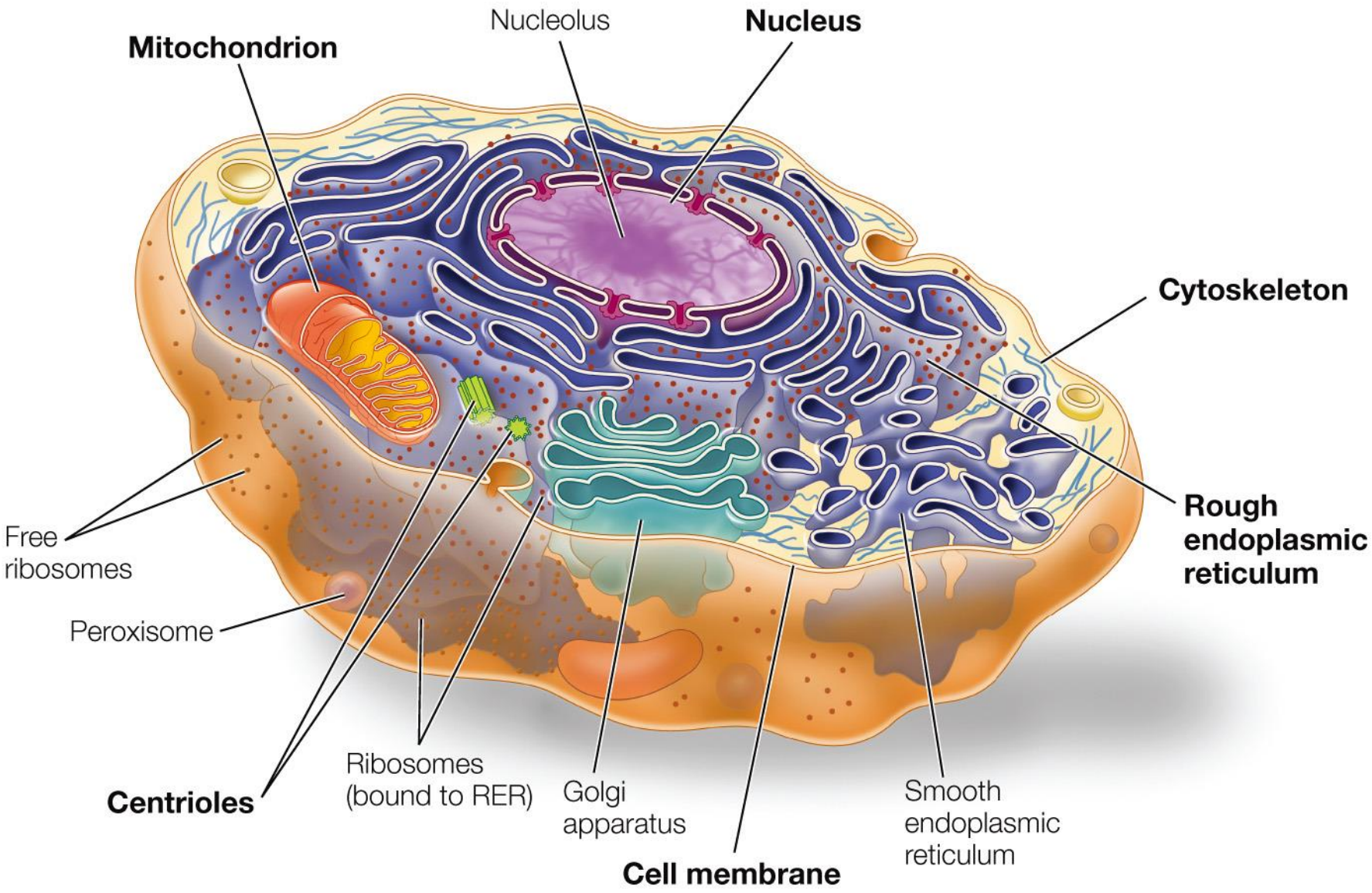
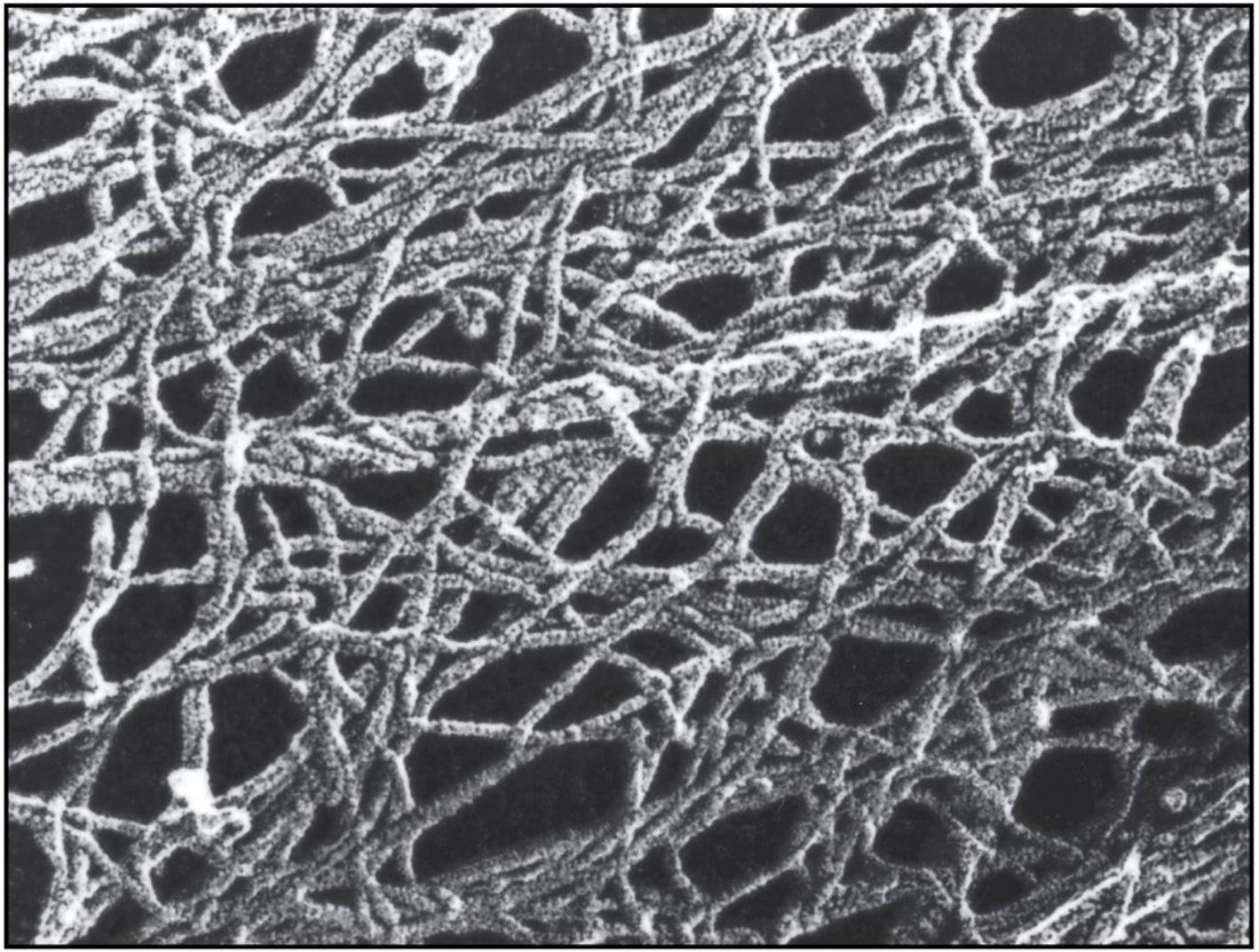
Cytoskeleton

Figure 5.7 Eukaryotic Cells (Part 1)



LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 5.7 (Part 1)

Figure 5.7 Eukaryotic Cells (Part 4)



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Cytoskeleton

25 nm

LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 5.7 (Part 4)

Cytoskeleton:

- Supports and maintains cell shape
- Holds organelles in position
- Moves organelles
- Involved in cytoplasmic streaming
- Interacts with extracellular structures to hold cell in place

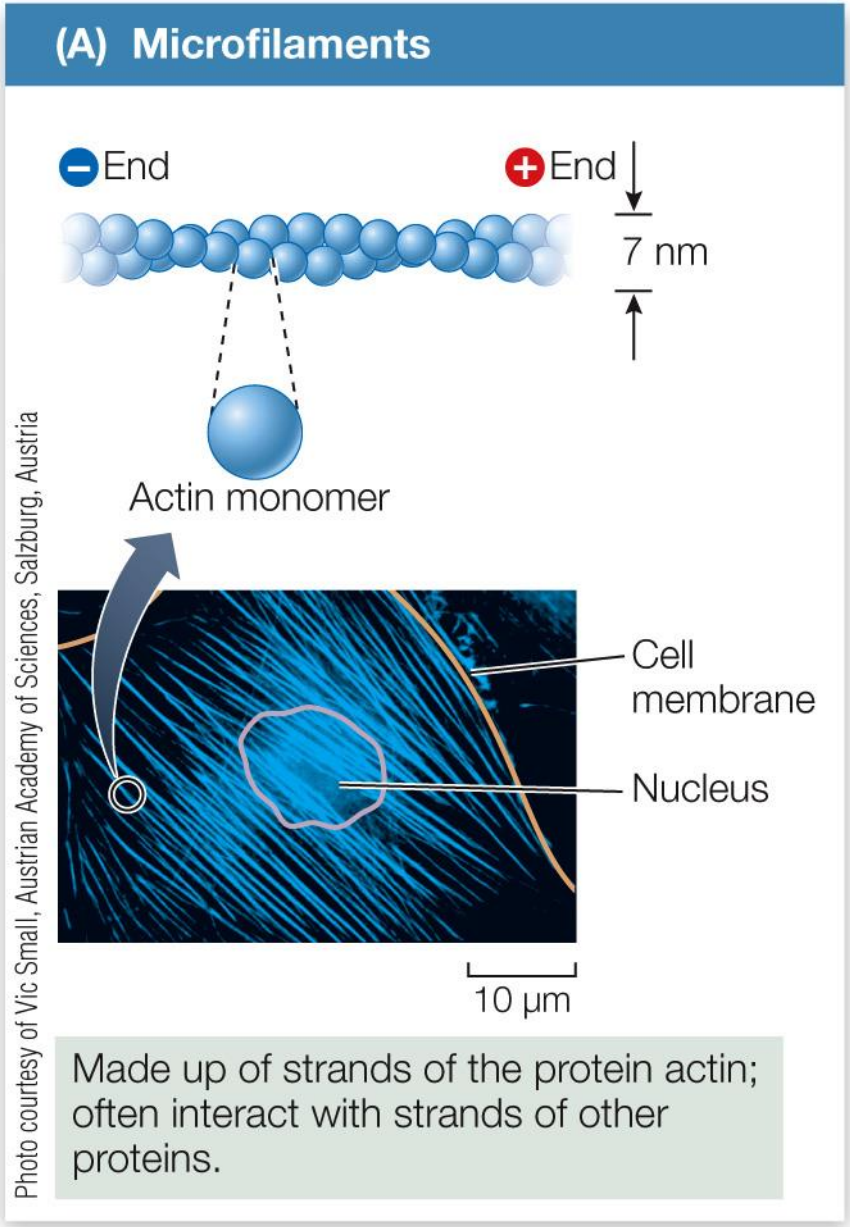
The cytoskeleton has three components:

- **Microfilaments**
- **Intermediate filaments**
- **Microtubules**

Microfilaments:

- Help a cell or parts of a cell to move
- Determine cell shape
- Made from the protein **actin**
- Actin polymerizes to form long helical chains (reversible)

Figure 5.14 The Cytoskeleton (Part 1)

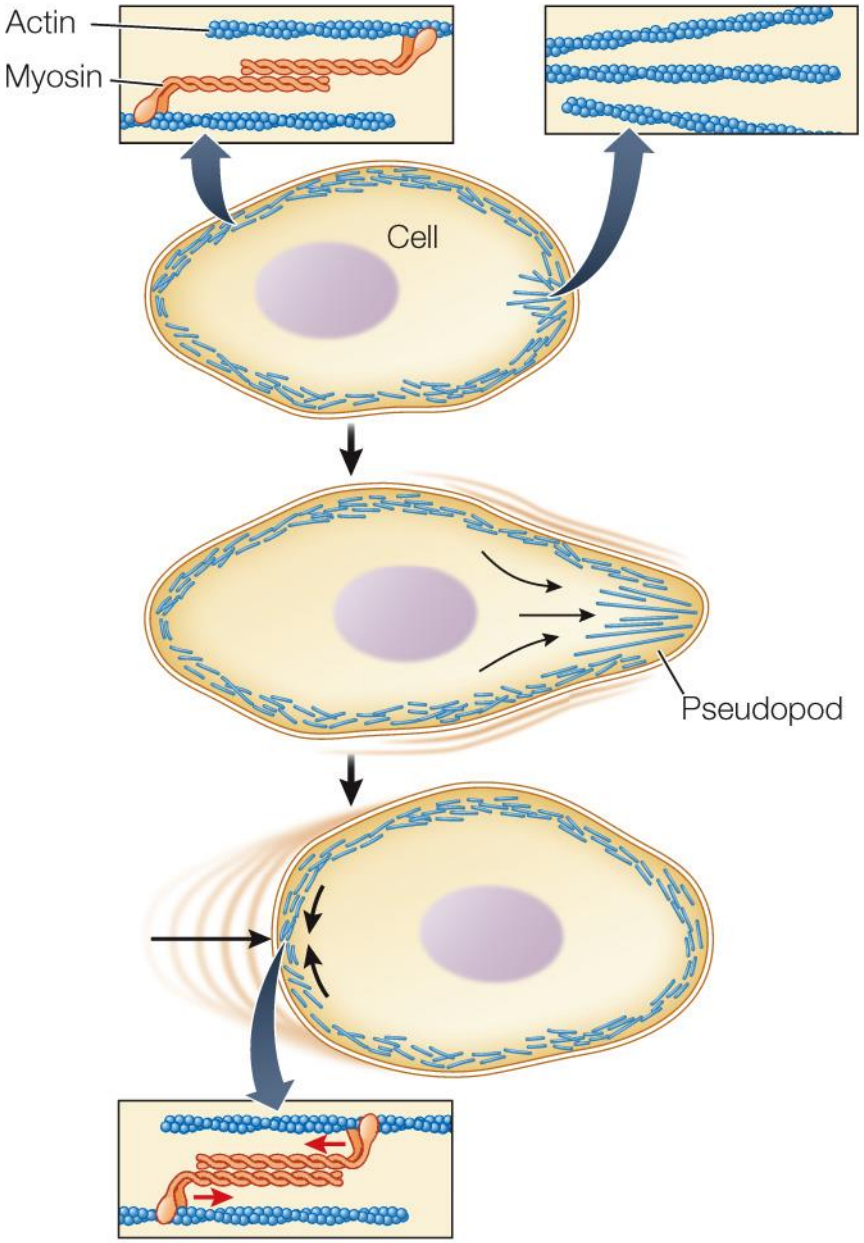


LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 5.14 (Part 1)

Actin filaments are associated with localized changes in cell shape, including cytoplasmic streaming and amoeboid movement.

Microfilaments are also involved in the formation of pseudopodia.

Figure 5.15 Microfilaments and Cell Movements

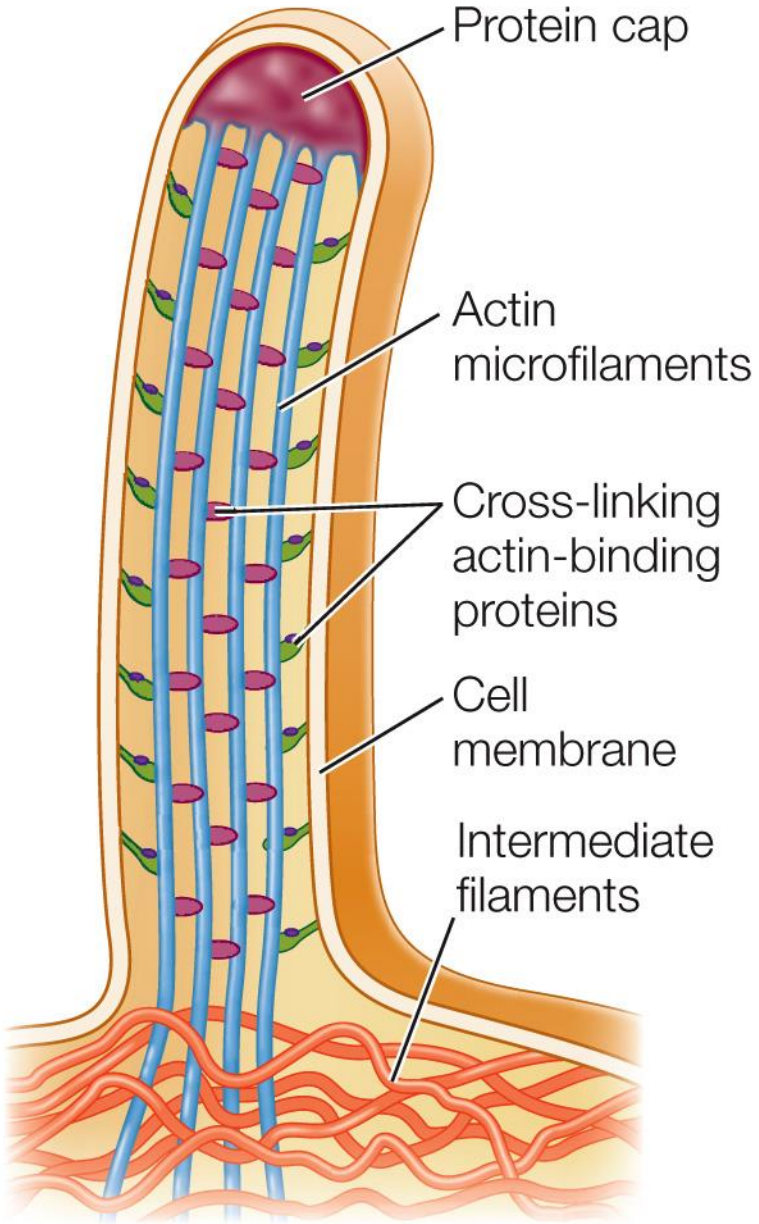


LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 5.15

In some cells, microfilaments form a meshwork just inside the cell membrane.

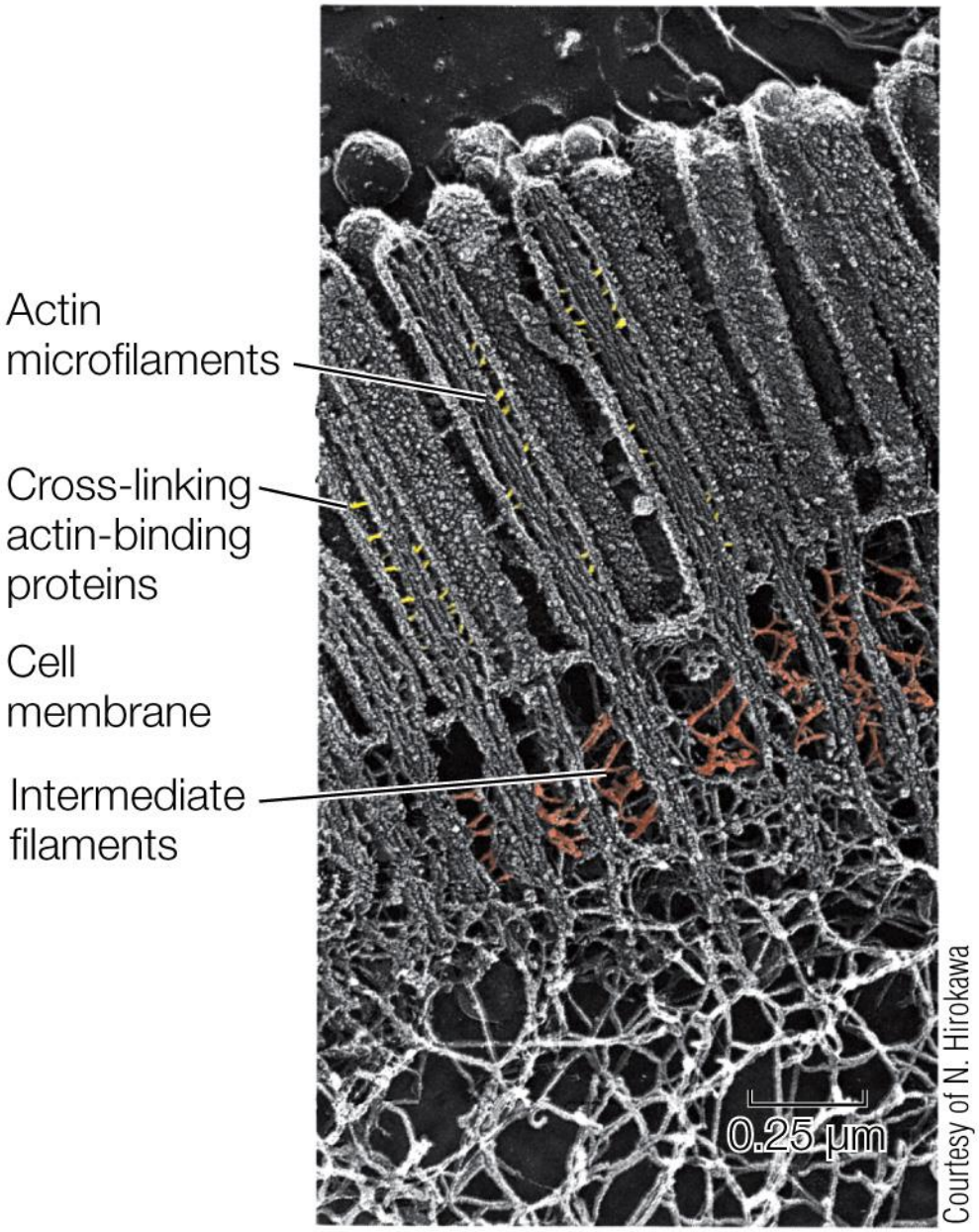
This provides structure, for example in the microvilli that line the human intestine.

Figure 5.16 Microfilaments for Support (Part 1)

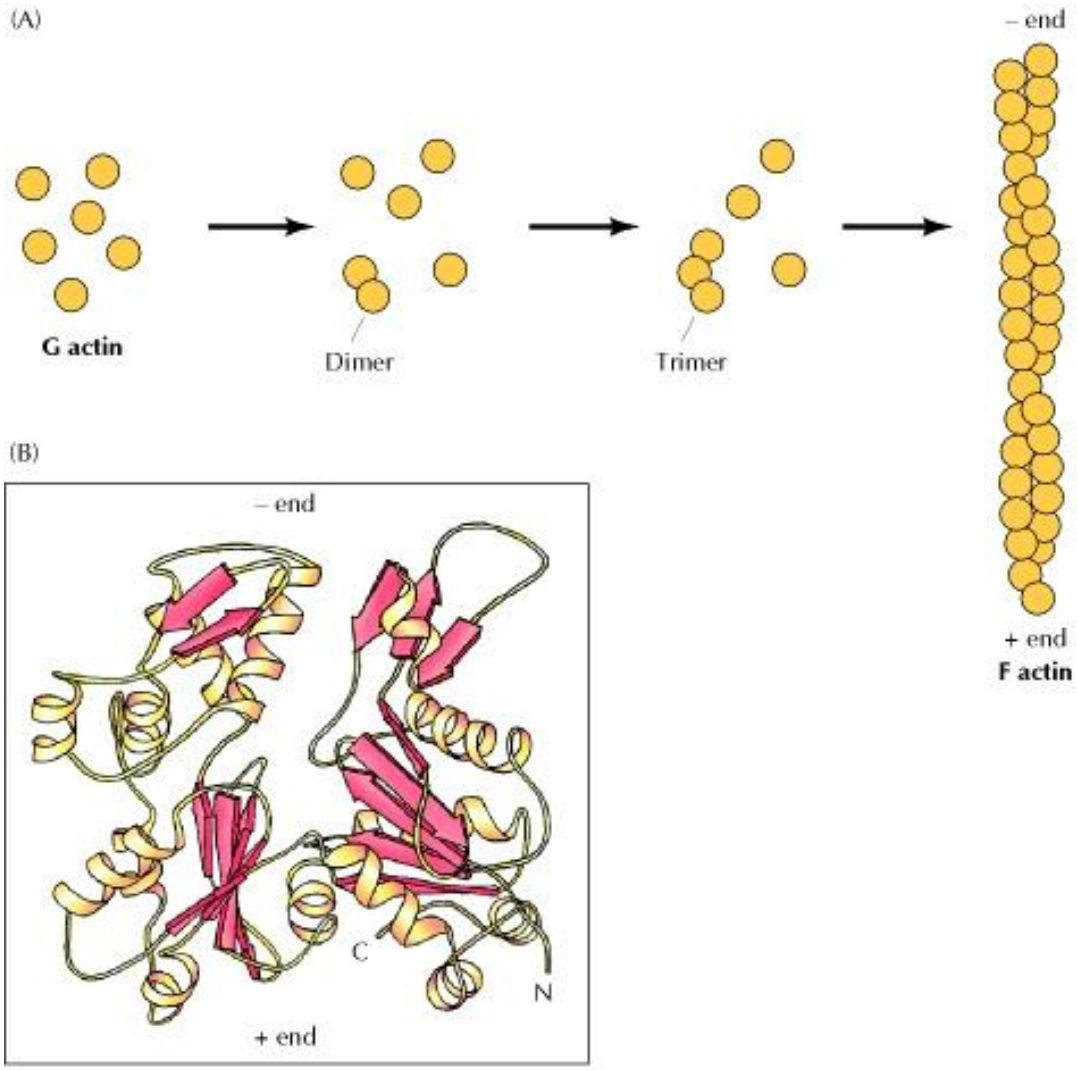


LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 5.16 (Part 1)

Figure 5.16 Microfilaments for Support (Part 2)

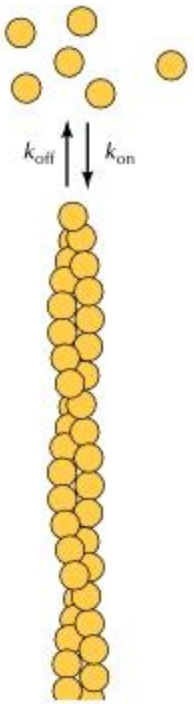


LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 5.16 (Part 2)



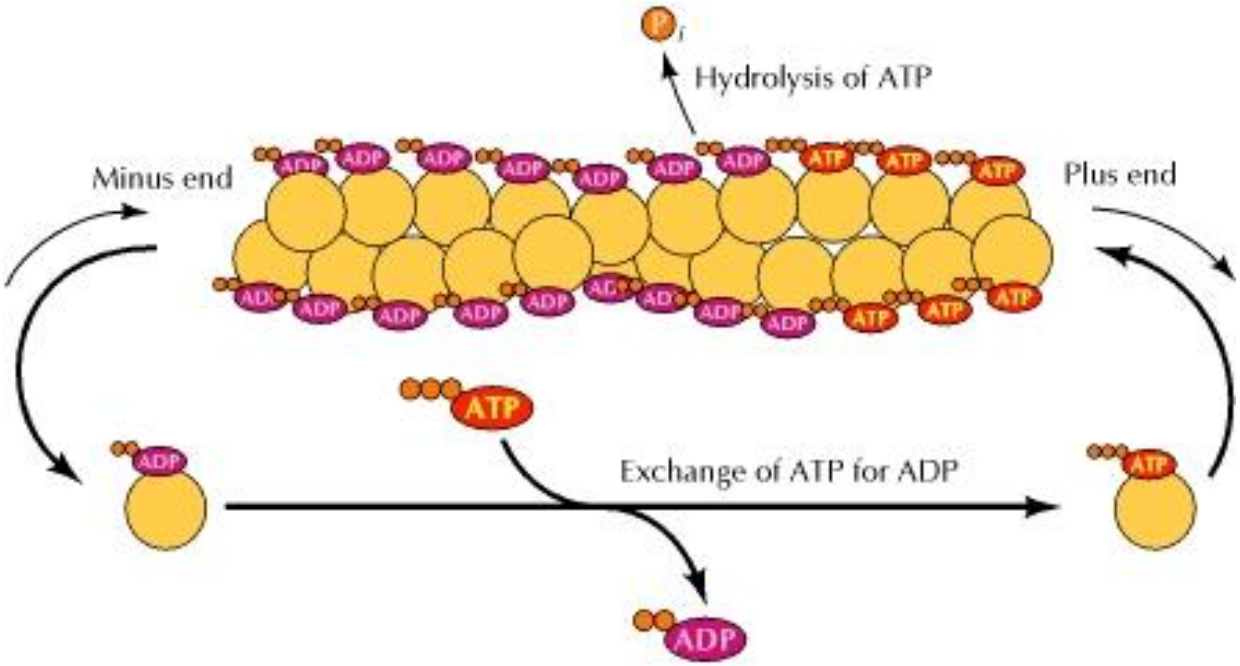
Assembly and structure of actin filaments

(A) Actin monomers (G actin) polymerize to form actin filaments (F actin). The first step is the formation of dimers and trimers, which then grow by the addition of monomers to both ends. (B) Structure of an actin monomer. (C) Space-filling model of F actin. Nine actin monomers are represented in different colors. (C, courtesy of Dan Richardson.)



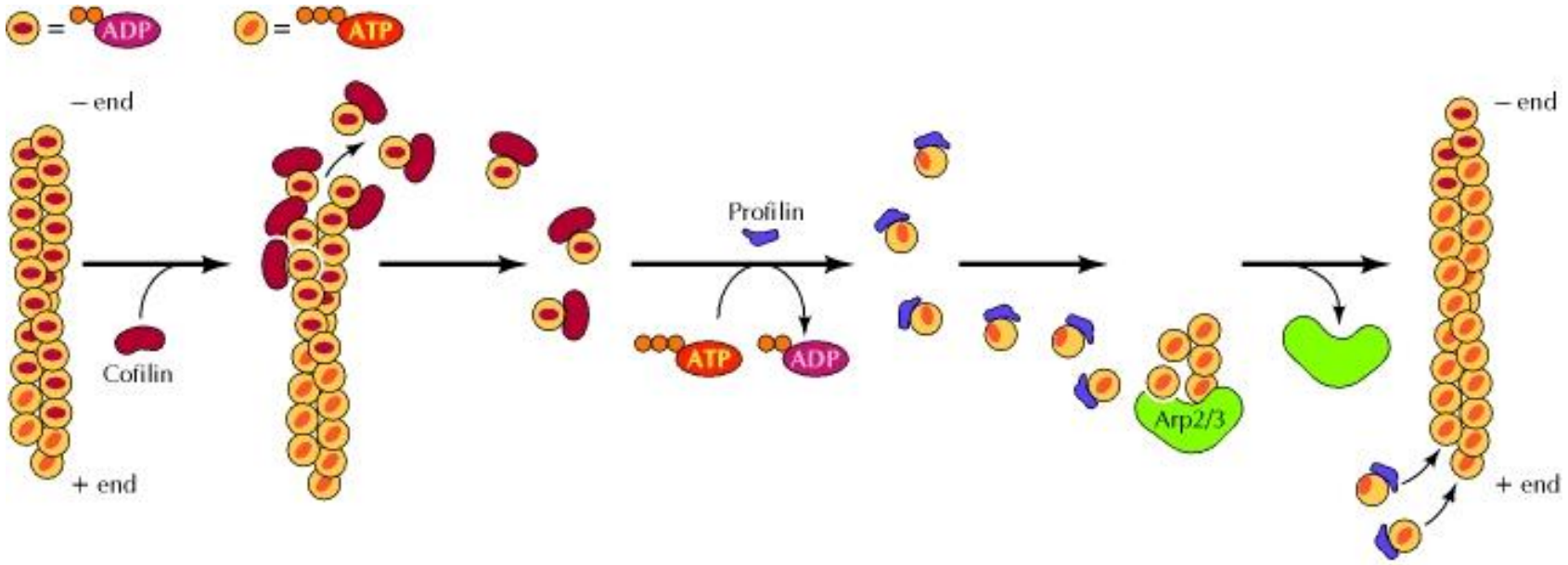
Reversible polymerization of actin monomers

Actin polymerization is a reversible process, in which monomers both associate with and dissociate from the ends of actin filaments. The rate of subunit dissociation (k_{off}) is independent of monomer concentration, while the rate of subunit association is proportional to the concentration of free monomers and given by $C \times k_{on}$ (C = concentration of free monomers). An apparent equilibrium is reached at the critical concentration of monomers (C_c), where $k_{off} = C_c \times k_{on}$.



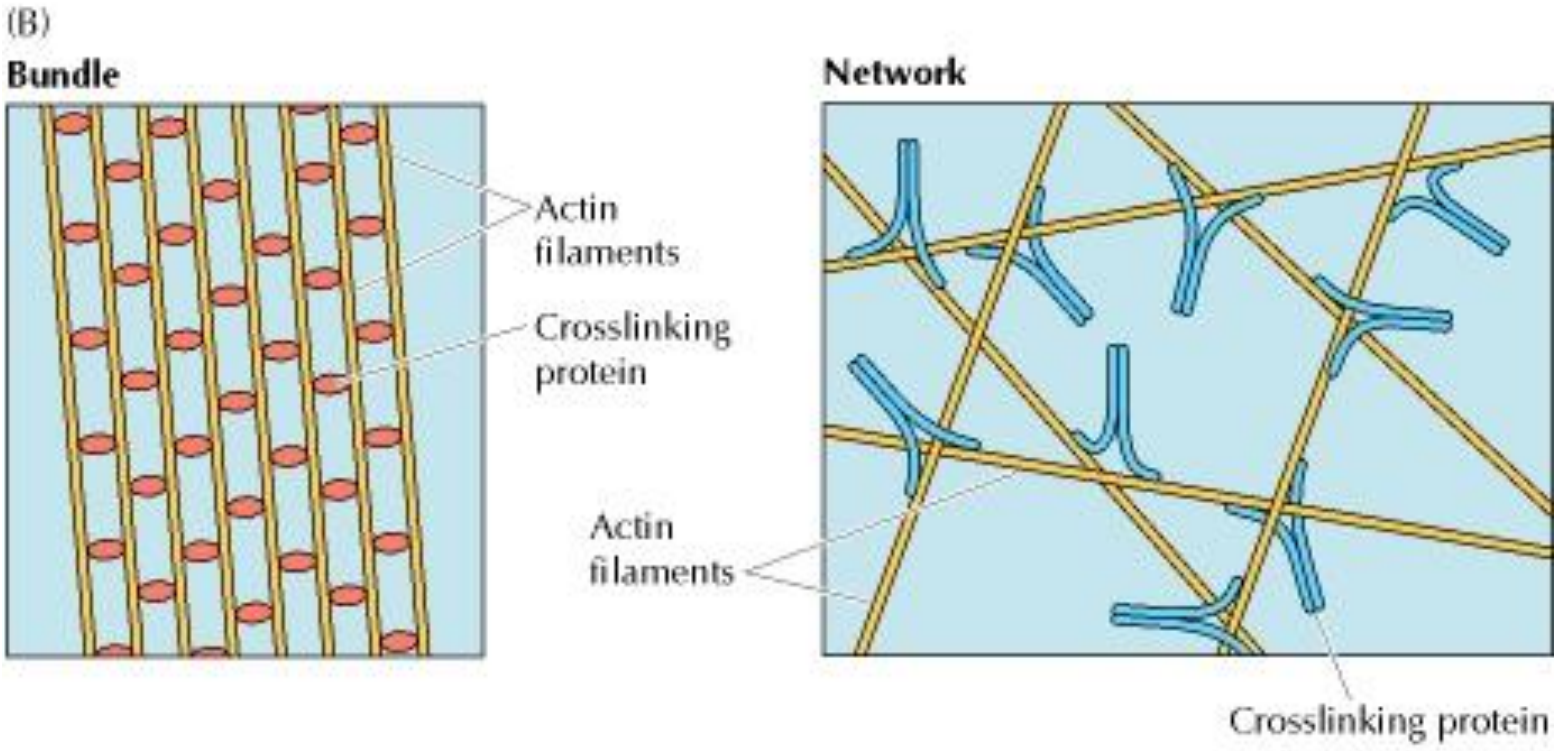
Treadmilling

The minus ends grow less rapidly than the plus ends of actin filaments. This difference in growth rate is reflected in a difference in the critical concentration for addition of monomers to the two ends of the filament. Actin bound to ATP associates with the rapidly growing plus ends, and the ATP bound to actin is then hydrolyzed to ADP. Because ADP-actin dissociates from filaments more readily than ATP-actin, the critical concentration of actin monomers is higher for addition to the minus end than to the plus end of actin filaments. Treadmilling takes place at monomer concentrations intermediate between the critical concentrations for the plus and minus ends. Under these conditions, there is a net dissociation of monomers (bound to ADP) from the minus end, balanced by the addition of monomers (bound to ATP) to the plus end.



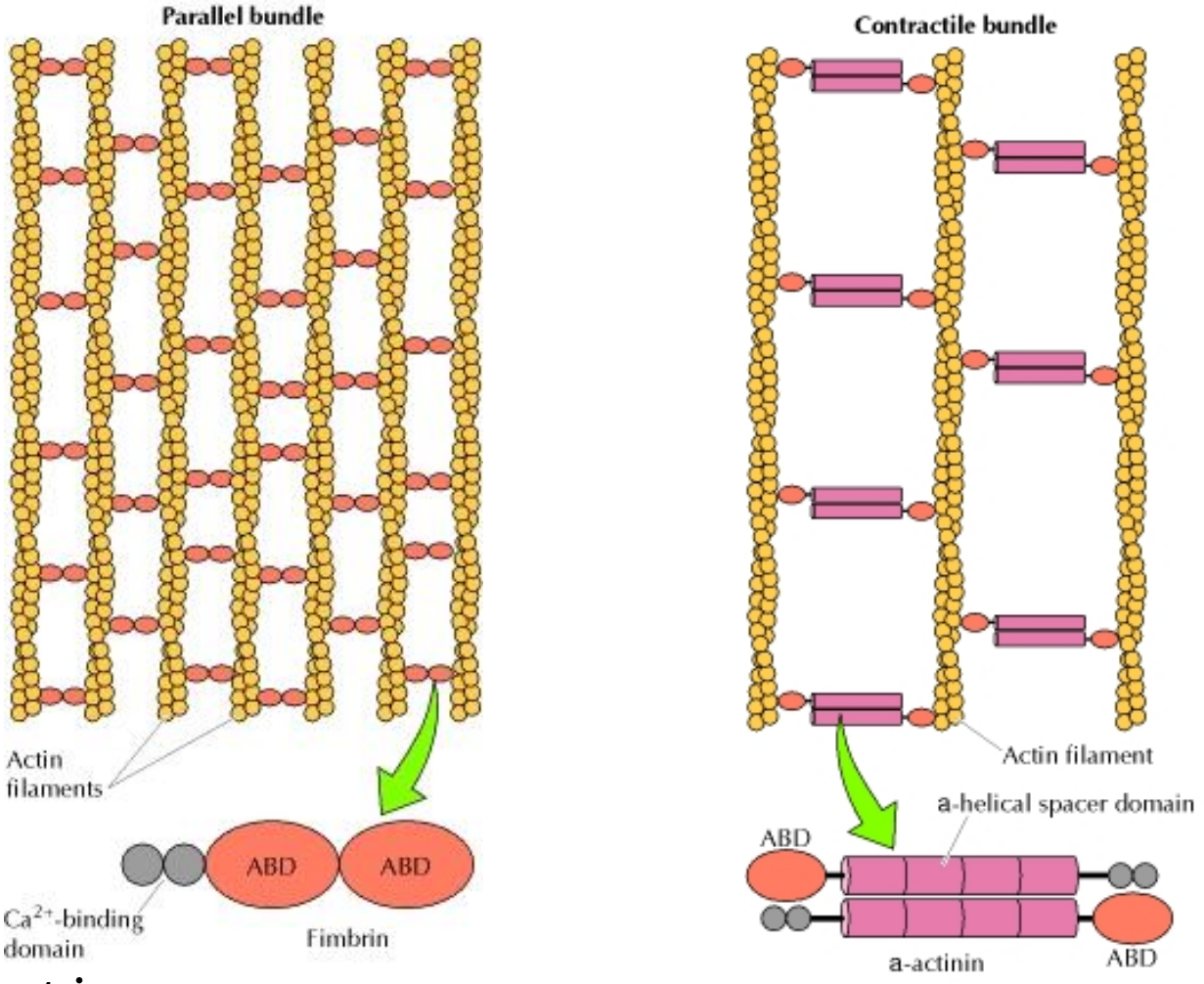
Effects of actin-binding proteins on filament turnover

Cofilin binds to actin filaments and increases the rate of dissociation of actin monomers (bound to ADP) from the minus end. Cofilin remains bound to the ADP-actin monomers, preventing their reassembly into filaments. However, profilin can stimulate the exchange of bound ADP for ATP, resulting in the formation of ATP-actin monomers that can be repolymerized into filaments, including new filaments nucleated by the Arp2/3 proteins.



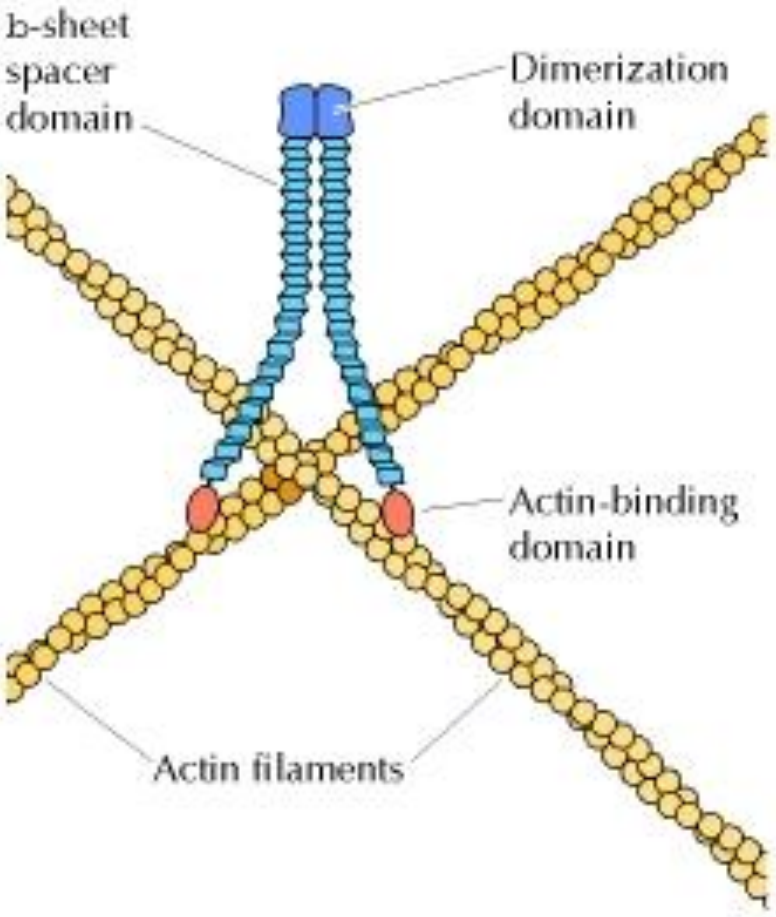
Actin bundles and networks

(A) Electron micrograph of actin bundles (arrowheads) projecting from the actin network (arrows) underlying the plasma membrane of a macrophage. The bundles support cell surface projections called microspikes or filopodia (see Figure 11.17). (B) Schematic organization of bundles and networks. Actin filaments in bundles are crosslinked into parallel arrays by small proteins that align the filaments closely with one another. In contrast, networks are formed by large flexible proteins that crosslink orthogonal filaments. (A, courtesy of John H. Hartwig, Brigham & Women's Hospital.)



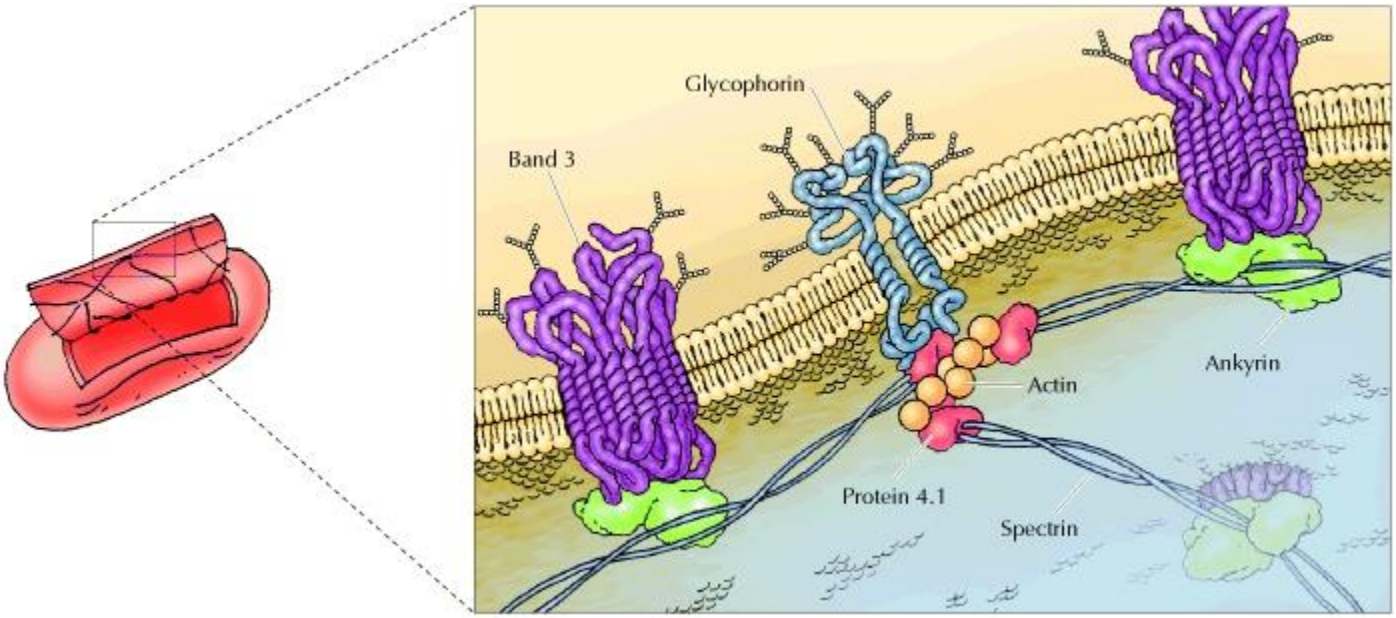
Actin-bundling proteins

Actin filaments are associated into two types of bundles by different actin-bundling proteins. Fimbrin has two adjacent actin-binding domains (ABD) and crosslinks actin filaments into closely packed parallel bundles in which the filaments are approximately 14 nm apart. In contrast, the two separated actin-binding domains of α-actinin dimers crosslink filaments into more loosely spaced contractile bundles in which the filaments are separated by 40 nm. Both fimbrin and α-actinin contain two related Ca^{2+}-binding domains, and α-actinin contains four repeated α-helical spacer domains.

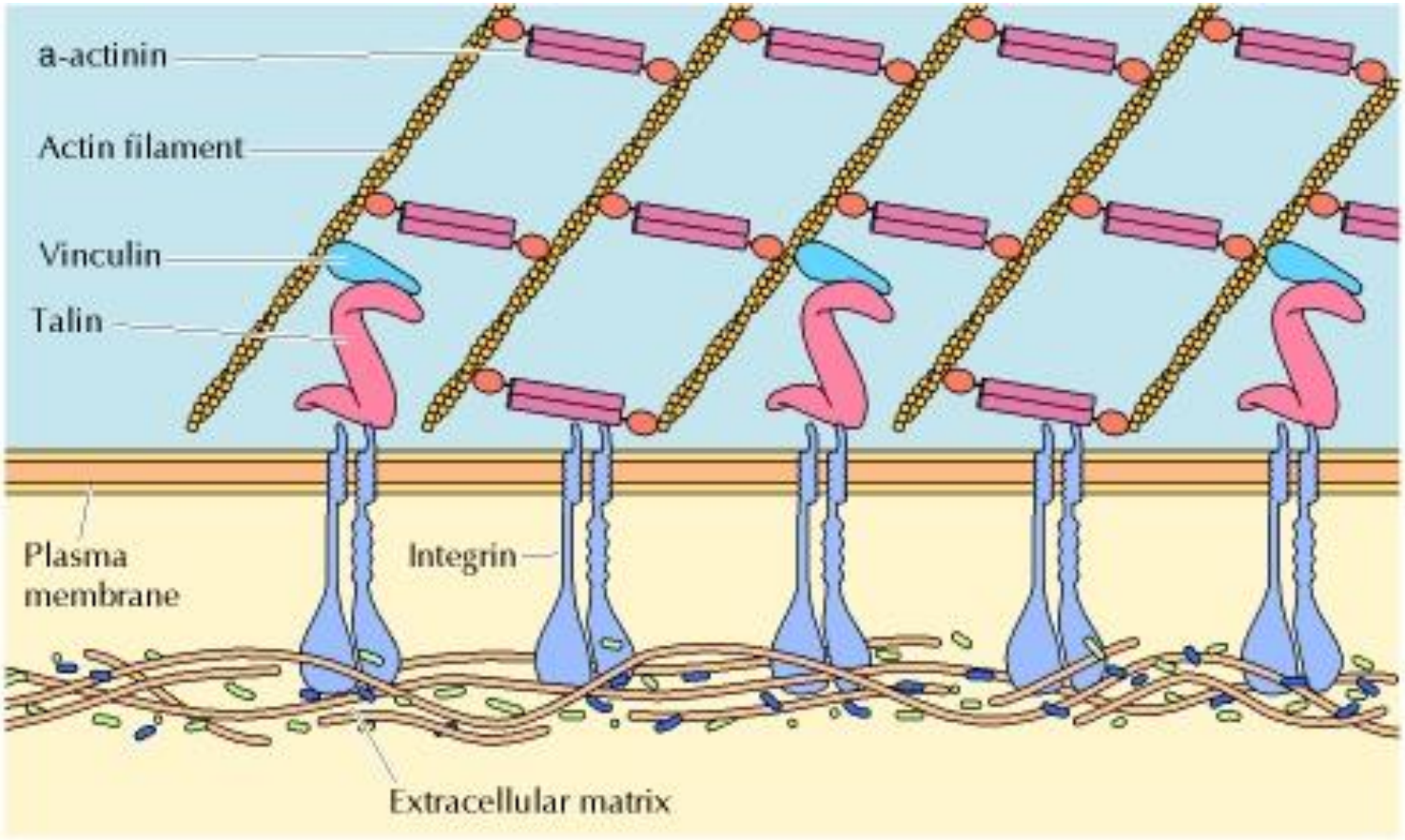


Actin networks and filamin

Filamin is a dimer of two large (280-kd) subunits, forming a flexible V-shaped molecule that crosslinks actin filaments into orthogonal networks. The carboxy-terminal dimerization domain is separated from the amino-terminal actin-binding domain by repeated β-sheet spacer domains.

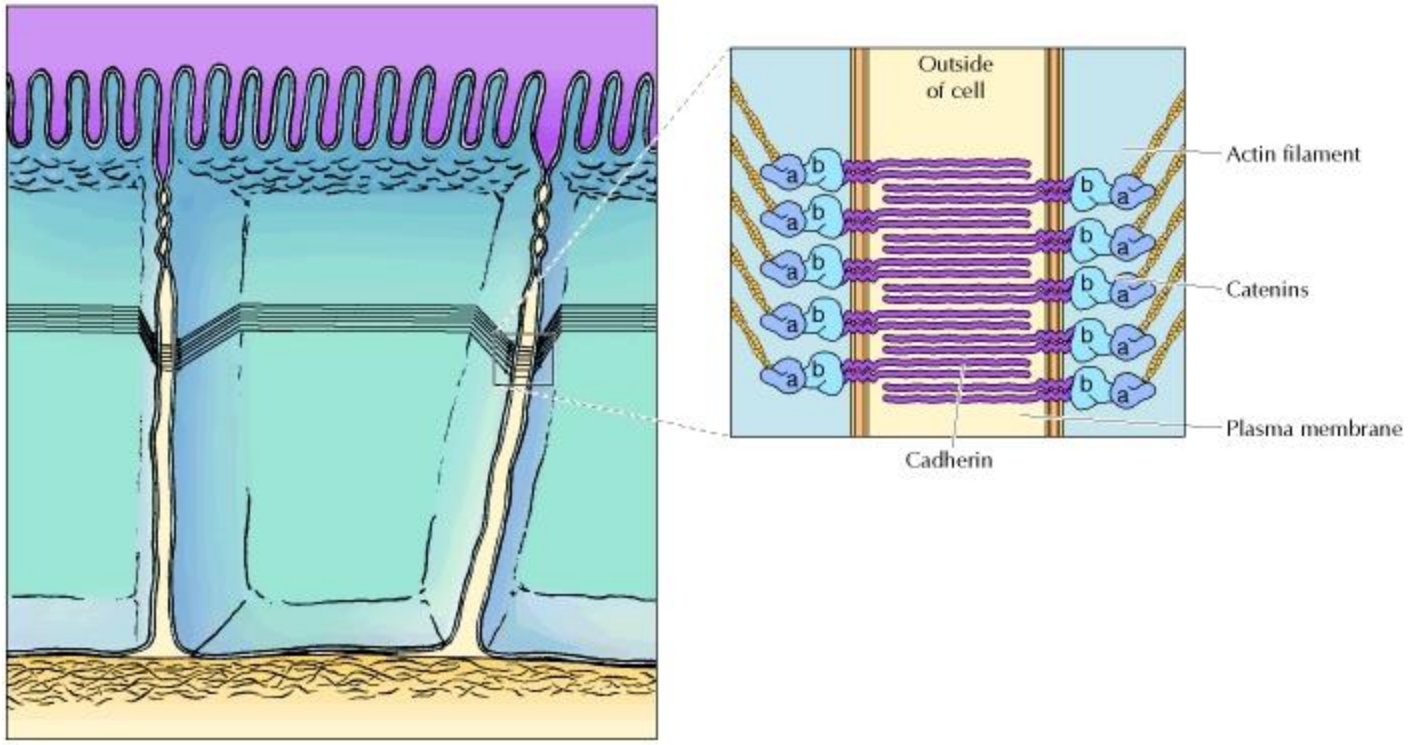


Association of the erythrocyte cortical cytoskeleton with the plasma membrane
The plasma membrane is associated with a network of spectrin tetramers crosslinked by short actin filaments in association with protein 4.1. The spectrin-actin network is linked to the membrane by ankyrin, which binds to both spectrin and the abundant transmembrane protein band 3. An additional link is provided by the binding of protein 4.1 to glycophorin.



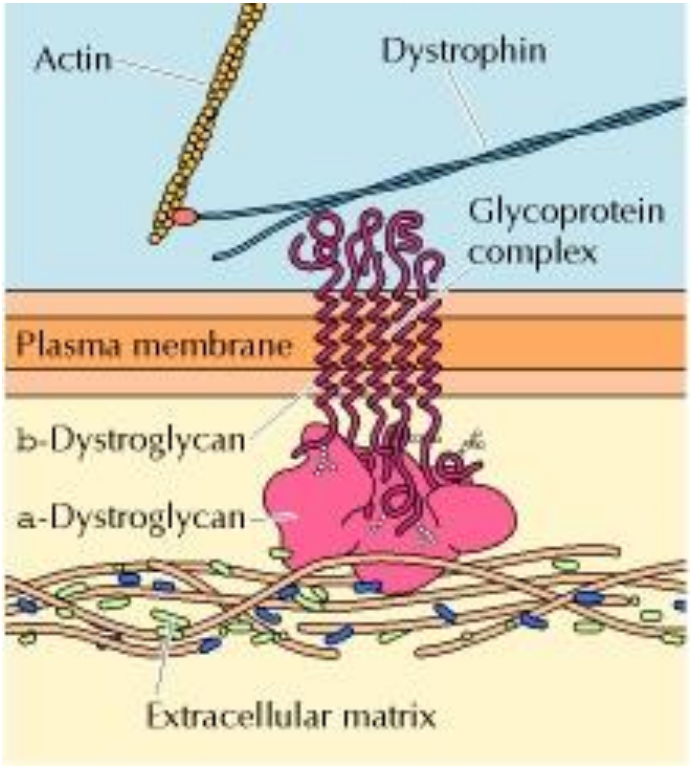
Attachment of stress fibers to the plasma membrane at focal adhesions

Focal adhesions are mediated by the binding of integrins to proteins of the extracellular matrix. Stress fibers (bundles of actin filaments crosslinked by α-actinin) are then bound to the cytoplasmic domain of integrins by complex associations involving a number of proteins. Two possible associations are illustrated: 1) talin binds to both integrin and vinculin, which in turn binds to actin, and 2) integrin binds to α-actinin. A number of other proteins (not shown) are also present at focal adhesions and may be involved in anchoring stress fibers to the plasma membrane.



Attachment of actin filaments to adherens junctions

Cell-cell contacts at adherens junctions are mediated by cadherins, which serve as sites of attachment of actin bundles. In sheets of epithelial cells, these junctions form a continuous belt of actin filaments around each cell. The cadherins are transmembrane proteins that bind β-catenin to their cytoplasmic domains. β-catenin interacts with α-catenin, which serves as a link to actin filaments.



The Disease

The muscular dystrophies are a group of hereditary diseases characterized by the progressive loss of muscle cells. Duchenne's muscular dystrophy (DMD) is the most common and severe form, affecting approximately one out of every 3500 male children.

Molecular and Cellular Basis

The much higher incidence of DMD and BMD in boys than in girls initially suggested that both diseases result from recessive sex-linked genes. This hypothesis was confirmed by genetic studies, which localized the DMD/ BMD gene to a specific region of the X chromosome. On the basis of its chromosomal position, the gene responsible for DMD and BMD was cloned by the research groups of Lou Kunkel and Ron Worton in 1986. Sequence analysis established that it encodes a 427-kd protein, called dystrophin, which is related to spectrin. Dystrophin is linked to the plasma membrane of muscle cells by a complex of transmembrane proteins. These transmembrane proteins in turn bind to components of the extracellular matrix, so dystrophin plays a key role in anchoring the cytoskeleton of muscle cells to the extracellular matrix. This anchorage is thought to stabilize the plasma membrane and enable the cell to withstand the stress of muscle contraction. The mutations responsible for DMD or BMD result either in the absence of dystrophin or in the expression of an abnormal protein, respectively, consistent with the severity of disease in DMD and BMD patients.

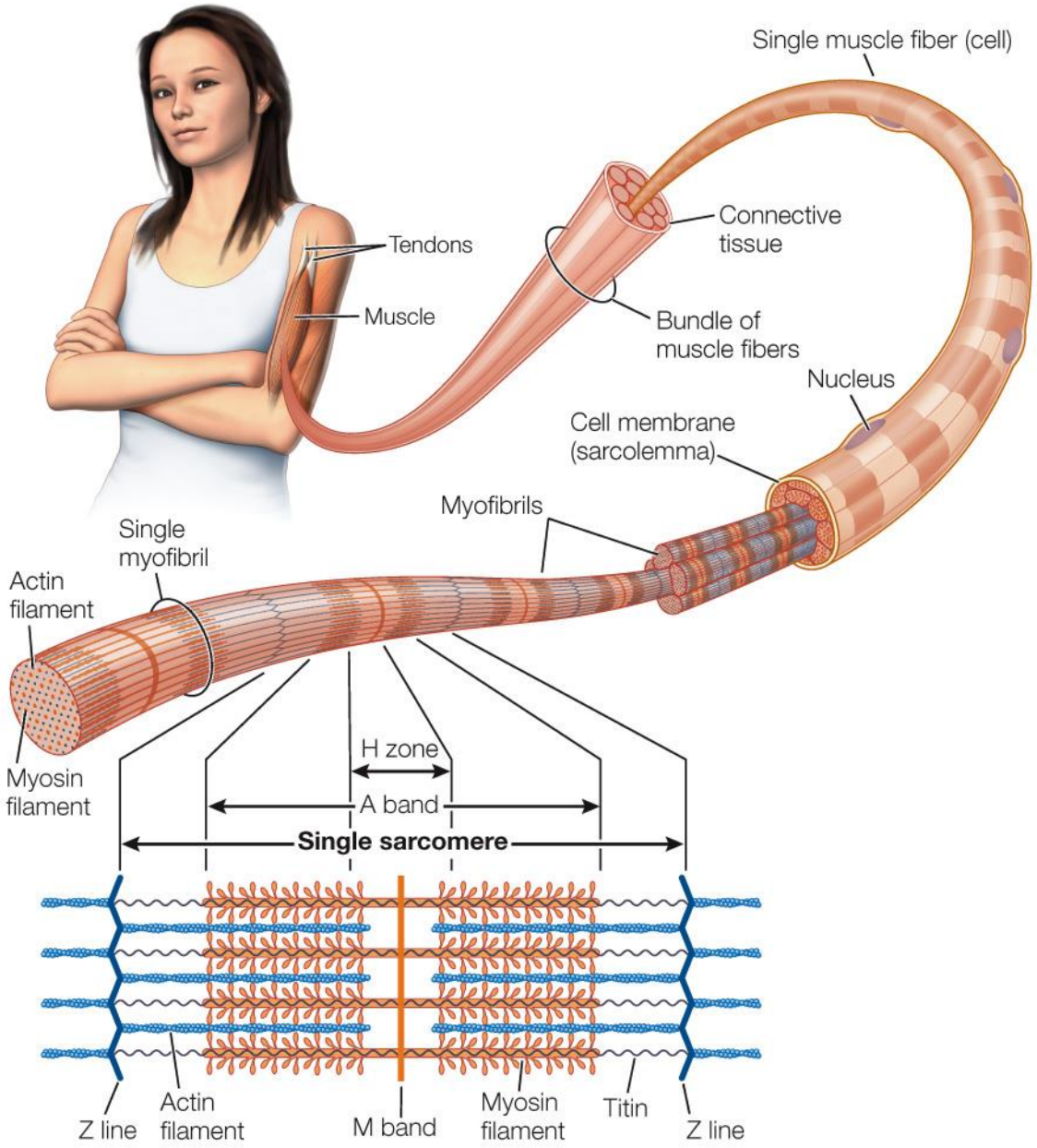
Interactions of Actin and Myosin Cause Muscles to Contract

- When skeletal muscle contracts, the sarcomeres shorten and the band pattern changes.
- Muscle contraction is due to repeated cross bridge forming, breaking, and reforming between the actin and myosin filaments causing them to slide past each other.

Skeletal muscle (striated):

- Cells are called **muscle fibers**—large and multinucleate.
- Form from fusion of embryonic myoblasts.
- One muscle consists of many muscle fibers bundled together by connective tissue.

Figure 47.1 The Structure of Skeletal Muscle (Part 1)



LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 47.1 (Part 1)

Contractile proteins:

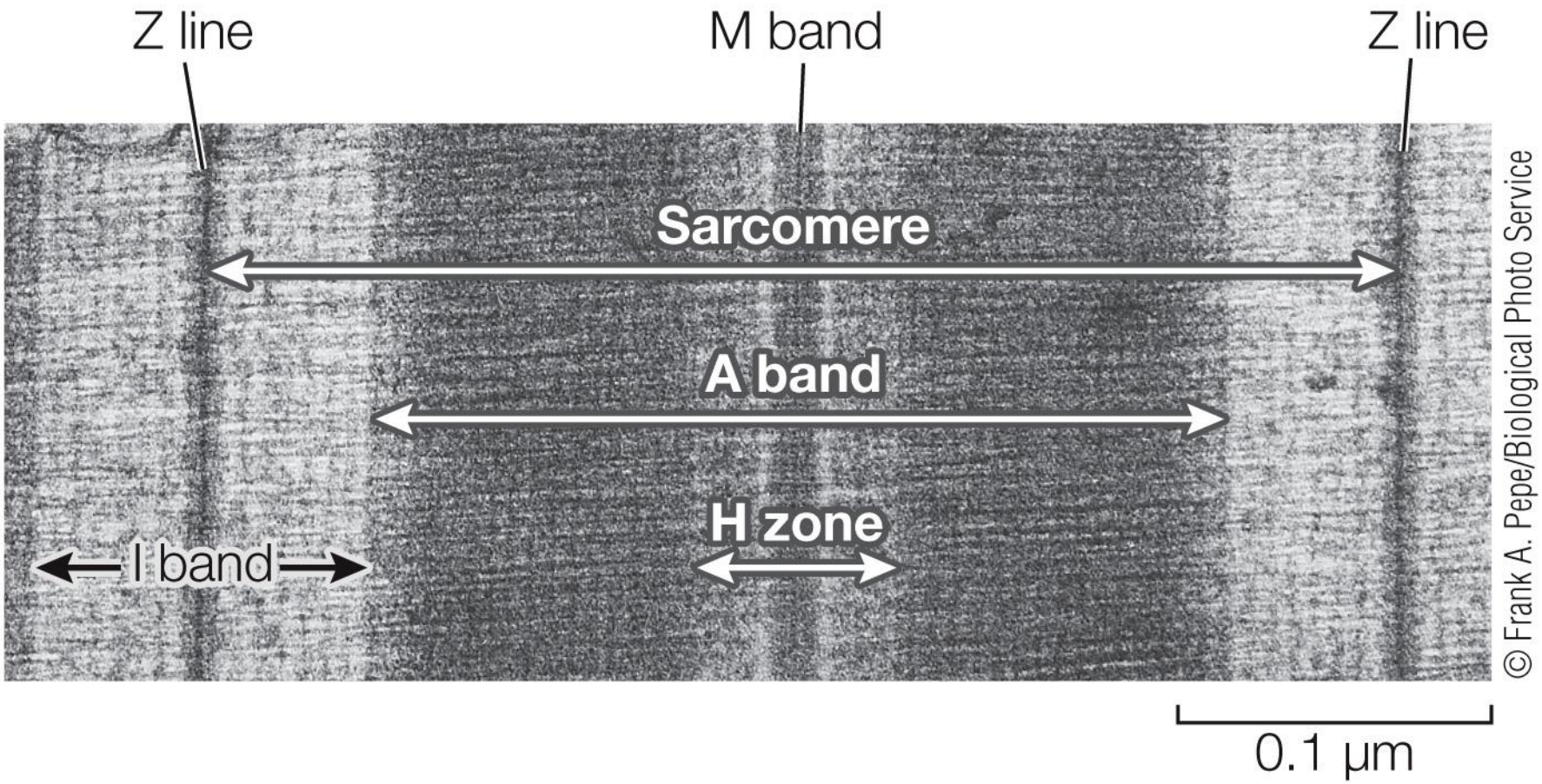
- **Actin**—thin filaments
- **Myosin**—thick filaments

Each muscle fiber (cell) has many **myofibrils**—bundles of actin and myosin filaments.

Each myofibril consists of **sarcomeres**—repeating units of overlapping actin and myosin filaments.

Each sarcomere is bounded by Z lines, which anchor the actin.

Figure 47.1 The Structure of Skeletal Muscle (Part 2)



LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 47.1 (Part 2)

Titin: The largest protein in the body; runs full length of the sarcomere.

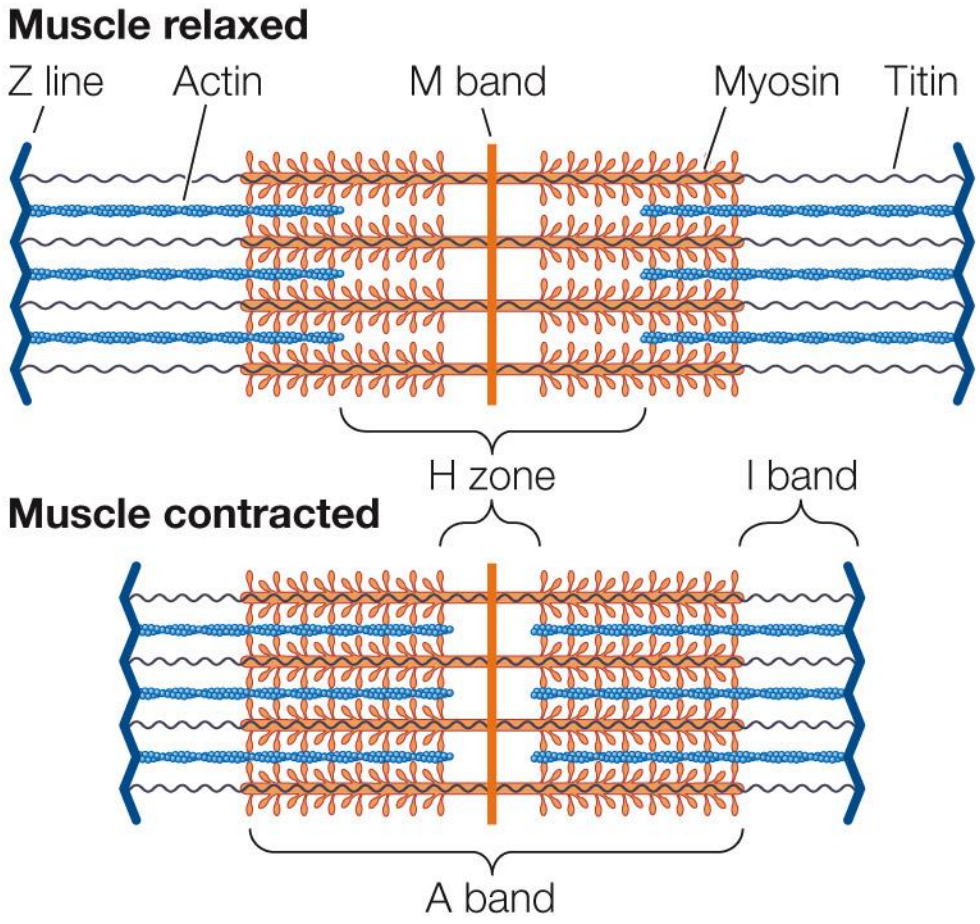
Each titin molecule runs through a myosin bundle. It is very stretchable.

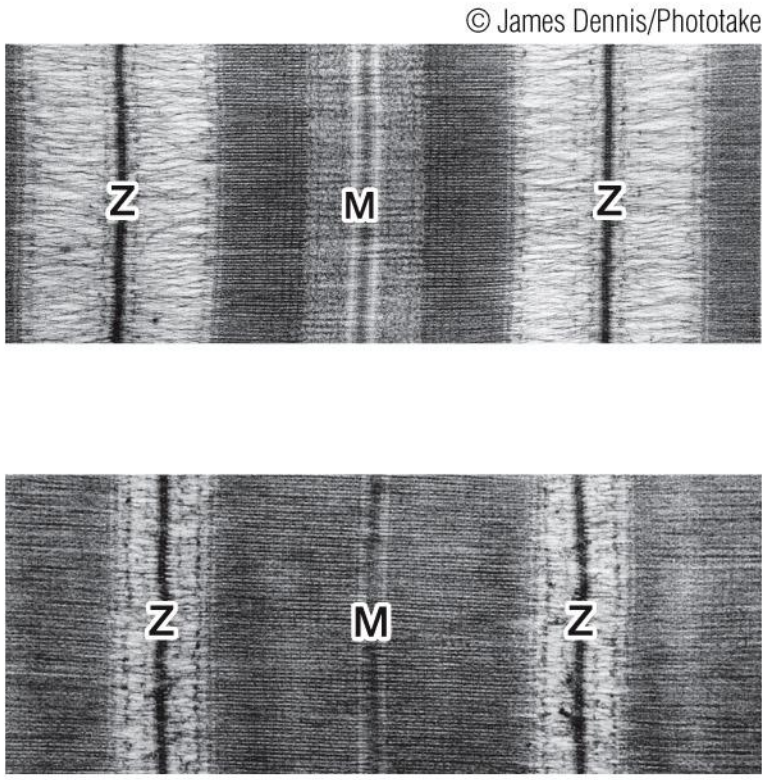
In a relaxed muscle, resistance to stretch is mostly due to elasticity of the titin molecules.

When a muscle contracts, sarcomeres shorten and the band pattern changes—the actin and myosin filaments slide past each other.

Observation of this led to development of the **sliding filament model** of muscle contraction.

Figure 47.2 Sliding Filaments

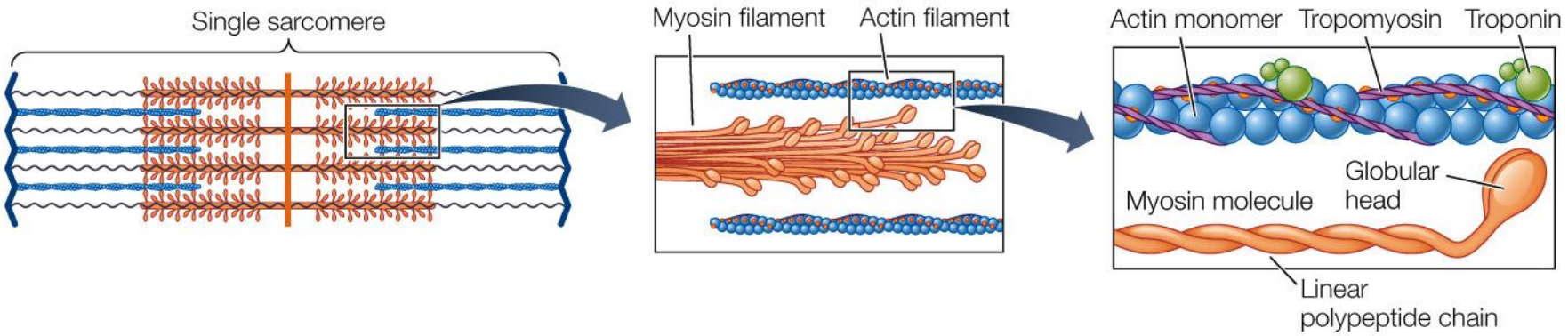




LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 47.2

Another protein, **tropomyosin**, twists around actin with **troponin** attached at intervals.

Figure 47.3 Actin and Myosin Filaments Overlap to Form Myofibrils



LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 47.3

Myosin heads bind to actin molecules to form cross-bridges.

The myosin head changes conformation and the head bends; causing the actin filament to slide 5–10 nm.

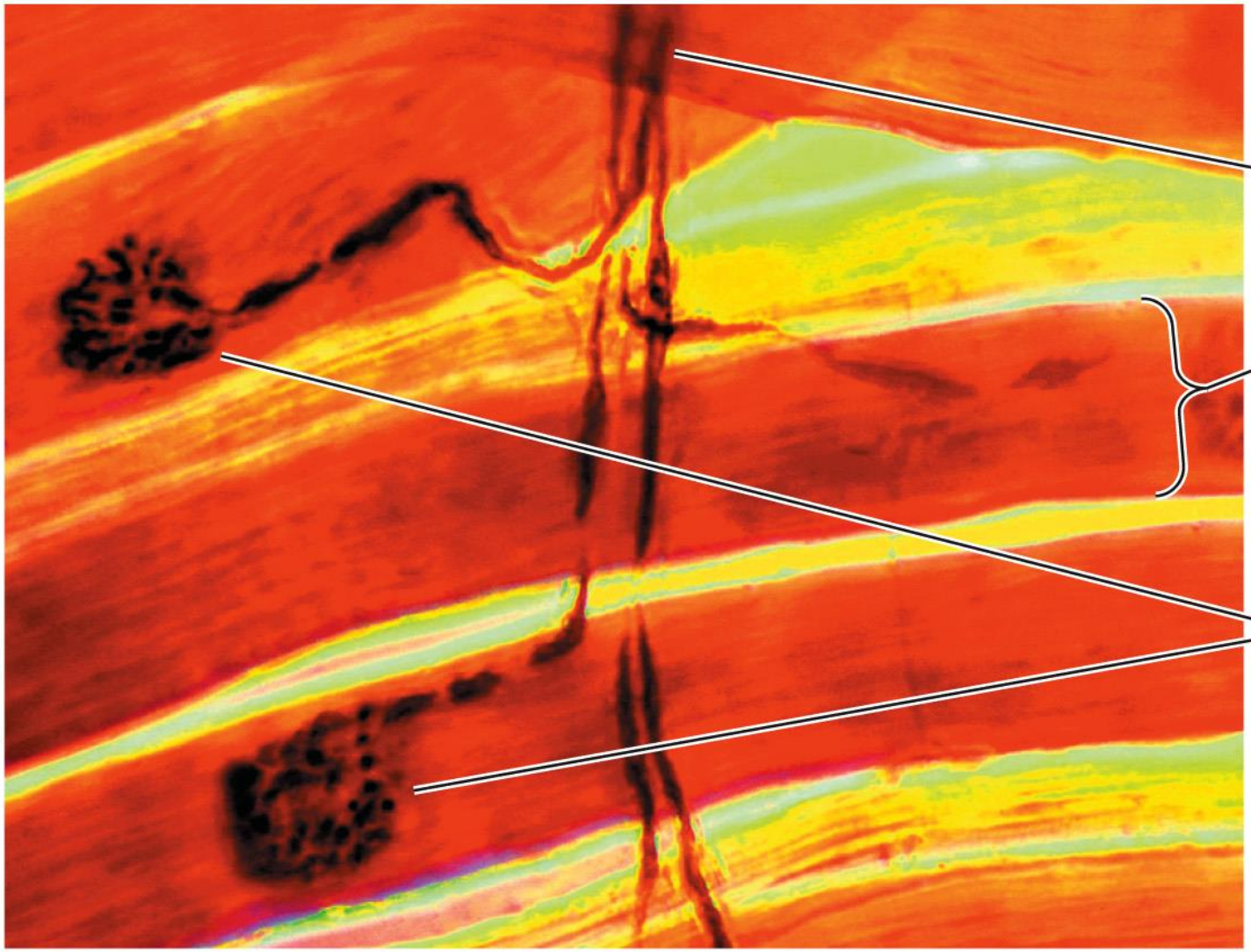
Myosin head hydrolyzes ATP, myosin changes conformation again, and releases the actin.

Muscle cells are excitable—the cell membranes can generate and conduct action potentials.

Contraction is initiated by action potentials from a motor neuron at the neuromuscular junction.

Motor unit: One motor neuron and all the muscle fibers it synapses with.

Figure 47.4 The Neuromuscular Junction



LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 47.4

At the neuromuscular junction, acetylcholine is the transmitter.

It binds to receptors in the postsynaptic membrane, ion channels in the motor end plate open, Na^+ flows in, and the motor end plate is depolarized.

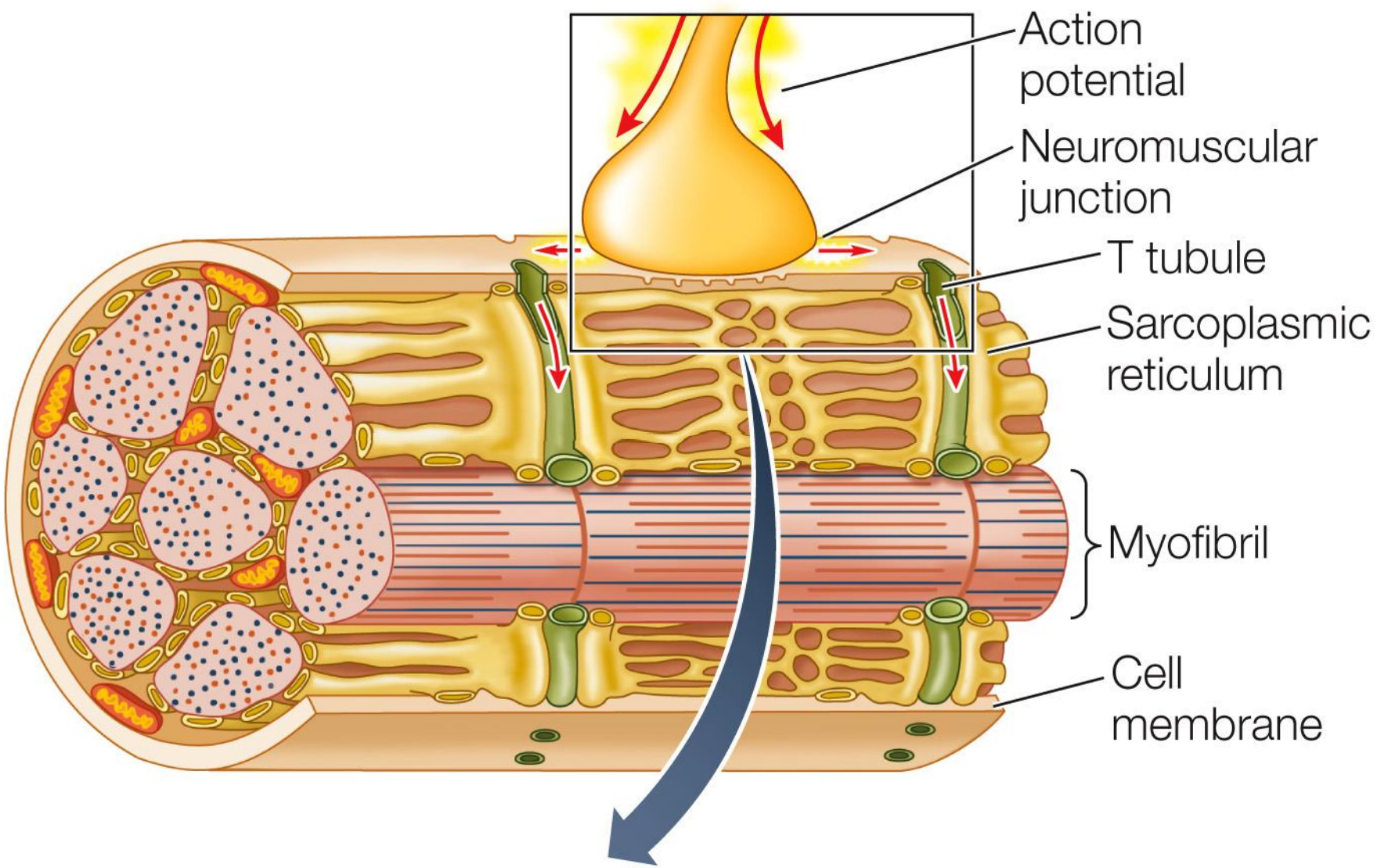
Depolarization spreads; when threshold is reached, the muscle fiber membrane fires an action potential.

Action potentials in muscle fiber also travel deep within the cell.

The cell membrane is continuous with **T tubules** that run through the **sarcoplasm** (muscle fiber cytoplasm).

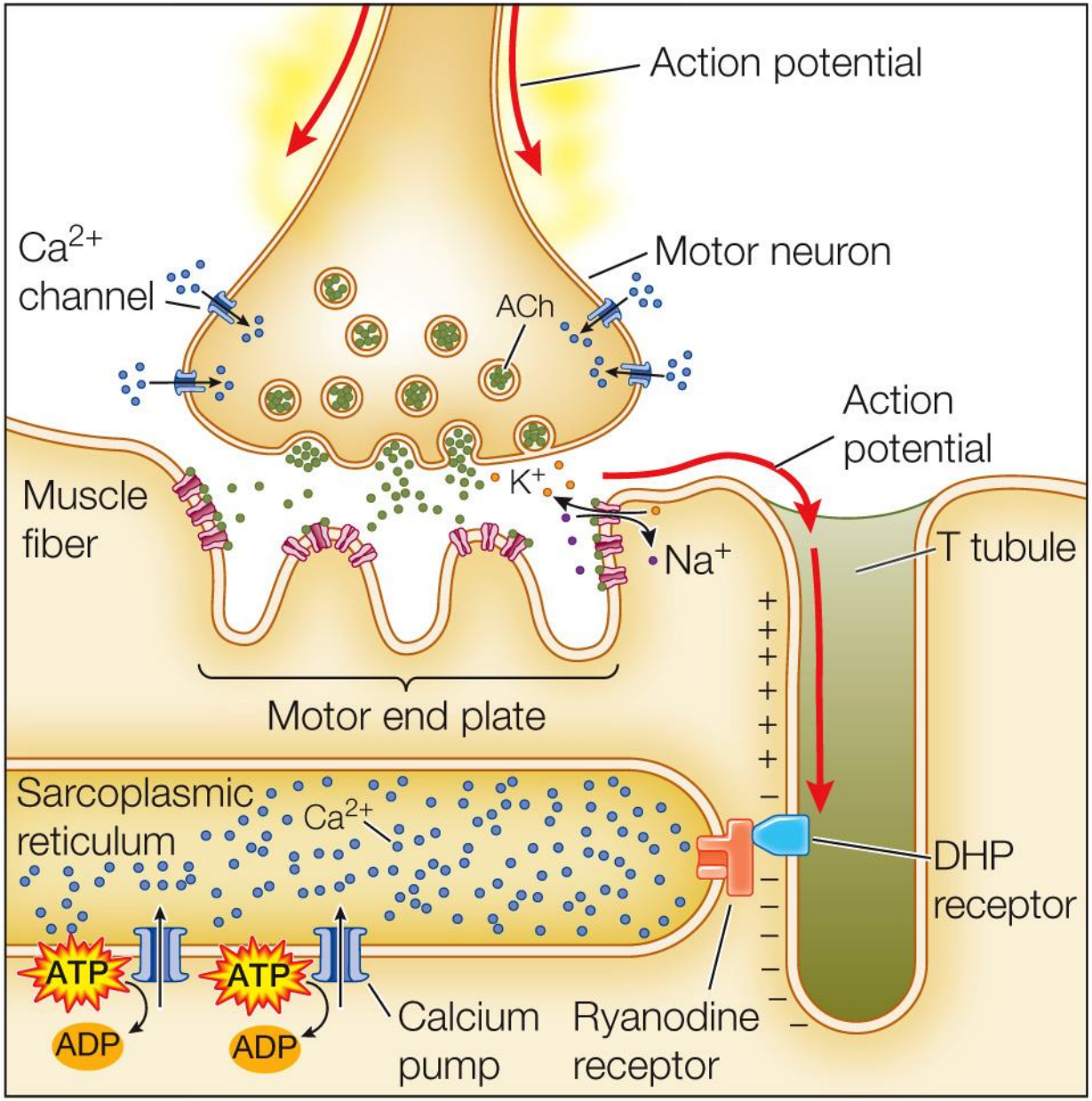
T tubules run close to the **sarcoplasmic reticulum** (muscle fiber ER) that surrounds every myofibril.

Figure 47.5 T Tubules Spread Action Potentials into the Fiber (Part 1)



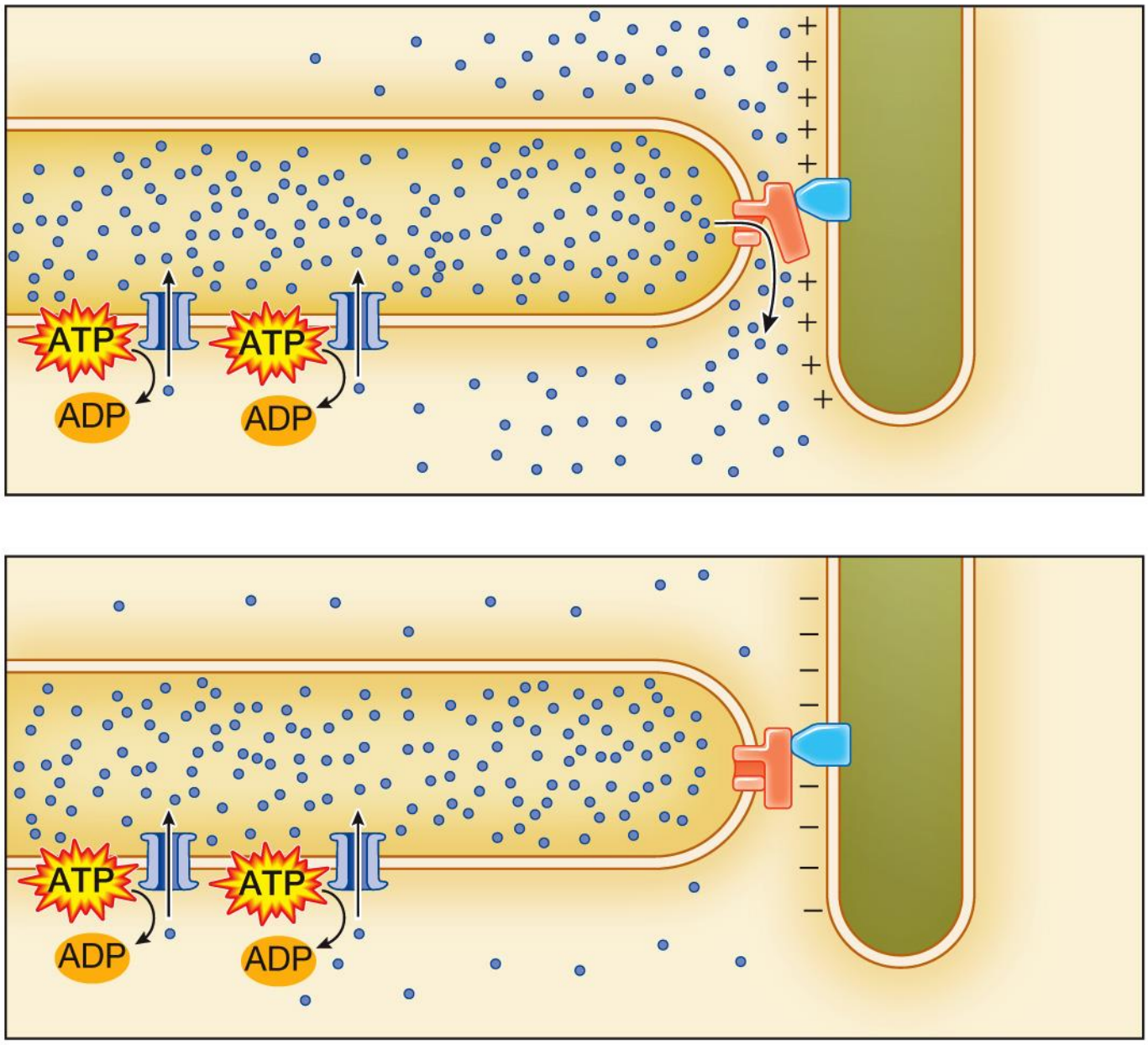
LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 47.5 (Part 1)

Figure 47.5 T Tubules Spread Action Potentials into the Fiber (Part 2)



LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 47.5 (Part 2)

Figure 47.5 T Tubules Spread Action Potentials into the Fiber (Part 3)

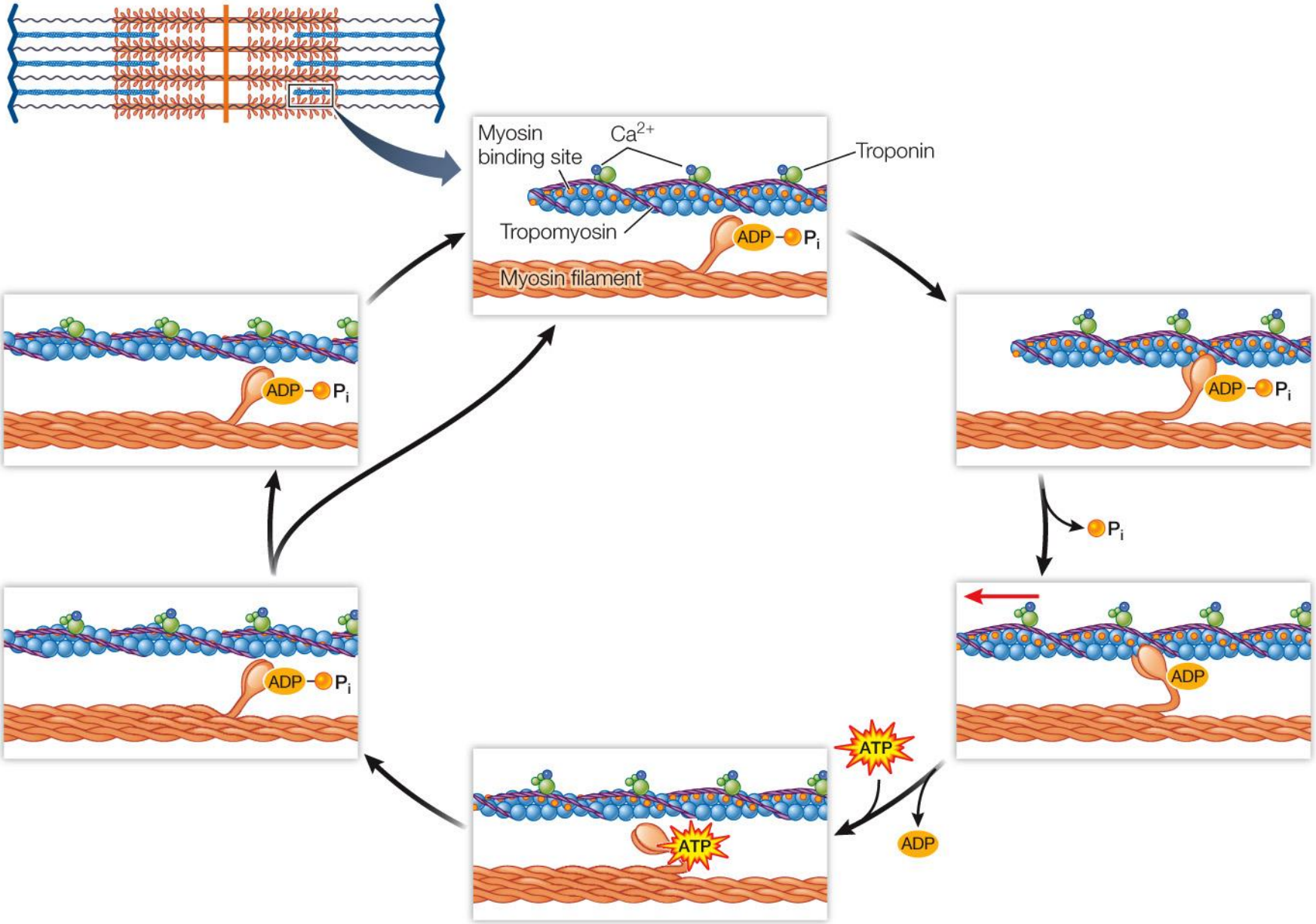


LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 47.5 (Part 3)

Ca^{2+} binds to troponin on the actin filaments—this twists the tropomyosin so that actin binding sites are exposed.

When Ca^{2+} pumps in the SR remove Ca^{2+} from sarcoplasm, contraction stops.

Figure 47.6 Release of Ca^{2+} from the Sarcoplasmic Reticulum Triggers Muscle Contraction



LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 47.6

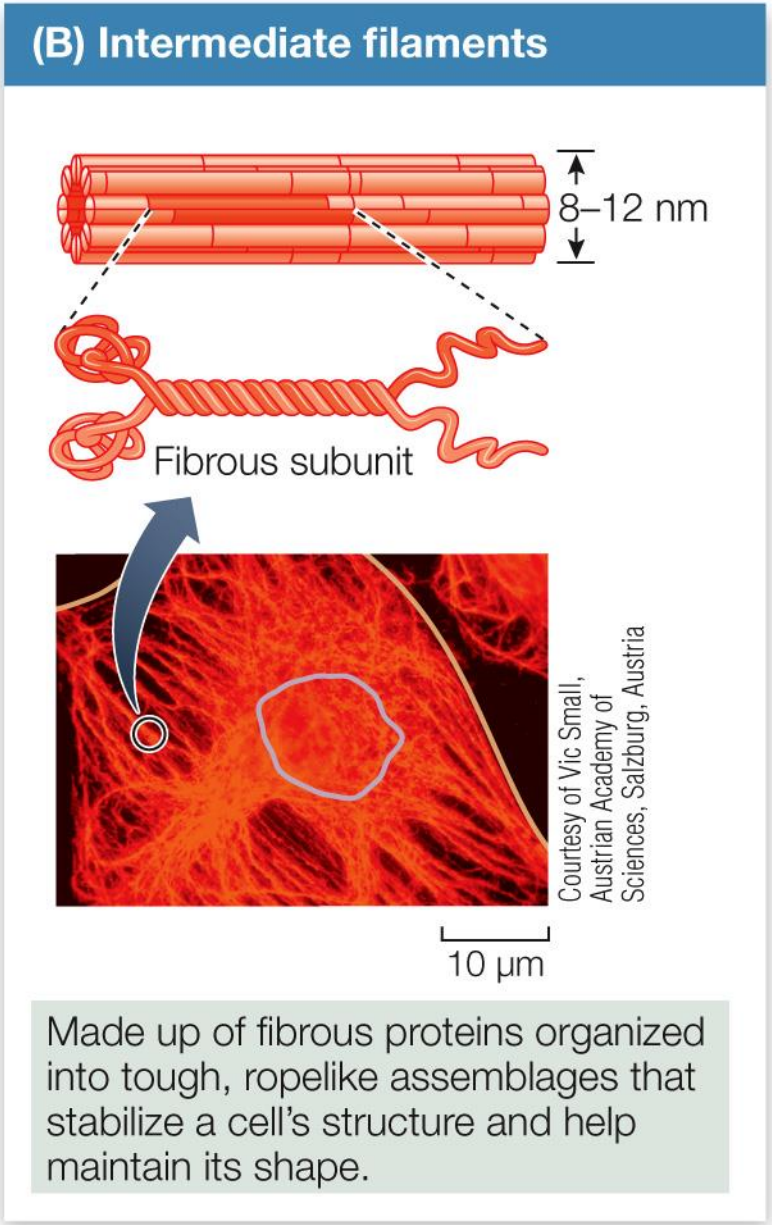
Intermediate filament

Intermediate filaments:

- 50 different kinds in 6 molecular classes
- Tough, ropelike protein structures
- Anchor cell structures in place
- Resist tension

Intermediate filaments have a diameter of about 10 nm, which is intermediate between the diameters of the two other principal elements of the cytoskeleton, actin filaments (about 7 nm) and microtubules (about 25 nm). In contrast to actin filaments and microtubules, the intermediate filaments are not directly involved in cell movements. Instead, they appear to play basically a structural role by providing mechanical strength to cells and tissues.

Figure 5.14 The Cytoskeleton (Part 2)

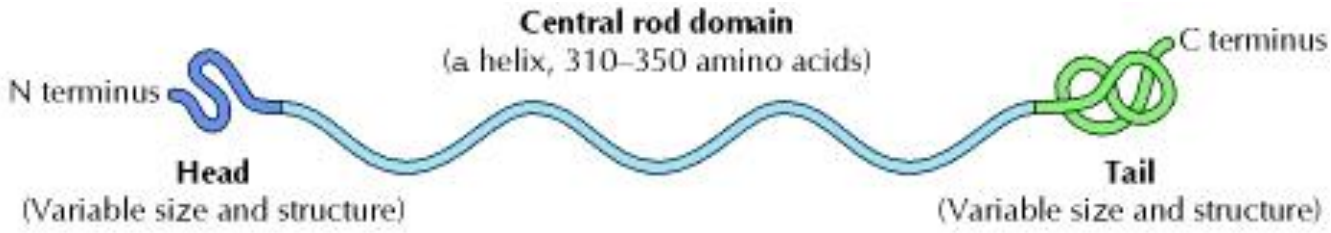


LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 5.14 (Part 2)

Table 11.1 Intermediate Filament Proteins

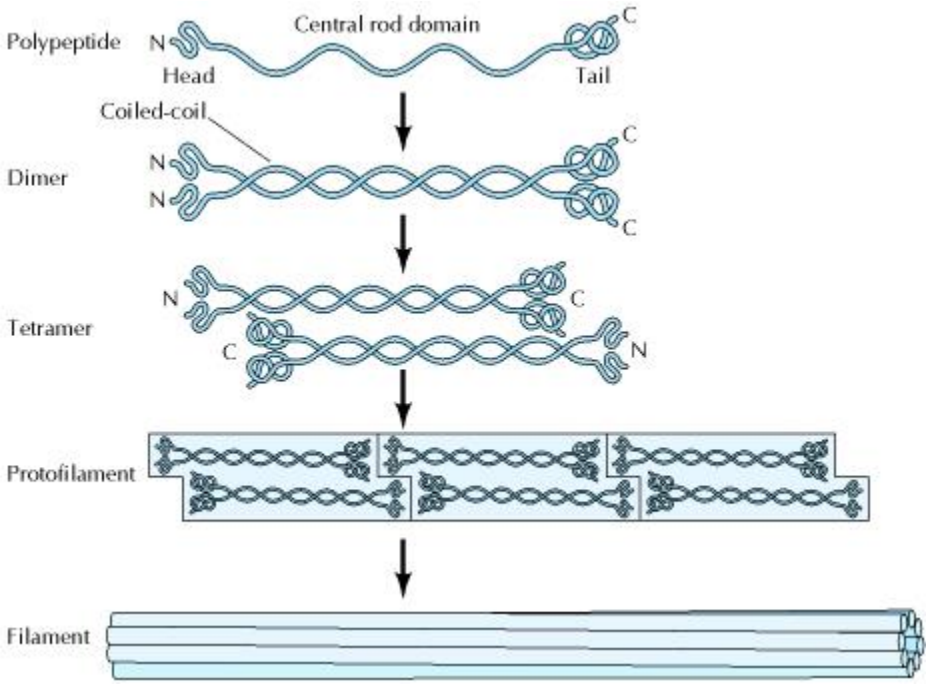
Type	Protein	Size (kd)	Site of expression
I	Acidic keratins	40–60	Epithelial cells
	(~15 proteins)		
II	Neutral or basic keratins	50–70	Epithelial cells
	(~15 proteins)		
III	Vimentin	54	Fibroblasts, white blood cells, and other cell types
	Desmin	53	Muscle cells
	Glial fibrillary acidic protein	51	Glial cells
	Peripherin	57	Peripheral neurons
IV	Neurofilament proteins		
	NF-L	67	Neurons
	NF-M	150	Neurons
	NF-H	200	Neurons
	α-Internexin	66	Neurons
V	Nuclear lamins	60–75	Nuclear lamina of all cell types
VI	Nestin	200	Stem cells of central nervous system

More than 50 different intermediate filament proteins have been identified and classified into six groups based on similarities between their amino acid sequences (Table 11.1). Types I and II consist of two groups of keratins, each consisting of about 15 different proteins, which are expressed in epithelial cells. The type III intermediate filament proteins include **vimentin**, which is found in a variety of different kinds of cells, including fibroblasts, smooth muscle cells, and white blood cells. The type IV intermediate filament proteins include the three **neurofilament (NF) proteins**. These proteins form the major intermediate filaments of many types of mature neurons. The type V intermediate filament proteins are the nuclear lamins, which are found in most eukaryotic cells. Rather than being part of the cytoskeleton, the nuclear lamins are components of the nuclear envelope.



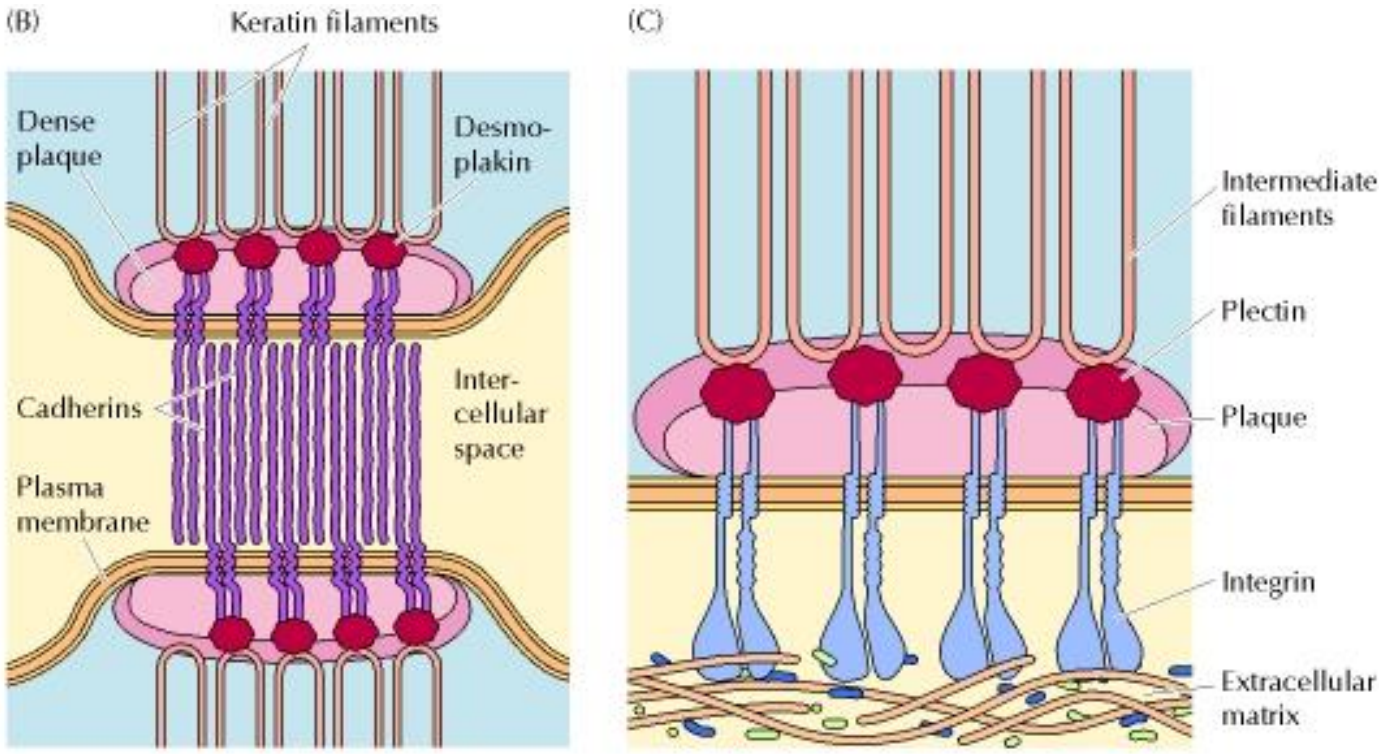
Structure of intermediate filament proteins

Intermediate filament proteins contain a central α-helical rod domain of approximately 310 amino acids (350 amino acids in the nuclear lamins). The N-terminal head and C-terminal tail domains vary in size and shape.



Assembly of intermediate filaments

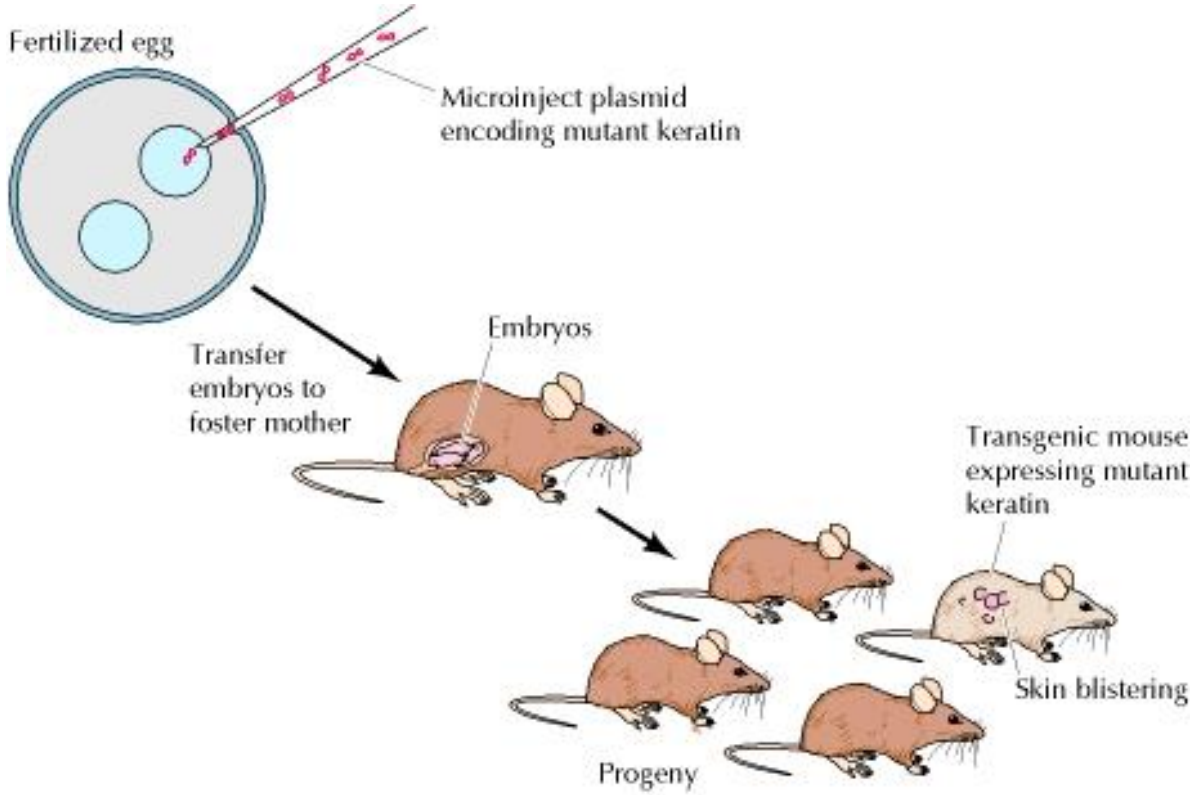
The central rod domains of two polypeptides wind around each other in a coiled-coil structure to form dimers. Dimers then associate in a staggered antiparallel fashion to form tetramers. Tetramers associate end to end to form protofilaments and laterally to form filaments. Each filament contains approximately eight protofilaments wound around each other in a ropelike structure.



Attachment of intermediate filaments to desmosomes and hemidesmosomes
(B) Schematic of a desmosome. Intermediate filaments are anchored to sites of cell-cell adhesion by desmoplaskin. (C) Schematic of a hemidesmosome. Intermediate filaments are anchored to an integrin by plectin. (A, Don Fawcett/Photo Researchers, Inc.)

In addition to linking intermediate filaments to cell junctions, some plakins link intermediate filaments to other elements of the cytoskeleton. Plectin, for example, binds actin filaments and microtubules in addition to intermediate filaments, so it can provide bridges between these cytoskeletal components (Figure 11.35). These bridges to intermediate filaments are thought to brace and stabilize actin filaments and microtubules, thereby increasing the mechanical stability of the cell.

Two types of intermediate filaments, desmin and the neurofilaments, play specialized roles in muscle and nerve cells, respectively. Desmin connects the individual actin-myosin assemblies of muscle cells both to one another and to the plasma membrane, thereby linking the actions of individual contractile elements. Neurofilaments are the major intermediate filaments in most mature neurons. They are particularly abundant in the long axons of motor neurons, where they appear to be anchored to actin filaments and microtubules by neuronal members of the plakin family. Neurofilaments are thought to play an important role in providing mechanical support and stabilizing other elements of the cytoskeleton in these long, thin extensions of nerve cells.



Experimental demonstration of keratin function

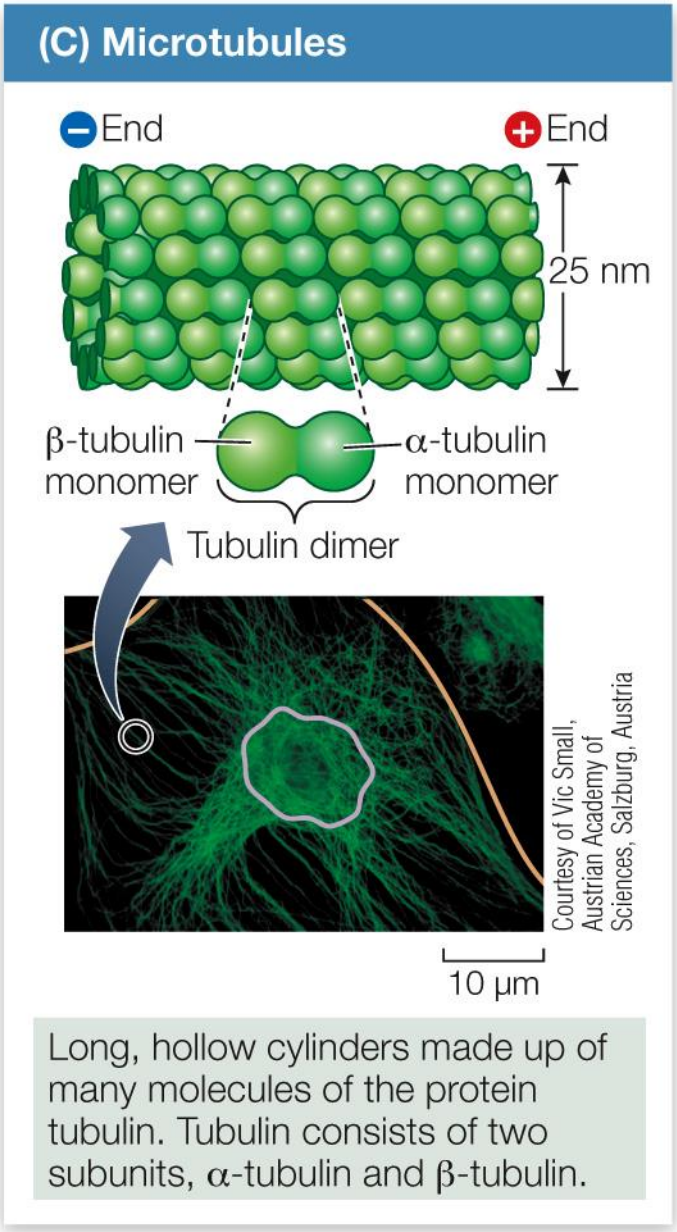
A plasmid encoding a mutant keratin that interferes with the normal assembly of keratin filaments was microinjected into one pronucleus of a fertilized egg. Microinjected embryos were then transferred to a foster mother, and some of the offspring were found to have incorporated the mutant keratin gene into their genome. Expression of the mutant gene in these transgenic mice disrupted the keratin cytoskeleton of cells of the epidermis, resulting in severe skin blistering due to cell lysis following mild mechanical stress.

microtubules

Microtubules:

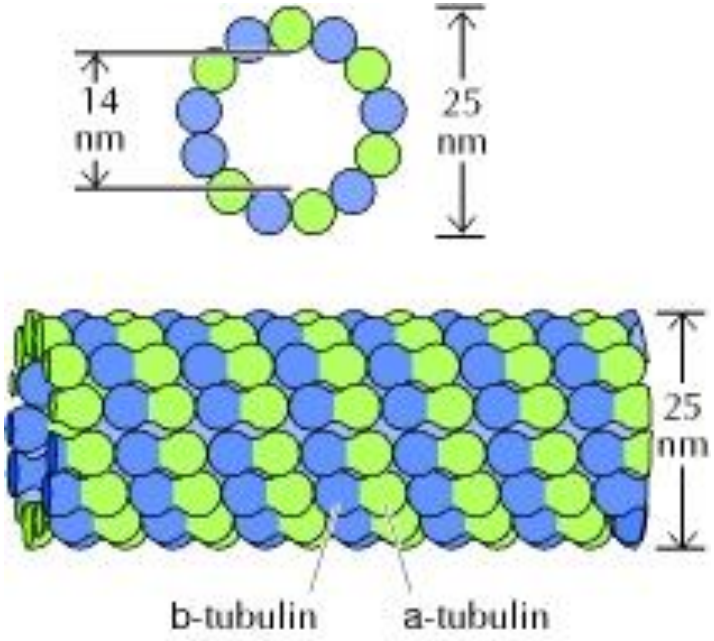
- Long, hollow cylinders
- Form rigid internal skeleton in some cells
- Act as a framework for motor proteins
- Made from the protein **tubulin—a** dimer
- Can change length rapidly by adding or losing dimers

Figure 5.14 The Cytoskeleton (Part 3)



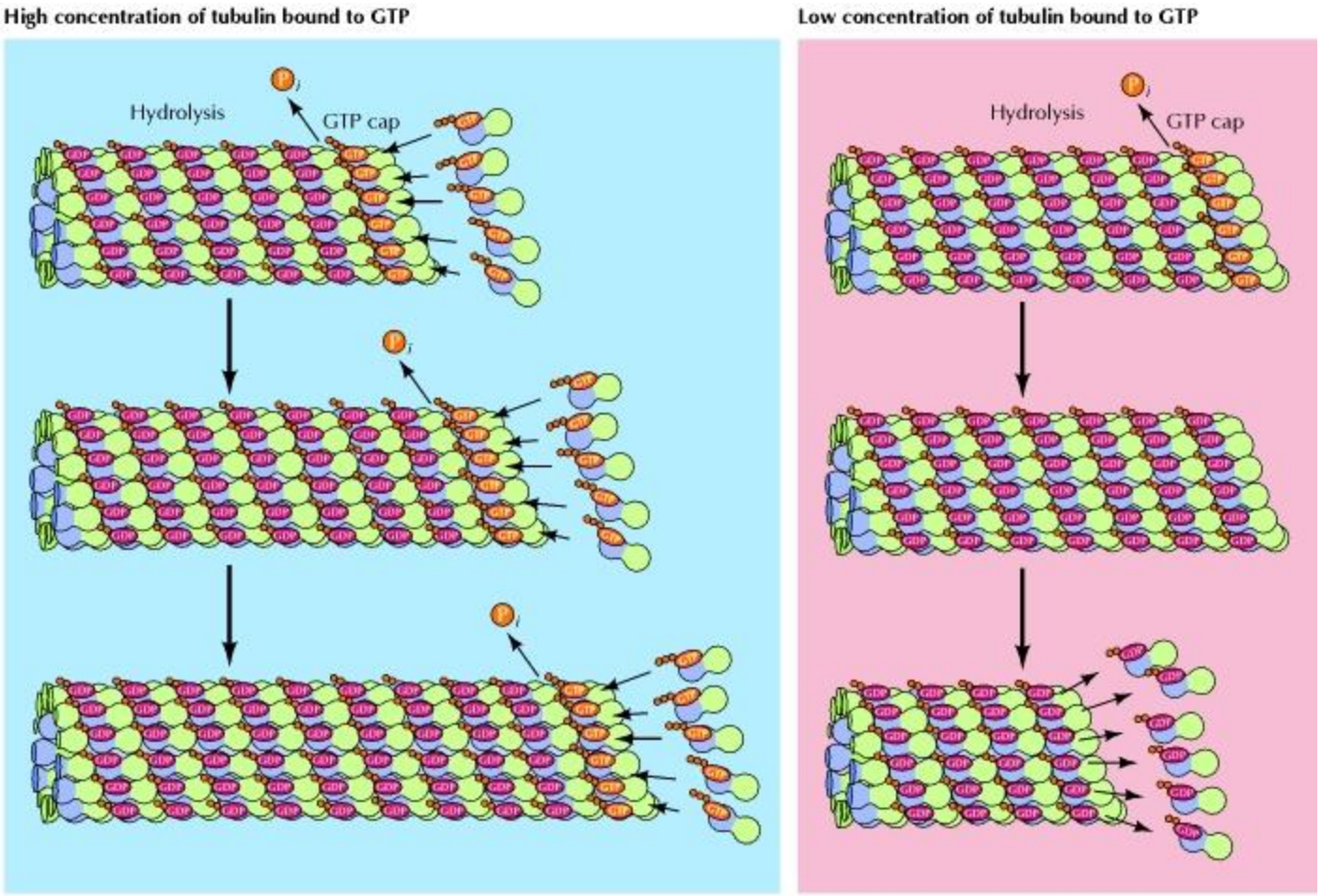
Long, hollow cylinders made up of many molecules of the protein tubulin. Tubulin consists of two subunits, α-tubulin and β-tubulin.

LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 5.14 (Part 3)



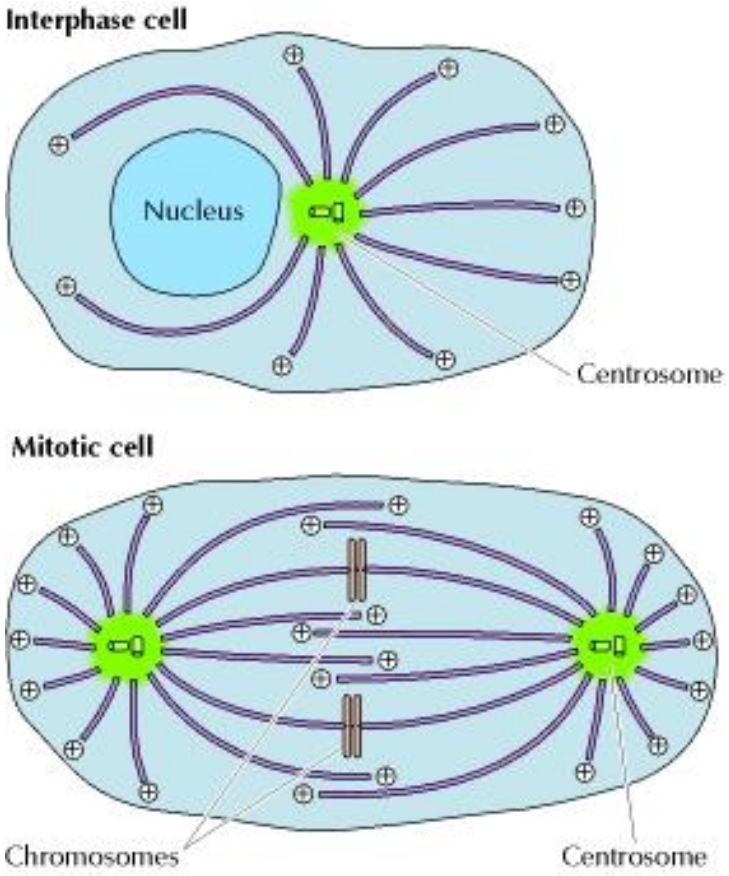
Structure of microtubules

Dimers of α- and β-tubulin polymerize to form microtubules, which are composed of 13 protofilaments assembled around a hollow core.



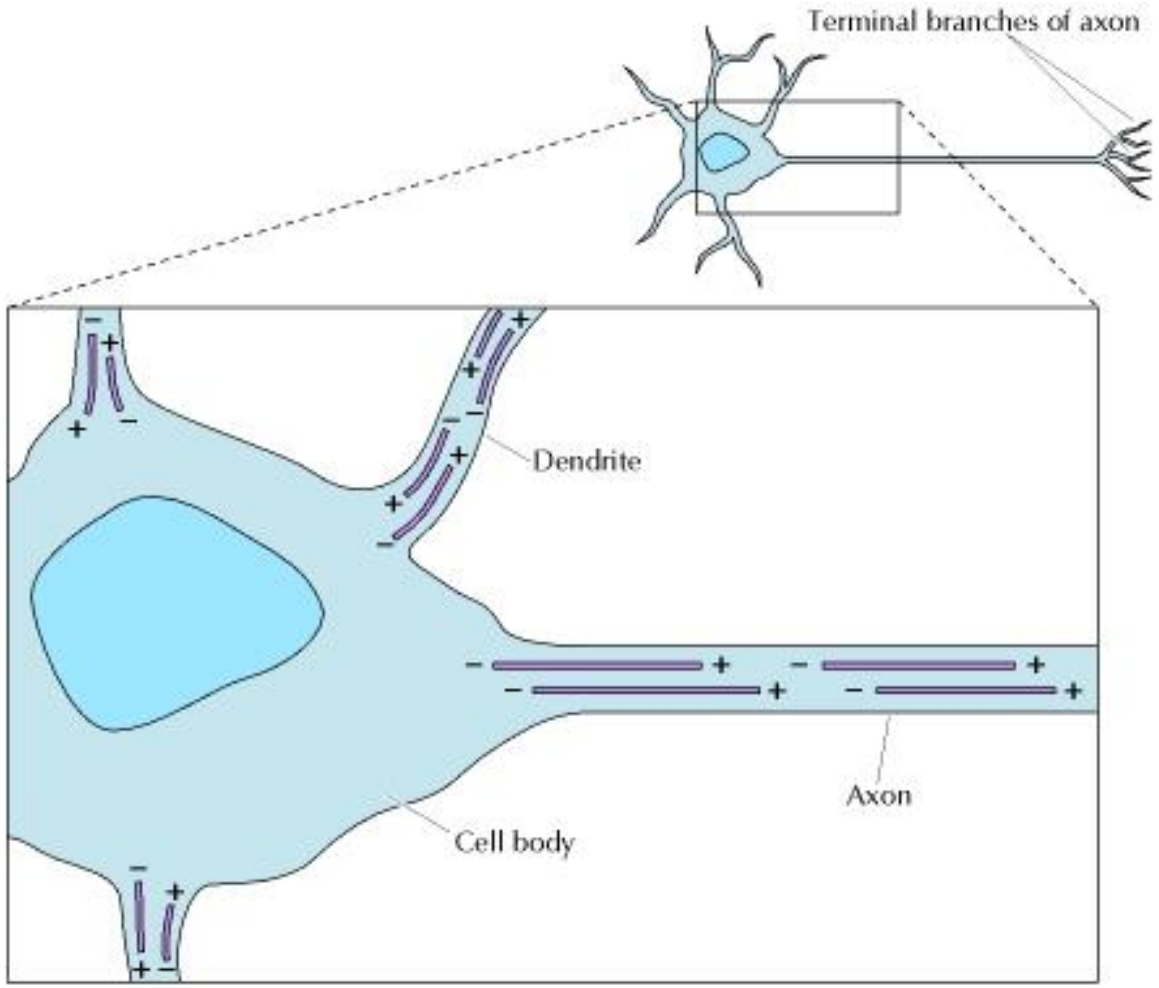
Dynamic instability of microtubules

Dynamic instability results from the hydrolysis of GTP bound to β-tubulin during or shortly after polymerization, which reduces its binding affinity for adjacent molecules. Growth of microtubules continues as long as there is a high concentration of tubulin bound to GTP. New GTP-bound tubulin molecules are then added more rapidly than GTP is hydrolyzed, so a GTP cap is retained at the growing end. However, if GTP is hydrolyzed more rapidly than new subunits are then added, the presence of GDP-bound tubulin at the end of the microtubule leads to disassembly and shrinkage. Only the plus ends of microtubules are illustrated.



Intracellular organization of microtubules

The minus ends of microtubules are anchored in the centrosome. In interphase cells, the centrosome is located near the nucleus and microtubules extend outward to the cell periphery. During mitosis, duplicated centrosomes separate and microtubules reorganize to form the mitotic spindle.



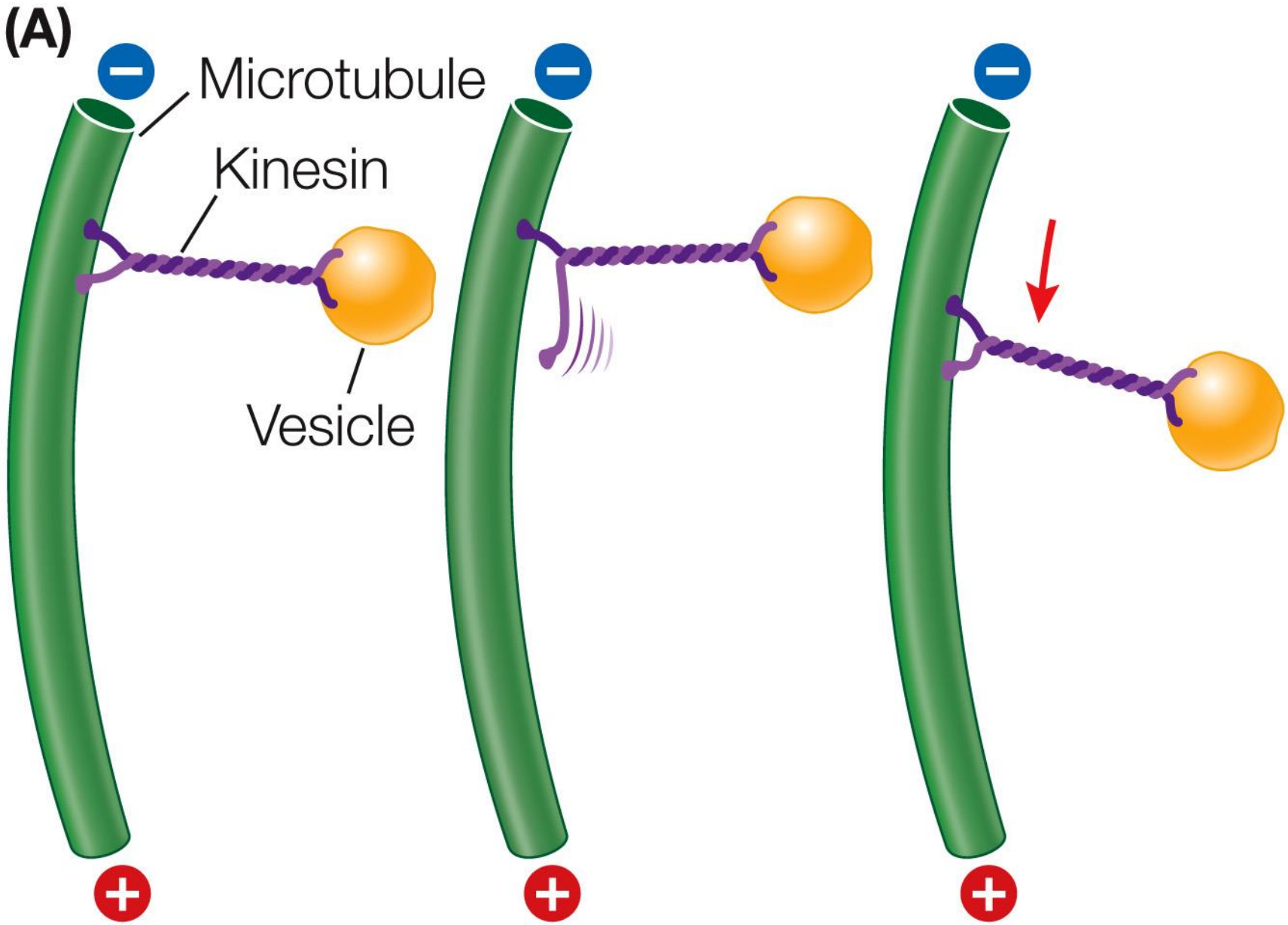
Organization of microtubules in nerve cells

Two distinct types of processes extend from the cell body of nerve cells (neurons). Dendrites are short processes that receive stimuli from other nerve cells. The single long axon then carries impulses from the cell body to other cells, which may be either other neurons or an effector cell, such as a muscle. Stable microtubules in both axons and dendrites terminate in the cytoplasm rather than being anchored in the centrosome. In dendrites, microtubules are oriented in both directions, with their plus ends pointing both toward and away from the cell body. In contrast, all of the axon microtubules are oriented with their plus ends pointing toward the tip of the axon.

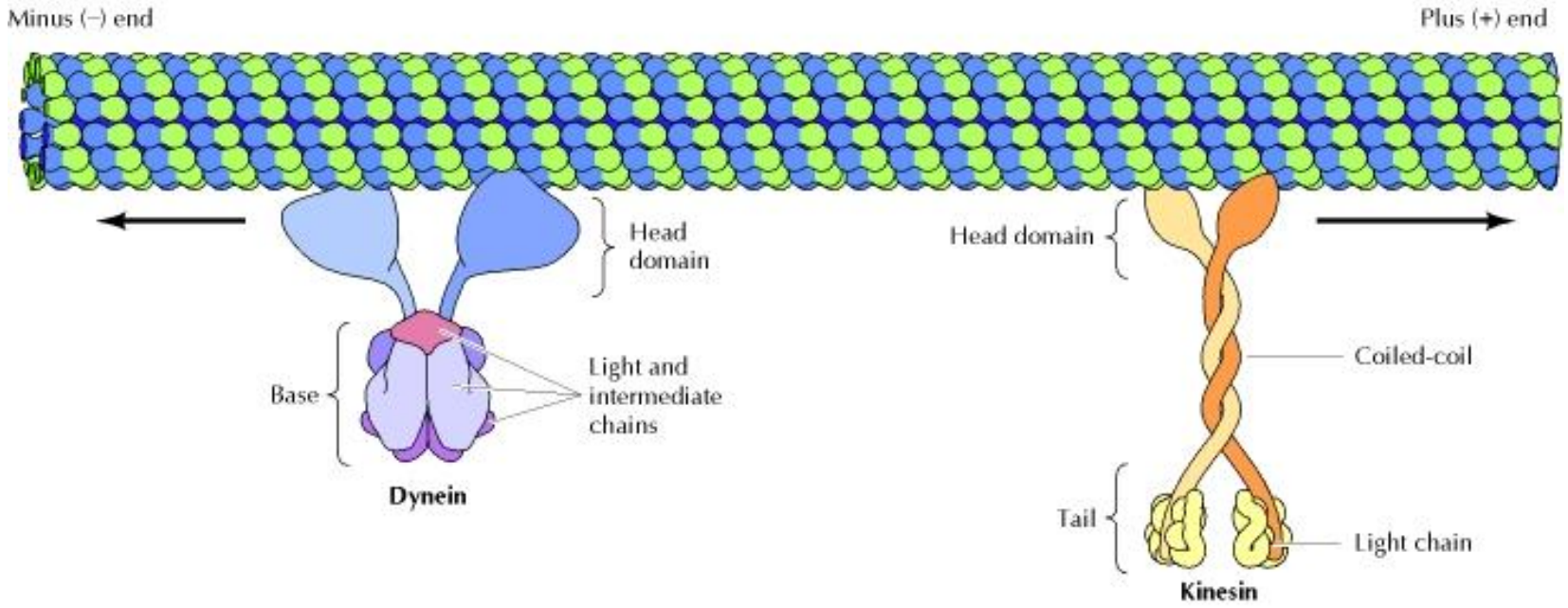
The motor protein kinesin moves vesicles or organelles from one part of a cell to another.

It binds to a vesicle and “walks” it along by changing shape.

Figure 5.19 A Motor Protein Pulls Vesicles along Microtubules (Part 1)

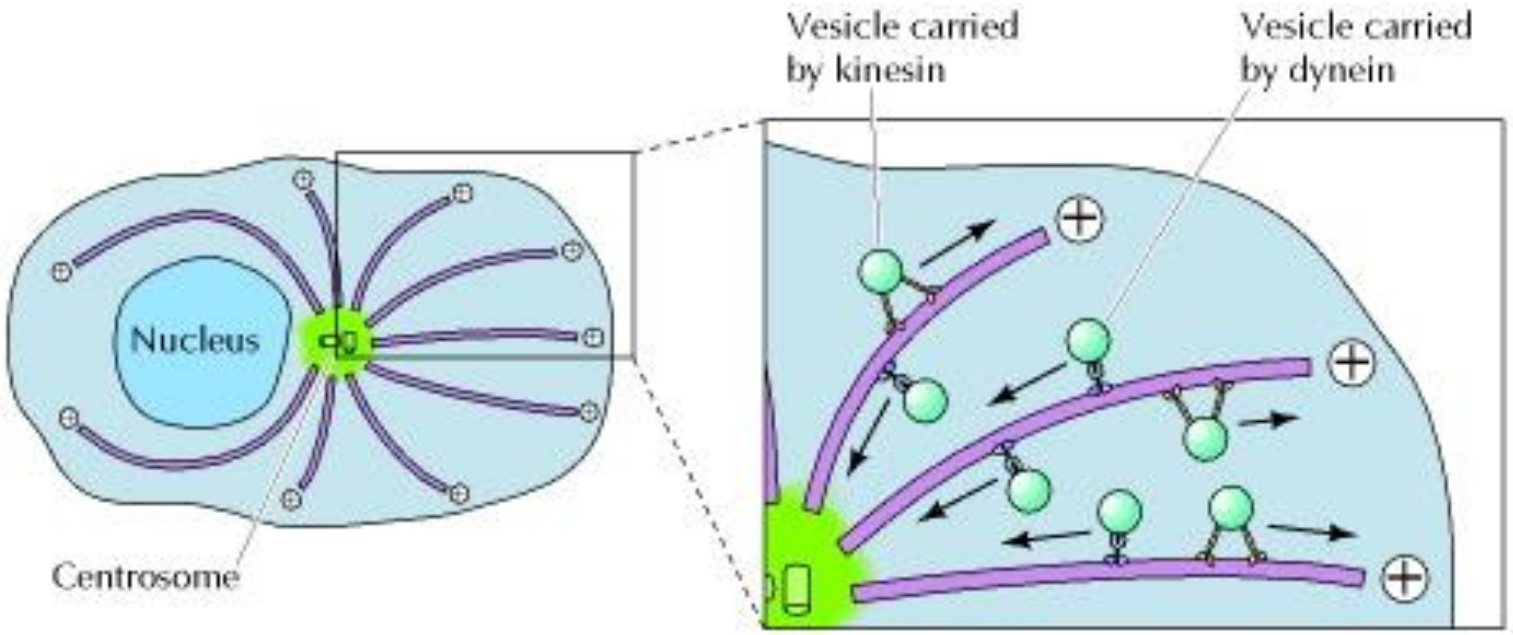


LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 5.19 (Part 1)



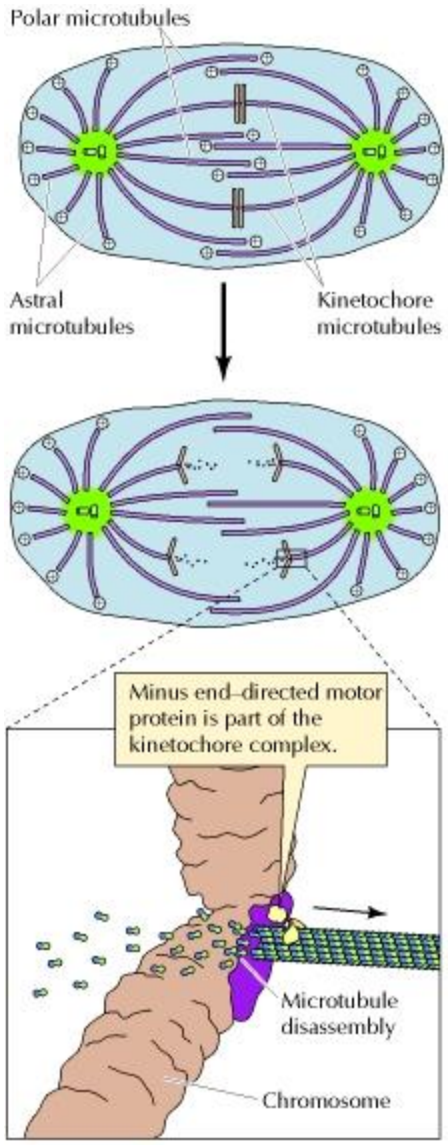
Microtubule motor proteins

Kinesin and dynein move in opposite directions along microtubules, toward the plus and minus ends, respectively. Kinesin consists of two heavy chains, wound around each other in a coiled-coil structure, and two light chains. The globular head domains of the heavy chains bind microtubules and are the motor domains of the molecule. Dynein consists of two or three heavy chains (two are shown here) in association with multiple light and intermediate chains. The globular head domains of the heavy chains are the motor domains.

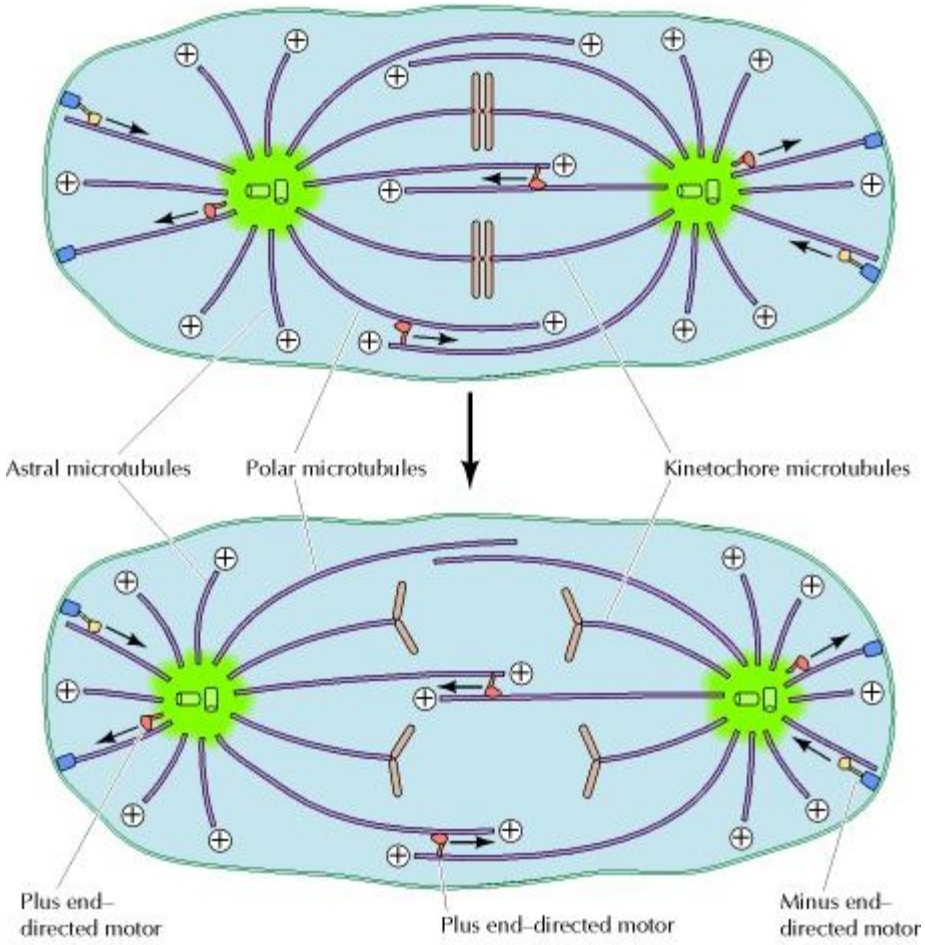


Transport of vesicles along microtubules

Kinesin and other plus end-directed members of the kinesin family transport vesicles and organelles in the direction of microtubule plus ends, which extend toward the cell periphery. In contrast, dynein and minus end-directed members of the kinesin family carry their cargo in the direction of microtubule minus ends, which are anchored in the center of the cell.



Anaphase A chromosome movement
Chromosomes move toward the spindle poles along the kinetochore microtubules. Chromosome movement is thought to be driven by minus end-directed motor proteins associated with the kinetochore. The action of these motor proteins is coupled to disassembly and shortening of the kinetochore microtubules.



Spindle pole separation in anaphase B

The separation of spindle poles results from two types of movement. First, overlapping polar microtubules slide past each other to push the spindle poles apart, probably as a result of the action of plus end-directed motor proteins. Second, the spindle poles are pulled apart by the astral microtubules. The driving force could be either a minus end-directed motor anchored to a cytoplasmic structure, such as the cell cortex, or a plus end-directed motor associated with the spindle pole.

Cilia and **flagella** are microtubule-based projections of the plasma membrane that are responsible for movement of a variety of eukaryotic cells. Many bacteria also have flagella, but these prokaryotic flagella are quite different from those of eukaryotes. Bacterial flagella (which are not discussed further here) are protein filaments projecting from the cell surface, rather than projections of the plasma membrane supported by microtubules.

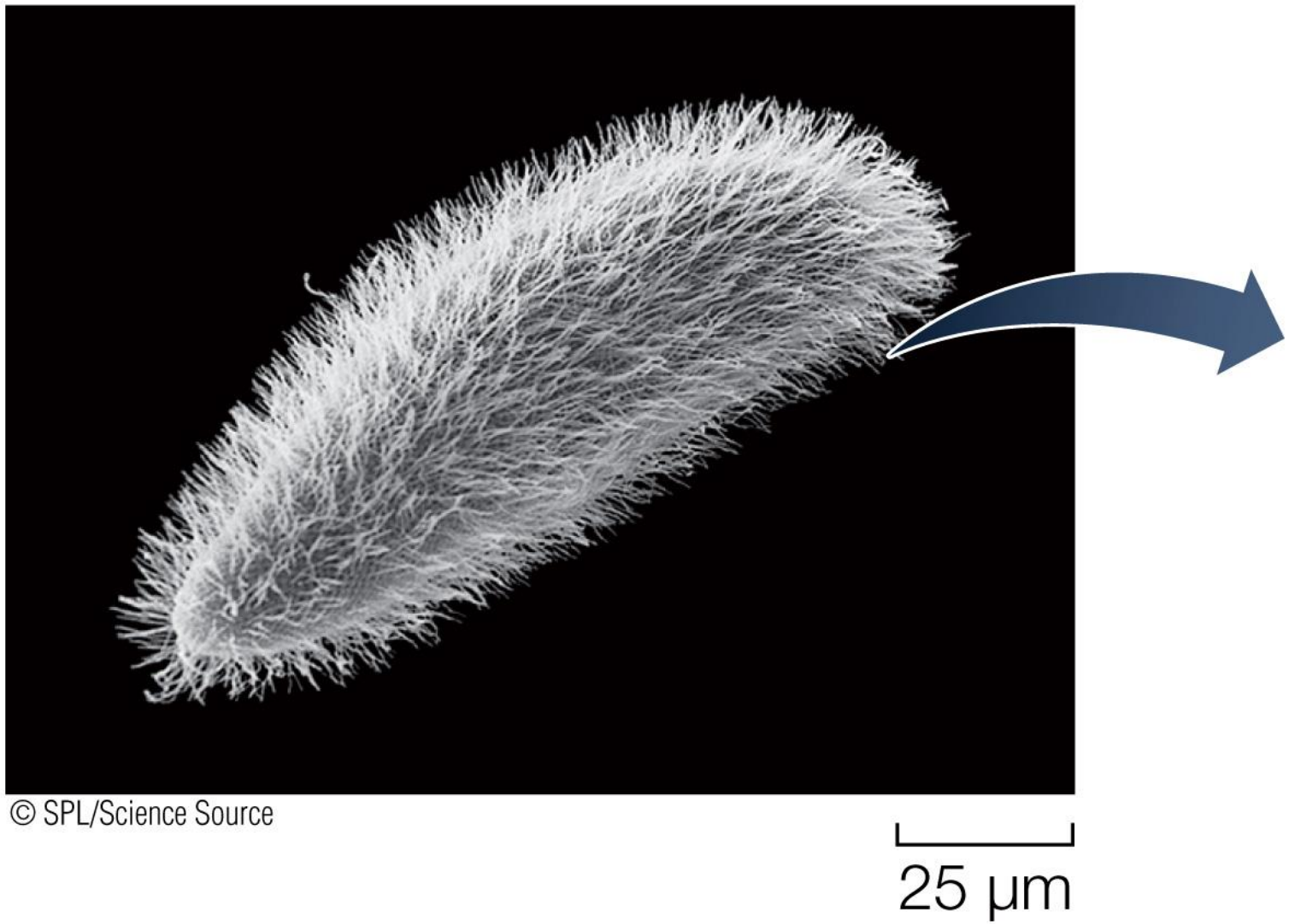
Eukaryotic cilia and flagella are very similar structures, each with a diameter of approximately 0.25 μm (Figure 11.50). Many cells are covered by numerous cilia, which are about 10 μm in length. Cilia beat in a coordinated back-and-forth motion, which either moves the cell through fluid or moves fluid over the surface of the cell. For example, the cilia of some protozoans (such as *Paramecium*) are responsible both for cell motility and for sweeping food organisms over the cell surface and into the oral cavity. In animals, an important function of cilia is to move fluid or mucus over the surface of epithelial cell sheets. A good example is provided by the ciliated cells lining the respiratory tract, which clear mucus and dust from the respiratory passages. Flagella differ from cilia in their length (they can be as long as 200 μm) and in their wavelike pattern of beating. Cells usually have only one or two flagella, which are responsible for the locomotion of a variety of protozoans and of sperm.

Cilia and eukaryotic **flagella** are made of microtubules in “9 + 2” array.

Cilia—short, hundreds on one cell, move stiffly to propel the cell or move fluid over a cell.

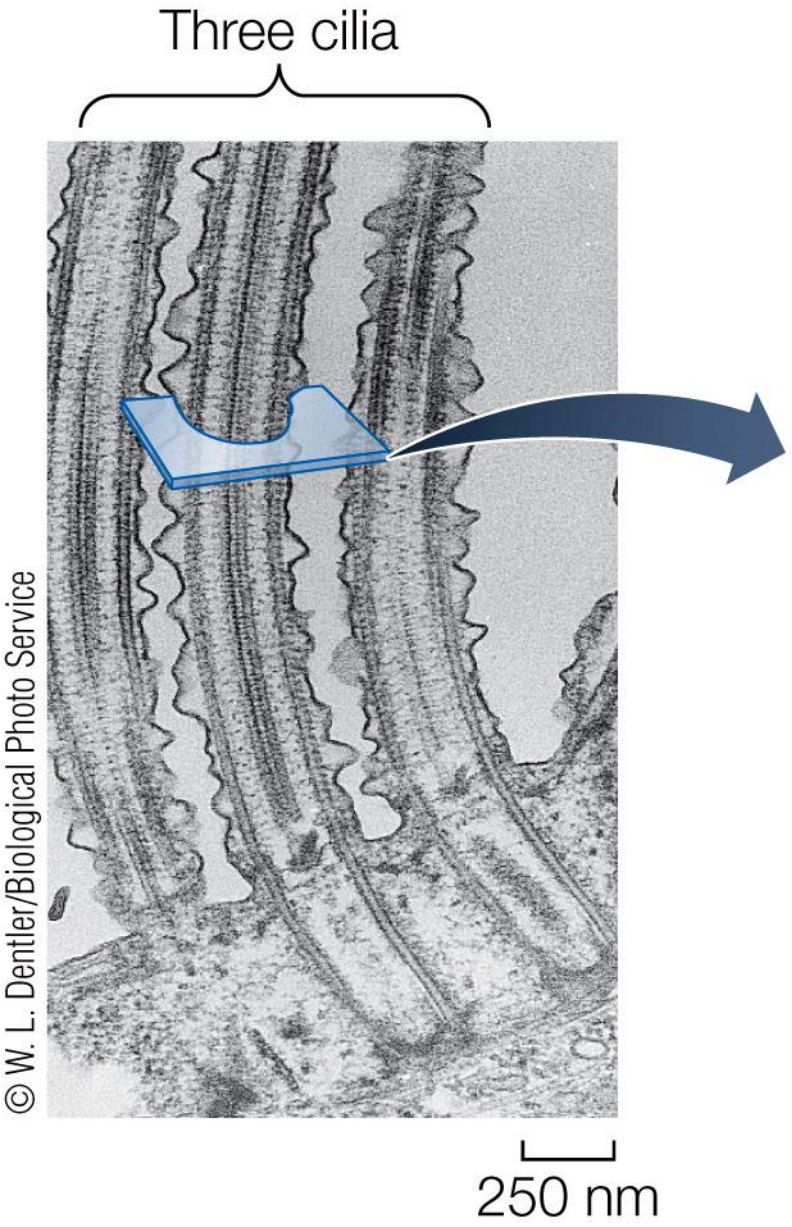
Flagella—longer, usually one or two present, movement is snakelike.

Figure 5.17 Cilia (Part 1)



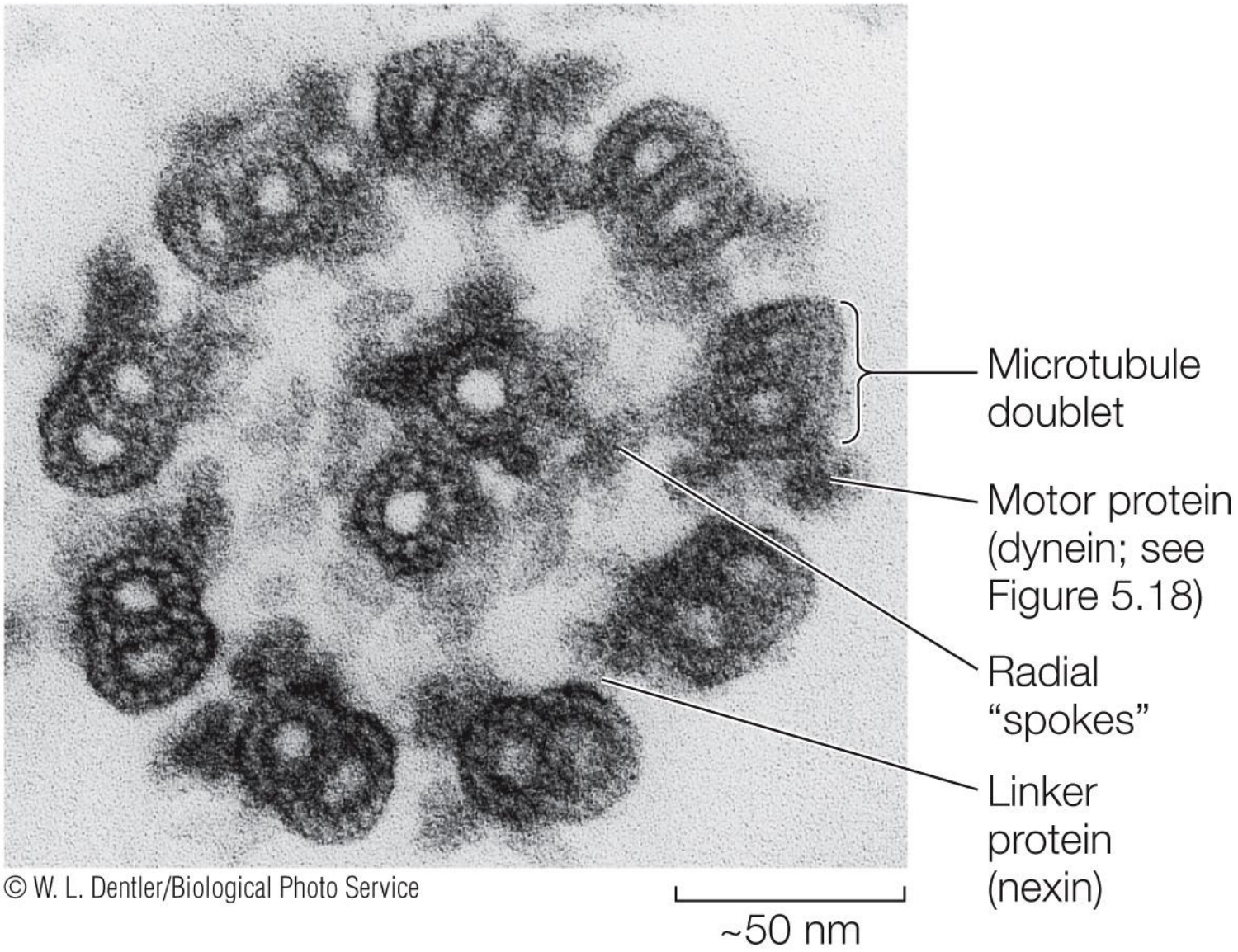
LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 5.17 (Part 1)

Figure 5.17 Cilia (Part 2)

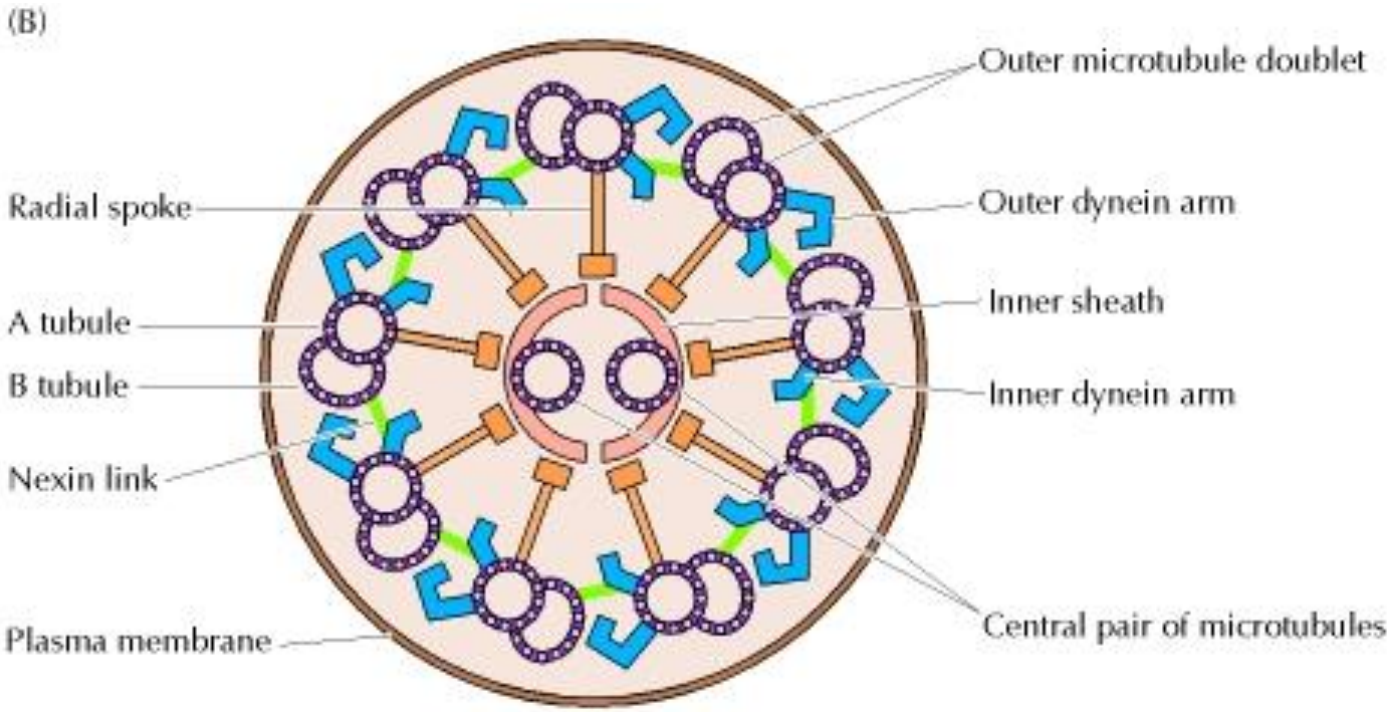


LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 5.17 (Part 2)

Figure 5.17 Cilia (Part 3)



LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 5.17 (Part 3)



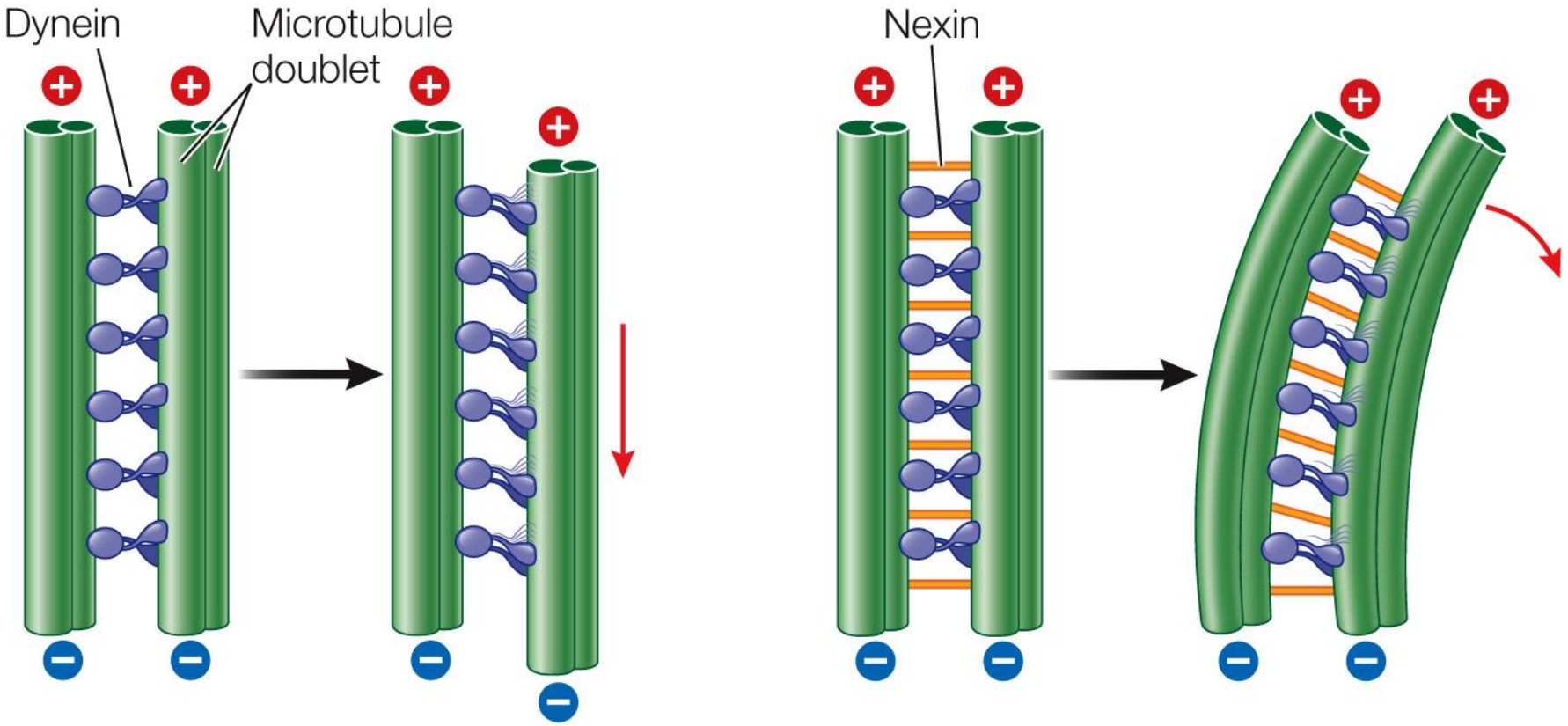
Structure of the axoneme of cilia and flagella

(B) Schematic cross section of an axoneme. The nine outer doublets consist of one complete (A) and one incomplete (B) microtubule, containing only 10 or 11 protofilaments. The outer doublets are joined to each other by nexin links and to the central pair of microtubules by radial spokes. Each outer microtubule doublet is associated with inner and outer dynein arms. (A, K. G. Murti/Visuals Unlimited.)

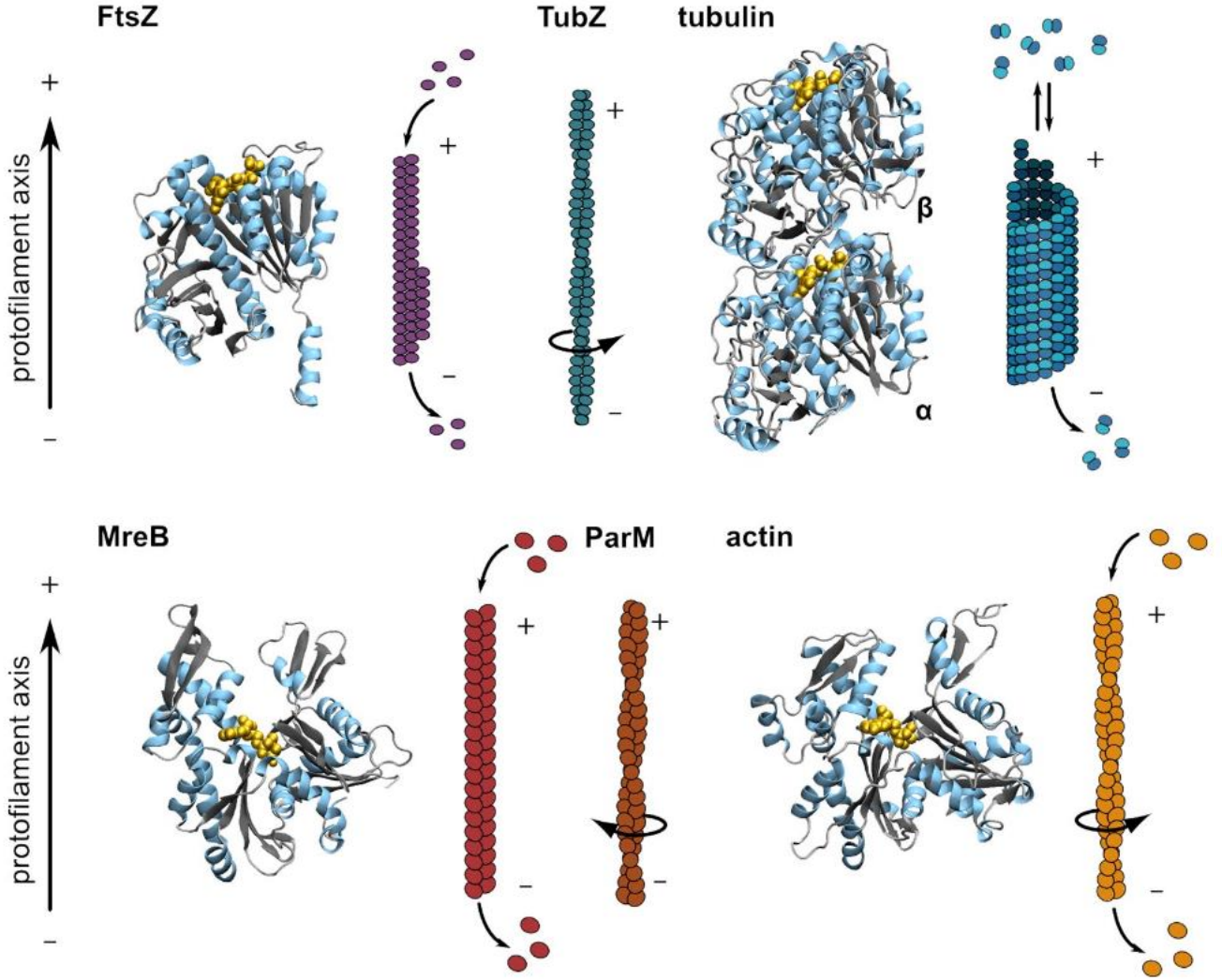
The motion of cilia and flagella:

- Dynein binds to microtubule doublets and allows them to slide past each other.
- Nexin cross-links the doublets and prevents them from sliding, and the cilium bends.

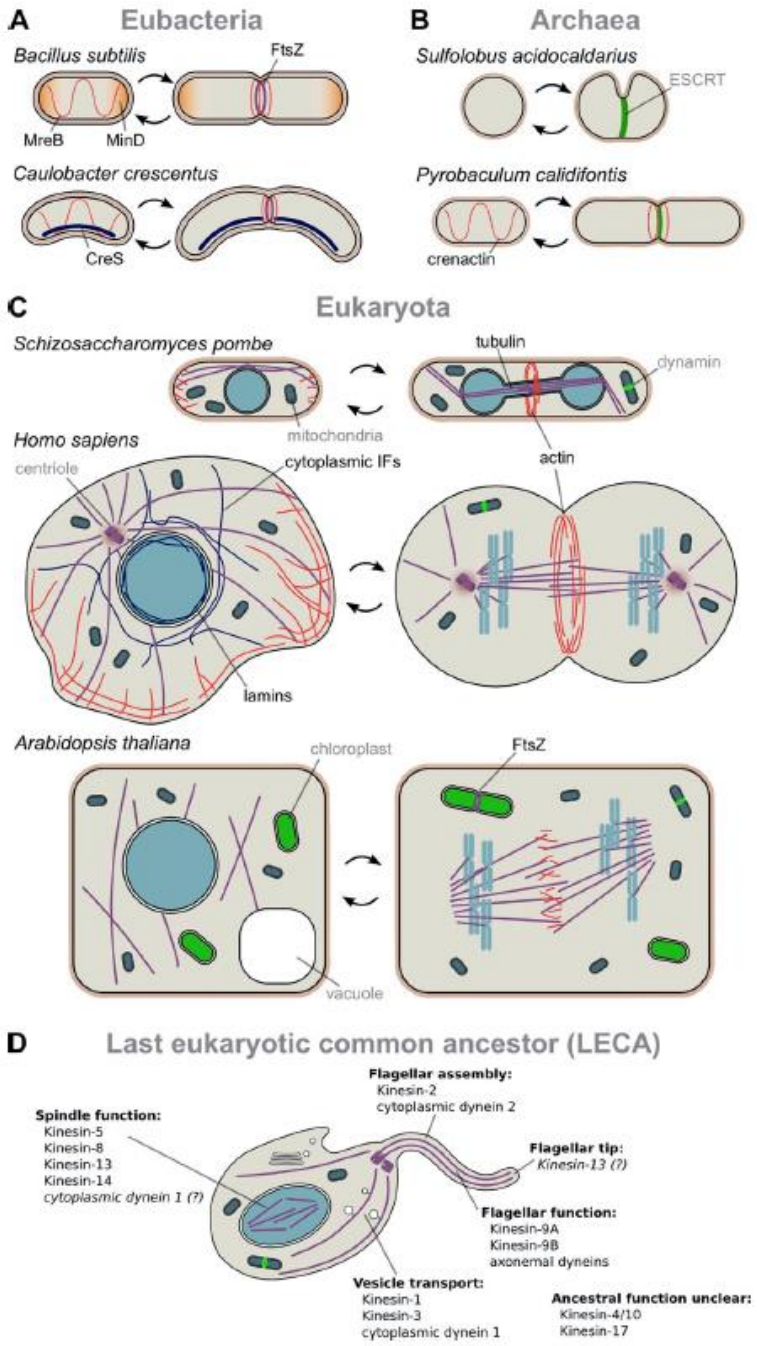
Figure 5.18 Motor Protein Moves Microtubules in Cilia and Flagella



LIFE: THE SCIENCE OF BIOLOGY 11e, Figure 5.18



Homology between prokaryotic and eukaryotic cytoskeletal filaments. Despite low levels of sequence similarity, the homologous cytoskeletal proteins FtsZ/TubZ/tubulin (top) and MreB/ParM/actin (bottom) have considerable conservation of folding and also longitudinal interaction. ParM and actin form similar helical filaments, but with opposite chirality (Orlova et al., 2007). Structures of filament subunits are derived from the following Protein Data Bank accession numbers: 1W5A (FtsZ; Oliva et al., 2004), 1JFF (α/β-tubulin; Löwe et al., 2001), 1JCG (MreB; van den Ent et al., 2001), and 1YAG (actin; Vorobiev et al., 2003).



Bacterial, archaeal, and eukaryotic cytoskeletons. Schematic representations are shown for a small number of model organisms from each of the three domains of life (A–C), showing the organization of the cytoskeleton in dividing and nondividing cells (right and left of each pair, respectively). Homologous filaments are colored similarly. Also shown is the possible organization of the cytoskeleton in the LECA (D), highlighting the ancestral families of microtubule motors.

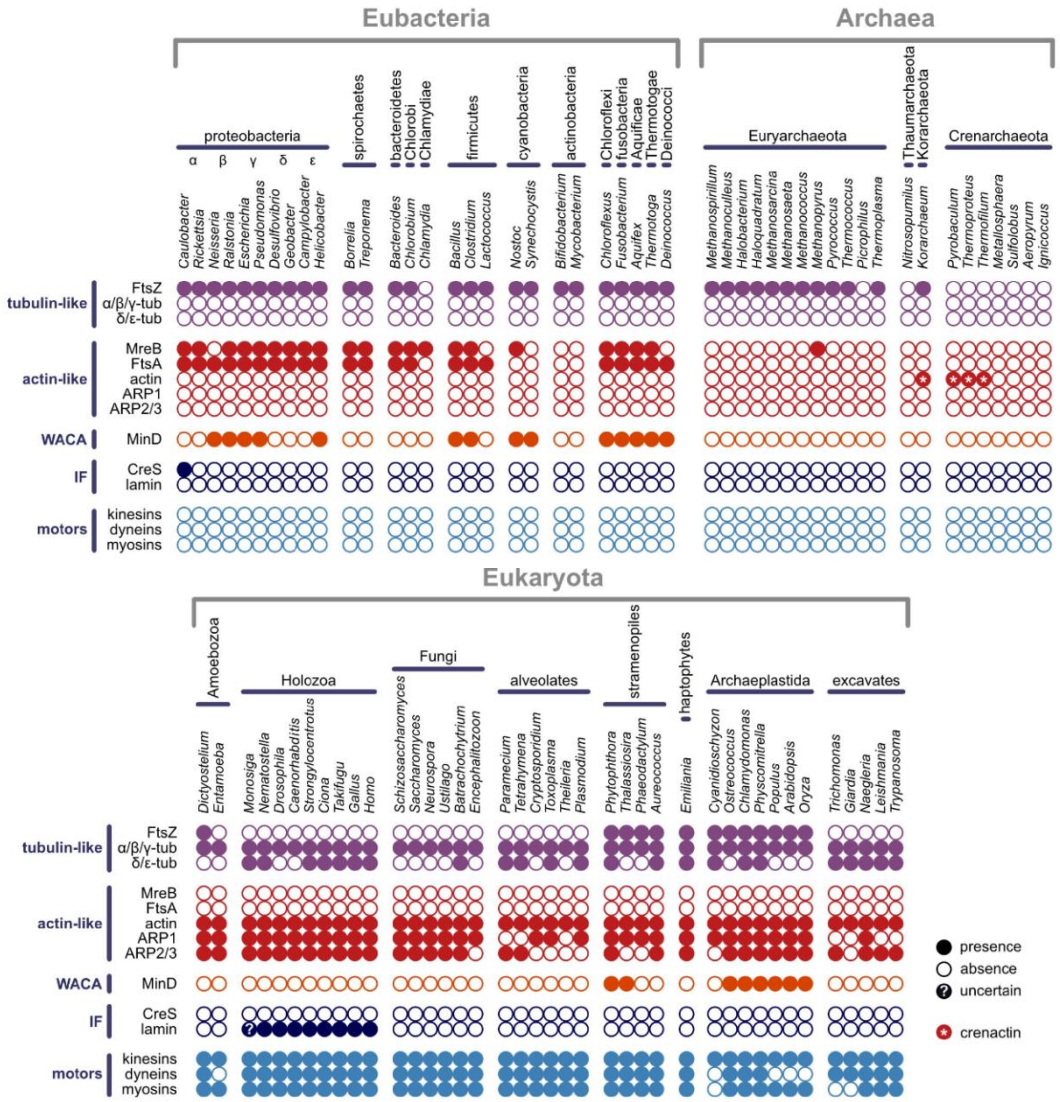


Figure 3. **The distribution of key components of the cytoskeleton across the tree of life.** Filled circle indicates presence of an identifiable member of a protein family in an organism; open circle indicates absence/not found. Organisms are identified by genera only and are grouped into higher taxonomic groups. *Emiliania huxleyi* has not been placed into one of the eukaryotic taxonomic groups in reflection of uncertainty as to the placement of Haptophyta. "MreB" includes MreB-like (Mbl/MreBH) sequences, which colocalize with MreB and are very similar in sequence (Carballido-López and Errington, 2003; Carballido-López et al., 2006). The archaeal sequences identified as MreB using the arCOG technique (arCOG04656; Makarova et al., 2007, 2010) have a closer affinity to Hsp70 sequences and are not included. Archaeal crenactin (*) is orthologous to the single common ancestor of eukaryotic actin and ARPs (Yutin et al., 2009; Ettema et al., 2011), but has been entered as actin for clarity. The distributions of the large number of prokaryotic actin-like proteins other than MreB and FtsA (such as AlfA, Alp6/7/8) are not included here because of current difficulties in resolution of individual families (Derman et al., 2009; Yutin et al., 2009). There are possible orthologues of MinD in Euryarchaeota (Leipe et al., 2002), but their true membership is still unclear.