LIPID UND LIPOPROTEIN-METABOLISMUS

FREDRICKSON, D. S. (National Inst. of Health, Bethesda): **Function and Structure of Plasma Lipoproteins**

Manuskript nicht eingegangen

SEIDEL, D. (Med. Univ.-Klinik Heidelberg): **Plasma Lipoproteins in Patients with Familial Plasma Lecithin: Cholesterol Acyltransferase Deficiency: Apolipoprotein Composition of Isolated Fractions**

Autoreferat

Familial lecithin: cholesterol acyltransferase (LCAT) deficiency has recently been described as an inborn error of metabolism. The enzyme acts upon circulating lipoproteins and catalyses the transfer of fatty acid from the beta position of lecithin to the 3-β-OH group of free cholesterol. Besides characteristic changes of plasma lipid concentrations, deficiency of this enzyme is followed by changes of the physicochemical properties of the plasma lipoprotein fractions. Heterogeneity and abnormality with regard to the apolipoprotein composition are most pronounced in the low density fraction (d 1,063 to 1,21 g/ml). Three different subfraction can be isolated from this density class. a) lipoproteins containing only apolipoprotein B; b) lipoproteins containing apolipoprotein B and apolipoprotein C and c) lipoproteins consisting of apolipoprotein C, lipoprotein are very similar to the abnormal lipoprotein properties of the third (c) lipoprotein are very similar to the abnormal lipoprotein (LP-X) characterizing cholestasis. Since liver disease is often associated with low LCAT activity an important metabolic relationship may exist between structure of plasma lipoproteins, LCAT activity and liver function. (Coauthors: Drs. E. Gjone and J. P. Blomhoff, Oslo.)

BROWN, W. V. (University of La Hoya, Calif.): **Some Functional Aspects of the Plasma Apolipoproteins**

Autoreferat

Human plasma very low density lipoprotein (VLDL) and Chylomicrons (Chyl) contain the same protein as low density lipoprotein (LDL) and an additional group of small apolipoproteins of approximately 10,000 Molecular Weight. These smaller apolipoproteins constitute approximately 55% of the protein in VLDL and over 90% of the protein in Chyl. The two major constituents of this latter group of apolipoproteins are designated by their carboxyl terminal amino acids as apoLp-Glu (glutamic acid) and apoLp-Ala (alanine). Both have marked effects on the triglyceride lipase (TGL) of lipoprotein lipase. Using milk lipoprotein lipase, 1to 2 ug of apoLp-Glu produces a ten-fold activation of TGL activity. ApoLp-Ala inhibits both the milk and plasma TGL activities at levels above 50 ug/ml. The inhibition occurs at levels of apoLp-Ala greater than 2% of the substrate mass. No differences in these properties have been observed with apoproteins purified from normal subjects, or Type III und Type V hyperlipoproteinemics. The inhibition of apoLp-Ala of lipoprotein lipase may be blocked in vitro by certain lipid factors. The properties of these lipids which block apoLp-Ala inhibition are presently under study in our laboratory.