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### A Manuscript on lipoprotein A: “Lp little a”

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**INTRODUCTION** Of any ethnic groups studied so far Asian Indian have highest rate of coronary artery disease (CAD). India accounts for approximately 60% of the world's heart burden despite being 20% world's population. It's nearly occurs early in age and generally follows a malignant course. Conventional risk factors like diabetes, insulin resistance, high triglyceride low high-density lipoprotein and abdominal obesity are important risk factor in are Genesis of coronary heart disease but cannot account for the 5-10-fold higher rate of CAD an individual under 40 years of age. Hence genetic will predisposition to CAD is questioned. LIPOPROTEIN(A) Lipoprotein(a) LP(a) was identified as a novel antigen by Kare Ingmar Berg Professor of medical genetics Norway in 1963. Lipoprotein little (a) is a heterogenous lipoprotein that shares many properties of low-density lipoprotein (LDL) and ten times as atherogenic as LDL. It consists of cholesterol rich LDL particle with one molecule of Apolipoprotein B100 (apoB100) and additional protein Apolipoprotein(a) [Apo(a)], attached by a disulfide bond on a 1:1 molar basis

**LIPOPROTEIN(A)-WHY IT IS DANGEROUS** The pathological processes associated with high levels of LP(a) start soon after birth or about 15-20 years earlier than other predisposing factors such as hypertension, cigarette, smoking, obesity and dyslipidemia it precis, the LP(a) strikes early.

**LIPOPROTEIN(A)-STRUCTURE** Lipoproteins better known as chylomicron are the biochemical assembly of tricyglycerols and cholesterol esters in the Core with an outer shell containing apoproteins and phospholipids. LP(a) is an atherogenic lipoprotein contain containing cholesterol rich LDL particles one molecule of ApoB100 and Apo(a) with Kringle IV type 2 repeats Apo(a) is a glycosylated protein and is composed of single peptide region repeating Kringle domain and a protease domain. There are different Apo(a) isoforms; over 500 that account for range of LP(a) molecular weight from 280 two 800 kDa

**ADDUCT LIPOPROTEIN (A) HYPOTHESIS** Apolipoprotein(a) is a sticky protein which act like a Patch of Velcro to normal Apo(a) is sites can bind to two neighboring LDLs and the LDL molecule hold a free docking site for another apo(a) protein thus forming giant Lp(a) aggregate. Small dense LDL with small apo(a) isoforms has highest atherogenic potential.

**LIPOPROTEIN(A) GENE – APOLIPOPROTEIN (A) POLYMORPHISM** LP(a) gene, located in chromosomes 6q26-27, codes for Apo(a) who's genetic heterogenicity is due to variations in the number of Kringle which in turns lead to size polymorphism (KIV-2VNTR) of Apo(a) a protein.

**ISOFORMS OF LIPOPROTEIN(A)** Depending on the size of Apo(a) 500 different isoforms of Lp(a) have been observed. Lp(a) size is Conversely related to plasma level, due to difference in hepatic secretion of Apo(a). More the Kringle repeats longer is the processing time for precursor apo protein. The outcome being small apo(a) isoforms secreted at a faster rate than the larger.

**ASSEMBLAGE DISTRUBUTION AND DEGRADATION** The Apo(a) and LDL particle assemble in the cell surface of hepatocytes. The Apo(a) is secreted in free form the endoplasmic reticulum which in turns buying to the cell surface. The ApoB100 containing lipoprotein complex binds to the cell surface bound Apo(a) causing conformational change releasing it from the cell surface. Half-life of Lp(a) in 3-4 days Lp(a) days is catabolized primarily by hepatic pathway update by LDL receptor adrenal pathway.

**Biography:** Sanjeev is a Medical Director from St Michael Hospital. Received latter appreciation & award from “Indian consulate, Philippines consulate, Poland consulate, Netherland consulate, Shanghai International school, Australian consulate Life member of the American Diabetic Association Life member of Indian Medical association

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