INTRODUCTION

INTRODUCTION AND AIM OF WORK

It has been suggested that the pathogenesis of minimal change nephrotic syndrome (MCNS) may be the result of disordered T-cell function (Shalhoub, 1974). Mitogen induced lymphocyte transformation is impaired in MCNS in relapse (Tomizawa et al.,1979; Sasdelli et al.,1980; Beale et al.,1980;and Fodor et al.,1982), and sera from these patients inhibit the transformation of lymphocytes from normal subjects (Moorthy et al.,1976; Beak et al.,1980;and Taube et al.,1981). When the patients enter remission, these abnormalities disappear (Tomizawa et al.,1979; Beale et al.,1980; and Fodor et al.,1982).

It has now been shown that the inhibitory effect of the nephrotic sera on normal lymphocyte function is not confined to MCNS, being found in other forms opposite to that reported by Moorthy et al.(1976) and Sasdelli et al.(1980). The inhibitory effect was of same degree as MCNS (Beale et al.,1980) or significantly less (Taube et al.,1981).

The impaired blastoid transformation in nephrosis was suggested to be due to an intrensic T-cell defect (Tomizawn et al.,1979; Sasdelli et al.,1980; and Fodor et al.,1982) and to a humoral factor circulating in

plasma (Moorthy et al.,1976; Tomizawa et al.,1979; and Sasdelli et al.,1980).

The inhibitory plasma factors were postulated to be hyperlipoproteinaemia (Beale et al.,1980; Taube et al.,1981; and Fodor et al.,1982), hypozincaemia (Reinold,1980; Fodor et al.,1982), and hypoalbuminaemia which proved to be of no significance (Fodor et al., 1982). Other unknown factors were suggested (Fodor et al., 1982).

The first purpose of this study, is to assess the T-cell function in nephrotic syndrome either corticosteroid responsive or resistant. The second aim is to investigate the effect of the immunosuppressive (Low density & very low density) lipoprotiens and the zinc concentration over the T-cell function.