Exam II Review Slides

Exam II! Whee!
I. **Announcements**  No lab today! Break for exam week! Next R Blood Chemistry. Thanks sincerely for helping us optimize safety by reading ≥ 2x Lab 5, LM pp 5-1 thru 5-6.

II. **Blood Form & Function**  LS ch 11, DC Module 5 pp 35-9  
A. Formed vs Nonformed/cells vs plasma LS fig + tab 11-1  
   Cell origin - bone marrow. What’s in plasma? LS p 297  
B. Red blood cells/erythrocytes: $O_2$ carrying LS p 299  
   Normal flexible vs fragile sickle cell LS p 301  
C. White blood cells/leukocytes: defense/immunity  
   differential + general functions LS pp 298, 309-12  
D. Platelets/thrombocytes: clotting LS pp 304-6 fig 11-6+7

III. **Blood Chemistry Lab: Basics**  LM + LS ch 11 & 17  
A. What’s blood typing? ABo System LS pp 302- 4  
   Rhesus factor? Erythroblastosis fetalis? LS p 303-4  
B. What’s blood glucose? Clinically healthy range?  
C. Diabetes + Treatment LS ch 17 pp 532-5

IV. **Exam Comments & Return**  
We survived the exam! Happy Halloween!! Remember nutrient ρ & have safe fun!
What's in Blood? Plasma & Blood Cells

- Plasma (55% of whole blood)
- Buffy coat: platelets and leukocytes (<1% of whole blood)
- Erythrocytes (45% of whole blood)

- Platelets
- Leukocytes (white blood cells)
- Erythrocytes (red blood cells)

Packed cell volume, or hematocrit
What a difference one amino acid can make!

Amino acid sequence of normal hemoglobin:
Val → His → Leu → Thr → Pro → Glu → Glu

Amino acid sequence of sickle-cell hemoglobin:
Val → His → Leu → Thr → Pro → Val → Glu
A & B Antigens
(Agglutinogens)
Erythroblastosis Fetalis?

eg, Rh- mom Rh+ baby

Erythroblastosis Fetalis or Hemolytic Disease of the Unborn/Newborn

Throw Blanket Over This Step!
Inject Mom with RhoGam ≤ 48-72 hr > each Rh+ Pregnancy

The Blanket is RhoGam → Masks the Mom’s Immune System!
I. **Announcements** To make Lab 5 educational, fun & safe for all, please read pp 5-1 thru 5-6 in LM twice before Thursday! Remaining exams & notebooks returned > lecture. Key posted in glass box in Huestis near 120 HUE? Estimate grade? Q?

II. **Blood Chemistry Review** LS ch 11 + 17, DC Module 5, Q?

III. **Blood Glucose, Insulin Diabetes Connections** DC Module 13+

IV. **Endocrinology Overview** LS ch 17, DC Module 13, SI Fox+

A. Vignette: Cushing's syndrome LS fig 17-20 p 521-2
B. Endocrine system DC p 103 fig 13-1, LS fig 17-1, tab 17-1
C. What’s an endocrine? + classes ~ LS pp 495 - 6
D. Hypothalamus (Master) – Pituitary (subcontroller)
   DC pp 104-6 + LS pp 499-506
E. Posterior pituitary + hormones DC p 108, LS fig 17-4 p 502
F. Anterior pituitary + hormones DC pp 105-7, LS pp 502-6
H. Peripheral endocrine organs DC pp 109-13, LS pp 513-36
   1. Pancreas (insulin – glucagon see-saw!) 2. Thyroid 3. Adrenals...This Thursday more fun & data about me! Heck yeah!!
No food, drink or gum in lab! Thanks sincerely!
Glucose: Sugar in Blood

Normal: 70-99
Pre-Diabetes: 100-125
Diabetes: ≥ 126 mg/dL

NB: Read & Record!
$1^0$ Q? Clumping in Any Wells?

Type AB+

Source: S Wong, BI 121 Lab, 2016
Diabetic & Normal Response to Glucose Load

Ingest Glucola or eat meal
Endocrine Pancreas: Insulin (I) & Glucagon (G)
See-Saw Hormones in Regulating Blood Glucose

Hormones (insulin, glucagon)

Duct cells secrete aqueous NaHCO₃ solution
Acinar cells secrete digestive enzymes

Exocrine portion of pancreas (Acinar and duct cells)

Endocrine portion of pancreas (Islets of Langerhans)

The glandular portions of the pancreas are grossly exaggerated.
Times of Plenty!!

NB: Diabetics have problems either here or here.

Store!

https://www.youtube.com/watch?v=8dgoeYPoE-0
<table>
<thead>
<tr>
<th></th>
<th>Type 1</th>
<th>Type 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percentage of cases</td>
<td>5–10%</td>
<td>90–95%</td>
</tr>
<tr>
<td>Age of onset</td>
<td>&lt;30 years</td>
<td>&gt;40 years(^a)</td>
</tr>
<tr>
<td>Associated characteristics</td>
<td>Autoimmune diseases, viral infections, inherited factors</td>
<td>Obesity, aging, inherited factors</td>
</tr>
<tr>
<td>Primary problems</td>
<td>Destruction of pancreatic beta cells; insulin deficiency</td>
<td>Insulin resistance, insulin deficiency (relative to needs)</td>
</tr>
<tr>
<td>Insulin secretion</td>
<td>Little or none</td>
<td>Varies; may be normal, increased, or decreased</td>
</tr>
<tr>
<td>Requires insulin</td>
<td>Always</td>
<td>Sometimes</td>
</tr>
<tr>
<td>Older names</td>
<td>Juvenile-onset diabetes, Insulin-dependent diabetes mellitus (IDDM)</td>
<td>Adult-onset diabetes, Noninsulin-dependent diabetes mellitus (NIDDM)</td>
</tr>
</tbody>
</table>

\(^a\) According to some sources, the percentage of Type 2 diabetes is as low as 80–85%.
<table>
<thead>
<tr>
<th>Warning Signs of Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>These signs appear reliably in type 1 diabetes and, often, in the later stages of type 2 diabetes.</td>
</tr>
</tbody>
</table>

- Excessive urination and thirst
- Glucose in the urine
- Weight loss with nausea, easy tiring, weakness, or irritability
- Cravings for food, especially for sweets
- Frequent infections of the skin, gums, vagina, or urinary tract
- Vision disturbances; blurred vision
- Pain in the legs, feet, or fingers
- Slow healing of cuts and bruises
- Itching
- Drowsiness
- Abnormally high glucose in the blood
Diabetics must constantly juggle diet, exercise & medication to control blood glucose!
Cushing’s Syndrome = Hypersecretion of Cortisol: Hypothalamic (CRH), Pituitary (ACTH), or Adrenal (Cortisol)

T = 0, near normal

T = 4 months later
ANP = Atrial Natriuretic Polypeptide

https://www.ted.com/talks/emma_bryce_how_do_your_hormones_work
https://www.youtube.com/watch?v=IRJE8c3ghRE
https://www.hopkinsallchildrens.org/Patients-Families/Health-Library/HealthDocNew/Movie-Endocrine-System
Hormone/Endocrine Classifications?

Exogenous

Endogenous

Amino Acid/PP/Protein

Thyroid

Steroid
Nervous Connection!!

Neurosecretory neurons

Hypothalamic-posterior pituitary stalk

Anterior pituitary

Hypothalamus

Posterior pituitary

Systemic arterial inflow

Systemic venous outflow

• = Vasopressin

• = Oxytocin
Hypothalamus-Anterior Pituitary Vascular Connection!

- Neurosecretory neuron
- Systemic arterial inflow
- Hypothalamic-hypophyseal portal system
- Anterior pituitary
- Posterior pituitary

Vascular Connection!!

- • = Hypophysiotropic hormones
- • = Anterior pituitary hormone
Pituitary Nourishing or Growth Hormones

Hypothalamus

Neurosecretory neuron

Systemic arterial inflow

Hypothalamic-hypophyseal portal system

RH + or RIH -

Releasing or Release-Inhibiting Hormones

Pituitary Nourishing or Growth Hormones

Systemic venous outflow

• = Hypophysiotropic hormones
• = Anterior pituitary hormone

Hypophysis = Pituitary

LS 2007
Paraventricular nucleus
Supraoptic nucleus
Median eminence
Portal system
Infundibulum
Hypothalamus
Anterior pituitary
Posterior pituitary

TSH

Prolactin

Mammary gland

Thyroid

Adrenal cortex

Growth hormone

Bone
Muscle
Adipose tissue

Gonadotropins

FSH
LH

Ovary
Testis

SI Fox 2008
cf: LS 2012 fig 17-5
Often, more than simply 1 feedback loop!
**GH/STH Effects: Insulin Resistance/Type II Diabetes?**

- ↑ Amino Acid uptake & Protein synthesis
- ↑ Lipolysis & Fatty Acid mobilization
- ↓ Glucose uptake (skeletal muscle & adipocytes)
- ↑ Glucose production (liver glycogenolysis)
- ↑ Insulin secretion

Mismatch!!
Increase GH naturally with exercise & sleep!!

Growth hormone (ng/ml plasma)

Time of day

ng/ml = nanograms per milliliter
BI 121 Lecture 11

I. **Announcements** Blood Chemistry Lab today! Fun!! Personal data!!! If you haven't already done so, please review Lab 5 in LM & in e-mail. Thanks! Q from last t?

II. **Safety & Techniques Review for Blood Chem Lab** Q?

III. **Endocrine Connections** Peripheral endocrine organs

   DC pp 109-13, LS pp 513-36

   A. Pancreas (insulin – glucagon see-saw!)
   B. Thyroid
   C. Adrenals

IV. **Introduction to the Nervous System** LS ch 5, DC Module 9

   A. Organization? LS fig 5-1 DC p 67
   C. Myelin? How does it help? DC fig 9-3, LS pp 83-5
   D. Brain structure & function DC fig 9-6 thru 9-10 pp 71-5+...
   E. Protect your head with a helmet! Bicycle head injury statistics *NHTSA & BHSI, 2013 & 2014*
Insulin Stores Sugar, Glucagon Mobilizes Sugar!

~ 4-6 hr of Stored Glucose

Peripheral Endocrine & Digestive Organ

Benjamin Cummings 2001

https://www.youtube.com/watch?v=y9Bdi4dnS1g
https://www.fuseschool.org
FIGURE 13-12
Adrenal Gland   The adrenal glands sit atop the kidney and consist of an outer zone of cells, the adrenal cortex, which produces a variety of steroid hormones, and an inner zone, the adrenal medulla. The adrenal medulla produces adrenalin and noradrenalin.
Stress Promotes Cortisol Secretion

Metabolic fuels and building blocks available to help resist stress:

- ↑ Blood glucose (by stimulating gluconeogenesis and inhibiting glucose uptake)
- ↑ Blood amino acids (by stimulating protein degradation)
- ↑ Blood fatty acids (by stimulating lipolysis)
Epinephrine 80%
Norepinephrine 20%

**Figure 77-1**

Secretion of adrenocortical hormones by the different zones of the adrenal cortex.
I. **Announcements** Thanks for your help with blood lab! Great job! No lab this week. Study for Exam II, Dec 7, Friday, 8 am!

II. **Introduction to the Nervous System** LS ch 5, DC Module 9  
A. How is the nervous system organized? LS fig 5-1 DC p 67  
C. What’s myelin? How does it help? DC fig 9-3, LS pp 83-5  
D. Brain structure & function DC fig 9-6 thru 9-10 pp 71-5 +…  
E. **Protect your head with a helmet!** Bicycle head injury statistics, *NHTSA & BHSI* from 2013 & 2014

III. **Autonomic Nervous System** LS ch 7 pp 178-85+…  
A. Sympathetic vs Parasympathetic branches LS fig 7-3  
B. Neurotransmitters & receptors LS fig 7-1 & 7-2, tab 7-2  
C. Actions LS tab 7-1  
D. **Fight-or-flight stories!**
Nervous System

CNS

PNS

input

output

https://www.youtube.com/watch?v=uU_4uA6-zcE&vl=ko
~99% of all neurons in humans! CNS ~100 billion interneurons!!
What is myelin? Why is it important?

Lipid insulative coat

↑ $\vec{v}$, conserves ions & ATP
Saltatory/Leaping Conduction!
Crucial Sensory & Motor Nerves

L. *saltare* to hop or leap! Fr. *salt*, sautier, *sauté*, leap, high air, vault

DC 2003
Motor

Sensory

M. Supplementary motor area (on inner surface—not visible; programming of complex movements)

M. Premotor cortex (coordination of complex movements)

M. Primary motor cortex (voluntary movement)

S. Primary sensory cortex (sensation)

Central sulcus

A. Prefrontal association cortex (planning for voluntary activity; decision making; personality traits)

M. Broca’s area (speech formation)

A. Limbic association cortex (mostly on inner and bottom surface of temporal lobe; motivation and emotion; memory)

A. Posterior parietal cortex (integration of somatosensory and visual input; important for complex movements)

A. Wernicke’s area (speech understanding)

A. Parietal-temporal-occipital association cortex (integration of all sensory input; important in language)

S. Primary visual cortex surrounded by higher-order visual cortex (sight)

LS 2006, cf: LS 2012 fig 5-8a
~ 500,000 bicyclists/yr visit emergency rooms

As of 2014, the population estimate of
State of Wyoming 584,153
   Albany OR 51,980
   Corvallis OR 54,953
   Springfield OR 60,263

~ 26,000 traumatic brain injuries

743 of ~900 cyclist deaths, 2013 ≡ ~ 2% of all traffic fatalities
13% of deaths children ≤ 14 yr, 87% σ
11% involved wrong-way riding!

Bicycle crashes & injuries are under reported,
since majority not serious enough for ER visits.

Helmets may reduce head & brain injury risk by 85%!

~$2.3 billion/yr = indirect injury costs from not using helmets!

Helmets Cheap, Brains Expensive!!
Use Your Head, Get a Helmet!!

http://www.bhsi.org/stats.htm
Homeostasis is a dynamic balance between the autonomic branches.

- **Rest-and-digest:** Parasympathetic activity dominates.
- **Fight-or-flight:** Sympathetic activity dominates.
I. **Announcements** No lab today – Study for Exam II!! Optional Lab notebook check after last Lab 6, Mac pulmonary function testing (PFT) next Thursday. Q?

II. **Nervous System Connections** NS organization video. [https://www.youtube.com/watch?v=qPix_X-9t7E](https://www.youtube.com/watch?v=qPix_X-9t7E)

Brain + spinal cord (CNS). What disease involves the basal nuclei? **Protect your head with a helmet!** Bicycle head injury statistics *NHTSA & BHSI, 2014 data*

III. **Peripheral Nervous System** LS sections of ch 3, 4, & 7

A. Autonomic NS: Branches, neurotransmitters, receptors, actions, fight-or-flight stories ch 7 pp179-85
B. Why are nerve & muscle unique? ch 4 p 71
C. How do excitable cells signal? ch 3 pp62-7; ch 4 pp74-83
D. How does the signal cross the nerve-muscle gap? ch 7 p 185-92 fig 7-5 p 190
   1. Ca2+ bones!…but what else? p 190
   2. What do black widow spider venom, botulism, curare & nerve gas have in common? Botox pp 189-92
Why overlap or dual innervation?

Fine-tune control & safety!

cf: LS 2012 fig 7-3
Homeostasis is a dynamic balance between the autonomic branches.

Rest-and-digest: Parasympathetic activity dominates.

Fight-or-flight: Sympathetic activity dominates.
**Parasympathetic**

Ach = Acetylcholine

- = Nicotinic Receptor

- = Muscarinic Receptor

**Sympathetic**

NE = Norepinephrine

- = α Receptor ($\alpha_1$, $\alpha_2$)

- = β Receptor ($\beta_1$, $\beta_2$)
Nicotine activates **both** Sympathetic & Parasympathetic post-ganglionic neurons!

Like hammering the gas pedal & brake at the same time!!
Hormonal Adrenaline Surge Reinforces Nervous Outflow & Accesses Tissues Not Directly Innervated!!

80% Epinephrine/Adrenaline (E)
20% Norepinephrine (NE)

Output to blood

Adrenals = Paired organs above kidneys
<table>
<thead>
<tr>
<th>Organ</th>
<th>Effect of Sympathetic Stimulation</th>
<th>Effect of Parasympathetic Stimulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart</td>
<td>Increases heart rate and increases force of contraction of the whole heart</td>
<td>Decreases heart rate and decreases force of contraction of the atria only</td>
</tr>
<tr>
<td>Blood Vessels</td>
<td>Constricts</td>
<td>Dilates vessels supplying the penis and the clitoris only</td>
</tr>
<tr>
<td>Lungs</td>
<td>Dilates the bronchioles (airways)</td>
<td>Constricts the bronchioles</td>
</tr>
<tr>
<td>Digestive Tract</td>
<td>Decreases motility (movement)</td>
<td>Increases motility</td>
</tr>
<tr>
<td></td>
<td>Contracts sphincters (to prevent forward movement of tract contents)</td>
<td>Relaxes sphincters (to permit forward movement of tract contents)</td>
</tr>
<tr>
<td></td>
<td>Inhibits digestive secretions</td>
<td>Stimulates digestive secretions</td>
</tr>
<tr>
<td>Urinary Bladder</td>
<td>Relaxes</td>
<td>Contracts (emptying)</td>
</tr>
<tr>
<td>Eye</td>
<td>Dilates the pupil</td>
<td>Constricts the pupil</td>
</tr>
<tr>
<td></td>
<td>Adjusts the eye for far vision</td>
<td>Adjusts the eye for near vision</td>
</tr>
<tr>
<td>Liver (glycogen stores)</td>
<td>Glycogenolysis (glucose is released)</td>
<td>None</td>
</tr>
<tr>
<td>Adipose Cells (fat stores)</td>
<td>Lipolysis (fatty acids are released)</td>
<td>None</td>
</tr>
<tr>
<td>Exocrine Glands</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exocrine pancreas</td>
<td>Inhibits pancreatic exocrine secretion</td>
<td>Stimulates pancreatic exocrine secretion (important for digestion)</td>
</tr>
<tr>
<td>Sweat glands</td>
<td>Stimulates secretion by sweat glands important in cooling the body</td>
<td>Stimulates secretion by specialized sweat glands in the armpits and genital area</td>
</tr>
<tr>
<td>Salivary glands</td>
<td>Stimulates a small volume of thick saliva rich in mucus</td>
<td>Stimulates a large volume of watery saliva rich in enzymes</td>
</tr>
<tr>
<td>Endocrine Glands</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adrenal medulla</td>
<td>Stimulates epinephrine and norepinephrine secretion</td>
<td>None</td>
</tr>
<tr>
<td>Endocrine pancreas</td>
<td>Inhibits insulin secretion</td>
<td>Stimulates insulin secretion</td>
</tr>
<tr>
<td>Genitals</td>
<td>Controls ejaculation (males) and orgasm contractions (both sexes)</td>
<td>Controls erection (penis in males and clitoris in females)</td>
</tr>
<tr>
<td>Brain Activity</td>
<td>Increases alertness</td>
<td>None</td>
</tr>
</tbody>
</table>
I. **Announcements** Last Lab 6, Pulmonary Function Testing + Optional notebook ✓ this Thurs. Exam II Fri Dec 7, 8am Q?

II. **Nervous System Connections** LS ch 3, 4 & 7; DC Module 9
   A. Why nerve & muscle unique? How do they signal?
      LS pp 62-67, 74-83
   B. How does the signal cross the nerve-muscle gap?
      LS p 185-92 fig 7-5 p 190; DC pp 69-71 fig 9-4
      1. Ca2+ bones!...but what else? LS p 190
      2. What do black widow spider venom, botulism, curare & nerve gas have in common? Botox LS pp 189-92

III. **Muscle Structure & Function** LS ch 8 + DC Mod 12
   A. Muscle types: cardiac, smooth, skeletal LS fig 8-1
   B. How is skeletal muscle organized? LS fig 8-2, DC fig 12-2
   C. What do thick filaments look like? LS fig 8-4, DC fig 12-4
   D. Thin filaments? Banding pattern LS fig 8-5, 8-3, 8-7
   E. How do muscles contract? LS fig 8-6, 8-10
   F. What's a cross-bridge cycle? LS fig 8-11 +...
Why are nerve & muscle unique?

They are excitable!!
Action Potentials ≡ Spikes ≡ Impulses

Ultra-short reversal of membrane potential
Only in nerve and muscle cells
Maintains strength over distance
Primary way nerves & muscles communicate!
"Resting"/Membrane Potential?

Cells are slightly negative inside!
Stimulate Cell @ Rest

Thermal

Mechanical

Electrical

Chemical
Action Potential has occurred!

Brief (1-2 ms) reversal to + inside cell!
Other Links That May Be Helpful!

https://www.youtube.com/watch?v=6RbPIOq0O3w
https://www.youtube.com/watch?v=mltV4rC57kM
https://www.youtube.com/watch?v=WhowH0kb7n0
http://sites.sinauer.com/psychopharm2e/animation03.01.html
https://www.youtube.com/watch?v=VitFvNvRIly
Axon of motor neuron
Myelin sheath
Action potential propagation in motor neuron
Terminal button
Vesicle of acetylcholine
Ca^{2+}
Calcium channel
Action potential propagation in muscle fiber
Plasma membrane of muscle fiber
Acetylcholine receptor site
Cation channel
Acetylcholinesterase
Motor end plate
Contractile elements within muscle fiber
Motor fiber
Local current flow between depolarized end plate and adjacent membrane
Campbell's Classics
3
4
Pulmonary Function Testing today! Hooray!...

BI 121 Lecture 15

I. **Announcements** Optional notebook ✓ + Lab 6
   Pulmonary Function Testing today. Q?

II. **Pulmonary Function Lab Overview**

III. **Muscle Structure & Function** LS ch 8, DC Module 12

   A. How is skeletal muscle organized? LS fig 8-2, DC fig 12-2
   B. What do thick filaments look like? LS fig 8-4, DC fig 12-4
   C. How about thin filaments? LS fig 8-5
   D. Banding pattern? LS fig 8-3, fig 8-7
   E. How do muscles contract? LS fig 8-6, 8-10
   F. What's a cross-bridge cycle? LS fig 8-11 +…
   G. Summary of skeletal muscle contraction
   H. Exercise adaptation variables: *mode, intensity, duration, frequency, distribution, individual & environmental char...*?
   I. Endurance vs. strength training continuum? fiber types...
Skeletal Muscle Histology: Microscopic Anatomy

Muscle fiber or cylindrical cell

“Threads” ≡ Myofibrils

Nuclei

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

x1000

H Howard 1980.
Organ = Muscle

Cell = Myocyte = Fiber

Subcellular = Cytoskeleton

Molecules = Actin & Myosin
Golf Club Analogy?

(a)

Actin binding site
Myosin ATPase site

Heads

Tail

100 nm

Cross bridges

Myosin molecules

LS 2006, cf:
LS 2012 fig 8-4
Actin molecules

Binding site for attachment with myosin cross bridge

Actin helix

Tropomyosin

Troponin

Thin filament

LS 2006, cf:
LS 2012 fig 8-5
Triad ≡ T tubule abutting cisternae

Mitochondria

Sarcomere

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

I Band = Light Band
Isotropic = Light Can Shine Through
Sarcomere

Z line

H zone

I band

A band

Z line

Relaxed

H zone shorter

I band shorter

A band same width

Contracted

Sarcomere shorter

Thick filament

Thin filament
I. **Announcements** Notebooks? Exam II, Dec 7th Friday 8 am. Review session in class next Thurs. Q?

II. **Muscle Contraction & Adaptation** LS ch 8, DC Mod 12
   A. Banding pattern? LS fig 8-3, fig 8-7
   B. How do muscles contract? LS fig 8-6, 8-10
   C. What's a cross-bridge cycle? LS fig 8-11 +…
   D. Summary of skeletal muscle contraction
   E. Exercise adaptation variables: *mode, intensity, duration, frequency, distribution, individual & environmental char...*?
   F. Endurance vs. strength training continuum? fiber types...

III. **Respiratory System** LS ch 12, DC Module 7, Fox +…
   A. Steps of respiration? External vs. cellular/internal?
      LS fig 12-1 pp 345-347
   B. Respiratory anatomy LS fig 12-2 p 347, DC, Fox +…
   C. Histology LS fig 12-4 pp 347-349, DC
   D. How do we breathe? LS fig 12-12, fig 12-25 pp 349-356, pp 373-378
A Band = Dark Band
Anisotropic = Light Can’t Shine Through

I Band = Light Band
Isotropic = Light Can Shine Through
Cross-Bridge Cycle

1. Energized
   - ATP (Mg++)

2a. Binding
   - Energy
   - ADP
   - P_i

2b. Resting
   - Energy
   - ADP
   - P_i
   - No Ca++

3. Bending (power stroke)
   - Energy
   - ADP
   - P_i
   - Ca++ present (excitation)

4a. Detachment
   - Fresh ATP available

4b. Rigor complex
   - No ATP (after death)
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.
Excited: Calcium Triggers Cross-Bridge Binding

(b) Excited

1. Muscle fiber is excited and Ca\(^{2+}\) is released.

2. Released 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.
Summary
We are almost there!

https://www.youtube.com/watch?v=Ktv-CaOt6UQ
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.
Adaptations to Exercise?
Mode, Intensity, Duration, Frequency, Distribution of Training Sessions? Conditions of Environment? Individual?
Cardiac Adaptations to Exercise:

1. Endurance vs. 2. Strength Training

NB: \( 1 \) \( \rightarrow \) ↑ LBM

1 + 2
**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>

---

LS 2012 tab 8-1 modified
> VP Lombardi 1989
Muscle Changes 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)?
Muscle Changes 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 ($\beta$-oxidation) of fatty acids
- Myoglobin (enhances $O_2$ transport)
- Resting energy levels which inhibit sugar breakdown
- Aerobic capacity of all three fiber types.
I. **Announcements**  Exam II next Friday, Dec 7th @ 8:00 am!
   12 n lab section go to 5 KLA; 1 pm lab section go to 13 KLA; 2 pm lab section go to 21 KLA. Discussion-Review Thurs. Q?

II. **Respiratory System**  LS ch 12, DC Module 7, SI Fox +...
   A. Respiratory system anatomy  LS fig 12-2 p 347, DC, SI Fox+...
   B. Histology  LS fig 12-4 pp 347-9, DC fig 7-4 p 54
   C. How do we breathe?  LS fig12-12, fig12-25 pp 349-56, 373-8
   D. Gas exchange  LS fig 12-19 pp 362-5
   E. Gas transport  LS tab 12-3 pp 365-70

III. **Physiology of Cigarette Smoking**
   A. ANS, autonomic nerves & nicotine? Route of chemicals,...
   B. Emphysema? 2nd-hand smoke?... LS pp 356, 365
   C. UO Smoke-Free since Fall 2012! Help is available!
NB: In vivo, Cupola or peak of each lung goes into neck > clavicle line!
16-20 C-shaped bars of hyaline cartilage to prevent collapse
No Gas Exchange

1st alveolar outpouching!

Gas Exchange
The last cilium on a smoker's lung

Shoot... If only I had a red five.
NB: Diaphragm is the chief muscle of ventilation!
**Inhale** (active)

Contract & flatten diaphragm

**Exhale** (passive @ rest)

Relax & pouch up diaphragm!
Brain stem = Control Center for automatic breathing!

Respiratory control centers in brain stem

- Pons respiratory centers
- Medullary respiratory center

- Pneumotaxic center
- Apneustic center
- Pre-Bötzinger complex
- Dorsal respiratory group
- Ventral respiratory group

Medulla
**Respiratory membrane** separates air from blood, is 6 layers, yet 1/50th thickness of tracing paper!

LS 2012 fig 12-4a
cf: DC 2013 fig 7-4
Across pulmonary capillaries:
O₂ partial pressure gradient from alveoli to blood = 60 mm Hg (100 → 40)
CO₂ partial pressure gradient from blood to alveoli = 6 mm Hg (46 → 40)

Across systemic capillaries:
O₂ partial pressure gradient from blood to tissue cell = 60 mm Hg (100 → 40)
CO₂ partial pressure gradient from tissue cell to blood = 6 mm Hg (46 → 40)

Numbers are mm Hg pressure.
$O_2$ is carried mainly by red blood cell hemoglobin!

Each hemoglobin molecule carries 4 $O_2$ on 4 iron-containing disks!

Carbon monoxide, CO, binds $\geq 200x$ more powerfully to these same sites, thus poisoning the hemoglobin!
## TABLE 12-3

Methods of Gas Transport in the Blood

<table>
<thead>
<tr>
<th>GAS</th>
<th>METHOD OF TRANSPORT IN BLOOD</th>
<th>PERCENTAGE CARRIED IN THIS FORM</th>
</tr>
</thead>
<tbody>
<tr>
<td>$O_2$</td>
<td>Physically dissolved</td>
<td>1.5</td>
</tr>
<tr>
<td></td>
<td>Bound to hemoglobin</td>
<td>98.5</td>
</tr>
<tr>
<td>$CO_2$</td>
<td>Physically dissolved</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>Bound to hemoglobin</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>As bicarbonate ($HCO_3^-$)</td>
<td>60</td>
</tr>
</tbody>
</table>

Cigarette Smoking: #1 Preventable Cause of Premature Death in the US

Cardiovascular Mortality (average annual incidence per 1,000)

- None: 7
- Less than 20: 8.4
- 20: 10.2
- More than 20: 12.4
- Quit One Year: 7

Cigarettes smoked per day
Not only the Lungs, but the Heart, Brain & 100s of Other Tissues & Organs Adversely Affected!

Tobacco smoke = Deadly mix of > 7000 chemicals!

http://www.cdc.gov/tobacco/data_statistics/sgr/50th-anniversary/index.htm#fact-sheets
Cigarette + Smoke: > 7000 Chemicals; ~600 Tobacco Company Additives
Atherogenic, Carcinogenic (C), Tumor Initiating, Tumor Promoting (TP),
Toxic (T), Cornucoppia of Unknowns, Synergistic, Reactive...?

<table>
<thead>
<tr>
<th>Chemical</th>
<th>Type</th>
<th>Concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>4-aminobiphenyl</td>
<td>C</td>
<td>140 ng per cigarette...</td>
</tr>
<tr>
<td>benz(a)anthracene</td>
<td>C</td>
<td>40-200 ng</td>
</tr>
<tr>
<td>benzene</td>
<td>C</td>
<td>400 μg</td>
</tr>
<tr>
<td>benz(o)pyrene</td>
<td>C</td>
<td>40-70 ng</td>
</tr>
<tr>
<td>carbon monoxide</td>
<td>T</td>
<td>26.8-61 mg</td>
</tr>
<tr>
<td>formaldehyde</td>
<td>C</td>
<td>1500 μg</td>
</tr>
<tr>
<td>hydrazine</td>
<td>C</td>
<td>90 ng</td>
</tr>
<tr>
<td>hydrogen cyanide</td>
<td>T</td>
<td>14-110 μg</td>
</tr>
<tr>
<td>2-naphthylamine</td>
<td>C</td>
<td>70 ng</td>
</tr>
<tr>
<td>nitrogen oxides</td>
<td>T</td>
<td>500-2000 μg</td>
</tr>
<tr>
<td>N-nitrosodimethylamine</td>
<td>C</td>
<td>200-1040 ng</td>
</tr>
<tr>
<td>N-nitrosodiethanolamine</td>
<td>C</td>
<td>43 ng</td>
</tr>
<tr>
<td>N-nitrospyrrolide</td>
<td>C</td>
<td>30-390 ng</td>
</tr>
<tr>
<td>phenol</td>
<td>TP</td>
<td>70-250 μg</td>
</tr>
<tr>
<td>polonium 210</td>
<td>C</td>
<td>0.5-1.6 pCi</td>
</tr>
<tr>
<td>quinoline</td>
<td>C</td>
<td>15-20 μg</td>
</tr>
<tr>
<td>O-toluidine</td>
<td>C</td>
<td>3 μg</td>
</tr>
</tbody>
</table>

**Sources:** US Surgeon General's Office, American Cancer Society, American Heart Association.
Ammonia converts nicotine, the additive agent in tobacco, into a more volatile form, Pankow said. “Ammonia is the thing that helps tobacco companies hook the smoker by providing a means of delivering the nicotine.”

Last October, a former tobacco industry employee revealed that secret industry documents indicated that ammonia was added to tobacco to double the impact of nicotine. Research now indicates that ammonia can boost nicotine availability up to 100x! The Oregon Graduate Institute (now a part of OHSU) was the 1st to research!

http://pubs.acs.org/doi/abs/10.1021/es970402f
http://www.nasw.org/users/sperkins/nicotine.html
Cigarettes ≡ **Patient-Assisted Drug-Delivery System**
Inhaling Bypasses the Systemic Circulation & Is Powerfully Reinforcing!
Tracing the Route of Cigarette Smoke Puff to Brain Time 5 to 8 seconds!!

Mouth
↓
Pharynx
↓
Larynx
↓
Trachea
↓
Bronchi
↓
Bronchioles
↓
Alveoli

Blood

Pulmonary Veins
↓
Left Atrium
↓
Left Ventricle
↓
Aorta
↓
Heart
↓
Brain

Systemic Circulation

Respiratory Membrane
Cigarette smoking is the most important preventable cause of premature death in the U.S. accounting for 443,000 annual deaths.

http://www.cdc.gov/tobacco/data_statistics/fact_sheets/health_effects/tobacco_related_mortality/#cigs
Cigarette smoking causes 87% of lung cancer deaths and is responsible for most cancers of the larynx, oral cavity & pharynx, esophagus, & bladder.
Emphysema ≡ Corrosion of Alveolar Walls with ↓ SA & Labored Breathing

Internet Journal of Pathology
Mayo Clinic Health
On the Pill & Smoke?

Increased Risk of:

1. Blood Clots
2. Heart Attack
3. Strokes!

You figure an occasional cigarette can't hurt, and you really don't want to listen to the "Stop smoking" lecture from your doctor. But if you want any type of hormonal birth control, smoking is a vitally important issue.

Hormonal birth control is a prescription drug and while the risks are rare, they can be serious, and smoking even a little increases the risks, especially if you're over 35.

Risks include blood clots, stroke, and heart attack. If you have a history of these conditions or certain cancers, you shouldn't use hormonal birth control.

Of course, you should tell your healthcare provider if you could be pregnant, and because hormonal birth control doesn't protect against HIV or sexually transmitted diseases, learn how to stay safe and healthy.

Hormonal birth control has been used safely by millions of women for 45 years, and is 99.9% effective when used correctly. It could be a good choice for you. To find out, talk to your healthcare professional. And to help you get started, there's a list of questions to ask. Visit www.orthohealth.com

Be smart about your body.
Be smart about your birth control.
Breathing 2nd-hand smoke for as little as ½ hr activates platelets almost as much as if you were a pack-a-day smoker.
2nd-hand smoke is the 3rd leading preventable cause of death in the US!

“Mind if I smoke?”

“Care if I die?”

Each year ~45,000 Americans die due to 2nd-hand smoke exposure!
Health risks of e-cigarettes emerge

Vaping pollutes lungs with toxic chemicals and may even make antibiotic-resistant bacteria harder to kill

By JANET RALOFF 4:31PM, JUNE 3, 2014

https://www.sciencenews.org/article/health-risks-e-cigarettes-emerge
SMOKING ≡ ASTHMA?

Ugh!!
Cough!!

Petri-dish Effect
Nicotine Addiction & Help Quitting Smoking

http://www.cancer.org/healthy/stayawayfromtobacco/guide
toquittingsmoking/guide-to-quitting-smoking-help-phys-nrt

2nd-Hand Smoke or ETS & 3rd-Hand Smoke?
http://www.cancer.org/cancer/cancercauses/tobaccocancer/
secondhand-smoke

2nd-Hand Smoke Addictive?
%20smoke%20addictive