

## INTRODUCTION AND AIM OF THE WORK

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Epidemiological studies have provided extensive evidence that the premature development of atherosclerosis and ischaemic heart disease is associated with elevated total serum cholesterol level (a reflection of increased LDL cholesterol) and decreased concentration of HDL cholesterol (Castelli et al. 1977; and Levy, 1983).

It is now apparent that the distribution of cholesterol in plasma is a complex function of both the relative and absolute concentrations of a variety of apolipoproteins.

Widespread studies have revealed that premenopausal women have lower levels of VLDL and higher levels of HDL than age -matched males (Kannel et al., 1976). They, also, experience a significantly lower risk of developing atherosclerosis (Gordon et al., 1977).

The decrease in estrogen that occurs following menopause is accompanied by an elevation in the ratio of VLDL: HDL and relatively abrupt increase in atherosclerotic risk. Taken together, the studies would suggest that in women normal physiological concentration of estrogen confers some protection against the development of atherosclerosis, while somewhat paradoxically the recent Framingham study has

linked elevated levels of estrogen in men with premature development of heart disease (Phillips et al., 1983). It is clear from other studies that higher concentrations of hormones, such as those found during pregnancy and during therapy with some types of oral contraceptives, can induce marked increase in VLDL: HDL ratio and result in hypertriglyceridemia (Barclay et al., 1965; Wynn et al., 1969).

So the aim of this work is to study the serum estrogen levels in patients with ischaemic heart disease and to correlate it with the different lipid parameters.