Chapter 1

Introduction

Bisphenol A, abbreviated as BPA, is a man-made organic compound with two phenol functional groups. It is the building block of several plastics and plastic additives. The commercial use of the monomeric form of BPA in the production of polycarbonate plastics and epoxy resins (Burridge E, 2003) and as a non-polymer additive to other plastics (Chitra KC et al., 2002; Hernandez RG et al., 2007) is increasing enormously. Polycarbonate plastics due to its clarity and toughness has been in use to make various types of day to day use products like water bottles, baby feeding bottles, soft and hard drink containers etc. Besides, BPA is widely used to manufacture other plastic products like- eyeglass lenses, medical equipments, CDs, DVDs, electrical equipments, sports safety equipments and many more household appliances. Epoxy resin is used for lining metal cans to maintain quality of canned food and beverages due to its chemical resistance. However, recent studies have revealed that BPA leaches from the protective internal epoxy resin coatings of canned foods and from consumer plastic products into the food contents (Larroque M et al., 1998; Fielding M et al., 1999). The degree of leaching of BPA from polycarbonate bottles into its content has been found to depend more on the temperature of the liquid or bottle, than the age of the container. Repeated washing of the bottle with harsh detergent also enhances the leaching process (Hunt PA et al., 2003). Besides, polymers made from BPA can be hydrolyzed in high temperature, acidic and basic conditions, leading to leaching and migration of BPA into the internal food and beverage contents (Biles JE et al., 1997; Brotons JA et al., 1995; Brede C et al., 2003; Factor F et al., 1996; Nerin C et al., 2003; Tan BL et al., 2003; Welshons et al., 2006).

Widespread and continuous human exposure to BPA occurs mainly through dietary intake (Stahlhut RW et al., 2009), with additional exposure through dental sealant, dermal exposure and inhalation of indoor and outdoor dusts (Olea N et al., 1996; Matsumoto H et al., 2005; Rudel RA et al., 2010). In recent times BPA is considered as a controversial chemical for its dual characteristics regarding toxicity. It exerts multi-system toxicity in animal models. It has been reported that BPA exerts its toxicity on the neurological system by causing adverse effects on fetal and infant brain development and behavior, process of learning and memory etc. (Bucher J et al., 2012; Richter C et al., 2007; Ogiue IM et al., 2008). The mode of action of BPA appears to mimic that of the female hormone estrogen. Therefore, BPA can be classified to a
group of chemicals termed ‘endocrine disruptor’ that disrupts the chemical messenger system in the body. Thus, BPA can be said as a man made endocrine disrupting chemical.

Scientists have reported that BPA can affect haematological system of animals by impairing the structural and functional properties of cellular components of blood (Ha MH et al., 2008; Sajiki J, 2003; Fang X et al., 2011; Furukawa F et al., 1994). Besides, BPA exerts its toxicity on the pancreatic β-cells, adipose tissue by causing metabolic disorders (Miyawaki J et al., 2007; Ropero AB et al., 2008).

Further, BPA is reported to cause oxidative stress induced damage of the liver, kidney and testis of experimental animals by producing reactive oxygen species (ROS) or by inhibiting the activities of antioxidant enzymes related with the scavenging of the ROS (Bindhumol V et al., 2003; Kabuto H et al., 2004; Mourad IM et al., 2012). From other studies it has been seen that the co-administration of Vitamin C with BPA ameliorates the BPA induced damage of the organs of male rats (Korkmaz A et al., 2010; Aydogan M et al., 2008; Bindhumol V et al., 2003; Chitra KC et al., 2003; Murmu S et al., 2011).

Moreover, it has been reported that BPA alters the function of coronary smooth muscle by activating Maxi-K (KCa.1.1) channels (Asano S et al., 2010). Besides, the studies of Pant J and associates have revealed that BPA causes depression of the atrial contractility in rat through NO-dependent guanylyl cyclase signaling pathway (Pant J et al., 2011). Further the study of Liang Q et al reported that BPA progressively increase sarcoplasmic reticulum (SR) Ca\(^{2+}\) release and Ca\(^{2+}\) reuptake and inhibited the L-type Ca\(^{2+}\) current (I\(_{\text{CaL}}\)) in cardiac myocytes (Liang Q et al., 2014). The effect of BPA on the function of heart and cardiovascular variables had not been reported clearly at the cellular level before to undertake the project. The present study has been undertaken to examine the effect of BPA on some core haematological and cardiovascular variables in rat and toad models. Results obtained from the study reveal that BPA impairs the haematological and cardiovascular functions by (i) decreasing the red blood cell (RBC) count and haemoglobin (Hb.) content and increasing mean corpuscular haemoglobin (MCH) content in whole blood; (ii) increasing the levels of serum calcium, glucose, total protein and lipid profile susceptible to cardiovascular disorder; (iii) enhancing the activities of transaminases; and (iv) depressing the movement of heart presumably by (a) reducing the availability of free Ca\(^{2+}\) within the ventricular myocytes, (b) promoting oxidative stress induced damage of the heart ventricular tissue, (c) inhibiting the activity of acetylcholinesterase (AChE) in cardiac cell membrane at acetylcholine (ACh) binding sites; and (d) facilitating the relaxation of cardiac myofibrils through nitric oxide mediated pathway.


