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EFFECTS OF HUMAN CHORIONIC GONADOTROPIN ON ENDOMETRIAL STROMAL CELLS DECIDUALIZATION

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Synchronized biochemical cross-talking between embryo and endometrium is critical for successful embryo implantation. One of the first embryo-secreted factors which signal endometrium about embryo presence is human chorionic gonadotropin (hCG). It was shown that hCG contributes to endometrium decidualization, differentiating tissue to a receptive state, and regulates maternal immunotolerance of the embryo and vascularization of the tissue [1-3].

This study analyzed how hCG affects endometrial stromal cell properties depending on its concentration *in vitro*. For endometrial stromal cells' decidualization, it was used established in vitro decidualization protocol by using db-cAMP and MPA. hCG effect in different concentrations was evaluated alone and in combination with decidualization stimulus.

Firstly, hCG and decidualization effects for cellular metabolic activity and viability were assessed. Gene expression analyses were conducted to evaluate hCG impact for decidualization markers (*IGFBP1*, *PRL*), apoptotic factors (*BAX*, *BCL2*, *BAK1*), DNA methylation regulators (*TET1*, *TET2*, *TET3*), and signaling molecules (*IL-11*, *IL-6*, *IL-1β*). Finally, ELISA was performed to confirm the results for prolactin. Results revealed that decidualization induction together with hCG increased the metabolic activity of cells, however, more prolonged exposure with hCG decreased it. Moreover, decidualization markers gene expression analysis revealed that an additive exposure with hCG slightly elevated these genes' expression compared to cells treated only with decidualization stimulus.

In conclusion, we demonstrated that hCG affects endometrial stromal cells and influence decidualization process in dose- and time-dependent manner.

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