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Effects of 4-*tert*-Octylphenol on Transcription of Steroidogenic Enzyme in Mouse Testis

Kim Suel-Kee[1,3], An Su-Yeon[1], Kim Myo-Kyung[2], Ko Duck-Sung[2], Yoon Yong-Dal[3] and Lee Ho-Joon[1]

[1]Department of Physiology, [2]Eulji Researh Insitute, Eulji University School of Medicine, [3]Department of Life Science, Hanyang University

Environmental estrogens (xenoestrogen) are synthetic compounds that are abundant in the environment and are known to mimic or inhibit the function of endogenous hormone. 4-*tert*-octyphenol (OP) has been shown to reduce reproductive hormone production and disturb testicular development. However, its precise mechanism for the impairment of reproductive system are virtually unknown. This study was designed to evaluate the effects of OP on steroidogenesis and feedback system in male mice. We identified the changes of gene expression of StAR, SF-1 and P450 isoenzymes such as CYPscc, $CYP_{17\alpha}$, and CYP19. Postnatal (15-day-old) and adult (8-week-old) mice were injected with OP (2, 20, and 200 mg/kg) for five days. No morphological abnormalities were detected in adult testis, however, reduced Leydig and Sertoli cells were observed in mice postnatally exposed to OP. A significant decrease occurred in the expression of StAR, CYPscc, and $CYP_{17\alpha}$ in postnatally exposed mice but not in adult mice. In parallel, serum testosterone concentration were reduced only in mice postnatally exposed to OP. These results suggest that OP induced-inhibition of testosterone production is related to decreases in the expression of StAR, CYPscc and $CYP_{17\alpha}$ and Leydig and Sertoli cells are more sensitive to OP during pubertal differentiation than in adulthood. The expression of SF-1 was not changed in all the treated groups. As the expressions of estrogen and androgen receptor were not changed in all the groups, OP may exerts its action indirectly to these receptors. In conclusion, OP treated at low concentration and during short exposure time to adult mice did not affect steroidogenesis and differentiation of testis. However, postnatal treatment of OP can severely reduce the expression of StAR and several P450 enzymes and decrease the production of testosterone. This impairment of steroidogenesis has an adverse affect on development and differentiation of Leydig and Sertoli cells and reproductive system in male mice. (This study was supported by grant 2001-ED-15 from KDFA)