Effects of Vitamin D Metabolites and Vitamin K2 on Menstrual Effluent-Derived Endometrial Stromal Cell Viability and Decidualization

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Endometriosis is associated with infertility in 30% to 50% of the millions affected worldwide. Decidualization, a key endometrial differentiation process that facilitates embryo implantation, is dysregulated in patients with endometriosis. While recent studies using natural supplements Vitamin-D and Vitamin-K2 have shown promise in female reproductive health, their direct impact on regulating decidualization remains unknown. This study aims to close this gap, investigating the individual and synergistic effects of Vitamin-D metabolites (calcitriol, calcifediol, and cholecalciferol) and menaquinone-4 (MK-4), a Vitamin-K derivative, on decidualization. Cryopreserved menstrual effluent-derived endometrial stromal cells (ME-eSCs) obtained from control participants (n=15) in the Research OutSmarts Endometriosis (ROSE) Study were cultured and treated with vehicle, MK-4, calcitriol, calcifediol, or cholecalciferol and then, decidualized using dibutyl cyclic adenosine monophosphate and medroxyprogesterone. Cell supernatants were analyzed by ELISA for decidualization marker IGFBP-1, and cells were analyzed by neutral red for cytotoxicity. Low MK-4, calcifediol, calcitriol, and cholecalciferol doses were not cytotoxic, whereas Calcitriol ($p<0.01$) and calcifediol ($p<0.05$) significantly increased IGFBP-1 production, suggesting enhanced decidualization. While synergistic effects between Vitamin-D/MK-4 were not observed in this study, future studies should explore menaquinone-7 (MK-7), a derivative with a longer biological half life. These findings support the potential therapeutic efficacy of calcitriol and calcifediol in promoting the decidualization of ME-eSCs, possibly ameliorating defects, and identifying therapeutic targets for endometriosis-associated infertility.

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