Bisphenol A (BPA) is a synthetic organic compound. Chemically it is one of the derivatives of phenols. It is used commercially to produce polycarbonate plastics to make water and baby bottles, impact-resistant safety equipments, medical devices etc. and epoxy resins to make inner coating of the cans used to store food items and drinks (soft and heavy) (NTP, 1982; Inoue et al., 2003; Biles et al., 1997; Erickson et al., 2008; Biedermann et al., 1998). It has been studied that humans are exposed to the BPA as a result of the consumption of BPA-tainted foods, water and drinks. Some dental sealants and composites may also play as important sources of BPA for human exposure (Biles et al., 1997; Olea et al., 1996; Faccioio et al., 2002). The possible health hazards of BPA have been studied in some animal models as well as in humans. The endocrine disrupting function of BPA has been reported by some researchers (Gould et al., 1998; Kuiper et al., 1998; Pennie et al., 1998; Wetherill et al., 2007; Alonso-Magdalena et al., 2005; Maruyama et al., 2013; Cao et al., 2013; Melzer et al., 2011). The reproductive, developmental, behavioral and neurotoxic effects of BPA in laboratory animal models have also been reported by some research groups (Le et al., 2008; Lang et al., 2008; Lee et al., 2007; Vom Saal et al., 2005; Okada et al., 2008; Paris et al., 2002; Choi et al., 2007; Nakazawa and Ohno, 2001; Miyatake et al., 2006; Honma et al., 2006; Miyagawa et al., 2007; Tanabe et al., 2006; Noguchi et al., 2002; Golub et al., 2010; Sall et al., 2012; Zhong et al., 2013; Kong et al., 2013). The role of BPA in inducing oxidative stress in different organ tissues and the efficacy of vitamin C to counteract the BPA induced oxidative stress in tissues have been studied discriminately by some scientists (Kabuto et al., 2003; Aydogan et al., 2008; Bindhumol et al., 2003; Nakagawa et al., 2000; Chitra et al., 2003; Rashid et al., 2008). Further, the effects of BPA on the functions of heart have been reported by few researchers (Pant et al., 2011; Yan et al., 2011; Aboul et al., 2013; Asano et al., 2010). From the study of Pant et al., 2011 it has been reported that BPA depresses the atrial contractility presumably through NO-mediated signal pathway.
Humans are predominantly exposed to BPA through dietary consumptions of canned food and beverages. In that case gastrointestinal mucosae are directly exposed to BPA. The gastrointestinal tract serves two major functions-one is digestive and the second one is motor. The gastrointestinal motility causes the movements of ingested food stuffs from stomach to intestine for digestion in sequence and absorption of digested food stuffs from intestine to the intestinal blood vessels and lymphatic ducts. The principal motor of the intestinal wall is the smooth muscle, arranged circularly and longitudinally in the muscular layer of the intestinal wall. The motor functions of the smooth muscle are locally controlled by Myenteric (Auerbach’s) plexus and centrally by sympathetic and parasympathetic efferent fibres that innervates the Myenteric central neural pool. There was no report about the possible toxic effects of BPA on the motor function of the intestine before to undertake this study. Therefore, the aim of the present study was to examine the effect of BPA on the function of intestinal smooth muscle in rat model at the molecular physiological level. In order to examine the effect of BPA on the motor function of the intestine, the movement of duodenum (as representative of intestine) has been studied in ante-mortem and post-mortem treatment experiments. Reports of the work have already published in journal and conference proceedings since 2011.

Results obtained from this study reveal that, BPA exerts toxic effects on the motor function of the intestine presumably by promoting the relaxation of the intestinal smooth muscle principally through nitric oxide-mediated and in part by α-adrenergic signal pathways. At the cellular level, BPA produced the activation of the acetylcholinesterase (AChE), over expression of the nitric oxide synthase (NOS), facilitation in the deposition of chelated free intracellular Ca^{2+}, and induction in oxidative stress along with an increase in the degree of membrane lipid peroxidation in intestinal smooth muscle. The effect of vitamin C in the counteraction of the BPA induced oxidative stress in intestinal smooth muscle has also been studied. Vitamin C showed significant counteraction in the BPA-induced oxidative stress in intestinal smooth muscle cells.