Work away! Thanks sincerely for making our poster sessions tops!...

**BI 199 APWT Discussion 13**...Lucky!!!

I. **Announcements** Poster Session Group I next Tuesday, May 20! Q? No Discussion this Thursday designed for time to work on posters! Hooray!!

   A. Are deep squats a safe & viable exercise? B Schoenfeld & M Williams.
   B. Exploring the front squat. SP Bird & S Casey.

III. **How Skeletal Muscles Work & Adapt**
   Please see resources on reserve in Science Lib
   A. What is muscle made of?
   B. How is muscle organized?
   C. What do thick filaments look like?
   D. What do thin filaments look like?
   E. How do muscles contract?
   F. How do muscles adapt?
      Hypertrophy, atrophy, fiber types

IV. **Questions/Discussion?**
Muscle fiber or cylindrical cell

“Threads” ≡ Myofibrils

Nucleii

Dark-Light...bands ≡ Overlapping thick & thin filaments

x1000
**Golf Club Analogy?**

(a)

- Actin binding site
- Myosin ATPase site
- Heads
- Tail
- 100 nm

(b)

- Cross bridges
- Myosin molecules

LS 2006, cf: LS 2012 fig 8-4
Broccoli Analogy?

Myosin Heads

Myosin Tails

Bare Zone

Myosin Heads
Rope Climb or Tug of War
Grasp, then Regrasp!
Summary
1. Acetylcholine released by axon of motor neuron crosses cleft and binds to receptors/channels on motor end plate.

2. Action potential generated in response to binding of acetylcholine and subsequent end plate potential is propagated across surface membrane and down T tubules of muscle cell.

3. Action potential in T tubule triggers $Ca^{2+}$ release from sarcoplasmic reticulum.

4. Calcium ions released from lateral sacs bind to troponin on actin filaments; leads to tropomyosin being physically moved aside to uncover cross-bridge binding sites on actin.

5. Myosin cross bridges attach to actin and bend, pulling actin filaments toward center of sarcomere; powered by energy provided by ATP.

6. $Ca^{2+}$ actively taken up by sarcoplasmic reticulum when there is no longer local action potential.

7. With $Ca^{2+}$ no longer bound to troponin, tropomyosin slips back to its blocking position over binding sites on actin; contraction ends; actin slides back to original resting position.
Relaxation Phase

1. Excitation by nerve fiber
2. Conduction by T-tubules
3. \( Ca^{2+} \) release by SR

Contractile Phase

SOURCE: VPL + Diann N Laing, 1989
A. Malcolm Campbell
Davidson College, Davidson, NC
http://www.bio.davidson.edu/courses/movies.html

Musclcp.mov
Adaptations to Exercise?

Body Levels of Organization?
Which Body System?
Adaptations to Exercise?
Mode, Intensity, Duration, Frequency, Distribution of Training Sessions? Conditions of Environment? Individual?
Adaptations to Skeletal Voluntary Muscle
Atrophy
 decrease in size & strength

Hypertrophy
 increase in size & strength
Skeletal Muscle

- Hypertrophy
- Hyperplasia
- Atrophy
Women & Hypertrophy?
What happens in muscles at cellular & subcellular levels?
Hypertrophy: Increased Number of Myofibrils
Thick & Thin Filaments
Myosin & Actin Molecules
# Characteristics of Skeletal Muscle Fibers

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Slow Oxidative (Type I)</th>
<th>Fast Oxidative (Type Ila)</th>
<th>Fast Glycolytic (Type IIb)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myosin-ATPase Activity</td>
<td>Low</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>Speed of Contraction</td>
<td>Slow</td>
<td>Fast</td>
<td>Fast</td>
</tr>
<tr>
<td>Resistance to Fatigue</td>
<td>High</td>
<td>Intermediate</td>
<td>Low</td>
</tr>
<tr>
<td>Aerobic Capacity</td>
<td>High</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Anaerobic Capacity</td>
<td>Low</td>
<td>Intermediate</td>
<td>High</td>
</tr>
<tr>
<td>Mitochondria</td>
<td>Many</td>
<td>Many</td>
<td>Few</td>
</tr>
<tr>
<td>Capillaries</td>
<td>Many</td>
<td>Many</td>
<td>Few</td>
</tr>
<tr>
<td>Myoglobin Content</td>
<td>High</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Color of Fibers</td>
<td>Red</td>
<td>Red</td>
<td>White</td>
</tr>
<tr>
<td>Glycogen Content</td>
<td>Low</td>
<td>Intermediate</td>
<td>High</td>
</tr>
</tbody>
</table>
Changes in Muscle Due to Endurance Training

↑ Mitochondria, # & size
↑ Mitochondrial (aerobic) enzymes
  including those specific for fat burning
↑ Vascularization of muscles (better blood flow)
↑ Stores of fat in muscles accompanied by
↓ Triglycerides/fats in bloodstream
↑ Enzymes: activation, transport,
  breakdown (β-oxidation) of fatty acids
↑ Myoglobin (enhances O₂ transport)
↑ Resting energy levels which inhibit
  sugar breakdown
↑ Aerobic capacity of all three fiber types.
Changes in Muscle Due to Strength Training

↑ Size of larger fast vs smaller slow fibers
↑ CP as well as creatine phosphokinase (CPK) which enhances short-term power output
↑ Key enzymes which help store and dissolve sugar including glycogen phosphorylase (GPP) & phosphofructokinase (PFK)
↓ Mitochondrial # relative to muscle tissue
↓ Vascularization relative to muscle tissue
Splitting of fast fibers? Hyperplasia?
With growth hormone (GH), androgenic-anabolic steroids (AAS)?
Which end of continuum?

+ 

Which energy nutrient/s?
+ Which specific muscles?
Discussion
Comments
Questions?