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# Neuroprotective Effects of Coenzyme Q10 on Bisphenol-A-Induced Neuroinflammation, Oxidative Stress and Cerebellar Cortex Toxicity Wistar Rats

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## ABSTRACT

This study evaluated the neuroprotective effects of coenzyme Q10 (CoQ10) supplementation on Bisphenol-A (BPA)-induced cerebellar cortex toxicity in Wistar rats. Thirty-six male Wistar rats (150–200 g) were divided into six groups (n=6) and fed standard diet for 28 days. Group A (control) received corn oil (10 ml/kg), while Groups B and C (CoQ10 controls) were administered CoQ10 at 10 mg/kg and 20 mg/kg orally, respectively. Group D (BPA control) received BPA at 50 mg/kg orally. Groups E and F were co-administered BPA (50 mg/kg) with CoQ10at 10 mg/kg and 20 mg/kg respectively. Weekly weight changes were recorded. At the end of the experiment, animals were sacrificed, and cerebellar tissues were either homogenised for biochemical analysis of oxidative stress markers, inflammatory markers, and antioxidant enzyme activities or processed for general histology. Results showed that exposure to BPA group D) caused significant weight loss, significant increase in malondialdehyde (MDA) levels, and significant decrease in antioxidant markers (SOD, GSH, TAC, CAT) compared to the control ($p<0.05$). Inflammatory markers (IL-1β and TNF-α) significantly increased in Group D. CoQ10 supplementation in Groups E and F significantly reduced antioxidant markers (SOD, GSH, TAC, CAT), MDA and inflammatory markers, and mitigated weight loss. Histological analysis showed neuronal loss in Group D, which was reversed in Groups E and F. CoQ10 supplementation protects against BPA-induced cerebellar cortex toxicity by reducing oxidative stress, inflammation, and neuronal damage, highlighting its potential as a neuroprotective agent.

**KEYWORDS: Antioxidants; Bisphenol-A; Neurotoxicity; Neuroprotection; Toxicant; Ubiquinone**

## 1. Introduction

Bisphenol A (BPA) belongs to chemicals that are produced in large quantities worldwide [1, 2]. It is widely utilized as a monomer in polycarbonate synthesis, a plasticizer in epoxy resin production, and an additive to neutralize excess hydrochloric acid during polyvinyl chloride (PVC) manufacturing [1]. Bisphenol A (BPA) is widely used in the production of polycarbonate plastics and epoxy resins, leading to extensive human exposure through multiple routes, including

oral ingestion, inhalation, and transdermal absorption [3]. Oral ingestion is the most common route, primarily due to BPA leaching from food packaging materials such as cans and plastic containers, especially when exposed to heat or acidic conditions [4,5]. Inhalation exposure occurs through indoor air and dust containing BPA, which can be significant in environments with high levels of plastic products or industrial emissions [6]. Transdermal absorption can occur through direct contact with materials like thermal paper receipts, healthcare equipment, toys, and articles designed for children and infants [7]. After exposure, BPA undergoes extensive metabolism in the liver, where it is primarily converted to bisphenol A glucuronide, a water-soluble form that is largely excreted in the urine. While this biotransformation reduces the bioactivity of BPA, unmetabolized BPA in the body can still exert significant biological effects due to its phenolic structure [8]. This structure enables BPA to interact with oestrogen receptors (ERα and ERβ), acting either as an agonist or antagonist depending on the context, and disrupts oestrogen receptor-dependent signaling pathways [9]. BPA's endocrine-disrupting properties have been implicated in a range of health disorders. For instance, BPA exposure has been linked to infertility in both males and females by disrupting gametogenesis and altering reproductive hormone levels [10]. It has also been associated with precocious puberty, likely due to its oestrogen-mimicking activity that accelerates the onset of puberty [11]. Furthermore, hormone-dependent cancers, including breast and prostate cancers, have shown associations with BPA exposure, as it promotes cell proliferation and inhibits apoptosis through oestrogen receptor-mediated mechanisms [12]. BPA is also implicated in metabolic disorders such as polycystic ovary syndrome (PCOS) by interfering with insulin signaling and exacerbating insulin resistance, a core feature of PCOS [13]. Additionally, evidence suggests that chronic BPA exposure may contribute to obesity, diabetes, and cardiovascular diseases, highlighting its role in broader metabolic and endocrine disruption [14]. Therefore, BPA has been implicated in the development of various endocrine disorders, including infertility in both males and females, precocious puberty, hormone-dependent cancers like breast and prostate cancer, as well as metabolic conditions such as PCOS [15] Due to its impact on the endocrine system, BPA was initially examined for effects on sexual dysfunction, malformation, and cancers of reproductive origin. This chemical exists as colourless crystals or powder and can leach from plastic items into food and beverages under heat and acidic or basic conditions. Exposure of polycarbonate plastics to elevated temperatures, such as when cooking food in containers or infant bottles, enhances the rate of BPA migration into the human body. Furthermore, exposure to acidic or basic substances, in addition to high concentrations of sodium chloride or vegetable oils, facilitates the release of BPA from polymeric materials. Studies indicate that BPA disrupts spermatogenesis and adversely affects male reproductive function [2]. Sperm motility is adversely impacted by BPA in humans, mice, bovines, chickens, and fish [16]. Exposure to BPA has been linked to a heightened risk of hypertension and cardiovascular illness in both humans and rats, although the underlying processes remain ambiguous [17]. BPA influences glucose metabolism, the initiation and advancement of various cancers, and immunological function by interacting with distinct receptors, altering transcription factors, and causing epigenetic modifications [17]. The majority of these findings have been derived from studies involving people, animals, and cellular cultures. Public apprehension regarding the potentially detrimental health impacts of BPA led to the prohibition of numerous plastic items, especially those intended for new-borns and young children [18]. BPA can induce aggression, anxiety, cognitive deficits, and learning-memory impairment [19-21].

In the last few decades, the beneficial effects of nutritional status, diet and the use of dietary supplements like CoQ10 in reducing the incidence and burden of neurodegenerative diseases and neurotoxicity have been the focus of a number of studies [22,23]. Coenzyme Q10 is a component of the electron transport chain that modulates mitochondrial generation of adenosine triphosphate (ATP). The reduced form of CoQ10 has been reported to have antioxidant properties, which are beneficial in the management of neurodegenerative diseases and neurotoxicity [24]. Results of a number of studies in rodents [24] and humans [25]. The objectives of this study were to assess the effects of the Coenzyme Q10 on the biochemical and histomorphological changes in the cerebellum of rats administered Bisphenol A.

## 2. Materials and Methodology

### 2.1 Chemicals and Drugs

Bisphenol A. (Anhui Sinotech Industrial Co. Ltd) was procured from Sumther Pharmacy, Ogbomoso. Assay kits for lipid peroxidation (Randox Laboratory Ltd. UK), Tumour necrosis factor-α, Interleukin 1beta, Superoxide Dismutase, Glutathione, Total Antioxidant Capacity, Catalase. (ENZO Life Sciences, U.S.A).

### 2.2 Animals

Thirty-six adult, male Wistar rats, weighing between 150–200 g, were procured from the Animal House in Igbo Sai, Ogbomoso, Oyo State, Nigeria. The rats were housed in plastic cages within a temperature-controlled environment (23–26°C) under a 12-hour light/dark cycle, with lights on daily at 7:00 a.m. They were fed commercial standard chow (TOP Feeds Nigeria Ltd). Animals were weighed and acclimatized for the

period of two weeks before the commencement of the study. During the experimental period, animals had free access to food and water *ad libitum*. All experimental procedures were conducted in accordance with the approved institutional protocols and within the provisions for animal care and use.

### 2.3 Experimental Methods

Thirty-six (36) Male Wistar rats weighing between 150-200g was randomly assigned into 6 groups (A-F) of 6 animals each. All groups were fed standard diet throughout the experimental period of 28 days, group A, served as the control and was administered corn oil at 10 mls/kg, group B and C served as CoQ10 control group and was administered CoQ10 at 10 mg/kg and 20mg/k respectively, groups D (Bisphenol A control group) received Bisphenol A at 50 mg/kg. Groups E was administered Bisphenol A at 50 mg/kg and CoQ10 at 10 mg/kg while group F received Bisphenol A at 50 mg/kg and CoQ10 at 20 mg/kg body weight [26, 27]. Body weight of the animals were measured weekly using an electronic Mettler weighing balance (Mettler Toledo Type BD6000, Switzerland). At the end of the experimental period, animals were sacrificed through cervical dislocation, the brain was dissected and weighed, the cerebellar tissues were excised, processed, sectioned at 5 µm and stained using haematoxylin and eosin and Cresyl fast violet stain. Homogenate of the cerebellar cortex was used for the assessment of Total Anti-Oxidant Capacity (TAC), Superoxide dismutase (SOD), Glutathione (GSH), Catalase (CAT), malondialdehyde (MDA), inflammatory markers (IL-1beta, and TNF-α), Catalase (CAT), activity to determine the neurologic effects of Co enzyme Q10 on Bisphenol A treated rats.

### 2.4 Determination of body weight

Body weights of animals in all groups were measured daily using electronic Mettler weighing balance (Mettler Toledo Type BD6000, Switzerland). The relative change in body weight for each animal was calculated using the equation below, and then the results for all animals were averaged to determine the statistical mean.

$$\frac{\text{Final body weight (or feed intake)} - \text{Initial body weight (or feed intake)}}{\text{Initial body weight (or feed intake)}} x100$$

### 2.5 Tissue Homogenisation

Cerebellum of rats in each group were homogenised has previously described by Onaolapo et al [28]. Homogenate was separated by centrifugation at 3500 rpm for 10 minutes using a general laboratory centrifuge (JICA Japan). The supernatant was assayed immediately or stored (-20$^{o}$).

### 2.6 Biochemical Test

#### 2.6.1 Determination of glutathione activity

Glutathione (GSH) level was assayed based on Ellman's reagent's (DTNB) reaction with free thiol groups. Sample were mixed with 0.4M Tris-HCL buffer (pH 8.9) and 0.01M DTNB as previously described [Onaolapo et al [29]. GSH activity was determined by absorbance at 412nm, and expressed as nM of GSH.

#### 2.6.2 Determination of lipid peroxidation levels

The lipid peroxidation kit was used to assess the level of malondialdehyde (MDA) in samples by measuring thiobarbituric acid-reactive species (TBARs) following the instructions of the manufacturer. Levels of malondialdehyde (MDA) a marker of lipid peroxidation was assayed from plasma by measuring thiobarbituric reactive species. Reactive substances of the thiobarbituric acid react with thiobarbituric acid producing a red coloured complex which is measured at an absorbance of 532nm as previously described [30].

#### 2.5.2. Determination of Superoxide Dismutase activity

The principle for determining superoxide dismutase (SOD) activity is based on the ability of SOD to inhibit the auto-oxidation of adrenaline (epinephrine) at an alkaline pH. Superoxide radicals (O2-) are generated through the auto-oxidation of adrenaline at pH 10.2. Superoxide dismutase catalyses the dismutation of O2- into oxygen (O2) and hydrogen peroxide (H2O2). The degree of inhibition of adrenaline auto-oxidation by SOD is measured spectrophotometrically at 480 nm. Superoxide dismutase activity in this study was measured following protocols as previously described by [29]

#### 2.6.3 Determination of Catalase activity

Catalase activity was measured following the directions of the kit manufacturer

#### 2.6.4 Determination of Total Antioxidant Capacity

Total antioxidant capacity was measured using the Trolox Equivalent Antioxidant Capacity Assay that is based on the ability of antioxidants within a sample to react with oxidized products as previously described by [30].

#### 2.6.5 Determination of Tumour Necrosis factor-α and Interleukin-1β

Tumour necrosis factor-α and interleukin 1β were measured using enzyme-linked immunosorbent assay (ELISA) techniques with commercially available kits (Enzo Life Sciences Inc. NY, USA) designed to measure the 'total' (bound and unbound) amount of the respective cytokines as previously described [30]. Interleukin1 β level was assayed using enzyme-linked

immunosorbent assay (ELISA) techniques with commercially available kits (Enzo Life Sciences Inc. NY, USA).

### 2.7 Tissue Histology

The brain of each of the animals was dissected out and weighed and cerebellum was removed, fixed in 10% formol-calcium by total immersion for 24 hours after which it was were trimmed to about 3 to 5mm thick sections and processed via paraffin wax embedding method. The tissue was dehydrated at room temperature through ascending grades of alcohol. Dehydrated tissue was cleansed at room temperature in two changes of molten paraffin wax using a multi block plastic embedding mold. The paraffin blocks were then trimmed and mounted on wooden block for sectioning on a rotary microtome (Bright B5143, Huntington, England), the section was transferred to water bath (40C) to allow spreading of the folded sections. These sections were mounted on new clean glass slides which are later dried on a slide drier to enhance adherence of section to slide.

### 2.8 Photomicrography

Representative haematoxylin and eosin, and cresyl fast violet-stained histological slides of the cerebellum were examined using a Carl Zeiss microscope (Axioscope 40, Germany) with a digital camera attached. Photomicrographs of the cerebellar sections were taken at different magnifications which were done at the histopathology laboratory of the Department of Medical Laboratory Sciences, Faculty of Basic Medical Sciences, Ladoke Akintola University of Technology Ogbomoso.

### 2.9 Statistical Analysis

Statistical analysis of the body weight and the biochemical was carried out using Chris Rorden's ezANOVA for windows. Data obtained were subjected to analysis of variance (ANOVA) and post-hoc tests (Tukey HSD). Results are expressed as Mean ± S.E.M., $p<0.05$ was taken as the accepted level of significant difference from control.

## 3. Results

### 3.1 Effect of Coenzyme Q10 on Body Weight

**Figure 1** shows the effect of dietary Coenzyme Q10 on percentage change in body weight in rats treated with Bisphenol A. (BPA). There was a significant ($p <0.001$) decrease in percentage weight gain in the groups administered BPA and BPA+Coenzyme Q10 [BPA/$CoQ_{10}$] at 10 and 20 compared to control. Compared to BPA, body weight increased with BPA/$CoQ_{10}$10 and BPA/$CoQ_{10}$20 respectively.

### 3.2 Effect of Coenzyme Q10 on oxidative stress parameters

**Table 1** Shows the effects of Coenzyme Q10 on Malondialdehyde (MDA), Superoxide Dismutase (SOD), Catalase, Glutathione (GSH), and Total Antioxidant Capacity (TAC) levels in BPA-treated rats. Malondialdehyde levels increased with BPA, BPA/$CoQ_{10}$10 and BPA/$CoQ_{10}$20 compared to control. Compared BPA, MDA levels decreased with BPA/$CoQ_{10}$10 and BPA/$CoQ_{10}$20. Superoxide dismutase activity and levels of GSH, and TAC decreased significantly with BPA, BPA/$CoQ_{10}$10 and BPA/$CoQ_{10}$20 compared to control while compared to BPA, these antioxidants increased with BPA/$CoQ_{10}$10 and BPA/$CoQ_{10}$20. Catalase levels did not differ significantly in any of the groups compared to control or BPA.

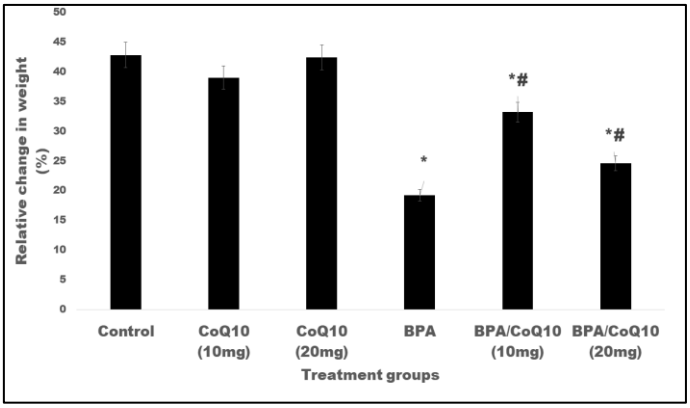


**Figure 1:** Effect of Coenzyme Q10 on relative change in body weight in Bisphenol A. (BPA) treated rats. Each bar represents Mean ± S.E.M, $^{*}p < 0.05$ vs. control, #p<0.05 significant difference from BPA, number of rats per treatment group =5. BPA: Bisphenol A, CoQ10: Coenzyme Q10.

### 3.3 Effect of Dietary Coenzyme Q10 on Inflammatory Cytokines

**Table 2** shows the effect of Coenzyme Q10 on inflammatory markers; interleukin 1β and tumour necrosis factor-α in Bisphenol A treated rats. Interleukin 1β, and TNF-α levels increased significantly with BPA, BPA/$CoQ_{10}$10 and BPA/$CoQ_{10}$20 compared to control. Compared to BPA, interleukin-1β and tumour necrosis factor-α levels decreased significantly with BPA/$CoQ_{10}$10 and BPA/$CoQ_{10}$20.

**Table 1: Effect of coenzyme-Q10 on oxidative stress markers**

| Groups | MDA nmol/g | SOD U/mg | CAT µM/mg) | GSH (pg/mg) | TAC(TE) |
|---|---|---|---|---|---|
| Control | 35.16±51 | 17.27±11 | 0.02±01 | 2.16±04 | 23.39±45 |
| $CoQ_{10}$ 10 | 34.60±28 | 17.02±37 | 0.02±01 | 2.21±02 | 23.43±44 |
| $CoQ_{10}$ 20 | 36.30±40 | 18.32±17 | 0.03±01 | 2.52±04 | 23.61±31 |
| BPA | 54.14±67* | 12.28±21* | 0.01±01 | 1.11±04* | 14.97±09* |
| BPA+$CoQ_{10}$10 | 37.28±38*# | 14.38±31*# | 0.01±01 | 2.06±02*# | 20.11±08*# |
| BPA+$CoQ_{10}$20 | 36.72±22*# | 13.98±4*# | 0.01±01 | 2.07±02*# | 20.08±13*# |

Data presented as Mean ± S.E.M, $^{*}p < 0.05$ vs. control, #p<0.05 significant difference from BPA, number of rats/ per group =10, MDA: Malondialdehyde, SOD: Superoxide Dismutase, CAT: Catalase, GSH: Glutathione, TAC, Total Anti-oxidant Capacity.

### 3.4 Effect of coenzyme Q10 cerebellar histomorphology

Plates show representative photomicrograph of haematoxylin and eosin (Plate 1A-F) and cresyl fast violet (Plate 2A-F) stained sections of the rat cerebellum. Examination of the haematoxylin and eosin-stained slides revealed discreet layers of the cerebellum showing the molecular (M), granular layer (G), and the Purkinje cell layer which appear normal in groups 1A, 1B, and 1C. While in group D, the molecular contains showed numerous pyknotic neurons also observed was an area of focal inflammatory cell infiltration. Groups E and **F** showed normal cerebellar features though an area of focal inflammatory cell infiltration was also observed.

**Table 2: Effect of coenzyme-Q10 on inflammatory cytokines**

| Groups | Interleukin 1 β(pg/ml) | TNF-α(pg/ml) |
|---|---|---|
| Control | 18 .71±35 | 19.87±27 |
| $CoQ_{10}$ 10 | 18.40±14 | 20.10±45 |
| $CoQ_{10}$ 20 | 18.90±22 | 21.15±51 |
| BPA | 35.96±29* | 40.67±68* |
| BPA+Co $Q_{10}$10 | 24.90±66*# | 27.59±38*# |
| BPA+Co $Q_{10}$20 | 24.42±26*# | 27.44±13*# |

Data presented as Mean ± S.E.M, *p < 0.05 vs. control, #p<0.05 significant difference from BPA, number of rats per group =10, Interleukin 1β and TNF-α (Tumour Necrosis Factor)

The pink-staining background, known as the neuropil, is clearly visible in slides stained with cresyl fast violet. The cresyl fast violet-stained slides showed pink-staining background with deeply stained granule cells and neuroglia in the control group and Co-Q10 administered groups (Plate 2 A, B, C). However, in the BPA group (Plate 2D), numerous degenerating granule cells with pale staining, and shrunken, nuclei were observed. Conversely, in the groups treated with Co-Q10 (3.2 E-F), a protective effect against BPA-induced neuronal damage was observed.

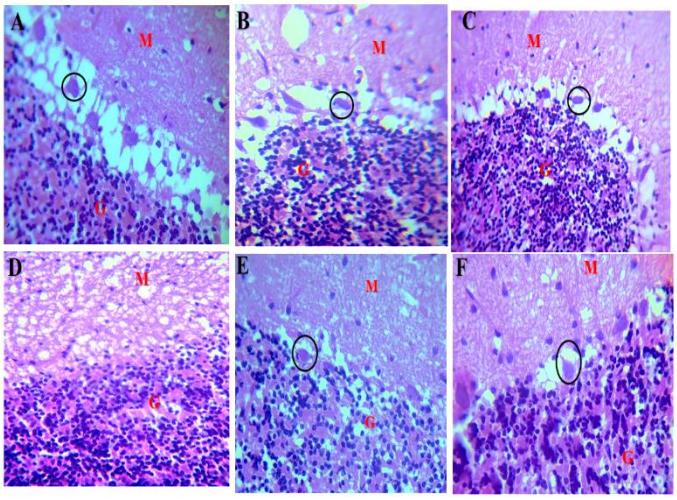


**Plate 1:** Photomicrograph of the Haematoxylin and Eosin-stained sections of the cerebellar cortex showing a distinct layer of the cerebellum. Molecular (M), granular layer (G), and cerebellar cortex appear normal with normal folia in groups (**A, B, and C**). With group **D,** the molecular contains reduced normal neurons, with few pyknotic neurons, an area of focal inflammatory cell infiltration was observed. Groups **E** and **F** show normal cerebellar features though an area of focal inflammatory cell infiltration was observed. **(Mag: X100).**

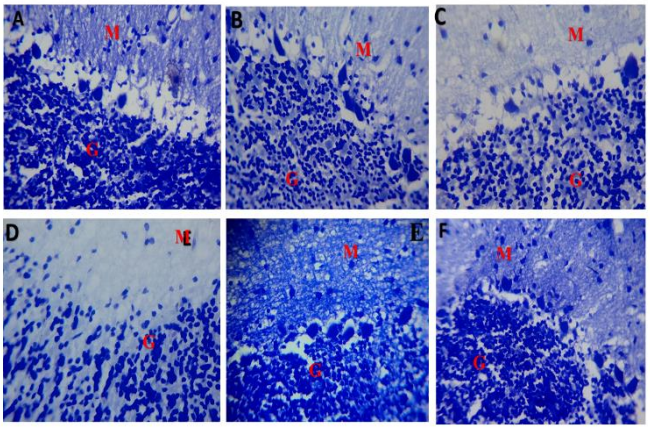


**Plate 2**: Photomicrograph of the Cresyl fast violet-stained sections of the cerebellar cortex. The pink-staining background is more clearly visible in the control group (2 A), and in the BPA control group (2 D), many degenerating neurons that exhibited pale staining, shrunken, and lightly stained nuclei were observed. Conversely, in the groups treated with Co-Q10 (2 **E-F**), a protective effect against BPA-induced neuronal damage was observed. **(Mag: X100).**

### 3.5 Effect of Coenzyme Q10 on cerebellar morphometry

**Figure 2** shows the effect of Coenzyme Q10 on number of pyknotic neurons in the cerebellar cortex. There was a significant increased in number of pyknotic neurons with BPA, BPA/$CoQ_{10}$10 and BPA/$CoQ_{10}$20 compared to control. Compared to BPA, pyknotic neurons decreased significantly with BPA/$CoQ_{10}$10 and BPA/$CoQ_{10}$20.

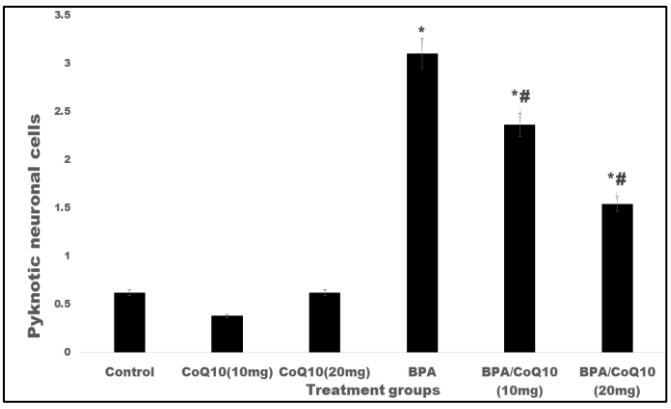


**Figure 2:** shows the Image J morphometry analysis of the cerebellum-. Effect of Coenzyme Q10 on relative change in body weight in Bisphenol A. (BPA) treated rats. Each bar represents Mean ± S.E.M, *p < 0.05 vs. control, #p<0.05 significant difference from BPA, number of rats per treatment group =5. BPA: Bisphenol A, CoQ10: Coenzyme Q10.

## 4. Discussion

This study investigated the possible effects of CoQ10 on histomorphological and biochemical markers of inflammation

and oxidative stress in rats treated with Bisphenol A. (BPA). The results showed that CoQ10 ameliorated BPA induced changes in body weight, oxidative stress, inflammation and cerebellar cortex histoarchitecture.

In this study, percentage change in weight decreased significantly in the groups administered BPA compared to control. Weight loss observed with BPA administration in this study was in tandem with a number of studies that had reported similar effects [31, 32]. Coenzyme Q10, a lipid soluble compound belonging to the vitamin category though not classified under the B-complex vitamins on the other hand reversed the weight loss effects observed with BPA. While a number of studies examining the effects of Coenzyme Q10 on body weight have reported it had no significant effects on body weight [33-35]. In this study we observed that in health CoQ10 did not significantly alter body weight corroborating the results of these studies; however in the background of BPA induced weight loss we observed a reversal of the weight loss, suggesting that in disease its ability to mitigate oxidative stress and other metabolic parameters [36] could reduce stress and thereby facilitate weight gain.

The prooxidant effects of BPA have been reported severally [37]. Results of this study revealed that BPA administration was associated with an increase oxidative stress consistent with these studies [37-39]. The administration of Coenzyme Q10 reversed these changes. Coenzyme Q10's ability to scavenge the reactive oxygen species (ROS) and increase the activity of various antioxidant enzymes, including SOD, and GSH, have been suggested as possible mechanisms through which it could reverse the oxidative stress associated with exposure to BPA in this study. BPA has also been reported to be associated with cellular damage and dysfunction, which can also further exacerbate oxidative stress [40]. The results of the TAC in this study showed an increase with COQ10 corroborating the results of studies that had reported similar effects [41-43]. In this study however no significant changes were observed with catalase. The stability of catalase levels with oxidative stress associated with BPA and ameliorating effects of CoQ10 exposure in contrast to other anti-oxidative enzymes (SOD and GSH) that increased in this study can possibly be attributed to a number of factors including differential regulation, substrate availability, tissue-specific responses, oxidative stress thresholds, and compensatory mechanisms. Catalase expression and activity can vary across different tissues and cell types. In some tissues with high metabolic activity or specific functional roles in antioxidant defence such as the brain, catalase could be more resilient to changes in oxidative stress compared to SOD and GSH, which could possibly explain why its levels remain unchanged compared to the others [44]. Moreover, there have also been suggestions that there could be a threshold level of oxidative stress above which SOD and GSH levels rise, but catalase levels do not change until a more severe level of stress is reached.

Also, in this study the administration of BPA was associated with an increase in the levels of pro-inflammatory cytokines (IL 1beta and TNF-α) also corroborating reports of studies that had associated BPA exposure with development of a proinflammatory response [45]. Treatment with CoQ10 reversed the associated inflammation caused by BPA; reducing the levels of these pro-inflammatory cytokines. There have been suggestions that CoQ10 has the ability to modulate inflammatory pathways as previously reported [36, 46, 47].

The histological findings in this study (Plates 1 and 2), revealed the preservation of normal cerebellar cortex architecture in the control group with evidence of neuronal injury with BPA exposure evidenced by decreased molecular and Purkinje cell layers, with few pyknotic neurons and slight inflammatory cell infiltration. In groups administered CoQ10 amelioration of BPA-induced disruption of the histoarchitecture of the cerebellum was observed.

## Conclusion

This study demonstrates that CoQ10 effectively mitigates BPA-induced weight loss, neuronal damage, oxidative stress, and neuroinflammatory processes in rats. Key biochemical markers, including malondialdehyde superoxide dismutase, glutathione interleukin-1β, and TNF-α, showed significant improvements with CoQ10 intervention. Histomorphological analysis further confirmed cerebellar preservation following treatment. These findings highlight CoQ10's potential as a therapeutic agent for managing weight loss and neurotoxicity associated with BPA exposure.

**Funding**.

None

**Availability of data and materials**

Data generated during and analysed during the course of this study are available from the corresponding author on request.

**Declaration of Ethics approval**

Research ethical approval was obtained from the Ethical research committee of Faculty of Basic Medical Sciences, Ladoke Akintola University of Technology with ERC approved number ERCFBMSLAUTECH:035/05/2024.

**Competing interests**

None

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