INTRODUCTION

Pre-eclampsia is a disease occurring in the second half of pregnancy and is characterized by hypertension, proteinuria and often oedema, if untreated progress to eclampsia, a condition in which generalized convulsion occurs **(Chesley et al., 1968).**

It is now established that in normal pregnancy there is a maternal immune response to trophoblastic antigens that involves both cell mediated and humoral factors **(Maroni Parrot, 1973).** In pre-eclamptic toxaemia an immunological disturbance is suggested by studies showing correlation between the incidence of the disease and the degree of maternal/fetal histocompatibility **(Stevenson and Davidson, 1971).**

Immunoglobulin and complement have also been detected in the renal glomeruli of pre-eclamptic patients immediately after delivery **(Petrucco et al., 1974).**

Maternal antibody production to paternal transplantation antigens in a first pregnancy could be considered analogous to primary immunization, whereas subsequent pregnancies would elicit a form of secondary immune response in which the antibody titre would be higher than in the first pregnancy, part of the function of such antibodies might be to neutralize soluble trophoblastic antigens by forming antigen-antibody complexes which would be eleminated from the

circulation by the reticuloendothelial system. If the amount of antibody available were insufficient to combine effectively with soluble trophoblastic antigen, as might occur in a first pregnancy, pathogenic complement-fixing antigen-antibody complexes would be formed. Such complexes would predispose to the development of a type III hypersensitivity state. In support of this hypothesis, the clinical features of preeclamptic toxaemia are consistent with a vasculitic process similar to that seen in immune-complex diseases. Also, the incidence of pre-eclamptic toxaemia is higher in the first pregnancies, declining in subsequent pregnancies **(Thomson et al., 1976)**.

Pre-eclampsia is exclusive to pregnancy.An immunological basis for the disease has been suggested but not proven **(Jenkins, 1976)**. Unlike rhesus iso-immunization, which is the best-defined immunological disease of pregnancy and inapporpiate maternal hyperresponse state which becomes more common with each pregnancy,pre-eclampsia is most commonest in primigravidae. Although this differece discouraged immunological studies , pre-eclampsia has recently been thought to represent a form of homograft rejection -in this case of a natural graft - but there is little evidence to support this. However , previous blood transfusion or previous pregnancy may help to prolong renal homograft survival **(Van Hoof et al., 1976)**, moreover ,transfusion

before pregnancy is associated with reduced incidence of pre-eclampsia (**Feeny et al., 1977**).

The possibility that the aetiology of toxaemia might be immunologic has been held over 70 years. In the past decade numerous studies have been instituted in an attempt to verify the possible role of immune system in this disease (**Gusdon, 1977**).

Knox et al. (1978) stated that CICs have been proposed as possible important in the pathogenesis of pre-eclampsia.

Birkeland and Kristofferson (1979) suggested that the cause of pre-eclampsia may be due to combination of maternal and paternal hyporesponsiveness together with fetal hyperresponsiveness, they found an important immunochanges at the beginning of the second trimester after serial lymphocyte counts and function tests during and after preeclamptic pregnancies.

Pattillo (1980) stated that the genetic make-up of an individual may determine the capacity to combat successfully cancer and other diseases such as abnormal pregnancy, abortion, infertility and pre-eclampsia, these factors appears to be based upon the individuals immunogenetic.

Pre-eclampsia is usually a disorder in the first pregnancy. Previous pregnancies, even those ending in spontaneous or induced abortion, have been associated with

reduced frequency of pre-eclampsia. Also previous blood transfusion protects against its development **(Alanen and Lassila, 1982).**

Gleichner et al. (1982) reported that IgG immune complexes may represent the normal physiologic situation of pregnancy whereas IgM containing immune complex (I.C) may represent pathologic and therefore abnormal state.

Moore et al. (1983) reported that pre-eclampsia might result from a disorder in the maternal immune response to the paternally inherited fetal antigens.

Gusdon et al. (1984) have made an effort to determine whether or not there is any change in subpopulation of lymphocytes in normal pregnancy and pre-eclamptic pregnancies using monoclonal antibody markers. They found that no significant differences were noted in OKT_3, OKT_4, OKT_8, OKT_{11} or OKIa cellular population. However, **Baker et al. (1987)** disagree these findings, demonstrating a significant reduction in helper/suppressor ratio.

Bolis et al. (1987) stated that the aetiology of pre-eclampsia is so far unknown, neverthless both clinical and experimental findings suggest the possibility that immunogenetic factors may operate in this disorder.