INTRODUCTION

Calprotectin is a major calcium- and zinc- binding protein produced in myelomoncytic cells. By sequestering zinc, calprotectin has the potential to inhibit the growth of many zinc- dependant microorganisms and the activity of zinc- dependant enzymes. As well as the ability to induce apoptosis in both human and animal cells in vitro. Among the enzymes that can be inhibited is collagenase B (MMP-9), known to be important during angiogenesis and invasive tumour growth. Increased levels of calprotectin are found in plasma during bacterial infections and in inflammatory processes such as rheumatoid arthritis and systemic lupus erythematosus. It is detectable in body fluids and tissues and the fecal calprotectin level is found to represent a valuable marker of gastroenterologic pathology ***(Poullis et al. 2003).*** Recently, an elevated calprotectin level in amniotic fluid has been found to be associated with preterm labor and with intra-amniotic inflammation ***(Espinoza et al. 2003).***

Pre-eclampsia is a serious complication of pregnancy characterized clinically by maternal hypertension and proteinuria. The etiology of pre-eclampsia is still uncertain, but an insufficient trophoblast invasion into the maternal endometrium (decidua) leading to suboptimal development of the placenta with reduced placenta perfusion is assumed to be a predisposing factor for pre-eclampsia. The maternal symptoms are believed to be caused by maternal endothelial dysfunction associated secondary to release of

substances shed from poorly perfused placental tissue ***(Roberts et al. 1989)***.

Redman et al., 1999 have suggested that the endothelial dysfunction in pre-eclampsia is the consequence of an excessive systemic maternal response to pregnancy, including involvement of leukocytes as well as the clotting and complement systems. Infectious triggering has been proposed as a possible explanation for placenta pathology in pre-eclampsia ***(Von Dadelszen and Magee, 2002; and Trogstad et al., 2001).***

Several investigators have shown that pre-eclampsia is associated with maternal leukocyte activation and because calprotectin is derived predominantly from activated neutrophils and monocytes, it will be elevated in pre-eclampsia ***(Barden et al. 1997; Sacks et al. 1998 and Gervasi et al. 2001)***.