Chapter 6

Summary of the results

1. BPA impairs the haematopoietic mechanism in rat by decreasing the number of red blood cells and haemoglobin concentration. Besides, it is also seen that the mean corpuscular haemoglobin (MCH) increases in BPA treated rats. The insufficient number of red blood cell or insufficient haemoglobin content per RBC with higher MCH may result in the occurrence of macrocytic anemia.

2. BPA impairs the blood metabolic variables linked to cardiovascular function through enhancing the levels of calcium, glucose, total protein and lipid profile, susceptible to cardiovascular disease.

3. BPA depresses the cardiac function through promoting the oxidative stress in the heart ventricular tissue by inhibiting the activities of antioxidant enzymes like SOD, CAT, GPX and GR and enhancing the degree of peroxidation of membrane lipids. Besides, it has also been observed that the maximum duration of ‘observable oxidative stress effects of BPA’ is 5 days in 20 days treatment duration and the minimum duration for the ‘no observable effect’ on oxidative stress is 10 days in the same treatment duration.

4. In order to know the oxidative stress induced damage of the ventricular myocytes, cytoarchitectural examination has been done. Degeneration of sarcolemma, intercellular detachment by the formation of lacunae, morphological changes in nucleus have been seen in the heart ventricular tissue of BPA exposed rat. From the result it has been suggested that BPA depresses the cardiac function presumably by promoting oxidative stress induced cardiac injury.

5. To confirm the BPA induced cardiac damage, the activities of transaminases, the cardiac damage marker enzymes, have been studied. The enzyme activities were increased significantly in BPA treated rat. The results suggest that BPA inhibits the cardiac function by causing the damage of cardiac myocytes.

6. To examine the involvement of acetylcholine (ACh) in the BPA induced cardiac depression, the activity of acetylcholinesterase (AChE) has been assayed. It has been seen that BPA inhibits the AChE activity in cardiac cell membrane at the ACh binding sites. The result suggests that BPA in part depresses the cardiac function by inhibiting the activity of AChE and thus prolongation of the action of ACh in the cardiac tissue.
7. To understand experimentally the involvement of probable pathway behind the action of BPA on cardiac function the pharmacodynamic study has been carried out. The results suggest that BPA depresses of heart function principally by promoting the production of NO and activating the NO-linked downstream second messenger pathway in the cardiac cells.

8. To examine the availability of free calcium in the ventricular myocytes of BPA exposed animals the calcium histochemical examination has been performed. It has been observed that calcium was deposited at the external side of the ventricular wall as calcium salt in the matrix of the ventricular tissue of BPA treated rats. The results revealed that BPA depresses the cardiac function as a result of the reduction in the availability of free Ca\(^{2+}\) in the ventricular myocytes by causing chelation of Ca\(^{2+}\) and inducing the formation of Ca\(^{2+}\) plaques in the cardiac cell.

9. To examine the ameliorative effect of antioxidant vitamin C on the BPA induced depression of cardiac function, the role of Vitamin C on the oxidative stress linked cardiac depression has been studied. It has been seen that Vitamin C significantly counteracts the BPA induced oxidative stress linked inhibition of heart function.

**Figure 6.1:** Probable mechanism of toxic effects of BPA on haematological and cardiovascular functions in animals which may lead to the occurrence of cardiovascular stress. The role of vitamin C in counteracting BPA induced toxic effect has been mentioned.