

MU-Varna Cytology • CYTOPLASM

Exam-perfect summary aligned to MU-Varna "EXCEPT" logic. This version is audited against your master checklist: definitions, dimensions, protein-level anchors, trap rules, and clinical relevance.

How to avoid losing points

Use this on every MCQ

Mechanism-first answering

For each option ask: **Where is it? What does it do? Which proteins enable it? What fails clinically?**

EXCEPT pattern (most common MU-Varna)

The wrong option is usually a **true term placed into the wrong system** (e.g., clathrin → protein synthesis, caveolae → ATP synthesis, cristae → outer membrane).

I. Plasma Membrane (Plasmalemma)

Dimensions, structure, glycocalyx trap, stability proteins

Dimensions & visibility

- **Thickness:** .
- **Light microscopy:** not visible.
- **Electron microscopy:** appears as a **trilaminar** structure (dark–light–dark).

Core structure

- **Asymmetric phospholipid bilayer.**
- **Lipids:** phospholipids (amphipathic) + **cholesterol** (modulates fluidity, restricts movement).
- **Proteins (~50% by weight):** integral (often multipass) + peripheral.

Key distinction (tested)

Peripheral proteins are more loosely attached and are often described as **extractable with salt solutions**, compared with integral proteins.

The glycocalyx (surface “fuzzy coat”)

- External carbohydrate-rich coat of **glycoproteins, glycolipids**, and **proteoglycans**.
- Functions: recognition, adhesion, receptor interactions, antigenic properties.

● MU-VARNA EXAM ALERT

- **Lipoproteins** are a classic distractor: treat “lipoproteins are part of glycocalyx” as **false** in exam logic.

Stability proteins

- **Spectrin:** critical for **RBC membrane stability**.
- **Dystrophin:** links **actin filaments** to muscle cell membrane (sarcolemma).

Clinical relevance

Defects in membrane-stability proteins manifest as mechanical fragility of cells (classic examples: RBC shape/fragility, muscle membrane instability).

II. Vesicular Transport: logic & exceptions

Endocytosis types, clathrin/caveolae rules

Endocytosis types

- **Phagocytosis:** “cell eating” of large particles (bacteria, debris) by macrophages/neutrophils.
- **Pinocytosis:** “cell drinking” of extracellular fluid; small vesicles.
- **Receptor-mediated endocytosis:** selective uptake via **clathrin-coated pits** (classic ligand: **LDL**).
- **Caveolae:** caveolin-based invaginations; important in **signal transduction** and **transcytosis** (bulk transfer across a cell).

● MU-VARNA RULES (must be automatic)

- **Clathrin** is for **transport**, **NEVER** for protein synthesis.
- **Caveolae** are for signaling/transport, **NEVER** for ATP synthesis.

Mechanism cue

If the stem says “coated pits/vesicles” think **receptor-mediated endocytosis**. If it says “signal transduction + transcytosis in thin cells” think **caveolae**.

III. Organelle function–coupling

RER vs SER vs Golgi, sorting direction, M6P

Rough ER (RER)

- Prominent in **protein-secreting cells**.
- **Basophilic** due to high RNA (ribosomes/polyribosomes).
- **Nissl bodies** in neurons = RER-rich regions.
- Synthesizes **secretory proteins, lysosomal enzymes, and membrane proteins**.

● Trap

- **Free ribosomes** synthesize cytosolic, nuclear, and mitochondrial/peroxisomal proteins, **not secretory** proteins.

Smooth ER (SER)

- No ribosomes.
- Functions: **lipid/steroid synthesis, detoxification** (Cytochrome P450), and **Ca²⁺ storage** (sarcoplasmic reticulum).

Clinical

Underdeveloped SER in neonates contributes to **neonatal jaundice** (reduced detox capacity).

Golgi apparatus

- Post-translational modification and sorting.
- **Logic: Cis** face receives vesicles from RER → **Trans** face ships secretory vesicles/lysosomes.

Lysosomal tag

- **Mannose-6-phosphate** targets enzymes to lysosomes.
- Defect in tagging/sorting → **I-cell disease**.

IV. Lysosomes vs proteasomes

Do not mix membrane digestion with ubiquitin quality control

Feature	Lysosomes	Proteasomes
Membrane	Membrane-bound	Non-membranous
Mechanism	Acid hydrolases ()	Ubiquitin-dependent degradation
Target	Extracellular material, endocytosed particles, old organelles (autophagy)	Misfolded/short-lived cytosolic (and nuclear) proteins

● Trap

- Proteasomes are not "digestive vesicles". Lysosomes are not "ubiquitin barrels".

V. Cytoskeleton (MU-Varna high-yield)

Diameters, subunits, junction links, constants

Component	Diameter	Subunit	Key associations
Microtubules		α & β tubulin	Cilia, mitotic spindle, axonemes; intracellular transport
Microfilaments (Actin)		G-actin \rightarrow F-actin	Adherens junctions, focal adhesions, microvilli, cytokinesis
Intermediate filaments		Varies (e.g., keratins, vimentin; lamins in nucleus)	Desmosomes, hemidesmosomes; mechanical stability

Microtubule constants

13 parallel protofilaments form the microtubule wall (classic numeric tested fact).

Clinical

Dynein defect \rightarrow Kartagener syndrome (immotile cilia phenotype).

Motor proteins (direction logic)

Kinesin = anterograde/forward transport. **Dynein** = retrograde/backward transport.

VI. Mitochondria & peroxisomes

Membrane logic, cristae trap, catalase, Zellweger

Mitochondria

- Double-membrane organelles.
- **Inner membrane** contains **cristae** (folds) and ATP synthase stalk-like complexes.

● Trap

- **Cristae are only on the inner membrane**, never the outer.

Peroxisomes

- Contain **catalase** to break down .
- Perform **β -oxidation** of long-chain fatty acids.

Clinical

Zellweger syndrome: peroxisomal biogenesis/enzymatic deficiency pattern.

VII. Inclusions (temporary deposits)

PAS+, pigments, "never inclusions" rule, hemosiderosis

Types

- **Glycogen (PAS+)**
- **Lipid droplets**
- **Pigments:** melanin, lipofuscin, **hemosiderin**

● EXAM ALERT

- **Centrioles and ribosomes** are organelles/structures, **never classified as inclusions**.

Clinical

Hemosiderosis = iron accumulation (hemosiderin), often after excessive RBC lysis or iron overload states.

MU-Varna scoring tip (cytoskeleton association)

Fast elimination in "NOT associated" questions

High-yield rule

If the question asks for a structure **NOT associated with the cytoskeleton**, the best answer is often **Gap junctions**: they mediate communication but do not have the same cytoskeletal anchoring logic as adherens junctions, desmosomes, or focal adhesions.

Junction	Association
Adherens junction	Actin filaments
Focal adhesion	Actin filaments
Desmosome	Intermediate filaments
Hemidesmosome	Intermediate filaments
Gap junction	No primary cytoskeletal anchoring in this exam mapping