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ANTIOXIDANT EFFECTS OF *SOLANUM AETHIOPICUM* EXTRACT ON OXIDATIVE STRESS MARKERS IN POTASSIUM BROMATE-TREATED WISTAR RATS

Okon Victoria Edem

Department of Physiology, University of Cross River (UNICROSS).

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ABSTRACT: *The aim of this study was to investigate the potential role of Solanum aethiopicum (GEL) on glutathione peroxidase (GPX) and malionaldehyde (MDA) in potassium bromate-induced cell toxicity in Wistar rats. In the method, a total of thirty (30) Wistar rats weighing 140-150 g were used for the experiment. The rats were grouped into four (5) groups according to their body weight. Each group contains five (6) Wistar rats. Group 1 was a normal control group fed with standard rat pellets and 0.2 ml of normal saline; Group 2 was administered potassium bromate (50 mg/kg); Group 3 was administered a low dose of GEL (300 mg/kg); Group 4 was administered a high dose of GEL (600 mg/kg); and Group 5 was administered vitamin C (100 mg/kg) using oropharyngeal cannulas. Potassium bromate was administered for 2 weeks for groups 3,4 and 5 before administration of the extracts. All the animals had free access to food and water. Administration was carried out every day for the period of 14 days, after which the animals were sacrificed. Serum GPx and MDA were assayed using spectrophotometric methods, and data were analyzed by one-way ANOVA with Tukey's post hoc test. Results showed that potassium bromate administration reduced GPX activity and MDA levels relative to control. Co-treatment with low-dose GEL significantly ($P<0.05$) enhanced GPX activity and normalized MDA levels, while the high-dose GEL did not differ compared to the control. Conversely, vitamin C markedly increased MDA and suppressed GPx activity, demonstrating a paradoxical pro-oxidant effect. These findings suggest that S. aethiopicum leaf extract exerts dose-dependent antioxidant effects, with the low dose providing a greater protective modulation.*

KEYWORDS: Anti-oxidants, Solanum aethiopicum, Vitamin C, Oxidative stress, Glutathione, Malionaldehyde.

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INTRODUCTION

Oxidative stress, an imbalance between the generation of reactive oxygen species (ROS) and the capacity of antioxidant defenses (Ozougwu, 2016), plays a central role in the pathogenesis of many diseases, including neurodegenerative disorders, cardiovascular disease, kidney injury, and cancer (Pham-Huy *et al*., 2008). ROS include free radicals such as superoxide ($O_2^{\bullet -}$) and hydroxyl radical (•OH), as well as non-radical species such as hydrogen peroxide (H_2O_2) (Ozougwu, 2016). Under physiological conditions, ROS act as signaling molecules in processes like cell proliferation, adaptation to stress, and immune defense; however, when ROS generation exceeds the capacity of enzymatic and non-enzymatic antioxidant systems, oxidative damage to lipids, proteins, and DNA may occur (Pham-Huy *et al*., 2008).

Potassium bromate ($KBrO_3$) is a well-known oxidizing agent and has been used industrially (e.g., in food processing, dye, and cosmetics). However, it is also a potent inducer of oxidative stress and toxicity in experimental animals (Al-Mareed *et al*., 2022). In biological systems, $KBrO_3$ can yield bromide radicals (Br•) and other reactive intermediates, which in turn react with biomolecules and generate further ROS. This cascade can overwhelm cellular antioxidant defenses, resulting in peroxidation of membrane lipids, oxidation of proteins, and DNA damage (Ahmad *et al*., 2014). One commonly used biomarker of lipid peroxidation is malondialdehyde (MDA), whose elevated levels reflect oxidative injury to cell membranes (Salami *et al*., 2021). Additionally, $KBrO_3$ exposure often leads to the depletion or inhibition of endogenous antioxidant enzymes, thereby amplifying oxidative damage (Salami *et al*., 2021; Al-Mareed *et al*., 2022).

One of the critical enzymatic defenses against ROS is glutathione peroxidase (GPx). GPx catalyzes the reduction of hydrogen peroxide (H_2O_2) and organic hydroperoxides (ROOH) to water (H_2O) and corresponding alcohols (ROH), using reduced glutathione (GSH) as the electron donor (Arthur, 2001; Prabhakar *et al*., 2005). This activity helps prevent Fenton-type reactions and further lipid peroxidation. A decline in GPx activity is often observed under conditions of oxidative stress or exposure to pro-oxidants and is considered an indicator of impaired antioxidant defense (Arthur, 2001; Yan *et al*., 2021; Lubos *et al*., 2011).

Given the harmful effects of $KBrO_3$, there is increasing interest in identifying compounds that can ameliorate its oxidative damage. In this context, medicinal plants rich in bioactive antioxidant compounds have gained attention. *Solanum aethiopicum* (commonly referred to as African eggplant or garden egg) is widely cultivated and consumed across Africa. It is rich in flavonoids, phenolic acids, alkaloids, carotenoids, and ascorbic acid, compounds known for free radical scavenging and redox-modulating properties (Faraone *et al*., 2021; Choi and Choi, 2024; Han *et al*., 2021). Studies on *S. aethiopicum* or its extracts have demonstrated antioxidant, anti-inflammatory, antibacterial, anti-diabetic, and hepatoprotective effects (Faraone *et al*., 2021; Choi and Choi, 2024; Han *et al*., 2021). For example, in a short-term administration experiment in Wistar rats, ethanol extract of *S. aethiopicum* fruit (SAFE) significantly reduced lipid peroxidation and increased antioxidant parameters such as superoxide dismutase (SOD) activity and glutathione concentration (i.e., endogenous non-enzymatic antioxidant) without producing histological abnormalities in the liver (Iheagwam & Chinedu, 2024). Similarly, in vitro antioxidant assays on leaf extracts of *S. aethiopicum* have shown dose-dependent scavenging of nitric oxide and hydrogen peroxide radicals, which likely arise from its phytochemical constituents such as flavonoids and alkaloids (Ilechukwu *et al*., 2025).

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Despite these promising findings, there is a paucity of studies specifically addressing the protective role of *S. aethiopicum* against $KBrO_3$-induced oxidative damage in vivo, particularly in terms of GPx and MDA as oxidative stress markers. $KBrO_3$ has been shown in multiple models to increase MDA levels and suppress antioxidant enzyme activity, including GPx, SOD, and catalase, thereby confirming its oxidative mode of toxicity (Al-Mareed *et al*., 2022; Agu *et al*., 2023; Aygörmez *et al*., 2025). In one study, $KBrO_3$ administration increased brain MDA and nitric oxide (NO) concentrations in rats, while co-treatment with antioxidants attenuated these changes (Agu *et al*., 2023). Therefore, the current study aims to investigate whether *Solanum aethiopicum* extract can attenuate oxidative stress in $KBrO_3$-treated Wistar rats, focusing on changes in MDA (as a marker of lipid peroxidation) and GPx activity (as a measure of enzymatic antioxidant defense). Specifically, we hypothesize that pre- or co-treatment with *S. aethiopicum* extract will (i) reduce $KBrO_3$-induced elevation of MDA levels, and (ii) restore or enhance GPx activity relative to $KBrO_3$-only treated animals. The findings may provide insight into the therapeutic potential of *S. aethiopicum* as a natural antioxidant agent against chemical-induced oxidative injury.

MATERIALS AND METHODS

Materials

Iron cages
Water bottle
Hand gloves
Plates
Digital weighing balance
Sample bottles
Centrifuge
Chloroform
Dissecting set
Sawdust
Paper tape
Garden Egg Leaf Extract
Potassium bromate
Vitamin C
Desiccator
Cotton wool
Syringe (5 ml)
Book, Pen and Paper
Distilled water
Tissue paper
Measuring cylinder
Laboratory coat
Electronic scale
Ethanol
Marker pen
Tween 80

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Experimental Animals

Thirty (30) healthy adult male Wistar rats weighing between 140 g and 150 g were procured from the animal house of the Department of Physiology, University of Ilorin. The animals were housed in well-ventilated cages under standard laboratory conditions (12-hour light/dark cycle, temperature 25 ± 2°C, and relative humidity 45–55%). They were fed standard rat pellets and water ad libitum. The animals were acclimatized for 14 days before the commencement of the experiment. All experimental procedures were conducted in compliance with the National Institute of Health (NIH) guidelines for the care and use of laboratory animals.

Plant Material Collection and Extract Preparation

The leaves of *Solanum aethiopicum* (garden egg) were obtained from a local farm market in Okuku, Cross River State. The fruits were washed, shade-dried, and ground into fine powder. Extraction was carried out using the cold maceration method. Briefly, 500 g of powdered sample was soaked in 70% ethanol for 48 hours with intermittent shaking. The mixture was filtered using Whatman No. 1 filter paper, and the filtrate was concentrated using a rotary evaporator at 40°C. The extract obtained was stored in an airtight container at 4°C until use.

Experimental Grouping and Treatment

The Wistar rats were selected according to their body weight and divided into 5 groups containing male Wistar rats in each iron cage. All the Wistar rats had free access to food and water. The drugs were administered according to their body weight.

The Wistar rats were grouped as follows:

Group I: Control group, treated with only feed and 0.2 ml of distilled water.

Group ii: The standard group, treated with potassium bromate (50 mg/kg)

Group iii: Potassium bromate and garden egg leaf extract low-dose group, treated with 0.3 ml/100 g body weight of the animals (300 mg/kg)

Group iv: Potassium bromate and garden egg leaf extract high-dose group, treated with 0.6 ml/100 g body weight of the animals (600 mg/kg)

Group v: Potassium bromate and Vitamin C (100 mg/kg)

Administration of the extract and Vitamin C was done for 2 weeks after 2 weeks of administration of Potassium bromate

Administration of Extracts and Duration

Treatment lasted for 28 consecutive days. All administrations were done orally using an orogastric tube once daily. The dosages of *S. aethiopicum* extract and vitamin C were determined based on previous literature and preliminary LD_{50} studies (Eshetu, 2015).

Sample Collection

At the end of the experimental period, the rats were anesthetized using light chloroform inhalation. Blood samples were collected via cardiac puncture into EDTA bottles for

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biochemical analysis. The kidneys were excised, rinsed in cold saline, blotted dry, and preserved in 10% formalin for histological analysis.

Biochemical Assays

Glutathione Peroxidase (GPx) Activity

Determined using spectrophotometric assay methods as described by Arthur (2001). Glutathione peroxidase (GPx) is a critical antioxidant enzyme that catalyzes the reduction of hydrogen peroxide and organic hydroperoxides to water or corresponding alcohols, using reduced glutathione (GSH) as a substrate. Its activity is commonly determined spectrophotometrically by coupling the enzymatic reaction with glutathione reductase (GR). In this method, GPx first reduces peroxides at the expense of GSH, producing oxidized glutathione (GSSG). Subsequently, GSSG is rapidly recycled back to GSH by GR in the presence of nicotinamide adenine dinucleotide phosphate (NADPH). The consumption of NADPH, which accompanies its oxidation to $NADP^+$, is monitored at 340 nm, and the rate of absorbance decrease is directly proportional to GPx activity. The assay is typically conducted in a reaction mixture containing phosphate buffer, EDTA, sodium azide (to inhibit catalase interference), GSH, GR, NADPH, and the chosen peroxide substrate (usually hydrogen peroxide or cumene hydroperoxide). One unit of GPx activity is defined as the amount of enzyme that catalyzes the oxidation of 1 μmol of NADPH per minute under standard assay conditions. This method provides a reliable and sensitive assessment of GPx activity, making it widely applicable in studies of oxidative stress, redox regulation, and antioxidant defense mechanisms.

Malondialdehyde (MDA) Levels

Determined as an index of lipid peroxidation using the thiobarbituric acid reactive substances (TBARS) method. Malondialdehyde (MDA) is one of the most widely used biomarkers of lipid peroxidation and oxidative stress. Its levels are commonly estimated using the thiobarbituric acid reactive substances (TBARS) assay, which relies on the reaction between MDA and thiobarbituric acid (TBA) under acidic and high-temperature conditions. In this method, MDA reacts with two molecules of TBA to form a stable pink-colored MDA–TBA adduct. The intensity of this chromogen is measured spectrophotometrically at 532–535 nm, and the absorbance is directly proportional to the concentration of MDA in the sample. The assay typically involves preparing a reaction mixture consisting of the biological sample (e.g., serum, plasma, or tissue homogenate), TBA solution, trichloroacetic acid (TCA), and hydrochloric acid. The mixture is heated in a boiling water bath for about 15–30 minutes to allow the reaction to proceed. After cooling and centrifugation, the supernatant containing the MDA–TBA complex is collected, and its absorbance is measured. The MDA concentration is then determined using a standard curve prepared with 1,1,3,3-tetramethoxypropane, a compound that hydrolyzes to MDA under assay conditions. This method provides a reliable index of lipid peroxidation, making it essential in evaluating oxidative damage and the efficacy of antioxidant therapies.

Statistical Analysis

Data were expressed as Mean ± Standard Error of Mean (SEM). Statistical analysis was performed using one-way Analysis of Variance (ANOVA) followed by Tukey's post hoc test. A p-value < 0.05 was considered statistically significant.

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RESULTS

Serum Malondialdehyde (MDA)

The mean serum MDA concentration in the control group was 0.79 ± 0.01 µmol/L. Potassium bromate (KBr) administration resulted in a significantly lower MDA level (0.67 ± 0.03 µmol/L) when compared to control ($p < 0.05$). Co-treatment with low-dose GEL (0.80 ± 0.01 µmol/L) and high-dose GEL (0.81 ± 0.01 µmol/L) restored MDA to values that were statistically comparable with the control group ($p > 0.05$). In contrast, the KBr + Vitamin C group recorded the highest MDA value (1.31 ± 0.06 µmol/L), which was significantly greater than all other groups ($p < 0.05$). Thus, when comparing across treatments, vitamin C markedly elevated lipid peroxidation, while GEL extract effectively normalized MDA relative to control.

Serum Glutathione Peroxidase (GPx) Activity

The control group recorded GPx activity of 38.67 ± 0.37 U/L, whereas KBr treatment slightly reduced the activity to 37.33 ± 0.48 U/L. Co-administration of low-dose GEL significantly increased GPx activity (39.33 ± 0.37 U/L) compared to both control and KBr groups ($p < 0.05$), while the high-dose GEL group (37.33 ± 1.02 U/L) showed values similar to KBr. Vitamin C treatment produced the lowest GPx activity (30.00 ± 0.63 U/L), significantly lower than all groups ($p < 0.05$). In direct comparison, low-dose GEL exhibited the strongest positive modulation of GPx, whereas vitamin C showed the most pronounced suppression.

Table 1: Serum antioxidant parameters in potassium bromate-treated Wistar rats administered *Solanum aethiopicum* extract

Groups	Treatment	MDA (mMol/L)	GPx (u/l)
1	Control	0.79 ± 0.01^{b}	38.67 ± 0.37^{bc}
2	KBr	0.67 ± 0.03^{a}	37.33 ± 0.48^{b}
3	KBr + GEL LD	0.80 ± 0.01^{b}	39.33 ± 0.37^{c}
4	KBr + GEL HD	0.81 ± 0.01^{b}	37.33 ± 1.02^{b}
5	KBr + VIT C	1.31 ± 0.06^{c}	30.00 ± 0.63^{a}

Figure 1: Chart showing the effect of treatments on MDA

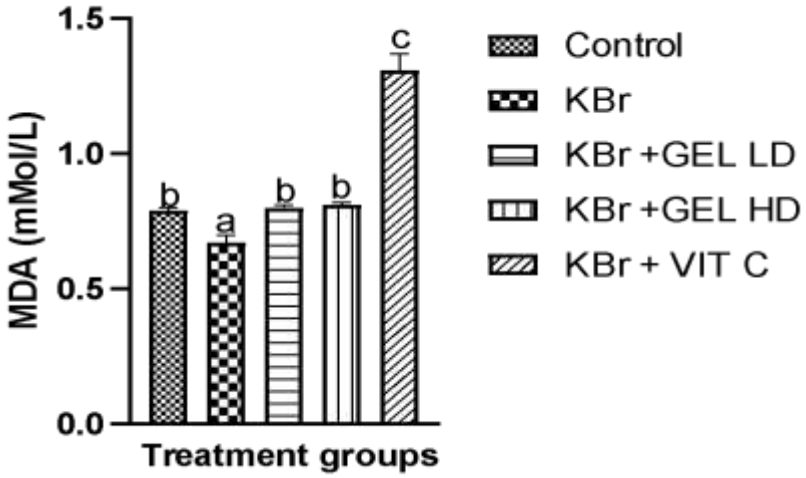

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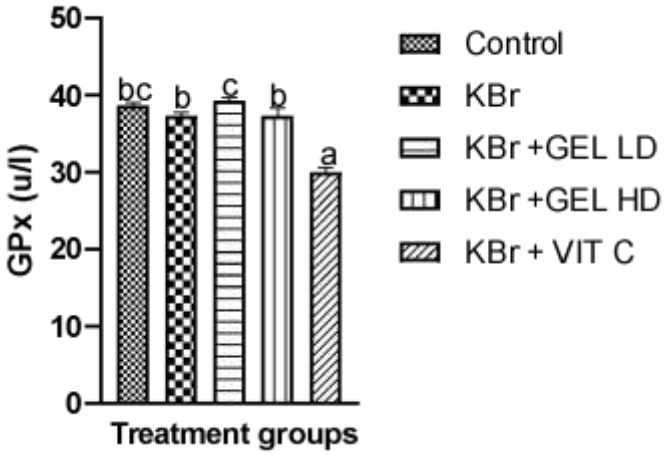
Figure 2: Chart showing the effect of treatments on GPx



DISCUSSION

The present study investigated the effects of potassium bromate (KBr), GEL extract, and vitamin C on serum oxidative stress markers, specifically malondialdehyde (MDA) and glutathione peroxidase (GPx) activity. The findings provide important insights into the antioxidant or pro-oxidant modulation by these agents. In this study, KBr administration resulted in a significantly lower MDA concentration compared with control rats. This observation is somewhat unexpected, as several previous studies have reported that KBr induces oxidative stress and markedly elevates lipid peroxidation levels (Ahmad & Mahmood, 2012). The observed modulation of MDA levels across the treatment groups aligns with the known capacity of *Solanum aethiopicum* to mitigate lipid peroxidation. The restoration of MDA levels in groups co-treated with the extract (GEL LD and GEL HD) to near-normal or control levels strongly suggests that the plant extract offered protection against potassium bromate-induced oxidative damage to lipids (Iheagwam & Chinedu, 2024). This finding is corroborated by multiple studies on closely related species. For instance, an aqueous extract of Solanum melongena was shown to significantly decrease testicular MDA levels in rats with mercury chloride-induced toxicity, confirming the anti-lipid peroxidative properties of this plant group (Adelakun *et al*., 2020). Similarly, research on *Solanum aethiopicum* fruit extract specifically reported a significant reduction in lipid peroxidation in rat models, reinforcing that this bioactivity is a direct consequence of its phytochemical composition (Iheagwam & Chinedu, 2024). The protective effect is attributed to the high content of phenolic compounds, flavonoids, and other antioxidants in the fruit, which neutralize reactive oxygen species (ROS), thereby preventing the peroxidation of polyunsaturated fatty acids in cell membranes (Faraone *et al*., 2022; Vicidomini *et al*., 2024; Choi & Choi, 2024). Interestingly, vitamin C co-treatment produced a significant increase in MDA, which was higher than all other groups. Although vitamin C is widely considered a potent antioxidant, it has also been shown to exert pro-oxidant effects under certain conditions, especially at high doses or in the presence of transition metal ions (Podmore *et al*., 1998; Niki, 2014). This finding contrasts with studies such as Nwanjo (2007), which reported protective effects of vitamin C against oxidative stress in diabetic rats, but is in agreement with studies that highlighted its dual role depending on experimental context (Arrigoni & De Tullio, 2002).

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For GPx activity, the KBr-treated group showed a slight but non-significant reduction compared with the control group. This is consistent with earlier findings that KBr exposure can impair enzymatic antioxidant defenses (Kurokawa *et al*., 1990). However, the suppression was not as pronounced as previously reported, suggesting that either the dose or duration of KBr exposure in this study was insufficient to markedly inhibit GPx activity. The activity of glutathione peroxidase (GPx) across the treatment groups indicates that *Solanum aethiopicum* extract helps in maintaining the endogenous enzymatic defense system under oxidative stress. The preservation of GPx activity in the extract-treated groups, comparable to the control, suggests a supportive role for the extract in the body's antioxidant machinery (Iheagwam & Chinedu, 2024). This is in agreement with the findings of Oboh and Rocha (2007), who reported that dietary antioxidants from African vegetables upregulate antioxidant enzymes, including GPx. A similar investigation by Adelakun *et al*. (2020) on the intervention of Solanum melongena in mercury chloride-induced toxicity reported that the plant extract was associated with increased activity of GPx, among other antioxidant enzymes. Furthermore, short-term administration of *Solanum aethiopicum* fruit extract has been shown to significantly increase glutathione (GSH) concentration in rats, which is intrinsically linked to the GPx pathway, as GSH serves as a essential co-substrate for the enzyme (Iheagwam & Chinedu, 2024). The enhancement of these enzymatic and non-enzymatic antioxidants is a key mechanism through which plant polyphenols exert their cytoprotective effects against xenobiotics (Vicidomini *et al*., 2024). The bioactive compounds in *S. aethiopicum*, such as chlorogenic acid and rutin, are known to upregulate endogenous antioxidants, potentially through pathways like Nrf2, thereby fortifying the cellular defense against oxidative insult (Nwanna *et al*., 2025). In contrast, vitamin C administration caused a marked reduction in GPx activity, the lowest among all groups. This suppression indicates that vitamin C, under the experimental conditions of this study, did not enhance antioxidant enzyme defense but rather aggravated oxidative imbalance. This is contrary to studies by Nwanjo (2007) and Arivazhagan *et al*. (2000), which found that vitamin C supplementation elevated antioxidant enzyme activities. However, it supports reports by Podmore *et al*. (1998) that vitamin C can, under oxidative conditions, act as a pro-oxidant and disrupt endogenous defense mechanisms.

CONCLUSION

This study has shown that potassium bromate administration produced mild oxidative changes, reflected by reduced MDA and slight suppression of GPx activity. GEL extract demonstrated a protective effect by normalizing lipid peroxidation and enhancing GPx activity at low doses, supporting its potential as a natural antioxidant. In contrast, vitamin C supplementation significantly elevated MDA and reduced GPx, indicating a paradoxical pro-oxidant role under the conditions of this experiment.

Conflict of interest

No conflict of interest.

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