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DETRIMENTAL EFFECTS OF BISPHENOL A ON DEVELOPMENT AND FUNCTIONS OF THE MALE REPRODUCTIVE SYSTEM IN EXPERIMENTAL RATS

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ABSTRACT

Bisphenol A (BPA) is widely used in manufacturing industries. It is commonly detected in the environment and was reported to exert oestrogenic effects which may be harmful to the reproductive system. The present study was carried out to observe the effects of oral administration of BPA on the development of the reproductive organs and plasma sex hormone levels in prepubertal male Sprague-Dawley (SD) rats. Prepubertal male SD rats (n=8 in each group) were administered BPA in the doses of 1, 5, 10 and 100 mg/kg BW (body weight) via oral gavage for a period of 6 weeks. The control animals received the vehicle for BPA (Tween 80 in distilled water). Following 6 weeks of BPA exposure, the rats exhibited less evidence of spermatogenesis. There was seminiferous epithelial damage which included disruption of intercellular junctions and sloughing of germ cells into the seminiferous tubular lumen. Furthermore, the lumina of the seminiferous tubules and the epididymis of these animals were filled with immature germ cells and cellular debris. This damage may lead to the significant reduction in the seminiferous tubular diameter in BPA-treated animals. These findings were associated with the significant lower plasma testosterone and 17β-oestradiol levels. There was no significant difference between the body weight gain, the absolute as well as relative testis weight or epididymal weight of BPA-treated animals when compared to the control animals. The findings provided further evidence of the detrimental effects of BPA on the male reproductive system.

Keywords: Bisphenol A, testis, anatomy, spermatogenesis, testosterone, 17β-oestradiol

INTRODUCTION

Various endocrine disrupting chemicals (EDCs) are commonly used in daily life. These chemicals are also present in the environment and prove to be harmful to human and animals. The environmental contaminants affect normal functions of the endocrine and reproductive systems either by mimicking or inhibiting endogenous