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Long-term exposure of Bisphenol A impairs metabolism and develop insulin resistance

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Abstract

Bisphenol A (BPA) is a well-recognized endocrine disruptor which has wide applications in current age. This is an industrial compound which is being used in the preparation of juice cans, water containers and various other household plastic products. The main objective of this study was to investigate the mechanism of BPA-induced metabolic pathways disturbance and other key risk factors that can may lead to the development of impaired insulin secretion from β -cells of pancreatic islets and insulin resistance peripheral tissues. The methodology involved the oral administration of BPA in different doses i.e., 50, 500, 2500 and 5000 for 3 months. The predefined BPA induced metabolic risk factors that lead towards the insulin resistance were measured by using ELISA kits. The association of long-term exposure of BPA with, significant reduction ($P < 0.05$) of antioxidant enzymes, such as superoxide dismutase (SOD), glutathione (GSH), catalase (CAT) and, considerable higher levels ($P < 0.05$) of inflammatory mediators, including tumor necrosis factor- α (TNF- α), interleukin-6 (IL-6), can exacerbate the chances of insulin resistance and impaired insulin secretion. Furthermore, we found a significant correlation ($P < 0.05$) between the long-term exposure of BPA and various enzymes that control the metabolism of carbohydrates such as α -glucosidase, glucose-6-phosphatase, hexokinase, α -amylase. The same correlation ($P < 0.05$) was also found to be existed in between the enzymes involved in the biosynthesis of cholesterol such as HMG-CoA reductase and long-term exposure of BPA. It can be concluded that long-term exposure of BPA can disrupt the various metabolic pathways by inducing the oxidative stress which provokes various inflammatory responses that ultimately lead to impair the β -cells of pancreatic islets and insulin resistance peripheral tissues.

Keywords: Bisphenol-A, oxidative Stress, metabolic pathways, inflammatory response, insulin resistance.

Abbreviations:

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