DIFFERENTIAL EXPRESSION OF PLASMINOGEN ACTIVATION CASCADE COMPONENTS IN HUMAN PRETERM DELIVERY WITH AND WITHOUT PRETERM PREMATURE RUPTURE OF THE FETAL MEMBRANES

***C. J. Nowell*[1,2], *P. Murthi*[1,2], *B. Kalionis*[1,2], *E. K. Moses*[1,2], *S. P. Brennecke*[1,2]**

[1]Obstetrics and Gynaecology, University of Melbourne, Carlton, VIC, Australia; [2]Department of Perinatal Medicine Pregnancy Research Centre, Royal Women's Hospital, Carlton, VIC, Australia

Previous studies from our laboratory have shown that plasminogen activation cascade (PAC) components are involved in the rupture of fetal membranes at term. The aim of this study was to test the levels of 4 key components of the PAC in fetal membranes collected from preterm deliveries (PTD) with and without preterm premature rupture of the fetal membranes (PPROM). In contrast to previous studies of the PAC in fetal membranes which have focused on labour at term, this study has examined PTD with PPROM (n = 10) and PTD without PPROM (n = 10).

Using immunohistochemistry, immunoblotting and zymography, we examined the expression and activity levels of key components of the PAC; plasminogen, PAI-2, uPA and uPAR for expression and plasminogen, plasmin and uPA for activity. The data presented here show significant changes in the expression and activity of PAC components in PTD + and – PPROM samples. When compared to term labour control samples both PTD + and – PPROM show increased expression and activity of PAC components. When PTD + and – PPROM are compared to each other differences in expression and activity can be seen. uPA expression and activity increases in PTD – PPROM, relative to term delivery samples, and increases further in PTD + PPROM samples. Immunohistochemical analysis reveals the uPAR is expressed at very low levels in the amnion epithelium, basement membrane and mesenchyme/intermediate zone of PTD + PPROM but is highly expressed in PTD – PPROM.

These data demonstrate differential expression of the PAC in PTD + PPROM cases compared with PTD – PPROM cases, which suggests different aetiologies and/or mechanistic pathways for the two types of PTD. These differences may be of importance in defining optimal clinical management of the two situations.

10.1071/SRB04Ab287