Studies on the Effects of Environmental Estrogens on Spermatogenesis

XIN Zhong Cheng, M.D.

Andology Center of Peking University Hospital Beijing, China, 100034

It was noted that male's sperm parameters decreased significantly in last fifty years and there are 15% of married couples suffered from infertility and 50% of them are caused by male factor. Studies on etiological bases on male infertility, there are more than 30% of patients were difficult to identify the etiological causes.

Due to rapidly development modern technical and industrial engineering, the adverse effects of environmental population on man's health has been investigated in worldwide. In was hypothesized that endocrine estrogens and particularly synthetic estrogenic environmental contaminants (xenoestrogens) could be an important etiologic factors in the global decrease of human sperm counts. Bases on these hypotheses studies showed that environmental estrogen could adversely affected testicular function, including androgen secretion and spermatogenesis, by pre-testicular, testicular and post-testicular mechanism; 1) studies on pre-testicular target showed that Bisphenol A (BPA) ,an environment estrogen, were exposed to rats suppressed serum LH and testosterone levels, the inhibition of testicular steroidogenesis by the xenoestrogen BPA is associated with reduced pituitary luteinizing hormone secretion. 2) Studies on testicular target showed that selective Leydig cell damage is well known in animal experiment with the exposure to environmental estrogen, but there was no human counterpart of this model exhibiting selective Leydig cell toxocity. An animal study showed that HPTE causes a direct inhibition of testosterone biosynthesis by Leydig cells at all stages of development. Beside the endocrine effects of xenoestrogens, they also have directly effect on spermatogenesis. Study found that elimination of spermatogenic cells via apoptosis occurs spontaneously under normal physiologic conditions and is often aggravated after chemical estrogen induced testicular impairment. There is still no research about the direct toxicity of environment estrogen on sertoli cell, but xenoestrogen exposure alters expression of SF-1/Ad4BP secreted by sertoli cell in the fetal rat testis. 3) Studies on post-testicular target showed that most post-testicular toxicity has been discovered during the search for novel chemical contractors for men. The polyamine biosynthesis in the hamster epididymis can be affected by DES, a xenoestrogen. Administration of the xenoestrogen, nonylphenol, to male newborn rats could disruption of male reproductive tract development. PCBs and PEs may be instrumental in the deterioration of semen quality in infertile men without an obvious etiology. However, the xenoestrogen p-nonylphenol has no biochemical effects at low dosages on epididymal markers in rats exposed. Although it was found that some xenoestrogen adversely affected testicular function, however, the detailed mechanism need more reaches to illustrate.

The related toxicity biomarker and look for methods of prevent and treatments for environmental estrogen on spermatogenesis still need more and more effort to be search and it will be an important project for future study.