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Oxidative and Hormonal Disruptions Underlie Bisphenol A: Induced Testicular Toxicity in Male Rabbits

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Abstract: The presence of endocrine-disrupting compounds, such as bisphenol A (BPA), in the environment can cause serious health problems. However, there are controversial opinions. This study investigated the reproductive, metabolic, oxidative and immunologic-disrupting effects of bisphenol A in male rabbits. Rabbits were divided into five groups. The first four rabbit groups were administered oral BPA (1, 10, 50, or 100 mg/kg/day) for ten weeks. The fifth group was administered corn oil as the vehicle. BPA significantly decreased serum testosterone, estradiol and the free androgen index (FAI) and significantly increased sex hormone binding globulin (SHBG) compared with the placebo group. The higher doses of BPA showed a significant decrease in follicular stimulating hormone (FSH) and luteinizing hormone (LH). A significant increase in blood glucose levels was identified in the BPA groups. The non-significant difference in insulin levels is a novel finding. The cumulative testicular toxicity of BPA was clearly demonstrated by the dose-dependent decrease in absolute testes weight, primary measures of semen quality and a significant increase in testicular malonaldehyde (MDA). Moreover, BPA significantly decreased total antioxidant capacity (TAC) and significantly increased immunoglobulin G (IgG) at the highest concentration. Our results suggest that BPA, especially at higher doses, is associated with many adverse effects on metabolism, oxidative stress, immunity, sperm quality and markers of androgenic action. These results may reflect the estrogenic effects of BPA, which we hypothesize could be related, in part, to an inhibitory effect on testicular steroidogenesis. The induction of oxidative stress by BPA may play an additional role in testicular toxicity. These results suggest that BPA poses a threat to endocrine and reproductive functions.

Keywords: bisphenol A, oxidative stress, rabbits, semen quality, steroidogenesis

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