

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 \leadsto sarcoplasm
- bind troponin \therefore tropomyosin moves
- \leadsto cross-bridges form

PROCESS

1. AP arrives motor end plate \rightarrow diff $\text{Ca}^{2+} \leadsto$ neurone \rightarrow vesicles a-choline fuse presyn. memb
 2. Acetylcholine = released & diff across synaptic cleft
 3. Ach bind receptors sarcolemma \leadsto open Na^+ ch
 $\therefore \text{Na}^+$ 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 filamenb.
 7. Myosin heads form cross-bridges w actin fil $\hookrightarrow \text{ATP} \rightarrow \text{ADP} + \text{P}_i$
 8. \hookrightarrow tilt \therefore move actin \rightarrow 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 \S 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]

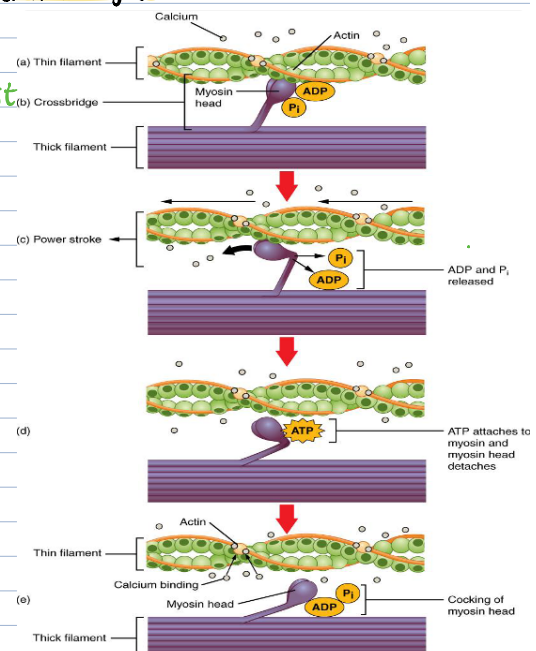
ATPase • breaks \downarrow ATP \therefore prod E
 \hookrightarrow to break actin-myosin cross-bridges

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
- 8 X Ca²⁺ come

Control:

- Ringer solⁿ alone
- show it by self to 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 b4 (same time)
5. use same mass + SA fibres
6. add same vol ATP solⁿ for each conc & add 1cm³ on slide
7. ATP solⁿ = irritant \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 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)

