Original Article

Bisphenol A-induced morphological alterations in Sertoli and spermatogenic cells of immature Shiba goats in vitro: An ultrastructural study

BIBIN BINTANG ANDRIANA,1,3* TAT WEI TAY,1 RYUJI HIRAMATSU,1 MOHAMMAD ABDUL AWAL,1 YOSHIAKIRA KANAI,1 MASAMICHI KUROHMARU1 and YOSHIHIRO HAYASHI2
1Department of Veterinary Anatomy, 2Department of Global Animal Resource Science, Graduate School of Agricultural and Life Sciences, The University of Tokyo, Tokyo, Japan and 3Faculty of Animal Husbandry, Bogor Agricultural University, Kampus Darmaga Bogor, Indonesia

Background and aims: There is no information currently available regarding the effects of bisphenol A (BPA) on testes in ruminants. Therefore, to establish and clarify the effects of BPA in ruminants, testicular tissue cultures were obtained from immature Shiba goats.

Methods: The testes of 2-month-old Shiba goats were cut into smaller pieces and seeded in medium. At 1, 3, 6 and 9 h after administration of various concentrations of BPA, the specimens underwent light and transmission electron microscopic observations.

Results: At 1 h after BPA treatment, vacuolization and nuclear membrane rupture appeared within the nucleolus and cytoplasm of Sertoli cells. Such alterations tended to gradually increase in number in time- and dose-dependent manners. Thus, because of BPA treatment, apoptotic spermatogenic cells, necrotic spermatogenic cells, apoptotic Sertoli cells and necrotic Sertoli cells could be identified. Particularly in the Sertoli cell: ruptured vesicles could be found within the multivesicular nuclear body.

Conclusion: The treatment with BPA at a low concentration tends to lead spermatogenic and Sertoli cells to apoptosis, whereas a higher concentration tends to lead spermatogenic and Sertoli cells to necrosis. Therefore, this study showed that testicular tissue culture is an advantageous avenue for screening the testicular toxicity of chemicals in ruminants. (Reprod Med Biol 2004; 3: 205–210)

Key words: Bisphenol A, Sertoli cell, spermatogenic cell.

INTRODUCTION

Many chemicals with different structures have been shown to exhibit weak estrogen-like functions.1 Various groups of environmental chemicals have estrogenic activity.2 Several studies have proven that various environmental chemicals may act as endocrine disruptors, most often as hormone antagonists. Some endocrine disruptors, such as pesticides, plasticizers, and raw materials for polycarbonate and epoxy resins, are discharged into the environment and bring unexpected unfavorable effects on wildlife and human beings.3–4 Bisphenol A (BPA) behaves like a weak estrogen in classic bioassays, exhibiting such signs as cellular proliferation and cornification of the vaginal epithelium.5 BPA is a monomer of polycarbonate plastics and epoxy resins.6 Polycarbonate is used in a wide range of plastic products, such as toys and livestock tools, and it leaches out from such products at a rate that increases with repeated use.7 For example, one report showed that BPA was always released from a plastic apparatus into one reagent during repeated autoclaving.8 Presently, plastics manufacturers in the USA have the capacity to produce over a billion pounds of BPA.9

Estrogen interacts with other steroid hormones to regulate the normal development of the reproductive system and other tissues.3 Many chemicals, including endocrine disruptors, have a binding affinity to estrogen receptor (ER) α and ER β.1 Plastics and pesticides are examples of products that contain an estrogenic endocrine-disrupting chemical, which can interfere with mammalian development by mimicking the action of the sex hormone estradiol.1

Most of the research regarding endocrine disruptors has been carried out in vivo (in rodents, such as rats,