I. **Announcements** Lab 6, Pulmonary Function Testing (PFT) + optional notebook check today. Exam II Dec 8 Monday, 8 am!

II. **Introduction to PFT Lab 6** Pulmonary Function Testing

III. **Connections: Muscle Contraction+Adaptation** DC Mod 12+

A. Review of structure + banding pattern? LS fig 8-3, fig 8-7

B. How do muscles contract? LS fig 8-6, 8-10, 8-11 +…

C. Summary of skeletal muscle contraction with videosCourtesy David Bolinsky, XVIVO & Malcolm Campbell, Department of Biology, Davidson College, NC +…

D. Exercise adaptation variables LS ch 8 pp 210-214

*mode, intensity, duration, frequency, distribution of training sessions, individual & environmental factors*

E. *Endurance vs. Strength* training continuum? fiber types…
Hey baby, what's your sign?

Cancer.
Lab 6 Review: Pulmonary Function Testing (PFT)
NB: Should be able to blow out ≥ 75 - 85% of VC/FVC in 1 second! That's FEV$_{1.0}$/FVC ≥ 0.75 – 0.85. If less, may indicate asthma or other lung disease.
PFT measures all lung volumes & capacities (sum of > 2 volumes). Subject relaxes & breathes normally into and out of tank.
Spirogram graphing complete PFT from computer simulation.

TV = Tidal volume (500 ml)
IRV = Inspiratory reserve volume (3,000 ml)
IC = Inspiratory capacity (3,500 ml)
ERV = Expiratory reserve volume (1,000 ml)
RV = Residual volume (1,200 ml)
FRC = Functional residual capacity (2,200 ml)
VC = Vital capacity (4,500 ml)
TLC = Total lung capacity (5,700 ml)
Vitalometer → Can only measure Vital Capacity (VC). No graph paper, so no time component.
Inhale air in room maximally!

NB: noseclip & mouthpiece!
Exhale into tube maximally!
More modern-day computerized Pulmonary Function Testing

Complete with HH!
Happy Helpers!
How to put together?
Viola!!
Sample subject setup
Thoughtful, identical twin, group partner with incredible quickness, speed & agility!
Q about lab?

Sample data!

Max I

FVC

Max E
Questions/Discussion?
Sarcomere

Triad \equiv T \text{ tubule abutting cisternae}

Mitochondria

Myofibril

Sarcomere
A Band = Dark Band
Anisotropic = Light Can’t Shine Through

I Band = Light Band
Isotropic = Light Can Shine Through
What do we guess happens at the molecular level?
Relaxed: No Cross-Bridge Binding

(a) Relaxed

1. No excitation.
2. No cross-bridge binding because cross-bridge binding site on actin is physically covered by troponin-tropomyosin complex.
3. Muscle fiber is relaxed.

LS 2012 fig 8-6a
Excited: Calcium Triggers Cross-Bridge Binding

(b) Excited

1. Muscle fiber is excited and $\text{Ca}^{2+}$ is released.

2. Released $\text{Ca}^{2+}$ binds with troponin, pulling troponin–tropomyosin complex aside to expose cross-bridge binding site.

3. Cross-bridge binding occurs.

4. Binding of actin and myosin cross bridge triggers power stroke that pulls thin filament inward during contraction.

LS 2012 fig 8-6b
Rope Climb or Tug of War
Grasp, then Regrasp!
Summary
Brain

neurons in motor cortex

Spinal Cord

anterior motoneuron

Muscle

neuromuscular junctions

peripheral nerve
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 passively slides back to original resting position.
David Bolinsky, XVIVO
Rocky Hill, CT
http://www.xvivo.net/

http://www.youtube.com/watch?v=BMT4PtXRCVA

http://www.vetmed.wsu.edu/van308/muscleanimation.htm

A. Malcolm Campbell
Davidson College, Davidson, NC
www.bio.davidson.edu/courses/movies.html
http://www.bio.davidson.edu/misc/movies/musclcp.mov

Musclcp.mov
Adaptations to Exercise?
Mode, Intensity, Duration, Frequency, Distribution of Training Sessions? Conditions of Environment? Individual?
Adaptations to Exercise?

Body Levels of Organization?
Which Body System?
Echocardiography documents hypertrophy...
Cardiac Adaptations to Exercise:  
1 Endurance vs. 2 Strength Training

NB: ① > ↑LBM
As muscles tug on bones, bones get stronger, too!...many systems adapt!!
Muscle Adaptations to Exercise
Atrophy
*decrease in size & strength*

Hypertrophy
*increase in size & strength*
Skeletal Muscle

- Hyperplasia
- Hypertrophy
- 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 IIa)</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 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)?
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.
Which end of continuum?

+ 

Which energy nutrient/s?
+ Which specific muscles?
Dancing can be super aerobic exercise, too, & you don’t have to be a star!
Extremes of the energy continuum!