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Melatonin administration ameliorate testicular mitochondrial oxidative damage caused by bisphenol A in adult mice

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Bisphenol A (BPA) monomer of polycarbonate plastic used to manufacture plastic baby bottles and lining of food cans. BPA is also used in dental fillings and sealants. Has endocrine-disrupting potential and exerts both toxic and estrogenic effects on mammalian cells. The aim of this study was to investigate if BPA induced oxidative stress and toxicity in the testicular mitochondria of adult male mice, is ameliorated by co-administration of melatonin. Mice exposed to standardized dose of BPA (10 mg/kg body weight), orally for 14 days caused lipid peroxidation (LPO) and decrease in reduced glutathione (GSH) content of testicular mitochondria. BPA also caused decrease in activities of marker mitochondrial enzymes such as succinate dehydrogenase, malate dehydrogenase, isocitrate dehydrogenase. Besides, it also affected activities of antioxidant enzymes such as superoxide dismutase, glutathione reductase and glutathione peroxidase. Concomitant melatonin administration (10 mg/kg body weight; intraperitoneally for 14 days) lowered mitochondrial lipid peroxidation. It also restored the activity of mitochondrial marker enzymes and ameliorated decreased enzymatic and non-enzymatic antioxidants of mitochondria. Melatonin acts as an antioxidant. These results demonstrate the prowess of melatonin in ameliorating BPA-induced mitochondrial toxicity and the protection is due to its antioxidant property or by the direct free radical scavenging activity.

Biography

Since joining the ecotoxicology lab, dept of toxicology for Ph. D Sameya Anjum has been involved with studies related to toxic effects of environmental contaminants on human beings, also on the climate change. Sameya conducted tests and studies to detect and determine changes caused by endocrine disruptors on mice model. Also studied the protective effect of melatonin.

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