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Estradiol Dependant Molecular And Biochemical Changes in Human Spermatozoa

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ABSTRACT

Many of the physiological effects of biphenyl A (BPA) have been described in the context of its ability to interact with classic estrogen receptors. BPA can have estrogenic activity, but it should not be considered to act only as an estrogen or even as a selective estrogen receptor modulator. The available data show that BPA's biochemical and molecular interactions are complex, involving classic estrogen receptors as well as a variety of other receptor systems and molecular targets. The complex of BPA's interactions and concentration ranges at which the observations have been made make it challenging to conclude whether a given in vivo finding is biologically plausible based on consistency and potency of a response compared with estradiol alone.

KEYWORDS:

Introduction

Few toxicological studies on mammals have been performed on dyes specific to leather industry and no specific conclusion could be made concerning the impact of these dyes on human health. However, many of the dyes are aromatic amines-a class of compound comprising a large number of suspected carcinogens. Chromium is produced from chrome tanning process and subsequent in wet processing steps, trivalent chrome which is the only form normally present. Hexavalent chromium in the tanneries is practically non-existent. Chromium can produce lung tumors when inhaled and induces skin sensitization. Large dose of chromate have corrosive effects on the intestinal tract and cause inflammation of kidney. A minimum level of chromate ion that has no effect on man has not been determined. The toxicity of chromium salts to fish and aquatic life varies widely with the species, temperature, pH, valency of chromium and synthetics or antagonistic effects, especially those of hard water. Fish appears to be relatively plerant to chromium. Many other harmful chemicals are used in tanneries and there are multiple effect or these harmful chemicals on biological system. It is necessary to know their mode of extent and extent of toxicity particularly under varied physiological ad environmental conditions.

Literature Review

Considering the current evidence and what has been mentioned, it is probable that the changes in hormonal levels that occur *in vivo* in the reproductive tract of the female of each species during the reproductive cycle subtly regulate the AR, retarding the onset or favoring its timely occurrence. As regards this delicate hormonal modulation that AR could be subject to, it is necessary to mention again the case of steroid hormones, about which more information is available: in the cervix, the estradiol present in the per ovulatory cervical mucus could be exerting the role of AR inhibitor since its onset in such regions of the female reproductive tract would not make fertilization possible (Ceric et al., 2005; Vigil et al., 2008; Vigil et al., 2009b). The inactivation of the nucleus, and thus the absence of transcription, confers the male gamete an advantage as a study model for the non-genomic action of steroid hormones. For this reason, the knowledge obtained from experiments on spermatozoa could explain the non-genomic response of somatic cells as regards these and other types of hormones.

The variations in the concentrations of cytoplasmic calcium, cAMP and the phosphorylation of protein residues are currently some of the non-genomic effectors of steroid hormones. Such signaling components have been described both in somatic cells and in spermatozoa (Baldi et al., 2009). On the contrary, in the distal third of the Fallopian tube, there are high levels of progesterone coming from the follicular fluid, which could promote AR precisely when the Spermatozoa and the oocyte are close to encounter, favoring successful fertilization (Morales et al., 1992; Vigil et al., 2008; Vigil et al., 2009c).

CHROMIUM: Chromium is produced from the chrome tanning process and subsequent wet processing steps, trivalent chrome is the only form normally present. Since hexavalent chrome used in tanneries is practically nonexistent. Chromium can produce lung tumors when inhaled and induces skin sensitization. Large dose of chromate have corrosive effects on the intestinal tract and cause inflammation of the kidney. A minimum level for the chromate ion that has no effect on man has not been determined. The toxicity of chromium salts to fish and aquatic life varies widely with the species, temperature, pH, valence of the chromium and synergistic or antagonistic effects, especially those of hard water.

SULFIDES: This is introduced in the form of sharpeners in the unhearing process. Sulfide compounds are used extensively in beam houses for the unhearing process and these are found in tannery effluent.

NITROGEN AND AMMONIA: Both nitrogen forms are generated in conjunction with the unhearing and bating processes. There are evidences that chromium expert toxic effects on all aquatic lives depending upon the pH, dissolved oxygen level and the total concentrations of nitrogen in the water.

PHENOL: Phenol and substituted phenolic are generated mainly in conjunction with coloring of tanned hide. These sources may include biocides (e.g., pentachlorophenol 2, 4, 5- trichorophenol), synthetics and natural vegetable tannins, carriers for dyes, and number of other adverse sources, all of which contribute to significant level of phenol in raw waste waters. Phenol acts as nerve poison, causing too much blood to get in to the gills and to the heart cavity. The human ingestion of a concentrated phenol solution results in severe pain, renal irritation, shock and possible death.

DYES: No positive conclusion could be made concerning the impact of leather industry dyes on human health and it is well known that they are suspected carcinogens e.g. basic orange 2, direct blue 14 etc.

RESEARCH METHODOLOGY: Physiology of Estrogens It is well known that estrogens, such as 17 β - estradiol (E2), play pivotal roles in the regulation of sexual development and fertility in both males and females (Couse and Korach, 1999a; Couse and Korach, 1999b; Nef and Parada, 2000; O'Donnell et al., 2001). Estrogens also regulate metabolic processes in fat, liver, and bone tissues (DeCherney, 1993; Vaananen and Harkonen, 1996). In addition to these roles in normal physiological processes, estrogens also play pivotal roles in many disease states. For example, estrogens can act as potent mitogens in some cancers (e.g., breast, uterine) causing hormone-dependent growth and proliferation (Foster et al., 2001; Prall et al., 1998; Sommer and Fuqua, 2001).A variety of synthetic estrogen antagonists ("antiestrogens") have been developed and are used clinically to reverse the mitogenic action of estrogens in estrogen - dependent cancers (e.g., Tamoxifen, Tam; Raloxifene, Ral). Interestingly, these same compounds may have estrogen – like agonistic activities in some tissues (e.g. , bone, endometrium), functioning more like tissue - or cell type specific "selective estrogen

receptor modulators" (SERMs) than pure antagonists (Harper and Walpole, 1967; McDonnell et al., 2002; Paech et al., 1997; Webb et al., 1995). Gaining a greater understanding of the molecular actions of estrogens and SERMs will aid in the development of new compounds that are even more effective in the treatment of breast cancers.

Estrogen Receptors the molecular actions of estrogens are mediated through estrogen receptor (ER) proteins which bind the hormones, dimerize, and regulate the transcription of estrogen responsive genes. ERs exist as two isoforms, ER α and ER β (Warner et al., 1999) which are members of a conserved super family of nuclear receptors that function as transcription factors (Mangelsdorf et al., 1995). These is forms have ER dependent Transcriptional Regulation in the Nucleus: Direct DNA Binding Upon hormone stimulation, ERs dissociate from nuclear chaperone proteins, dimerize, and bind to DNA sequences known as estrogen response elements (EREs). Estrogen dependent transcriptional regulation through the ERE pathway involves a variety of cofactors that fun ction with liganded ERs to modify histones, alter chromatin structure, and recruit the RNA polymerase II (Pol II) transcriptional machinery (Kraus and Wong, 2002).

Summary

Many of the physiological effects of BPA have been described in the context of the ability of the active aglycone form to interact with classic E Rs. BPA can have estrogenic activity, but it should not be considered to act only as an estrogen or even a SERM. Depending on the system studied and the dose, BPA may exert pleiotropic cellular and tissue-type specific effects and can exhibit non-monotonic dose–response relationships at cellular and intracellular levels. When BPA acts as a ligand of the nuclear ERs, the influence on responsive genes is not identical to that of endogenous estrogens (e.g. E2) or other natural or synthetic ligands (e.g. DES). Comparison of gene-centric data for BPA with those of E2 and two potent estrogenic compounds (EE and DES) lends additional support for this conclusion. In one study, the transcriptomal signature profiles of MCF7 cells were compared following a 48-hour incubation with E2 at 30 pmol/l or BPA at 10 nmol/l ; mRNA levels of a similar number of genes were changed following treatment with BPA (2102 genes) and E2 (2164 genes), but only 668, or approximately 30%, were affected in common.

A large number of in vitro studies have helped elucidate specific molecular interactions of BPA in cell systems. In vitro studies summarized in Wetherill et al. (2007) used female reproductive tissue (LOECs 0.0001–0.1 µmol/l), breast cancer cells (LOECs 0.0001–1 µmol/l), male reproductive tissue (LOECs 0.0001–150 µmol/l), pancreatic/adipose tissue (LOECs 0.0001–10 µmol/l), pituitary tissue (LOECs 0 .000 001–1 µmol/l), neural cells or tissues (LOECs 0.000 000 1–2.5 µmol/l), immune cell s (LOECs 0.0001–10 µmol/l) and embryonic cultures (LOECs 0.1–1 µmol/l). The estrog enic potency of BPA ranges over about 8 orders of magnitude but is generally 1000-fold le ss than that of positive control estrogens in vitro and 1000- to 10 000-fold less based on in vivo models (Chapin et al., 2008).

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