

SLIDING FILAMENT THEORY

- Muscles only pull \therefore work pairs = **antagonistic muscles**
(muscle bends (flex) = flexor muscle)
straightens joint = extensor muscle

Role sarcopl. ret. in contracⁿ: (2)

- Ca^{2+} ch open
- \hookrightarrow released \rightsquigarrow sarcoplasm
- bind troponin \therefore tropomyosin moves
- \rightsquigarrow cross-bridges form

PROCESS

1. AP arrives motor end plate \rightarrow diff Ca^{2+} \rightsquigarrow neurone \rightarrow vesicles a.choline fuse presyn. memb
2. Acetylcholine = released & diff across synaptic cleft
3. Ach bind receptors sarcolemma \rightsquigarrow open Na^+ ch
 $\therefore Na^+$ diff in \rightarrow depolⁿ (spreads along memb)
4. AP spread along memb & down T-tubules
5. Ca^{2+} diff out sarcopl. ret
6. \hookrightarrow bind troponin Δ shape
 \therefore tropomyosin moves = expose myosin-bind-sites on actin filament.
7. Myosin heads form cross-bridges w actin fil $\xrightarrow{ATP \rightarrow ADP + P_i}$
8. \hookrightarrow tilt \therefore move actin $\rightarrow \leftarrow$ centre sarcomere (shortens) = **power stroke**
9. E from hydrolysis ATP cause myosin heads 'reset' o.g position w ATPase = break cross-bridge
M-heads bind actin further along \hookrightarrow as ATP bind
 \hookrightarrow ratchet mov repeats

ATP & ATPASE

Uses ATP \wedge :

1. actin-myosin cross bridges
2. power stroke (mov. myosin heads) [hyd. ATP]
3. Detachment myosin heads [new ATP attaches]
4. recovery stroke (reset m-heads) [hyd. ATP]

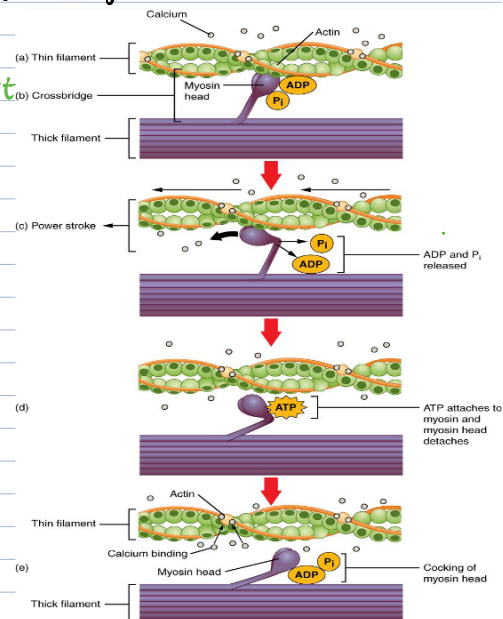
ATPase \cdot breaks \downarrow ATP \therefore prod E
 \hookrightarrow to break actin-myosin cross-bridges

Q: Why max tension x when sarcomere length = shortest

- max tension = max interacⁿ
- sarcomere shortest when overlap actin fil & length myosin \downarrow
- \downarrow cross-bridges formed

Q: How sarcomere shortens

- act-my cross bridge formed
- tilt m-head
- pull actin closer (moves)
- req ATP
- process repeats = ratchet



PRACTICAL

1. Fibres of fresh meat
2. In Ringer's solⁿ (w glucose vs w ATP)
3. Place on microscope slide
4. Record Δ length (%)
5. Add 1 drop ATP solⁿ
6. Control = distilled water

After death, cross bridges remain bound ^{rigor mortis}
Suggest why:

- respⁿ stops
- x ATP prod
- = req for break cross bridges
- x Ca²⁺ come

Control:

- Ringer solⁿ alone
- \therefore showⁿ it by self + cause contracⁿ (only w ATP)

Method

1. Dil & 5 dil %
2. Control w Ringer's solⁿ alone
3. Ruler mm measure Δ length fibres
4. Leave fibres 2 mins but (same time)
5. use same mass + SA fibres
6. add same vol ATP solⁿ for each conc & add 1cm³ on slide
7. ATP solⁿ = imitant \therefore gloves
8. Repeat x3 + mean.

Why results may x be valid

- may be dead
- Δ thickness strips (variable length)

Q: Tropomyosin role 'SFT'

1. covers myo-binding sites on actin
2. When Ca²⁺ bind troponin, tropomyosin moves
3. allow form cross-bridges (myo bind act.)

Q: Role myosin 'SFT'

1. hyd ATP \rightarrow ADP + Pi
2. \therefore \sim m-head tilt
3. m-head bind act \therefore form cross-bridges w act
4. ADP & Pi detach
5. m-head swings back \rightarrow prev. position
6. power stroke occurs (moves actin \rightarrow \leftarrow)
7. (new) ATP binds
8. cross-bridges break (\therefore m-head detatches from actin)

