Lecture 2

The importance of knowing the ins-and-outs of the body aids to effectively and properly provide the means to maximize human performance. Physiology, the study of the body down to the cells, allows us to understand the functions of the human body, and, when applied properly, can help us achieve almost any fitness goal we have. However, not all bodies are the same. The fundamentals of a human body are similar in each person, but the presence of a previous or current injury or any underlying health disease can affect how a personal trainer or a physical therapist services their patient. For example, a patient whose goal is to beat their mile time, but has a cardiovascular disease, might not be able to run at a high intensity compared to a person who has no medical history of a heart disease. This enforces the concept of individuation, and that each person needs their own exercise routine that aligns with their body and their goals.

My current goals touch upon 3 of the 4 types of goals. One of the goals I have is to gain muscle and endurance because I want to get into jiu jitsu, and develop a more toned body. Another goal is to develop healthy habits to alleviate any pain that can be caused from poor lifestyle choices. I would say that the categories of physical appearance, athleticism, and health/longevity are split fairly evenly.

Lecture 3

The function of a skeletal muscle depends on the orientation and insertion of the muscle, and the fiber direction. The longer muscles shorten faster and have a larger amount of muscle that is shortened compared to short muscles, but short muscles create a larger force. Each muscle is surrounded by sheaths. The epimysium is the outer layer that surrounds the whole muscle. The perimysium surrounds the fascicles or groups of fibers that make up the muscle. The last type of sheath that surrounds individual muscle fibers is the endomysium.

The muscle fibers that make up the muscle are composed of myofibrils, which in turn are composed of sarcomeres. Those sarcomeres are made up of myofilaments, actin and myosin. At the M line of the myosin are proteins, connective tissue and structural structures that help determine where the M- line is. The z-disks mark the beginning and end of each sarcomere.

Each person has a different amount of fibers in the same muscle, and each muscle has a different amount of muscle compared to another muscle in the body.

Tropomyosin and troponin make up actin. The configuration of the three polypeptides found in troponin, which are T, C, and I, determine whether tropomyosin is attached to the binding sites or not. If calcium is present, it binds to the polypeptide troponin C, which causes a confirmation change of the troponin complex. This causes tropomyosin to move off the binding sites, and myosin to attach to those binding sites.

Through a process called cross bridge cycling, the muscles in our body contract. Cross bridge cycling causes the sarcomeres to shorten as the z-disks are being pulled together by the binding and unbinding of the myosin heads on the actin binding site. ATP is needed to fuel this routine, which causes the myosin heads to detach. The reattachment and attachment of the myosin heads make it appear as if the myosin is walking on the actin. This is called the sliding filament theory.

Lecture 4

Lecture 4 covered the basis of how the brain signals the body to move through the use of the nervous system and action potentials. What I found interesting was that each type of muscle had a different refractory period. For example, the video mentions that cardiac muscle had a longer refractory

period than skeletal muscle. From a logical viewpoint, it makes sense that cardiac muscle would need a larger refractory period. If the heart experiences tetanus contractions, it would be impossible for the heart to fill all the chambers with enough blood needed to prevent cell death.

Allowing skeletal muscles to have a shorter refractory period provides our bodies with the ability to continue to contract muscle over periods of time. An example is maintaining correct sitting posture. The skeletal muscles found on the body's posterior side assist in keeping one's back straight.

What also needs to be taken into consideration is the number of motor units recruited into keeping one's back straight and upright. Both the rate coding, the frequency of action potentials, and the amount of motor units recruited allow us to determine the force created by the muscles to perform the action of sitting up straight.

Lecture 5

In the fifth lecture the concept of muscle spindles and how they are a part of a reflex arc fascinated me. The muscle spindles detect the length of the muscle, the rate at which the muscle is lengthening, and the tension. These spindles are involuntary or intrafusal fibers, which send signals to the central nervous system. In muscle spindles, the spinal cord plays a huge role as the interneurons bypass the brain and connect the afferent and efferent pathways to create a reflex arc. The result of this reflex arc is the activation of muscle tissue to prevent the tearing of muscle that is stretched. This reflex arc uses gamma motor nerves instead of alpha motor nerves.

The importance of the muscle spindles being involuntary is to prevent major tears or damage to the muscle. If used correctly, muscle spindles can help with flexibility and/or generate more force. Some examples are vertical jumping or kicking off the pool walls. Both have the same position of quickly going down into a squat and then an explosive push through the soles of the feet. This creates a greater amount of force compared to just jumping up without using muscle spindles.

This is a prime example why knowing and understanding the human body is necessary. By using the information about muscle spindles and the reflex arc, we can target certain goals effectively.

Lecture 6

There are different kinds of fiber types present in the body. The most well known fiber types are type I and type II. Type I muscle fibers are energy efficient and red colored, while type II muscle fibers are white colored and consume large amounts of ATP. Therefore, the other pseudonyms for type I and type II are sensible. Type I is also called slow twitch muscle fiber, and type II is called fast twitch muscle fiber. Both of these fiber types have subcategories such as type Ic or type IIa. It is interesting to note that it is possible to alter fiber types; however, the muscle cells contain some remnants of the fiber type it was before.

The pattern of where the muscle fibers are located is also fascinating. The study mentioned in the lecture came to the conclusion that the deeper the muscle fibers are in the body, the larger the amount of slow twitch muscle fibers- type I- are present. At the superficial lever, there are more type II muscle fibers present.

Lecture 7

Postactivation potentiation (PAP) is a short-term boost in performance causing a more forceful contraction of the muscles than the previous contraction. The mass recruitment of motor units from doing heavy load exercises is the main cause for this reaction; however, the phosphorylation of the regulatory

light chain also plays a part in PAP. The phosphorylation of the regulatory light chain makes the thin and thick filaments sensitive to calcium, enhancing cross bridge cycling in the muscle.

Hennman's size principle and Denny-Brown D. and Pennybacker J. explain the reasoning why postactivation potentiation works. Denny-Brown D. and Pennybacker J. discovered the principle of orderly recruitment. Both principles, Hennman's principle and the principle of orderly recruitment, have the same base where there is an order to which muscle units and fibers are recruited first, but Hennman's principle takes it a step further by including the size of the motor unit also determines the order. By knowing that motor units with a higher ratio of type I fibers to type II are the first ones to be recruited followed by the size of each motor unit, we can understand how postactivation potentiation works.

If we do heavy load exercise, more motor units will be recruited starting from the small motor units primarily consisting of type I fibers all the way to the large motor units that are composed of type II muscle fibers. When a large portion of the muscle units are recruited and type II fibers are activated, that is where peak activity and a stronger forceful muscle contraction occurs. This allows for the improvement of performance mentioned in the first paragraph. During this phase, type I muscle fibers are not contributing to the action of the muscle.

Factors that inhibit PAP are cold, pain, and fatigue. MVC is one of the ways to improve your PAP. Isometric holds can be another way to improve PAP, but there is lack of appropriate time under tension.

Lecture 8

Size principle is not based solely on the load. For example, the lecture mentions participating in an exercise that can recruit most or nearly all of the motor units with a load that is not even 100% of one's maximum amount of strength.

I found that the use of GABA is important for self preservation; however, too much inhibition from GABA can be detrimental. There are two types of inhibition, which are reactive and proactive inhibition. Reactive inhibition is an unanticipated inhibition, whereas reactive inhibition is the opposite.The older one is, the more difficult reactive inhibition is compared to proactive inhibition. Also, the older one gets, the less type II fibers are present in your body, and a decrease of motor units occurs.

Something I learned from this lecture video is the futile use of static stretching to prevent any injuries. In fact, it ruins the performance of the athlete. Static stretching can affect the muscle, and can cause the muscle to not recoil as well. It can be used to help with posture though. Dynamic stretching can be helpful as it warms up the athlete.

Lecture 9

There are different types of ways for the body to adjust to the environment that it is placed in. Adaptation, accomodation, and habituation, although ways to preserve the body, are different ways the body tailors to its surroundings. There is overlap between the terms though. Accommodation is a process where the body temporarily adjusts to the environment, while adaptation is a relatively permanent biological alteration. Generally, adaptation follows accommodation if the subject undergoes longer periods of stress. Habituation is like accomodation, but it can be recaptured. Accommodation is readily reversible. Adaptation still occurs after the stress of the environment is removed; however, accomodation is more favored than adaptation. It costs more energy and proteins to create muscles or any modifications to the body in order to adapt compared to the body just accommodating the environment around it. Adaptation is also specific. When the body adapts to its surroundings, only the muscles or bones that

directly undergo the stress in the environment adapt. The whole body does not adapt. That would cost too much energy, and it might not be beneficial to adapt all parts of the body in that particular environment.

Lecture 10

Adaptation is relative to the type of stress the body undergoes. Different variations of stress incite specific biological adaptations; however, even though specific parts of the body are targeted, there is a small amount of overlap into what muscles, tissues, or nerves are affected. The process of adapting is intended for survival. Fitness is also self-preservation. All living organisms, cells, etc. continue to adjust to their surroundings.

Angular specificity is another type of adaptation that is related to isometric strength training. This phenomenon is when a person can maintain an isometric hold only at a specific angle. Any degree off the defined angle can or will cause the person to have difficulty to preserve the isometric hold.

Another type of specificity accounts Mike Tyson's experience when fighting an amature after his release from incarceration. During his sentence, Mike Tyson exercised, and his muscles adapted to the environment. However, he never got physical with anyone during his time in prison. When he finished his sentence and fought an amature boxer, his previous tolerance for taking punches decreased after he was released. His body became used to an environment of exercise and no physical contact, and it adapted to that environment. This concept backs up the fact that the body does not act to different stresses in the same way, which is a concept that was speculated by Hans Selye. Instead, adaptation of the body depends on the stressors in the environment.

Lecture 11

The topic of what type of shoes are better continues to be a trending topic. Are minimalist or no shoes better than supported or maximal shoes? There is no definite answer to this question. The type of shoe that is deemed "better" is relative to the user of said shoe. For example, a person who suffers from a pronated gait would need a shoe with better support than to simply run barefoot. The supported shoe can help correct the position of the foot when the person is running. Someone who uses minimal footwear or no shoes might not even need supportive shoes if they do not have a supinated or pronated gait.

Minimalist type shoes are those that cause little to no change between the interaction of one's feet and the ground. Maximal type shoes are the opposite of minimalist shoes. It is important to note that people who are switching from supportive or maximal shoes to minimalist shoes or barefoot should adjust to the minimalist shoes or being barefoot properly to avoid injury.

Daniel Lieberman makes a valid point that our ancestors did not have shoes and performed persistence running. The introduction of shoes weakens our feet and can cause injuries or overpronation. It also gradually dulls neuromuscular balance. Certain types of shoes can have a greater impact on the foot. Flip-flops and heels are some examples. Flip-flops are maximalist type shoes that can cause a deformity of the feet. Heels can cause damage to the posture, shorten the gastrocnemius, and even shorten the hamstrings.

Lecture 12

Biomechanics is how components interact to create movement. There are always consequences if one changes the impact of a certain body part. For example the surgery to assist patients with cerebral palsy lengthens the leg, but results in a damaged knee flexion and the patient dragging their feet.

Statics is the study of bodies at rest, and dynamics is the opposite of statics. Kinematics is the study of the movement itself, and kinetics is the cause of the motion.

There are three types of joints. The first one being uniaxial, the second one biaxial, and the third one is multiaxial. The uniaxial joint moves in a hinge-like motion. The biaxial joint can move up, down, left or right. It will not be able to rotate like the multiaxial joint though.

Fibrous attachment is the type of attachment that is commonly associated with muscles. The fibrous attachment is weak at the myotendinous junction, which lies between the tendon and the muscle. Fleshy attachment is where the epimysium is continuous with the periosteum, and is weaker than fibrous attachments.

Classes of levers range from one to three. Class two levers are mechanically advantageous, and require less effort, but a slower motion is created. The third class of levers is always mechanically disadvantaged. This means that it requires more effort, but produces more speed.

Robin McKenzie and Dr. Paul Williams have theories in regards to alleviating spinal pain, but they are the complete opposites of each other. Robin Mckenzie believes in spinal extension to help spinal pain, and Dr. Paul Williams believes spinal flexion will help with spinal pain.

Lecture 13

The body is mostly composed of mechanical disadvantages with some mechanical advantages. This means that there are more third class levers present in the body than second class levers (mechanical advantages). However, some muscles in the body are not levers, i.e., the tongue. With the body composition consisting mostly of third class levers, swift movements are achievable. Some examples of brisk actions are punching or bicep curls.

The location of the insertion of the muscle is an interesting topic. Depending on the point of insertion of the muscle, the angle created from the starting position or amount of motion can be large or small. If the velocity and every other aspect of a muscle contracting is the same in two subjects, but the point of insertion is different between the two, the muscle that is inserted farther from the joint has a small amount of motion when it flexes compared to a muscle that is inserted closer towards the joint. The muscle that has a point of insertion closer towards the joint behaves like a third class lever. This means that it has more speed than the other muscle with the point of the insertion farther away from the joint. The muscle that has the insertion point more distal to the joint can pick up larger loads easier than muscle insertions more proximal.

Lecture 14

Each sport or activity requires different mechanical demands, and one's body develops specific adaptations to the stresses that are imposed on the body. A certain activity could involve more load applied or different muscles used compared to a different activity. The muscles, organs, and other parts of the body that are involved in repetitive actions hypertrophy while the other parts of the body do not develop. The lack of equal growth and development throughout the body causes some parts of the body to be more vulnerable to injury.

If a person is injured, the body will deliberately or unintentionally avoid the pain that stems from the injury. Other muscles will be recruited or the muscle itself will be recruited differently to alleviate the pain. A disadvantageous adaptation is formed from this phenomenon, and properly using the right muscles to prevent injury will need to be relearned. By learning the most beneficial way to use muscles,

one can preserve the muscle tissue; however, there still is a high risk of injury if there is previous injury history.

The pain received from any affliction causes the neurological pathway to be affected, too. The muscle spindles and the efferent pathways might act differently based on the afferent pathways of pain. Any altered firing patterns cause an increased risk of injury.

Lecture 15

Tropomyosin receptor kinase A (TrkA) is a receptor on the nerve that mediates nerve growth factor. The purpose of the nerve growth factor is for the function, formation and survival of the neurons. If no TrkA is present in the body, the production of sweat or the ability to experience pain is not possible. TrkA also mediates the presence of Aδ and C fibers in the body.

The Aδ fibers and the C fibers are nociceptors. The Aδ fibers are thinly myelinated and signal sharp immediate pain to the body. Unlike Aδ fibers, C fibers are not myelinated and are slow at signaling. These fibers also linger for a longer period of time compared to Aδ fibers, and are not precise in the location of pain. People rub the spot where they got injured to relieve the pain of the injury by replacing it with a greater stimulus.

Many substances can trigger the depolarization of a nerve. Histamine, bradykinin, prostaglandin, and ATP are some of the substances in one's body that can activate the nociceptors. Substance P can cause a release of histamine from mast cells or neutrophils to bind to the nerve endings. The use of CGRP and/or substance P from the nerves to elicit an inflammatory reaction is called neurogenic inflammation.

Lecture 16

After an injury, the body goes through phases of healing. The first phase can be generalized as the inflammatory phase. This is a stage where the injured should not engage in rigorous activity. The tissues in the inflammatory phase do not have structure, and can, therefore, undergo further damage in a stressful environment. The second part of healing is the repair phase. In this phase, the athlete or patient does an optimal amount of load. The load that each person performs is catered to their injury or strength they have. Any amount of load that is above or below the optimal range results in harm to the body. The importance of adding loads to this phase of healing is to maintain muscular and cardiovascular function, and to prevent any deterioration of the joints. The last phase of healing is the remodeling stage. More load is added during this period, and the tissue is more structured than in the previous phases. Most of the collagen III in the inflammatory phase will be replaced with collagen I in the remodeling phase.

Lecture 17

The procedure of restoring the body to its natural state after an injury first starts off with the blood vessels leaking around the site of trauma, and a small period of vasoconstriction. After that stage, the fibrin and fibronectin form a mesh-like structure. Fibrinogen can be soluble or insoluble depending on the location. Together, platelets, fibrin, and fibronectin cause the blood to coagulate and form a clot. The activated platelets release the enzyme thrombin, which turns fibrinogen into fibrin. Before platelets activate, the formation of fibrin starts with prothrombin, which is turned into thrombin if calcium is present. After the enzyme is created, thrombin changes fibrinogen into fibrin. The platelets continue to engage in positive feedback of producing thrombin until the clot is large enough to prevent bleeding.

Platelets also continue to degranulate and cause chemotaxis, which brings neutrophils and other cells to the site.

After clotting is finished, the inflammatory response starts. The area of injury becomes red, the person feels pain, heat is produced from the site of injury, and swelling occurs. The heat produced during the inflammatory phase is caused by the metabolic processes occurring at the place of trauma. Ions and other substances move from the blood and lymph vessels into the surrounding tissue due to the increased permeability of the vessels. With the ions, water moves out of the blood into the surrounding tissues. This causes swelling around and at the injured site.

Substances like complement proteins, kinins, prostaglandins, and others cause the blood and lymph vessels to leak. Allowing large substances and molecules to pass through these vessels enable the body to repair and to stop bleeding loss.

The last part of recovery for an injured area is the fibroblast synthesizing collagen to rebuild any damaged tissue.

Lecture 18

When the body is undergoing repair, the first cells to come in are the neutrophils. These cells arrive at the site of trauma and release reactive oxygen species to sterilize the injury. The neutrophils also break down any tissues with protease including damaged tissues. After the neutrophils complete their job, macrophages phagocytose the dead neutrophils. Once the macrophages clean the area of any dead cells, the macrophages secrete growth factors. From there, the fibroblasts start to reconstruct any tissue that was damaged. Fibroblasts create collagen and stick to the fibrin part of the fibrin and fibronectin mesh structure. The collagen created by the fibroblasts is temporary during the first part of reconstruction. Labeled type III collagen, the formation of its fibers is non-linear and lacks structural stability. This means that it can easily tear. Collagen III is eventually replaced with collagen I, which has a sturdier structure and a linear formation. Through extracellular and intracellular digestion, type III collagen is replaced with type I.

The best type of inflammatory response is brief with the appropriate amount of applied mechanical healing. The response usually lasts around 72 hours. In order for a tissue to heal, the presence of cells, blood, mechanical stress, and tissue touching are needed.

Lecture 19

Metabolism regulates blood flow. When the body flexes or contracts a muscle, metabolism occurs. Just after a muscle contracts, the arterioles vasodilate, which increases the blood flow to the muscle or motor units participating in the metabolic action. Capillaries contain no smooth muscle. This makes it impossible for them to vasodilate or vasoconstrict. Therefore, the constriction or dilation of the arterioles determines the flow of blood.

Biologically, a motor unit does not have all its innervated muscle fibers in the same location. Instead, the muscle fibers are spread throughout the muscle just like capillaries are. When a single motor unit is recruited to contract a muscle, various arterioles vasodilate to provide blood to the engaged muscle fibers in the motor unit. Capillaries that supply blood to a single muscle fiber or a single motor unit may be connected to different arterioles. This causes multiple arterioles to vasodilate.

Capillaries distribute blood to muscle fibers, tissues, and other organs, and they can also relay information about to help assist the blood flow. They can detect pH changes, lactate build-up, and any other chemical presence in the body.

Lecture 20

The transition from acute pain to chronic pain has some responsibility attributed to PKA signaling in the nerves, CGRP, and possibly prostaglandins. Substance P is related to the transition from acute pain to chronic pain in the body. It causes neurogenic inflammation, vasodilation, and increases permeability of the blood and lymph vessels. Substance P also assists in immune responses by directing monocytes to the injured area. This process is called chemotaxis, which is the movement of cells to a chemical signal in the body. Re-injury of an injured part of the body can cause acute pain to become chronic if there is no time for recovery of the tissue.

Lymphatic and blood circulation disruption can happen anywhere in the body. When the fibronectin and fibrin create a mesh structure to prevent blood loss, platelets adhere to the fibrin and fibronectin net via von Willebrand factor. Without the adherence of platelets and the inflammatory response like neutrophils and macrophages, recovery of tissue would not be possible.

Lecture 21

The hypothalamus, located in the brain, controls most of the neuroendocrine system; however, there are other autocratic organs and glands in the body that control the distribution of certain hormones. A part of the body, the adrenal medulla, releases neurotransmitters, too.

An example of a self-governing organ is the pancreas. The pancreas distributes insulin and glucagon throughout the body to regulate the glucose content in the blood. Insulin is an anabolic polypeptide hormone that starts a signaling cascade in the cell when it binds to a receptor located on the cell membrane. Glucagon is a catabolic polypeptide hormone that also binds to the receptors on the cell membrane.

Through the relationship of the hypothalamus and pituitary gland in the brain, the body is able to maintain homeostasis. The hypothalamus releases hormones that target the pituitary gland, and stimulates the pituitary gland to release a certain hormone. The hormones released from the pituitary gland target specific cells and receptors, which allow the body to function accordingly.

Lecture 22

Polypeptide hormones cause a signaling cascade to activate in the cell when the polypeptide hormones bind to the receptors on the cell membrane. This phenomenon is the second messenger system. Second messenger systems work can be understood using the example of glucagon binding to its receptor.

As the polypeptide, glucagon, attaches to the protein receptor on the cell membrane, the g-protein undergoes a conformational change. Adenylate cyclase is then activated and converts ATP to cyclic AMP (cAMP). The cAMP is the second messenger ergo the name second messenger system. Afterwards, four cAMP adhere to protein kinase A to activate protein kinase A (PKA). PKA then phosphorylates phosphorylase kinase, which in turn activates phosphorylase. The phosphorylase cleaves off parts of the glycogen chain to produce more sugar in the bloodstream and maintain homeostasis. This process is called glycogenolysis. The cell also turns off glycogen synthase while glycogen is being broken down to glucose. Depending on the location and polypeptide hormone, different substances will be catabolized.

Insulin, growth hormone, catecholamines, cortisol, and a few others regulate lipolysis. The presence of insulin can inhibit the catabolic reaction of PKA in the cell by using phosphodiesterase to decrease the amount of cAMP available in the cell. Phosphodiesterase, PDE3, converts cAMP to AMP in the cell. AMP does not bind to PKA, which prevents PKA signaling.

If a copious amount of insulin is present in the blood, insulin sensitivity is low.

Lecture 23

Lifestyle choices impact some of the hormones present in the body. The concentration of androgens, which are steroid hormones made from cholesterol, is influenced by a person's diet. For example, a diet with low fats and high protein reduces the amount of androgens that exist in the body. A decrease in the intake of fats results in the decrease of cholesterol in the body. Since cholesterol is needed to make androgens, a lack of cholesterol leads to a lack of androgens. If a person decides to eat a low fat and high fiber diet, there might be a decrease of androgen concentration in the body, which will never return back to the original concentration pre-diet. Since testosterone is an androgen, the decrease of cholesterol also impacts the presence of testosterone.

Sleep and exercise also affect hormone concentration. Growth hormone is affected by both of these factors. Women, after vigorous exercise, produce more growth hormone; however, robust exercise does not incite an increase of growth hormone in men. Sleep also increases the concentration of growth hormone in the body. A lack of sleep results in a decrease of growth hormone in the body.

Lecture 24

Ergogenic aids are any substance or material that enhances an athlete's performance.There are three types of aids. One is mechanical aids. Mechanical aids are materials like running shoes and glasses or contacts. Another ergogenic aid is psychological aid. Cheering, listening to music, and positive self-talk are all examples of psychological aid. The last type is physiological aid. This can be substances like caffeine, creatine and water. Another example of a physiological aid are steroids.

Androgens are a steroid, and they can be exogenous or endogenous. Some examples of an endogenous steroid are testosterone and DHEA. Stanozolol is an exogenous androgen.

There are three types of performance enhancing drugs. What category the drug is placed in determines if an athlete is allowed to use that substance. If the ergogenic aid is totally regulated, then the athlete is free to use the drug, and is able to get it without a prescription. A drug that is conditionally regulated needs a prescription from a health professional or doctor. Objects like an inhaler are conditionally regulated. A schedule III controlled substance is a drug that is illegal, and can lead to arrest if in one's possession.

Lecture 25

The media on steroids dictates the opinions people develop. Most of the population has negative opinions about the usage of steroids without understanding how they work. The rebuttal to the argument of steroids negatively affecting an athlete's health brought up some key points many people avoid bringing up in a conversation. For example, tylenol has a higher chance of causing liver failure than a steroid dosage. A sedentary lifestyle has the potential to cause more damage to the body than taking steroids.

Even though we have medical journals and articles that can easily be accessed, the general public has not developed the skill of reading medical literature. When an article that claims oral administration of a steroid has negative effects surfaces, the untrained reader might misinterpret all steroids in all forms as harmful. Instead, the reader should nitpick the fallacies of the article or the study that is described.

Although the oral administration of steroids has a negative effect, injecting the steroids into the body has no adverse effects. Through oral administration, the steroids consumed go to the liver where it can cause a decrease in cholesterol in the body. By injecting the steroids, the liver is bypassed.

Lecture 26

There are seven arguments against the use of steroids. The first argument states that steroid usage affects the athlete's health. The second claims that steroids create an unnecessary risk for harm, and may be coercive. However, the athlete has the choice to take or not take the drug. No one is physically being forced to take a drug. Another argument is that steroids are unfair. The person stating this claim fails to realize that life is not an even playing field. Genetic lottery is a huge example as to why the concept of even playing field does not exist.

Steroids undermine the soul of the sport is another assertion; however, this claim does not hold water because sports are constantly being modified to keep the audience captivated. Also, warriors or gladiators used whatever concoctions or means necessary to win.

Natural versus unnatural as a counter to steroid usage fails to address any problems with steroids. Steroids, like testosterone, that are injected into the body are naturally made from the endocrine system.

Rules is another claim people use countering steroids; however, rules are socially influenced, and not definite. For example, in a certain country chewing gum is illegal, but here no one has any qualms about it. Interacial marriage used to be banned in the U.S., but now it is accepted.

There are a few more common claims that have been made, but the root of why steroid use does not sit well with people lies in the belief that people are getting something for free. Steroids do not enhance the ability of the user without the user putting in the effort to use it properly. What I mean is that an athlete or user has to work out, and continue their routine to reap any benefits. If the drug is taken without the user putting in any effort, the drug holds no effect.

Lecture 27

Enzymes are catalysts that speed up the reactions.They work by lowering the activation energy of the products causing a quick reaction to occur. Enzyme binding sites are specific, and can only bind with specific substrates.

Inhibition happens when an inhibitor, a compound similar to the shape of an enzyme, binds to the enzyme. This causes the enzymatic reaction to be negated. There are different types of inhibition. Allosteric inhibition is when an inhibitor binds to a site on the enzyme and alters the substrate binding site. This prevents the substrate from attaching to the enzyme, which nulls the reaction. Competitive inhibition occurs when the inhibitor and the substrate have the same structure, and can bind on the same binding site. Whether the reaction is inhibited or not depends on whether the inhibitor of the substrate attaches to the binding site. When an inhibitor has its own binding site for inhibition, it is called noncompetitive inhibition. Uncompetitive inhibition requires the binding of the substrate and the enzyme, which allows the inhibitor to attach to the enzyme. The phenomenon when an inhibitor binds permanently to the enzyme is called suicide inhibition.

Accumulation of product causes negative feedback when there is too much of a substance produced. The excess product inhibits the enzyme that helps create it.

The phosphorylation of an enzyme can activate or deactivate it. Phosphorylation of an enzyme can happen at numerous sites. An example of phosphorylation of an enzyme is glycogen synthase. When this enzyme is phosphorylated, the enzyme turns off.

Lecture 28

The signaling cascade that happens when insulin binds to a receptor prevents PKA being activated. As insulin binds to a receptor, PI3K becomes activated. PI3K then stimulates the PKB, which

phosphorylates PDE. PDE is an enzyme that converts cAMP into AMP. Cyclic AMP concentration decreases in the cell, which inhibits PKA, an enzyme that needs cAMP to break down molecules.

CAMP is produced when epinephrine or glucagon binds to its cell receptor. In the case of epinephrine, the molecule binds to a β- adrenergic receptor. This receptor activates adenylate cyclase, which creates cAMP. PKA binds to cAMP, activates HSL, and phosphorylated perilipin. Note that anabolism always trumps catabolism.

MTOR is a kinase or enzyme that regulates protein turnover in the body. In order for mTOR to be stimulated, an anabolic molecule must bind to its receptor on the cell wall. Akt, which is another name for PKB, deactivates tuberine, a protein complex that inhibits mTOR. MTOR phosphorylates P7056K, which turns on, and 4EBP1, which turns off after being phosphorylated. P7056K promotes protein synthesis when activated, and 4EBP1 breaks down proteins when mTOR is inhibited.

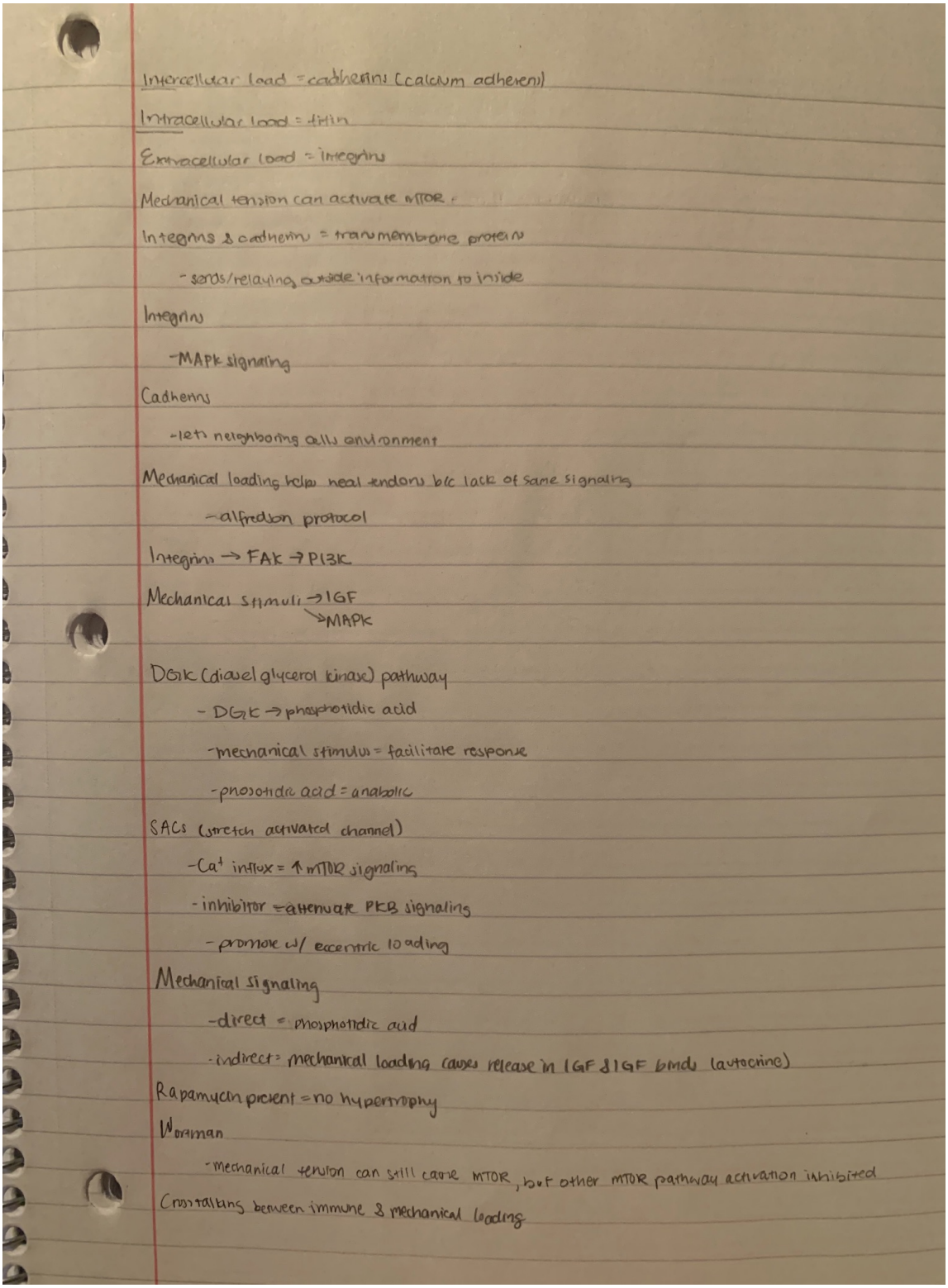
Lecture 29

MTOR is present anywhere in the body. It is a regulator of muscle metabolism. There are two types of mTOR complexes, type I and II. Complex I controls the growth of muscle and can be found downstream to complex II. The mTOR complex II can activate the PKB in complex I, but mTOR needs more sources to activate PKB than just complex I.

MTOR complex I involves three main proteins. These proteins are mTOR, raptor, and MLST8. While rapamycin inhibits mTOR to activate, raptor assists mTOR binding. Raptor is an abundant protein in skeletal muscles, and it recruits substrates for mTOR to phosphorylate. Depending on the site where raptor is phosphorylated, raptor can be inhibited or promoted. If raptor is phosphorylated at the MAPK site, then raptor is activated. At the AMPK site, raptor is inhibited.

Rheb turns on the mTOR when it is bound to GTP, and aids in raptor phosphorylation. Tuberine, which is activated by AMPK, inhibits activation of mTOR by hydrolyzing the GTP bound to Rheb. However, RSK prevents tuberine from inhibiting mTOR, and can phosphorylate many other proteins involved in the anabolism and catabolism of the body. RSK also stops BAD, which is an agonist of cell death.

Lecture 30



Intercellular load = cadherins (calcium adherens)

Intracellular load = titin

Extracellular load = integrins

Mechanical tension can activate mTOR

Integrins & cadherins = transmembrane proteins

- sends/relaying outside information to inside

Integrins

- MAPk signaling

Cadherins

- let's neighboring cells environment

Mechanical loading helps heal tendons b/c lack of same signaling

- alfredson protocol

Integrins → FAK → PI3K

Mechanical stimuli → IGF
→ MAPK

DGK (diacel glycerol kinase) pathway

- DGK → phosphotidic acid

- mechanical stimulus = facilitate response

- phosotidic acid = anabolic

SACs (stretch activated channel)

- Ca^+ influx = ↑ mTOR signaling

- inhibitor = attenuate PKB signaling

- promote w/ eccentric loading

Mechanical signaling

- direct = phosphotidic acid

- indirect = mechanical loading causes release in IGF & IGF bmds (autocrine)

Rapamycin present = no hypertrophy

Norman

- mechanical tension can still cause mTOR, but other mTOR pathway activation inhibited

Crosstalking between immune & mechanical loading

- part of regulation of tissue damage
- IL2 → PI3K
  - FOXO shut off
  - BAD & glycogen synthase kinase turned off ← inhibited = glycogen synthesized

Interferon γ (gamma)
- endogenous
- required for efficient skeletal muscle regeneration
- cytokine

Myostatin
- myokine (cytokine)
- catabolic
- if bonded to follistatin = inhibited
- inhibition of PKB

TNF-α
- anabolic

Wnt
- shuts down glycogen synthase kinase 3
  - ↳ GSK3 = promotes tuberine

Reactive oxygen species = acute fatigue
- antioxidants suppress benefits of working out
  - -insulin sensitive
  - -potential mTOR signaling

Mechanical Tension
- mechano receptors
- mechanotransduction
- ideal for fitness
- integrin = transmembrane protein
  - relay information from the extracellular matrix to inside the cell
- titin is also part of mechanotransduction

Focal adhesion kinase
- point of relay between integrin & interior of the cell

Cadherin & catenin in cell junction

most take PI3K pathway

Video 30

Anaerobic training not as destructive to aerobic training

- destructive to specificity of adaptation

Aerobic training

- more destructive to anabolism compared to anaerobic trains to boost of aerobic

MTOR tightly regulated to preserve fitness (in tune w/ environment)

- specificity of adaptation = alignment w/ environment
- critical regulated hub
- tons of extracellular events
  - cytokines
  - loads
  - Growth factors
  - insulin / IGF

10~20% of phospholipid bilayer = aracodonic acid

- 20 carbon fatty acid
- polyunsaturated
- abundant in skeletal muscle

PLA II release aracodonic acid

to cyclooxygenase converts it to prostoglandin

- aspirin = irreversible inhibitor / suicide inhibitor for cyclo oxygenase
- upregulate another pathway if blocked
  - asthmatic inflammation from leukotrines

MAPK

- prostoglandins activate

Gastrointestinal problems if COX1 blocked

- COX1 present in gastrointestinal tissue

Prostoglandins have own receptor

- activate MAPK signal cascade

RAS-GDP → Ras-GTP → activates raf (kinase) → activates MEK (mitogen activated protein kinase, kinase) → ERK/MAPK → RSK

Raptor major hub for crosstalking

Interleukin-15

- cytokine
- abundantly released during tissue damage

Lecture 31

Estrogen binding to an estrogen receptor inside the cell starts a signaling cascade. The non-genomic effect of estrogen causes tuberine to be inhibited. Estrogen causes hypertrophy; however, it also stimulates LKB1. The steroid hormone also promotes Rheb.

LKB1 activates AMPK in the cell. AMPK is also stimulated by exercise AMP. Testosterone inhibits LKB1, which inhibits the proliferation of cells.

Both estrogen and testosterone result in an increase of calcium mobilization. Testosterone has two pathways that lead to the activation of mTOR. These pathways are PI3K and MAPK signaling.

For mTOR to function, it must be sent to a lysosome. The lysosome is a storage site for amino acids, and nutrition helps us move mTOR to the lysosome. In order for mTOR to be recruited, the lysosome has to have an adequate amount of amino acids present. Rag GTPase binds to the raptor protein on the mTOR complex, and transports mTOR to the lysosome for protein synthesis.

Lecture 32

A kinase called AMPK regulates the energy of the muscle, inhibits raptor, and activates tuberin. Both raptor and tuberin are phosphorylated by AMPK, which turn on or off the protein. LKB1 activates AMPK by binding to its alpha subunit. However, in order for LKB1 to bind to the alpha subunit on AMPK, AMP needs to attach to the gamma subunit. Exercise also activates AMPK, and helps create AMP by breaking down ATP for energy. AMP binding to the gamma unit promotes the binding of a second one. When two AMPs are bound to AMPK, LKB1 can bind to the alpha subunit.

When ATP is used, the products are ADP, phosphate, and hydrogen atoms. In order to provide AMP to the AMPK pathway, adenylate kinase phosphorylates two ADP to create ATP and AMP. The ATP is a competitive inhibitor to the AMP AMPK enzyme-substrate complex. The ratio of ATP to AMP determines whether AMPK will activate or not.

Present glycogen binds to the beta subunit of AMPK. When glycogen attaches to AMPK, GLUT 4 is transported into the cell. This complex also assists in glycolysis by promoting hexokinase and PFK. However, chronic AMPK activation may cause glucose synthase because of the excess amount of G6P.

Lecture 33

Insulin is one of the hormones that activate mTOR through the P13K pathway. Insulin dependent glucose pathway uses this process, which has an anabolic outcome. Non independent glucose uptake involves exercise, and stimulates AMPK. The pathway to activating AMPK involves breaking down ATP for energy, and using the AMP that is created from the phosphorylation of two ADP. Both mTOR and AMPK pathways mobilize the GLUT 4, but mTOR promotes glucose synthesis, and AMPK inhibits glucose synthesis.

AMPK might have some influence in enhancing healing during the early stages, but, in the terminal stages, AMPK has little to no activation to support the repair and growth of tissue. Metformin, a drug, inhibits AMP deaminase. If AMP deaminase is activated, then AMPK is inhibited.

Eccentric loading activates MAPK. Isometric and concentric loading also activate MAPK, but eccentric loading stimulates more MAPK than isometric and concentric.

Low loads and high duration does not cause mTOR to be activated, but high loads and high duration does. High loads and low duration can work, but not as well as the former.

It is important to know that human bodies are unable to excel in all aspects of athleticism. If someone is talented in marathon running or endurance, it is hard for them to show the same level of professionalism in weight lifting.

Lecture 34

Supplements, if used correctly, can enhance mTOR activation. In order to properly use supplements to assist in anabolic reactions, one must understand what that supplement targets. A signaling cascade inside the cell stimulates protein synthesis. Depending on the supplement, different enzymes or hormones are targeted to enable protein synthesis.

HMB is a supplement derived from leucine. Although it is a derivative of leucine, it is different in a few ways. HMB is not as effective in triggering protein stimulation compared to leucine, and creates a more anti catabolic effect. In a study where HMB was given to bedridden patients, lean body mass was preserved in those who took the supplement.

Another supplement is phosphatidic acid. This supplement is taken orally, and gives the same stimulation as mechanical loading. Phosphatidic acid directly activates mTOR, which results in muscle or tissue growth. Mechanical loading uses an indirect pathway, but still has the same effect as phosphatidic acid.

There are many more supplements that promote hypertrophy. The former examples are just a few.

Lecture 35

Dopamine is a neurotransmitter that increases performance if the proportion of dopamine to serotonin is high. Serotonin causes lethargy, and can mostly be found in the gastrointestinal tract. Tryptophan is an amino acid that assists in the production of serotonin when it crosses the blood brain barrier. However, for tryptophan to cross the blood brain barrier, the concentration of amino acids that are competing against tryptophan needs to be lowered.

There are two different categories of amino acids that are transported across the blood brain barrier. The two groups are BCAA, branch chain amino acids, and AAA, aromatic amino acids. Tryptophan is an AAA, and competes with the other amino acids to be carried across the barrier. Exercise decreases the amino acids, BCAA, competing with tryptophan and promotes the increase of free tryptophan in the bloodstream. When tryptophan is not attached to albumin, tryptophan can cross the blood brain barrier. During exercise, lipolysis occurs, which increases the concentration of free fatty acids in the bloodstream. The free fatty acids have a higher affinity for the albumin, which causes the tryptophan that is loosely bound to the albumin to become free. The increase of free tryptophan creates less competition between the amino acid and the BCAA. Serotonin can then be created and released, which makes the body feel enervated.

Lecture 36

Nerves do not take breaks

- ganzfeld procedure
- constantly seeking internal & external signals

Involuntary & Voluntary responses to environment

↓ automatic ↓ conscious

H^+ ions / protons

- depolarize nerves
- pH

Hurt = charge behavior

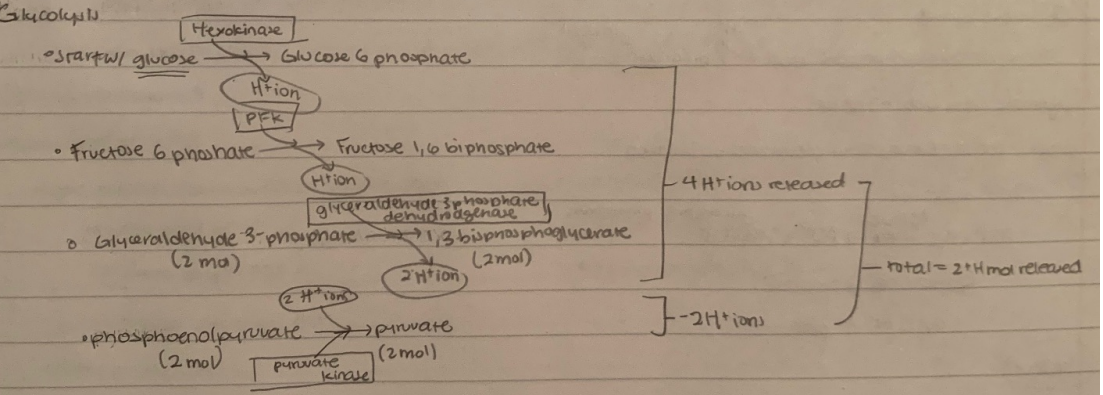
Rhabdomyolysis = muscle fiber lyse & release contents into bloodstream → kidney

- hurts, but not burning sensation

Burning

- pH changes & nerve senses it = burning
- anaerobic metabolism & lactic acid blamed, but not cause

Glycolysis



If you begin w/ glucose & end w/ lactate = net = 0 H^+ ions

↳ no change in pH

If you start w/ glycogen = skip hexosenase

↳ net change of -1 H^+ ions

↳ become more basic

We inhibit glucose consumption during exercise

glycogen consumed instead

↳ pH = basic during exercise

- pyruvate goes into krebs cycle or anaerobic/lactate
  - Daemon = acytle CoA
    - pyruvate $\xrightarrow[\text{pyruvate dehydrogenase complex } (\varepsilon_1+\varepsilon_2+\varepsilon_3)]{}$ Acetyl-CoA + CO_2
      - 1st enzyme (ε_1) = CO_2
      - 2nd enzyme (ε_2) = Acetyl-CoA
      - 3rd enzyme (ε_3) = H^+ ion

Krebs cycle

- $2CO_2$ made
- If no oxaloacetate + Acetyl-CoA bind = Keto
- at end
  - per acetyl CoA
    - $3CO_2$ + 1 from pyruvate
    - $3H^+$ +1 from pyruvate

CO_2 in the blood

carbon dioxide $\leftrightarrow$ carbonic acid $\leftrightarrow$ bicarbonate (H^+, in blood)

1 pyruvate from krebs = $7H^+$ ions

What is the largest source of metabolic byproducts?

- ATP hydrolysis
- $ATP + H_2O \xrightarrow{ATPase} ADP + HPO + H^+$ (HPO: inorganic phosphate)
  - burning sensation
    - bulk of H^+ production

Fatigue

- Isometric fatigue ≠ jogging fatigue
- RM fatigue differs
- major cause of fatigue
  - ATP : ADP ratio
  - pH (H^+)
  - phosphate
  - increase of ADP, phosphate, H^+ = fatigue
- more cause of fatigue
  1. # Ca^+ released from sarcoplasmic reticulum
  2. how sensitive muscle is to released Ca to cause cross bridge

③ how much force cross-bridge generate
- length tension relationship
- impair relationship
- delay reaction

④ Central fatigue
- brain says no b/c of signals
- cellular mechanisms

Intense exercise ↑ rate of energy > ATP can be resynthesized
- glycolysis slow
- creatine fast
- oxidative slow

Size principle
- energy efficient muscles recruited 1st, and energy inefficient recruited last
  - ATP depletion found @ high threshold motor units
  - not going to see huge ATP depletion
- If you isolate type II muscle = more ATP used compared to whole

ADP concentrations ↑

Consequence of ↓ ATP
- rigor mortis / cross bridge cycling stops
  - ↳ weak bond = ATP
  - ↳ strong bond = ADP
  - (power stroke)
- sarcoplasmic reticulum can be leaky & Ca pump impaired w/ too much ADP
- ADP outcompetes ATP for RyR
- just one fiber uses much ATP
- ATPase use ATP
  - myosin ATPase
  - sarcoplasmic reticulum Ca-ATPase
    - ↳ Ca^{+} pump back in
  - Na^+/K^+ pumps

ADP / ATP competition for releasing Ca^{2} into cytosol

AMP deaminase to maintain ratio
- IMP & NH_3 created
  - becomes slightly basic & picks up H^+ ions accumulated
- no energy released
- causes central fatigue b/c ammonia can cross blood brain barrier
  - ↳ more intense = ↑ NH_3 crossing
- carbs attenuate NH_3 crossing blood brain barrier

Lecture 37

ATPases

-bulk of ATP consumption = myosin ATPase

↳ cross bridge cycling

↳ H^+ ions created in big amounts

H^+

at rest: • blood = 7.4 • muscle = 7.0

not at rest: blood = 7.1, muscle = 6.8 → accumulate H^+ ions = ↓ pH

Intramuscular → blood for exercise

blood → Intramuscular for central nervous system maybe

-exercise intensity

protons detected by nociceptors = burn

pH might contribute to muscle fatigue

↑ proton concentration

- sarcoplasmic reticulum/calcium kinetics effected
  - ↳ Ca^{++} leave Sarcoplasmic reticulum = H^+ ions enter
    - under abnormal condition = H^+ ions staying in SR after Ca^+ ions pumped back in
  - ↳ Ca^{++} back into SR can slow depending on pH
  - ↳ release of Ca^{++} affected w/ pH
- effect on cross-bridging
- possibly help electroactivation of cells
  - ↳ activation of muscle = lose K^+
    - more - = refractory period ↑
    - need large excitatory stimulus
  - ↳ intracellular ↑ H^+, K^+ ↓, tubules less permeable to Cl^-
    - charge doesn't decrease b/c (-) charge not leaking in
    - refractory period not severe
- Baking soda affect
- cause of fatigue

Maybe something

earliest cause fatigue = Type II fibers & CNS & anaerobic ← crossbridge impairment

Late fatigue CNS (Tryptophan)

↑ phosphate concentration ← weak bond w/ ADP

- no release of phosphate on myosin = no muscle contraction
- ATP ↓ = P_i binds to Ca^{++} = ↓ free Ca^{++} to cause contraction

- total # Ca^{++} in SR high
- most Ca^{++} bound to calsequestrin (binding protein)
  - 1 mol = 50 calcium units
  - low affinity
- Ca^{++} & Pi high affinity = hard to release Ca^{++}
- caffeine enhances Ca^{++} release, might be countered by Pi bound

H^+ ion = burning.
- maybe fatigue centrally

HPO = no burning
- def. fatigue

ADP = maybe burning
- def. fatigue

General timing of muscle fatigue

Early = cross bridge impairment

Late = Sarcoplasmic reticulum Ca^{++} release

Mg
- ATP complexed w/ Mg^{2+}
- free Mg^{2+} ↑ w/ ↓ ATP
  - ADP, AMP, IMP ↓ affinity w/ Mg^{2+}
- compromise release of Ca^{2+}
  - compete against Ca^{2+} binding sites in Ca^{2+} induced Ca^{2+} released when Mg^{2+} free
  - ryanodine receptor

ROS
- antioxidants inhibit
- fatigue => can't handle work load
- superoxide & nitric oxide → peroxide
  - bad
- H_2O_2 might impair depolarized-induced Ca^{++} release
- SR might be affected
- Ca^{++} sensitivity might be affected

(binder of short)

pH found to cause fatigue by proton buffers & test performance

ROS found to produce fatigue
- ROS scavenging compounds = ↑ performance
- fc, & no effect

Inflammation
- induce some fatigue

- skeletal muscle release stuff
- probably CNS fatigue

Heat cause of fatigue

- enzymes function @ certain C°
- blood distribution
- hemoglobin O_2 drop off @ hot places
- higher temp = ↑ ROS
- electrolyte balance across sarcolemma
- cardiac drift (SV↓, ↑HR) = heart works harder
- ↑ RPE, ↓ central drive

Carbs

- glucose & fructose different pathways
- fructose retained in liver
- fructose can cause fatty acid synthesis