Function

Learning Objectives

- Objective 1:** Describe the fluid mosaic model
- Objective 2:** Explain membrane lipid and protein components
- Objective 3:** Relate membrane structure to selective permeability

The fluid mosaic model (Singer and Nicolson, 1972) pictures the membrane as a phospholipid bilayer with embedded proteins drifting laterally like icebergs. Phospholipids, with hydrophilic heads and hydrophobic tails, self-assemble into bilayers; cholesterol tunes fluidity; glycolipids and glycoproteins form the glycocalyx for recognition. The membrane is asymmetric: the two leaflets differ in lipid and protein composition.

Membrane proteins do the work: integral proteins span the bilayer (channels, transporters, receptors), peripheral proteins attach to surfaces (enzymes, cytoskeletal anchors). Functions include selective transport, enzymatic activity, signal transduction, cell-cell recognition, and attachment. The sodium-potassium pump, burning ATP to expel Na^+ and import K^+ , maintains the electrochemical gradients underlying nerve impulses.

Selective permeability emerges from chemistry: small nonpolar molecules cross freely, small polar ones slowly, ions and large polars need proteins. Very important: fluidity depends on temperature, fatty-acid saturation (kinks from double bonds increase fluidity) and cholesterol (buffers both extremes); examiners love asking how cold-adapted membranes differ.

Key Definitions

Fluid mosaic model is defined as the membrane as a fluid lipid bilayer with embedded drifting proteins.

Selective permeability is defined as allowing some substances through while blocking others.

Glycocalyx is defined as the carbohydrate coat on the cell surface for recognition.

Key Points to Remember

- Bilayer: phospholipids (heads out, tails in); cholesterol modulates fluidity; proteins do the work.
- Protein classes: integral (span) vs peripheral (surface); functions: transport, enzymes, signaling, recognition.
- Na^+/K^+ pump maintains gradients at ATP cost.
- Permeability follows chemistry; fluidity follows temperature, saturation, cholesterol.

Unit Summary

The fluid mosaic membrane's lipid bilayer hosts functional proteins, achieving selective permeability with tunable fluidity.

Exam Pointer

Very important: draw and label the fluid mosaic model; explain how unsaturated fatty acids affect fluidity.

Unit 3: Membrane Transport

Learning Objectives

- Objective 1:** Distinguish passive and active transport
- Objective 2:** Explain diffusion, osmosis and facilitated diffusion
- Objective 3:** Describe endocytosis and exocytosis

Passive transport moves substances down their gradients without ATP: simple diffusion (O₂, CO₂ across the bilayer), facilitated diffusion via channel or carrier proteins (glucose through GLUT transporters), and osmosis, water's diffusion across selectively permeable membranes toward higher solute concentration. Tonicity predicts cell fate: animal cells lyse in hypotonic and shrivel in hypertonic solutions; plant cell walls resist lysis, generating turgor.

Active transport moves against gradients using ATP: primary (directly ATP-driven, like the Na⁺/K⁺ pump and proton pumps) and secondary (using an ion gradient built by primary transport, e.g. Na⁺/glucose symport). Coupled transport comes as symport (same direction) and antiport (opposite). The energy cost is the price of maintaining the internal order life requires.

Bulk transport moves large cargo in vesicles: endocytosis (phagocytosis for solids, pinocytosis for fluids, receptor-mediated for specific ligands like LDL) brings material in; exocytosis ships secretory products out, also adding membrane. Very important: receptor-mediated endocytosis of LDL and its link to cholesterol regulation is a classic applied question. 

Key Definitions

Osmosis is defined as diffusion of water across a selectively permeable membrane toward higher solute concentration.

Active transport is defined as ATP-driven movement of substances against their concentration gradient.

Receptor-mediated endocytosis is defined as selective uptake via receptors clustering in coated pits.

Key Points to Remember

- Passive: diffusion, facilitated diffusion, osmosis; no ATP; down gradients.
- Tonicity: hypotonic lyses animal cells; walls give plants turgor.
- Active: primary (ATP direct) and secondary (gradient-driven); symport/antiport.
- Bulk: endocytosis in (phago-, pino-, receptor-mediated), exocytosis out.

Unit Summary

Membranes transport passively down gradients or actively at ATP cost, with vesicular bulk transport for large cargo.

Exam Pointer

Very important: predict cell behavior in different tonicities and explain Na⁺/glucose symport as secondary active transport.

Unit 4: The Endomembrane System

Learning Objectives

- Objective 1:** Trace the secretory pathway through organelles
- Objective 2:** Distinguish rough and smooth ER
- Objective 3:** Explain Golgi processing and vesicular trafficking

The endomembrane system, nuclear envelope, ER, Golgi, lysosomes, vacuoles and plasma membrane, functions as an integrated manufacturing and shipping network. The nuclear envelope's double membrane with pores connects to the ER, linking gene expression to protein production physically.

The rough ER, studded with ribosomes, synthesizes secretory and membrane proteins, folding them with chaperones and starting glycosylation. The smooth ER synthesizes lipids and steroids, detoxifies drugs, and stores calcium (as sarcoplasmic reticulum in muscle). Proteins leave the ER in COPII vesicles for the Golgi, with COPI handling retrograde return of escaped residents.

The Golgi apparatus, stacked cisternae with cis, medial and trans faces, modifies (glycosylation), sorts and packages: lysosomal enzymes get mannose-6-phosphate tags, secretory proteins concentrate in vesicles. Very important: the secretory pathway order (ER → Golgi → vesicle → outside/lysosome) is among the most tested sequences; learn the vesicle coats (COPII forward, COPI backward, clathrin for endocytosis/exocytosis).

Key Definitions

Endomembrane system is defined as the integrated set of internal membranes: nuclear envelope, ER, Golgi, lysosomes, vacuoles, plasma membrane.

Secretory pathway is defined as the route of proteins from rough ER through Golgi to secretion or organelles.

Glycosylation is defined as attachment of carbohydrate chains to proteins, beginning in the ER and refined in Golgi.

Key Points to Remember

- Rough ER: protein synthesis/folding; smooth ER: lipids, detox, Ca²⁺ storage.
- Vesicle coats: COPII (ER→Golgi), COPI (retrograde), clathrin (endocytosis/secretion).
- Golgi: cis→medial→trans processing; mannose-6-phosphate tags lysosomal enzymes.
- Pathway order ER→Golgi→destination is core exam material.

Unit Summary

The endomembrane system manufactures, modifies and ships proteins and lipids through the ER-Golgi-vesicle pathway with specific molecular tags and coats.

Exam Pointer

Very important: trace a secretory protein's journey naming coats and tags; distinguish rough vs smooth ER functions.

Unit 5: Mitochondria and Chloroplasts: Energy Organelles

Learning Objectives

Objective 1: Describe mitochondrial and chloroplast structure

Objective 2: Outline cellular respiration stages

Objective 3: Outline photosynthesis stages

Mitochondria, double-membraned with a folded inner cristae membrane, house aerobic respiration: glycolysis (cytosol, glucose to pyruvate, net 2 ATP), pyruvate oxidation and the Krebs cycle (mitochondrial matrix, CO₂ and NADH/FADH₂), and oxidative phosphorylation (cristae: electron transport chain pumps protons; ATP synthase harnesses the gradient, ~26-28 ATP). The chemiosmotic mechanism, proton-motive force driving ATP synthase, is Mitchell's Nobel-winning insight.

Chloroplasts capture light: thylakoid membranes hold photosystems II and I; light reactions split water, release O₂, and make ATP and NADPH; the Calvin cycle (stroma) fixes CO₂ into sugar using that ATP and NADPH (Rubisco as the carbon-fixing enzyme). The two organelles are complementary: photosynthesis stores energy, respiration releases it.

Both retain bacterial-like DNA, ribosomes and double membranes, the endosymbiosis evidence from Unit 1. Very important: energy accounting questions (ATP yields, where each stage occurs) are perennial; also know that chemiosmosis unifies both organelles' ATP production. 

Key Definitions

Chemiosmosis is defined as ATP synthesis driven by a proton gradient across a membrane via ATP synthase.

Krebs cycle is defined as the mitochondrial matrix cycle oxidizing acetyl-CoA to CO₂, yielding NADH/FADH₂.

Calvin cycle is defined as the stromal reactions fixing CO₂ into sugar using ATP and NADPH.

Key Points to Remember

- Respiration: glycolysis (cytosol) -> Krebs (matrix) -> ETC/ATP synthase (cristae).
- Chemiosmosis: proton gradient -> ATP; Mitchell's mechanism, common to both organelles.
- Photosynthesis: light reactions (thylakoids: ATP, NADPH, O₂) -> Calvin (stroma: CO₂ fixation).
- Endosymbiotic traits: own DNA, 70S-like ribosomes, double membranes.

Unit Summary

Mitochondria release energy via respiration and chloroplasts capture it via photosynthesis, both using chemiosmotic ATP synthesis and bearing endosymbiotic signatures.

Exam Pointer

Very important: tabulate respiration stages with locations and yields; explain chemiosmosis in both organelles.

Unit 6: Lysosomes, Vacuoles and Peroxisomes

Learning Objectives

Objective 1: Explain lysosomal digestion and autophagy

Objective 2: Describe vacuole functions in plants

Objective 3: Outline peroxisomal reactions

Lysosomes are the cell's demolition crew: membrane sacs of acid hydrolases (active at pH ~5, maintained by proton pumps) digesting macromolecules. They handle phagocytosed material, worn organelles via autophagy, and, in development, programmed tissue removal. Tay-Sachs disease, missing a lysosomal enzyme, shows the cost of failure: undigested substrate accumulates fatally in neurons.

Plant central vacuoles are multifunctional: storing water, ions and pigments; maintaining turgor pressure against the wall; sequestering toxins; and aiding growth by expansion. Contractile vacuoles in freshwater protists pump out excess water. The vacuole's size, up to 90% of a plant cell, reflects its importance.

Peroxisomes oxidize fatty acids and detoxify: they produce hydrogen peroxide as a byproduct and immediately destroy it with catalase. In leaves, they participate in photorespiration. Very important: contrast lysosomes (acid hydrolases, digestion) with peroxisomes (oxidative reactions, H₂O₂ handling); confusing them is a common error.

Key Definitions

Lysosome is defined as a vesicle of acid hydrolases digesting macromolecules and worn organelles.

Autophagy is defined as lysosomal degradation of the cell's own components for renewal.

Peroxisome is defined as an oxidative organelle producing and detoxifying hydrogen peroxide.

Key Points to Remember

- Lysosomes: acid hydrolases (pH~5); digestion, autophagy; Tay-Sachs shows failure cost.
- Plant vacuoles: storage, turgor, toxin sequestration; contractile vacuoles expel water.
- Peroxisomes: fatty-acid oxidation, H₂O₂ made and destroyed by catalase.
- Lysosome vs peroxisome: digestion vs oxidation; do not confuse.

Unit Summary

Lysosomes digest, vacuoles store and pressurize, peroxisomes oxidize: three specialized compartments with distinct chemistries.

Exam Pointer

Exam pointer: contrast lysosomes and peroxisomes precisely; explain autophagy and turgor with examples.

Unit 7: The Nucleus and Chromatin

Learning Objectives

- Objective 1:** Describe nuclear structure and the pore complex
- Objective 2:** Explain chromatin organization: DNA to chromosome
- Objective 3:** Distinguish euchromatin and heterochromatin

The nucleus houses the genome behind a double nuclear envelope perforated by nuclear pore complexes, massive selective gates importing proteins and exporting RNA. Inside, the nucleolus manufactures ribosomal RNA and assembles ribosome subunits. The envelope's inner lamina organizes chromatin and breaks down during mitosis.

DNA packaging is hierarchical: DNA wraps histones forming nucleosomes ('beads on a string'), which coil into the 30-nm fiber, loop onto scaffolds, and condense into metaphase chromosomes, achieving ~10,000-fold compaction. Histone modifications (acetylation, methylation) regulate access: acetylation generally opens chromatin for transcription, an epigenetic layer atop sequence.

Euchromatin is loosely packed, gene-rich and transcriptionally active; heterochromatin is condensed and mostly silent (constitutive at centromeres, facultative like the inactivated X). Very important: link structure to function explicitly; examiners ask why packaging matters, expecting the access-regulation answer.

Key Definitions

Nucleosome is defined as DNA wrapped around a histone octamer; the basic chromatin unit.

Euchromatin is defined as loosely packed, transcriptionally active chromatin.

Heterochromatin is defined as condensed, largely silent chromatin.

Key Points to Remember

- Nuclear pores: selective transport; nucleolus: ribosome production.
- Packaging: DNA → nucleosomes → 30-nm fiber → loops → chromosome.
- Histone acetylation opens chromatin (epigenetic regulation).
- Euchromatin (active) vs heterochromatin (silent); structure serves access control.

Unit Summary

The nucleus organizes DNA through hierarchical chromatin packaging, with pore-mediated transport and epigenetic regulation of access.

Exam Pointer

Very important: draw the packaging hierarchy and explain how histone modification regulates transcription.

Unit 8: Ribosomes and Protein Synthesis

Learning Objectives

Objective 1: Describe ribosome structure

Objective 2: Outline translation: initiation, elongation, termination

Objective 3: Explain co-translational targeting to the ER

Ribosomes are rRNA-protein machines: large and small subunits (80S in eukaryotes, 70S in prokaryotes and organelles) with A, P and E sites choreographing tRNA. The ribosome is a ribozyme: rRNA catalyzes peptide-bond formation, a relic of the RNA world. Free ribosomes make cytosolic proteins; bound ribosomes (rough ER) make secretory and membrane proteins.

Translation proceeds in three phases. Initiation: the small subunit, initiator tRNA and factors assemble at the start codon (AUG). Elongation: codons are read sequentially, aminoacyl-tRNAs enter the A site, peptide bonds form, and the ribosome translocates, consuming GTP. Termination: release factors recognize stop codons, freeing the polypeptide. The genetic code's degeneracy (multiple codons per amino acid) buffers mutations.

Proteins destined for secretion carry an N-terminal signal peptide; the signal recognition particle (SRP) pauses translation and docks the ribosome to the ER translocon, achieving co-translational import.

Very important: antibiotics exploit ribosome differences (70S vs 80S); this applied link frequently appears in exams. 

Key Definitions

Translation is defined as ribosomal synthesis of protein from mRNA codons.

Codon is defined as a three-nucleotide mRNA unit specifying an amino acid or stop.

Signal peptide is defined as an N-terminal tag directing nascent proteins to the ER.

Key Points to Remember

- Ribosome: rRNA catalyzes peptide bonds (ribozyme); 70S (prokaryote) vs 80S (eukaryote).
- Phases: initiation (AUG assembly), elongation (A/P/E cycling, GTP), termination (release factors).
- Code degeneracy buffers mutation; free vs bound ribosomes by protein destination.
- SRP-mediated co-translational ER targeting via signal peptides.

Unit Summary

Ribosomes translate mRNA through initiation, elongation and termination, with signal peptides routing secretory proteins to the ER co-translationally.

Exam Pointer

Very important: outline translation phases and explain antibiotic selectivity via 70S/80S differences.

Unit 9: The Cytoskeleton

Learning Objectives

Objective 1: Compare microtubules, microfilaments and intermediate filaments

Objective 2: Explain motor proteins and intracellular transport

Objective 3: Describe cell motility: cilia, flagella, amoeboid movement

The cytoskeleton is the cell's dynamic scaffolding. Microtubules (tubulin, 25 nm) are hollow tracks for vesicle transport, form the mitotic spindle, and build cilia/flagella (9+2 axoneme); they grow and shrink by dynamic instability. Microfilaments (actin, 7 nm) drive muscle contraction, cytokinesis (contractile ring) and amoeboid crawling. Intermediate filaments (keratins, lamins; 10 nm) are stable ropes resisting tension, anchoring organelles and the nucleus.

Motor proteins walk the tracks: kinesins mostly carry cargo toward microtubule plus ends, dyneins toward minus ends (and drive ciliary beating), myosins walk actin (muscle contraction via sliding filaments). ATP powers each step. Axonal transport in neurons, moving vesicles meters along microtubules, shows the system's importance.

Cilia and flagella beat via dynein-driven microtubule sliding in the 9+2 arrangement; defects cause primary ciliary dyskinesia. Amoeboid movement extends actin-rich pseudopods. Very important: the structure-function pairing (which filament, which motor, which movement) is the exam's focus; tabulate it.

Key Definitions

Dynamic instability is defined as microtubules' rapid growth and shrinkage enabling reorganization.

Motor protein is defined as an ATP-driven protein walking cytoskeletal tracks carrying cargo.

Axoneme is defined as the 9+2 microtubule arrangement of cilia and flagella.

Key Points to Remember

- Microtubules (tubulin): transport tracks, spindle, cilia; dynamic instability.
- Microfilaments (actin): contraction, cytokinesis, crawling.
- Intermediate filaments: stable tension-resisters (keratins, lamins).
- Motors: kinesin (+end), dynein (-end, cilia), myosin (actin); cilia = 9+2 dynein sliding.

Unit Summary

Three filament systems with dedicated motors provide structure, transport and motility, from vesicle trafficking to ciliary beating.

Exam Pointer

Exam pointer: tabulate filament, protein, diameter, motor and function; explain the 9+2 ciliary mechanism.

Unit 10: The Cell Cycle and Mitosis

Learning Objectives

Objective 1: Outline cell-cycle phases and checkpoints

Objective 2: Describe mitotic stages in detail

Objective 3: Explain cell-cycle regulation: cyclins, CDKs, checkpoints

The cell cycle comprises interphase (G1 growth, S DNA replication, G2 preparation) and the M phase (mitosis + cytokinesis). Checkpoints guard transitions: G1/S (DNA damage? sufficient growth?), G2/M (replication complete?), and the spindle assembly checkpoint (chromosomes attached?). p53, the 'guardian of the genome', arrests or kills damaged cells at G1/S; its mutation underlies many cancers.

Mitosis distributes duplicated chromosomes: prophase (chromatin condenses, spindle forms), prometaphase (envelope breaks, microtubules capture kinetochores), metaphase (chromosomes align at the plate), anaphase (sister chromatids separate, pulled poleward), telophase (nuclei reform), then cytokinesis (contractile ring in animals, cell plate in plants). Each stage is visually distinctive, a favorite of diagram questions.

Regulation uses cyclin-CDK complexes: cyclin levels oscillate, activating cyclin-dependent kinases that phosphorylate targets driving the cycle forward. Growth factors signal through these; their dysregulation causes uncontrolled proliferation. Very important: connect checkpoints to cancer explicitly; examiners reward the regulation-to-disease link.

Key Definitions

Checkpoint is defined as a control point halting the cycle until conditions are satisfied.

Cyclin-dependent kinase (CDK) is defined as an enzyme driving cell-cycle transitions when bound to its cyclin.

Kinetochores are defined as the chromosomal attachment sites for spindle microtubules.

Key Points to Remember

- Interphase (G1, S, G2) + M; checkpoints at G1/S, G2/M, spindle assembly.
- Mitosis: prophase → prometaphase → metaphase → anaphase → telophase → cytokinesis.
- Cyclin-CDK complexes drive transitions; p53 guards G1/S.
- Checkpoint failure → genomic instability → cancer.

Unit Summary

The cell cycle's ordered phases are guarded by checkpoints and driven by cyclin-CDKs, with mitosis ensuring equal chromosome segregation.

Exam Pointer

Very important: draw and label mitotic stages; explain two checkpoints and their cancer connection.

Unit 11: Meiosis and Sexual Reproduction

Learning Objectives

Objective 1: Contrast meiosis with mitosis

Objective 2: Explain crossing over and independent assortment

Objective 3: Relate meiosis to genetic variation

Meiosis produces haploid gametes through two divisions. Meiosis I is reductional: homologous chromosomes pair (synapsis), cross over, and separate, halving the number. Meiosis II resembles mitosis, separating sister chromatids. The result: four genetically distinct haploid cells from one diploid parent.

Prophase I's stages (leptotene, zygotene, pachytene, diplotene, diakinesis) host synapsis and crossing over, exchanging segments between homologs and creating recombinant chromatids. Independent assortment at metaphase I (maternal/paternal homolog orientation is random) multiplies combinations: 2^n possibilities for n chromosome pairs. Together with crossing over and random fertilization, meiosis generates the variation natural selection needs.

Errors cause aneuploidy: nondisjunction (homologs or chromatids failing to separate) yields trisomies like Down syndrome (trisomy 21), risk rising with maternal age. Very important: the three variation sources (crossing over, independent assortment, fertilization) are a standard list question; nondisjunction mechanics suit diagram questions.

Key Definitions

Meiosis is defined as two divisions producing four haploid genetically distinct cells.

Crossing over is defined as exchange of segments between homologous chromatids in prophase I.

Nondisjunction is defined as failure of homologs or chromatids to separate, causing aneuploidy.

Key Points to Remember

- Meiosis I reductional (homologs separate), meiosis II equational (chromatids separate).
- Variation: crossing over, independent assortment (2^n), random fertilization.
- Prophase I sub-stages host synapsis and crossing over.
- Nondisjunction -> aneuploidy (e.g. trisomy 21); maternal-age risk.

Unit Summary

Meiosis halves chromosome number while generating variation through crossing over and independent assortment; errors produce aneuploidy.

Exam Pointer

Very important: list the three variation sources and diagram nondisjunction consequences.

Unit 12: Cell Signaling

Learning Objectives

Objective 1: Outline signaling stages: reception, transduction, response

Objective 2: Compare receptor types

Objective 3: Explain second messengers and signaling cascades

Cell signaling follows reception, transduction, response. A ligand binds a receptor (reception); the signal is relayed and amplified intracellularly (transduction); the cell responds (gene expression, enzyme activation, cytoskeletal change). Signaling spans distances: autocrine (self), paracrine (neighbors), endocrine (hormones via blood), and synaptic (neurons).

Receptors come in three main types. G protein-coupled receptors (largest family; adrenaline, odorants) activate G proteins that modulate enzymes. Receptor tyrosine kinases (growth factors like EGF) dimerize and autophosphorylate, launching kinase cascades. Ligand-gated ion channels (neurotransmitters) open pores directly, fastest. Intracellular receptors (steroid hormones) act as transcription factors.

Second messengers (cAMP, Ca²⁺, IP₃, DAG) diffuse the signal; kinase cascades (like MAPK) amplify it enormously, one receptor activating thousands of targets. Termination matters equally: phosphodiesterases degrade cAMP, phosphatases reverse phosphorylation. Very important: signal amplification explains how tiny hormone amounts cause massive responses; exams ask for the amplification logic.

Key Definitions

Second messenger is defined as a small intracellular molecule relaying and spreading a signal (cAMP, Ca²⁺).

G protein-coupled receptor is defined as a seven-pass membrane receptor signaling via G proteins.

Signal cascade is defined as a relay of activating kinases amplifying the original signal.

Key Points to Remember

- Stages: reception → transduction → response; distances: auto-, para-, endocrine, synaptic.
- Receptors: GPCR, receptor tyrosine kinase, ligand-gated channel, intracellular.
- Second messengers + kinase cascades amplify; termination by degradation/dephosphorylation.
- Amplification explains massive responses to tiny signals.

Unit Summary

Cells receive signals through diverse receptors, amplify them via second messengers and cascades, and terminate them precisely.

Exam Pointer

Exam pointer: compare the three receptor types with an example each; explain amplification quantitatively.

Unit 13: Cell Death, Cancer and Stem Cells

Learning Objectives

- Objective 1:** Distinguish apoptosis from necrosis
- Objective 2:** Explain the cellular basis of cancer
- Objective 3:** Describe stem cells and their potential

Apoptosis is programmed cell death: orderly, ATP-dependent, with cell shrinkage, chromatin condensation, blebbing and phagocytosis of apoptotic bodies without inflammation. Caspases execute it, triggered extrinsically (death receptors) or intrinsically (mitochondrial cytochrome c release when Bcl-2 family balance tips). Necrosis, by contrast, is messy traumatic death with inflammation. Apoptosis sculpts development (digit separation) and deletes damaged cells.

Cancer is cell-cycle regulation broken: proto-oncogenes mutate into oncogenes (accelerators stuck on), tumor suppressors like p53 and Rb are lost (brakes removed), enabling hallmarks: sustained proliferation, evading apoptosis, angiogenesis, invasion, metastasis. Carcinogenesis is multistep, requiring several mutations, which is why cancer is largely age-related. Therapies target these: chemotherapy hits dividing cells, targeted drugs block specific oncoproteins, immunotherapy unleashes immune attack.

Stem cells (totipotent zygote, pluripotent embryonic/inner cell mass, multipotent adult) self-renew and differentiate, maintaining tissues. iPS cells reprogram adult cells to pluripotency (Yamanaka factors).

Very important: connect apoptosis failure to cancer (evading death is a hallmark) and stem-cell logic to both renewal and cancer stem cells. 

Key Definitions

- Apoptosis** is defined as programmed, orderly cell death via caspases without inflammation.
- Oncogene** is defined as a mutated proto-oncogene driving uncontrolled proliferation.
- Pluripotent** is defined as able to form all body cell types (embryonic stem cells, iPS).

Key Points to Remember

- Apoptosis: caspase-executed, tidy, anti-inflammatory; vs necrosis: traumatic, inflammatory.
- Cancer: oncogenes on + tumor suppressors off; multistep; hallmarks include evading apoptosis.
- Therapies: chemo (dividing cells), targeted (oncoproteins), immuno (unleash immunity).
- Stem hierarchy: toti- > pluri- > multipotent; iPS reprogram adult cells.

Unit Summary

Apoptosis orderly removes cells; its failure enables cancer's hallmarks; stem cells renew tissues and model disease, with iPS technology reprogramming fate.

Exam Pointer

Very important: contrast apoptosis and necrosis in a table; explain the oncogene/tumor-suppressor logic of cancer.