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Impact of Vitamin C Supplementation in BPA Induced Swiss Albino Mice (*MUS MUSCULUS*)

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Abstract: ***This study aimed to evaluate the effects of Bisphenol A BPA and Vitamin C supplementation on oxidative stress levels in the blood of Swiss Albino mice. The study measured the expression of key antioxidant enzymes and assessed the preventive effects of Vitamin C on oxidative damage. The results indicated that BPA caused cellular dysfunction and increased the production of reactive oxygen species. However, supplementation with Vitamin C, both immediately and for a prolonged period, significantly reduced the damage caused by oxidative stress. These findings highlight the potential of Vitamin C as a preventive measure against BPA-induced oxidative stress.***

Keywords: BPA, Oxidative stress, Vitamin C , Antioxidants

1. Introduction

In recent years, considerable attention has been focused on endocrine disrupting compounds and their impacts on the environment and human health, raising questions about their levels of exposure. Bisphenol A (BPA) is one of the world's highest production volume chemicals (Ritter, 2011)used in polycarbonate plastics in many consumer products and epoxy resins lining of food and beverage containers .BPA is a kind of potential endocrine disruptor. The human population is continuously and inevitably exposed to low doses of BPA in daily life (Schoen felder, 2010; Carwile and Michels, 2011) Importantly, BPA has been detected in amniotic fluid, cord blood and human breast milk, which demonstrates the potential of this compound to pass from mother to fetus (Groff, 2010). Animal experiments showed that prenatal BPA exposure resulted in abnormal glucose metabolism in future life (Alonso-Magdalena *et al*.**,** 2010; Batista *et al*.**,** 2012).

The global population is subjected to repeated exposure to BPA, primarily through packaged food but also through drinking water, dental sealants, dermal exposure and inhalation of household dusts (Lakind and Naiman, 2008) with detectable concentration of metabolites in the urine of > 90% of the population worldwide (Calafat *et al.*,2008). Heat, repeated washing of polycarbonate products and contact with either acidic or basic compounds accelerate hydrolysis of the ester bond linking BPA molecules in polycarbonate plastics and resins resulting in an increase in the rate of leaching of BPA (Lim *et al.*, 2009). In addition, another potential source of human exposure to BPA is water used in plastic covers for drinking or bathing as per studies conducted in Japan (Kawagoshi *et al*.**,** 2003) and in the United States.

Several studies reported the occurrence of oxidative toxicity after BPA exposure in rats and mice (Chitra *et al*.**,**2003; Gong and Han, 2006). BPA can cause liver, kidneys, brain, and other organs injury by forming Reactive oxygen species (ROS). Moreover, the study of (Bindhumol *et al*.**,** 2003) revealed that low doses of BPA generate ROS by decreasing the activities of antioxidant enzymes and increasing lipid peroxidation thereby causing oxidative stress in liver of rats. ROS are cytotoxic agents causing oxidative damage by attacking cell membrane and DNA. The liver has a range of antioxidant defense system.ROS are scavenged by the endogenous antioxidant defense system, including superoxide dismutase (SOD), catalase (CAT) and reduced glutathione (GSH) in cells. When the capacity of this antioxidant system decreases, the level of inactivated ROS rises. Antioxidants are scavengers by produced cell and tissue that could be expected to result in cellular damage and disease.

Vitamin C is considered one of the most important dietary antioxidant in biological systems due to its association with cell membrane and its ability to act directly on reactive oxygen species (ROS) preventing peroxidation. Besides the well characterized function of vitamin C as antioxidant alternative roles such as that of a membrane stabilizer, and a regulation of membrane fluidity have been proposed. The protective role of vitamin C on the oxidative stress develops due to antileprosy chemotherapy in leprosy patients was recorded. The present study is carried out to evaluate the impact of vitamin C supplementation on the oxidative stress during BPA administration.

2. Materials and Method

2.1 Animals

This study was conducted on 24 apparently healthy male and female Swiss Albino mice (*Mus musculus*) aged between 8 to 12 weeks with an average body weight of 32 to 40 grams. Lighting 12 hour light and 12 hour dark cycle. Animals were kept under the controlled temperature at 23°C degree celsius ± 2 degree celciusand relative humidity (55% +5%) conditions. Animals were acclimatized to the laboratory conditions. The animals were fed on certified pelleted rodent food, purchased from, Thansi Pets Super Stores, No. 22, South Mada street, Kolathur Chennai- 600 099.

2.2 Experimental Design

The rats were divided into 6 groups comprising of 6 animals in each group.
Group I: Control-sham operated animals (C)
Group II: BPA induced (by oral) for 30 days in animals-Control (EC)
Group III: BPA was induced to animals for 30 days ,Thereafter, 31 to 60 days Vitamin C was given.
Group IV: BPA was induced to animals for 1 to 3 days and immediately after 4 th to 34 day BPA and Vitamin C was given.

The study was carried out to assess the oxidative stress, antioxidant enzymes, Glutothione S Transferase (GST), Glutothione peroxidase (Gpx), TBARS, Super oxide dismutase (SOD), Catalase (CAT) in the BPA induced animal blood and under the influence of BPA induced and vitamin-C capsule which was mixed with water for administration.

2.3 BPA induction: (Nimisha Balakrishnan and Sendhilvadivu M , 2016) BPA was given to the albino mice orally (20mg/ kg body weight) for 30 days.

2.4 Vitamin C supplementation (Wilson Magdy*et al*., 2016)
Mice were fed with Vitamin C (200 mg/kg body weight).

2.5 Sample Preparation

The experimental animals were sacrificed by cervical dislocation at the end of the appropriate experimental period. The various experimental groups viz, Group – I Control (C), Group – II Experimental Control (EC) BPA induced animals, Group – III BPA induced animals for 30 days and from 31 to 60 days Vitamin C was given. Group – IV BPA induced animals for 1 to 3 days and immediately after 4 th to 34 days BPA and Vitamin C was given. The mice were dissected after the experiment and heart was punctured and the blood samples were collected for analysis.

2.6 Oxidative Stress Analysis

Oxidative damage was analysed by the levels of antioxidant enzymes present in the blood samples of mice by measuring the levels of TBARS, Glutathione S Transferase (GST), Glutathione Peroxidase (GpX), Super oxide Dismutase (SOD) and Catalase (CAT).

2.6.1. Glutathione S-Transferase (Habig *et al*, 1974)
Glutathione S Transferase (GST) enzyme was assayed in all the blood samples of mice viz C,EC,ET vitC(31 to 60days), ET vitC immediate (4^{th} to 34 th day) by using the method of Habig *et al.*,(1974).

2.6.2. Glutathione Peroxidase (Rotruck *et al*, 1973)
Glutathione Peroxidase (GpX) enzyme was assayed in all the blood samples of mice viz C,EC,ET vitC(31 to 60days), ET vitC immediate (4^{th} to 34 th day) by using the method of Rotruck *et al.*,(1973).

2.6.3. Tbars (MDA)
TBARS (MDA) was measured in all the blood samples of mice viz. C, EC,ET vitC(31 to 60days), ET vitC immediate (4^{th} to 34 th day) treated animals by using the method of Ohkawa (1979).

2.6.4 Super oxide dismutase (EC. 1.151.1)
Super- oxide-dismutase enzyme was assayed in all the blood samples of mice viz. C, EC,ET vitC(31 to 60days), ET vitC immediate (4^{th} to 34 th day) treated animals by using the method of Beauchamp and Fridovich (1973).

2.6.5 Catalase (EC. 1.11.1.6)
Catalase was measured in all the blood samples of mice viz. C, EC,ET vitC(31 to 60days), ET vitC immediate (4^{th} to 34 th day) treated animals by using the method of Chance and Machly (1955).

3. Statistical Analysis

The statistical analysis of the various parameters of the present study in all animal groups was carried out. Student 't' test was conducted to test the difference between two sample means by using given formula as suggested by Gupta (1978).

4. Results

The present study aimed to evaluate the oxidative stress effect of BPA in blood whether co-administration of vitamin C in both prolonged (30 Days BPA, 31 – 60 days Vitamin – C) and immediate (1-3 days BPA, 4 to 34 th day BPA and Vitamin C) form can ameliorate this oxidative damage in albino mice.

In the Experimental Control animals (EC) after administration of BPA without any treatment, The activity level of Thiobarbituric acid (TBARS) reactive substances was significantly elevated to- 219.78 %when compared to that of Control (Table I). The activity level of antioxidant enzymes in Group – II viz. Glutathione S transferase (GST), Glutathione Peroxidase (GPx), Super oxide dismutase (SOD), Catalase (CAT), was found to be reduced to 88.33%, 82.09 %,81.60 %, 51.03 % respectively in the BPA induced albino mice when compared to that of Control (Table-I).

After supplementation with Vitamin C by both immediate and prolonged treatment, Group – III – (Treatment BPA 30 days), After inducing the mice with BPA, They are supplemented with Vitamin C from 31 – 60 days. The results on antioxidant enzymes revealed significant improvement in the levels of GST, GPx, SOD and CAT in the Blood Samples of Vitamin C fed BPA induced albino mice, the elevation of activity levels of enzymes ranges 58.76 %, 46.57% and 48.84%. and 6.20% (Table-II). The TBARS value has a significant decrement 40.70%indicating lowered lipid peroxidation in the BPA induced albino mice blood supplemented with Vitamin C. (Table II).In Group – IV (Immediate Treatment group), BPA was administered from 1 to 3 days followed by 4th to 34 days BPA + Vitamin C was given. The results on antioxidant enzymes revealed significant improvement in the levels of GST, GPx, SOD and CAT in the blood samples of Vitamin C fed BPA

induced albino mice, the elevation of activity levels of enzymes ranged 35.04 %, 20.74 % and 39.72 %. and -14.48 % (Table-II). The TBARS value has a significant decrement 48.53 % indicating lowered lipid peroxidation in the BPA induced albino mice blood supplemented with Vitamin C.

5. Discussion

Impacts of BPA in the blood have been demonstrated by (Tyl *et al*., 2008 and Al-Griw *et al*., 2022). There are various routes of human exposure to this substance such as oral, by inhalation and transdermal. The main sources of exposure to BPA include food packaging and dust, dental materials, healthcare equipment, thermal paper, toys and articles for children and infants. BPA is metabolized in the liver to form bisphenol A glucuronide and mostly in this form is excreted with urine.

Vitamin C is an important antioxidant known to decrease damaging effects of reactive oxygen species. The study was aimed to explore the potential role of vitamin C as an antioxidant on Bisphenol (BPA) induced oxidative stress in mice.

Vitamin C (ascorbic acid) is an important non-enzymatic antioxidants. Human and other primate are incapable to synthesizing Vitamin C, therefore it has to be gained from diet. Fruits and vegetables are a prominent dietary source of vitamin C for human and animals. Vitamin C is an essential micronutrient for humans, with pleiotropic functions related to its ability to donate electrons. It is a potent antioxidant and a cofactor for a family of biosynthetic and gene regulatory enzymes. Vitamin C contributes to immune defense by supporting various cellular functions of both the innate and adaptive immune system. The majority of studies on BPA have focused on its endocrine disrupting and potential adverse effects on the developing reproductive system. However, there is only limited information concerning the effects of it on other tissues, so that the present study aimed to evaluate the oxidative stress effect of BPA in the blood of mice and whether co-administration of vitamin C can ameliorate this oxidative damage in albino mice.

Glutathione S Transferase (GST) protects cells or tissues against oxidative stress and damage by detoxifying various toxic substrates derived from cellular oxidative processes (Sharma *et al*., 2004). The current data showed a increase in the activity of GST in Group – III (prolonged exposure to Vitamin C) and little progression in Group – IV (immediate exposure to Vitamin C) compared to Control Group. Our results showed that the activity level of GST in the Vitamin C treated groups were significantly elevated as compared to the Control group of Albino mice. In Table – I, (Group – I & II) GSTenzyme level of the BPA induced mice was found to reduced to (0.041µg) as compared to the control value of (0.3156 µg) The values were found to be significant at $p < 0.05$ level .In Table – II, (Group – I & III) the GSTenzyme level of the Vitamin C treated mice was found to be (0.145µg) as compared to the control value of (0.3516µg). The values were found to be significant at $p < 0.05$ level. In Group – IV, The GST enzyme level of the Vitamin C (immediate 4 th to 34 th day) treated mice was found to be (0.2283µg) as compared to the the control value of (0.3516µg).The values were found to be significant at $p < 0.05$ level. The treatment supplemented with Vitamin C both prolonged and immediate showed little progression in the level of antioxidant enzyme Glutathione S Transferase in the blood of albino mice.

GPx catalyze dismutation of the Superoxide anion (O_2-) into hydrogen peroxide (H_20_2) which then convert hydrogen peroxide to water, In this manner, providing protection against reactive oxygen species (Aydogan *et al*., 2010).In Table – I, (Group – I & II) GpX enzyme level of the BPA induced mice was found to reduced to (0.0916µg) as compared to the control value of (0.5166 µg) The values were found to be significant at $p < 0.05$ level. In Table – II, (Group – I & III) the GpX enzyme level of the Vitamin C treated mice was found to be (0.273µg) as compared to the control value of (0.5166µg). The values were found to be significant at $p < 0.05$ level. The GpX enzyme level of the Vitamin C (immediate 4 th to 34 th day) treated mice was found to be (0.405µg) as compared to the control value of (0.5116 µg). The values were found to be significant at $p < 0.05$ level. The treatment supplemented with Vitamin C both prolonged and immediate showed little progression in the level of antioxidant enzyme Glutathione per oxidase (GpX) in the blood of albino mice.

The Oxidative stress in the blood of BPA induced mice was indicated by the elevated levels of TBARS. In Table – I (Group – I & II) The activity level of TBARS enzyme in Group – II (BPA induced mice) is significantly elevated (1.636µg) to higher levels which clearly indicates the Oxidative stress in the blood of Albino mice as compared to control mice (0.5116 µg). In Table - II (Group – I & III) The activity level of TBARS enzyme (0.3033 µg) was significantly decreased in the treated mice as compared to the control group of Albino mice, (0.5116 µg). The values were found to be significant at $p < 0.05$ level in the present study. In Group – IV, The activity level of TBARS enzyme (0.2633µg) was significantly decreased in the treated mice as compared to the control group of Albino mice, (0.5116 µg). The values were found to be significant at $p < 0.05$ level in the present study. The treatment supplemented with Vitamin C both prolonged and immediate treatment significantly reduced the damage caused by TBARS enzyme and the level get reduced in the blood of treated mice.

Super oxide Dismutase (SOD) constitute a very important antioxidant defense against oxidative stress in the body. SOD is an enzyme that alternately catalyzes the dismutation of the superoxide radicals into ordinary molecular oxygen and hydrogen peroxide. In Table – I ,(Group – I & II) The activity level of SOD in the BPA induced group was significantly decreased as compared to the control group of Albino mice in the present study. The activity level of SOD was found to be decreased to (0. 145µg) as compared to the control, (0.7883 µg) respectively. In Table – II, (Group – I & III) The activity level of SOD was found to be (0. 4033 µg) as compared to the control, (0.7883 µg) respectively. In Group – IV, The activity level of SOD was found to be(0.4753 µg) as compared to the control, (0.7883 µg) respectively. The treatment supplemented with Vitamin C

both prolonged and immediate showed little progression in the level of antioxidant enzyme Super oxide dismutase (SOD) in the blood of albino mice.

Catalase catalyze dismutation of the Superoxide anion (O_2-) into hydrogen peroxide (H_20_2) which then convert hydrogen peroxide to water, In this manner, providing protection against reactive oxygen species (Aydogan *et al*., 2010). In Table – I, (Group – I & II) CAT enzyme level of the BPA induced mice was found to reduced to (0.0716µg) as compared to the control value of (0.145 µg) The values were found to be significant at $p < 0.05$ level . In Table – II, (Group – I & III) the CAT enzyme level of the Vitamin C (31-60 days) treated mice was found to be(0.1366µg)as compared to the the control value of (0.145 µg). The values were found to be significant at $p < 0.05$ level. In Group – IV, The CAT enzyme level of the Vitamin C (immediate 4^{th} to 34^{th} day) treated mice was found to increased to (0.1666µg) as compared to the control value of (0.145 µg). The values were found to be significant at $p < 0.05$ level. The treatment supplemented with Vitamin C both prolonged and immediate significantly increased the level of antioxidant enzyme Catalase in the blood of albino mice.

6. Conclusion

The study findings demonstrate that BPA exposure causes cellular dysfunction and increased production of reactive oxygen species, leading to oxidative stress. However, supplementation with Vitamin C, both immediately and for a prolonged period, significantly reduces the damage caused by oxidative stress. These results suggest that Vitamin C has a protective role against BPA-induced oxidative stress. Further research is needed to explore the potential mechanisms underlying this protective effect and to assess the applicability of these findings in human populations.

Conflicts of Interests

The authors declare no conflict of interest.

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Table I Showing Parameters of antioxidant enzymes in Control (Group- I) and Experimental BPA induced Group (Group – II). Values are mean ±SD. The values were found to be significant at $p < 0.05$ level

S. No	Parameters	Control (Group-I)	Experiment (Group-II)	% of Changes
1	GST	0.3516 ± 0.0147	0.041±0.008	88.33 %
2	GPX	0.5116 ±0.00371	0.0916 ± 0.018	82.09 %
3	TBARS	0.5116± 0.0365	1.636 ± 0.036	- 219.78 %
4	SOD	0.7883± 0.0278	0.145 ± 0.033	81.60 %
5	CAT	0.145± 0.0137	0.071± 0.013	51.03 %

Table II Showing Parameters of antioxidant enzymes in Control (Group- I) , BPA induced (Group – II) and BPA (1 to 30 days), Vitamin C (31-60 days) treated Group (Group –III) and BPA (1 to 3 days), Vitamin C (4^{th} - 34 days) treated Group (Group –IV). Values are mean ± SD. The values were found to be significant at $p < 0.05$ level

S. No	Parameters	Control Group – I	Experiment Group – II	% of Changes	Experiment Group – III	% of Changes	Experiment Group – IV
1.	GST	0.351±0.014	0.041± 0.008	58.76 %	0.145 ± 0.015	35.04 %	0.228 ± 0.017
2.	GPX	0.511 ± 0.003	0.091± 0.0186	46.57 %	0.273± 0.012	20.74 %	0.405± 0.019
3.	TBARS	0.511± 0.036	1.636 ± 0.036	40.70 %	0.303± 0.012	48.53 %	0.263± 0.012
4.	SOD	0.788± 0.027	0.145 ± 0.033	48.84 %	0.403± 0.018	39.72 %	0.475± 0.016
5.	CAT	0.145 ± 0.0137	0.071 ± 0.013	6.20 %	0.136± 0.015	- 14.48 %	0.166± 0.016

[GST – Glutathione S Transferase , GpX – Glutathione Peroxidase, TBARS – Thiobarbituric acid reactive substances, SOD – Super Oxide Dismutase, CAT – Catalase]

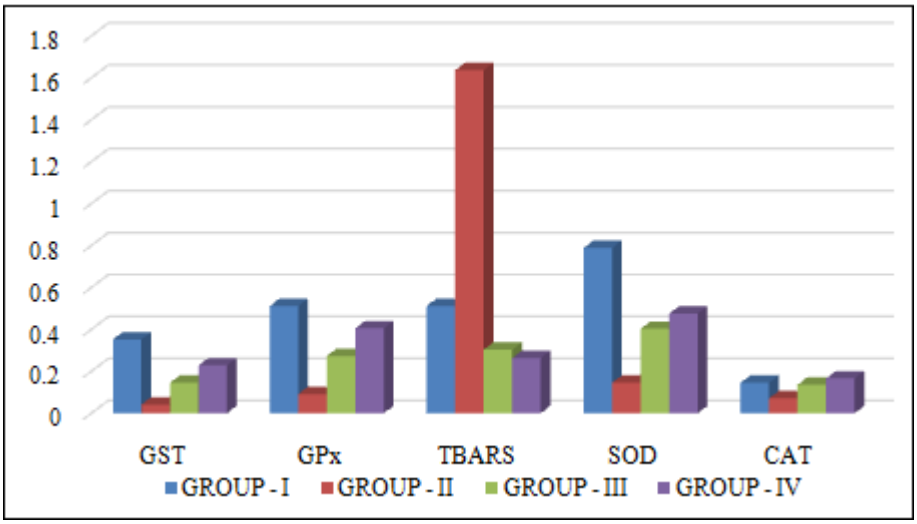

Figure 4: Bar chart showing Comparison of Mean values in antioxidant enzyme Glutathione S Transferase (GST), Glutathione Peroxidase (GpX), TBARS, Superoxide Dismutase (SOD) and Catalase (CAT)