Cell Biology

Unit 3 · Lesson 4 · Cytoskeleton and motors

The notes for Lesson 4 of Unit 3: chapters 3.8 to 3.9 of the unit book, with their figures, worked examples, exam traps and quizzes, in one file. The full unit book, with every chapter, the exam question bank and the glossary, is linked at the end.

Lesson 4 of 4 | 2 chapters | Chapters 3.8 to 3.9

About these notes

These are the notes for **Lesson 4 · Cytoskeleton and motors** of the University free n easy video lessons on Cell Biology Unit 3, Cell Organelles and Cytoskeleton. They are chapters 3.8 to 3.9 of the unit book, unchanged, so a page here is the same page in the full book.

3.8 Cytoskeleton: microtubules, actin and intermediate filaments

The cytoskeleton is a network of protein filaments that runs through the cytoplasm of every eukaryotic cell. It gives the cell its shape, resists stretching, places the organelles, lays tracks for transport, pulls chromosomes apart in mitosis and makes the cell crawl. There are three kinds of filament, each built from many copies of one small protein subunit: **microtubules** (tubulin), **actin filaments** or microfilaments (actin), and **intermediate filaments** (a family of fibrous proteins). Unlike a building's skeleton, two of the three are rebuilt all the time, in seconds to minutes.

You need: protein structure (α-helix, subunits) from your biochemistry course. Motor proteins that move along these filaments are in Chapter 3.9.

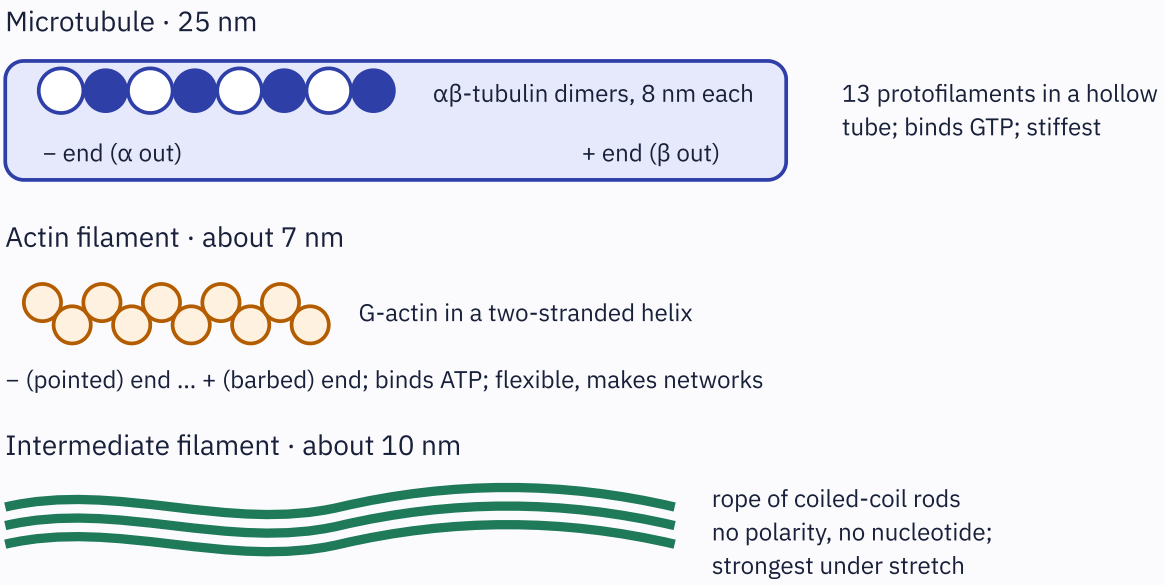


Figure 3.8.1 The three filaments drawn side by side (not to scale along their length). Microtubules and actin filaments have a plus and a minus end because their subunits all point the same way; intermediate filaments do not. Notice that only the first two bind a nucleotide, which is what lets them grow and shrink quickly.

Microtubules

A microtubule is a hollow tube about 25 nm across, made of **αβ-tubulin dimers** lined up head-to-tail into **protofilaments**; usually 13 protofilaments form the wall. Since every dimer points the same way, the tube has a **plus end** (β-tubulin exposed, grows fast) and a **minus end** (α-tubulin exposed). Each tubulin binds GTP. The GTP on β-tubulin is hydrolysed to GDP soon after the dimer joins the tube.

In animal cells most microtubules grow out from the **centrosome**, the main **microtubule-organising centre** (MTOC), where **γ-tubulin ring complexes** start new tubes and hold the minus ends. Plus ends point out towards the cell edge. Microtubules show **dynamic instability** (Mitchison and Kirschner, 1984):

1. A growing plus end keeps a **GTP cap** of newly added dimers, which bind each other tightly.
2. If hydrolysis catches up with addition and the cap is lost, the GDP-tubulin protofilaments curl outward and the tube shrinks rapidly. This switch is a **catastrophe**.
3. If GTP-tubulin adds again before the tube is gone, it starts growing once more: a **rescue**.

This lets microtubules search the cell and be captured where they are needed, for example by chromosomes in mitosis. **Microtubule-associated proteins** (MAPs) tune this: tau and MAP2 stabilise microtubules in neurons, while proteins such as stathmin and kinesin-13 take them apart. Microtubules form the mitotic spindle (Unit 4), tracks for organelle transport, and the core of cilia and flagella (Chapter 3.9). **Drugs:** colchicine and nocodazole stop assembly; vinblastine

and vincristine also block assembly and are used in cancer treatment; **paclitaxel** (taxol) does the opposite, freezing microtubules so they cannot shrink, which also stops mitosis.

Actin filaments

Actin is one of the most abundant proteins in many animal cells. A globular monomer (**G-actin**) binds ATP; monomers join into a two-stranded helical filament (**F-actin**) about 7 nm thick. Its fast-growing plus end is called the **barbed end** and its minus end the **pointed end**, after the arrowhead pattern myosin heads make when they bind it.

Assembly starts slowly because a nucleus of three monomers is unstable (the **lag phase**), then grows until free monomer falls to the **critical concentration** C_c: the free-monomer concentration at which adding and losing subunits balance. Above C_c an end grows; below it, the end shrinks. With ATP-actin, C_c is lower at the plus end than at the minus end. At monomer levels between the two, the filament adds at the plus end and loses at the minus end at the same rate, so its length stays the same while subunits flow through it: **treadmilling**.

Cells control actin with helper proteins:

- **Nucleators:** the **Arp2/3 complex** starts branches on existing filaments, building the meshwork of the **lamellipodium**; **formins** make long unbranched filaments for **filopodia**, stress fibres and the contractile ring of cell division.
- **Monomer binders:** profilin helps monomers add at plus ends; thymosin β4 holds a reserve of monomers.
- **Cappers and severers:** capping protein stops plus-end growth; cofilin cuts old ADP-actin filaments.

Actin forms the **cell cortex** under the plasma membrane, microvilli in the gut lining, and with myosin it drives muscle contraction and cytokinesis. A crawling cell such as a neutrophil or a fibroblast moves in three steps: it **protrudes** its front by actin polymerisation, **attaches** it to the surface through integrins at focal adhesions, and **contracts** its rear with myosin II. **Drugs:** cytochalasins cap barbed ends, latrunculin binds monomers, and phalloidin (a mushroom toxin) stabilises filaments; fluorescent phalloidin is the standard stain for F-actin.

Intermediate filaments

Intermediate filaments are about 10 nm thick, between actin and microtubules, hence the name. Each subunit has a long central **α-helical rod**. Two rods wind into a **coiled-coil dimer**; two dimers pair side by side in opposite directions (**antiparallel tetramer**); about eight tetramers pack into a **unit-length filament**, which joins end to end. Because the tetramer is antiparallel, the filament has **no polarity**, binds no nucleotide and carries no motors. It bends easily but is very hard to break, so it protects cells from mechanical stress.

CLASS	PROTEIN	WHERE
I and II	acidic and basic keratins	epithelial cells, hair, nails
III	vimentin, desmin, GFAP	fibroblasts; muscle; astrocytes
IV	neurofilament proteins	nerve axons
V	lamins	nuclear lamina (Chapter 3.1)

Cells regulate them mainly by phosphorylation, as with lamins at mitosis. Mutant keratins 5 or 14 cause **epidermolysis bullosa simplex**, in which skin blisters from slight rubbing. Pathologists use the intermediate filament type to tell which tissue a tumour came from.

WORKED EXAMPLE

Question (8 marks). (a) For ATP-actin, C_c = 0.12 µM at the plus end and 0.60 µM at the minus end. Predict what each end does when the free G-actin concentration is held at 0.05 µM, 0.30 µM and 1.0 µM. (b) A microtubule is 10 µm long and has 13 protofilaments. Each αβ-dimer is 8 nm long. How many dimers does it contain?

Solution.

1. **0.05 µM:** below both C_c values, so both ends lose subunits and the filament shrinks.
2. **0.30 µM:** above 0.12 (plus end grows) but below 0.60 (minus end shrinks). The filament treadmills.
3. **1.0 µM:** above both, so both ends grow, the plus end faster.
4. Dimers per protofilament = 10 µm ÷ 8 nm = 10,000 nm ÷ 8 nm = 1,250.
5. Total = 1,250 × 13 = **16,250 dimers** (32,500 tubulin monomers).

Answer: (a) shrinks; treadmills; grows at both ends. (b) 16,250 αβ-dimers. In a steady state the free monomer settles between the two C_c values, which is why treadmilling is seen in cells.

EXAM TRAPS

- Actin binds ATP; tubulin binds GTP. Intermediate filaments bind neither.
- Plus end does not mean "the end that only grows". It is the faster end; both ends can grow or shrink depending on monomer level.
- Colchicine and taxol both stop mitosis, but in opposite ways: one prevents assembly, the other prevents disassembly.
- Lamins are intermediate filaments, even though they sit in the nucleus.
- Cytochalasin acts on actin, colchicine on microtubules. Examiners like to swap them.

CHECK YOURSELF

– REMEMBER **Give the diameter and subunit of each cytoskeletal filament.** 3 marks

Microtubule about 25 nm, αβ-tubulin; actin filament about 7 nm, G-actin; intermediate filament about 10 nm, proteins such as keratin, vimentin or lamin.

– REMEMBER **What is the main MTOC of an animal cell?** 1 mark

The centrosome.

– UNDERSTAND **Explain dynamic instability.** 4 marks

Growing microtubules carry a GTP cap at the plus end. If GTP hydrolysis catches up and the cap is lost, GDP protofilaments peel apart and the tube shrinks quickly (catastrophe). Regaining GTP-tubulin restarts growth (rescue). Individual microtubules therefore switch between growth and shrinkage.

– UNDERSTAND **Why do intermediate filaments have no polarity?** 2 marks

The two coiled-coil dimers in each tetramer point in opposite directions, so both ends of the tetramer, and of the filament, are the same.

– APPLY **A cell is treated with cytochalasin D. Which of these stop: mitosis, crawling, cytokinesis?** 3 marks

Crawling and cytokinesis stop because both need actin. The spindle (microtubules) still forms, so chromosomes separate, but the cell cannot divide and may end with two nuclei.

– APPLY **How many tubulin dimers are in a 4 µm microtubule with 13 protofilaments?** 2 marks

4,000 ÷ 8 = 500 per protofilament; × 13 = 6,500 dimers.

– ANALYZE **Explain why paclitaxel is useful against cancer but causes nerve damage as a side effect.** 4 marks

It stabilises microtubules so the spindle cannot work and dividing cancer cells arrest in mitosis. Neurons do not divide, but they depend on microtubules for long-distance transport in axons; freezing them disturbs transport, which gives peripheral neuropathy.

BEYOND THE SYLLABUS

Super-resolution microscopy (STORM, PALM, expansion microscopy) now shows single cytoskeletal filaments inside cells, and cryo-electron tomography shows their arrangement in 3-D. Tau that falls off microtubules forms the tangles of Alzheimer's disease. Microtubule drugs such as eribulin and antibody-drug conjugates carrying tubulin poisons are current cancer treatments. Search "expansion microscopy cytoskeleton" and "antibody-drug conjugate tubulin". Not for the exam.

3.9 Molecular motors and centrioles

Molecular motors are proteins that turn the chemical energy of ATP into movement along a cytoskeletal track. Three families do almost all the work: **kinesins** and **dyneins** walk on microtubules, and **myosins** walk on actin. They carry vesicles and organelles, pull chromosomes apart, beat cilia and contract muscle. **Centrioles** are small barrels of microtubules that organise the centrosome and form the base of every cilium, so they decide where many of the motors' tracks begin.

You need: microtubule and actin polarity from Chapter 3.8.

How a motor works

Every motor has a **head** (motor domain) that binds both the track and ATP, and a **tail** that binds the cargo. One ATP cycle drives one step:

1. The head binds ATP (or, for myosin, ADP + P_i after hydrolysis) and its grip on the track changes.
2. Hydrolysis and release of phosphate cause a shape change in the head, the **power stroke**, which swings a lever or linker.

3. The head lets go of ADP and the cycle resets.

Because the track is polar, each motor family moves in a fixed direction. A motor is **processive** if it takes many steps without falling off; this needs two heads working in turn, so that one is always attached.

The three families

Kinesins mostly move towards the **plus end** of microtubules, from the cell centre outward. Conventional **kinesin-1** has two heads that walk **hand over hand**, each step 8 nm (the length of one tubulin dimer) and using one ATP. It carries vesicles, mitochondria and mRNA particles down nerve axons (**anterograde transport**). Other kinesins work in mitosis: kinesin-5 pushes spindle poles apart, and kinesin-13 does not walk at all but takes microtubule ends apart. A few kinesins (kinesin-14) move towards the minus end.

Dyneins move towards the **minus end**. They are very large; the motor is a ring of six ATPase domains with a long stalk that touches the microtubule. **Cytoplasmic dynein**, working with the adaptor complex **dynactin**, carries cargo back to the cell centre (**retrograde transport** in axons), keeps the Golgi near the centrosome and helps position the spindle. **Axonemal dynein** forms the arms on the microtubules of cilia and flagella and makes them beat.

Myosins move along actin, almost all towards the **plus (barbed) end**. **Myosin II** has two heads and long tails that bundle into thick filaments; in muscle it pulls actin thin filaments to shorten the sarcomere, and in dividing cells it closes the contractile ring. In its **cross-bridge cycle**, ATP binding releases the head from actin, hydrolysis cocks it, the head rebinds and releasing P_i triggers the power stroke. Muscle structure is in **Unit 6, Muscle cells**. **Myosin V** is a processive cargo carrier with large steps of about 36 nm; **myosin VI** is unusual in walking towards the minus end.

MOTOR	TRACK	DIRECTION	TYPICAL JOB
Kinesin-1	microtubule	plus end (outward)	anterograde axonal transport
Cytoplasmic dynein	microtubule	minus end (inward)	retrograde transport, Golgi position
Axonemal dynein	microtubule doublets	minus end	beating of cilia and flagella
Myosin II	actin	plus end	muscle contraction, cytokinesis
Myosin V	actin	plus end	vesicle and pigment transport

Centrioles, centrosomes, basal bodies and cilia

A **centriole** is a short cylinder about 0.2 µm across and roughly twice as long, whose wall is made of **nine triplets** of microtubules (called A, B and C, with only A a complete tube). A cartwheel-like structure at one end sets the ninefold pattern. Two centrioles lying at right angles, surrounded by a cloud of **pericentriolar material** rich in γ-tubulin ring complexes, form the **centrosome**, the main MTOC of animal cells (Chapter 3.8). The centrioles organise the material; the material starts the microtubules.

Centrioles **duplicate once per cell cycle**, starting around the G1-to-S transition under control of the kinase PLK4. Each old centriole grows one new daughter at right angles, so each of the two centrosomes that separate in mitosis has one old and one new centriole (Unit 4). Higher plants and most fungi have no centrioles and still build spindles, which shows that centrioles are not needed for the spindle itself.

When a centriole moves to the plasma membrane and anchors a cilium, it is called a **basal body**. The cilium's core, the **axoneme**, grows from it: its triplets continue as **nine doublets**. A **motile** cilium or flagellum has **9 + 2**: nine outer doublets around a central pair of single microtubules. Axonemal dynein arms on each doublet walk on the next doublet, and **nexin links** stop the doublets sliding past each other, so the sliding turns into bending. A **primary cilium** has **9 + 0** (no central pair) and does not beat; it acts as a sensor and an antenna for signals, and most human cells carry one.

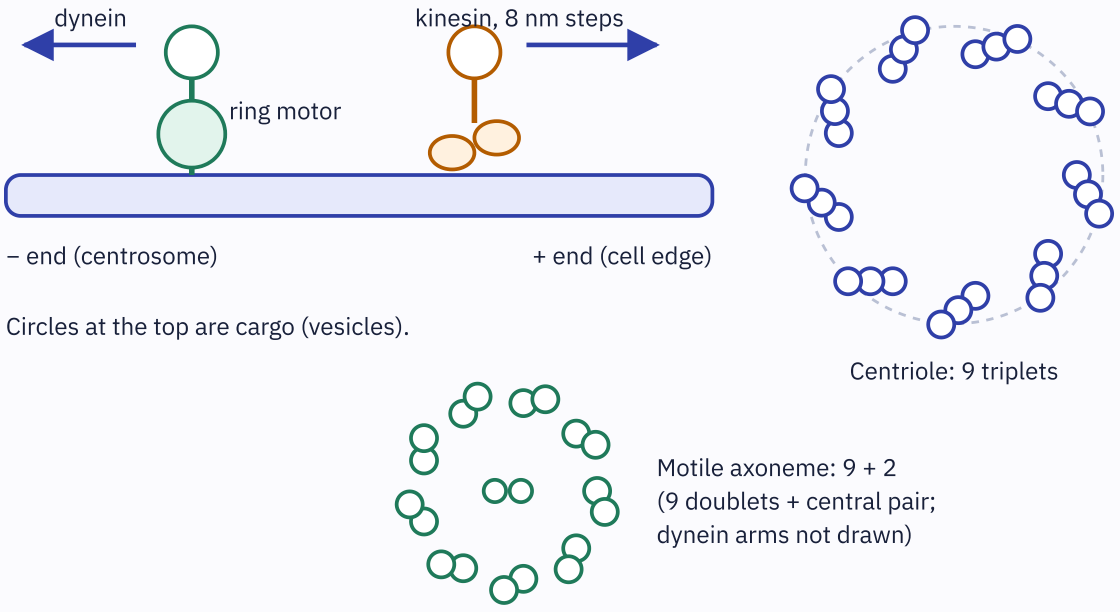


Figure 3.9.1 Top left, two motors on one microtubule carry cargo in opposite directions: kinesin towards the plus end, dynein towards the minus end. Right, a centriole in cross-section with its nine triplets. Bottom, a motile axoneme with nine doublets and a central pair; the triplets of the basal body continue as these doublets.

WORKED EXAMPLE

Question (6 marks). A motor neuron runs from the spinal cord to a toe, an axon about 1.0 m long. A kinesin-1 motor takes 8 nm steps, uses one ATP per step, and takes 100 steps per second. (a) What is its speed? (b) How long does it take to carry a vesicle the full length? (c) How many ATP molecules does one motor use for the trip? (d) Which motor brings worn-out material back, and in which direction relative to microtubule polarity?

Solution.

1. Speed = 8 nm × 100 s^{-1} = 800 nm s^{-1} = **0.8 µm s^{-1}**.
2. 1.0 m = 1.0 × 10^6 µm. Time = 1.0 × 10^6 ÷ 0.8 = 1.25 × 10^6 s. One day is 86,400 s, so the time is about **14.5 days**.
3. Steps = 1.0 m ÷ 8 × 10^{-9} m = 1.25 × 10^8, so **1.25 × 10^8 ATP**.
4. In axons, microtubule plus ends point away from the cell body. Kinesin goes out towards the plus ends; **cytoplasmic dynein** brings material back towards the minus ends in the cell body.

Answer: 0.8 µm s^{-1}; about 14.5 days; 1.25 × 10^8 ATP; dynein, minus-end directed. Diffusion of a vesicle over a metre would take far longer than a lifetime, which is why long cells depend on motors.

EXAM TRAPS

- Kinesin to plus, dynein to minus, myosin on actin. A memory aid: "dynein drives down to the centre".
- A cilium's axoneme is 9 + 2 doublets; a centriole or basal body is 9 + 0 triplets. Do not draw triplets in a cilium.
- Centrioles are not needed to make a spindle: plant cells have none.
- Myosin binds actin, not microtubules; the only common minus-end myosin is myosin VI.
- Kartagener syndrome (primary ciliary dyskinesia) comes from defective axonemal dynein: chronic chest infections, male infertility, and in about half of patients organs on the wrong side.

CHECK YOURSELF

– REMEMBER **Name the three motor families and their tracks.** 3 marks

Kinesins and dyneins on microtubules; myosins on actin filaments.

– REMEMBER **What is the microtubule pattern of a motile cilium and of a centriole?** 2 marks

Motile cilium: 9 + 2 (nine doublets, central pair). Centriole: nine triplets, no central pair.

– UNDERSTAND **Why does a processive motor need two heads?** 2 marks

One head must stay bound to the track while the other steps forward; with one head the motor would fall off after each step.

– UNDERSTAND **How does dynein sliding turn into bending of a cilium?** 3 marks

Dynein arms on one doublet walk on the next, trying to slide it. Nexin links and the basal body hold the doublets together, so they cannot slide freely and the axoneme bends instead.

– APPLY **A myosin V takes 36 nm steps at 10 steps per second. How far does it move in a minute?** 2 marks

36 nm × 10 × 60 = 21,600 nm ≈ 21.6 µm.

– APPLY **A drug stops cytoplasmic dynein. Predict the effect on the Golgi and on retrograde axonal transport.** 3 marks

The Golgi, normally held near the centrosome by dynein, scatters through the cytoplasm. Retrograde transport stops, so material from the axon tip, including signals for the cell body, is not returned.

– ANALYZE **A patient has chronic sinus and lung infections and immotile sperm. Link the symptoms to one cellular defect.** 4 marks

Motile cilia of the airway sweep mucus out, and the sperm tail is a flagellum; both use the 9 + 2 axoneme with dynein arms. A dynein-arm defect (primary ciliary dyskinesia) stops both, so mucus and bacteria stay in the airways and sperm cannot swim.

BEYOND THE SYLLABUS

Single-molecule methods such as optical tweezers measured the 8 nm kinesin step directly, and they are now used to test new motor drugs. Diseases of primary cilia, called ciliopathies, affect the kidney, eye and brain, and are an active field. Synthetic biologists are building artificial motors from DNA and engineering kinesins for nanoscale transport on chips. Search "optical tweezers kinesin" and "ciliopathies". Not for the exam.

Key terms of this lesson

TERM	MEANING OR FORMULA	CHAPTER
Critical concentration	free monomer level where an end neither grows nor shrinks; treadmilling between Cc(+) and Cc(−)	3.8
Motor directions	kinesin → plus end; dynein → minus end; myosin on actin	3.9
Centriole and axoneme	centriole 9 triplets; motile cilium 9 + 2; primary cilium 9 + 0	3.9

Glossary (terms used in this lesson)

Axoneme The microtubule core of a cilium or flagellum; 9 + 2 in motile cilia.

Basal body A centriole at the base of a cilium, from which the axoneme grows.

Centrosome Two centrioles with pericentriolar material; the main microtubule-organising centre of animal cells.

Critical concentration Free-subunit concentration at which a filament end neither grows nor shrinks.

Dynamic instability Switching of microtubule ends between growth and rapid shrinkage.

Lamina Mesh of lamin filaments under the inner nuclear membrane.

Processive motor A motor that takes many steps without letting go of its track.

Treadmilling Steady addition at the plus end and loss at the minus end, keeping filament length constant.

The full unit book

Every chapter of Unit 3, the unit summary, the exam question bank with model answers and the full glossary: Cell Biology · Unit 3 · Cell Organelles and Cytoskeleton (one file, works offline).

Sources and further reading

Standard textbooks

- Bruce Alberts and others, *Molecular Biology of the Cell* (W. W. Norton / Garland Science): chapters on intracellular compartments and protein sorting, intracellular membrane traffic, energy conversion in mitochondria and chloroplasts, and the cytoskeleton. (from memory; check)
- Harvey Lodish and others, *Molecular Cell Biology* (W. H. Freeman / Macmillan). (from memory; check)
- Geoffrey M. Cooper, *The Cell: A Molecular Approach* (Sinauer / Oxford University Press). (from memory; check)
- Gerald Karp, *Cell and Molecular Biology: Concepts and Experiments* (Wiley). (from memory; check)
- P. S. Verma and V. K. Agarwal, *Cell Biology, Genetics, Molecular Biology, Evolution and Ecology* (S. Chand), widely prescribed in Indian universities. (from memory; check)

Open resources

- OpenStax, *Biology 2e*, Chapter 4 "Cell Structure", section 4.3 "Eukaryotic Cells" and 4.4 "The Endomembrane System and Proteins" (CC BY 4.0): an open textbook covering the organelles of this unit at an introductory level. Reached through the course pages below; section titles from memory; check.
- Penn State DuBois, Biology 110 course pages built on OpenStax *Biology 2e*: psu.pb.unizin.org/biol110/part/cell-structure/
- City Tech OpenLab, Biology 1101 Course Hub, Lecture 11 "The Eukaryotic Cell", with readings and slides: openlab.citytech.cuny.edu/bio1101coursehub/?p=3597

Numbers in this book (pore size limit, ribosome protein counts, mtDNA size, filament diameters, motor step sizes) are standard textbook values written from memory; check them against your prescribed book. Worked-example inputs such as step rates and translation speeds are given in the question and are for practice.

Syllabus: the University free n easy universal syllabus, built from top universities' syllabi.

Built for the University free n easy YouTube channel from the universal syllabus of Cell Biology (Oct 2026). Written with top universities' syllabi and standard textbooks; check a formula against your prescribed book before an exam, and report mistakes on the channel.