Derica Stone, Biology

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Faculty Mentor: Dr. Fred vomSaal, Biological Sciences
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Bisphenol A alters messenger RNA expression in prostate mesenchyme cells

Exposure to abnormal levels of estrogen and estrogen-mimicking chemicals during fetal life can alter proper development of tissues. These alterations are initiated during fetal life and include increased adult predisposition to cancer, heightened tendency towards obesity, earlier onset of puberty in females, decreased sperm count in males, and affected normal tissue growth. The prostate is a tissue that is sensitive to alterations in sex hormone levels during development. The development of this tissue is androgen-dependent, but can also be influenced by estrogens. Experiments have shown that fetal exposure to estrogen and estrogen-mimicking chemicals alters prostate growth in mice, but the molecular mechanism by which this occurs is still unclear. In order to answer the above question, this study looked at alterations in gene expression in the mesenchyme of the developing prostate due to exposure to different amounts of estradiol (E2) and the estrogen-mimicking chemical bisphenol A (BPA). The mesenchyme tissues were collected from male fetuses on gestation day 17. These cells were cultured and treated with varying concentrations of E2 and BPA. We examined gene expression of the androgen receptor (AR), estrogen receptor (ER), and insulin-like growth factor-1 (IGF-1). Over the dose range studied, BPA caused an increase in AR (1.6 fold), ER (2.85 fold) and IGF-1 (2.5 fold). E2 increased AR (2.2 fold) and ER (3.36 fold) gene expression, but did not effect IGF-1 gene expression. Our findings show how development of the prostate may be regulated by alterations to estrogen levels in the body. They also show how environmental chemicals can have a disruptive effect on tissue by inducing changes in the expression of certain genes.