

Lipidology Apo B is Important? Become Atherogenic? In Brain, Heart, Vascular, How?

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LDL-C is causative of cardiovascular disease. It is also the attack factor. The metabolism of apo B contains lipoproteins, these are absorbed in intestinal cells, such as chylomicrons, these chylomicrons, will be metabolized in the liver as LDL, vldl1, VLD L2, and LDL [1,2]. How does hyperlipidemia produce? atherosclerosis apo B contains lipoproteins that cause atherosclerosis. There is consensus that low-density lipoproteins cause atherosclerotic cardiovascular disease. There is genetic, epidemiological evidence, and clinical studies, low-density lipoproteins cause cardiovascular, pathophysiological, genetic disease. As lipoproteins enter the arterial wall by a mechanism called transcytosis, it is a passive filtration, through the endothelium. Transcytosis, involves Alk1, SRB1 and caveolin Estrogens inhibit LDL, endothelial this explains why women are protected from cardiovascular disease before menopause. Experimental studies indicate that hypercholesterolemia and hyperglycemia can increase LD transcytosis L through the endothelium. There is one in the apo B 100 has positive receptors that mediate the union with the arterial wall in Proteoglycans. Chylomicrons and VLDL cannot enter the endothelium because they are heavy, only LDL particles that are less than 70 nanometers. Triglycerides are cleaned by lipolysis as well as cholesterol esters. Lipidic postponement dial metabolism is governed by Apo B 48 containing particles that are secreted not only by chylomicrons but also by the cell since about half of Apo B 48 contains deb LDL particles derived from lipolysis chylomicrons, and half by direct secretion. Apo B of 100 and apo B of 48 both from the liver and the intestine, can raise triglycerides, the remnant, or with the retention of lipoproteins, are those that initiate atherosclerosis modifying the MLL making a cellular response and causing inflammation, of the macrophage. Oxidized phospholipids have been found in which they form a but non-enzymatic oxidation of lipids in inflamed tissues, including atherosclerotic lesions, which are caused by the accumulation of atherosclerosis in the absence of LDL - does the trial songs testify that if the anti-inflammatory therapy with Canakinumab a monoclonal antibody, which neutralizes interleukin one sees does not lower the incidence of cardiovascular events. Canakinumab was effective in reducing adverse cardiac events over 3.7 years and methotrexate did not result in any cardiovascular effect. Retained lipoproteins causes early lesion development and accelerates accelerated retention and aggregation of LDL C. The deposition of lipids infiltrate the macrophages in the arterial wall. The mechanism of progression of a stable cardiovascular lesion is apolipoprotein, macrophage inflammation, inflammatory mediators, fibrous cap. In unstable lesions to see an accumulation of retained apolipoprotein, there will be an effect on necrotic cells, inflammatory mediators, fibrous layer will be thin. Subsequently, the rupture mechanism will be more apolipoprotein accumulation of necrotic cells, ruptured plaque. This triggers Oxidative stress proteolysis, lipolysis, and activation. In conclusion, at mononucleosis is that there will be pro-inflammatory mediators, cell necrosis in the stable plaque, later the plaque will progress, formation of necrotic nuclei, metalloprotein loops, and rupture of the plaque, producing the thrombus. Then the mechanism will be stable lesion, advanced lesion, plaque rupture, with thrombosis, and in conclusion the ApoB v can be decreased to avoid plaque rupture. LDL receptors in the liver to be degraded by

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the lysosomes and incorporated into cells. Imagine a Y inside a cell and then its evolution. The Pck 9 are placed below the Y is introduced inside the cell and destroys itself the statins does the same effect increases synthesis and degradation. Pharmacotherapy with these tubs, tpck9, will include a Increase the number of LDL in the liver, recent studies say that its administration for six months reduces by 50% in LDL levels while Pck9 reduces them from 50 to 70%, ezetimibe 15% and statins 30 to 40%. Currently there is treatment for homozygous autosomal dominant hyperlipidemia, from birth, familial heterozygous familial hyperlipidemia., Mutant gene: receptor, apoB, pck9, LDL-*rap1*, protein. LDL recep, Nobel price 1985. Ultrasound is the method to observe the progression or regression of the atheroma plaque as shown in the slide in our laboratory. There are studies with evolocumab c ro auction tina with intracoronary ultrasound to observe the progression and regression of the plaque. 67 to 57, or prog 48 to 58. Atherogenesis is initiated by retention of its endothelial Apple ve containing lipoproteins, these induce inflammation within the arterial wall. And the best strategy is to reduce those with the drugs LDL, eZetimibe, pck9, inclisiran, bempedoic acid. Risk factors such as smoking, hypertension, diabetes, elevated triglycerides, decreased HDL and elevated lipoprotein are risk factors Duth lipid Network classification criteria; which is definitive, probable, and possible, definitively eight points probable, 68 and possible three to five. It is also important that the lipoprotein an elevated by Apple a can cause coronary atherosclerosis, infarction and aortic stenosis. The incidence of myocardial infarction with lipoprotein A has been described by Lannsted in 2016. One in every 200 people may have hypercholesterolemia family. the most aggressive form of homozygous familial hypercholesterolemia is that with a negative LDL receptor mutation. Heterozygous familial hypercholesterolemia in the general population has a range from one to 200 to one to 300. We can conclude Novelties in the treatment of familial hypercholesterolemia and hyperlipidemia by lowering LDL receptors. The Fourier study decreased Lipo A by 27% in 59%, as well as cardiovascular events with evolocumab, as well as Ebbinghaus. Alirocumab with odyssey trial the future is now we can control hyperlipidemia with novel and interesting drugs [3-5].

Ethical Responsibilities

The authors declare that they have no conflicts of interest when writing the manuscript.

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