MCQ's

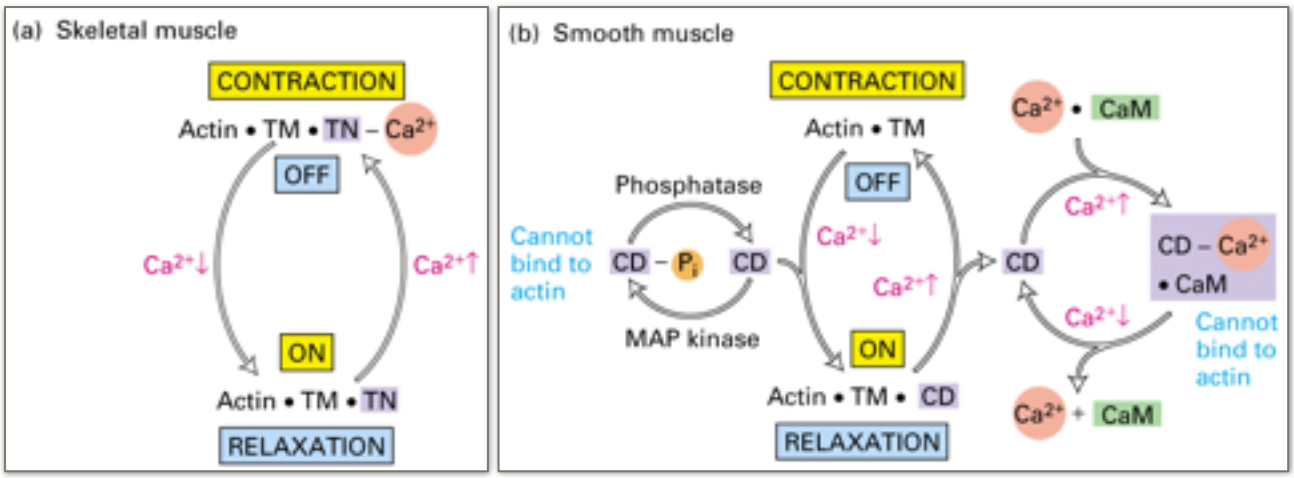
Muscle

Skeletal Muscle

- Ca^{2+} dependent reaction
- TM (Tropomyosin) & TN (Troponin) are lying over globular Actin — Ca^{2+} moves them out of the way to attach the Myosin head-group and activate contraction (need ATP)
- As $[Ca^{2+}]$ ↑, the TM-TN-Calcium complex moves out the way, and vice versa
- Isotonic (shortening)

Smooth Muscle

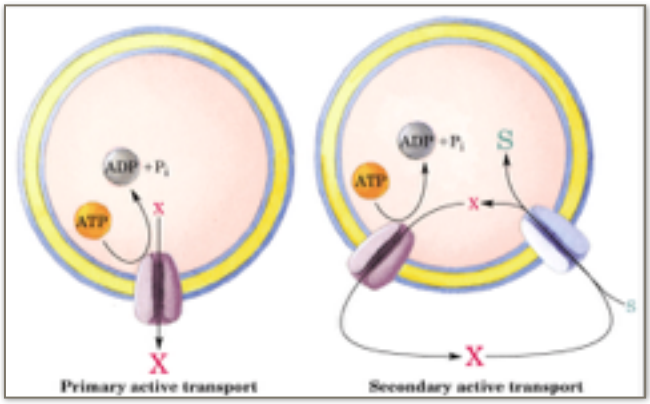
- Ca2+ dependent reaction
- Same concept applies when it comes to ↑ & ↓ in $[Ca^{2+}]$, but also depends on Caldesmon (CD), in that it cannot contract if it's bound (presence or absence is opposite to Ca^{2+}
  - CD phosphorylation prevents its binding, so when it's not phosphorylated it binds to Actin —> ∴ relaxation
  - Also, ↑ Ca^{2+} will take off CD and bind to it



Membrane Transports

- Uniport —> transporting only 1 molecule/ion at a time
- Symport —> transports 2 or more molecules/ions, in the same direction
- Antiport —> transport 2 or more molecules/ions, in opposite directions

- Different antiporters work at different pH's
- Secondary active transport —> using a uniport molecule to function a symport transporter



Calcium

Required for:

- each coagulation pathway (clotting)
- both skeletal & smooth muscle contraction (Ca2+ released from the Sarcoplasmic reticulum (SR) in the muscle cell)
- movement of cells due to signals (chemotaxis)
- Ca^{2+} levels in the cell is orders of magnitude lower compared to blood
- Towards the synaptic cleft, changes from using Na+ channels to Ca2+ channels, as it causes fusion of the membrane

Characterisation

- Nuke Blue —> used to stain dsDNA
- Pyronin Y —> used to stain RNA
- Nucleolus is a nuclear sub-compartment where most of the cell's rRNA is synthesised
- Tissue are fixed in Paraffin & Resin when observing thin slices under a microscope
- Resolving power —> ability to distinguish between 2 pixels/lights
- ↑ to ↓ resolution: TEM —> SEM/Fluorescence —> Light microscope

Type	Probe	Technique	Best Resolution	Penetration	Constraints
SEM	Electrons	Detects e^- back-scattered by the sample & it's focused using electromagnets	~1 nm	Surface	Sample must be in a vacuum

- Light Microscopy:
  1. Brightfield
  2. Phase Contrast
  3. Differential Interference Contract (DIC)
- GFP can be introduced into a plasmid and produce a fusion gene —> making a fusion protein
- Immunofluorescent microscopy:
  1. Sample chemically fixed to slide
  2. Primary antibodies added
  3. Fluorochrome-conjugated secondary antibody added
  4. Observed under microscope
- Fluorochrome Quantum Yield (QY) —> no. of emitted photons / no. of absorbed photons
- FRAP (Fluorescence Recovery After Photo-bleaching) —> bleaching refers to losing fluorescence and it tried to get back its original intensity but because it contains inactivated GFP, the fluoresce isn't as strong
- FRET (Fluorescence Resonance Energy Transfer) —> where the emission wavelength of one probe gets absorbed by the probe next to it
- FLIM (Fluoresce Lifetime Imaging Microscopy) —> the emitted wavelength is higher than the absorbed
- Time Domain Lifetime Imaging —> measuring the decrease in intensity of the photons emitted (decay)

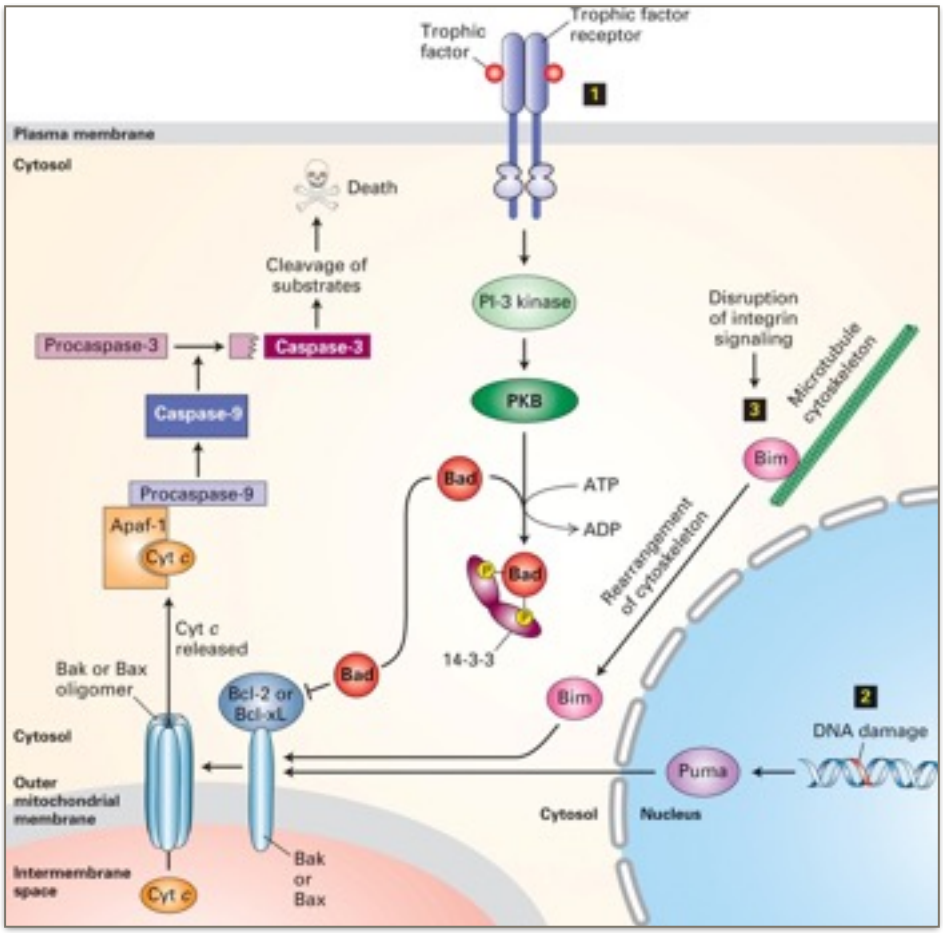
- Fluorescence Correlation Spectroscopy —> measures concentration and diffusion rates quantitatively, in order to analyse molecule interactions and transport process within living cells
- RICS (Raster Imaging Correlation Spectroscopy) —> fluorescence imaging analysis technique, enabling tracking of diffuse particles (spread over a wide area)

Apoptosis

- A process to manage the cell and recycle its contents
- Doesn't cause an inflammatory response
- Phagocytes engulf the cells after death

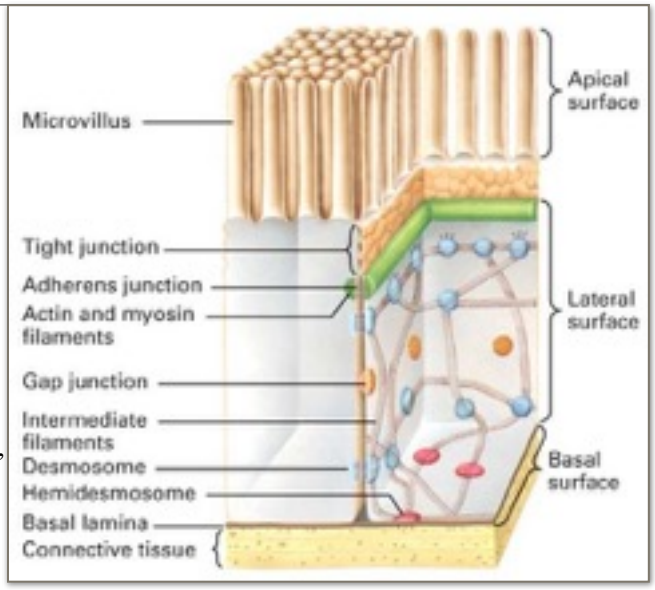
There are 3 ways it can get initiated:

1. Absence of Trophic Factors —> in its presence, it causes PKB to phosphorylate Bad (using ATP), which forms a complex with 14-3-3, hence unable to bind to Bcl-2. So in its absence, unphosphorylated Bad binds to Bcl-2, releasing Bax and allowing them to form the oligomeric channel, in turn allowing Cyt C to get out —> ∴ Apoptosis
2. DNA damage —> leads to induction of Puma, which binds to Bax, allowing it to form the oligomeric channel and let Cyt C out —> ∴ Apoptosis
3. Disruption of intern signalling —> removal of substratum induces Bim, having the same affect as Puma



- After Cyt C is out it binds to the CED4—Apaf-1 complex —> forms a circle with ~7 of them —> requires more caspases (they cleave > 100 different cell target proteins —> inducing apoptosis)
- Bcl-2 promotes cell survival (by preventing activation of Apaf-1, which Cyt C binds to after release)
- Apoptotic cells express Phosphatidylserine on the outer surface
- Autophagic Cell Death does not require Caspases

Tissue & Cell Junctions



- **Tight junctions** —> so things don't get between cells
  - epithelial cell's membranes are connected together via a honeycomb fashion
- **Gap junctions** —> allow small molecules through, e.g. Ca^{2+} & ions
  - SHUT: high Ca^{2+} or low pH
  - OPEN: low Ca^{2+} or high pH
- **Desmosomes** —> welding cells together (if tugged, adjacent cells also tug)
  - plaque is holding them together (in the ECM)
  - intermediate filaments extend out and connect to the cytoskeleton
- **Adherens junctions** —> similar to desmosomes, but used Cadherins and Actin instead of plaque and Intermediate Filaments, respectively
- **Hemidesmosomes** —> work between the cells and the matrix
- **Basal lamina** —> sheet of proteins (Laminin) which allow molecules in and out
  - Laminin is a T-shaped molecule: either side bind to Collagen and the top binds to Integrins

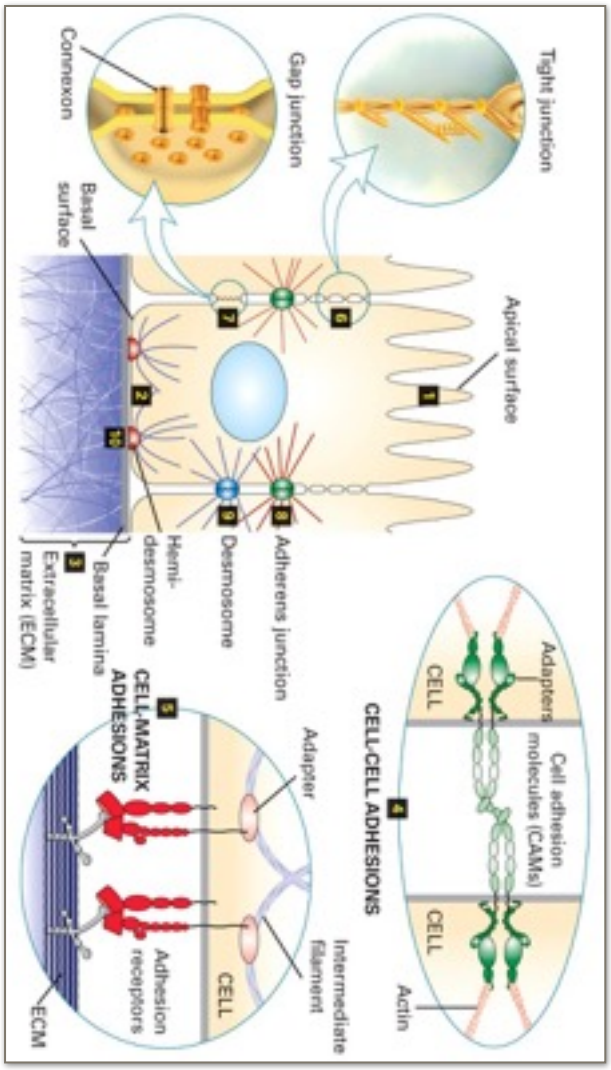


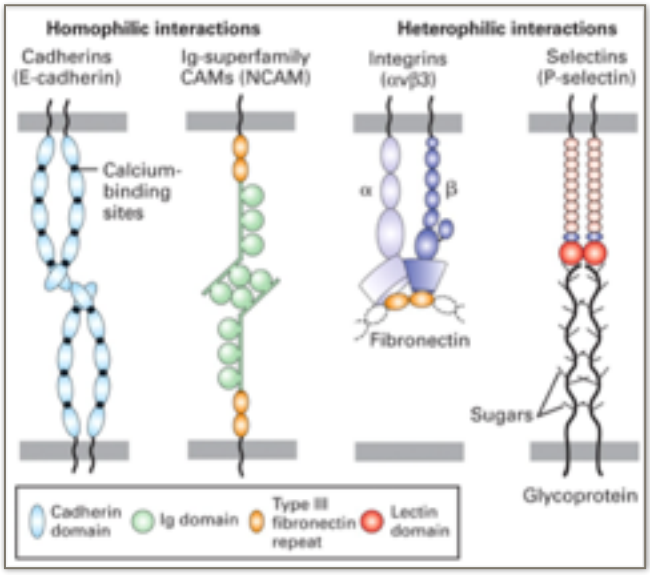
TABLE 19-2 Cell Junctions

JUNCTION	ADHESION TYPE	PRINCIPAL CAMs OR ADHESION RECEPTORS	CYTOSKELETAL ATTACHMENT	FUNCTION
Anchoring junctions				
1. Adherens junctions	Cell-cell	Cadherins	Actin filaments	Shape, tension, signaling
2. Desmosomes	Cell-cell	Desmosomal cadherins	Intermediate filaments	Strength, durability, signaling
3. Hemidesmosomes	Cell-matrix	Integrin ($\alpha 6\beta 4$)	Intermediate filaments	Shape, rigidity, signaling
Tight junctions	Cell-cell	Occludin, claudin, JAMs	Actin filaments	Controlling solute flow, signaling
Gap junctions	Cell-cell	Connexins, innexins, pannexins	Possible indirect connections to cytoskeleton through adapters to other junctions	Communication: small-molecule transport between cells
Plasmodesmata (plants only)	Cell-cell	Undefined	Actin filaments	Communication: molecule transport between cells

Cell Adhesion Molecules (CAMs)

- Homophilic: similar interaction proteins
- Heterophilic: different types of interaction proteins

1. **Cadherins** —> uses Ca^{2+} to stick together and they're mobile on the lipid bilayer
2. **Ig-superfamily** —> have binding domains that allow sticking together
3. **Integrins**
   - stick to:
     - Fibroblasts (ligand: Fibronectin)
     - T-lymphocytes (ligand: ICAM-1, ICAM-2)
4. **Selectins** (P-selectin) —> stick to glycoproteins



Epithelium

- **Simple cuboidal** —> single row of cubed-shaped cells, absorption & secretion, produced mucous
- **Simple columnar** —> single row of tall narrow cells, absorption & secretion, secretion of mucous
- **Stratified cuboidal**—>2 or more layers of square cells, secretes sweat,ovarian hormones, produced sperm
- **Keratinized stratified squamous** —> (has layer of dead cells packed with Keratin) multilayered epithelium, prevents water loss and penetration of organisms
- **Non-keratinized stratified squamous** —> (lacks the layer of dead cells) multilayered epithelium forming abrasion-resistant (scraping resistant) moist, slipper layer

Collagen & Biosynthesis of Fibrous Collagen

- Collagen in Basal Lamina is Collagen type IV
- N-terminal: forms Tetramers
- T-terminal: forms Dimers