

Cell Bio Exam 1 Review Study Guide

- Protein Basics:
 - Composition:
 - Alpha-carbon (central)
 - Amino group
 - Carboxyl group
 - R group/side chain
 - Joined by peptide bonds in dehydration-synthesis reactions to form a polymer
 - Assembly starts at the amino-terminal end of the amino acid chain
- Cellular Structures and Functions (Basics):
 - Lysosomes: organelles dealing with cellular waste management, digestion, and disposal
 - Peroxisomes: organelles breaking down toxic hydrogen peroxide into H₂O
 - Vesicles: membrane-bound sacs transporting food particles, waste, etc. throughout the cell
 - Nuclear membrane: organelle regulating entry/exit to the nucleus and protecting genetic material inside
 - Nuclear pores: organelles allowing materials to enter or exit the nucleus
 - Phospholipid bilayer: cellular membrane protecting the inside of the cell and regulating entry and exit of materials, waste, etc.
 - Golgi apparatus: post-translational protein modifier and packager
 - Ribosomes: RNA-protein complexes synthesizing intracellular and extracellular proteins
 - Rough ER: endoplasmic reticulum with ribosomes attached to it synthesizing proteins; makes post-translational modifications to the proteins
 - Smooth ER: endoplasmic reticulum without ribosomes attached dealing with cellular detoxification
- Actin: protein important to cellular movement
 - Important in:
 - Cell motility
 - Cell structure
 - Cell division
 - Muscular contraction
 - Can be monomeric or filamentous
 - Monomeric actin is known as *G-actin* or *globular subunits*
 - Filamentous actin is known as *F-actin* or microfilaments
 - ❖ Important for cellular motility
 - G-actin is added to the plus end (end of microtubule where actin polymerizes) of F-actin
 - Polymerization of actin filaments occurs at the leading edge of a cell
 - Globular subunits tend to depolymerize at the minus end of a microfilament
 - ✓ Treadmilling: phenomenon occurring where the rate of actin polymerization at the plus end is equal to the rate of actin depolymerization at the minus end
 - Polymerization requires the binding of ATP (replaces bound ADP)
 - Actin structures:

- Lamellipodia: flattened, sheet-like extensions of cell membrane formed by actin polymerization
 - Filopodia: spiked extensions of cell membrane formed by actin polymerization
 - Actin-Associated Structures:
 - Cortex: special region of actin filament-rich cytoplasm just beneath the cell membrane
 - Serves to support the cell membrane
- Cellular Movement:
 - Overall transduction pathway: signal → receptor → GTP proteins → ARP complex → actin polymerization
 - GTP proteins: proteins binding GTP acting as molecular switches for signal transduction pathways (*Rho protein family* most common)
 - ❖ Cdc42 regulates actin polymerization in filopodia
 - ❖ Rac regulates actin polymerization in Lamellipodia
 - Enhances nucleating activities of the ARP complex and promotes uncapping of plus ends of microfilaments
 - Powered by GTP hydrolysis
 - ❖ Loss of phosphate to become GDP turns off signaling pathways
 - ARP complex: actin-related proteins that promote branching points for actin polymerization
 - ❖ Activated via signal transduction pathways
 - Surface contacts:
 - Extracellular matrix: non-cellular proteins and other materials on the outside of the cell
 - ❖ Linked to the cytoskeleton via integrin proteins (esp. *vinculin*)
 - Focal contacts: adhesions by which a cell attaches to the underlying surface
- Muscle Contraction:
 - Sarcomere: basic unit of muscular structure
 - Bind in fibers to form *myofibrils*
 - Structure:
 - ❖ Thin filaments: filamentous actin chains stabilizing muscular contraction
 - ❖ Thick filaments: myosin filaments that contract and pull thin filaments during muscular contraction
- Cytokine Signaling: Integrated Example with CD8
 - T cells target infections/infected cells
 - Neutrophils: cells initiating and maintaining the immune response
 - ❖ First immune cells to migrate out of blood vessels and target infected tissues
 - ❖ Recruit T cells for bacterial and viral infections
 - ❖ Leave a molecular trail by depositing chemokine molecules for T cells to follow
 - Initiates a signal transduction pathway in T cells promoting actin polymerization and cellular movement
 - Chemokine molecule in question is CD8

- Angiogenesis: formation/sprouting of new blood vessels from existing ones
 - Growth stimulated by growth chemicals such as VEGF
 - Initiates a signal transduction pathway promoting actin polymerization and the growth of filopodia toward the source of the signal
 - ❖ VEGF Binds to an extracellular receptor
 - ❖ G-proteins are phosphorylated and activate ARP complexes
 - ❖ ARP complexes promote actin polymerization
 - ❖ Vesicles in the forming stem fuse to form a lumen
- Microtubules: strands of polymerized tubulin protein
 - Functions:
 - Gives the cell its polarity (shape/orientation in a given direction)
 - Cellular support and motility (helps form the cytoskeleton)
 - Separate chromatids during mitosis/meiosis
 - Helps form the mitotic spindle
 - Form the cilia and flagella (utilize a *9+2 structure*)
 - Transport of organelles and other materials along a microtubule track
 - Organize organelles
 - Structure:
 - Basic piece is an alpha-beta heterodimer tubulin protein
 - Heterodimers associate among themselves to form a protofilament
 - ❖ 13 protofilaments in a ring form a microtubule
 - Formation:
 - Beta subunit binds GTP and provides energy for polymerization (depolymerization caused by GTP hydrolysis)
 - ❖ Dynamic instability: rapid polymerization and depolymerization of tubulin subunits
 - Occurs at the microtubule organizing center (MTOC)
 - ❖ Centrosome along with a gamma-tubulin complex nucleates microtubules at the plus ends of the strands
 - Plus ends can be stabilized by the addition of capping proteins
 - ❖ Centrioles are not required for nucleation
 - Does assemble the *kinetochore* (protein complex found on chromosomes → utilized during cell division?)
- Motor Proteins: proteins transporting organelles and other materials along microtubules
 - Types:
 - Kinesins: motor proteins travelling toward the plus ends of microtubules
 - Dyneins: motor proteins travelling toward the minus end of microtubules
 - Move along the microtubule via “walking”
 - Driven by the binding of ATP molecules
- Intermediate Filaments: protein filaments giving vertebrate cells great tensile strength
 - Made up of 8 associated protein complexes
 - Dimers associate into tetramers in opposite directions to make a filament
 - Where they are located:
 - Desmosomes: cell-cell junctions

- Hemidesmosomes: linkages between the cell to the extracellular matrix
 - Help prevent cellular lysis (rupture)
- Phagocytosis: “cellular eating”
 - Process:
 - Chemical signal received by the cell
 - Molecular switch activated by GTP protein activation
 - ARP complex activated
 - Actin polymerization powers creation of filopodia
 - Filopodia come together to form a phagosome (vesicle enclosing material formed by the closing of filopodia)
 - Phagosome carried by the endocytic pathway to endosomes
 - Material reached lysosomes and is digested
 - Lysosomes:
 - Contain many different kinds of digestive enzymes (sulfatases, proteases, etc.) that work at a low pH
 - Hydrolyze ATP into ADP to power a proton pump that creates the low intra-lysosomal pH
 - Produce H_2O_2 as a byproduct
 - Peroxisomes:
 - Break down fatty acids and hydrogen peroxide
 - Also involved in the synthesis of cholesterol and myelin lipids
- DNA → RNA → Protein Basics:
 - DNA consists of regulatory, coding, and noncoding regions
 - RNA polymerase binds to a promotor sequence to begin the transcription of a template strand
 - ❖ Made 5' to 3'
 - ❖ Creates a complementary and antiparallel sequence of mRNA (uracil instead of thymine)
 - ❖ 3-letter codons (methionine start codon)
 - Ribosomes use tRNA to “read” and translate mRNA into protein
 - tRNA anticodons bind to mRNA
- Mutation Basics:
 - Tyoes:
 - Silent: no change in the amino acid sequence
 - Missense: mutation causing a single amino acid change
 - Nonsense: mutation causing the insertion of a premature stop codon
 - Frameshift: mutation causing the reading frame of the mRNA to be shifted and the entire amino acid sequence following the mutation to be altered
- Ribosomes:
 - Consist of a large and small subunit:
 - mRNA binds to the small subunit
 - Three major sites in the large subunit:
 - ❖ A site: aminoacyl-tRNA binding site
 - ❖ P site: peptidyl-tRNA binding site where the polypeptide chain is made

❖ E site: tRNA exit site

- tRNA:
 - Structure:
 - Amino acid attached to the 3' end by an ester bond
 - Anticodon located on the *anticodon loop*
 - Charged with the proper amino acid by aminoacyl-tRNA synthases (enzymes)
- In-Depth Translation:
 - Small subunit binds to translation initiation factors
 - mRNA binds to the small subunit and *initiator tRNA* moves along the mRNA looking for the first AUG start codon
 - Initiation factors dissociate and the large subunit binds after the first AUG is found
 - Aminoacyl tRNA binds to the A as initiator tRNA moves to the P site
 - Peptide bonds form at the P site
 - Elongation factor tu brings in the aminoacyl tRNA to the A site
 - Large subunit displaces as peptide bonds are formed
 - Moves tRNA at A site to P site, and P site to E site
 - Elongation factor G restores the normal ribosomal conformation and allows another aminoacyl tRNA to bind at the A site
 - Often many ribosomes translate the same mRNA molecule at one time (polyribosomes)
- DNA Packaging:
 - Packaged in the nucleus with histone proteins as chromatin
 - Nucleosome: combined DNA-protein loop
 - 6 nucleosomes are packed on top of each other to form chromatin → condenses into chromatids and chromosomes
- DNA Replication:
 - Occurs at multiple replication forks in eukaryotes
 - Creates two DNA strands identical to that of the parents
 - Nucleotides added 5' → 3' at the 3' hydroxyl end
 - Process:
 - Helicase enzyme separates the single DNA strands
 - DNA polymerase III binds to each strand along with a sliding clamp to relieve pressure
 - RNA primase and single-stranded binding proteins bind to the lagging strand to make *Okazaki fragments* on the lagging strand
 - DNA polymerase I replaces the RNA primers
 - Ligase joins the parental and daughter strands together
 - End-replication problem: gap of the end of the lagging strand cannot be copied (no primer)
 - Solved by the use of telomerase enzyme
 - Hayflick limit: differentiated cells in culture divide about 50 times before dying
 - Associate with shortening of telomeres
 - Cellular life:
 - ❖ Rapid cell division
 - ❖ Mitosis slows
 - ❖ Senescence (ceasing of cell division)

- ❖ Apoptosis (programmed cellular death)
 - Cancerous cells fail to undergo apoptosis