Anatomy and Physiology
Muscular System
Part # 1
Anatomy, Contraction Needs, Fatigue

Fall 2008
Properties of Muscle Tissue

- Myo = muscle; sarco = muscle
- Can contract/shorten
- Lengthening is passive
- Can only pull
- Ligaments attach bone to bone
- Tendons attach muscle to bone
- Fuse with bone periosteum
Basic Muscle Anatomy
• Tendon (fascia) fuses with periosteum
• Muscle is bundle of fibers (protein)
• Each fiber – **fasciculus**
• Each fasciculus is bundle of fibers – **fibrils**
  Each fibril is one muscle cell
  Histology?
  Multinucleated, striated
  Cytoplasm – sarcoplasm
  ER – Sarcoplasmic reticulum
• Each fibril is bundle of fibers – **myofibril**
• Each myofibril is bundle of fibers – **filaments**
  Filaments – 2 types
  **Actin** – thin, light
  **Myosin** – thick, dark
  Myosin have tiny extensions - crossbridges
Basic Muscle Anatomy
Sarcomeres

- **Sarcomere** – repeating pattern of arrangement of actin and myosin fibers
- Billions extend along fibril (cell) length
- Actual site of contraction

Muscle Anatomy
(a) Sarcomere

(b) Thin (actin) filaments

Thick (myosin) filament
Other Proteins in Muscles

- Dystrophin – protein fiber that holds actin in place, supportive
- Can be missing in MD (Muscular Dystrophy) patients
- Genetic
- Several forms of the disease exist
- Vary in severity, age of onset, prognosis
- Muscles weaken, degenerate, replaced by fat and connective tissue
- Can be fatal
- Duchenne: sex-linked form
Inheritance of Duchenne MD
“Working Out”

- “Stress” on muscle tears tiny myofibrils
- Result? Mild soreness
- Stimulated to make more filaments of actin and myosin, NOT more cells
- Muscle increases in diameter = **hypertrophy**
- Muscle also develops more mitochondria, capillaries and **myoglobin** = protein that bonds to oxygen (gives muscle its red color)
- Result?
  - Stronger, fatigues less quickly; “fit”
  - Repair process takes about 48hrs
  - Larger fibrils torn results in excess soreness and longer repair time
Steroids

- Males – more muscle mass and strength due to testosterone
- Steroids – class of hormones
- Often synthetic testosterone injected
- Does build more muscle mass
- Negative consequences
  Male: reduction in natural testosterone can stunt growth, early hair loss, sterility
  Female: effects tend to masculine
  Both: organ damage – kidneys, liver, heart, elevated cholesterol, atherosclerosis, psychiatric symptoms – depression, delusions, violence
Muscle Atrophy

• What happens to muscles not worked?
• “Dormant” for long periods of time?
• Ex. Cast on broken arm
• **Atrophy**
• Breakdown of actin and myosin
• Loss of mitochondria, capillaries, myoglobin
• Shrinkage in diameter, weaker, can be infiltrated with adipose
Muscle Tone

• Some fibrils remain in state of contraction without fatiguing
• Working out increases the number of these cells
• Muscle is firm, “fit”
Muscle Needs for Contraction

1. Nervous Stimulation
   - **Motor Unit** = motor neuron and its controlled fibrils
   - **Motor Point** = place where motor neuron “enters” the muscle
   - **NMJ (Neuromuscular Junction)** = space between neuron and muscle
   - Axon releases neurotransmitter - acetylcholine
Neuromuscular Junction

Axon of motor junction

Muscle fiber

Capillary

Mitochondrion

Myofibrils

Presynaptic terminal

Synaptic vesicles

Sarcolemma

Synaptic cleft

Postsynaptic membrane
• **2. Proper pH** – not too acidic for proper enzyme function

• **3. Oxygen bonded to myoglobin**
  (Dark meat vs. White meat)
  Includes number of capillaries present

• **4. Calcium ions** – “unlock” filaments allowing contraction
• **5. Energy**
  # 1 energy source - Carbohydrates (glucose)
  Glucose stored in liver and muscle as glycogen
Glucose

Figure 3.4C Three representations of the ring form of glucose

- Structural formula
- Abbreviated structure
- Simplified structure
Glycogen
Cellular Respiration

- Occurs in mitochondria
- \[ C_6H_{12}O_6 + O_2 \rightarrow CO_2 + H_2O \]
- Energy from glucose molecule “bonded” to molecule of ATP
More on ATP

- Glucose “burnt” in mitochondria
- Released energy (originally from where?)
- Bonded inside ATP molecule

Energy → ATP
ATP
Breakdown of ATP

- Adenosine
- Energy from Glucose
- ATP

Lots of energy (originally from glucose) is stored here!
ATP Continued…..

- ATP high energy bond between the last 2 phosphates can be broken to release the energy that was originally in glucose
- Creates ADP + energy for “cell work”
- I.e. Contract a muscle, pump the heart, produce enzymes, cell division, etc…….
“Banking of ATP”

Muscle cells can store ("bank") some ATP; usually do so when at rest; more "fit" = more "banked"

High demands during contraction
Impossible to have enough "banked"

Muscle tries to keep up with demand while active
Two Types of Cellular Respiration

• 1. Aerobic
  under high oxygen levels
  1 glucose yields 36 ATP

• A. Muscle at rest
  Cells make lots of ATP
  Some glucose stored as glycogen
  Some stored bonded to a protein – creatine
    to create creatine/phosphate - CP

• “Banked” ATP
What if not enough glucose is available?

- Example: Atkins diet
- Eat few carbs, “burn” fatty acids (fat) instead
- 1 fatty acid “burnt” creates 17 ATP
- Energy levels?
Contraction begins.....(Next, depends on “fitness” level)

I. Moderate Activity

Average muscle can “run” about 17 seconds on “banked” ATP and CP reserves and stored oxygen.

All ATP produced is used, no excess.

Continues aerobically if oxygen diffuses in fast enough.
II. Intense Activity

Muscle starts glycogen breakdown to glucose

Oxygen can’t diffuse in fast enough

**Oxygen debt** develops

Not enough oxygen for aerobic

Switches to second type of cellular respiration
• 2. Anaerobic respiration
  under low oxygen levels
  much less efficient
  1 glucose yields 2 ATP
  Process also results in high levels
  of an acid called pyruvic acid
  which further breaks into lactic acid
Effects?
  pH drops, enzymes can’t function
  Fatigue results
  Wastes glucose
  Less ATP produced
  Contracture (Cramp) may result – severe ATP depletion
Example of Anaerobic Respiration in Contracting Muscle Cells

Repeated contraction of *Tibialis Anterior* leg muscle

![Diagram of leg muscles](image)
Rigor Mortis

- Occurs after death ("stiff")
- Time dependent on temperature
- Muscles "lock up"
- Why?
- Eventually relax
- Why?
Muscle Fatigue

- Muscle can’t contract or does so weakly
- Even with neural stimulation
- Causes:
  - Depletion of ATP and CP
  - Depletion of glycogen, fatty acid reserves (common in a marathon)
  - Severe Oxygen Debt
  - Lactic acid pH drop
  - Physical fiber damage (Marathon)
Muscle Fatigue Recovery

- **A. Lactic Acid**
  1. Diffuses and actively transported out
  2. Travels to liver
  3. Recycled back to glucose
     
     **Drawback:** requires input of oxygen and ATP
  4. Glucose released back to blood and back to muscles
B. Oxygen Debt
(Bank Debt Analogy)
Occurs during contraction when depleted
Needs to be restored to normal levels and bonded to myoglobin
Debt size depends on level of activity and ATP usage
Repaid how?
During and after activity – breathe deeper and faster
Can take minutes, hours or even days to repay
• C. Glucose and ATP
  Oxidize glucose to rebuild ATP and CP reserves
  Rebuild glycogen and fatty acid reserves
  Can take minutes, hours or days
• D. Muscle Damage
  Cells do protein synthesis to repair filaments and may rebuild more new ones
  Importance of waiting 48 hours between workouts