Kine 4010
Lecture 0

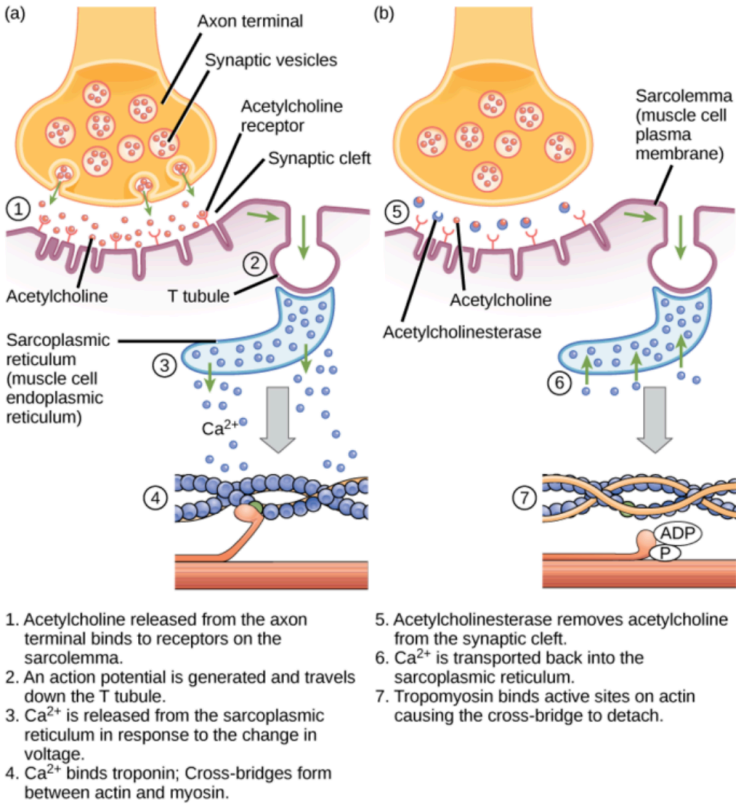
- Textbook is recommended edition 10 exercise physiology powers, S.K
- Doesn't take questions from this book but information is required
- COURSE MANUAL AND LAB MANUAL REQUIRED
- Go to York u bookstore, go to e books, find course, add to cart
- When you get access code, go to McGraw website
- Then it will take u to vital source
- Lab requires attention, must attend
- Labs are not tested on the midterms
- Final exam has lab material

Lecture 1
- Starts on page one of lecture manual
- Starts with metabolic system; today we will talk about the three systems of energy production
- Dive deep into the molecular system involved (cellular mechanisms that control)
- Coronary heart disease; very prevalent, exercise and heart disease, risk factors for heart disease, physical inactivity is a risk factor that can be modified. Inactivity wasn't on the list for a long time, now it is, and it is a big factor. SINCE, regular exercise lowers blood pressure, cholesterol won't be high (modified babying physical), likely not going to be a smoker if you're active. THUS, being physically active can determine other factors. THEREFORE, physical inactivity puts u at higher risk for heart disease
- Obesity and physical inactivity weren't on the list before, now they are.
- High fat diet is part of the being obese and high serum cholesterol risk factors
- Look into high blood pressure calculation
- Most people are just not active, 59% Americans are not active
- Anything above 240 units is high serum cholesterol
- Break down serum cholesterol to understand it more,
- Lipoproteins exist in blood, lipid containing proteins, they move cholesterol and triglycerides around the body
- Classes of lipoproteins are shown on the slide (circles on the slide);
- Chylomicron, Very low density lipoproteins.. etc.
- When fat is absorbed in the gut it is absorbed as triglycerides largely and it is packaged in vesicles called chylomicron (they are loaded with TG^)
- Chart under the circles show what is inside each one, example chylomicron has 98% lipid, 2% protein, density <0.95 (its going to float on water, density of water=1)
- Only time you see a Chylomicron (chylomicron is made in the gut) is when you eat a meal containing fat (a lipid) and you absorb those lipids and it travels in the portal circulation and it goes to the liver. Thus, It travels from gut to liver.
- The liver then produces other type of lipoproteins that make up total cholesterol. The brakes in read encompasses what we measure as total cholesterol. (VLDL, LDL, HDL) they differ in their density and in their composition
- VLDL(loaded with triglycerides, not as much as chylomicron.)
- LDL, lipid found in LDL is cholesterol (there is a lot of it) (this is the worst one)
- VLDL and HDL transfer lipids (triglycerides and cholesterol) to blood vessels and tissues and deposit them on the walls of the blood vessels (leading to acerolscorosis plaque, which can obstruct blood flow (this is when it becomes dangerous)
- HDL is made in the liver however it transports cholesterol from the tissues to the liver to be metabolized. Thats why it is the good cholesterol. (These are high in protein and cholesterol and the particles are smaller they are higher density cuz more protein) HDL reduces possibility for plaque build up.
- How to figure out density (centrifuge tube) separates things by their density.

- Going back to the total cholesterol levels, you want you HDL to be 25% or more of your total cholesterol levels.
- Effects of exercise training on your total cholesterol, levels of VLDL and LDL goes down and levels of HDL goes up. = lowers risk of heart disease.
- How active we are as Canadians; what west coast province is the most active province in the west (B.C)
- Canadians are a bit better than Americans in physical activity rates
- History' exercise physiology began in early 1900, used frog muscle to study the contraction processes, and metabolism (using heat)
- 1962 muscle biopsy technique (taking a piece of muscle and analyzing it, how we can measure ATP, this revolutionized exercise physiology
- 1980 there was a medical awakening, HDL
- Present, health benefits of exercise
- Physical education used to be only important or required to get a degree in it, or to be a gym teacher.
- Now you can do more by learning exercise physiology, nursing, wellness, medicine, research, nutrition, therapy.

Lecture 2
- discussing energy metabolism during exercise
- Today: ATP-PC system off energy
- Review from last class; How does LDL differ from HDL? 1. HDL is smaller it is filled with protein and cholesterol as opposed to LDL which contains just cholesterol. 2. High levels of HDL is good and high levels of LDL is bad. 3. LDL carried the lipids from the liver to the tissues as opposed to HDL, which carries the lipids from the tissues back to the liver to be metabolized.
- ATP is the energy currency of the cell. ATP structure (In the box in the slide)
- ATP**ase** cuts ATP. The name changes every time u cut ATP, ATP then ADP then AMP then Adenosine (composed of only a ribose and an adenine) when the phosphates have been sliced off).
- When an ATP is broken down to ADP and the free phosphate (inorganic phosphate Pi)
- Metabolism is generating ATP
- Myosin (a muscle protein) myosin ATPase breaks down ATP, breaks up atp for muscle contraction
- First few seconds of exercise; if you are in the sprint running position and the coach tells you to walk not run, you will have a lower energy (ATP) demand (because it is lower intensity) than if he tells you to run which would be high intensity and you would require a higher energy (ATP) demand. (Square function, you start and you end at resting)
- Lets say he will be running for a few minutes, the start needs to be high that starts up quickly, which is the ATP-PCr system. It covers the energy that is needed quickly, the other 2 systems (glycolysis and the aerobic system pick up from the starting gun but start up more slowly "sluggish")
- How does this system work? (ATP-PC system): Well, when you need energy quickly, like the sprinter. ATP is required immediately and is broken down by myosin ATPase in the cross bridge of myosin. The breakdown products are ADP(f) and Pi. (f)= free
- CPK (creatine phosphokinase reaction); uses the ADP from the previous reaction to regenerate ATP by combining phosphocreatine (PCr) and ADP, PCr donates a phosphate to ADP making ATP as a result and a creatine (Cr) is left.
- A lot of CPK in the cytoplasm of muscle cells that is ready to combine phosphocreatine (Also in the cytoplasm) with ADP. Regenerating ATP, by product is creatine.
- If we didn't replenish ATP the runner would not be able to keep running, first replenishment is the creatine phosphokinase (CPK)
- During exercise Phosphocreatine levels go down (to help replenish ATP), Cr levels go up.

- What gets Myosin ATPase started? Calcium! Calcium activates myosin ATPase.
- What happens when the runner stops? There is no more activation of muscles, reactions go in the opposite direction. Creatine phosphokinase CPK goes both ways (it is a equilibrium reaction) can drive the reaction back to the right to regenerate phosphocreatine.
- When you stop exercise creatine (Cr) levels go down to replenish phosphocreatine.
- No more demand for myosin ATPase, therefore that ATP at the beginning of the reaction is used with creatine to replenish phosphocreatine.
- The ATP that is used to replenish phosphocreatine after you stopped exercise is coming from mitochondria.
- Muscle structure: Muscles develop in our bodies from myoblasts during embryonic development. When we are in the uterus our muscle cells are not fully develop. They go from single nuclei myoblasts and they fuse together to form tube like cells (long multi-nucleated mature muscle cells= muscle fibres=myofibres)
- Review structure of a muscle fibre.
- Sarcomere
- Z line
- Thick and thin filaments
- Myosin crossbridge
- Excitation-contraction coupling in skeletal muscle: when we start to exercise our commands come from the motor cortex. Motor cortex sends impulses down to the spinal cord and from the spinal cord with have a synapse (we have an alpha motor neuron) which goes out to a peripheral muscle and ends with a neural muscular junction.
- We have vesicles inside the alpha motor neuron containing acetylcoline. When an action potential occurs, the acetylcholine is released out the junction and goes out into the synaptic cleft. Acetylcholine binds to the receptors and opens the channel, sodium comes into the cell. (receptor operated sodium Chanels)
- Membrane potential is negative, therefore sodium rashes In since it is positive.
- Black channels are voltage gated channels, they open and close due to changes with positive and negative.
- Look for a better explanation for this..
- Depolarization
- T-tubule continuous membrane?
- Black dot is zoomed up at the bottom of the page (in the square) how calcium comes in.



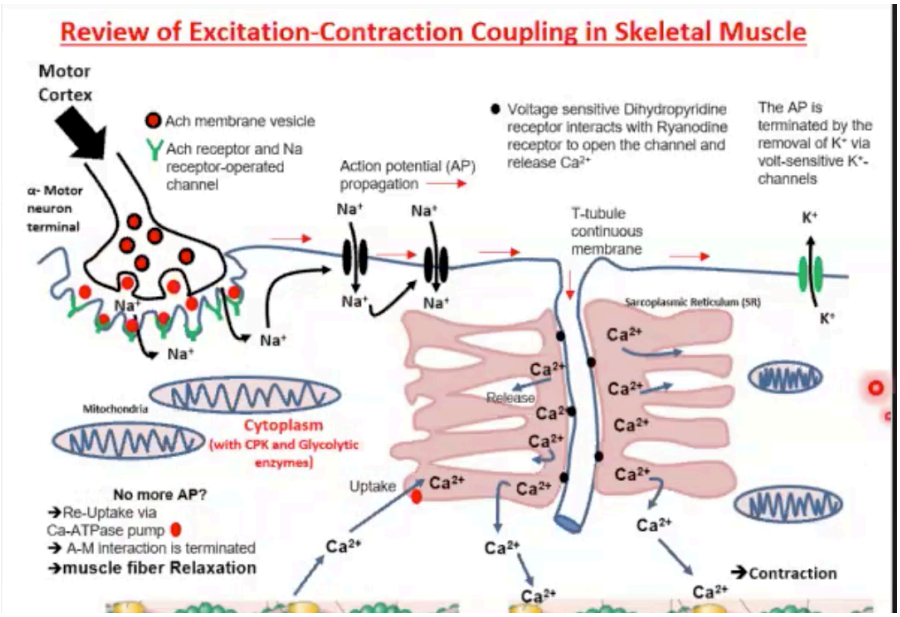
1. Acetylcholine released from the axon terminal binds to receptors on the sarcolemma.
2. An action potential is generated and travels down the T tubule.
3. Ca^{2+} is released from the sarcoplasmic reticulum in response to the change in voltage.
4. Ca^{2+} binds troponin; Cross-bridges form between actin and myosin.
5. Acetylcholinesterase removes acetylcholine from the synaptic cleft.
6. Ca^{2+} is transported back into the sarcoplasmic reticulum.
7. Tropomyosin binds active sites on actin causing the cross-bridge to detach.

- Q: ATP-PCr starting, the muscle contraction starts this reaction, calcium activates it.
- Q: phosphokinase is an enzyme

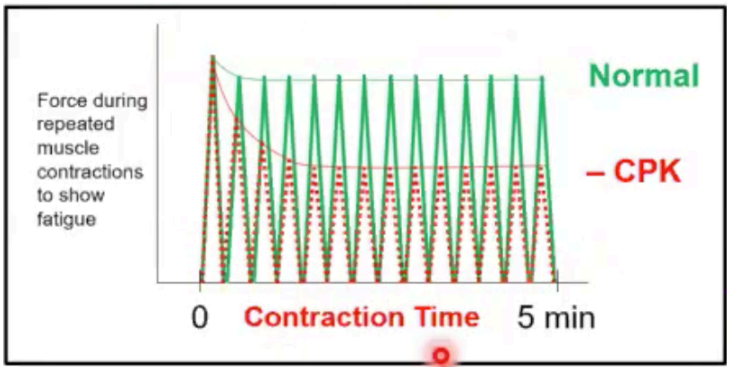
Lecture 3
- pages 9-14
- Today; creatine
- Quiz: what drives CPK in 1 direction or another? When you need energy to exercise right away, calcium will drive the reaction of breaking down ATP by myosin ATPase into ADP, then

phosphocreatine will combine with ADP to replenish ATP by donating its phosphate and creating creatine and ATP. (Phosphocreatine is already in large amounts in the cell, whenADP builds up in the cell thats when creatine phosphokinase CPK will start (to the left). When you stop exercise you want to replenish the phosphocreatine, ATP is not getting used anymore so it will donate a phosphate to creatine and produce phosphocreatine. (when creatine levels and ATP levels are high, it will drive the reaction to the right.)

- What does the Ryanodine receptor do? Black dots in the diagram are the ryanodine receptor. (Ca release channel) (EXCITATION-CONTRACTION COUPLING IN SKELETAL MUSCLE)
- Ryanodine receptors mediate the release of calcium ions from the sarcoplasmic reticulum and endoplasmic reticulum, an essential step in muscle contraction. ... However, as the concentration of intracellular Ca2+ rises, this can trigger closing of RyR, preventing the total depletion of SR.



- Facts about ATP and PCr; available for immediate use, located in the cytoplasm. Phosphocreatine is 3 to 4 times higher in concentration than ATP, If you relied only on ATP you would only be able to take a few steps. Phosphocreatine is there to make sure ATP is not being depleted. Phosphocreatine utilization depends on exercise intensity.
- To see how much phosphocreatine there is you need to take a biopsy of the muscle, graph; looking at different exercise intensity levels and levels of phosphocreatine. Highest level of PCr is at rest, they found that the harder one workouts the more PCr is depleted. Why doesn't it keep depleting? There are other systems that help. They pick up the pace. The level (Platos). Use phosphocreatine at first and then it Platos.
- How fast can you get phosphocreatine back? Its replenished very rapidly, you get almost all of it back very shortly after.
- Experiment, they blocked blood flow in the leg (like the arm cuff) which blocked oxygen delivery which prevents the mitochondria function which prevented phosphocreatine recovery. Which means that the ATP that is donating its P to creatine is going from the mitochondria.
- ATP-PC system is used for all exercise types however it is very important for events requiring high power output. Not plentiful its rapid.
- Another example why ATP-PC system is important at the start of exercise: experiment used genetically modifies mice. Knockout animal is an animal that has had its gene medically



modified on purpose. Useful in medical science, here, they knocked out the cpk gene thereby completely knocking out the atp-pc system. Cant use cuz the enzyme is not there. They made the muscle contract and they say that there was a drop (fatigue), but overtime will be normal Cuz other 2 systems take over. That initial drop shows the use for ATP-PC system.

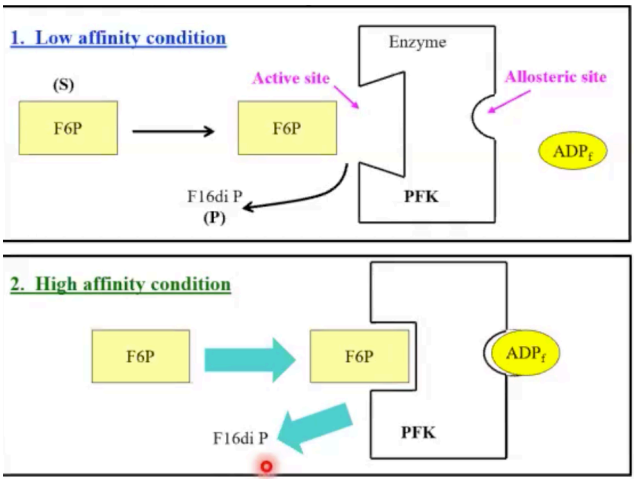
- Knockout vs transgenic (transgenic more copies of gene, check this)
- Benefits of creatine supplements: idea of taking creatine is to make more phosphocreatine, people who eat less meat (meat already contains creatine) will have a more drastic % increase of PCr.
- You are trying to synthesize the PCr when you take creatine supplements.
- Creatine ingestion has an osmotic effect on muscle cells- may give a false impression of Its anabolic effects. Creatine has an osmotic effect on the cell. Muscle cell I n the yellow, when you take 3g of creatine, it is taken up by the muscle, it induces an osmotic effect, water follows creatine, because there is a desire equilibrium, water follows and swells the cells. Blue arrow shows the expanding of the cell. Muscles look bigger because of this. It's not an anabolic effect its an osmotic effect creatine doesn't have the anabolic it has a water retention effect.

Lecture 4:

- Dashed lines around cell show the expansion from water.
- Creatine should be ingested with glucose to maximize uptake into muscle. Why? When you are glucose in any form, blood glucose goes up and when this happens it stimulated insulin to get secreted. (beta cells of the pancreas secrets it) insulin will bind to its receptor (on its muscle cell) and stimulate the uptake of glucose from the blood into the cell. Glucose out of the blood into the muscle cell. The insulin affect reduces blood glucose back down to normal. This is important for exercise and for diabetes.
- Green transporter on slide, called flute-4. Transports glucose from the blood into tissues (like the muscle cells). Important in Facilitated diffusion across the cell for muscle cells. Insulin doesn't just stimulate glucose uptake it also stimulated amino acid uptake. By activating the amino acid transporter (the square on the slide). When creatine is elevated in the blood, creatine levels go up in the blood, the amino acid transporter will facilitate the diffusion of creatine into the muscle cell. More affective way to get a more creatine into the cell.
- When you do take a supplement: normal vs supplement on slide, only 10% higher. Don't deplete as much since u start at a higher level. Faster recovery when u take a supplement. This is important for repeated bouts (interval training).
- Creatine supplement can allow you to work harder. You're sparing PRc during exercise and better resynthesis during recovery (gets replenished quicker).
- Load up on carbs when u do long distance performance.
- Summary on creatine; slide read it.
- Free ADP is useful for activating all 3 systems of energy production: 1) substrate for CPK 2) activator of glycolysis via PFK, 3) Rate-limiting substrate for the aerobic system (mitochondrial respiration).
- How does ADP activate PFK and thus the glycolytic pathway?
- Glycolysis has about 13 reactions, highlights the most important steps: Glucose —- fructose 6 is catylized by this PFK enzyme (phosphofructokinase), product fructose 1,6, di P. ADP free activated PFK, PFK is the rate limiting step. If PFK was faster, it would make more ATP, but more lactic acid…. We'll talk about this later. Every step is catalyzed by an enzyme. Substrate ——-(enzyme)——- product . (Michaelis- menten kinetics). Twice as much substrate, twice as much product. With more substrate you will make more product and velocity will increase but then velocity will come to a plateau, since a cell has a certain number of enzymes, when there is a plateau you have saturated all of the enzymes (Vmax)
- Vmax (is also theoretical number) helps characterize enzymes, another way to characterize enzymes, is with a different number, Km, a theoretical number that represents the substrate concentration that elicits 50% of the Vmax for the reaction. It gives you 50% of the Vmax. (If

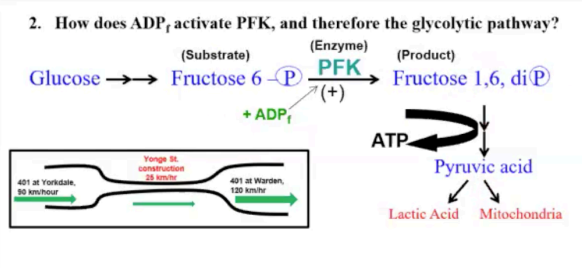
you look at the graph, if you go down to 50% on the y axis, you go a cross to the x axis to find the Km, what the substrate is at that point.

- The Vmax can change if the numbers of enzyme molecules increases or decreases.
- When the muscle is at rest you don't have that free ADP, when your muscle contracts, you have the free ADP.
- How does ADP activate PFK enzyme? Enzymes have compartments or (active) sites that interact with the molecule. F6P is going to go into the active site and convert to F 16di P, (low affinity condition- this is when muscle is at rest. F6P may stay in the site or may fall out because of its shape, not being held onto tight enough, more and more substrate you will produce more and more product, until you use all of the enzymes.
- High affinity condition; when you have free ADP, free ADP binds to PFK and this changes the conformation (shape) of PFK, the shape becomes perfect for F6P, so that it fits perfectly and it doesn't slip out like in low affinity condition.



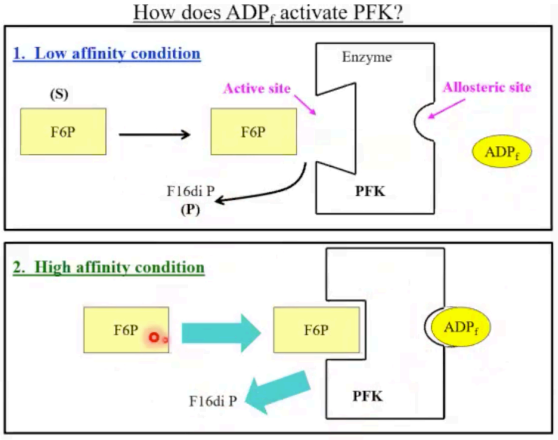
- So if you look at the graph, when you have free ADP, PFK works more effectively. \
- The lower the Km number, the higher the strength of the interaction between the enzyme and the substrate. (illustration on the right).

-so the addition of ADP will increase the reaction speed and make ATP faster.

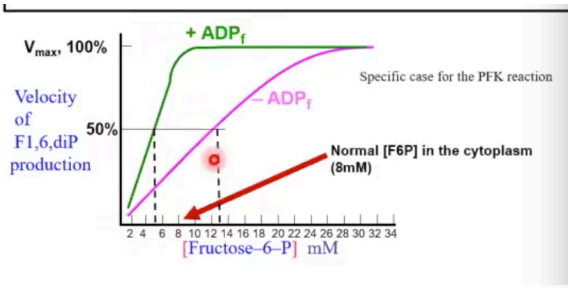


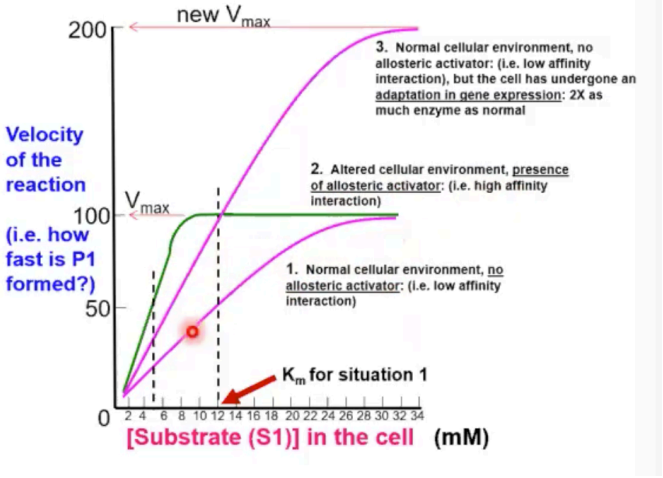
Lecture 5

- Aerobic system
- Quiz questions? 1. How does acute exercise modify a)PFK kinetics, b)Vmax of PFK 2. Hoe many cycles are there in the b-oxidation of a 14 C fatty
- Page 16, how free adp activates glycolysis and in order to do that you have to activate the rate limiting step (phosphofructose kinase)
- We need this when we start to exercise
- The Vmax is a reflection how many enzyme molecules you have, if you have more, the max would be higher. Less would be lower.
- Km has do to with the strength of the interaction between the substrate and the enzyme. The lower the Km the higher the strength of the interaction, we call that high infinity and low infinity.
- Low affinity condition, high affinity condition (if you see on page 16, it is more productive, the shape fits exactly.



- Graph on the bottom of the page, if you increase the substrate you would increase the amount product formed (on vertical axis).
- When you saturate all of the enzymes you get the Vmax.
- Because high affinity condition is more affective you'd not need as much substrate to produce product. (from pink to green).
- For example at 8mM how fast are you making product, around 30% but if in the presence of free adp, about 95%. (probably moving more lactic acid).
- 3 situations, 1. 2. 3. (Vmax has not changed between 1 and 2, km changes.
- 3. Has a higher max (adaptation in gene expression)
- We undergo transcription and translation to make more protein.
- Acute exercise on PFK, to increase the level of free adp, the adp binds to the site on pfk and it changes the confirmation of pfk, allows for the substrate to bind to the active site more easily, more efficiently, makes pathway go faster. PFK is the rate limiting enzyme in the pathway.
- Low affinity, there is still binding but not as efficiently
- High affinity (Shifting curve to the left)

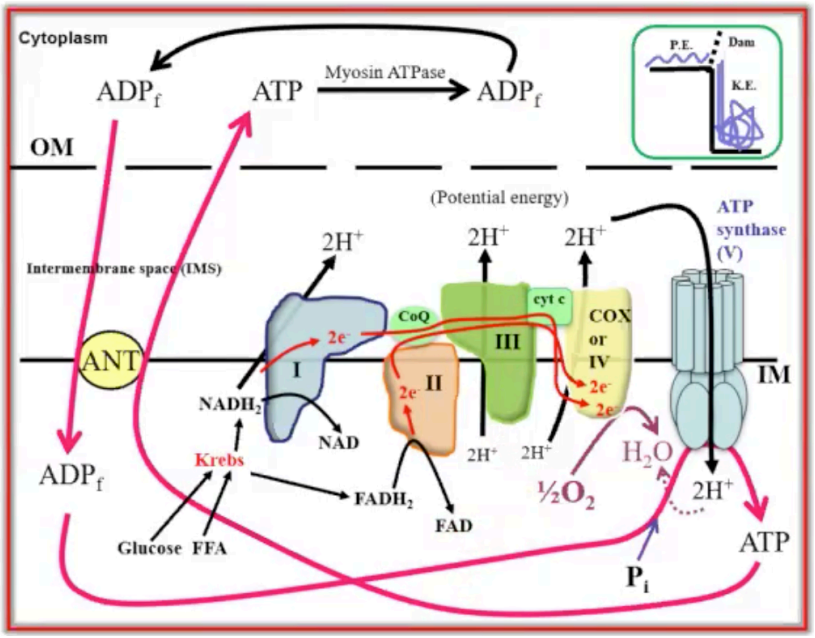




- Overview of the aerobic system; we metabolize 2 substrates during pro longed exercise, fats and carbohydrates.
- Carbohydrate break down on the right, Fat breakdown on the left.
- Glucose is C6H12O6.
- Fist the C6 is broken down into 2 carbon fragment. Then the it is broken down in to 2C (Acetyl CoA) and CO2 (we breathe it out) is another product by pyruvate dehydrogenace.
- Acetyle CoA enters the Kreb's cycle, First step: the 4C (oxaloacetic Acid, (OAA) combines with acetyl CoA that came from glucose and it produces 6C (Citric acid(citrate)). Through this reaction CO2 is produces, FADH2, NADH2, and ATP is also produced.
- Fatty acids, anything between 16C to 24C. Fatty acids enter the muscle and go through a process called beta oxidation. In this process, there are enzymes that cut off 2C at a time, they form Acetyl CoA (2C). So for example C18 would go to C16, then C14, then goes through cycle again, C12,C10,C8,C6,C4,C2 and they enter the Kreb's cycle like the one from glucose.
- Every time there is one cycle of beta oxidation, you produce one NADH2, one FADH2, and one acetyl CoA (the NADH2 and FADH2 produced go into the electron transport chain, and the CoA goes into the Krebs cycle). Thus breakdown of fat is important.
- How many CoA are you going to make at C18, 9!!! How many cycles did it take? 8!!
- Electron transport chain.
- Mitrochondria is the p over house of the cell it produces a lot of ATP. The cell has a porus outer membrane, can allow for some stuff to come in (selevtive), intermembrarne space is inside the cell, mitochondria has a very compact membrane, doesn't allow for things to come through.
- Adenine nucleotide transclocase is a transporter to bring free ADP into the mitochondria, does that by exchanging one in one out, with what? With a newly made ATP.
- We are breaking down ATP and have to re make it.

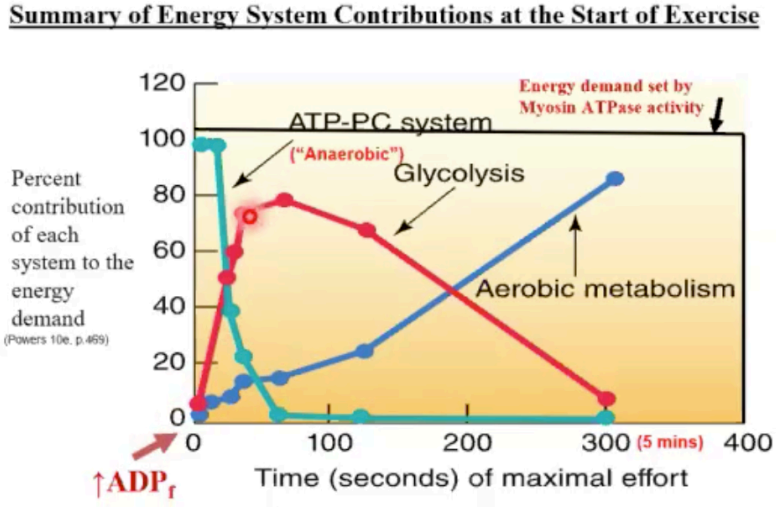
- Electron transport chain, makes water and ATP.
- NADH2. Step one you are going to split the atom. If we take H2 and we split it into 2 protons and 2 electrons, The protons are pumped into the inter membrane space. 2electrons to CoQ then through complex 3 then complex 4 then the electrons combine with the protons and produce water.1/2O2 is for every 2 electrons and 2 protons. (see full explanation 45 min lecture 5.

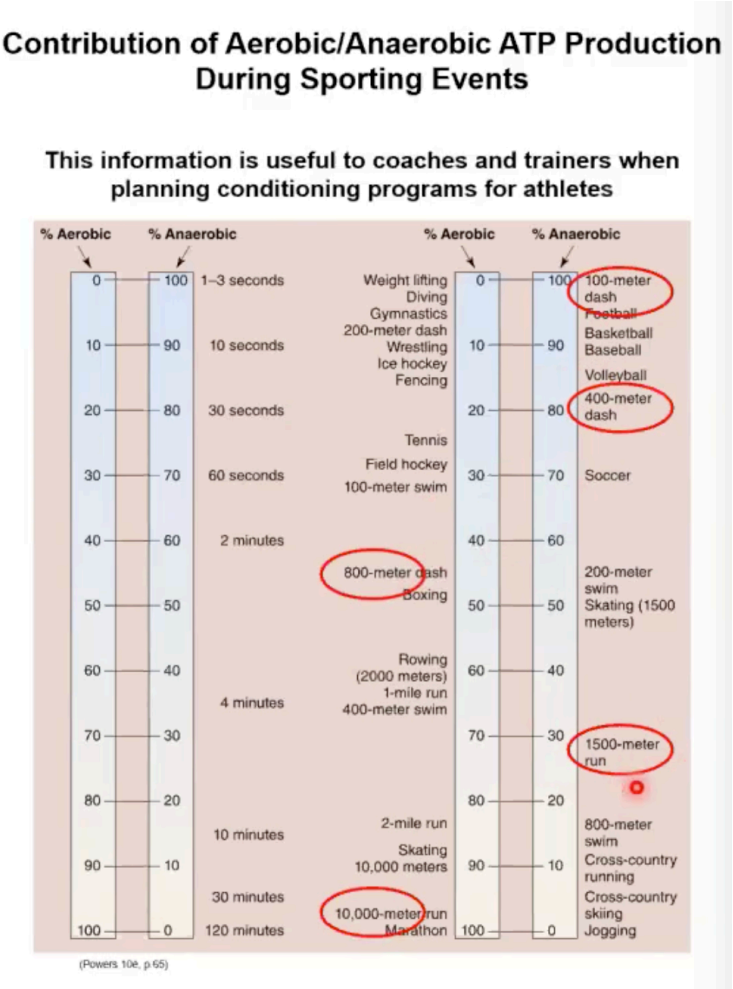
Lecture 6
- Page 20 review
- How were converting free adp that comes into the mitochondria into ATP.
- As protons move, electrons are pumped
- They are pumped to get a proton
- Where does the phosphate come from? It comes from a carrier in the membrane (theres a lot of phosphate in the matrix) it doesn't limit the reaction always plenty of it, whats limiting is the availability of ADP.
- If we had no oxygen here this reaction could not take place. Aerobic system requires oxygen. (No oxygen would mean that the electrons get backed up) aerobic system gets shut down, no atp made, cells die. (does this happen? Is oxygen the limiting factor during exercise? And therefore do u have to use glycolysis and atp phosphokratine? NO! Under most conditions oxygen is not a limiting factor. It is only limited if you have a blood flow problem (air up in the mountains).
- Cyanide inhibits the enzyme in IV channel, which would cause backing up of electrons, thus cells die.
- FADH2 is oxidized at complex 2
- Cytochromes- contain the red pigment heme, and heme contains iron, which accepts and donates electrons in the ETC. (oxidized means it has lost an electron, reduced means it has gained an electron).
- Red blood cells are red because they contain heme which has red pigment.
- Free ADP controls how fast.
- Cytochromes; channel 3,4 and cytochrome C contain cytochromes
- Google where electrons and protons go during the electron transport chain.



- ANT (transporter, takes one ADP in and Takes one ATP out)
- How is the water formed? Google this
- Summary of energy system contributions at the start of exercise
- Chart on percent contribution of each system to the energy demand.
- ATP-PC system, Glycolysis, Aerobic metabolism
- ATP-PC system is activated right away, largely used then quickly diminishes, glycolysis then picks up, and a little bit of aerobic metabolism (the last to activate, slowest).

- Same systems are activated through ANY type of exercise (gym Orr getting out of bed, etc.. just at different intensities)
- They are all activated at the same time but its how quickly they get warmed up (ATP-PC is able to quickly work)
- Anaerobic, oxygen is not used to make ATP (ATP-PC system). Not because there is no oxygen… there is always oxygen.
- Dont memorize the table, (table is example of when you use the aerobic and anaerobic (when it kicks in) Contribution of aerobic/ anaerobic ATP production during sporting events. (photo on the bottom right)
- During interval exercise (repeated) u use glycolysis and lactic acid can build up.
- High intensity interval training, 10-20 seconds (if you look at graph) its in the right bottom corner. You get to acidosis (lactic acid build up) when you're focusing on exercise intervals of about 45-75 seconds (This is when glycolysis is being used predominantly, you are basically relying on glycolysis here).
- Interval training, pick a time to avoid acidosis.
- Effect of interval exercise on metabolism and the CV system.
- Conditions are shown (seconds being active vs recovery time). Recovery is twice as long as exercise time.
- The longer the exercise in seconds and recovery time, the more blood and muscle lactate (thus more acidosis).
- Muscle lactate will always be a little higher than blood lactate.
- Lots of lactate no harm, but you get higher fatigue. More you train less lactate you make. No long term harm with acidosis.

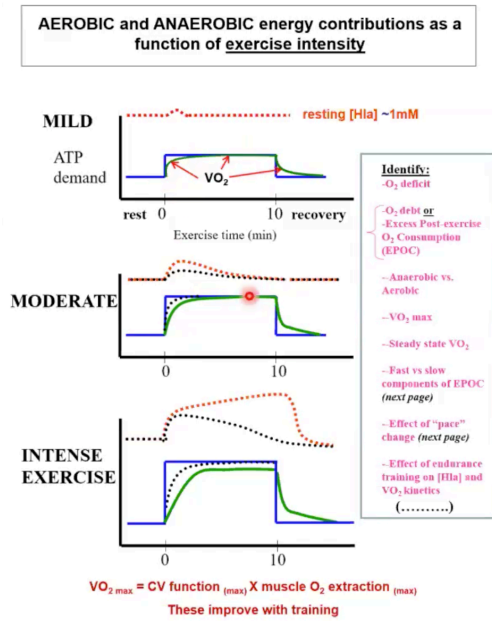




Lecture 7
- Review on energy systems
- Interval exercise
- Aerobic and anaerobic energy
- Make sure you know terms on page 24
- Lactic acid is an indicator of glycolysis
- Lactic acid makes you tired
- Blood glucose doesn't change during exercise (more important to maintain in comparison to lactate. We have systems that

maintain glucose levels).

- Our endocrine system maintains our blood glucose level of 5mM (millimolar)
- Heart rate, not enough time to go all the way down to resting during the recovery phase.
- You're using anaerobic systems largely for those bouts of exercise. (interval)
- H.I.I.T high intensity interval training. (aerobic benefit, your cardiorespiratory fitness will improve)
- Pick duration exercise that suits your event but if u can pick which to do It is better to pick high intensity low duration exercise you can avoid acidosis.
- 3 different types of intensities and the different type of atp demand relative to the change in intensity. What are the differences? ATP demand depends on how hard you're exercising.
- Lets say you are walking (it goes up until the amount of energy required, then when u stop it stops.)
- The blue box is how much atp Is required, you have to fill this box. You are going to use anaerobic energy to start. Aerobic is going to slowly climb (showed by green line).
- How do we measure the aerobic system? We measure oxygen consumption (VO2).
- We measure VO2 at the mitochondria
- Aerobic system ends up matching the exercise (steady state) , but before it does, there is a triangle (space where it does not match), thats the energy that is supplied by our aerobic systems (glycolysis and ATP-PC system)
- The plateau that we see is called the steady state.
- Little triangle area (oxygen deficit), muscles are never out of oxygen (except for special circumstances) so this name is unfortunate.

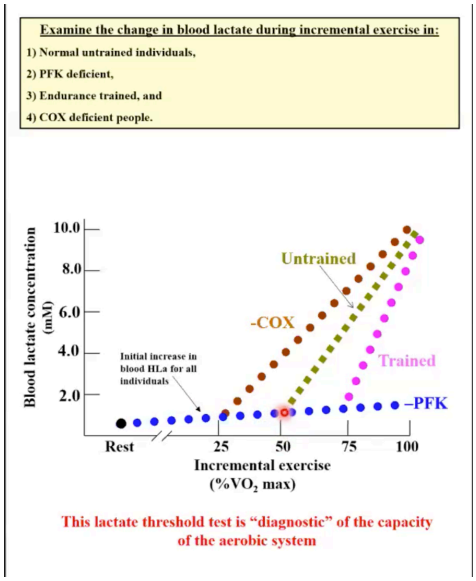


- There is also triangle at the end when u stop exercising (where the green line is above the blue square this time) you don't shut off like a switch, slowly shuts down, rapid stage and a slower phase).
- This end triangle is called a debt or Excess post-exercise)2 consumption (EPOC)
- Fast vs slow slow downward will be explained.
- Lactic acid is the dotted lines, if you look at the mild exercise circumstances for example if you get up from a chair and start walking around you use a bit of that anaerobic system (glycolysis) thus there is a little lactic acid that is invoked. (about 2mM of lactic acid)
- Lactic acid at rest is one mM (millie molar)
- Bigger beginning triangle (space between blue and green lines) means more glycolysis is being used thus more lactic acid and it will take longer for the lactic acid to go away.
- Intense exercise, (example pushing yourself to the max for 10 minutes, your oxygen consumption cant match the energy demand. You picked a work load that you cant match, the steady state is below the energy demand. In this case, its more than a triangle, you're going to have an anaerobic contribution throughout the entire 10min period. Since your aerobic system cant match the demand you've decided to do. Lactic acid thus continue to rises throughout the exercise until about 2 min after u stop exercising. And it will eventually come back down again.
- What does training do to this, effect of endurance training on lactic acid and VO2 kinetics; if you continuously do this type of exercise, you will adapt to it. Your oxygen consumption will be higher (green line). Your VO2 max will increase.
- What is VO2 max, it is a product of cardiovascular function and the ability of muscle to extract oxygen. Both of these things improve with training.

- If you look at the photo ^ the green line will shift up closer to the blue square (making a smaller triangle at the beginning) due to consistent training).
- With training your aerobic system is more easily turned on. (you can turn on mitochondria more quickly because you have more mitochondria. Your VO2 max will you p (10-15%)
- Thus, the deficit after is smaller with training (the triangle). You're also sparing phosphocreatine in this case and you're not making as much lactic acid. (they will start to go up but then go down, less acidosis)
- Fast and slow component:
- Relationship between VO2 core temperature and PCr during exercise and recovery: your oxygen consumption goes up when you start, when you stop you start to recover and your excess post oxygen consumption goes down rapidly at first then comes down more slowly over time.
- Phosphocreatine goes up then down when you exercise and then hits a plateau when the other systems kick in. The pattern of recovery of phosphocreatine is fast when you stop working out. How do we re synthesize phosphocreatine? We use oxygen consumption in mitochondria to drive that reaction back to phosphocreatine. ATP needed to re synthesize phosphocreatine is coming from O2 being consumed and converted to water by the mitochondria.
- Drop in body temperatures compared to the EPOC rate. (the higher your temperature the faster your reaction rates go)
- What is oxygen consumption? Series of reactions in the mitochondria. SO the higher your body temperature, the rate of consumption is higher.
- As you cool down reactions rates slow down. (correlation)
- We've been talking about exercise at a constant load (not reality)

Lecture 8
- Page 24. Trying to meet energy demand with the supply (anerobic and aerobic)
- VO2 max, change pace to get to maximum
- We are going to learn how to preform a VO2 max test.
- You have VO2 per Liter per minute on the y axis and mins on the x axis.
- We are measuring oxygen consumption (plotting on y axis)
- We are also measuring blood lactate levels at various times at each work load.
- Work load is changing progressively.
- In an average sized person, your oxygen consumption at rest is about 1 quarter of a liter, per minute. (.25 liters per minute)
- When you are representing your oxygen consumption always use the plateau (steady state) as the number.
- When your VO2 max doesn't change when you increase your work load that means, it is you VO2 max. (not able to consume more oxygen) Maximum capacity to take up and use oxygen.
- 2 criteria on how you know if you reached VO2 max; 1. Plateau (oxygen consumption doesn't change when you increase work load), and lactic acid (increases when u hit VO2 max) look at graph on page 28. What percent of your VO2 max does the lactate threshold occur at.
- Based on the percentage for the lactate threshold, you will be able to tell if the person is well trained, physically active, or quite sedentary.
- If your aerobic system is poor, your lactate acid threshold will be low. And if your aerobic system is good, your lactic acid threshold will be high.
- Second criteria of VO2 max, its attaining a blood lactic acid during the test of around 10 mM (if person gets around 8-10mM, its additional criteria saying that they reached VO2 max)
- To get to VO2 max you need your heart and oxygen extraction to be working
- Heart rate times stroke volume gives you cardiac output (how we measure cardiovascular function)
- Oxygen extraction part (how well your muscles are taking up the oxygen being delivered.)

- Incoming units of arterial oxygen (A) are 20 units coming in! And at rest 15 units coming out on the veinous side (V) . Extraction is (A - V)02 (20-15=5) units was extracted to make ATP at rest. When you start to exercise and go to VO2 Max the extraction increases dramatically, from 7,8,10,15,17 … mitochondria requires the oxygen to make ATP.
- The fitter you are, the longer you will live.
- Benefit of being fit (or having a high VO2 max), Epidemiology study,
- Higher your level of fitness the lower your mortality rate
- Study done on men working out, they were less likely to die from anything, less likely to develop cardiovascular disease. Probability of survival is much better
- The lactate threshold test is diagnostic of the capacity of the aerobic system. The more lactate you produce that tells you that your aerobic system is not that good, less lactate you produce means your aerobic system is good.
- Some people are born with deficiencies not allowing them to work out 100%.
- How do you compare a trained person to a untrained person when they have different levels of VO2 max? You call there maximum 100% whether its 3 or 4, you call it 100%. Now you can equate them at 100%
- Photo on right, the x axis is the capacity percentage, as you can see at around 50% the lactate for an untrained person goes up. Lactate threshold for a trainer person is higher than an untrained person. (better aerobic system) they don't have to rely on anaerobic systems. Reducing acidosis. Helps them perform better.
- A person with COX deficiency, they're aerobic system is not strong. They produce lactate at very low work loads. they have a lactate threshold of about 25-30% of VO2 max compared to trained person of 75%.



- PFK deficient, they cant use glycolysis very well since they don't have regular PFK activity. They have low PFK activity. When they start exercising they don't produce lactic acid, they cant use glycolysis and they cant use carbohydrates properly. Highly reliant of fat oxidation (u need a mixture for good performance)
- Three systems of energy productions… free ADP is used equally in all of the energy systems for an untrained person (on slide). And for a trained person, it will be used less by atp phosphocreatine system, free ADP will use glycolysis less (Less lactate acid produced), it has more mitochondria so it will be making atp aerobically. The way we make more mitochondria is through a change of gene expression (transcribing and translating more mitochondria)

Lecture 9