GENERAL SUMMARY

Alterations in thyroid function affect the activities of hepatic lipase (HL), lipoprotein lipase (LPL) and lecithin : cholesterol acyltransferase (LCAT) but in different ways. Low levels of thyroid hormones are associated with decreased activities of all three enzymes while increased concentration of thyroid hormones are accompanied by elevated activity of HL but not of LPL or LCAT. Besides quantitative alterations in LCAT activity changes in the lipoprotein substrate are of great importance for the endogenous cholesterol esterifying ability, which is decreased in hypothyroidism and increased in hyperthyroidism.

Plasma lipoprotein metabolism in hypothyroidism is characterized by an impaired degradation of triglyceride rich lipoproteins. Together with a defective elimination of LDL particles via specific LDL receptors, this leads to an increased concentration of lipoprotein particles in the LDL and the IDL fractions and of partly degraded VLDL particles. Also the eliminat on of cholesterol via the HDL system to the liver is impaired. This is reflected in an elevation of the concentration of the HDL_2 subclass although the total HDL cholesterol concentration is normal or only moderately increased.

Plasma lipoprotein metabolism in hyperthyroidism is characterized by an essentially normal degradation of triglyceride-rich lipoproteins. The cholesterol esterifying ability is increased. An increased activity of HL will facilitate the elimination of cholesterol from the HDL system. Along with an increased activity of the LDL receptor pathway, this will lead to decreased concentration of cholesterol mainly in the LDL, but also in the HDL fraction.

Of the different variables used to reflect thyroid hormone function (S-TSH, S-T_4, S-T_3 and free thyroxine index) S-T_3 turned out to give the best correlations to plasma lipoprotein concentrations and enzyme activities. This is in agreement with the view that tri-iodothyronine is responsible for the biological effects of thyroid hormones. Moderate but distinct disturbances of lipoprotein metabolism appear also in patients with moderate thyroid dysfunction, as in subclinical hypothyroidism.

There are no fundamental differences in the disturbances of lipoprotein metabolism in primary and secondary forms of hypothyroidism. The notion that the hypercholesterolemia in hypothyroidism of pituitary origin is less pronounced can probably be explained by the milder forms of thyroid hormone deficiency usually seen in secondary compared to primary forms of hypothyroidism.

Hyperthyroidism offers a possibility to study the effects on lipoprotein metabolism of a selective increase of HL activity. The findings in these patients are consistent with the concept that HL is a major determinant of HDL concentration in man.