INTRODUCTION

Rheumatic fever is an inflammatory disease that occurs as a delayed sequela to pharyngeal infection with group A streptococci ***(Bisno, 1993).*** Initial (primary) or recurrent (secondary) rheumatic fever does not occur without a streptococcal antibody response ***(Stollerman, 1964).***

Virtually every immune response, humeral and cellular, to all streptococcal antigens that have been studied shows a statistically significant increase in rheumatic subjects compared with non rheumatics recovering from streptococcal pharyngitis. The genetic control of immune response, however, and their strong association with certain HLA haptotypes in other rheumatic diseases make the issue of genetic predisposition still quite viable especially with regard to certain complications as mitral stenosis and chorea ***(Bisno, 1993).***

Several recent findings raise the possibility of a role for M protein in the pathogenesis of ARF. These include the existence of shared epitopes between M proteins of rheumatogenic group A streptococcal serotypes and human heart ***(Dale, 1985).*** The recognition of distinct structural differences between M proteins of rheumatogenic and non rheumatogenic types ***(Bessen et al., 1989),*** M protein functions as a superantigen capable of strongly activating broad range of T-lymphocytes ***(Bisno, 1993) (Tomai et al., 1990).***

Intercellular adhesion molecule (ICAM-1) also known as CD-54, is a cell-surface glycoprotein of endothelial cells. It is a member of the immunoglobulin supergene family of adhesion molecules, having five extra cellular immunoglobulin like domains of 100 amino acids ***(Bevillacqua, 1993).*** Serum levels of the soluble form of the intercellular adhesion molecule-1 (sICAM-1) have been advocated as a parameter of clinical