INTRODUCTION AND AIM OF THE [redacted]

INTRODUCTION AND AIM OF WORK

Serum lipoprotein (a) (Lp (a)) is a cholesterol-rich lipoprotein with structural feature of low density lipoprotein (LDL) and plasminogen that provides a link between atherosclerosis and thrombosis ***(Brown & Goldstein 1987, Utermann 1989).***

The apolipoprotein (apo) moiety of LP (a) includes apo B and apo (a). the latter being the antigenic determinant ***Gaubatz et al.(1987).*** Structurally apo (a) subunit contains multiple copies of kringle 4. a single copy of kringle 5 and a protease domain which are highly homologous to their plasminogen counterparts ***(Mclean et al., 1987).*** This suggests that Lp (a) may be involved in thrombogenesis ***(Jauhiainen et al., 1991)*** and could interfere with the process of plasminogen generation and inhibits fibrinolysis ***(Mclean et al., 1987 & Scanu, 1992).***

The serum concentration of Lp (a) is chiefly under genetic control ***(utermann et al., 1987).*** Although Lp (a) is synthesized independently of other apo B containing lipoproteins, its catabolism appears to follow the LDL pathways ***(Utermann, 1989). Scanu (1992)*** reported that the kidney could play a role in Lp (a) catabolism.

Numerous studies have indicated that high plasma Lp (a) are strongly associated with the development of atherosclerosis and coronary heart disease (CHD) ***(Genest et al., 1991; Jauhiainen et al., 1991; & Scanu, 1992).*** Lp (a) has been also detected from arterial lesions of CHD in a distribution detected from arterial lesions of CHD in a distribution

similar to LDL *(Armstrong et al., 1986).* R*hoads et al. (1986)* found it to be more atherogenic than LDL.

Watts et al. (1993). reported that Lp (a) is a risk factor for the presence of CAD in men. Independent of smoking, hypertension, diabetes, LDL and HDL cholesterol or apolipoprotein A1 and B levels.

Family studies revealed that Lp (a) is a common genetic disorder in patients with premature CAD and that it is highly heritable ***(Jacques et al., 1991).***

The pathological precursors of CAD begin in childhood. Prospective studies are needed to determine the level of Lp (a) in the prediction of CAD risk early in life. Relevant information concerning the future risk of disease can be obtained by comparing the relation of Lp (a) levels in children with the incidence and history of cardiovascular disease in their parents ***(Sathanur et al., 1991).***

Therefore this study aimed:

* To examine lipoprotein (a) levels in sera of sibling of myocardial infarction (MI) patients and to assess the presence of Lp (a) excess in that population.
* To examine the plasminogen activity and its relation to Lp (a) level.