

Adverse Effects of EDCs on Female Reproductive Organs

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Editorial

Endocrine Disrupting Chemicals (EDCs) are exogenous chemical substances or mixtures that interfere with synthesis, secretion, transport, metabolism, binding action, or elimination of natural endocrine hormones that are present in the body [1]. EDCs are widespread and one of component in persistent pesticides, herbicides, biocides, heat stabilizers, and chemical catalysts, plastic contaminants, pharmaceuticals, or dietary materials. Many EDCs are weakly estrogenic and elicit their actions through the estrogen receptors. The two mammalian receptors for estrogen (ER- α and ER- β) are widely distributed throughout the female reproductive tract [2,3]. Therefore, actions of estrogenic EDCs on estrogen receptors may promote abnormalities in estrogen receptor-present female organs such as ovary, uterus, breast, and placenta.

Recently we examined the effect of parabens that were known to have an estrogenic property on the development of ovary [4]. In this study, female pups were given with methyl-, propyl- and butylparabens during neonatal day 1 to 7. Parabens inhibited early phase of folliculogenesis in the ovaries by regulating expression of folliculogenesis associated genes, AMH, Foxl2, and KITL. In addition, the steroidogenesis in the ovary was also altered by parabens through reduction of StAR and Cyp11a1 gene expression. Since parabens are widely used as anti-microbial agents in cosmetic industries, these results suggest that maternal use of cosmetics containing parabens may cause abnormality in folliculogenesis of female infant [5,6].

The effects of EDCs on the uterus that is a major female reproductive organ also studied in our previous studies [7]. The uterus is composed of an endometrium, a muscular myometrium and an outer serosa layer. There are a number of studies showing that EDCs promote endometriosis by increasing proliferation of endometrial cells [8]. However, impact of EDCs in myometrium layer was not understood. In our previous study, we administered immature female rats with bisphenol A, and octylphenol that are well known estrogenic phenolic compounds. The tested EDCs increased expression and localization of contraction-associated proteins including oxytocin receptor and prostaglandin F receptor, and thereby adversely regulated contractile activity of muscular myometrium in the uterus. Since uterine contraction is related with physiologic function of uterus, exposure to bisphenol A and octylphenol may cause reproductive disorders such as myometriosis, leiomyoma, dysmenorrhea, infertility, preterm labor, abortion, and delayed labor [9-14].

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