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Muscle Physiology: Skeletal Muscle Contraction

Printable Flashcards — Pre-Med Biology

Sarcomere structure, neuromuscular junction, excitation-contraction coupling, cross-bridge cycle, ATP requirements, force control, motor units, fiber types, and muscle metabolism.

211 cards — Print double-sided, flip on long edge, then cut along dashed lines.

211 cards — Printable Flashcards

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1

Muscle contraction in one sentence (no fluff):

2

Sliding filament theory (the point of it):

3

The basic unit that actually contracts is the...

4

A muscle fiber is basically...

5

Skeletal muscle is called 'striated' because...

6

Skeletal muscle is voluntary because it's controlled by...

7

A motor unit is...

8

Why do we have small vs large motor units?

2

Muscle shortens because actin filaments slide past myosin; the filaments themselves don't get shorter.

1

A nerve signal releases Ca^{2+}, Ca^{2+} uncovers actin, and myosin pulls actin using ATP.

4

One muscle cell.

3

Sarcomere (Z line to Z line).

6

The somatic nervous system.

5

Sarcomeres create repeating light/dark band patterns.

8

Small units = fine control; large units = big force.

7

One motor neuron + all the muscle fibers it controls.

9

All-or-none rule applies to...

10

The whole muscle can contract 'more' or 'less' because...

11

The 2 big 'must-have' things for contraction are:

12

The functional unit of contraction is the {{c1::sarcomere}} (from {{c2::Z line}} to {{c3::Z line}}).

13

One motor neuron + all the fibers it controls =

14

Thick filament =

15

Thin filament =

16

Trap: troponin is part of the thick filament. True or false?

10

You recruit different numbers of motor units and change firing frequency.

9

A single muscle fiber (cell), not the whole muscle.

12

The functional unit of contraction is the sarcomere (from Z line to Z line).

11

Calcium (Ca^{2+}) and ATP.

14

Myosin.

13

Motor unit

16

False.

15

Actin + tropomyosin + troponin.

17

Z line is where... attaches.

18

M line is where... is anchored.

19

A band is...

20

I band is...

21

H zone is...

22

During contraction, which band stays the same length?

23

During contraction, what happens to I band and H zone?

24

During contraction, Z lines move...

18

Myosin (thick filaments) in the center.

17

Actin (thin filaments).

20

The thin-filament-only region (light band).

19

The length of the thick filament region (dark band).

22

A band.

21

The thick-filament-only region
in the center (no actin overlap).

24

Closer together.

23

They shrink.

25

Trap: myosin pulls actin toward the Z lines. True or false?

26

What actually shortens when a muscle contracts?

27

Actin filaments slide because myosin heads...

28

At rest, why can't myosin bind strongly to actin?

29

Ca^{2+} binds to... (this is always tested)

30

Trap: Ca^{2+} binds tropomyosin. True or false?

31

Troponin's job is basically to...

32

Tropomyosin's job is basically to...

26

Sarcomeres (Z to Z distance).

25

False.

28

Tropomyosin blocks the
myosin-binding sites on actin.

27

Attach, pivot (power stroke), detach, and repeat.

30

False.

29

Troponin (specifically troponin C).

32

Block myosin-binding sites on
actin when the muscle is relaxed.

31

Hold tropomyosin in place
and move it when Ca^{2+} binds.

33

Myosin head has two important abilities:

34

In the sarcomere, thick and thin filaments change length during contraction?

35

If a diagram shows a sarcomere getting shorter, what is actually changing?

36

Where is the 'no actin overlap' region located in the sarcomere?

37

Where is the 'no myosin' region located?

38

Why does skeletal muscle look striped (striated) under microscope?

39

A band (dark) length stays {{c1::the same}} during contraction; I band and H zone get {{c2::smaller}}.

40

Ca^{2+} binds {{c1::troponin}}, which moves {{c2::tropomyosin}} off actin's binding sites.

34

No. They keep the same length; overlap changes.

33

It binds actin and it has ATPase activity.

36

In the center: the H zone.

35

The overlap between actin and myosin increases.

38

Because A bands and I bands alternate.

37

Near the Z line: the I band (thin only).

40

Ca^{2+} binds troponin, which moves tropomyosin off actin's binding sites.

39

A band (dark) length stays the same during contraction; I band and H zone get smaller.

41

Band that stays the same length during contraction:

42

NMJ is the synapse between...

43

The neurotransmitter at the NMJ is...

44

Sequence at NMJ (fast version):

45

What triggers ACh vesicle release from the motor neuron terminal?

46

Trap: sodium entering the nerve terminal triggers ACh release. True or false?

47

ACh binds to what type of receptor on skeletal muscle?

48

What ion movement causes the end-plate depolarization?

42

A motor neuron and a skeletal muscle fiber.

41

A band

44

Nerve AP -> Ca^{2+} enters nerve terminal -> ACh released -> ACh binds receptor -> muscle depolarizes -> muscle AP.

43

Acetylcholine (ACh).

46

False.

45

Ca^{2+} entering the neuron through voltage-gated Ca^{2+} channels.

48

Mostly Na^{+} influx (some K^{+} efflux).

47

Nicotinic ACh receptor (ligand-gated cation channel).

49

End-plate potential is...

50

What stops the NMJ signal from lasting forever?

51

Trap: acetylcholinesterase is in the neuron terminal. True or false?

52

Botulinum toxin causes weakness because it...

53

Curare (classic example) causes paralysis because it...

54

If AChE is inhibited, what happens at the NMJ?

55

Most skeletal muscle fibers have... NMJs.

56

One motor neuron can control...

50

Acetylcholinesterase breaks down ACh.

49

A local depolarization at the motor end plate that can trigger a muscle action potential.

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52

Blocks ACh release from motor neurons.

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51

False (conceptually).

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54

ACh stays longer -> prolonged stimulation (can cause spasms then weakness).

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53

Blocks nicotinic ACh receptors on the muscle.

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56

Many muscle fibers.

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55

One (usually one motor end plate).

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57

The NMJ is designed to be reliable because...

58

After the muscle fiber action potential starts, the NMJ is done and the rest happens in the...

59

NMJ neurotransmitter is {{c1::acetylcholine}} (ACh). It binds {{c2::nicotinic}} receptors.

60

ACh release from the neuron terminal is triggered by {{c1::Ca^{2+}}} entering the terminal.

61

Enzyme that breaks down acetylcholine in the synaptic cleft:

62

Excitation-contraction coupling means...

63

Once the muscle action potential starts, it spreads along the...

64

T-tubules are...

58

Muscle fiber membrane/T-tubules/SR system.

57

One nerve AP usually triggers one muscle AP (high safety factor).

60

ACh release from the neuron terminal is triggered by Ca^{2+} entering the terminal.

59

NMJ neurotransmitter is acetylcholine (ACh). It binds nicotinic receptors.

62

Turning a muscle action potential into Ca^{2+} release and contraction.

61

Acetylcholinesterase

64

Inward folds of the sarcolemma that carry the action potential deep into the muscle fiber.

63

Sarcolemma (muscle cell membrane).

65

Sarcoplasmic reticulum (SR) is basically the...

66

When the AP travels down T-tubules, what is the immediate next big event?

67

What does Ca^{2+} do right after it's released from SR?

68

Triad (in skeletal muscle) is...

69

DHPR (dihydropyridine receptor) in skeletal muscle acts mainly as...

70

Ryanodine receptor (RyR) is basically the...

71

Skeletal muscle special point: the Ca^{2+} that triggers contraction comes mainly from...

72

Trap: skeletal muscle contraction requires Ca^{2+} to enter from the extracellular fluid every time. True or false?

66

SR releases Ca^{2+} into the cytosol.

65

Ca^{2+} storage organelle of muscle.

68

A T-tubule sandwiched by two SR terminal cisternae.

67

Binds troponin, moving tropomyosin and exposing actin binding sites.

70

Ca^{2+} release channel on the SR.

69

A voltage sensor in the T-tubule membrane.

72

False (for the basic concept).

71

The SR (internal store).

73

Why does Ca^{2+} rise and fall quickly in muscle?

74

SERCA pump is...

75

Trap: muscle relaxation is passive and doesn't need energy. True or false?

76

If Ca^{2+} stays high in the cytosol, what happens to contraction?

77

If SR can't release Ca^{2+}, then...

78

If SERCA pump is blocked, what happens?

79

The muscle AP travels along the membrane and down T-tubules to make sure...

80

AP down T-tubule -> SR releases {{c1::Ca^{2+}}} -> Ca^{2+} binds {{c2::troponin}} -> sites exposed -> contraction.

74

An ATP-powered pump that moves Ca^{2+} back into the SR.

73

Because SR releases it fast and SERCA pumps it back fast.

76

It keeps going (more cross-bridge cycling).

75

False.

78

Muscle has trouble relaxing because Ca^{2+} stays in cytosol longer.

77

Actin stays blocked and contraction can't start properly.

80

AP down T-tubule -> SR releases Ca^{2+} -> Ca^{2+} binds troponin -> sites exposed -> contraction.

79

All sarcomeres get the Ca^{2+} signal at the same time.

81

Main calcium storage structure in skeletal muscle:

82

Cross-bridge cycle is the repeating process where...

83

What has to happen first for a myosin head to bind actin?

84

The 'power stroke' is basically...

85

ATP does 3 big jobs in muscle:

86

Trap: ATP is needed for the power stroke itself. True or false?

87

Myosin detaches from actin when...

88

Trap: ATP makes myosin bind actin more strongly. True or false?

82

Myosin binds actin, pulls (power stroke), detaches with ATP, and re-cocks.

81

Sarcoplasmic reticulum (SR)

84

Myosin head pivoting and pulling actin toward the center of the sarcomere.

83

Actin binding sites must be exposed (Ca^{2+} -> troponin -> tropomyosin moves).

86

Sort of false / misleading.

85

Detach myosin from actin, re-cock myosin head (via ATP hydrolysis), and pump Ca^{2+} back into SR.

88

False.

87

ATP binds to the myosin head.

89

After ATP binds and myosin detaches, ATP is hydrolyzed to...

90

During the power stroke, what is released from myosin first?

91

If ATP is missing, what happens to the muscle?

92

Rigor mortis happens because...

93

Trap: rigor mortis happens because there is too much ATP. True or false?

94

If Ca^{2+} is low but ATP is present, what happens?

95

If Ca^{2+} is high but ATP is low, what happens?

96

How does one myosin molecule increase force?

90

Pi (and then ADP).

89

ADP + Pi, which 'cocks' the myosin head into a high-energy position.

92

ATP runs out, so cross-bridges can't detach (and Ca^{2+} can leak out).

91

It becomes stiff because myosin can't detach from actin (rigor).

94

Muscle relaxes because tropomyosin blocks actin sites.

93

False.

96

By having many heads cycling, and many myosin molecules working in parallel.

95

Cross-bridges form and then get stuck (can't detach well).

97

Why does force increase when more cross-bridges are attached at once?

98

If the muscle is stretched too far, force drops because...

99

If the muscle is extremely shortened, force can drop because...

100

Myosin is an ATPase. That means...

101

Trap: actin is an ATPase that powers the contraction. True or false?

102

If a question asks 'what causes detachment of cross-bridges?', the answer is usually...

103

If a question asks 'what exposes myosin-binding sites on actin?', the answer is usually...

104

Cross-bridge cycling stops when...

98

Actin and myosin overlap becomes too small for enough cross-bridges.

97

More parallel pulls = more total tension.

100

It can split ATP to provide energy for movement.

99

Filaments interfere/overlap poorly and cross-bridge geometry becomes less effective.

102

ATP binding to myosin.

101

False.

104

Ca^{2+} is removed from troponin and binding sites get covered again.

103

Ca^{2+} binding to troponin.

105

ATP binding to myosin causes {{c1::detachment}} from actin; ATP hydrolysis cocks the myosin head.

106

No ATP -> cross-bridges can't {{c1::detach}} -> stiffness (rigor).

107

Ion that binds troponin to start contraction:

108

Cross-bridge cycle order (simple):

109

What event directly ends a single power stroke?

110

If ATP can't bind to myosin, the muscle would be...

111

If ATP can't be hydrolyzed (split), then myosin can't...

112

Why does contraction require repeated cross-bridge cycles, not one big pull?

106

No ATP -> cross-bridges can't detach -> stiffness (rigor).

105

ATP binding to myosin causes detachment from actin; ATP hydrolysis cocks the myosin head.

108

1) Ca^{2+} exposes sites, 2) myosin binds actin, 3) power stroke, 4) ATP binds -> detach, 5) ATP hydrolysis -> re-cock.

107

Ca^{2+}

110

Stiff (myosin can't detach).

109

ADP leaves and the head is stuck until ATP binds to detach it.

112

Because each myosin head moves actin only a small distance per stroke.

111

Re-cock/reset properly for repeated power strokes.

113

Force is higher when... Ca^{2+} is higher because...

114

Trap: Ca^{2+} provides the energy for the power stroke. True or false?

115

In one line: Ca^{2+} is for... and ATP is for...

116

Scenario: Ca^{2+} release is normal, but ATP is low (no oxygen + no fuel). What happens?

117

Ca^{2+} is the '{{c1::on switch}}' (exposes sites). ATP is the '{{c2::motor fuel}}' (cycling + relaxation).

118

Relaxation starts when...

119

What actively removes Ca^{2+} from the cytosol in skeletal muscle?

120

Trap: Ca^{2+} diffuses back into SR without energy. True or false?

114

False.

113

More Ca^{2+} exposes more binding sites -> more cross-bridges can form.

116

Initial contraction happens, then relaxation fails and force drops (fatigue/stiffness risk).

115

Ca^{2+} exposes binding sites; ATP powers cycling and relaxation.

118

Ca^{2+} is removed from the cytosol (pumped back into SR).

117

Ca^{2+} is the 'on switch' (exposes sites). ATP is the 'motor fuel' (cycling + relaxation).

120

False.

119

SERCA pumps Ca^{2+} back into the SR (uses ATP).

121

If ACh release stops, does the muscle instantly relax?

122

What physically blocks actin sites again during relaxation?

123

Why does low ATP make relaxation hard even if the nerve stops firing?

124

Trap: AChE inhibitors always cause stronger muscles. True or false?

125

Relaxation needs {{c1::ATP}} because Ca^{2+} is pumped back into the SR by {{c2::SERCA}}.

126

Protein that blocks actin binding sites when relaxed:

127

A single brief contraction from one stimulus is called a...

128

Why can a second stimulus make a stronger contraction if it comes quickly?

122

Tropomyosin moves back to cover the sites (when Ca^{2+} leaves troponin).

121

Not instantly, but it starts: APs stop -> Ca^{2+} release stops -> Ca^{2+} is pumped back -> relaxation follows.

124

False.

123

You need ATP to detach myosin and pump Ca^{2+} back into SR.

126

Tropomyosin

125

Relaxation needs ATP because Ca^{2+} is pumped back into the SR by SERCA.

128

Because Ca^{2+} hasn't been fully cleared yet -> more cross-bridges can form (summation).

127

Twitch.

129

Summation means...

130

Trap: a stronger stimulus makes a bigger action potential in the same muscle fiber. True or false?

131

Two ways the body increases whole-muscle force:

132

Recruitment (spatial summation) means...

133

Size principle (high-yield idea): recruitment usually goes...

134

Tetanus is...

135

Incomplete vs complete tetanus:

136

Why does tetanus produce more force than a single twitch?

130

False.

129

Force increases when stimuli arrive before full relaxation.

132

Activating more motor units at the same time.

131

Recruit more motor units and increase firing frequency (summation).

134

A sustained strong contraction from very rapid stimuli (no time to relax).

133

Small motor units first, then larger ones as more force is needed.

136

Ca^{2+} stays high -> more cross-bridges engaged continuously.

135

Incomplete = tiny relaxations between twitches; complete = smooth sustained plateau.

137

Trap: tetanus means the muscle is dead. True or false?

138

Isometric contraction means...

139

Isotonic contraction means...

140

Concentric contraction means...

141

Eccentric contraction means...

142

Trap: eccentric contraction means the muscle isn't contracting. True or false?

143

In general, which can generate more force: eccentric or concentric?

144

Why does a whole muscle have smooth force output, not jerky twitches?

138

Tension changes but muscle length stays the same.

137

False.

140

Muscle shortens while generating force.

139

Muscle length changes while tension is roughly constant.

142

False.

141

Muscle lengthens while generating force (controlling a load).

144

Because motor units fire at different times and summation blends forces.

143

Eccentric (usually).

145

Muscle fatigue means...

146

Trap: fatigue means the muscle ran out of Ca^{2+}. True or false?

147

If you stimulate a muscle more often (higher frequency), force usually...

148

If you recruit more motor units, force...

149

Scenario: a person can still generate a twitch but can't sustain force with repeated contractions. Think...

150

Whole muscle force increases by {{c1::motor unit recruitment}} and {{c2::higher firing frequency}} (summation).

151

Isometric: length {{c1::same}}.
Isotonic: length {{c2::changes}}.

152

Sustained contraction from rapid repeated stimulation:

146

Usually false.

145

A decline in ability to maintain force.

148

Increases.

147

Increases (up to a max) because of summation/tetanus.

150

Whole muscle force increases by motor unit recruitment and higher firing frequency (summation).

149

Fatigue/ATP/metabolic limits rather than 'no NMJ signal'.

152

Tetanus

151

Isometric: length same. Isotonic: length changes.

153

Length-tension relationship (one idea):

154

If a muscle is stretched too far, force drops mainly because...

155

If a muscle is very shortened, force can drop because...

156

Force-velocity concept (simple): if the load is heavier, shortening velocity is...

157

If load becomes too heavy to lift, the contraction becomes...

158

Why can skeletal muscle go into tetanus but cardiac muscle can't (high-level)?

159

Trap: a stronger nerve AP makes stronger muscle fiber AP. True or false?

160

Why do small motor units get recruited first? (one reason)

154

Fewer cross-bridges can form (less overlap).

153

Max force happens at an optimal muscle length where actin-myosin overlap is best.

156

Slower.

155

Actin filaments overlap too much and myosin can't pull effectively.

158

Skeletal muscle has a short refractory period; cardiac has a long one.

157

Isometric (tension without shortening).

160

It's energy-efficient and gives smoother control.

159

False.

161

Muscle has only a tiny store of ATP. So during activity, it must...

162

Fastest way to regenerate ATP for a few seconds is...

163

Next quick ATP source when oxygen can't keep up is...

164

Most ATP for long-duration activity comes from...

165

Aerobic metabolism is slower to start because...

166

Lactate rises during intense exercise because...

167

Trap: lactate is pure 'poison waste' and useless. True or false?

168

Muscle soreness 2 days after exercise (DOMS) is mainly from...

162

Creatine phosphate (phosphocreatine) system.

161

Regenerate ATP continuously.

164

Aerobic metabolism (mitochondria).

163

Anaerobic glycolysis.

166

Anaerobic glycolysis is being used a lot.

165

It depends on oxygen delivery and mitochondrial pathways ramping up.

168

Micro-damage and inflammation, not lactate.

167

False.

169

Slow-twitch (Type I) fibers are best for...

170

Fast-twitch fibers are best for...

171

Why are slow-twitch fibers 'red'?

172

Fast glycolytic fibers fatigue faster mainly because...

173

In general, which fiber type has more mitochondria: slow or fast glycolytic?

174

In general, which fiber type has higher peak power: slow or fast?

175

Why do fast fibers contract faster? (basic)

176

Which energy system dominates in a 100 m sprint (roughly)?

170

Quick, powerful movements (sprinting, jumping).

169

Endurance: long, repeated activity.

172

They rely more on anaerobic metabolism and have fewer mitochondria/capillaries.

171

More myoglobin and more blood supply (capillaries).

174

Fast-twitch.

173

Slow-twitch (Type I).

176

Phosphocreatine + anaerobic glycolysis.

175

They have faster myosin ATPase activity and Ca^{2+} handling.

177

Which energy system dominates in a long run (endurance)?

178

Trap: aerobic metabolism makes ATP faster than phosphocreatine. True or false?

179

Simple comparison: phosphocreatine vs aerobic metabolism

180

Endurance fibers (Type I) have lots of {{c1::mitochondria}} and {{c2::myoglobin}} -> fatigue-resistant.

181

Immediate ATP backup: {{c1::phosphocreatine}}. Longer duration ATP: {{c2::aerobic metabolism}}.

182

Protein in muscle that stores oxygen and makes it look red:

183

Sarcolemma =

184

Sarcoplasm =

178

False.

177

Aerobic metabolism.

180

Endurance fibers (Type I) have lots of mitochondria and myoglobin -> fatigue-resistant.

179

PCr = very fast, short duration. Aerobic = slower start, huge endurance.

182

Myoglobin

181

Immediate ATP backup: phosphocreatine. Longer duration ATP: aerobic metabolism.

184

Muscle cell cytoplasm.

183

Muscle cell membrane.

185

Sarcoplasmic reticulum is basically... compared to regular cell organelles

186

Motor end plate is...

187

Trap: nicotinic ACh receptors are voltage-gated channels. True or false?

188

Trap: ACh binds and directly releases Ca^{2+} from the SR. True or false?

189

Myasthenia gravis (basic idea) causes weakness because...

190

Quick compare: botulinum vs curare

191

Trap: If ACh receptors are blocked, Ca^{2+} release from SR still happens normally. True or false?

192

If you see 'weakness that gets worse with repeated use', think NMJ problem like...

186

The specialized region of muscle membrane at the NMJ with lots of ACh receptors.

185

Specialized smooth ER that stores Ca^{2+}.

188

False.

187

False.

190

Botulinum blocks ACh release.
Curare blocks ACh receptors.

189

ACh receptors at the NMJ are blocked/damaged by antibodies -> weaker end-plate potentials.

192

Myasthenia gravis (classic pattern).

191

False.

193

Trap: ATP is only needed to start contraction, not to keep it going. True or false?

194

If a question asks 'what happens if Ca^{2+} can't bind troponin?', answer:

195

If a question asks 'what happens if tropomyosin can't move?', answer:

196

If a diagram shows A band shrinking during contraction, what should you think?

197

If a question says 'muscle gets shorter because myosin filaments shorten', that's...

198

Sarcolemma = muscle {{c1::cell membrane}}. Sarcoplasm = muscle {{c2::cytoplasm}}.

199

Scenario: voltage-gated Ca^{2+} channels in the motor neuron terminal are blocked. What happens?

200

Scenario: ACh is released normally, but nicotinic receptors are blocked. What happens?

194

Binding sites stay covered -> weak/no contraction.

193

False.

196

The diagram is wrong or you're misreading it (A band stays constant).

195

Actin sites stay blocked -> weak/no contraction.

198

Sarcolemma = muscle cell membrane.
Sarcoplasm = muscle cytoplasm.

197

Wrong. The filaments slide; they don't shorten.

200

No end-plate depolarization -> no muscle AP -> no contraction.

199

Less/no ACh release -> weak/no muscle action potential -> paralysis/weakness.

201

Scenario: muscle AP is normal, but ryanodine receptors on SR are blocked. What happens?

202

Scenario: Ca^{2+} release is normal, but SERCA is slow. What do you see?

203

Scenario: ATP is available, but Ca^{2+} never rises. What happens?

204

Scenario: Ca^{2+} is high, but ATP is missing. What happens?

205

Quick: which protein is the 'motor' that moves?

206

Quick: which protein is the 'track' that gets pulled?

207

If you forget everything, remember this chain:

208

Nerve AP -> {{c1::ACh}} -> muscle AP -> {{c2::SR Ca^{2+} release}} -> {{c3::troponin}} -> contraction.

202

Slow relaxation / cramp-like prolonged contraction.

201

No Ca^{2+} release -> no strong contraction.

204

Stiffness (cross-bridges can't detach) and poor relaxation.

203

No contraction (actin sites stay blocked).

206

Actin.

205

Myosin.

208

Nerve AP -> ACh -> muscle AP -> SR Ca^{2+} release -> troponin -> contraction.

207

Nerve AP -> ACh -> muscle AP -> SR Ca^{2+} release -> troponin -> cross-bridges -> force.

209

Neurotransmitter at skeletal muscle NMJ:

210

Protein that Ca^{2+} binds to (thin filament complex):

211

Band that shrinks during contraction (one answer):

210

Troponin

209

Acetylcholine (ACh)

211

I band (also H zone shrinks)