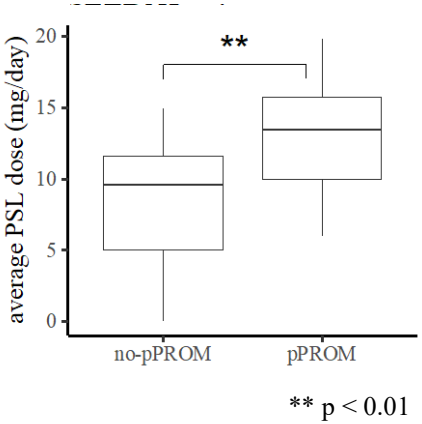
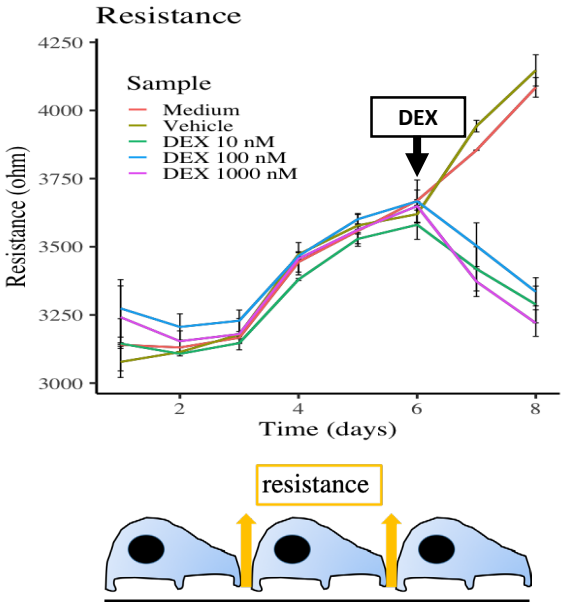
From Laboratory of Maternal-Fetal Medicine

Glucocorticoids increase the risk of preterm premature rupture of membranes (pPROM) possibly by inducing *ITGA8* gene expression in the amnion

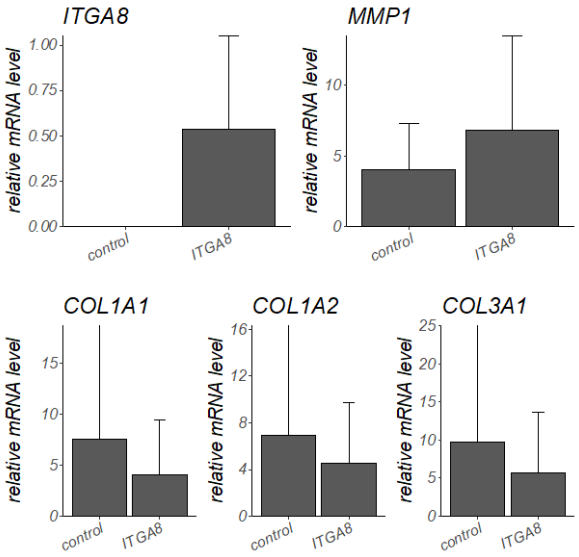
➢ The average prednisolone dose was higher in the pPROM group than in the no-pPROM controls.



➢ The resistance decreased in human amniotic mesenchymal cells treated with dexamethasone (DEX), which represents glucocorticoids weakened the cell-cell connections.



➢ Over expression of glucocorticoid target gene *ITGA8* decreased the collagen levels and increased *MMP* levels In human amniotic mesenchymal cells.



Based on the association between glucocorticoids and pPROM, we showed that glucocorticoid exposure weakened the cell-cell connections in human amniotic mesenchymal cells, and the glucocorticoid target gene *ITGA8* could be a primary molecule that triggers pPROM through collagen degradation in fetal amnion.

References: Yuka Okazaki, *et al.*, Placenta. 2022; 128: 73-82

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