Progestins Effect on an Endometriotic Cell Line

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Endometriosis is a chronic inflammatory disease which affects up to 1 in 10 women of reproductive age. It occurs when endometrial cells grow outside the uterus and is categorised based on the location of ectopic lesions, namely: ovarian, peritoneal, and deep infiltrating endometriosis. The exact cause of endometriosis is yet to be determined.

Nevertheless, for the last 50 years progestins (synthetic analogues of progesterone) have been used to treat endometriosis. Progestins are known to be anti-inflammatory by inhibiting the function of certain immune cells and affecting the balance of pro- and anti-inflammatory cytokines. However, the pharmacological action of progestins in endometriosis is not completely understood.

Previous unpublished data from our laboratory has identified IL-1, IL-10 and OPG signalling pathways involved in progestins mechanism of action. The aim of this study was to further assess the role of progestins on the pro-inflammatory IL-1 signalling pathway. We examined the effect of progesterone and three progestins (Medroxyprogesterone acetate (MPA), Dydrogesterone (D) and Dienogest (DNG)) in a model cell line of peritoneal endometriosis (12-Z). We utilised Real Time-qPCR to assess expression of 6 genes involved in the IL-1 signalling pathway. We found that gene encoding IL1 receptor (*IL1R*) was upregulated by MPA and DNG whereas repressors of IL-1 signalling pathway, *IL1R2* and *IL1RN,* were upregulated by all progestins. Genes *IL1A/B* showed changes depending on the progestin, and expression of IL1R1 co-receptor *IL1RAP* was not affected by any condition. Although progestins showed different effects on expression of the investigated genes, up-regulation of *IL1R2* and *IL1RN* suggest that all progestins decrease pro-inflammatory IL-1 signalling in endometriotic cell line 12-Z.

 ISBN: 978-81-954993-6-6; DOI: 10.21467/abstracts.125