Aldo-keto reductase activity after DEHP exposure in eutopic and ectopic endometrial cells: possibility of biomarker.

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Endometriosis is gynecological disease in reproductive age women, cause pelvic pain, dysmenorrhea and infertility. Endometriosis is a multifactorial disease and exact pathophysiology is still unveiled.
The molecular characteristics of endometriosis tissue are high prostaglandin in tissue and progesterone resistance. Human aldo-keto reductases are known to involve both mechanisms. They convert progesterone to the less potent metabolite, lead progesterone resistance and catalyze prostaglandin biosynthesis. In this study, we hypotheses the expression of aldo-keto reductase genes in human ectopic endometrium and eutopic endometrium would be altered expression by DEHP exposure, and used microarrays and western blot to study effects of DEHP. We also expect to check the possibility of aldo-keto reductase as an detection marker of endometriosis using Western blot and ELISA of aldo-keto reductase. For this, we cultured human endometrial cells from normal endometrium from women without endometriosis (NE), eutopic endometrium from women with endometriosis (EE) and ectopic endometrium from women with endometriosis (EC).
In Results, NE, EE and EC showed different genetic expression change after DEHP treatment. In EE, genes which changed its expression significantly after DEHP exposure were over 10 ~ 20 times more than in NE endometrial cells. DEHP leads up-regulated expression change in AKR1C1, AKR1C2, AKR1C3 and AKR1B10 of EE. AKR1B1 showed no expression change after DEHP exposure in NE, EE and EC. EC shows no additional gene expression change after DEHP, but show continuously increased expression of AKR1C3 protein in western blot, before and after DEHP exposure. This result suggests the possibility of AKR1C3 as a pathophysiologic gene of endometriosis. In ELISA data, serum AKR1C3 was significantly elevated in patients with endometriosis during secretory phase, not during proliferative phase. As conclusions, DEHP induced AKR activity in endometrium of endometriosis patients, and endometriosis patients showed significantly elevated AKR1C3 enzyme level during secretory phase.

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Authors :
References : , , ,

Authors

HyunHee HyunHeeCho 1,

1. Obgy, Catholic university medical colleage, Seoul, KOREA, REPUBLIC OF

Authors (raw format)

HyunHeeCho HyunHee - email : drrabbit@catholic.ac.kr Institution : Catholic university medical colleage Department : Obgy City : Seoul Country : KOREA, REPUBLIC OF Speaker : Yes