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## Apolipo proteins and its Role in Genetic Diseases

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### Commentary

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### INTRODUCTION

The main aim of the this topic is to know how apolipoproteins are influencing in causing the different genetic diseases like Atherosclerosis, Cardiovascular diseases, coronary heart diseases. Apolipoproteins are the lipid molecules which binds to proteins to form lipoproteins. The different types of lipoproteins includes HDL, LDL, VLDL and chylomicrons. Among these types HDL, LDL are primarily involved in causing the Atherosclerosis and Cardiovascular diseases.

Low plasma HDL will results in increasing the activity of Cardiovascular disease on patients who were already suffering from this condition. The causative factors to develop the CVD involves Apolipoproteins. Individuals with low HDL group will have higher concentrations of atherogenic particles, where as individuals with High HDL group will have small and VLDL. This conclude that low HDL individuals are at higher risk of for atherosclerosis and CVD as compared to high LDL category [1].

HDL has the ability to accept cholesterol from macrophage cells which reflects the role of HDL on Atheroprotection. Ex vivo cholesterol potentiality was studied from blood samples of a normal persons and from patients suffering with or without coronary artery disease. When compared with healthy volunteers patients with coronary artery disease will not only possess lower levels of HDL and apolipoprotein but also had lower cholesterol efflux capacity [2]. Cholesterol molecules present within the LDL particles will accumulate in macrophage and leads to the formation of large droplets which converts the macrophage to foam cell i.e Atherosclerotic lesion [3].

Schnyder corneal dystrophy is a condition which is characterized by accumulation of lipids in cornea. Some genetic disorders are also caused due to deposition of lipid molecules these lipid molecules will effect the function of HDL. HDL Associated apolipoproteins are accumulated more in this Schnyder corneal dystrophy. The main function of HDL is to remove cholesterol from tissue but in this condition the cholesterol removal in SCD is impaired. By identifying the defected gene in SCD and by knowing the different mechanisms involved in cholesterol mechanism it is possible to prevent this condition [4].

Most recent studies have recommended the part of Low-Density Lipoprotein Cholesterol (LDL-C) in the pathogenesis of atherosclerosis and the danger of Coronary Artery/Heart Disease (CAD/ CHD) occasions. The advancement of the "statins" class of medications gave the huge jump in the administration by pharmacotherapy of hyperlipidemia and CHD hazard lessening. Unmethodical clinical trials have given solid proof that bringing down plasma cholesterol with statins diminish the danger of causing cardiovascular/CHD condition.

Recently National Cholesterol Education Programme (NCEP) has given an update regarding high risk patients with lower HDL and those who belong to this category are recommended to be given a combination of folic acid or nicotinic acid instead of giving the LDL-C lowering agent. By giving such therapy to high risk patients it is possible to reduce the LDL-C levels by at least 30–40% [5].

Studies have demonstrated a connection in the middle of dyslipidemia and CHD for quite a few years and bringing down of low-thickness lipoprotein cholesterol (LDL-C) with statins has ended up a piece of the standard treatment regimen in patients with made CHD. Notwithstanding viability of momentum gauges of consideration (counting accomplishment of LDL-C, pulse and glucose objectives), patients with atherogenic dyslipidemia which is regular in patients with diabetes mellitus, metabolic disorder or cardiovascular maladies stay presented to a high lingering

danger of major cardiovascular vents and miniaturized scale vascular entanglements. Glossy silk treatment does not enough address vascular danger connected with hoisted triglycerides and low HDL-C levels. As studies demonstrates, the expansion of lipid adjusting movement of fenofibrate to statin treatment profited just certain subgroups of patients at expanded cardio metabolic danger. Albeit,there is still under treatment with statins, particularly in patients with CV sickness. Under 50% of patients were on focus for LDL-cholesterol. Patients who don't accomplish limits for statin treatment, however who are generally at high hazard for cardiovascular. Occasions, ought to in any case get statin treatment <sup>[6]</sup>.

Apolipoproteins also play a key role in development and regeneration of central nervous system. The genes encoding for this apolipoproteins and their receptors will cause various disturbances in our body and also effect the drug metabolism.It has been shown that it is not only the quantity of HDL particles, and thus HDL-cholesterol level in plasma, that matters, but their quality and impaired functionality as well. HDL from diabetic subjects also lose some of their antiatherogenic properties but a common feature of patients with diabetes type 2 is atherogenic dyslipidemia which is characterized exactly by low HDL-cholesterol and high triglycerides <sup>[7]</sup>.

Diabetic patients with such dyslipidemia are at high CVD danger and the consequences of studies, for example, ACCORD-Lipid proposed that in them treatment of these lipid anomalies might behelpfull. Treatment choices with fibrates, especially fenofibrate, and niacin are talked about based upon distributed trials, and also blend treatment with these prescriptions and other lipid-bringing down medications <sup>[8]</sup>.

### CONCLUSION

From past five years there are many cases with genetic diseases manly due to effects of apolipoproteins as these lipid molecules will increase the frequency of causing genetic diseases. Hence it is important to implement new techniques by knowing their complete structural, molecular and their binding properties to reduce the risk of causing diseases.

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