7 CONCLUSIONS

7.1. Low doses of BPA does not cause general systemic toxicity in adult male rats while long-term administration decreases the weights of testis, epididymis and accessory sex glands.

7.2. Low doses of BPA decreases the activities of antioxidant enzymes and induces oxidative stress in the testis of rats. Antioxidant enzymes viz. superoxide dismutase, catalase, glutathione peroxidase and glutathione reductase have favorable binding pockets for interactions with BPA. But catalase has the highest binding affinity among these enzymes.

7.3. Short-term administration of low doses of BPA does not affect cell integrity of testis whereas long-term exposure to BPA and estradiol causes loss of germ cells in the testis of rats.

7.4. Administration of low doses of BPA decreases the activities of steroidogenic enzymes and inhibits steroidogenesis in the testis of rats.

7.5. Short-term administration of low doses of BPA enhances glycolysis in the liver of rats whereas long-term administration downregulates glycolysis and upregulates glycogenolysis.

7.6. Short-term administration of low doses of BPA induces hyperinsulinemia and hypoglycemia in rats while long-term administration causes hyperglycemia possibly due to insulin resistance.

7.7. Short-term administration of low doses of BPA does not affect glycolysis in the testis of rats while long-term administration downregulates glycolysis.
7.8. Exposure to low doses of BPA downregulates insulin signaling molecules and glucose transporters, GLUT-2 and GLUT-8, in the testis of rats. Molecular docking studies confirm that BPA can directly interact with GLUT-2 and GLUT-8 proteins and inhibit their functions.

7.9. Exposure to low doses of BPA activates Fas and mitochondria-mediated apoptotic pathways in the testis of rats.