Hyperalphalipo proteinemia is associated with distinct of lipoprotein.

Roberta Cesaro*

Department of Obstetrics and Gynecology, University of California, Davis Sacramento CA, United States

Introduction

The major lipoprotein types, Very Low-Density Lipoprotein (VLDL), Low-Density Lipoprotein (LDL), High Density Lipoprotein (HDL), are composed of many subgroups Classifications are based on size density, or Apo lipoprotein (Apo) content or a combination of these and the subtractions that are isolated have distinct metabolic and other functional properties. Thus, it is entirely reasonable to think that subtractions of the major lipoproteins have diverse relationships to Coronary Heart Disease (CHD). Because the classical lipid risk factors by no means perfectly predict CHD in patients, lipoprotein sub fractionation has the potential to improve risk prediction [1].

Analytical ultracentrifugation is the original gold standard to which subsequent methods have been calibrated and validated. Analytical ultracentrifugation measures the flotation velocity of LDL in a gravitational field; the faster the flotation velocity, the more lipid rich the LDL. This method is currently available only in a few research laboratories worldwide. Preparative ultracentrifugation separates discrete LDL subtractions that can be quantified and studied for chemical composition and function. The classic method separates seven LDL density fractions and although it is time consuming, any laboratory that has an ultracentrifuge can do it [2]. Preparative ultracentrifugation is the method that defines the LDL subclasses, and it is the *de facto* gold standard in the field. No equilibrium density gradient ultracentrifugation uses the same principles as analytical ultracentrifugation, and is simpler and less labor intensive. The vertical auto profiling system is a well-validated example of this technique Gradient gel electrophoresis is a simple, readily available method to determine LDL size. It uses a drop of whole plasma or serum, and multiple samples can be processed together. It has been extensively validated using ultracentrifugation. Its limitation is that, in contrast to ultracentrifugation, it does not quantify the concentration of LDL particles of specific sizes, just the size of the predominant LDL species or the average size of LDL. Thus, changes in the predominant or average LDL size do not necessarily indicate changes in concentration of a particular species of LDL. For example, a selective reduction in large LDL concentration with no change in small LDL concentration would reduce the average size of LDL: this is often misinterpreted to mean an increase in the plasma concentration of small LDL particles. Nuclear Magnetic Resonance (NMR) measures the diameter and lipid concentration of LDL Diameter is determined by a signal from the phospholipid surface coat of LDL, and concentration by the number of methyl groups on the cholesterol ester and triglyceride molecules within each of the four LDL subtractions resolved. Average LDL size is then calculated by the weighted average of the LDL subtractions. NMR computes the concentration of LDL subtractions using typical lipid contents of LDL subtractions in the published literature [3].

NMR is the most rapid and convenient method for determining LDL size and subtraction concentration. However, it is limited by lack of published data on detailed procedures, calibration, and validation, which are expected when novel methods are established. The assumptions and calibration method that NMR uses to convert lipid signal intensity and size to LDL concentration have not been revealed, nor is it known whether these assumptions hold equally across diverse populations and during diet or drug therapy that affects the composition of LDL. For example, when LDL becomes cholesterol-ester poor and triglyceride rich, as in the generation of small LDL described above, the algorithms used may be incorrect as there is not likely to be a single triglyceride for cholesterol exchange in the final particle. Validation studies in large populations have not been published on LDL subtraction concentration by NMR and ultracentrifugation on the same samples, as have been long available for gradient gel electrophoresis [4].

Mechanistic support for small LDL having a special atherogenicity depends on atherogenic actions being greater for small than for intermediate or large LDL. This case has not been proven, however. Both large and small LDL compared with intermediate size LDL have reduced affinity for the LDL receptor which clears LDL from plasma Decreased clearance of these forms of LDL by the liver and steroid genic tissues is thought to lead to increased uptake by the arterial wall. *In vivo*, small LDL has a longer residence time in plasma than large LDL This may be caused by reduced exposure on small LDL of the region of Apo B that binds to the LDL receptor, an interaction that is necessary to clear LDL from the circulation. The long residence time in plasma for small LDL could foster atherosclerosis if small LDL entered the arterial intima more readily than other LDL. Although this was found in experiments in rabbits a study of Trans vascular transport of LDL *in vivo* in humans did not find a correlation with LDL size. This finding suggests

***Correspondence to:** Roberta Cesaro, Department of Obstetrics and Gynecology, University of California, Davis Sacramento CA, United States, E-mail: robertacesaro@gmail.com

***Received:** 24-Feb-2022, Manuscript No. AAATR-22-110; **Editor assigned:** 26-Feb-2022, Pre QC No. AATR-22-110(PQ); **Reviewed:** 12-Mar-2022, QC No. AATR-22-110; **Revised:**24-Mar-2022, Manuscript No. AATR-22-110(R); **Published:** 31-Mar-2022, DOI:10.35841/aatr-6.2.110*

***Citation:** Cesaro R. Hyperalphalipo proteinemia is associated with distinct of lipoprotein. Am J Transl Res.2022;6(2):110*

that for every unit of time, large LDL is just as likely as small LDL to enter the arterial intima. Because large LDL has more cholesterol ester than small LDL, a large LDL particle would deposit more cholesterol into plaque than small LDL. Small LDL binds to arterial proteoglycan in the arterial wall, but so does large cholesterol-rich LDL Proteoglycan exists on the endothelial cell surface as well as inside the intima. Proteoglycan binding on endothelium may facilitate lipoprotein entry into the vascular intima, and binding to arterial intimal proteoglycan activates or accelerates plaque progression. Thus, it appears that both large and small LDL share undesirable characteristics [5].

References

1. Wilson A, Marcil M, Clee SM, et al. Mutations in ABC1 in Tangier disease and familial high-density lipoprotein deficiency. Nat Genet. 1999;22(4):336-45.
2. Clee SM, Kastelein JJP, van Dam M, et al. Age and residual cholesterol efflux affect HDL cholesterol levels and coronary artery disease in ABCA1 heterozygotes**.** J Clin Invest. 2000;106:1263-70.
3. Marcil M, Boucher B, Krimbou L, et al. Severe familial HDL deficiency in French-Canadian kindreds: clinical, biochemical, and molecular characterization**.** Arterioscler Thromb Vasc Biol. 1995;15(8):1015-24.
4. Marcil M, Yu L, Krimbou L, et al. Cellular cholesterol transport and efflux in fibroblasts are abnormal in subjects with familial HDL deficiency**.** Arterioscler Thromb Vasc Biol. 1999;19(1):159-69.
5. Warnick GR, Wood PD. National cholesterol education program recommendations for measurement of high-density lipoprotein cholesterol: executive summary**.** Clin Chem. 1995;41(10):1427-33.

Citation: *Cesaro R. Hyperalphalipo proteinemia is associated with distinct of lipoprotein. Am J Transl Res.2022;6(2):110*