

Triclosan and estradiol effects on human prostate cells

Mariana Di Lorenzo¹, Angela Lucariello², Maurizio Forte³, Giuseppina Iachetta¹, Vincenza Laforgia¹, Maria De Falco¹, Antonio De Luca²

¹ Department of Biology, Section of Evolutionary and Comparative Biology, University of Naples, Naples, Italy

² Department of Mental and Physical Health and Preventive Medicine, Section of Human Anatomy, Second University of Naples, Naples, Italy - ³ National Institute of Biostructures and Biosystems, (INBB), Rome, Italy

Xenoestrogens are estrogen-mimicking compounds that are commonly found in personal care products and pesticides. The activity of xenoestrogens in the human body involves interference with estrogen receptor binding. Triclosan (TCS), a lesser-known xenoestrogen, is a broad-spectrum antibacterial commonly used in cosmetics, toothpastes, soap and other consumer products. The widespread use of TCS and its detection in human breast milk, urine and serum have raised concerns regarding its association with various health outcomes. Recent evidence suggests that TCS may play a role in cancer development, perhaps through its estrogenicity. In the present work we have studied the effects of TCS and Estrogen (E2) on human prostate adenocarcinoma epithelial cells (LNCaP) in order to highlight estrogen and xenoestrogen influence on human prostate. Although androgens are the most important hormones in the normal development of the male reproductive system, more recently, it has been suggested a central role for estrogen in male reproductive system and it has been hypothesized that high level of estrogens may disturb the endocrine control of the male reproductive capability. We examined the effects of TCS and E2 on the proliferation of the LNCaP through MTT assay. They were both able to increase cell proliferation at concentration of 10^{-8} M after 24h of treatment. In order to study estrogen receptor (ER) involvement, we evaluated the cellular localization and expression of ERs with immunofluorescence and western blot techniques after treatment with TCS and E2. Finally, through Real Time PCR analysis we have investigated gene expression of several molecular targets of estrogen pathway. We have observed that treatment with TCS and E2 induced an upregulation of Ki-67, cyclin D1 and cyclin E. We have also observed an upregulation of proinflammatory cytokines $IL-1\beta$ after TCS and E2 treatment. These results confirm the estrogenic activity of TCS and suggest that estrogen and xenoestrogens may interfere with molecular pathways of prostate physiology.