

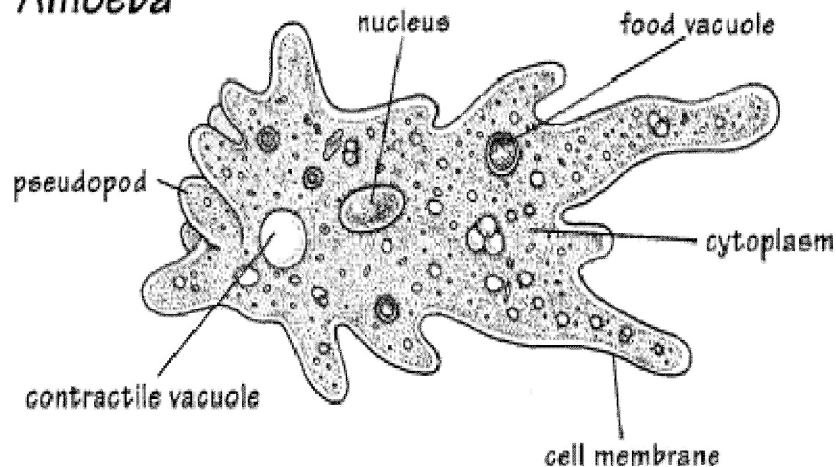
Locomotion in Protozoa

A. Amoeboid or Pseudopodial

B. Axonemal or Microfibrillar



Amoeba



Cilia and Flagella

□ Flagella

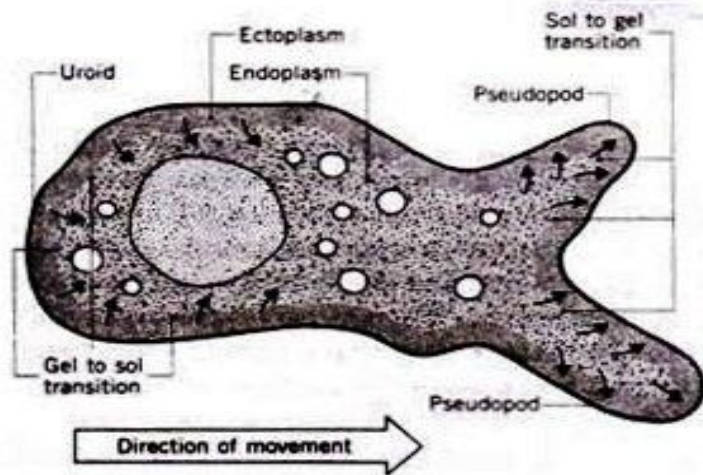
- Long, tail-like projection with a whiplike motion that helps a cell move through a watery environment

□ Cilia

- Short, numerous projections that look like hair and function in cell movement



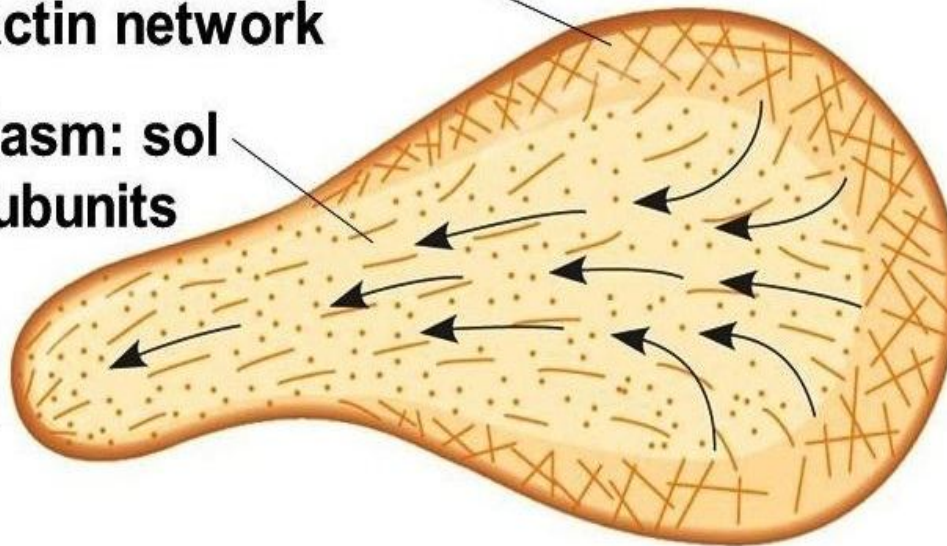
Amoeboid movement



Cortex (outer cytoplasm):
gel with actin network

Inner cytoplasm: sol
with actin subunits

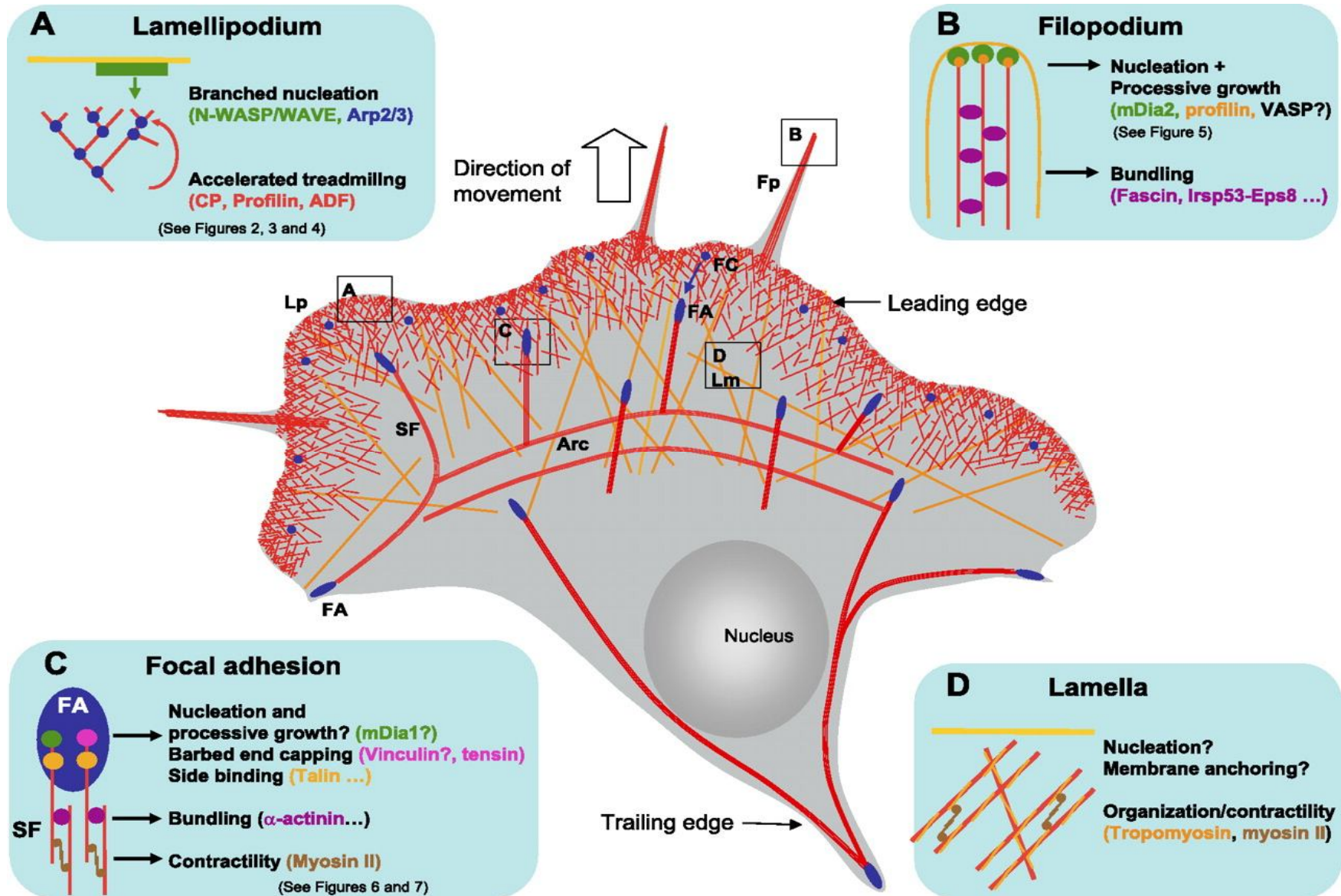
Extending
pseudopodium



Amoeboid movement

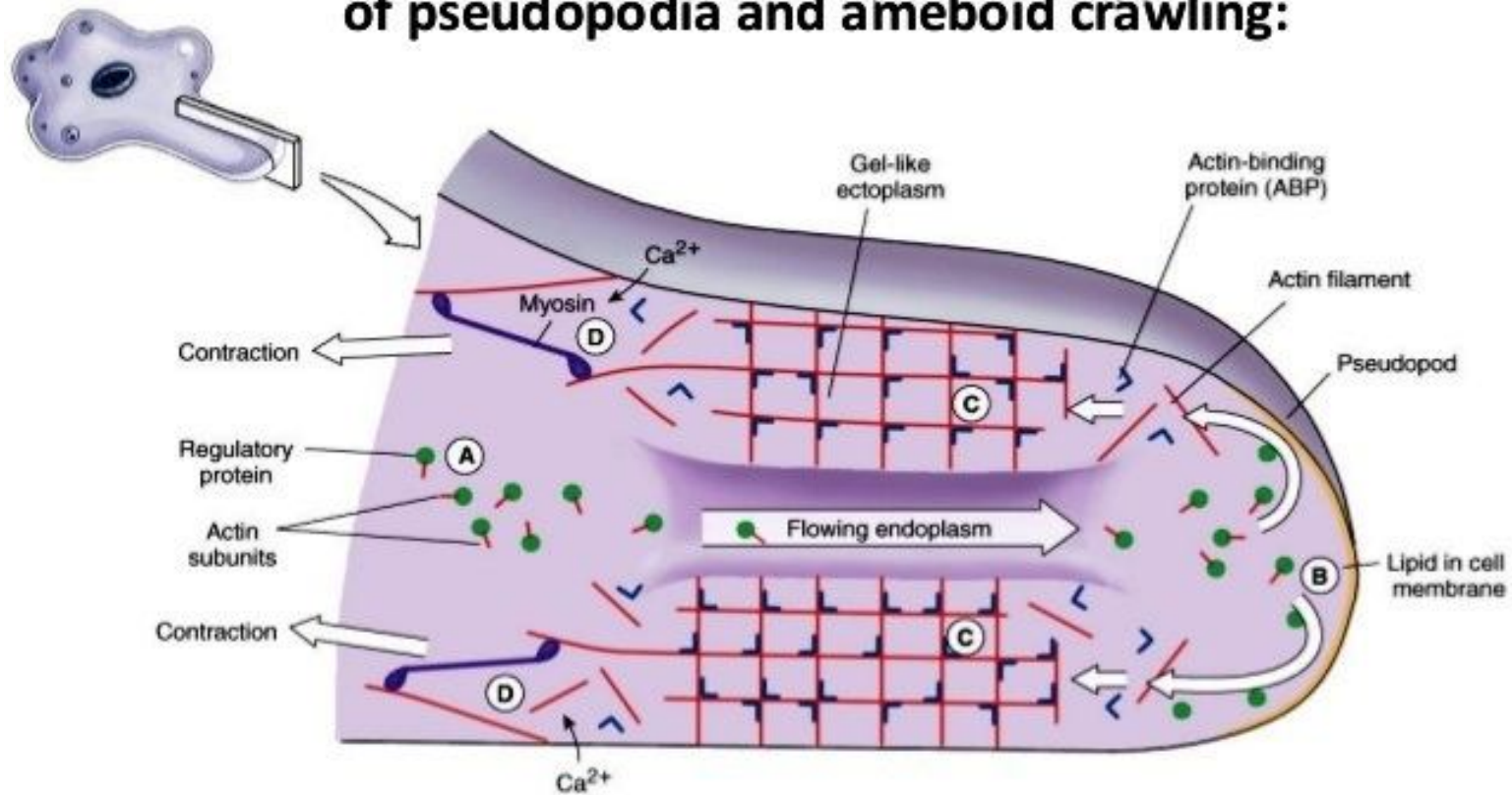
Cortex (outer cytoplasm) gel with **actin** network

Molecular Mechanism of Amoeboid Locomotion



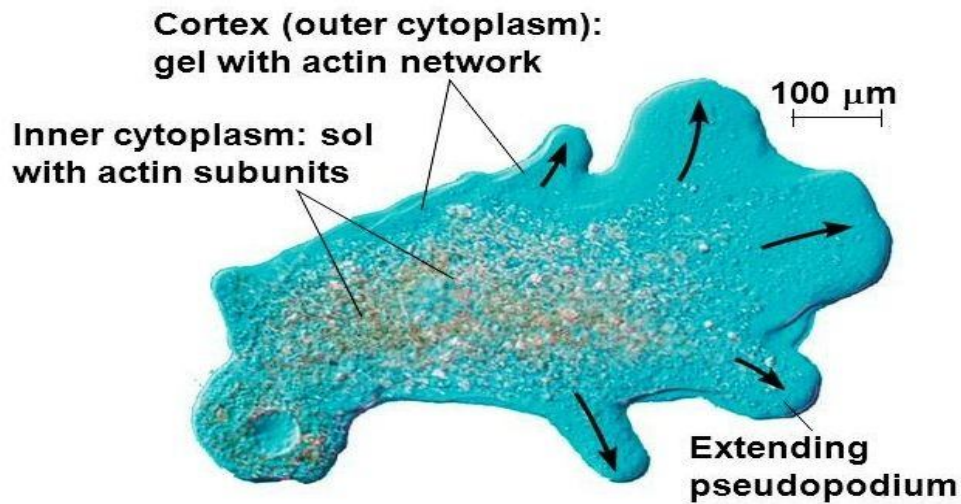
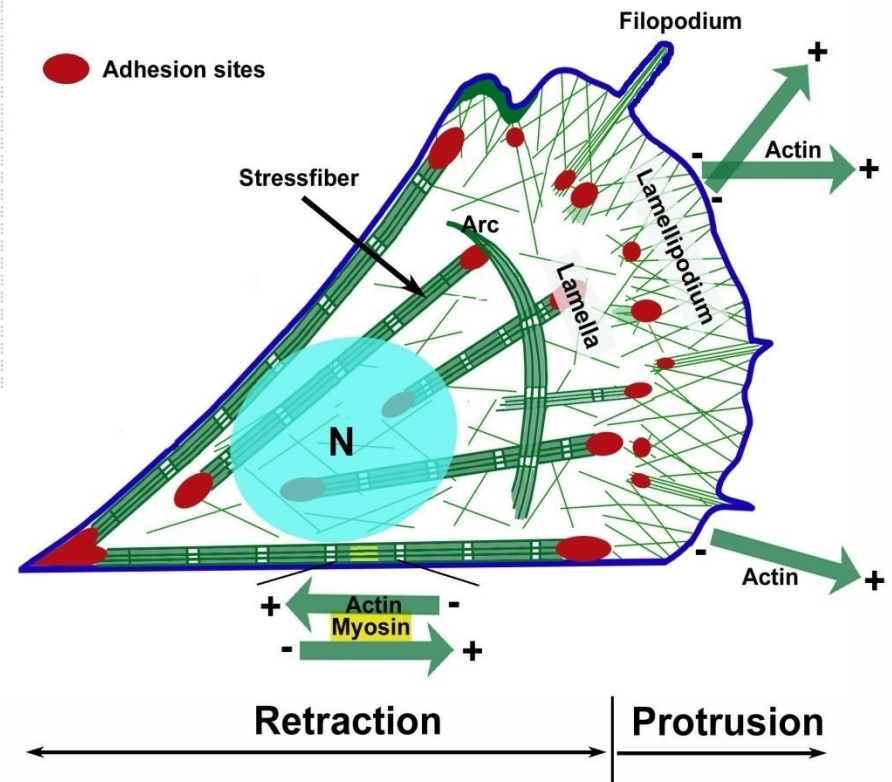
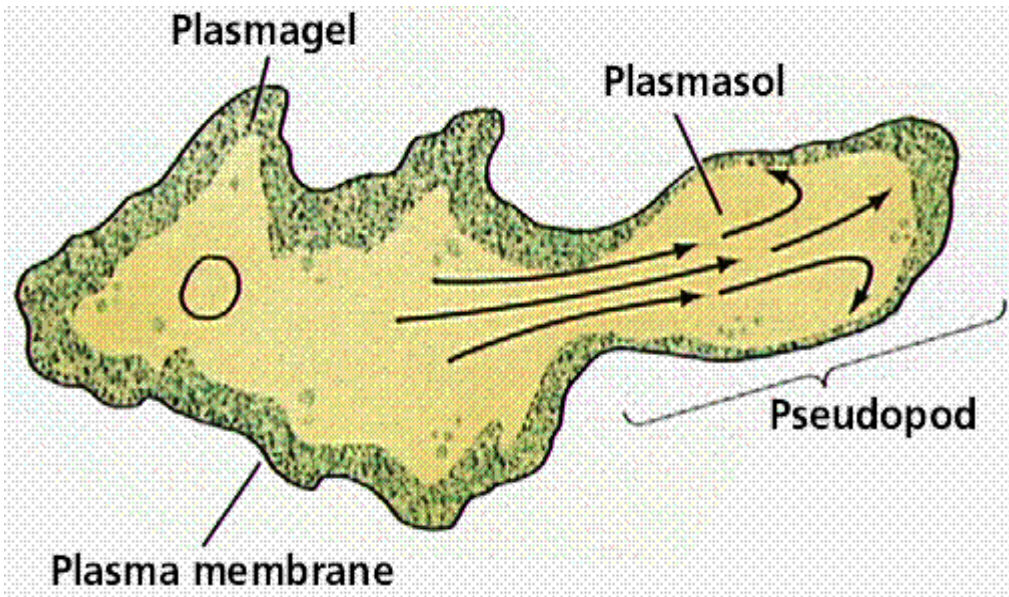
Role of Actin, Myosin & Ca-ion

Consensus model to explain extension and withdrawal of pseudopodia and ameboid crawling:



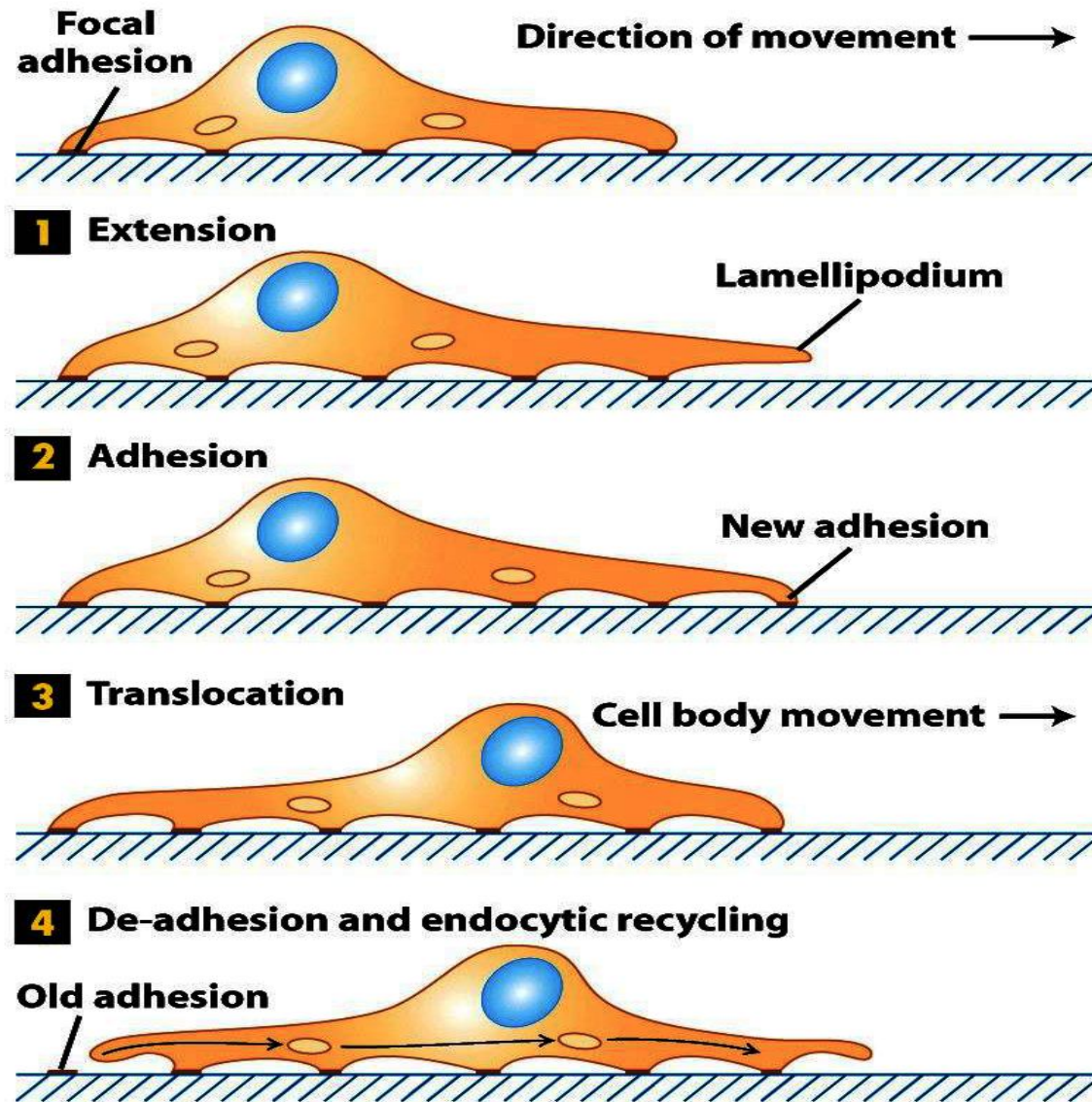
Ca²⁺ activate actin-severing protein

Amoeboid Locomotion : Mechanism

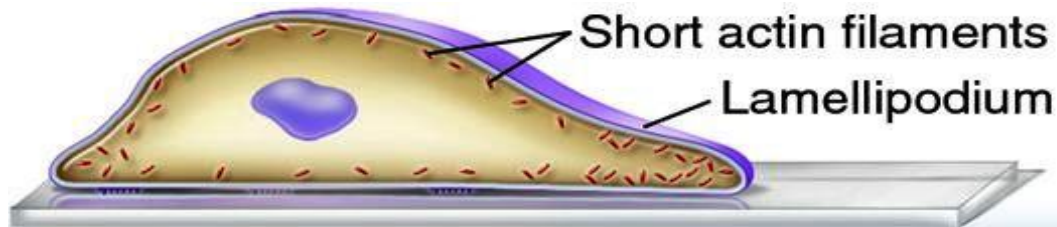


Amoeboid movement

Amoeboid Locomotion: a Model presentation

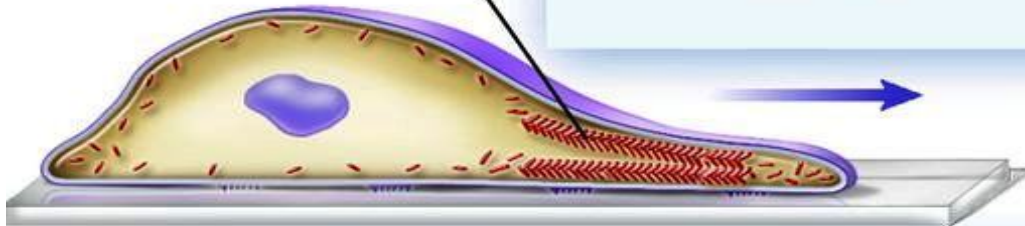


Molecular explanation of Pseudopodial locomotion

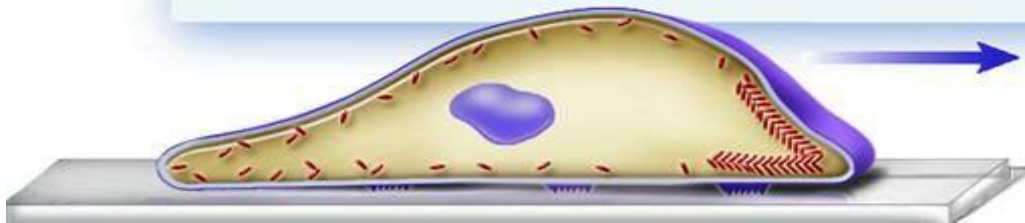


Actin polymerization

- 1 The formation of long actin filaments at the leading edge extends the lamellipodium.

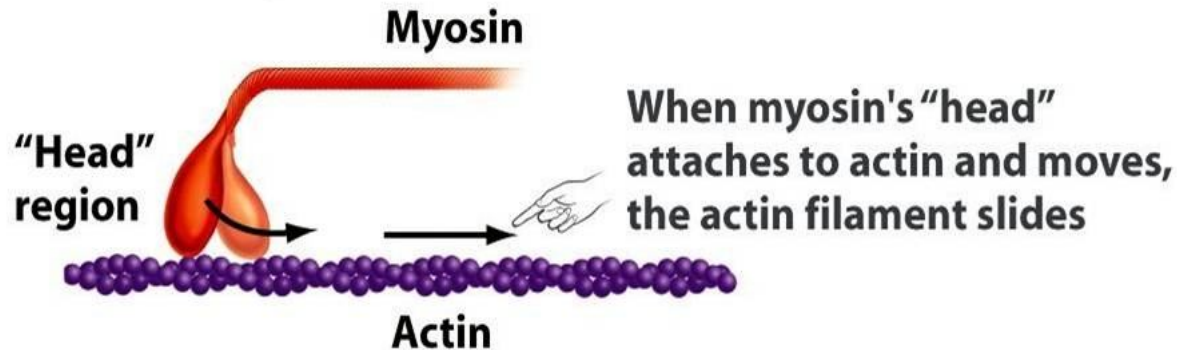


- 2 Rearrangement of the actin cytoskeleton in other regions of the cell causes the cell to be pulled toward the leading edge.



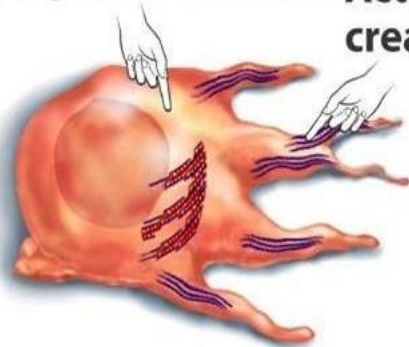
Actin & Myosin Interaction

(a) Actin and myosin interact to cause movement.



(b) Actin-myosin interactions produce several types of movement.

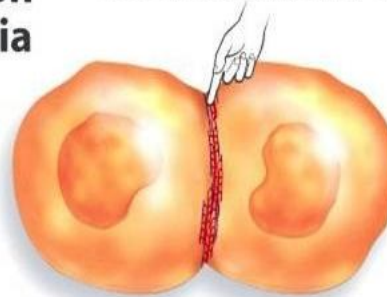
Actin-myosin interactions push cytoplasm forward



Cell crawling

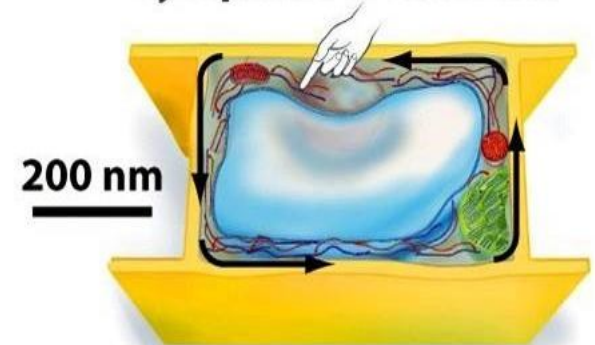
Actin polymerization creates pseudopodia

Actin-myosin interactions pinch membrane in two



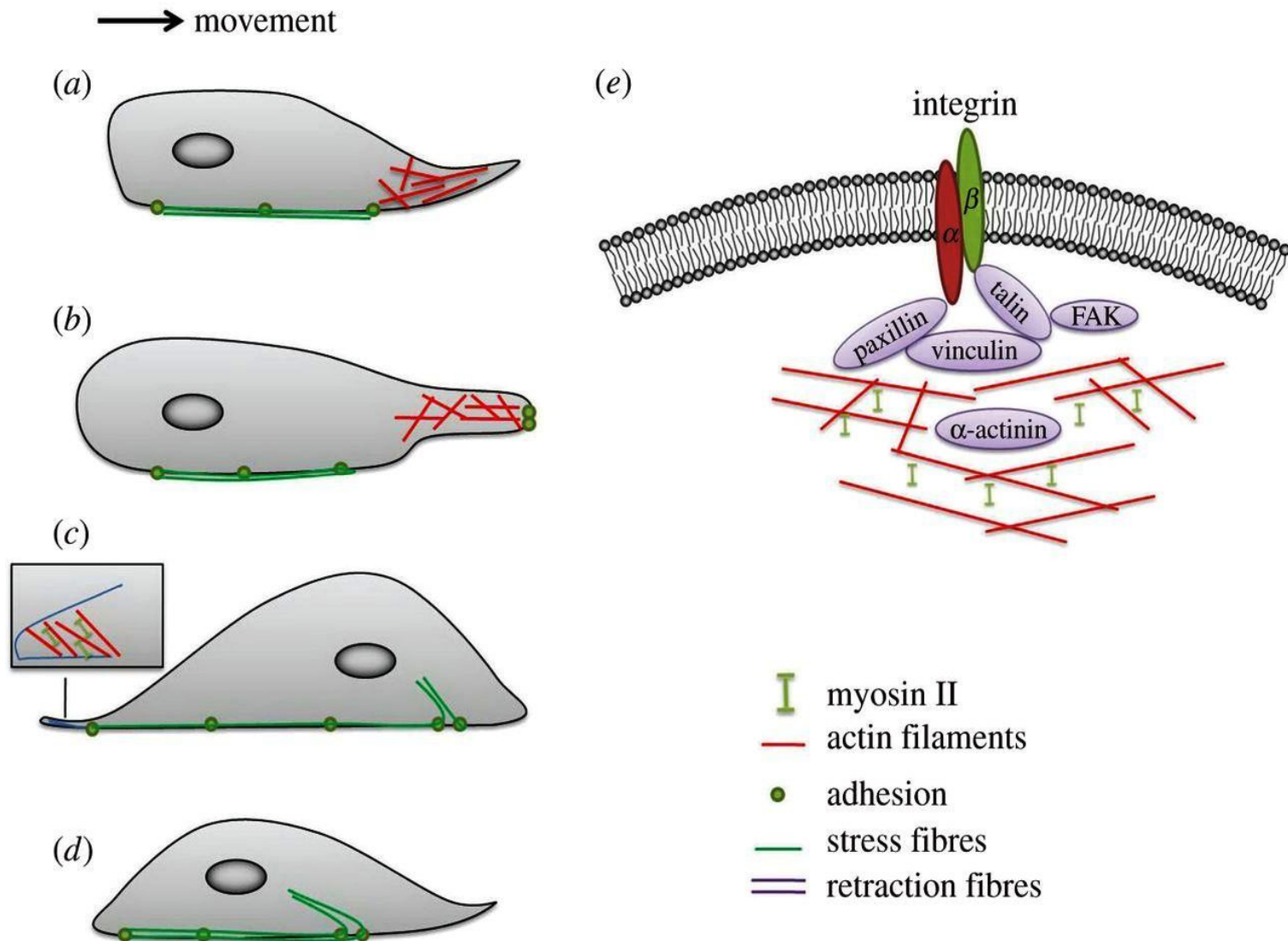
Cell division in animals

Actin-myosin interactions move cytoplasm around cell

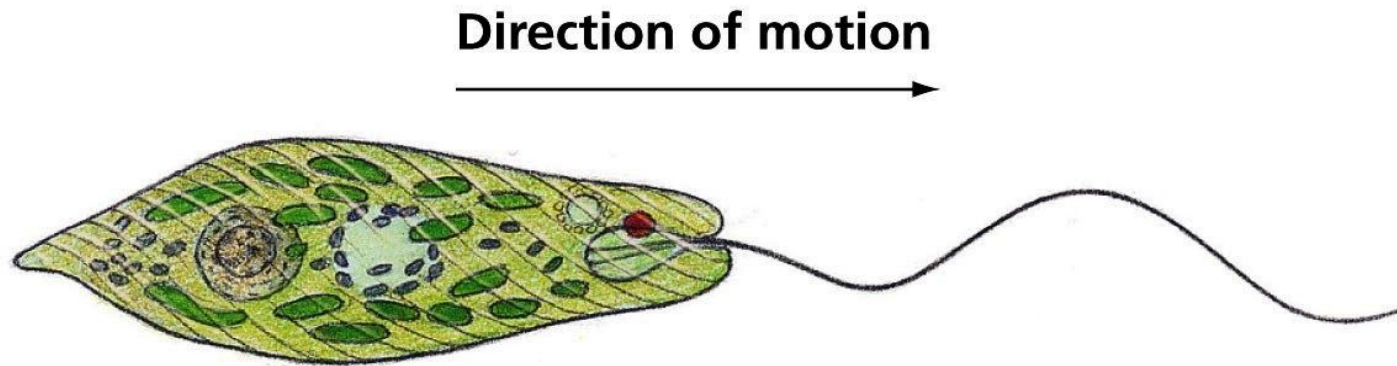


Cytoplasmic streaming in plants

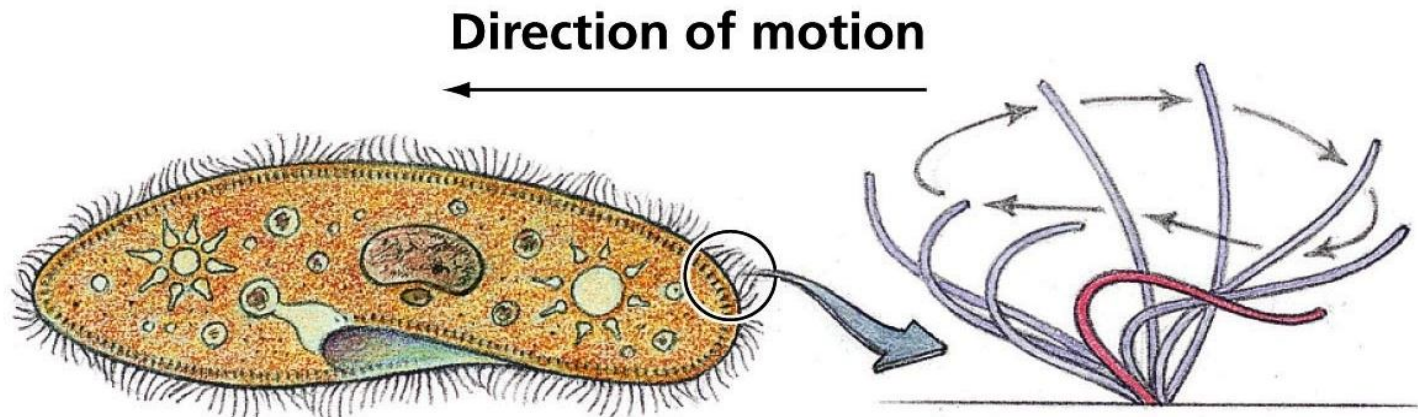
Sol-gel (forward) or Gel-sol (backward) conversion: Molecular modeling



AXONEMES : Flagellum & Cilium

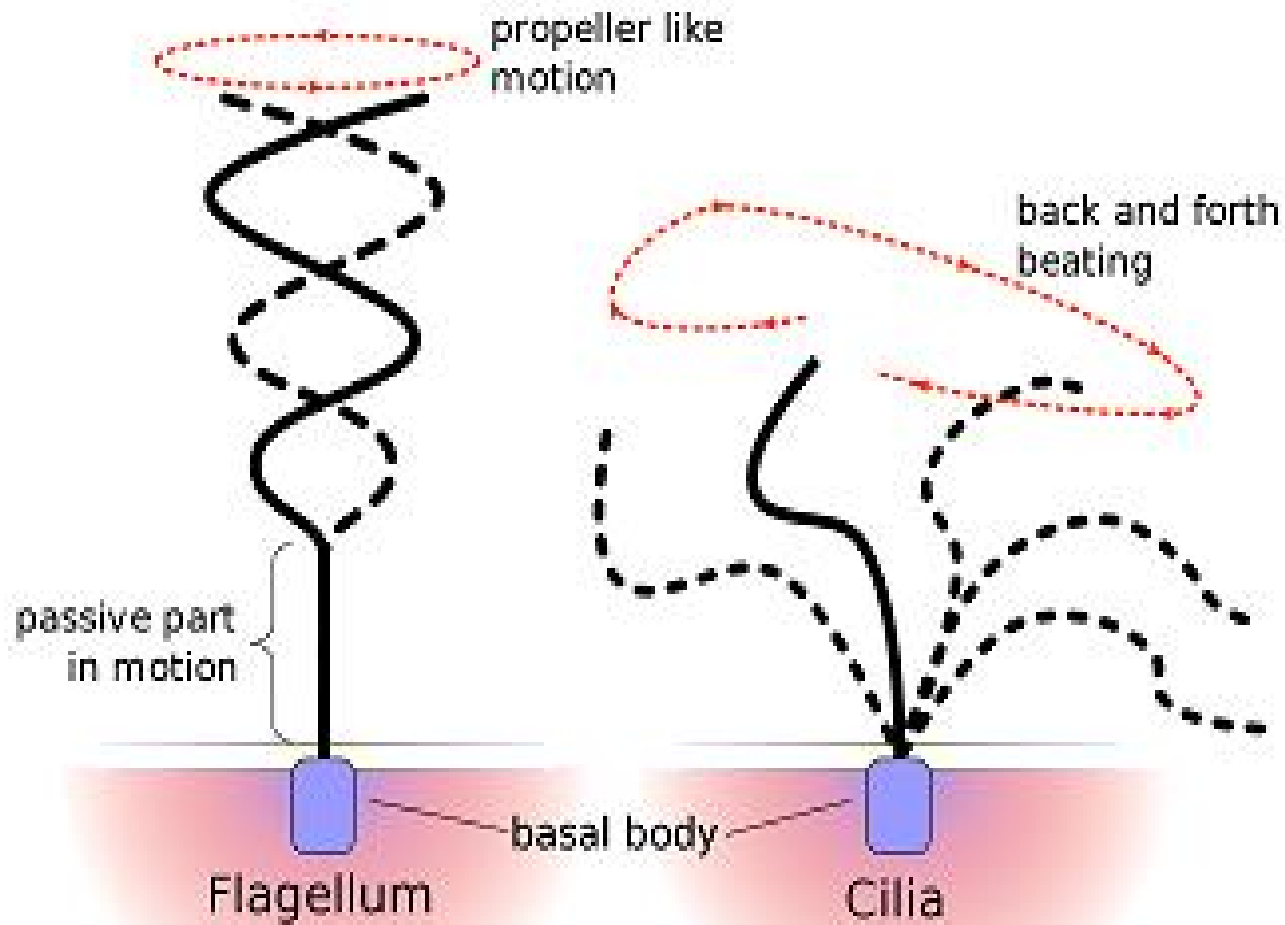


(a) Flagella



(b) Cilia

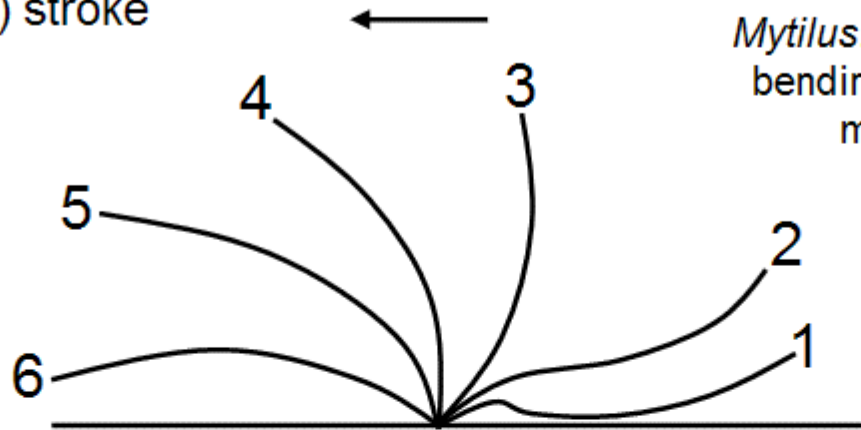
Axonemes: **Flagellum** & **Cilium**



Propeller Motion & Back-forth Motion

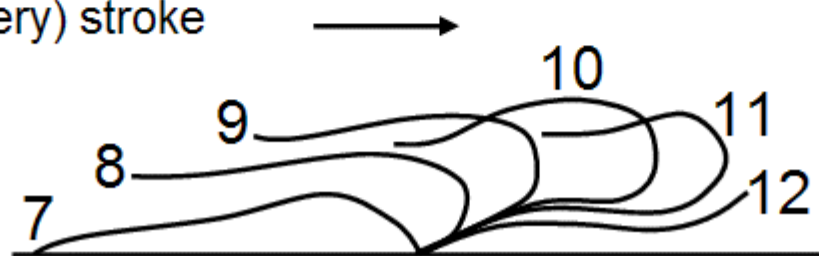
Forward & Backward Stroke by Cilia

Forward (power) stroke



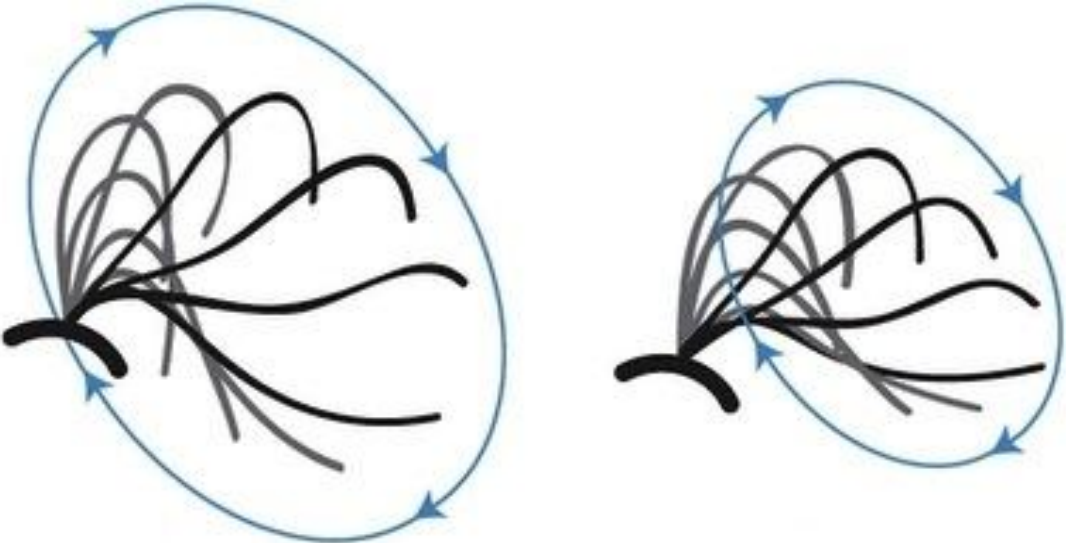
Metachronal rhythm of cilia in *Mytilus*. The cilia are flexible and bending starts at the base and move toward the tip.

Backward (recovery) stroke

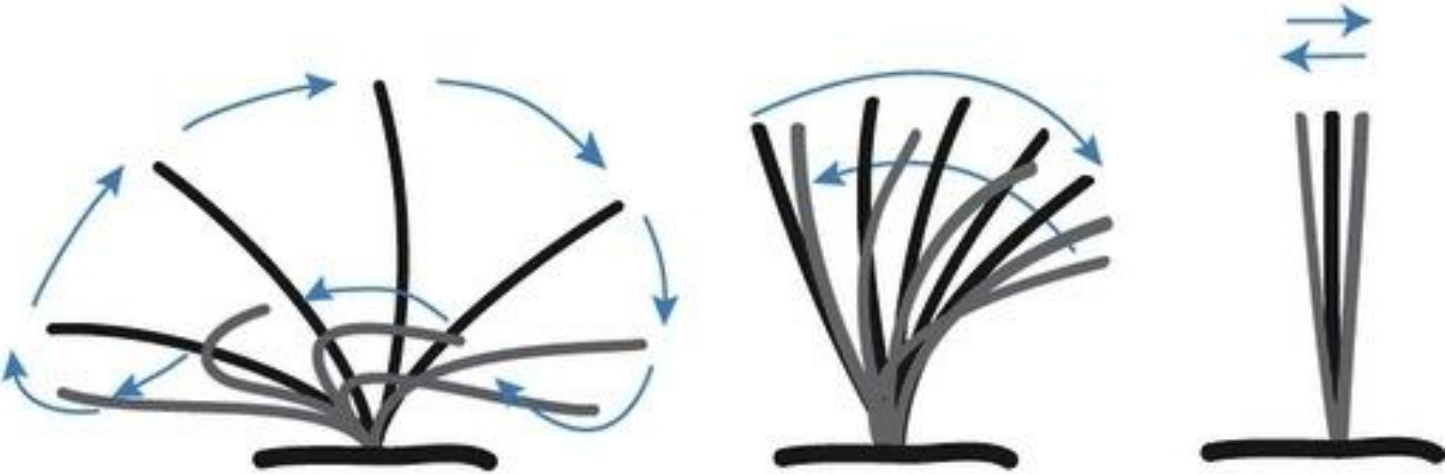


Model presentation of **Flagellar** vs. **Ciliary** Motion

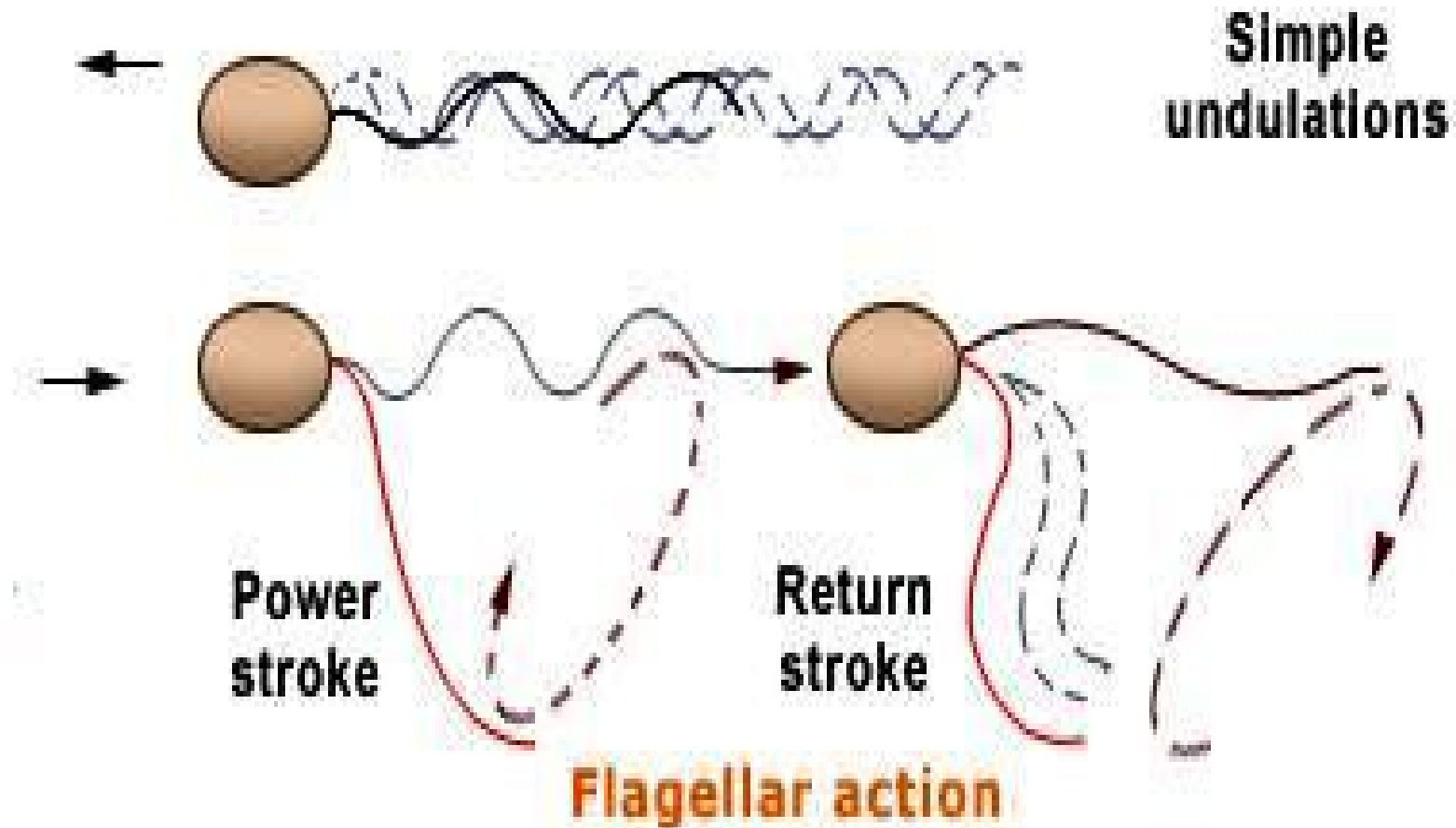
a



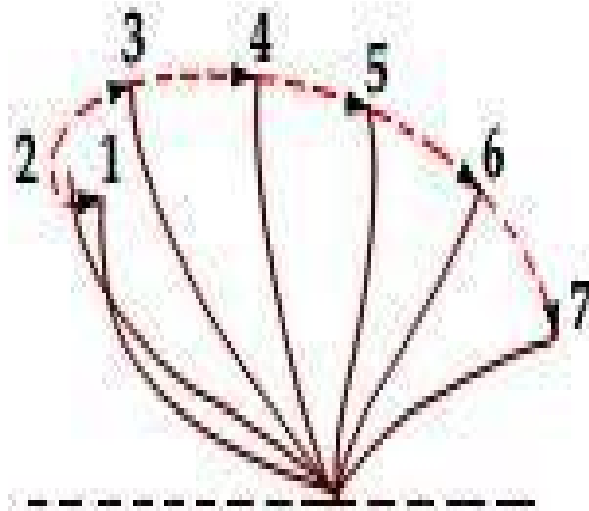
b



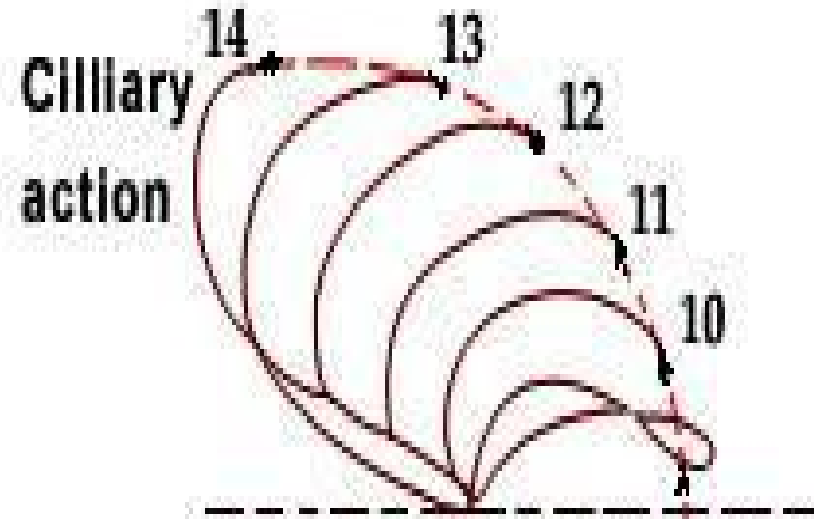
Undulatory Motion & Power-return Stroke



Ciliary Locomotion



Power stroke
(Maximum resistance)

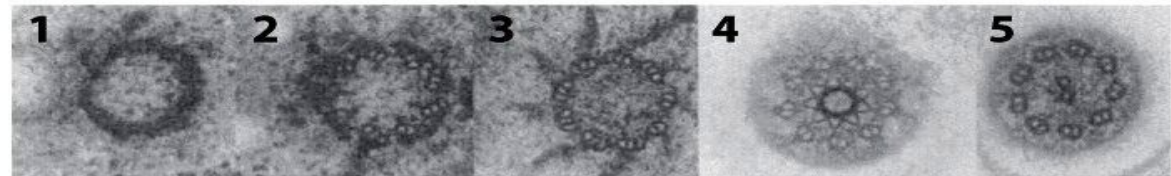
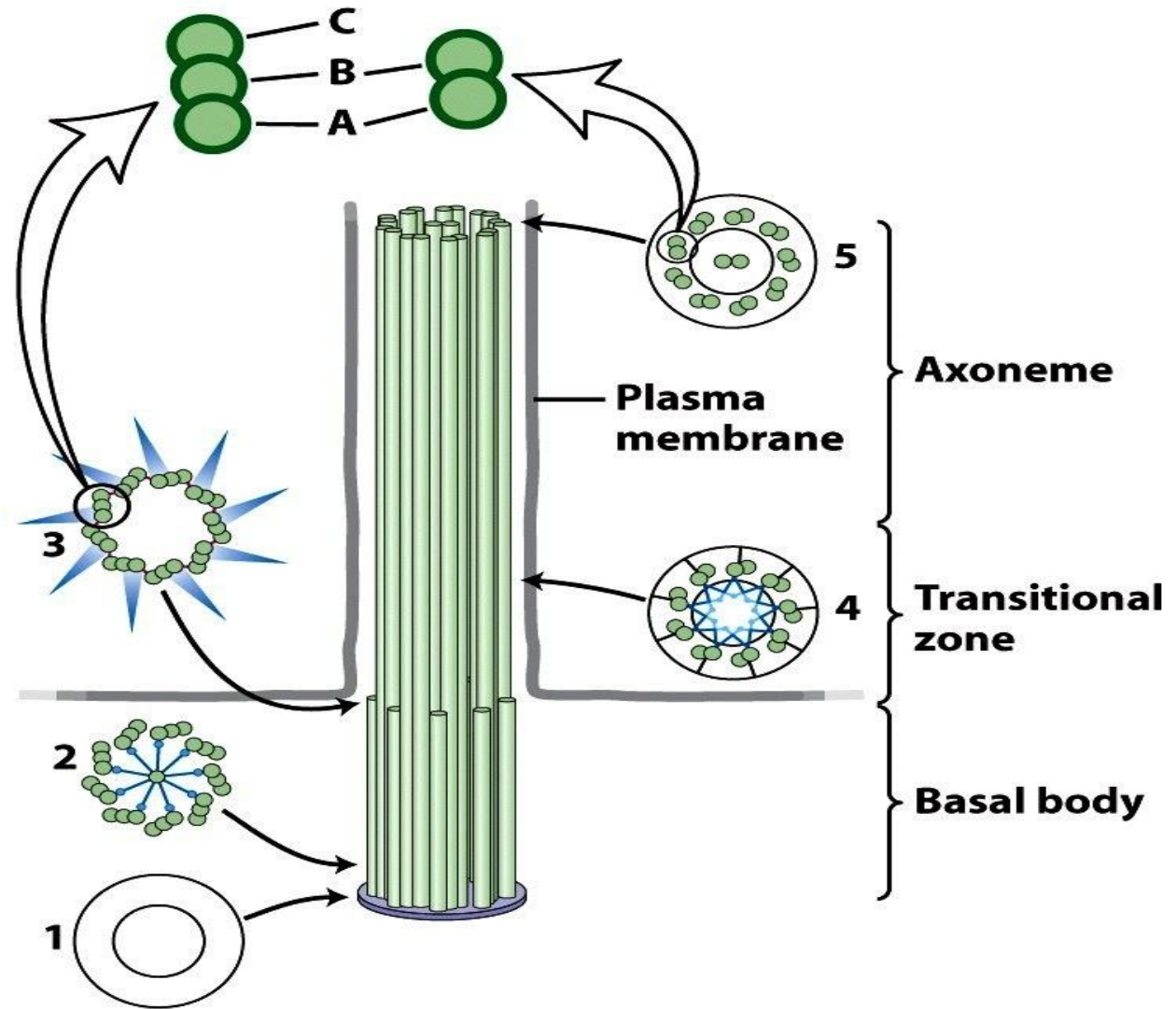


Return stroke
(Minimum resistance)

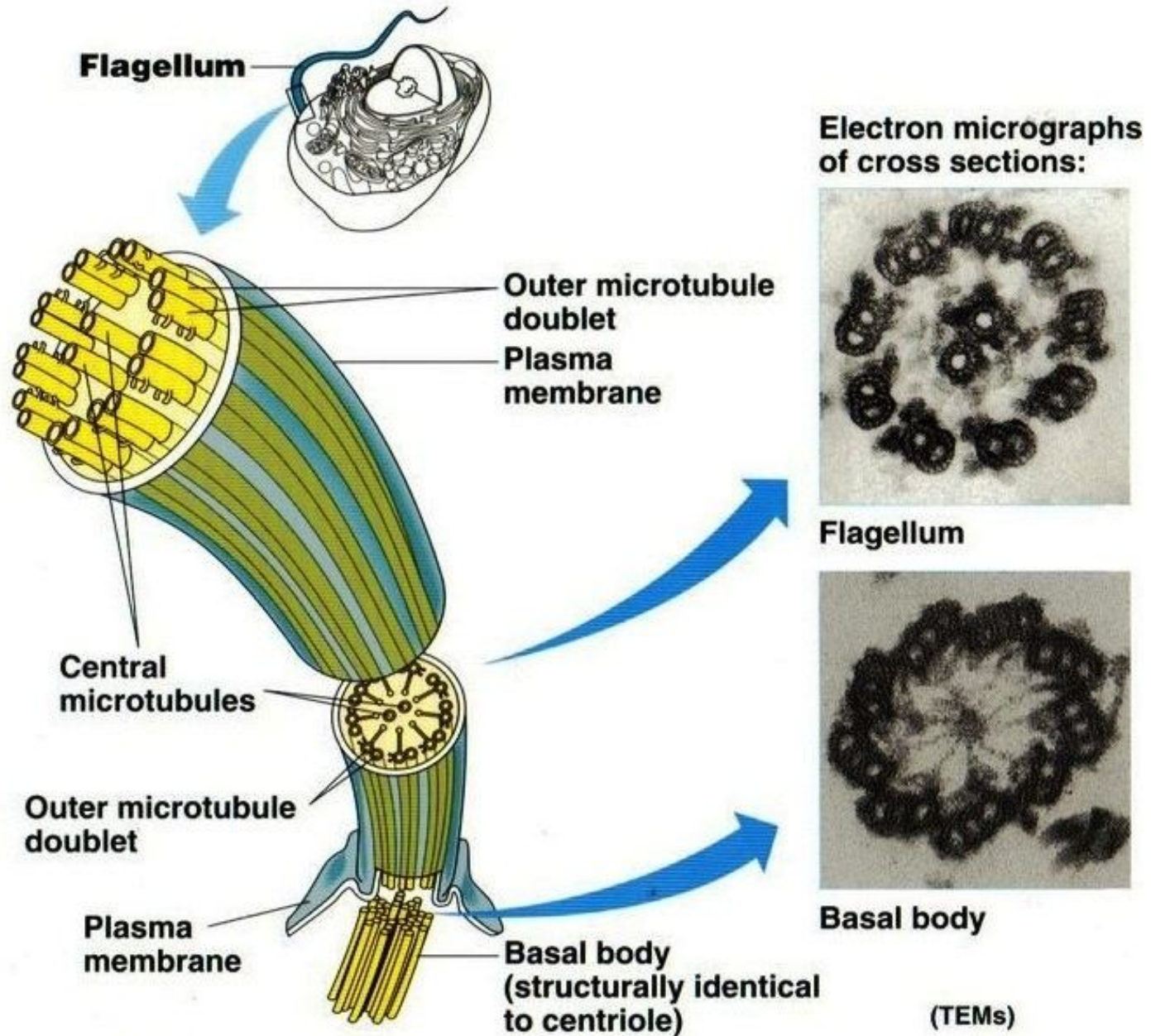
Effective stroke (a) and recovery stroke (b) of a cilium

Structural Model of Axoneme:

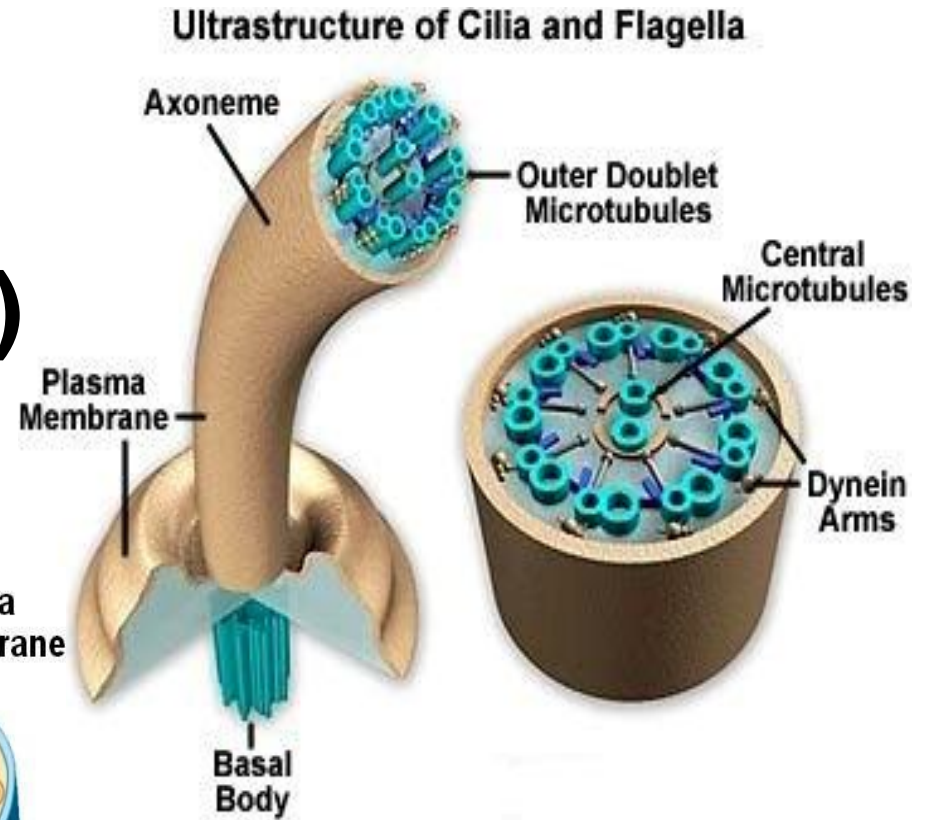
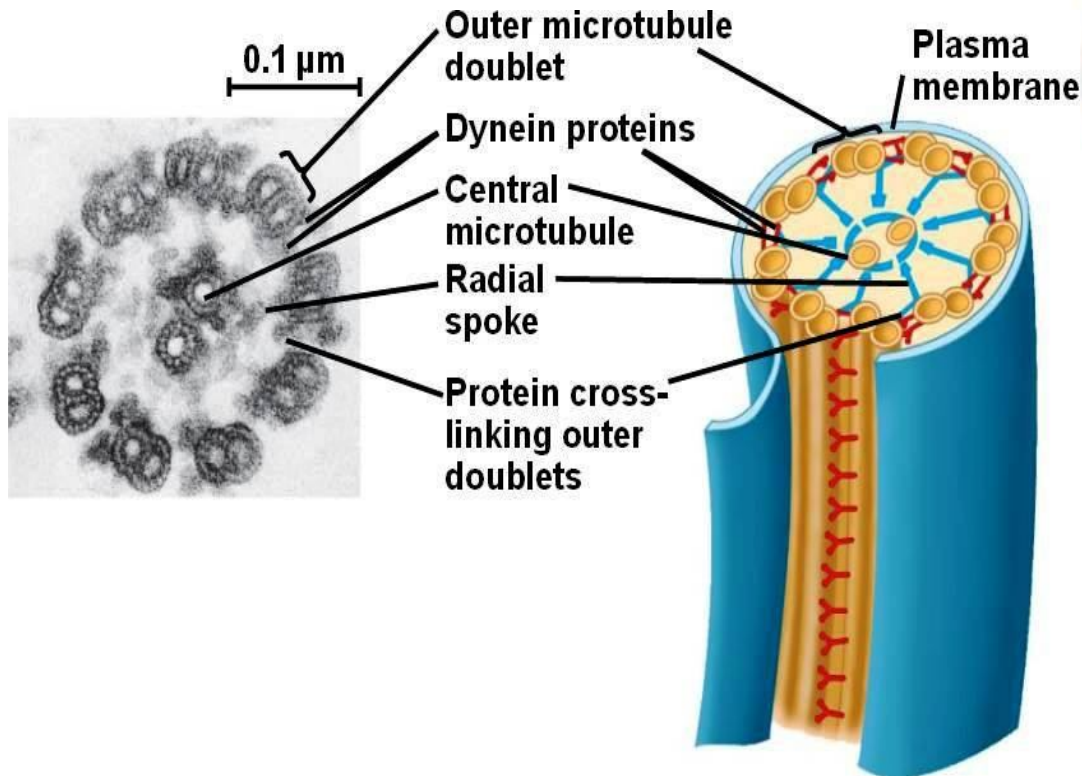
T.S. or C.S. through different zones



Ultrstructure of Flagellum or Cilium

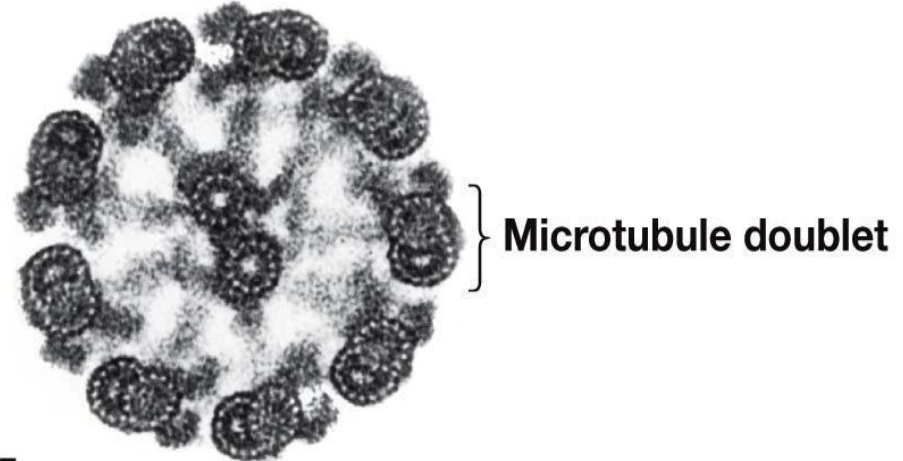


C.S. or T.S. of Axoneme (Cilium or Flagellum)



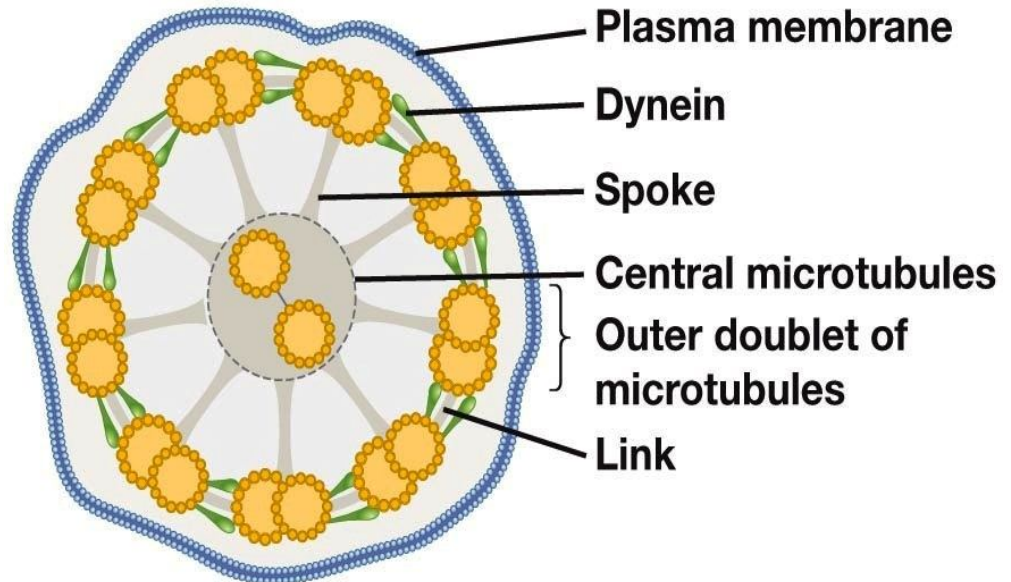
9+2
Arrangement
of
Microtubules
in
Axoneme

(a) Transmission electron micrograph of axoneme

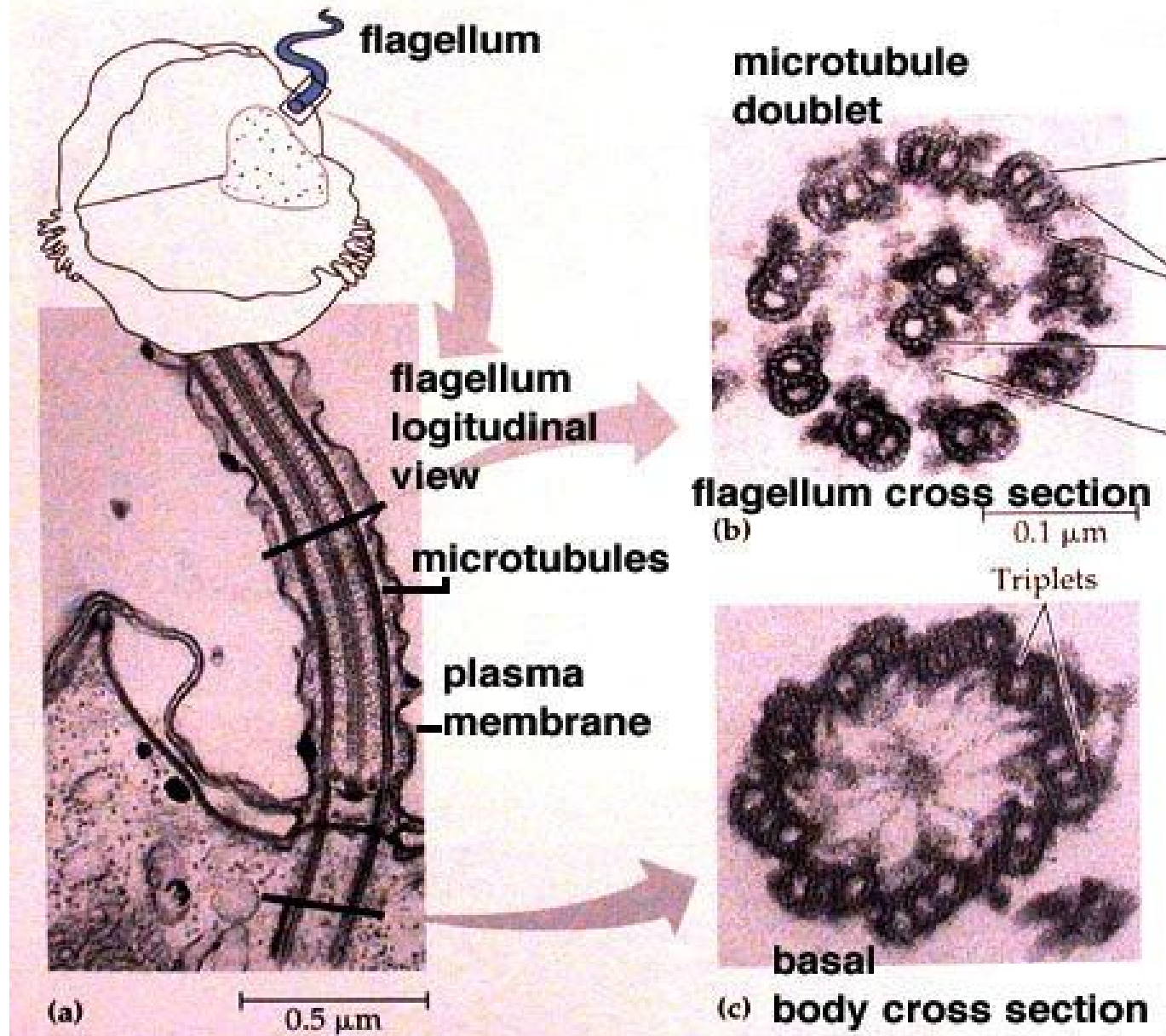


75 nm

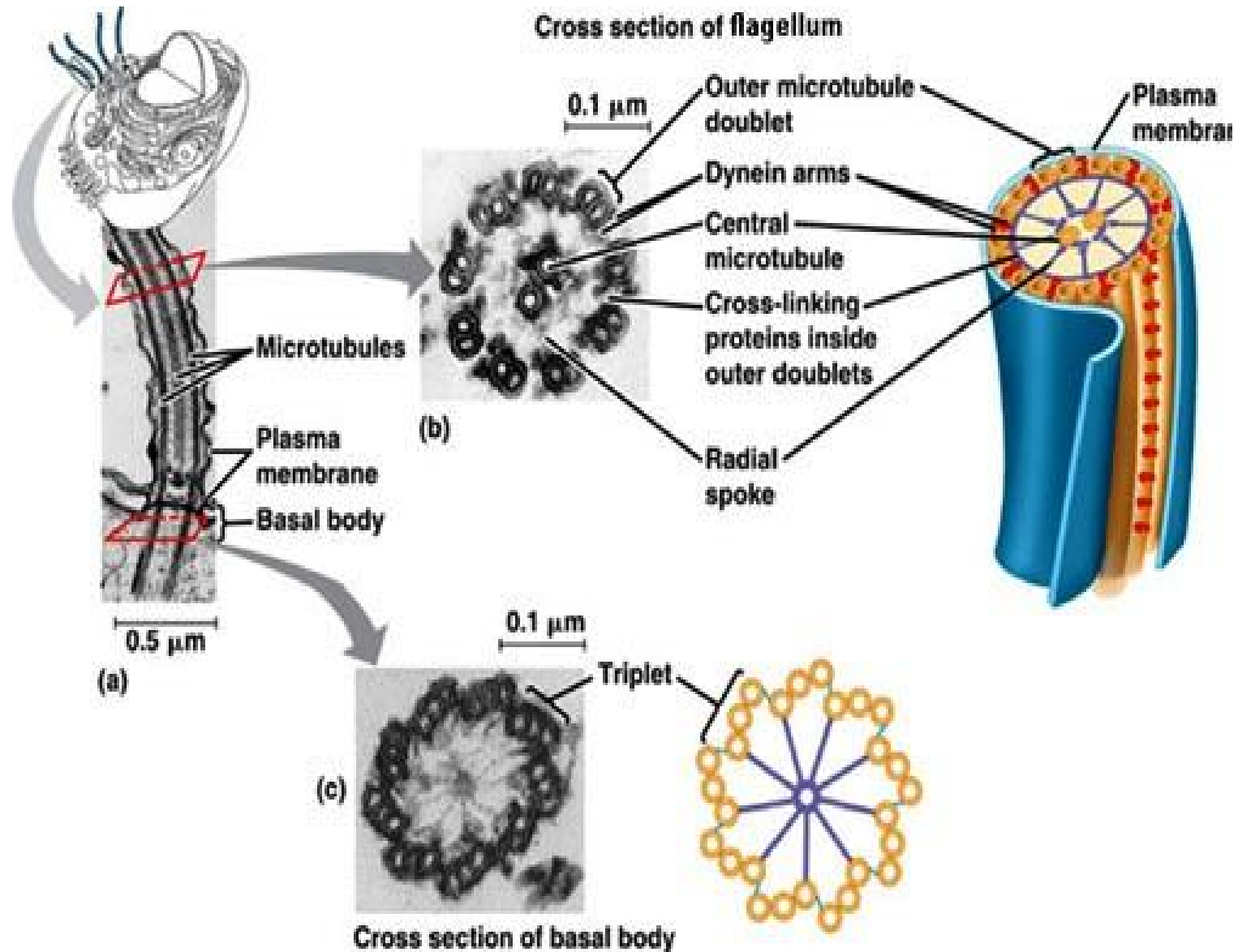
(b) Diagram of axoneme



Ultrastructure of exoneme in L.S. & T.S.

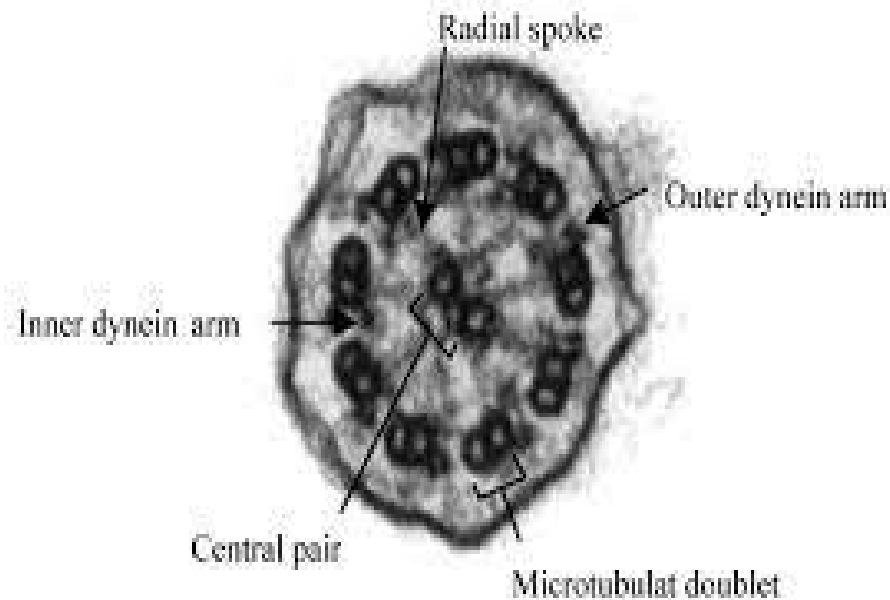


Cross Section of Axoneme

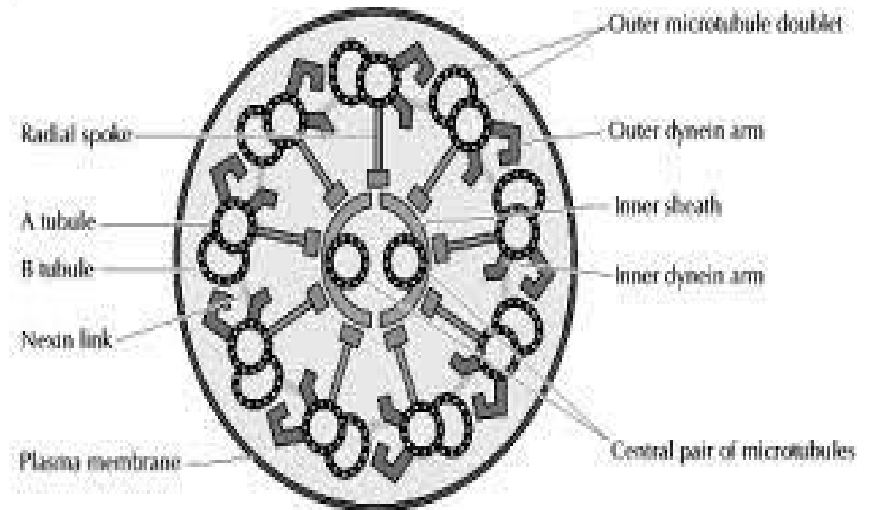


Cross Section of Axoneme

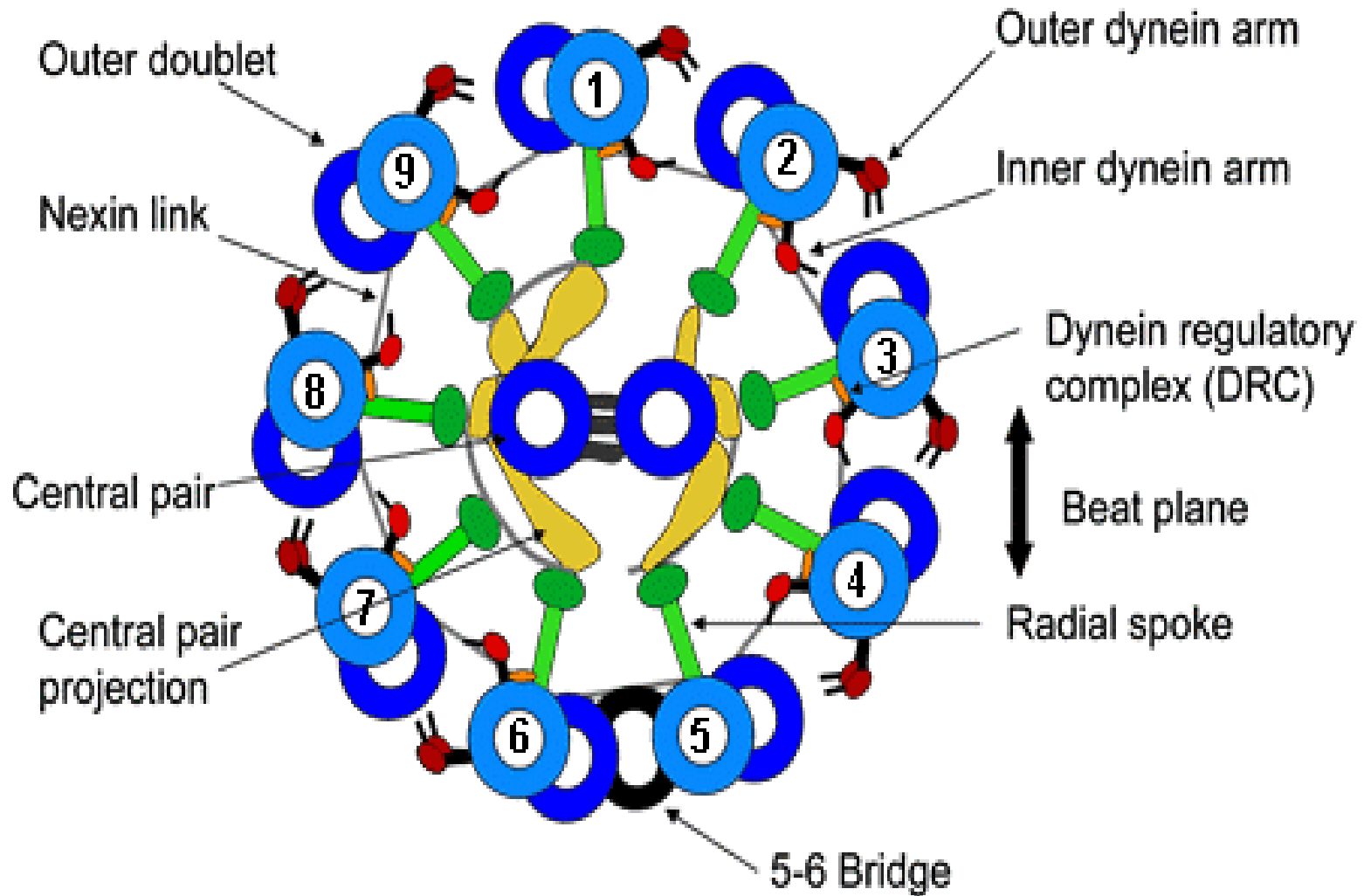
A. Real figure



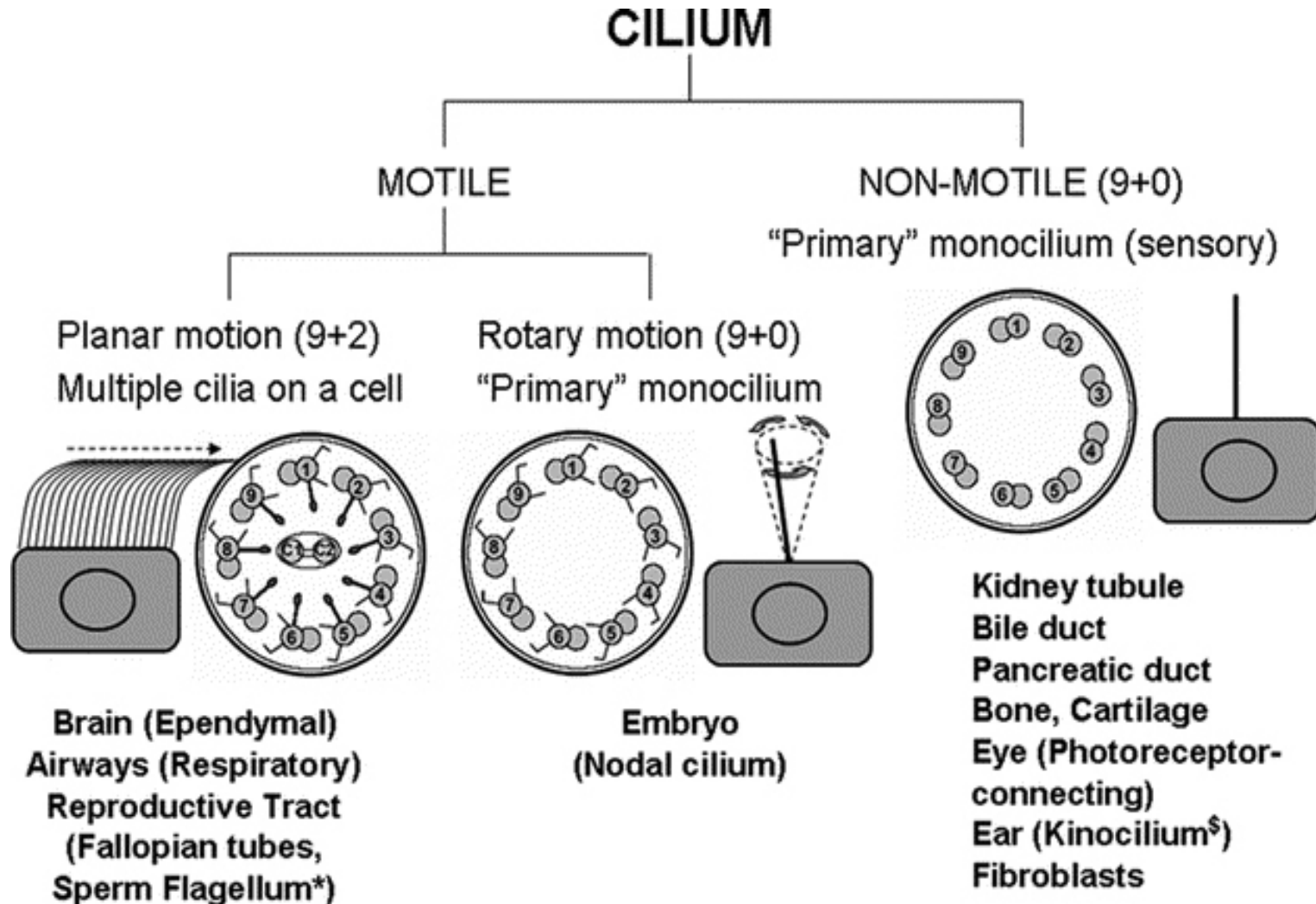
B. Model figure



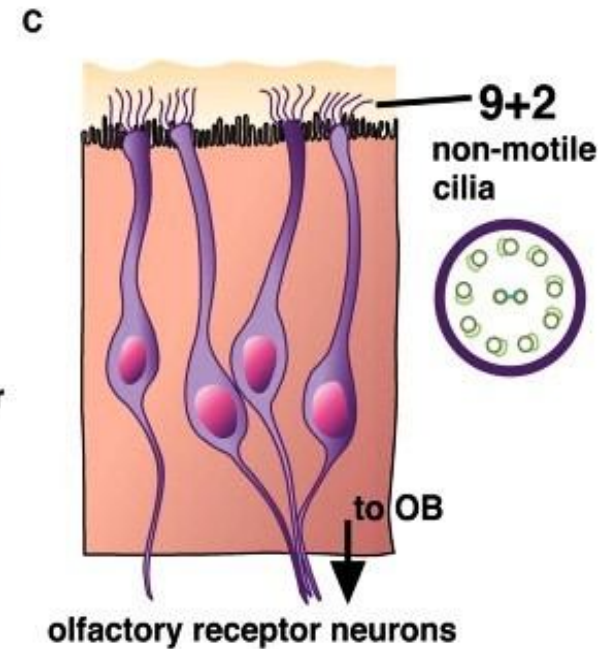
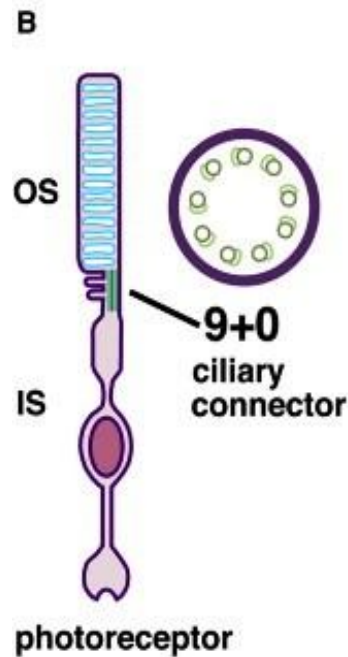
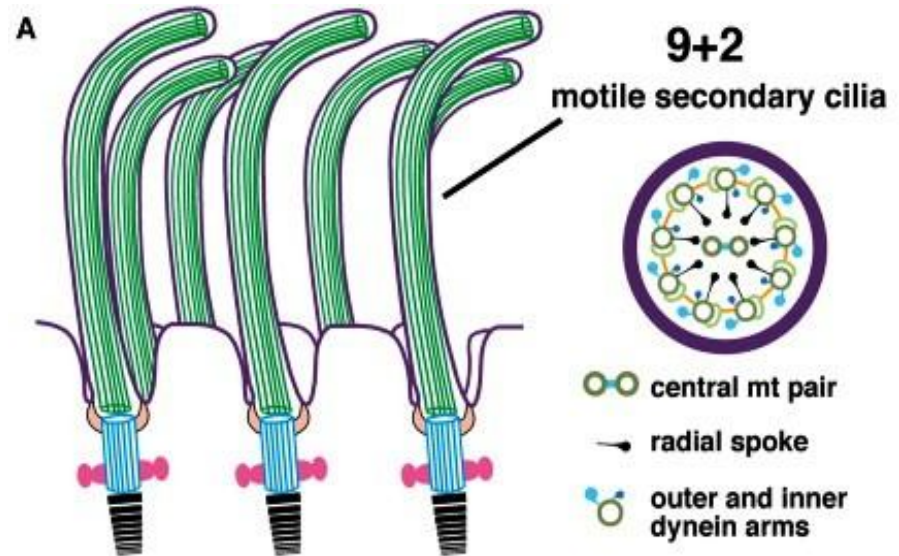
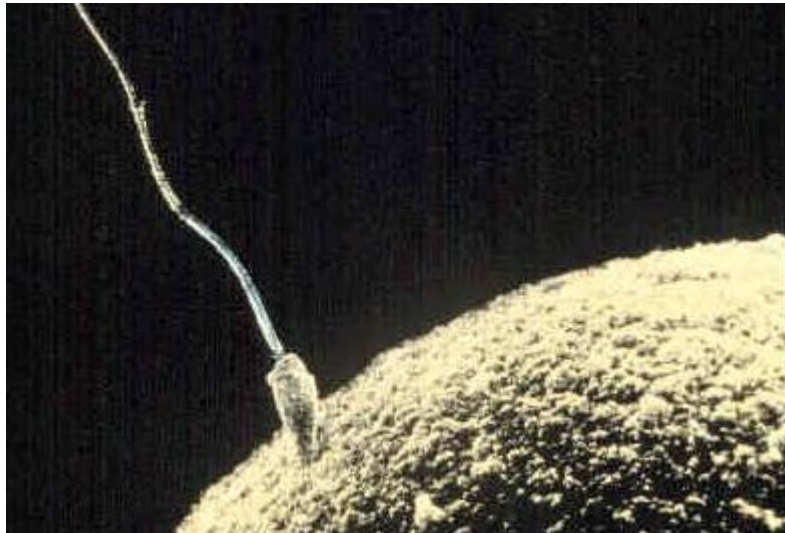
Details of microtubular arrangement: a Model



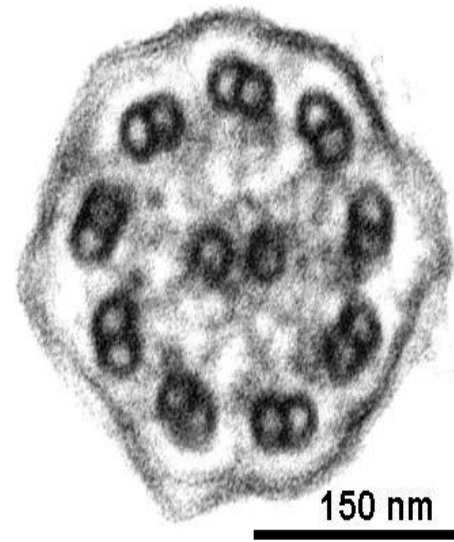
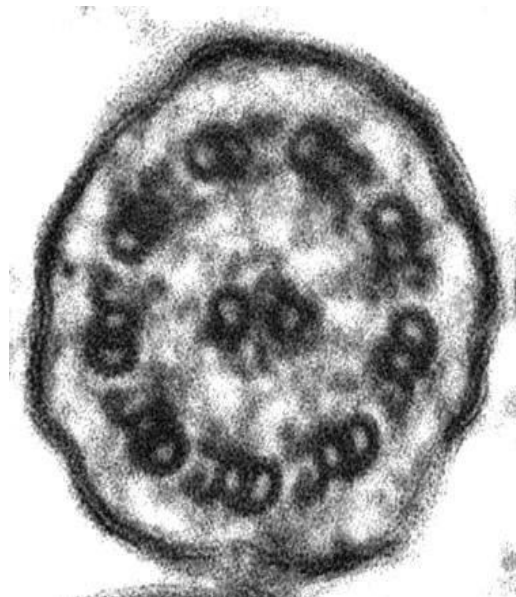
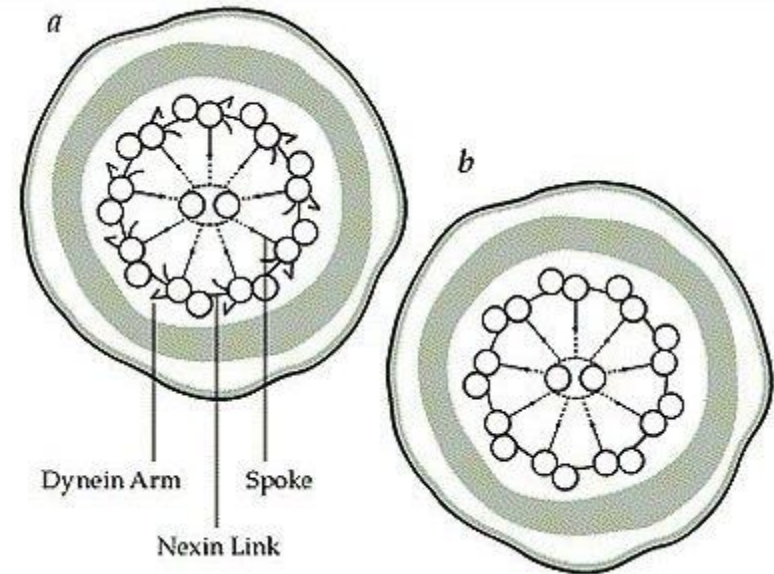
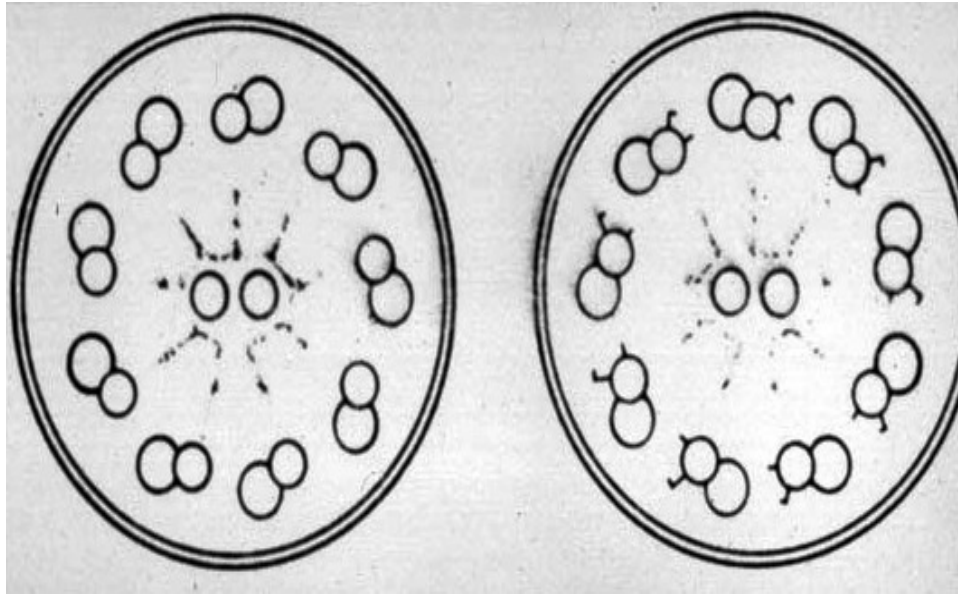
Deviation from (9+2) to (9+0) arrangement and lack of Dyneins that result to Ciliopathy



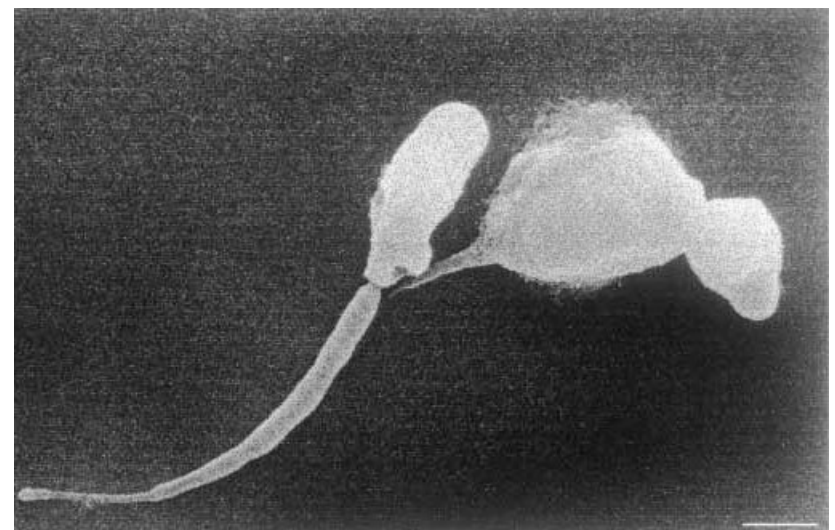
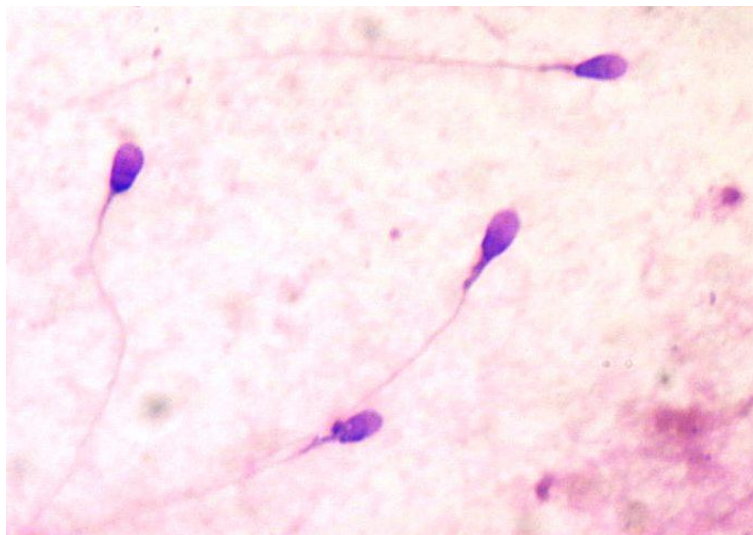
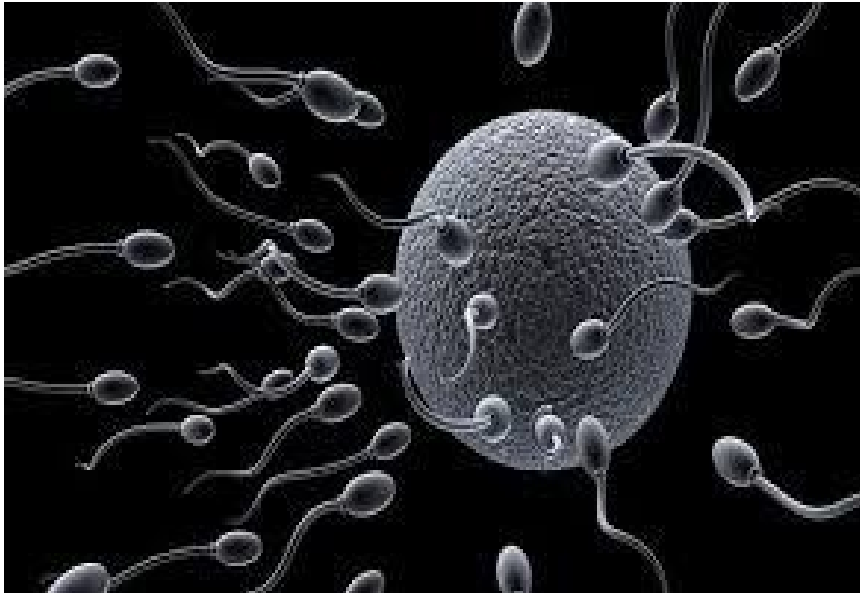
Human cilium & Flagellum



Abnormal Axonemal Configuration



Normal vs. **Abnormal Sperm**
Stumpy tail Syndrome *or* **Kartagener syndrome**



Axonemal Dysfunction

Dysfunction of the axonemal structure has been linked to the emerging class of disorders collectively known as **CILIOPATHIES**, which includes **Primary ciliary dyskinesia (PCD)/Kartagener syndrome**, **Bardet-Biedl syndrome**, **hydrocephalus**, **polycystic kidney disease**, **polycystic liver disease**, **nephrolithiasis**, **Meckel-Gruber syndrome** and **Joubert syndrome**.

PCD is a genetically heterogeneous disorder affecting motile cilia which are made up of approximately 250 proteins. Around 90% of individuals with PCD have ultrastructural defects affecting protein(s) in the outer and/or inner dynein arms which give cilia their motility, with roughly 38% of these defects caused by mutations on **two genes**, DNAI1 and DNAH5, both of which code for proteins found in the ciliary outer dynein arm.

Primary ciliary dyskinesia (PCD) Immotile Ciliary Syndrome (ICS)

Ultrastructural and functional defects of cilia result in the lack of effective ciliary motility, causing abnormal mucociliary clearance. This leads to recurrent or persistent respiratory infections, sinusitis, otitis media, and male infertility. Primary ciliary dyskinesia (PCD), also known as immotile ciliary syndrome (ICS), is a rare, ciliopathic, autosomal recessive genetic disorder that causes a defect in the action of the cilia lining the respiratory tract (lower and upper, sinuses, Eustachian tube, middle ear) and fallopian tube, as well as the flagella of sperm cells. In 50% of the patients, ICS is associated with situs inversus.

Kartagener Syndrome

- In 1933, Kartagener described a unique syndrome characterized by the triad of *situs inversus*, *chronic sinusitis*, and *bronchiectasis*, later termed as **Kartagener syndrome**. The moveable tails of sperm (flagella) are often also affected. Abnormality in sperm motility may result in male infertility.
- Male infertility is a common sign of Kartagener syndrome present in men. Women with Kartagener syndrome are often infertile because of ciliary immotility or immobility in/of the lining of the Fallopian tubes.

SEE YOU NEXT TIME

THANKS

Uploaded on 16-01-2019

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Sept, 2018