Healthcare Education in the Community

Brady Ulrich

Bachelor of Integrated Studies: Capstone Project

Weber State University

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I. Background

Many times I have reflected on the experiences I have had, and often pondered how I can use them to benefit others. The BIS program here at Weber State University has provided that path for me. The Capstone Project has helped me find a way that I can use the unique experiences to help improve the quality of life in any community. I had the opportunity to participate in a service mission in Japan on the island of Kyushu for two years. The increased learning and experiences I had while in Japan have fueled my desire for this project. In Japan, I made five trips to the hospital for myself, and many more for other accidents such as, bike crashes, sickness, and appendectomies with other missionaries. During these trips to the hospital there was one aspect that was the same with each visit; somehow I had to explain the situation in a second language, Japanese.

The language barrier was difficult to overcome. We made do with our pocket dictionaries and hand gestures, but had we had access to a reference pamphlet including translations, as well as explanations of situations that were occurring, it would have been a huge help. Time could have been saved, we would have felt more comfortable knowing that the doctor completely understood the problem, and the magnitude of cultural and language differences would have been minimized.

I have taken what I've learned from these situations and devised a project that will be a benefit to the English, and also the Japanese community. Near my home in Utah there is a high population of Japanese people in the area, festivals tailoring to the Japanese are becoming more prevalent each year, and travel to and from Japan is becoming more common. By making this pamphlet widely available individuals will not only be informed of three of the most common ailments people experience, but also have the reference when they need it.

II. Introduction to the Project

When you visit a doctor, hospital, or even pharmacy you may notice a pamphlet informing you on a certain condition. Similar to these pamphlets, in the pamphlet I have put together, three extremely common ailments are addressed: myocardial infarction (heart attack), traveler's diarrhea, and low back pain. Included in the pamphlet will be the detailed definition for each ailment, their signs/symptoms of the condition, and how to prevent or avoid these conditions.

Each year about 715,000 people have a heart attack in the United States. Of these, 525,000 are a first heart attack and 190,000 happen to people who have previously suffered from a heart attack.[1] Traveler's Diarrhea (TD) is the most common illness in persons traveling from resource-rich to resource-poor regions of the world.[2] The fear of developing diarrhea while traveling is common among travelers to any part of the world. This concern is realistic; 40%-60% of travelers may develop diarrhea.[3] Back pain is the second most common symptom-related reason for clinician visits in the United States. Up to 84% of adults have low back pain at some time in their lives.[4]

The need for the community to be aware of these ailments is obvious. Up until the time I began to experience lower back pain and travelers' diarrhea in Japan, I was not properly informed on how to handle these situations. A myocardial infarction is commonly talked about, though the signs and ways to prevent a heart attack are not widely known. The pamphlet will not only help at a time in need, but also is available to help inform people on how to avoid or prevent these conditions in the future.

III. Research

Myocardial Infarction and contributing factors

What is a myocardial infarction?

A myocardial infarction (MI), or heart attack, occurs when the blood flow to the heart is severely reduced or halted.[5] This happens because coronary arteries that supply the heart with blood slowly become thicker and harder/less elastic from a buildup of cholesterol and other substances, altogether called plaque.[5] If the plaque breaks open and a blood clot forms blocking the blood flow, the heart muscle is starved for oxygen and a heart attack occurs.[5]

What is cholesterol?

Cholesterol is one of many substances created and used by our bodies to help keep us healthy. Some cholesterol is produced naturally, and some comes from the food we eat. The two main components of cholesterol are high density lipoprotein (HDL) or "good" and low-density lipoprotein (LDL) or "bad".[6] It's important to note that the actual cholesterol in HDL and LDL is the same, it's the amount of cholesterol carried in each lipoprotein that is the difference.[6] Too much of one type, or not enough of another can increase the risk of a heart attack. Although the exact nature of the protective effect of HDL levels is unknown, high levels of HDL can help reduce the risk of heart disease.[6] For a healthy person the HDL/LDL ratio is 3.5.[6] The effects of HDL and LDL will be discussed in more detail later on in this section.

The lipoproteins that "house" cholesterol molecules are particles that function in cholesterol homeostasis, transporting the molecule from sites of synthesis to sites of use, and finally to the liver for excretion.[6] Lipoproteins contain hydrophobic (water repelling) lipids (triglycerides, cholesteryl ester, and cholesterol) stabilized by a surface film of polar (electrons

are shared, but not equally) phospholipids (membrane), cholesterol, and proteins (integrated in the membrane, Figure 1).[6] Traditionally, lipoproteins are classified on the basis of their densities of cholesterol. Also, cholesterol inserts into the phospholipid membrane of each lipoprotein by forming a hydrogen bond between the carbonyl (carbon and oxygen share a double bond) oxygen atom of the polar head, and the hydroxyl group of the cholesterol.[6] The hydrocarbon tail of cholesterol is then in the nonpolar core of the membrane (Figure 2,3).

HDL or "good cholesterol" is a lipoprotein with very few cholesterol molecules and functions as a shuttle that moves cholesterol throughout the body. It binds and esterifies cholesterol released from macrophages and the peripheral tissues and then transfers cholesteryl esters (compound of cholesterol and a fatty acid) to tissues that use cholesterol to synthesize steroid hormones, or to the liver where the cholesterol is converted into bile salts or excreted.[6] This process is called reverse cholesterol transport (RCT), and will be the only HDL function discussed.

The effect of HDL on RCT is likely the most important mechanism by which HDL reduces the risk of a heart attack. The steps involved in HDL metabolism begin when its primary structural protein, apolipoprotein A1 (ApoA1), is secreted by the liver into the blood.[7] It is then lipidated with phospholipids transported via ATP-binding cassette (ABCA1) in the intestines, which is essential for the stability of ApoA1.[7] Now the HDL particle is an acceptor of cellular cholesterol, and will return to the liver to be broken down and excreted.[7] This process enhances cholesterol removal and decreases the cellular cholesterol levels.[7] However, failure to effectively lipidate ApoA1 or HDL renders them ineffective in cholesterol transport and targets them for degradation in the kidneys.[8]

Therefore, HDL metabolism occurs by coordinated action of multiple tissues, while the heart attack preventing effects of HDL are due to its ability to transport cholesterol from macrophage cells to the liver where it can be broken down and excreted. Without HDL transporting cholesterol from macrophage cells, buildup and blocking of arteries would occur rapidly, and much more often. One critical aspect is that HDL routinely measured as a "snap-shot" of the amount of cholesterol associated with HDL particles and does not report the ability of HDL to facilitate RCT.[7] Direct measurement of the ability of HDL to facilitate cholesterol from macrophage cells is the only way to determine HDL functionality with respect to its role in RCT.[7]

LDL or "bad cholesterol" is a major carrier of cholesterol in blood and has a core of around 1500 cholesterol molecules esterified to fatty acids.[6] LDL differs from HDL in that instead of picking up cholesterol released from dying cells into the plasma, it transports cholesterol to peripheral tissues and regulates cholesterol synthesis. Excess LDL in the body will result in buildup of atherosclerotic plaques in arteries, and increase the risk of a heart attack.[6]

The liver is the primary site of cholesterol synthesis, and is a major site for control of cholesterol metabolism. There are many non-hepatic cells outside the liver and intestine that obtain cholesterol from the plasma rather than synthesizing it. More specifically, their primary source of cholesterol is LDL by a process of LDL–uptake called receptor–mediated endocytosis[6] (Figure 4).

Endocytosis is the process by which surface membranes pouch inward and pinch off to form vesicles.[9] As visualized in figure 4, receptor-mediated endocytosis begins when apoliprotein B-100 on the surface of an LDL particle binds to a specific receptor protein on the plasma membrane of non-hepatic cells.[10] The LDL receptors are localized in specialized regions

which contain a specialized protein. The receptor-LDL complex is then internalized by endocytosis to form a vesicle called an endosome.[9,10,11] The endosome is acidified which causes the receptor to release its cargo. The vesicle is then returned to the cell membrane in a recycling vesicle.[10] The protein component of LDL is then hydrolyzed to free amino acids; the cholesteryl esters in LDL are hydrolyzed by a lysosomal acid lipase (enzyme to break down the molecule).[9,10] The unesterified cholesterol that is released can be used for membrane biosynthesis or re-esterified for storage inside the cell.[10]

The LDL receptor plays a critical role in LDL concentration regulation. Too high of a concentration of LDL in the blood plasma will result in cholesterol deposits in various tissues. One particular concern is oxidation (loss of electrons) of excess blood LDL to form oxidized LDL (oxLDL), which is taken up by macrophages.[10] In an attempt to destroy oxLDL, macrophages surround the oxLDL and in turn become engorged to have a foamy cell appearance. These foam cells become trapped in the walls of blood vessels and contribute to the formation of plaques that cause arterial narrowing and lead to heart attacks.[9]

By understanding this mechanism we can see that the absence or deficiency of LDL receptors is a major contribution to high levels of LDL cholesterol, and also the buildup of plaque in arterial walls. On the other hand, if we have low levels of HDL then we won't be able to clean up the excess LDL that has been picked up by macrophages, and we will have a similar result as the absence or deficiency of LDL receptors. It is an art to be able to find out your own HDL/LDL ratio and effectively raise and lower what you need to. HDL/LDL ratio levels are also coupled with genetics, so it may be helpful to know your parent or grandparent's levels. A history of high LDL levels generally leads to risk of cardiovascular disease.

If you are worried about your own HDL/LDL levels, tests can be done by your physician to determine where your HDL/LDL levels are at. HDL or “good cholesterol”, to put it simply, picks up excess cholesterol (mainly LDL) in the tissues from macrophage foam cells, and delivers it to the liver to be converted into bile salts or to be excreted. LDL or “bad cholesterol” is necessary for the body, but if we produce more than needed it will be taken up by macrophages to form foam cells with the potential to become trapped in the walls of blood vessels, which may lead to a heart attack.

Traveler's Diarrhea

What is traveler's diarrhea?

Travelers' diarrhea (TD) is a digestive tract disorder that commonly causes loose stools and abdominal cramps, and is often caused by eating or drinking contaminated food or water.[12] Fortunately, TD usually isn't serious, just unpleasant. When you visit a place where the climate, social conditions, or sanitary standards and practices are different from yours at home, you have an increased risk of developing TD.

Being careful about what you eat and drink while traveling can reduce your risk of TD, and if you do develop TD, chances are it will resolve without treatment.[12] However, it's a good idea to have doctor-approved medications with you when you travel to high-risk areas in case diarrhea persists.[12]

During the course of this section, the two types of diarrhea mentioned will be secretory and malabsorptive diarrhea. Secretory diarrhea is an increase in active secretion, while malabsorptive diarrhea is the inhibition or inability to absorb nutrients. However, TD is more related to secretory diarrhea. For this reason the mechanism and prevention of secretory diarrhea will be explained at the microscopic level, while malabsorptive will have basic care and prevention methods.

Secretory diarrhea is a dysfunction involving cystic fibrosis transmembrane conductance regulator (CFTR) which is a cyclic-adenosine monophosphate (cAMP) regulated chloride channel located primarily at the luminal surfaces of secretory epithelium (cells that line the inner gut tube, or lumen) where it is normally inactive.[13] The CFTR plays a crucial role in transepithelial fluid homeostasis. cAMP is an important secondary messenger derived from adenosine triphosphate (ATP, with cAMP two phosphate groups have left ATP, Figure 5) and is

used for intracellular signal transduction in many different organisms (Figure 6).[13] Another key component to secretory diarrhea is protein kinase A (PKA) which is an enzyme (helps speed up reactions).[13]

The term phosphorylation will be used which is a chemical reaction where a phosphate group (PO_4^{3-}) is added to a protein. Phosphorylation is important because it can switch receptors "on" and "off" causing them to be activated or deactivated.[14] When a phosphate group is added to a protein it turns a hydrophobic (water repelling) portion, into a hydrophilic (water attracting) portion of the molecule.[14] In this way a conformational change is introduced into the structure of the protein due to interaction with other hydrophobic and hydrophilic residues in the protein, and the "switch is turned on".[15]

When CFTR is over activated in the gastrointestinal tract the result is secretory diarrhea, which is the leading cause of mortality in early childhood.[13] Hyper activation of CFTR commonly occurs when the gut lumen is exposed to various enterotoxins that cause excessive secondary messengers (cAMP) to be generated inside the cell.[14,15] This then unlocks a chain of cell signaling events because excessive cAMP activates the enzyme protein kinase A (PKA).[16,17] This is done by phosphorylating, or adding phosphate groups to the enzyme to turn it on.

Within the structure of PKA, two of the amino acids phosphorylated are serine and threonine[18] (Figure 7). This phosphorylation occurs via a common mechanism seen many times throughout the body, and is directly involved with cell signaling. A protein kinase must recognize between one and a few hundred phosphorylation sites in a background of approximately 700,000 potentially phosphorylatable residues.[19] The responsibility for the recognition of substrates by protein kinases appears to be distributed among a large number of independent, imperfect specificity mechanisms.[19] From this we can derive that there are many

different protein kinases that can be phosphorylated, and for each specific protein kinase structure there are specific features to help recognize substrates.

The cAMP-PKA pathway is one of the most studied and well-known signal pathways, and to maintain the high level of specificity this pathway is tightly regulated.[20] The specific recognizable structures for PKA are called A-kinase-anchoring proteins (AKAPs) which target PKA to specific substrates and distinct compartments providing specificity in the biological effects controlled by the cAMP-PKA pathway.[20] AKAPs also serve as scaffolding proteins that assemble PKA together with signal terminators, as well as components of other signaling pathways into multi-protein signaling complexes.[20]

Once PKA has found the proper phosphorylation site (substrate), the terminal phosphate group of ATP is hydrolyzed (exothermic reaction, releases energy), binding to the hydroxyl group on serine or threonine.[18] Thus, ATP loses one phosphate group and is converted to adenosine diphosphate (ADP), and releases energy or transfers energy to PKA to be used later in the signaling chain.[21]ADP can then absorb energy and regain the phosphate group, regenerating an ATP molecule, thus allowing ATP to store energy like a rechargeable battery.[21]

Now that PKA has been phosphorylated, PKA can in turn phosphorylate the CFTR channel to turn it on (Figure 5,8), and open the channel to chloride ion (Cl-) secretion across the epithelium, increasing the electrical and osmotic driving forces for flows of sodium (Na+) and water.[16,17] The net result is secretion of fluid and electrolytes across the epithelium into the gut lumen, namely secretory diarrhea and the resulting dehydration, which can be fatal if untreated.[16,17] (Figure 7).

Malabsorptive diarrhea however, is the opposite of secretory. Instead of over activation, there is inhibition or inability to absorb nutrients. Nutritional costs of malabsorption may pose a

major threat if diarrhea becomes chronic or recurrent. The majority of the detrimental effects are weight loss and inability to absorb nutrients such as sugars, fats, vitamins, and carbohydrates. However, it is important to note that it is the more extreme and rare cases that this occurs.[22]

Within malabsorptive diarrhea there are a handful of categories that can be broken down such as abdominal distention, intestinal infections, and chronic diarrhea. This can have a large impact on quality of life and overall health. At it's mildest, the condition may be an inconvenience; at it's worst, it may be disabling and even-life threatening, especially during early childhood.[23] If you have three or more bowel movements per day, consistently for three or more weeks you should seek medical attention. There are many different categories or causes of chronic diarrhea and your medical professional will take the necessary steps to help you specifically.

Treatment for travelers' diarrhea

As you travel you may show more signs and symptoms of secretory diarrhea. This can be caused by a variety of bacterial, viral, and parasitic organisms, most often transmitted by food and water which cause the hyper activation of the CFTR complex.[24] The epidemiology of TD does vary from location to location and with the season of the year. One important note is that spices in food and changes in climate **do not** cause TD, although variations in diet, temperature, or even time zones can alter the way a traveler feels and the stresses of travel may exhibit diarrheal symptoms.[25]

Treatment may include three common modes for TD. They are fluid replacement, antibiotics, and antimotility agents.[26] Fluid replacement is essential, while antibiotics and antimotility agents may be required depending upon the circumstances. Most cases are self-limited and resolve on their own within three to five days of fluid replacement only; while

antibiotics will shorten the duration to about one day, and antimotility agents may limit the duration to a period of hours.[26]

Fluid replacement is the primary and most important treatment of TD since the most significant risk is volume depletion.[27] Severe diarrhea should be treated with an oral rehydration solution; this replaces needed electrolytes in the appropriate concentrations.[26] These solutions were developed following the finding that glucose linked sodium absorption remains intact in most diarrheal diseases.[26] Thus, when excessive cAMP is generated by hyper activation of the CFTR complex, the intestine remains able to absorb water if glucose and salt are also present to assist in the transport of water from the intestinal lumen.[28] For mild diarrhea, the use of fluids is the critical factor, though the fluid does not need to be an oral rehydration solution as one study shows no difference between treatments.[29]

Antibiotics are often used to treat moderate to severe diarrhea. Generally while you are traveling you won't need to seek medical advice unless you develop a high fever, abdominal pain, bloody diarrhea, or vomiting.[26] When antibiotics are suggested, one of the most common antibiotics is Quinolones. Quinolones are antimicrobial agents effective in the treatment of acquired infections, and are usually administered orally.[30] Other antibiotics include Azithromycin (for children), Rifaximin, and a more recently known agent is bismuth subsalicylate (BSS, Figure 9).

BSS is a type of bismuth salt commonly found in Pepto-Bismol. The exact mechanism has not yet been determined, but BSS may exert its antidiarrheal action by stimulating absorption of fluid across the intestinal wall.[31] When BSS is hydrolyzed to salicyclic acid, the synthesis of prostaglandin responsible for intestinal inflammation is inhibited. Another product of hydrolyzation is bismuth oxychloride (chloride ions are abundant in the lumen due to CFTR

complex being hyper activated) and bismuth hydroxide; both are believed to have bactericidal (bacteria killing) action.[32]

Low Back Pain

What is low back pain?

Low back pain is a common complaint, most people in the United States will experience low back pain at least once during their lives, and low back pain is also one of the most common reasons for people go to the doctor or miss work.[33] Low back pain is pain in the lumbar (lower) region of your back. This pain can range from mild, dull, annoying pain, to persistent, disabling pain in the lower back. Pain in the lower back can restrict mobility and interfere with normal functioning.[34] Low back pain may result from skeletal irregularities, arthritis, muscle strain, or bulging or ruptured discs. Since I am currently experiencing low back pain from bulging disks, this will be the main topic discussed.

Discs act as cushions between the individual bones (vertebrae) in your spine. Vertebral discs have a nucleus (inner) and annulus (outer) portion (Figure 10). The inner nucleus is the cushy center of the intervertebral disc that is completely encased by the annulus. In young people the nucleus is made mostly of water (around 80% at birth), and dries out as we age. As discs dry out they lose height and shock absorption, this process continues until the ages between 60-70 when our discs are dried out and composed entirely or fiber.[35]

The annulus portion of a disc consists of several rings of tough fibrous cartilage. The fibers run on a diagonal angle with each separate layer running at a right angle to the fibers in the ring next to it. This design increases the strength of the annulus. The annular fibers seal the nucleus and evenly distribute pressure or force imposed on the structure. A tear or rupture in the annulus may be the source of a herniated disc. Most tears are caused by natural aging processes such as discs drying out and becoming brittle.[36] The back (vertebral column) is responsible for bearing most of a person's bodyweight and is susceptible to a great deal of wear over time.[36]

In regards to chemical composition both the nucleus and annulus portions of an intervertebral disc, they are made up of varying compositions of proteoglycan (protein and carbohydrate), collagen (cartilage), and water.[37] Proteoglycan refers to a family of differently-shaped proteins (isomers) that share similar chemical structure. Proteoglycans are formed of glycosaminoglycan's (GAGs) covalently (sharing of electrons) attached to proteins. They are also found in all connective tissues and on the surfaces of many cell types. GAGs are long unbranched molecules containing a repeating disaccharide (two sugars joined together) unit.[38] GAGs bind to proteins via a tetrasaccharide (four sugars joined together) bridge from GAG to the amino acids serine and threonine (forming an O-glycosidic bond) on the protein chain.[38]

These proteoglycans have several similar structural forms within the disc, but the most important arrangement is called aggrecan. The main function of aggrecan is to trap and hold water, which is what gives the nucleus its strength, elasticity, and can trap over 500 times it's weight.[37] Aggrecan is attached to GAGs through which an overall negative charge results and attracts water to attach to the GAGs.[37]

Aggrecan cushions compressive forces because the absorbed water enables it to spring back after having been deformed. When pressure is exerted, as when landing after jumping into the air water is squeezed from GAG, cushioning the impact, and when the pressure is released, the water rebinds.[37] A good analogy is pressure on a car tire. If pressure is placed on one spot on the tire, it is distributed throughout the rest of the tire, and once the pressure is released the tire bounces back to its original form. In this way intervertebral discs help cushion and lubricate our vertebral column with movement.

How does a herniated disc cause pain?

A bulging or herniated disc occurs when the nucleus herniates or slips through the surrounding annulus portion. Common causes for a herniated disc are suddenly lifting, twisting, direct injury, or it can occur gradually from degeneration as we age. The herniated nucleus can also press on nerve roots going to the spinal cord, causing a shock-like pain (sciatica) down the legs, weakness, or numbness.[39] This is a common cause of low back pain in many individuals.

Pain signals are recognized and sent to the brain via neurons which are electrically excitable cells that process and transmit information through electrical and chemical signals. Chemical signals occur via a synapse, or a junction (minute gap between two cells across which impulses pass). Neurons communicate with each other to form neural networks, the main "command center" is the central nervous system (CNS) made up of the brain and spinal cord. There is also a signaling network of peripheral nerves or fibers that stretch to every corner of the body, which relay their signal back to the CNS. A typical neuron will have a cell body, many dendrites used for picking up incoming signals, and only one axon used for sending signals. At the majority of synapses, signals are sent from the axon of one neuron to a dendrite of another[40,41] (Figure 11).

All neurons are electrically excitable, maintaining voltage gradients across their membranes by means of ion (charged atom or molecule) pumps in combination with ion channels embedded in the membrane. Alteration in intracellular (inside the cell) versus extracellular (outside the cell) concentration differences of ions such as sodium, potassium, chloride, and calcium will generate an action potential, or "signal".[42] The action potential travels very rapidly along the cell's axon, and activates synapses.

A great example of how an ion channel works is the potassium (K^+) ion channel. One important detail is that ion channels allow diffusion to occur, and each cell works to maintain a larger amount of K^+ inside the cell versus outside. Beginning from inside the cell, the channel opening is at its largest diameter. K^+ ions are bound to water molecules and can enter into the opening of the channel while still bound to water.[42] Three-quarters of the way through the ion channel, the diameter is decreased to 1/3 of the original, at which point the K^+ ions must shed their water molecules (decrease in size) and interact directly with carbonyl groups (carbon and oxygen share a double bond) of the channel protein.[42] Three K^+ ions are lined up inside the channel, and as another K^+ approaches the channel from either side it sends a "jolt" down the line of ions, similar to one billiard ball hitting two others lined up, and an ion pops out of the opposite side.[43] When enough positive ions cross the channel into the cell (known as depolarization) the chance of a neuron to fire an action potential and propagate a signal is increased.[44] While a negative charge inside the cell (known as polarization) results in no firing of an action.[44]

The generated action potential is sent along the cells axon and arrives at a synapse. A synapse is a small space between the axon of one neuron, and the dendrite of the other. Neurons do not make contact with each other to communicate (Figure 12). Each synapse will have a presynaptic ending that contains neurotransmitters (chemicals that transmit signals across a synapse) typically at the end of the axon, a postsynaptic ending containing receptor sites for neurotransmitters typically at the "beginning" of a dendrite, and a synaptic cleft space between pre and postsynaptic endings[45] (Figure 12). Although more than one type of neurotransmitter exists, the principal one is acetylcholine (ACh, an ester of acetic acid and choline). At the presynaptic ending an action potential will trigger the release of ACh into the synaptic cleft.[45]

ACh then binds to receptor sites on the post synaptic ending, and as a result changes the receptor's conformation to open the ion channel.[46] This allows positive ions to move across, with the net positive flow being inward generating an action potential to be continued through the dendrite of the neuron receiving the signal.

In this way action potentials, or signals are relayed from one neuron to another, quickly making their way back to the central nervous system where the signal is interpreted. With a bulging disc pressure is put on a nerve root in the peripheral nervous system (PNS). The signal would pass from the point of pressure through neurons in the PNS and relayed back to the CNS through neurons in the spinal cord, and eventually to the brain for interpretation.

IV. Conclusion

Throughout the various explanations in this project, many different areas have been observed. Research in heart attack, traveler's diarrhea, and low back pain is continuing, and in the near future we will know much more about how to help/prevent these conditions. By taking advantage of the research and pamphlet instructions everyone has the opportunity to be more informed on conditions that nearly ever human being will experience in some way. This project is not only for the young or elderly, but anyone wishing to be more informed. When we know about certain conditions that are a risk for everyone, we are able to make educational materials that help the general public be more knowledgeable. Which in turn helps with early treatment and care. Lives may be saved, family bonds strengthened, and the hope that by educating ourselves a long and happy life will be possible.

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VI. Figures

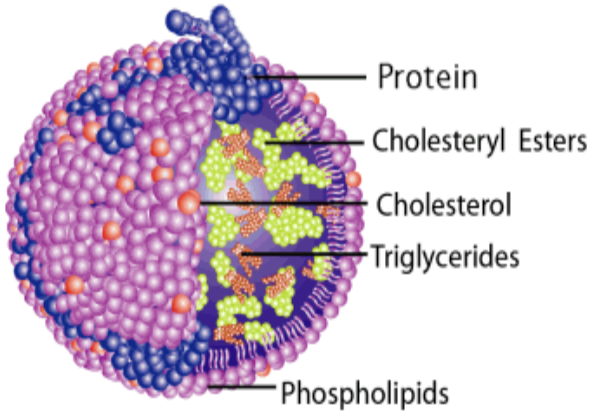

Figure 1. Visual structure of a lipoprotein. Proteins are integrated into the phospholipid membrane which houses the cholesteryl ester, cholesterol, and triglycerides (adapted from ref[6]).

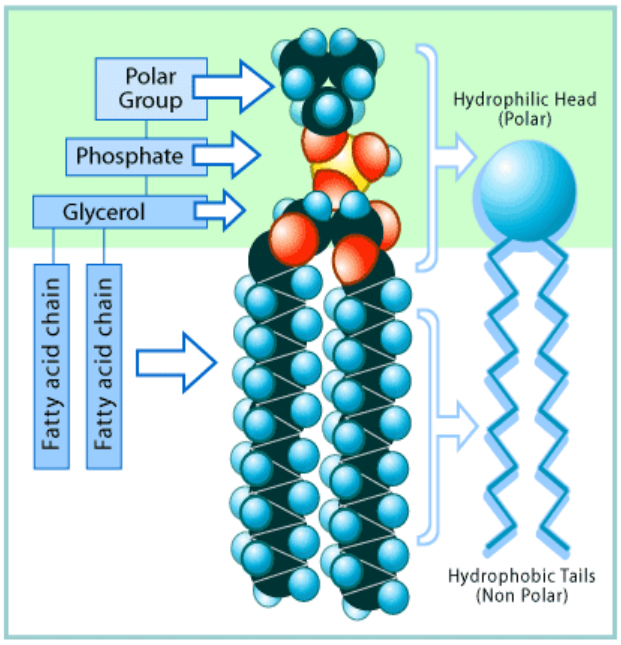

Figure 2. Structure of a phospholipid membrane. Phospholipids are amphipathic in that the "heads" are hydrophilic, or water attracting, and the "tails" are hydrophobic or water repelling. Phospholipids are major components of cell membranes. By being amphipathic flow in and out of the cell can be regulated.

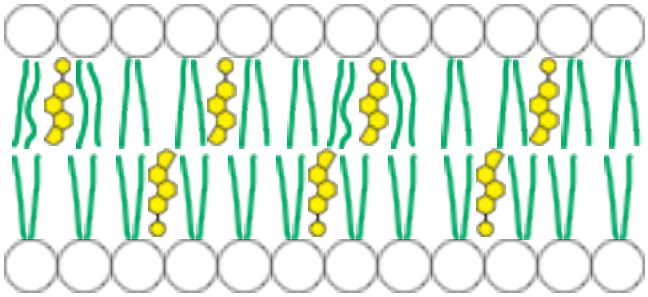

Figure 3. Structure of a phospholipid membrane with hydrogen bonded cholesterol molecules in yellow. The polar head (circles) of the phospholipid membrane forms a hydrogen bond with the hydroxyl (OH) group of cholesterol. (modified from ref[6]).

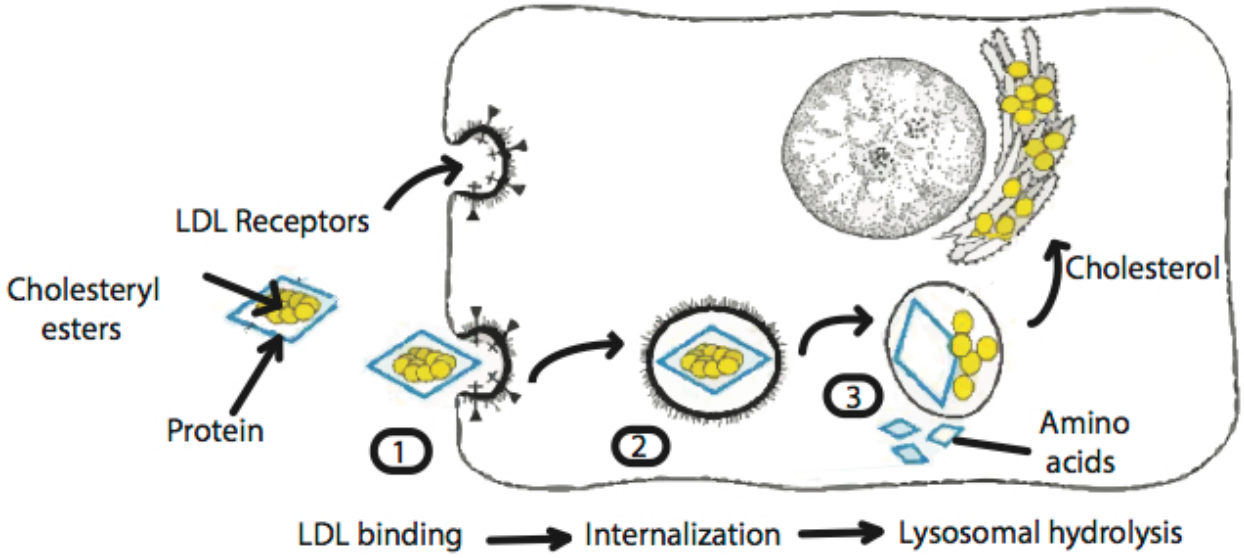

Figure 4. Receptor-mediated endocytosis. (1) – LDL binds to a specific receptor, the LDL receptor; (2) – this complex pouches inward to form an internal vesicle; (3) – after separation from its receptor, the LDL-containing vesicle fuses with a lysosome, leading to the degradation of the LDL and the release of the cholesterol (modified from ref[6]).

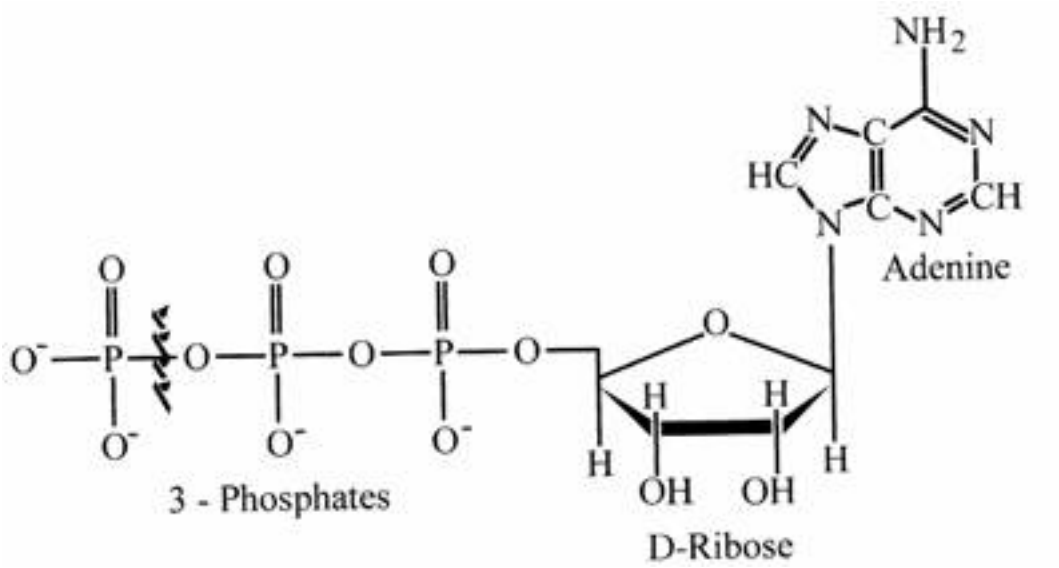

Figure 5. Structure of adenosine triphosphate (ATP). Used by kinases as the source of phosphate groups. Hydroxylation of the terminal phosphate group converts ATP to adenosine diphosphate (ADP). (adapted from ref[25]).

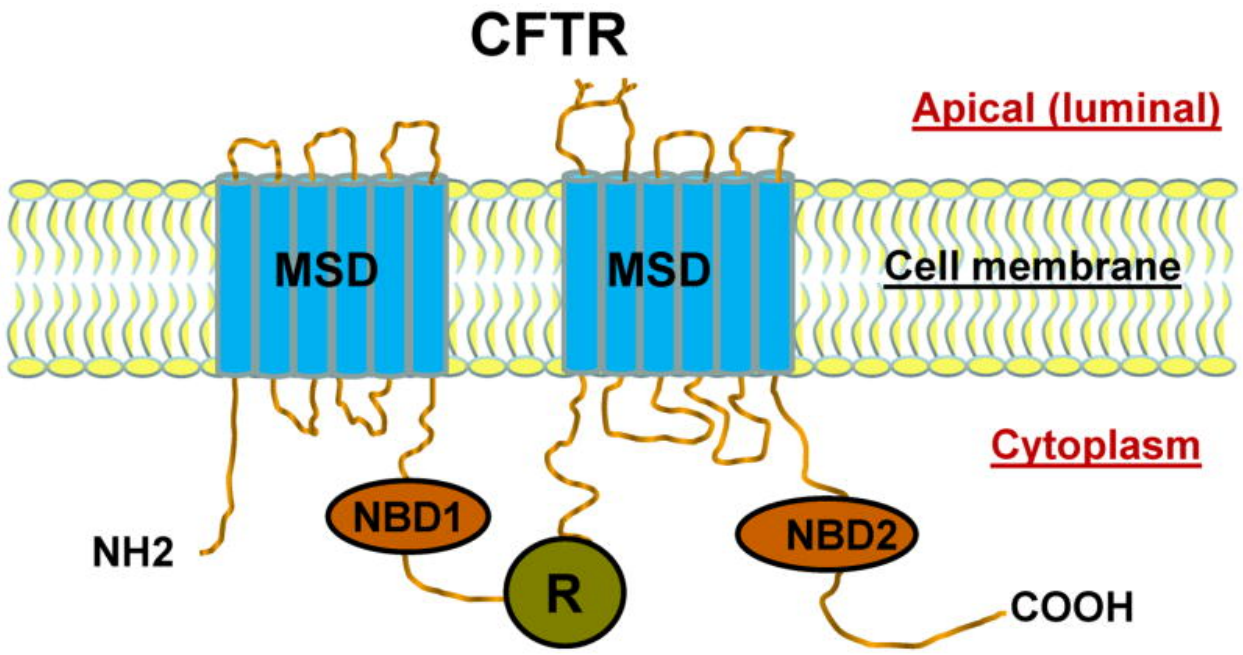

Figure 6. CFTR and its interactions. CFTR consists of two repeated membrane-spanning domains (MSD) containing six helices and a nucleotide binding domain (NBD), which can bind and hydrolyze ATP. The two MSD's are linked by a cytoplasmic regulatory (R) domain that contains a number of charged residues and multiple phosphorylation sites. The CFTR chloride channel can be activated through the phosphorylation of the R domain by various protein enzymes and by ATP binding to, and hydrolysis by, the NBD domain. Both the COOH and NH_2 terminal tails are cytoplasmically oriented and mediate the interaction between CFTR and a wide variety of binding proteins (adapted from ref.[10]).

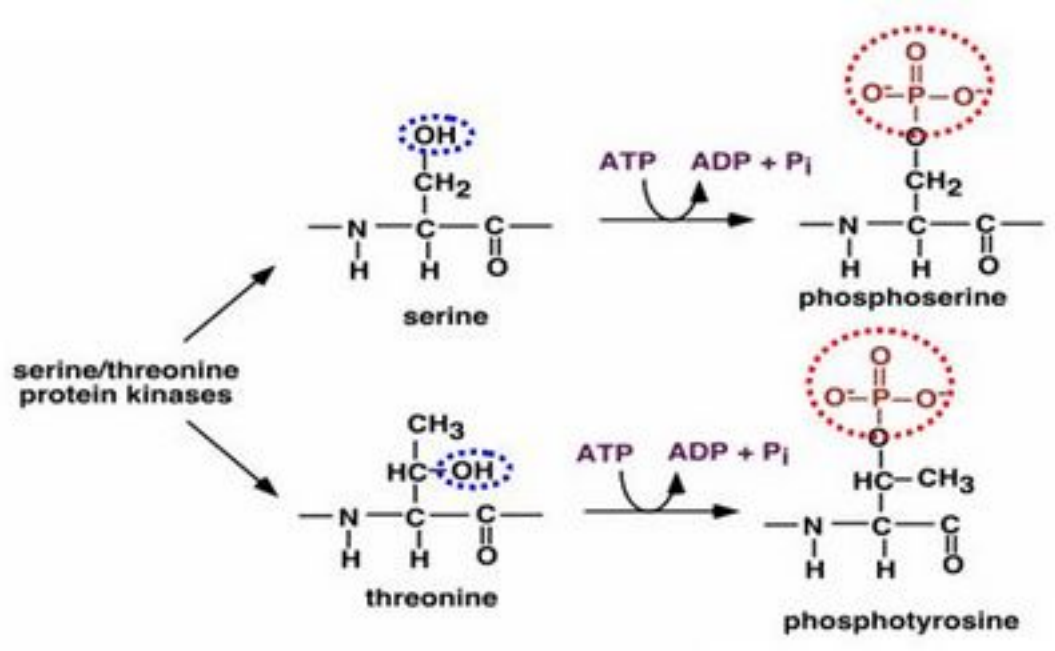

Figure 7. Mechanism for phosphorylation of serine and threonine. The conversion of ATP to ADP releases phosphorus groups causing phosphorylation. (adapted from ref[21]).

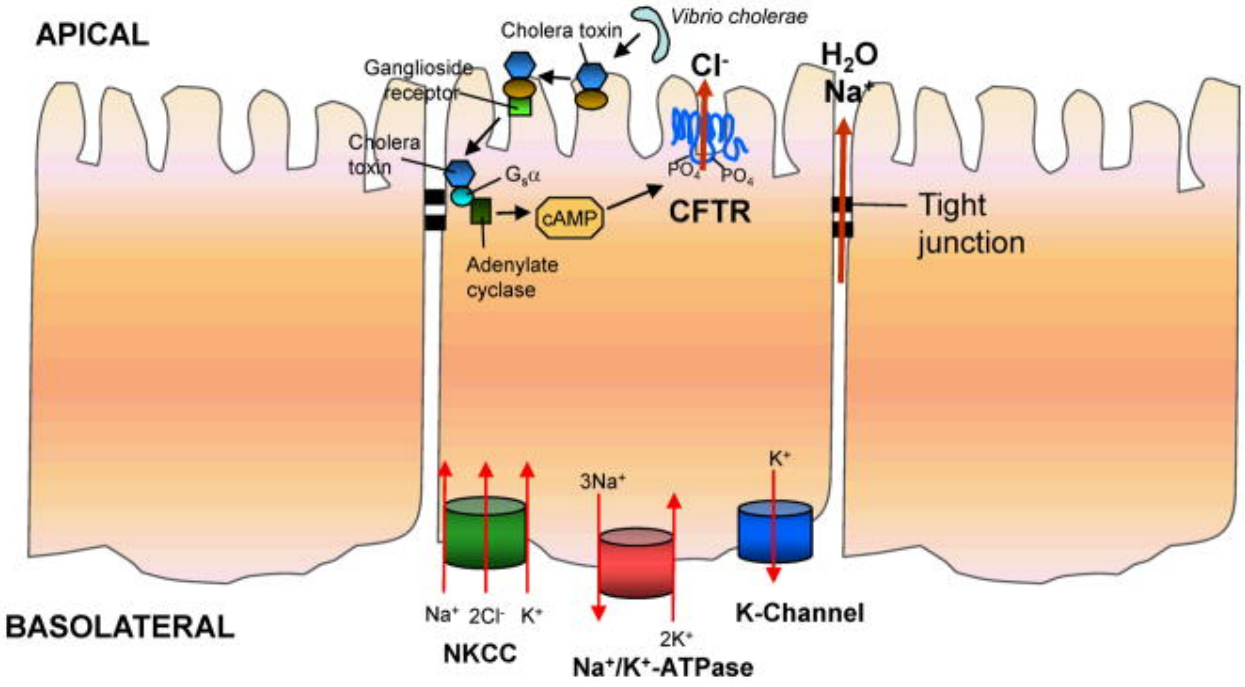

Figure 8. A model of secretory epithelial cell and secretory diarrhea. A heat stable toxin (in this case Cholera) increases the cAMP levels by activating the membrane-localized adenylate cyclase (AC). Increase in the cAMP leads to phosphorylation of the R domain of CFTR by PKA, which in turn activates the CFTR chloride channel, resulting in Cl- secretion into the lumen. As a result, Na+ and water enter the lumen through the paracellular transport mechanism. The net result is the secretion of fluid and electrolytes across the apical surface into the gut lumen. Cl- is taken up from the basolateral (blood) side by NKCC, potassium (K+) recycles through basolateral K+ channels, and Na+ is pumped out of the cell by Na+/K+ -ATPase (adapted from ref.[15]).

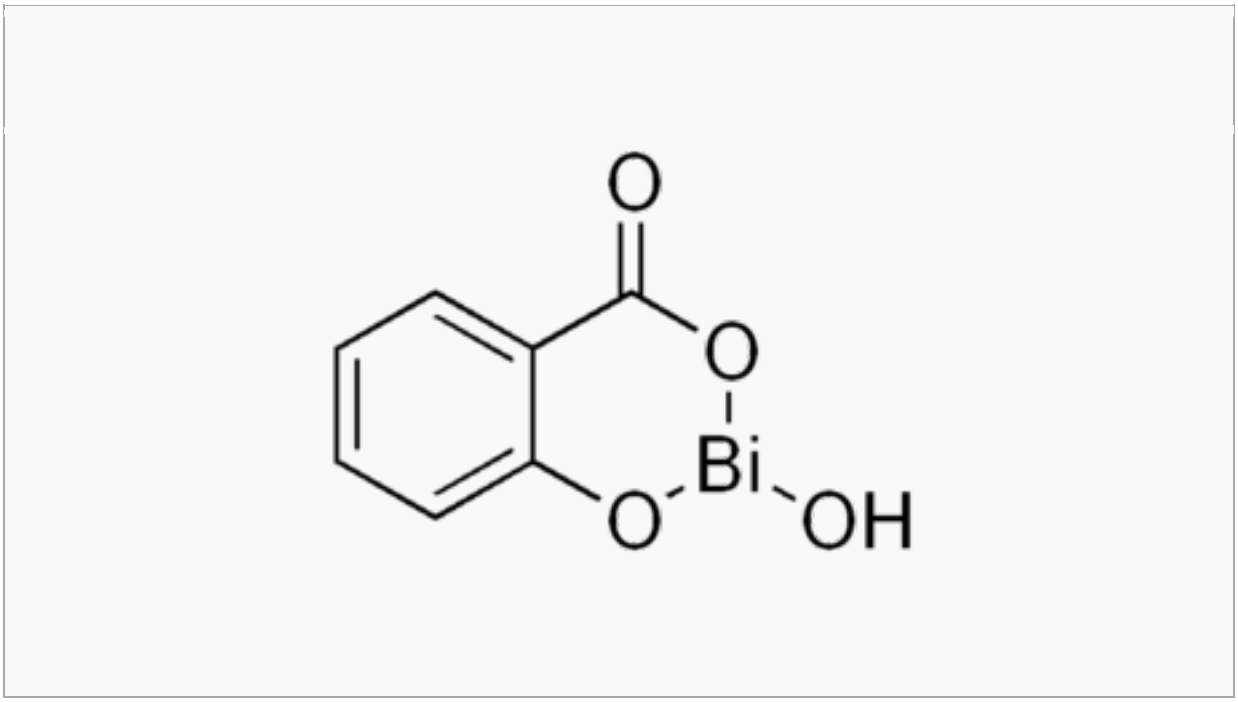

Figure 9. Bismuth Subsalicylate. A bismuth oxide core structure with salicylate ions attached to the surface. Commonly found in Pepto-Bismol.

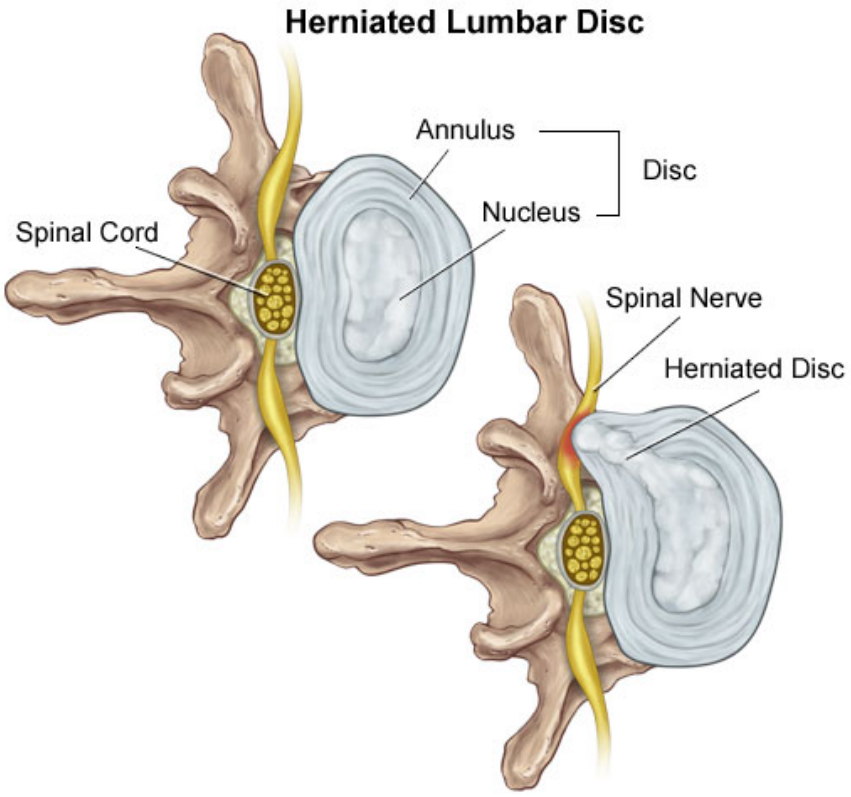

Figure 10. Overhead view of a Herniated Lumbar Disc. Each disc has an inner nucleus (nucleus pulposus) portion, and and outer annulus (annulus fibrosus) portion. When a disc herniates, pressure is put on the spinal nerve, which carries the pain signal to the brain.

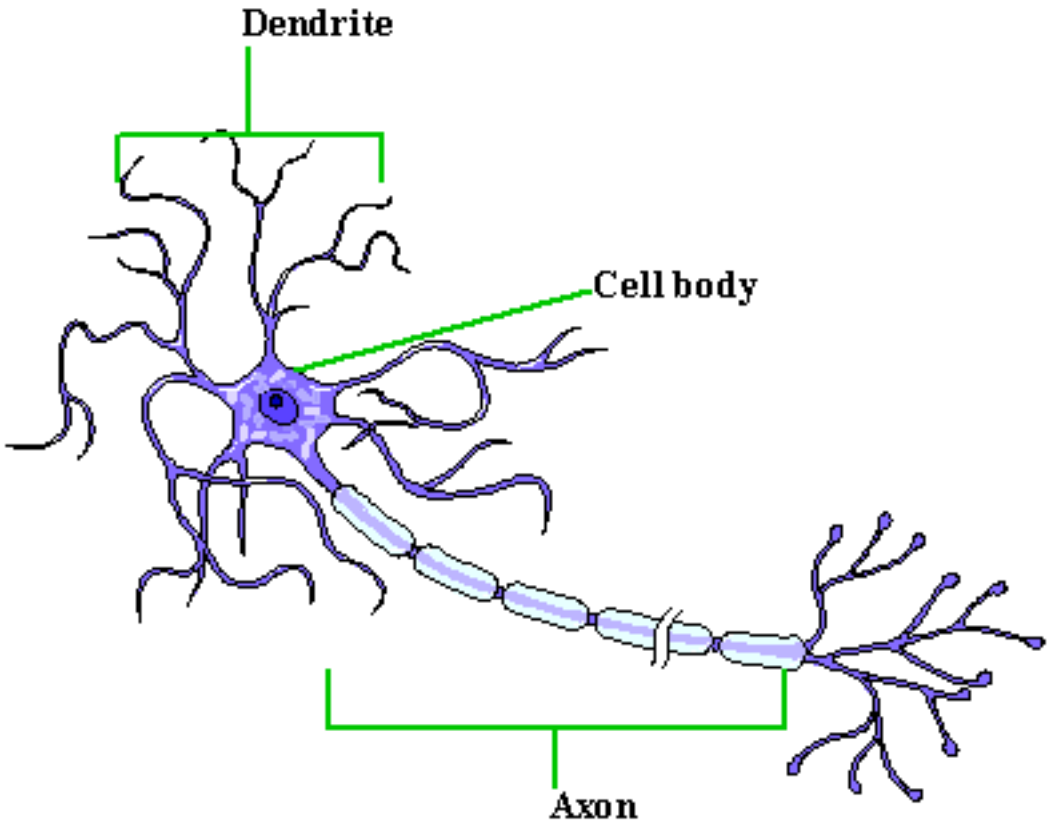

Figure 11. Structure of a neuron. The axon sends signals to other cells, the dendrites pick up signals from other cell axons.

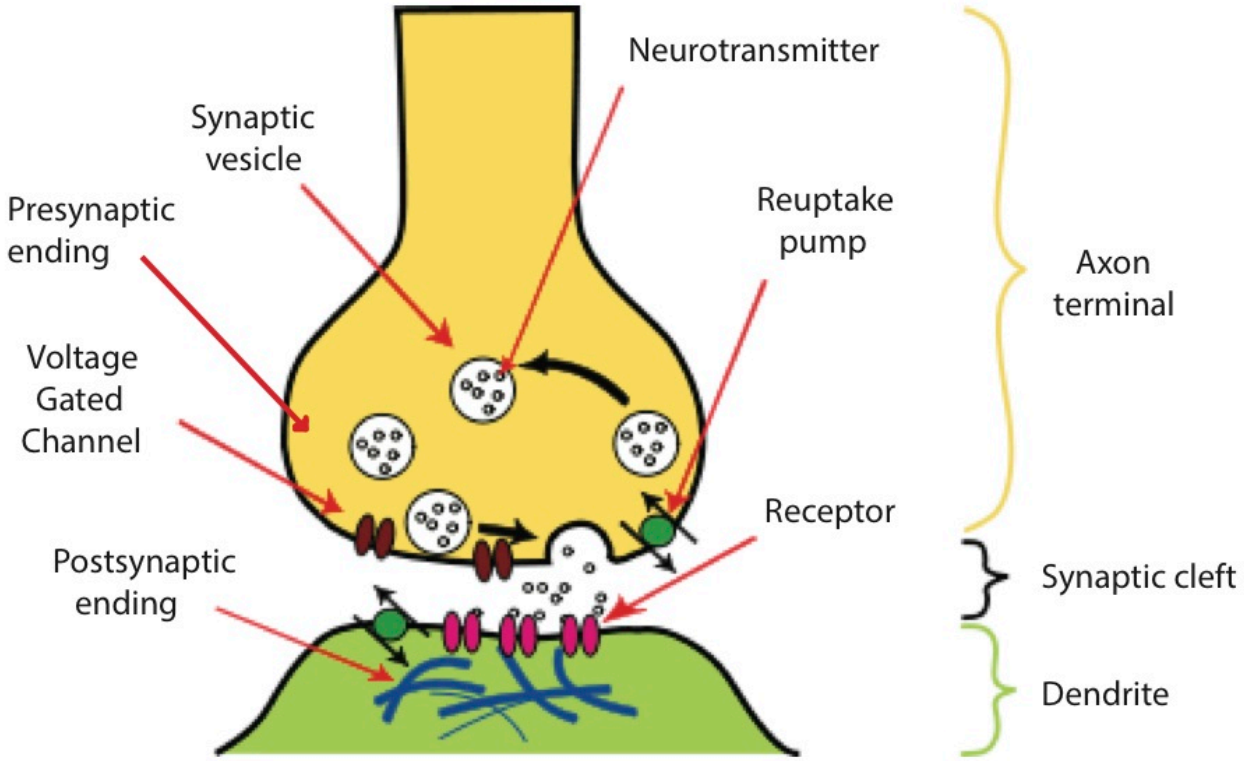

Figure 12. Diagram of a synapse. The presynaptic ending, or axon, receives the signal and releases neurotransmitters into the synaptic cleft. Neurotransmitters then bind to receptor sites on the postsynaptic ending, or dendrite, opening ion channels and a net positive flow inward generates the signal to be continued.