# Studentskrivna Föreläsningsanteckningar från

## Cell och Molekylärbiologi II

Skrivna av Jonas Elmwall (Student på Bioteknik) 2009

# Cell- och Molekylärbiologi 2

Föreläsning 1, 2008-01-19 Kap 10
Introduktion Magnus Holm

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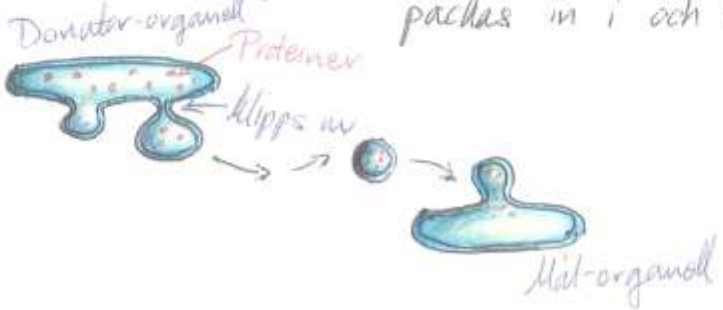
Kursen tar upp bla
- Transport mellan organeller (Intracellulär transport)
- Signaler mellan celler (Signal-transduktion)
- Struktur hos celler (Cytoskelett)

Membran: tunn struktur
hydrofil utsida, -fob kärna
Halva massan bestående av proteiner

Membrantransport (med/mot gradient)

Proteintransport
Transkription i cellkärnan därefter transporteras ut i cytosolen.
Hur transporteras de in i organeller?

Tre skilda typer av transport i cellen
- Reglerad (mellan kärna och cytosol)
- Transmembran (genom membran)
- Vesikeltransport (vesikel: ~~paket~~ paket som protein packas in i och skickas i)



Signaltransduktion

Hur signaler tas upp och tolkas

Cytoskelett

Tre olika typer

Hur kan de fungera som cellulära motorer?

# Cell- och Molekylärbiologi
Föreläsning 2, 2008-01-20
Membrantransport

Kap 11
Magnus Holm

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**Membranstruktur**

definierar cellers och organellers form
är en dynamisk och flexibel struktur

Fungerar som en 2-dimensionell vätska

Membranen hålls samman av hydrofoba interaktioner, där den hydrofoba delen riktas inåt. Bild 2

**Membrantransport**

Hydrofoba ämnen kan lätt diffundera igenom membranet.
Små oladdade molekyler kan diffundera igenom, och även större, men med större svårighet.
Joner och vattenlösliga molekyler har svårt att passera. Bild 3

**Membranproteiner**

50% av massan i ett membran består av proteiner, vilka sitter inbäddade i membranet. Dessa avgör vad membranet gör.

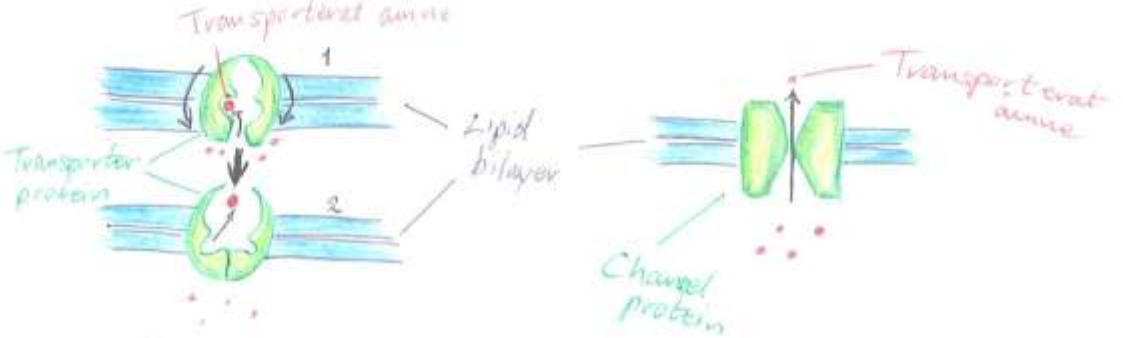
Funktioner: Transport. Funktionen för 30% av proteinen.
Förankring. Ex. fäste till omgivningen, som i ett lager av celler.
Signalering över membran.

Membran och proteiner kan skapa barriärer som kan skapa gradienta av koncentration och laddningar. Gradienterna kan användas som energikällor för transport el. signalering.
I jongrad. med $Na^+$ har man låg konc utanför, låg innanför, med $K^+$ tvärtom. Bild 4.

## Transportproteiner för passiv transport.

Det finns två typer av transportproteiner: transporter och channel proteins.

| Transporter | Kanal |
|---|---|
| - Binder till transporterat molekyl | - Tillåter nästan fri diffusion |
| - Flippar mellan två lägen | - Kan regleras (öppen/stängd) |
| - Transport sker långsammare, pga flippandet | - Mycket svagare interaktion |
| | - Snabbare transport |




- Transporter binder molekyl → struktur hos proteinet ändras, varpå det flippar → släpper molekylen på andra sidan.
- Underlättar diffusion som annars skulle ta tid, till en viss gräns beroende på konc.

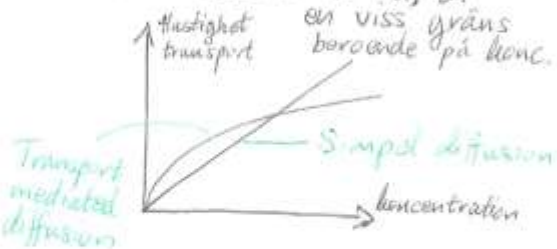



Bild 5, 6

Transporter protein finns i tre varianter:

Uniport: En molekyl transporteras.

Symport: Två olika molekyler transporteras, åt samma håll

Antiport: Två — " — , åt olika håll

Bild 7

## Elektrokemisk gradient

Att med laddningar skapa en membranpotential kan underlätta el. motverka transport av laddade molekyler:

- Motsatt potential (jmf laddad molekyl) ⇒ underlättar
- Omvänt ⇒ motverkar Bild 8

Koncentrationsgrad + Membran-pot. ⇒ Elektrokemisk grad

## Passiv / Aktiv transport

Passiv transport underlättar diffusion, samt följer grad.
Aktiv går mot grad. och kräver energi. Det kräus pumpar. Bild 9

## Aktiv transport

Tre typer av aktiv transp.:

- Kopplad: Pumpar för transport mot grad. av ett ämne drivs av energin hämtad från grad. av ett annat ämne.
- ATP-driven: genom hydrolys av ATP
- Ljus-driven: anv. energin hos foton. Bild 10

## Kopplad

Bild 11 visar transport mha $Na^+$-grad, co-transp, symport-typ.
Energi från grad. (från $Na^+$-grad) transp. glukos mot dess grad.
Med $Na^+$ bundet bildas ett hög-affinitetsområde för glukos.
Glukos bundet ⇒ flipp, svag bindning för glukos blir samma ⇒ släpps. Detta är viktad transport.
Bild 12: samma princip. Laktos transp mha $H^+$.
Förbjudet steg: kan ej flippa m. enbart $H^+$
Bild 13: En tarmcell vill ta upp näringsämnen och skicka vidare.
Låg konc. innanför och utanför tarmen och hög i cellen.
$Na^+$-driven transporter på tarmsidan och diff.-driven på utsidan skickar ämnet åt rätt håll.
Tarmen mot tät barriär, inget skickas åt fel håll.

## ATP-drivna pumpar

Tre typer: P-typ: autofosforyleras, P frigörs från ATP
Pumpar ex $Na^+$, $Ca^{2+}$
F-typ kallas ATP-aser. Använder $H^+$-grad. för att göra ATP av ADP
ABC-transportör

Bild 14

Bild 15: $Ca^{2+}$-pump. ER har hög $Ca^{2+}$-konc., så här används ATP för att pumpa $Ca^{2+}$.
Proteinet har ATP-bindande domän.

## $Na^+/K^+$-pump (P-typ)

Spenderar ca 1/3 av cellens energi. Antiport.
Medan $2K^+$ transporteras in, mot sin grad.,
transp. $3Na^+$ ut, med sin grad.
Viktig för att bibehålla grad. i plasma

Bild 16, 17

## Osmotiskt tryck

En blodcell har i isotonisk lösn. normalt utseende,
i hypertonisk mindre vatten, i hypotonisk mer vatten
(kan spräckas).
Lösn: Djur och bakterier pumpar ut inorganiska joner
Växters celler hindras av sina cellväggar
Protozoaner skickar ut vatten m. speciella vakuoler.

Bild 19

## ABC-transportör

När dessa proteiner binder ATP, öppnas de åt ena hållet,
och när ATP hydrolyseras öppnas de åt andra.
Bakt. använ. dem för import.
Euk för export.

Bild 19

## Gated Ion channels

Tre typer: Voltage-gated
Membran-pot. styr om öppen/stängd.
Ligand-gated
Mechanically gated
Bild 20

## Membranpotentital

$Na^+/K^+$-pump ger 1/10 av pot.
$K^+$ läcklage ger neg. joner på insidan, pos. på utsidan, vilket skapar pot.
Varierar mellan 20mV och 200 mV i djurcell.
Viktigt för nervsystemet
Bild 21

## Selektivitetsfilter

$K^+$ kan passera, större eller mindre kan inte ha interaktion m. syre.
Bild 22
Filter kan vara specifikt för en särskild jon
Bild 23

## Aquaporiner

Större delen av kanalen är hydrofob, men har hydrofil sid där $H_2O$ kan passera. Joner har svårt att passera $H^+$ kan ej passera.
Bild 25

## Nerceller, aktionspotentital

Mkt snabb transp. av signaler, m. stegen:
1) Depolarisering av plasma-membran.
2) Öppnar Voltage $Na^+$-kanaler, $Na^+$ släpps in.
3) ~~Fortsatt~~ dep., kastig
4) $Na^+$-kanaler inaktiveras
5) Fördröjda $K^+$-kanaler öppnas
6) Membr. polariseras
Signal slutar
Bild 25

Anteckningar skrivna av Jonas Elmwall
Lp3 2009

Membr.proteiner strävar efter så låg fri energi som möjligt. Proteiner "slappnar av" till den lägsta energin.
Portar öppnar vid depol. Depolariseringen får Bild 26
en riktning och kan bara gå åt ett håll. Bild 27
Nervceller är isolerade av gliaceller Bild 28
Depol. känns av mellan noder i nervceller.
Synaps membr.paket, öppnas vid depol ⇒ signalerar cell.
Acetylkolinreceptor: stängd om acetylkolin ej bundet.

En nervcell fungerar som sammanställning av av information.
Vissa övf depol, andra pol signaler.
Om depol. membr öppnas sätts $Ca^{2+}$ in
Mottagande cell mer känslig för glutamat.

Cell- och Molekylärbiologi
Föreläsning 3, 2008-01-26
Cytoskeleton dynamics, Ch 16

Kap 16
Julie Gramtham

## Cytoskeleton

Three types of cytoskeleton filaments

Intermediate filaments: Mechanical strength

Microtubules: Determine postions of organnelle, intracellular transport

Actin filaments: Shape of the cell's surface and cell locomotion.

Constructed from smaller subunits, hold together by weak, noncovalent interactions, forming polymers.

Can assemble and disassemble rapidly and diffuse rapidly trough cytoplasm ⟹ can undergo rapid structural reorganization.

[redacted]

Important for the flexibility and motion of the cell, as for the cell divison: polarized microtubili form a bipolar mitotic spindle, aligning and separating cromosomes. Actin filaments form a contractile ring, that pinches the cell in two at the center.

Bild 1-3

## Actin and Microfilament

Monomeric form - G-actin (globular → G)

Filamentous form - F-actin (filament → F)

Actin subunit: single globular polypeptid chain, a monomer.

Has a binding site för ATP (ADP when in filament)

Assemble head-to-tail, generating filaments with distinct structural polarity

Two parallell protofilaments, twisted in a right-handed helix.

Flexible, easily bent, but made as strong large-scale actin structure by accessory proteins

## Actin polymerization

Has three phases

Nucleation (lag phase): Initial process of nucleus assembly. Can take time, depending on how many units must come together to form a stable nucleus.

Elongation (growth phase): When stabilised more subunits can be added rapidly to the ends of the nucleated filaments

Steady State (equilibrium phase): Rate of addition balances the rate of subunit dissociation

Critical conc. ($C_c$): The conc when the rate of subunit addition equals to the rate of subunit loss.

There exists two types of filamentous structures, T-form (ATP, actin / GTP, tubilin) and D-form (ADP/GDP). Polymerize at both ends, then undergoing nukleotide hydrolysis

Plus end of actin filament grows faster, minus end slower, because of differences in structure between the ends

⇒ Subunit addition can be faster than the hydrolysis at +

— " — slower — " — at -.

Hydrolysis reduces binding affinity of the subunit

⇒ More likely to dissociate.

Therefore, the + end adds subunits and - end loses, if $C_c(T) < C < C_c(D)$ ⇒ treadmilling occurs, constant length

$C > C_c$ ⇒ both grow $C < C_c$ ⇒ both shrink (if the same units in both ends)

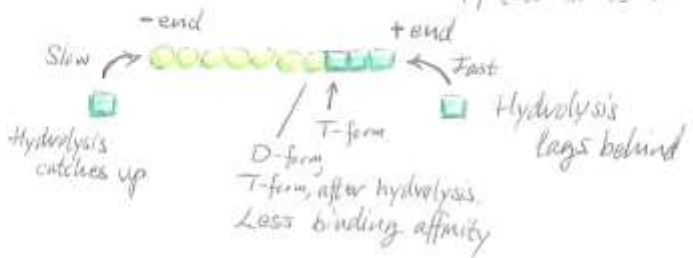



Bild 5-7

## Actin organisation

Actin interacts with many accessory proteins, ex. monomer binding, severing, capping, nucleation and cross-linking proteins.

### Cross-linking proteins

Two classes: bundling prot., cross-linking AF into parallell array.
gel-forming prot., " loosar meshwork

Ex of four types: fimbrin, α-actinin, filamin, spectrin.

Ex. of two types of formation of AF bundles:

α-actinin cross-links AF into loose bundles, allowing the motor prot. myosin II to enter, making AF contractile.

Fimbrin cross-links into tight bundles, excluding myosin II, why AF is not contractile.

The different spacing of α-actinins and fimbrin excludes one another.

Villin another example. Bundling prot. with two AF-binding sites close together in a single polypeptid chain. Helps 20-30 tightly bundled AFs in microvilli cross-link. Bild 8-12

### Filamin and cell motility Bild 13

Filamin promotes formation of a loose and high viscous gel. It forms a flexible, high-angle link between two adjacent AFs
Are used by some cells in order to extend thin sheet-like membrane projections, lamellipodia, helping them to crawl.

### Capping proteins

An uncapped AF can grow or depolymerize at both + and -, most rapidly at + end.

Binding of a + end capping prot. stabilizes the + end, blocking it, making it inactive.

AF can be capped at + or - or both.

ARP-complex (ARP 2/3 complex) Bild 15-16

Complex of proteins, including two actin-related proteins (ARPs), 45% identical to actin.

Catalyzes the nucleation needed to change the cell's shape and stiffness rapidly in respons to changes in the external environment. (also formins does this)

Nucleates AF from - end, allowing rapid elongation at + end.

Attaches of another AF, while bound to - end → building treelike web

Actin elongation and formins Bild 17-19

Formin: dimetric prot., nucleating prot.

Induces formation of actin bundles

Nucleates AF polymerization by capturing two monomers.

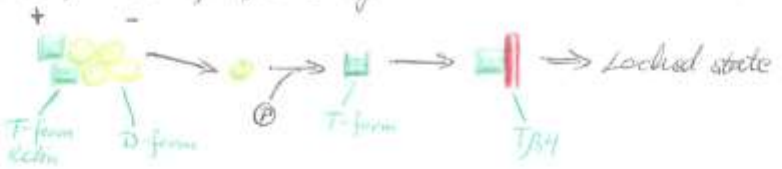
Remains associated at + end while growing rapidly. Other regions regulate activity and link to cell structures

Thymosin: sequesting prot. (binding to AM polymerization site)

Binds to AM, making it less favourable to polymerize

→ AM in locked state ⇒ remain soluble in subunit pool.

Ex. thymosin β4, binding to ATP-G-actin (T-form globular actin)




Profilin: recruits AM to AF, increase actin polymerization.

Binds to end associating with AF - end, exposing + binding end, and leaving ATP-cleft free. → Can bind to AF + end.

When AM binds to + end of AF, affinity for profilin is reduced → profilin falls off

Certain formins require that AM is bound to profilin for elongation. They have "whisker"-domains with sites for profilin or profilin-actin complex.
Gives higher rate of elongation.

## Cytoskeletal attachments to membranes

ERM-family prot. (named by three first members: ezrin, radixin, moesin)

Members of ERM required for maintenance of cell polarity and are involved in exocytosis and endocytosis

C-terminal binds to side of AF

N-terminal " cytoplasmatic face of transmembrane glyco-prot.

ex. CD44.

Attachments regulated by intra- and extracellular signals.

Two conformations

Active extended conf.: oligomerizes and binds to actin and transmemb. prot.

Folded conf.: N- and C-termini held together by intramolecular interactions.

## Tubulin and microtubules (MT)

Microtubules: subunit is tubulin, formed by α-tubulin and β-tubulin, held together by noncovalent bonds, (heterodimer), and has one site each for GTP/GDP.

Microtubules are hollow cylindrical structures, built from 13 parallell protofilaments composed of alterning α- and β-tubulin.

Has distinct structural polarity with an α or β in one direction, and opposite in the other.

## Thermal stability

Formation of filament of more than one protfilament

=> ends are dynamic, filament themselves resistant to thermal breakage. Addition/loss can occur at ends.

A breakage at the end: 1 longitudinal bond break, 2 lateral.

" in middle: Many bonds! => More energy required

=> Stability!

## Dynamic instability  Bild 21-23

$C_c(T) < C < C_c(D)$ => MT can undergo transitions between shrinking or growing state.

Intact MT has protofilaments of D-form subunits (with GDP), forced to linear conformation by lateral bonds in the wall.

T-form subunits (with GTP) formes a GTP-capped end, making the conformation stable.

Loss of cap could happen, if hydrolyzation of GTP into GDP is more rapid than subunit addition.

⟹ Conformational change in subunit conformation gives the protofilament a curved shape, less able to pack in MT.

•MT starts to shrink, called a catastrophe.

Regain of T-form subunit is still possible. If it is enough for a cap to be formed and growth can continue, it is called a rescue.

## Nucleation of microtubules

Are generally nucl at MTOC (microtubule-organizing center).

γ-TuRC (γ-tubilin ring complex): made of γ-tubilin, a tubilin specialized for MT growth.

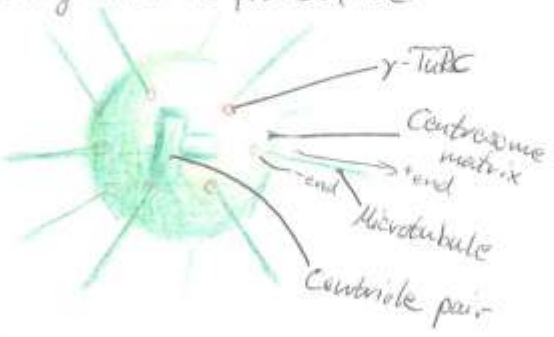
Two prot. binds to γ-tubilin, other helps create a ring of γ-tubilin.

Nucl. at −end, +end growing outward from MTOC.

## Centrosome

MTOC in animal cells, located near the nucleus.

MT nucl. at centrosome at -end, + end growing outward




Composed of centrosome matrix. Has more than 50 γ-TuRCs

Centrioles at center.

Centrioles: Pair of cyl. structures located at the center of a centrosome.
Organizes the centrosome matrix.
Duplicates during every cell cycle.
Consists of short cylinders of modified MTs, plus large number of accessory prot. Bild 27

Finding the center Bild 28
MTs allways arrange themselves with -end in the center of the cell.
Polymerization of the MTs, nucl by centrosome, pushes against walls, stabilizing centrosome at the center.

MAPs
Microtubule-associated proteins
Binds along the sides of MTs, stabilizing them against disassembly.
Can mediate interactions with other cell components.
MAP2: Has long projecting domain
Overexpressing => bundles of stable MTs, kept widely spaced
Tau: Shorter proj. domain
Overexpr. => bundles of closely packed MTs.

Katanin: MT severing prot.
Taxol: MT-specific drug. Binds to and stabilizes MT, causing a net increase in tubulin polymerization.

~~Microfilaments~~ Intermediate filaments Bild 34-36
Build up by individual polypeptides, elongated molecules with extended α-helical domain, forming a monomer.
A pair of monomers form a dimer, wounded into a coiled coil.
Two dimers line up, forming antiparallell tetramer. This is the soluble subunit.
Forms rope-like filament, packed in helical array with 16 dimers.

Keratin: most diverse IF family.
Ex. hair, nails, animal covering (claws, shells).

Neurofilaments (NF)
Found in high conc. along axons of vertebrate neurons.
Three types: NF-L
NF-M
NF-H

FtsZ protein
Tubilin homolog, found in procaryotes

Mbl
Actin homolog in bacteria

ParM
Actin homolog in bact.

4.1

Cell- och Molekylärbiologi 2
Föreläsning 4, 2009-01-27
Molecular Motors

Kap 16
Julie Grantham

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**Myosin Protein** → Motor prot. that moves along AFs. Some conformation. Most moves toward AF +end. Generates force for muscle

All kinds of myosin share similar motor domains.

C-terminal tail and N-terminal extensions are very diverse.

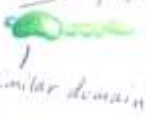



Bild 1

Ex. of functions: I: intracellular org (ex. petrusion) II: contractile activity in muscle and nonmuscle cells (cytokinesis and forward translokation of cell body). V: vesicle and organelle transp.

**Myosin II**

Structure: Globular head domain at N-term, followed by long aminoacid sequence forming extended coiled-coil (two chains wrapped). These domains form heavy chain.

Light chains: two chains binding close to N-term. head domain at each heavy chain.

The head domains have
- actin binding site
- ATP binding site

MyII can form bipolar thick filament by tail-tail interactions, with the heads on the outside, leaving a center bare zone without heads.

Bild 2, 3

**MyII motion → Motion**

The My heads bind ATP, hydrolyzing it. The energy is used to walk toward a AF plus end.

Stage 1: Myosin locked onto AF. Short-lived state
2: ATP bound to head → change in conformation, reducing affinity → AF released
3: Head displaced. ATP hydrolyzed
4: Binding AF → release of $P_i$ → Power stroke, releasing ADP and force-generating change in conformation, moving AF

→ New cykle, but new position

Bild 5

Speed of motion can be varied with ATPase rate, proportion of cykle time spent bound and with length of lever arm.
My V have longer arms than My II → longer swing → longer step. Bild 6

## Activation

Phosforylation of heavy or light chain can affect motor activity and thick filament assembly, in non-muscle cells.
Ex. Enz. MLCK causes My II to assume extended state
Phosforylation, in My II inactive state, at light chains, activates My II → Spontaneous self assembly, into bipolar filaments. Bild 7

## Muscle contraction

ATP-driven
Depending sliding network of Af arrays against My II arrays.

### Muscle cell

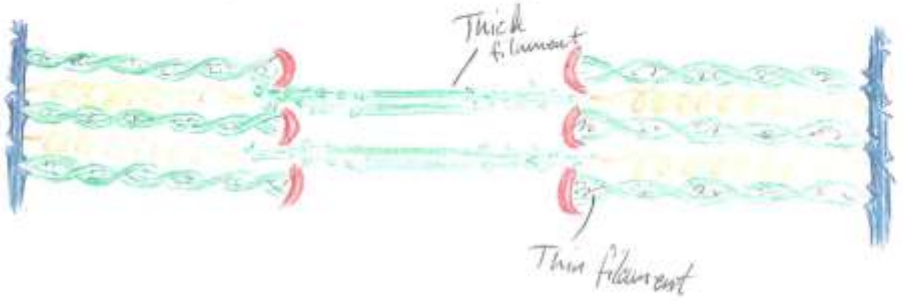
50 µm diameter, several cm long
Formed by fusion of many muscle cells.

### Myofibril

Cylindrical structure, often as long as the muscle cell.
Fibre made from repeated chain of tiny contractile units
= Sarcomeres
A muscle cell contains many myofibrils.

### Sarcomeres

Formed from array of parallell and partly overlapping thin filaments (actin) and thick filaments (myosin) Bild 8, 9



## Sarcoplasmatic Reticulum (SR)

SR is a kind of modified ER with huge surface area.

Incoming action potential activates $Ca^{2+}$-channel connected to T-tubule in T-tubule membrane → opening of $Ca^{2+}$-release channels in SR → $Ca^{2+}$ flooding into cytosol of muscle cell → Initiates contraction of each myofibril. Bild 11

## Tropnin effect on muscle contraction

$Ca^{2+}$-dependence of vertebrate skeletal muscle cell is due to a set of specialized accessory proteins. These are the following:

Tropomyosin: Elongated molecule, binding along the groove of actin helix.

Troponin: Complex of three polypeptides:

T - Tropomyosinbinding

I - Inhibitory

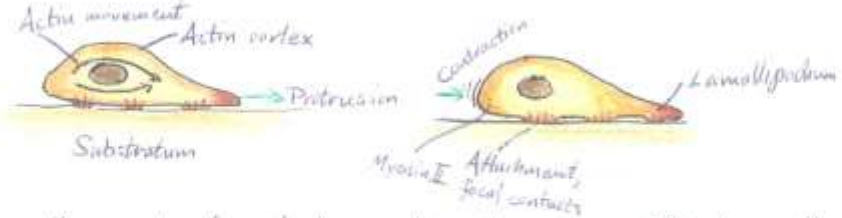
C - $Ca^{2+}$-binding

I binds actin and T. I-T complex pulls tropmyosin out of normal binding groove → Interferes with AF binding to My head.

$Ca^{2+}$ level raised → C, binding $Ca^{2+}$, causes I to release actin, letting tropomyosin go back to normal position

⟹ AF can interact with My head. Bild 12

## Cell movement




Cell crawling dependent on actin rich cortex beneath plasma membrane.

Protrusion: Actin rich structures push out at the front, attachment of lamellipodium moves edge forward.

Attachment: Actin cytoskeleton connects across plasma membrane to substratum.

Traction: Bulk of cytoplasm drawn forward by contraction at the rear, relaxing some tension.

Bild 14

Lamellipodium is a sheet like structure of cross-linked AF.
Advances at the front during cell movement.
During movement, ARP-complex mediates nucleation of AF.
Filaments elongate, pushing plasma membr. forward because of an anchorage at the behind. Cofilin disassembles older filaments. Bild 15

My motor proteins, especially MyII, generates traction forces.
MyII concentrated at the rear where it may help to push cell body forward. Bild 16
MyII bipolar filaments bind to AF in pulling them into a new orientation. Reorientation forms actin bundle recruiting more MyII. Bild 17

Activation of Rac, Rho (and Cdc42) and actin organisation
GTPases of Rho protein family triggers global structure rearrangements
Cdc42, Rac and Rho
Rho proteins act like switches controlling cell processes by cycling between active GTP-bound state and inactive GDP-bound state.
Cdc42: Triggers actin polymerization and bundling to form filopodia or microspikes. (Activated by G-prot-coupled recepter (yeast mating factor case))
Rac: Promotes actin polymerization at cell periphery → formation of lamellipodial extensions.
Rho: Promotes bundling of AF with MyII filaments and clustering of integrins and associated prot., forming focal contacts.
Bild 18-19
Primary determinants of cell polarity in budding yeast.
Bild 20, 21

## RNA localisation

To concentrate prot. at site of function, a cell can restrict synthesis of a prot. by localizing its mRNA molecules.
important when a cell divides
in some cells, mRNA transport is actin dependent.
The yeast mother and daughter cells ~~this~~ retain distinct identities, caused by regulatory protein, Ash1. Ash1 mRNA and prot are localized in the daughter cell (the growing bud)
Myosin V type is required. Bild 22

## Kinesin and Dynein

### Kinesin

Motor protein, moving along MT toward +end.
Have two heavy chains, two light chains per active motor. Motor domains form globular heads. Have elongated coiled-coil.
Most have motor d. at N-term, and C-term attaching to cargo.
(ex. Kinesin-1. Kinesin-13 opposite) Bild 23, 25

### Dyneins

→-end directed MT motor.
2 or 3 heavy chains, large variable number of intermediate and light chains.
Important for vesicle trafficking. Bild 23

### Kinesin motion

Kinesin moves its heads along MT in a hand-over-hand motion, like feet. One is allways bound, the other moves, depending on the binding of ATP/ADP.
High prossesivity. Bild 24

### Kinesin / Myosin

Motor domain of My larger. No amino acid similarities, but motor domains are built around nearly identical cores.

Motor protein function
- Has binding site for MT (kinesin) or AF (myosin)
- Has ATP-binding site
- Has machinery to translate ATP hydrolysis into conformational change.

Bild 26

| Muscle Myosin | Kinesin |
|---|---|
| Low processivity | High processivity |
| Moving by powerstroke | Walking stepwise along MT |
| ATP hydrolysis | ATP hydrolysis |
| Short time binding  | One head domain allways bound to MT  Time binding |
| Works as a group | Works alone |
| If bound to long → Interfering! | |

Dynein powerstroke

ATP-bound, stalk detached from MT.
ATP hydrolysis → attachment
Release of ADP + $P_i$ → Powerstroke

Bild 27

Kinesin and dynein cargo loading

Motor prot. mediate intracellular transp of organells.
For dynein, a large number of accessory prot. presence is required to associate with membrane enclosed organelles.

Bild 28

Kinesin vs Dynein

MT works as a highway system. - end inwards, + end outwards
Inward movement is rapid and smooth, outward jerky.
Caused by "war" between MT motor prot. kinesin and dynein


⊖ ⊕ Kinesin is stronger, winning the battle.

If kinesin light chain is phosforylated after ex. hormonal signal, kinesin is inactivated → Dynein can freely drag its cargo to the center.

Bild 29

## Cilia and flagella

Flaggella: found on sperm, and protozoa. Enables cells to swim through a liquid media, by the wave-like motion.

Cilia: shorter than flagella. Has a more whip-like motion, enabling a cell to rotate (propell) Bild 30

Axoneme: the bending of with causes movement of flagella or cilia. Composed of MTs, nine doublets in a ring around a single pair. Bild 31

Cilia dynein (axonemal) form bridges between doublet MTs. Bild 31

Activated (after ATP add) motor domain of dynein, dynein tries to walk along MT → Slide

Presence of linking prot. between MTs → Sliding prevented → Bending, waving motion. Bild 32

## Basal bodies

Nine sets of triplet MTs, forming lower portion of flaggella and cilia. Bild 33

~~Bot~~ them at cell surface.

## Microtubule organization

### Fibroblasts

The MTs +ends are pointed outward, with -ends at the middle, at the centrosome.

Kinesin attached vesicles move outward, dynein attached inward.

### Neurons

In the axon all MTs are oriented in the same direction: + ends forward toward terminals, -ends toward cell body. The MTs are staggered and overlapping.

In the dendrite MTs are parallell, but have mixed polarities.

AF line cortex of axons. IF forms neurofilaments, specialized IFs Bild 34-37

Cell- och Molekylärbiologi 2
Föreläsning 5, 2009-02-02
Intracellulär sortering

Kap 12
Magnus Holm

I en normal djurcell finns ca 1miljard proteiner, som består i 10-20 gener.
Alla proteiner måste transporteras till rätt ställe i cellen.
Det gör transport till en stor apparat i cellen. (cellulärsystem)
Man tror att tidiga eukaryoter hade sitt DNA i kontakt med cellmembranet, och processer skedde över detta. Fungerar för en liten cell.
Hos större prokaryoter kan man se invaginationer som fortfarande är i kontakt med plasmamembranet. En teori är att ett sådant avskiljts från membranet ⟹ ER.
Mitokondrier & och kloroplaster tros ha tagits upp av eukaryoter, och att ett symbiotiskt förhållande uppkommit.
Bild 3: Ribosomer vandrar längs RNA under translation. Bild 1, 2
Då prot-syntesen tar sin tid, börjar prot. vecklas ännu medan de skrivs.

Transporttyper.

Gated transport: Sker mellan kärnan och cytosolen, genom Nuclear Pore Complexes (NPCs). Selektiv.

Transmembrane transp: Protein Translocators transp specifika prot. från cytosol till ett topologiskt olikt område (avdelning) ex. ER. Prot. måste ofta vikas upp

Vesikulär transp: Membranomsluten transp Små sfäriska el. stora oregelbundna vesiklar. Knoppas av från lumen när de lastas, smälter ihop med målorganellen när de lastas av. Topologiskt lika! Bild 4