6 CONCLUSIONS

6.1 Exposure to bisphenol A (2 and 20 mg/ Kg body weight) for 7 days affects the epididymal functions of rats as evidenced by the decrease in sperm motility and sperm count.

6.2 Exposure to bisphenol A (0.2, 2 and 20 mg/ Kg body weight) for 4 and 7 days induces oxidative stress as evidenced by the increase in the levels of hydrogen peroxide and lipid peroxidation in testis, epididymal sperm and epididymis of rats.

6.3 Exposure to bisphenol A induces oxidative stress in liver of rats only at the dose of 20 mg/ Kg body weight whereas this doses does not induce oxidative stress in the kidney of rats.

6.4 Administration of low doses of bisphenol A (0.2, 2 and 20 µg/ Kg body weight) to adult rats for 30, 45 and 60 days affects epididymal sperm functions as evidenced by decreased sperm motility and sperm count.

6.5 Administration of low doses of bisphenol A (0.2, 2 and 20 µg/ Kg body weight for 60 days) causes necrosis and derangement of seminiferous tubule in testis and complete degeneration of epithelium in epididymis, seminal vesicles and ventral prostate of rats.

6.6 Administration of low doses of bisphenol A (0.2, 2 and 20 µg/ Kg body weight for 45 days) decreases the serum levels of LH, FSH, testosterone and estradiol while the levels of serum prolactin remains unchanged.

6.7 Administration of low doses of bisphenol A (0.2, 2 and 20 µg/ Kg body weight for 30, 45 and 60 days) induces oxidative stress in testis, epididymis,
epididymal sperm and liver of rats as evidenced by the decrease in the activities of antioxidant enzymes. However, bisphenol A could induce oxidative stress in kidney of rats only at the doses of 2 and 20 µg/ Kg body weight following 60 days of treatment.

6.8 Co-administration of bisphenol A along with vitamin C imparts protective effects against bisphenol A-induced toxicity.