Hertig,kathleen(1)

Nutrition

Hertig, Kathleen(1) The Importance of Lipoproteins and How They Effect Our Body and Through Lifestyle Decisions How Cardiovascular Disease Can Be Prevented and or Controlled. Insoluble in water lipids can be defined. To move lipids like fatty acid, triacylglycerols, steroids and fat soluble vitamins within the blood plasma, a mover protein is needed. Moved from the adipose tissue to the muscle, heart and liver tissues by serum albumin are fatty acids. Moved by the retinol binding protein is Vitamin A. There are steroid moverproteins that move steroids to the aimed cells. Majority of the body's lipids(phospholipids, triacylglycerols and cholesterol), are moved in the plasma by big complexes called lipoproteins. Lipoproteins makeup a center part of hydrophobic lipids encompassed by a shell of phosphotidyl glycerols and proteins. Protein parts of lipoproteins solubilize the hydrophobic lipids and include the cell targeting signals. Assorted according to their density are lipoproteins . The smallest density lipoprotein are the chylomicrons proceeded by the chylomicron remnants, very small density lipoproteins VLDLs, medium density lipoproteins, IDLs, small density lipoproteins, LDLs, and big density lipoproteins, HDLs. The densities of these lipoproteins are correlated to the relative parts of lipids to proteins in the complex. The bigger the protein amount the larger the density of the lipoprotein.(www. tamu. edu/faculty/bmiles/lectures/Lipid%20Transport. pdf). Chylomicrons: Moved from the intestinal mucosa cells to other tissues by lipoproteins that are referred to as chylomicrons, which are dietary lipids. Chlyomicrons are big and have the smallest protein to lipid ratio and therefore have the smallest density of all the lipoproteins. Chylomicrons include phospholipids and proteins on the surface so that the hydrophilic surfaces are in touch with water. The hydrophobic molecules are encompassed in the interior. The

major apoproteins of nascent chylomicrons are apo B-48, apo A-I, apoA-II and apoA-IV. In circulation, the nascent chylomicrons acquire apo-C and apo-E fromplasma HDL in replacement for phospholipids. The acquisitionof apo-CII fromHDL is substantial to start up lipoprotein lipase, LPL. Chylomicrons tie up to membrane bound lipoprotein lipases (LPLs). Lipase, LPL are placed on adipose and muscle tissues where the triacylglycerols are hydrolyzed into fatty acids. The fatty acids are moved into the adipose cell where they are again recombined into triacylglycerols and kept. In the muscle, the fatty acids are oxidized to give energy. As the tissues soak up the fatty acids, the chylomicrons gradually become smaller until they are decreased down to cholesterol enriched remains. As the chylomicron becomes smaller it moves a good portion of its phospholipids and apoproteins A and Cto HDL. The apo C proteins are continuously converted between chylomicrons and HDL. The remains missing apo A and C proteins will not bind to the LPLs in the capillaries. The remains are soaked up by the liver. Chylomicrons tie up to Lipoprotein Lipases in the capillaries of the tissues. Apo-CII is needed Hertig, Kathleen(2) to convert the LPLs. The LPLs hydrolyze the fatty acid ester bonds freeing glycerol and free fatty acids. The fatty acids are soaked up by the endothelial cells that line the capillary. LPL is serine esterase that is located mostly in muscle and adipose tissue. LPL is discharged out of the cell and is shifted to the lumenal surface of the endothelial cells lining the capillary where it is fastened to heparin sulfate. LPL is the most important enzyme responsible in the processing of chylomicrons and VLDLs. (dietheartpublishing. com/node/282). Very Small Densisty Lipoproteins: The liver combines fatty acids and cholesterol and wraps them up for movement into the blood plasma in VLDLs. The cholesterol is unesteried and instituted

as a surface component of the lipoprotein. A large cholesterol diet changes the composition of the VLDL with cholesteryl esters replacing for triacylglycerols as the major constituent of the lipid make up. The major apoprotein is B-100. The liver discharges VLDLs via exocytosis. VLDLs undergoes repeated changes in the plasma. First, the nacent VLDL obtains apo C and E fromHDL. VLDLs ties up to the same membrane bound lipoprotein lipases (LPLs) on adipose and muscle tissues where the triacylglycerols are hydrolyzed into fatty acids. The fatty acids are moved into the adipose cell where they are again recombined into triacylglycerols and kept. In the muscle, the fatty acids are oxidized to give energy. As the tissues soak up the fatty acids and monoacylglycerols, the VLDLs gradually become smaller making IDLs. As the VLDL becomes smaller it moves a good amount of its phospholipids and apoprotein C to HDL. IDLs can adhere to receptors of liver cells where they are soaked up in a manner to chylomicrons, or they can moreover be catabolized by LPLs, lastly unbinding apo-E to form LDLs. LDL, a cholesterol abundant lipoprotein which makes up apo B-100. LDL is the major plasma cholesterol mover. The concentration of LDLs absolutely correlates with coronary heart disease. LDL is sometimes referred to the bad cholesterol. Transporter of plasma cholesterol to the tissues is LDL. It serves as a source of cholesterol for the majority of the tissues of the body. Large levels of LDL are connected with the forming of atherosclerotic plaques that block blood vessels bring about heart attacks and strokes.(http://www. sciencedaily. com/articles/l/low_density_lipoprotein. htm) Small Density Lipoproteins: LDLs tie to particular cell receptors found on the plasma membrane of aimed cells Glycoprotein is the LDL receptor that has a domain with negative charged residues. The LDL binding domain

has electrostatic interactions withthe positively charged arginine and lysine residues of apo-B100. LDL receptors go to areas of the plasma membrane that are especially for endocytosis called coated pits. They get the name coated pits because of the clatharin protein coat on the cytoplasmic side of the membrane. When the LDL ties to the receptor, the clathrin proteins advances endocytosis. When the vesicle is in the cell, the clathrin voluntarily separates from the endosomal vesicle. PH of the vesicle is decreased to such that LDL separates from the receptor. LDL receptors are converted to a reusable material to the cell surface. The vesicle combines with a lysosome which then lowers the lipoprotein to its main components, amino acids, cholesterol, glycerol and fatty acids. The cholesterol is merged into Hertig, Kathleen(3) the intracellular cholesterol pool which is utilized for the membrane. (http://www. sciencedaily. com/articles/l/low_density_lipoprotein. htm) Large Density Lipoproteins: Discharged by the liver and intestinal cells are HDLs. Disk shaped, but they become round as they obtain free cholesterol from cell membranes and triacylglycerols from other lipoproteins are nascent HDLs. The major function of HDLs is to eliminate excess cholesterol and carry the excess to the liver to be metabolized into bile salts. The duty of cholesterol elimination from the tissues is the inverse relationship between the plasma concentration of HDLs and the prevalence of heart diseases. Commonly known as the good cholesterol HDL. It is the mover of plasma cholesterol back to the liver. Enzymes that contain either esterify cholesterol or move cholesteryl esters are HDLs. Enzyme that circulates with HDL is Lechithin-cholesterol(LCAT)that catayzes the movement of long chain fatty acids from phospholipids to cholesterol to make cholesteryl esters. The lipid core of the Cholesteryl esters occupy HDL .

Facilitation, keeping and movement of excess cholesterol is LCAT. It is activated by apo A-I. Exchanged between lipoproteins are Cholesteryl esters. Cholesteryl ester transfer protein (CETP) which is another protein that circulates HDL. Promotion the net movement of cholesterol esters from HDL to LDL, IDL and VLDL in exchange triacylglycerols is CETP. By this process, it converts VLDLs and IDLs into LDLs. HDLs increase in size they gain apo-E which enlarges the binding of the HDL heads to receptors in the liver. The liver then soaks up and catabolizes HDL.(www. ncbi. nlm. nih. gov/pubmed/2642759). Dietary Considerations for Prevention and Reduction of Cardiovascular Disease: Vegetable oils that contain trans fatty acids should be removed from diets because of their correlation to increased risk of cardiovascular disease. Saturated fats should be consumed in moderation in order to control or prevent cardiovascular disease. An even better combination would be mono-unsaturated and poly-unsaturated fats in place of saturated fats to reduce risk of cardiovascular diease.(Willett). A lifestyle of modifying risk factors can prevent and or control sudden cardiac death in in women. These factors would include not partaking in tobacco product use, weight that is healthy and maintained, and a diet that does not include any trans fat and limited saturated fat (Chiuve, Fung, Rexrode, Spiegelman, Manson, Stampfer and Albert). Not enough Vitamin D in our diet can negatively effect our musculoskeletal system and health. Since our heart is part of this system it can effect our cardiovascular health as well. Parathyroid hormone levels become increased with Vitamin D deficiency and insufficiency. This creates a chain reaction of events, insulin resistance becoming worse, which could cause systemic inflamatory process, high blood pressure, enlargement of left ventricle and diabetes. Increased cardiovasular

death, there is a correlation with it and decreased levels of 25-hydroxyvitamin D.(Shapees and Manson). Work Cited Adherence to a Low-Risk, Healthy Lifestyle and Risk of Sudden Cardiac Death Among Women Stephanie E. Chiuve, ScD, Teresa T. Fung, ScD, Kathryn M. Rexrode, MD, MPH, Donna Spiegelman, ScD, JoAnn E. Manson, MD, DrPH, Meir J. Stampfer, MD, DrPH, Christine M. Albert, MD, MPH JAMA. Carl S. Swisher Library. 2011; 306(1): 62-69. doi: 10. 1001/jama. 2011. 907. Web. 6, April 2013. www. dietheartpublishing. com/node/282. Web. 6, April 2013. Dietary fats and coronary heart disease. Detail Only Available (includes abstract) Willett WC; Journal of Internal Medicine, Carl S Swisher Library, CIANL. 2012 Jul; 272 (1): 13-24. (journal article - review) ISSN: 0954-6820 PMID: 2258305. Web. 6, April 2013. www. ncbi. nlm. nih. gov/pubmed/2642759. Web. 6, April 2013. http://www. sciencedaily. com/articles/l/low_density_lipoprotein. htm. Web. 6 April 2013 www. tamu. edu/faculty/bmiles/lectures/Lipid%20Transport. pdf. Web. 6, April 2013. Vitamin D Supplementation for Cardiovascular Disease Prevention–Reply Sue A. Shapses, PhD, JoAnn E. Manson, MD, DrPH JAMA. Carl S. Swisher Library. 2011; 306(14): 1546-1548. doi: 10. 1001/jama. 2011. 1466. Web. 6 April 2013. Paper title: Week 5 Part B Paper ID: 318310799 Author: hertig, kathleen The plagiarism detector has analyzed the following text segments, and did not find any instances of plagiarism: Text being analyzed Result binding domain has electrostatic interactions withthe positively charged arginine and OK acquisitionof apo-CII fromHDL is substantial to start up lipoprotein lipase, LPL OK lipoproteins VLDLs, medium density lipoproteins, IDLs, small density lipoproteins, LDLs, OK concentration of LDLs absolutely correlates with coronary heart disease OK Majority of the body’s lipids(phospholipids, triacylglycerols and cholesterol), are moved OK

better combination would be mono-unsaturated and poly-unsaturated fats in OK Dietary Considerations for Prevention and Reduction of Cardiovascular Disease: OK Vitamin D Supplementation for Cardiovascular Disease Prevention–Reply OK Results: No plagiarism suspected