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GASTRO-PROTECTIVE ROLE OF *SOLANUM AETHIOPICUM* ETHANOL EXTRACT IN POTASSIUM BROMATE-INDUCED TOXICITY IN RATS

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ABSTRACT: *This study was aimed at investigating the gastroprotective role of Solanum aethiopicum ethanolic extract on lipid profile in potassium bromate-induced toxicity in Wistar rats. The treatment lasted for 28 days. Thirty-five (35) rats weighing 140 g-150 g were used in this study and were separated into five groups of 7 rats. Groups 3 ,4 and 5 were pre-teated with 50 mg/kg of potassium bromate for 2 weeks. Group 1 (control) was given normal feed and 0.2 ml of normal saline. Group 2 was administered 50 mg/kg of potassium bromate for 2 weeks. Groups 3 and 4 received 300 mg/kg and 600 mg/kg of Solanum aethiopicum extract, respectively. Group 5 was administered 100 mg/kg of Vitamin C. After 28 days of administration, the animals were subjected to fasting for 24 hours and were anesthetized and sacrificed, and blood was collected by cardiac puncture, spinned and the serum used to carry out tests for lipid profile. In another set of fifty (50) male Wistar rats (150–190 g), they were randomly divided into five groups and were sacrificed to access pepsin activity and mucus secretion. Juice collected from the stomachs was also subjected to pH, pepsin activity and total acidity tests. Low doses (significantly $P<0.05$) decreased lipid profile compared to vitamin C. Statistical analyses were conducted using one-way analysis of variance (ANOVA) together with a post hoc test at $P<0.05$. The low dose of extract significantly ($P<0.05$) increased mucus secretion and pH level, reduced acidity and pepsin output in the stomach, and reduced intestinal motility.*

KEYWORDS: Solanium, acidity, pepsin, mucus, lipid, potassium bromate.

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BACKGROUND OF THE STUDY

The increasing prevalence of metabolic disorders such as dyslipidemia, obesity, cardiovascular diseases and gastric acid secretion has intensified the search for safe, effective, and affordable interventions. Synthetic drugs used to manage lipid abnormalities are often expensive and may be associated with adverse side effects, limiting their long-term use [1].

Medicinal plants have long been recognized as vital sources of therapeutic agents due to their bioactive compounds, which exhibit antioxidant, anti-inflammatory, and protective properties against various diseases [2]. Among such plants, *Solanum aethiopicum*, commonly known as African eggplant, is widely cultivated and consumed in sub-Saharan Africa, including Nigeria. While traditionally valued as a food crop, the leaves of S. aethiopicum are also used in ethnomedicine to treat a range of ailments such as liver disorders, hypertension, and metabolic imbalances.

Phytochemical studies of *S. aethiopicum* leaves have revealed the presence of alkaloids, flavonoids, saponins, tannins, and phenolic compounds [3]. These compounds are known for their antioxidant properties, which allow them to neutralize free radicals and reduce oxidative stress in the body. Oxidative stress is a major contributing factor to the development of metabolic disorders, including dyslipidemia, cardiovascular disease, and liver dysfunction. Therefore, plants like *S. aethiopicum*, rich in natural antioxidants, are of considerable interest for scientific research and potential therapeutic applications.

The ethanolic extraction of *S. aethiopicum* leaves has been particularly effective in concentrating these bioactive compounds, enhancing their pharmacological effects. Studies have demonstrated that ethanolic leaf extracts of *S. aethiopicum* can improve liver function, exhibit anti-inflammatory effects, and modulate lipid metabolism in animal models [2, 3]. These findings support the hypothesis that the plant extract may provide a protective effect against metabolic disturbances caused by oxidative stress. Potassium bromate is a known carcinogen and oxidative agent that can lead to mucosal damage and dysfunction in rats. *Solanum aethiopicum* has been previously identified as possessing anti-oxidative, anti-inflammatory and mucosal protective effects. Given its potential therapeutic properties, this study serves to determine if *Solanum aethiopicum* can mitigate the adverse effects of potassium bromate exposure in the gastrointestinal system.

Given its nutritional and medicinal properties, *S. aethiopicum* represents a promising natural agent for managing metabolic disorders and promoting general health. Investigating the effects of its ethanol leaf extract on specific biomarkers such as lipid profile parameters in potassium bromate-induced toxicity in Wistar rats could provide scientific validation for its traditional use and open avenues for developing plant-based therapeutic interventions.

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MATERIALS AND METHODS

Materials

The materials used for this study included iron cages, water bottles, hand gloves, Wistar rats, standard animal feed, ceramic plates, digital weighing balance, sample bottles, centrifuge, chloroform, dissecting set, sawdust, masking tape, fresh *Solanum aethiopicum* leaves, potassium bromate, vitamin C, desiccator, cotton wool, 5 mL syringes, cannulas, distilled water, tissue paper, measuring cylinder, laboratory coat, ethanol (98–99%), marker, and twine.

Collection and Preparation of Plant Material

Fresh leaves of *Solanum aethiopicum* were purchased from a local market in the Okuku Local Government Area of Cross River State, Nigeria. The leaves were identified and authenticated by a plant taxonomist in the Department of Botany, University of Cross River State (UNICROSS). The leaves were washed thoroughly with distilled water to remove dust and debris before further processing.

Preparation of *Solanum aethiopicum* Ethanol Leaf Extract

The leaves were air-dried under shade at room temperature (27 ± 2°C) for two weeks until a constant weight was obtained. The dried leaves were pulverized into fine powder using a mechanical grinder. About 500 g of the powdered sample was soaked in 1.5 L of ethanol (98–99%) for 72 hours with intermittent shaking. The mixture was filtered first using muslin cloth and then with Whatman No. 1 filter paper. The filtrate was concentrated using a rotary evaporator under reduced pressure and later dried in a water bath to yield the crude ethanolic extract, which was stored in airtight containers at 4°C until required for the experiment.

Ethical consent

Ethical consent of the research was approved by the Research and Ethics Committee, Faculty of Basic Medical Sciences, Okuku Campus, Cross River State, Nigeria. The approved number was UNICROSS/FBMSEC/2025-HP002

Experimental Animals

A total of 85 healthy adult Wistar rats weighing 140 g–150 g were obtained from the animal house of the Physiology Department, Faculty of Basic Medical Sciences, University of Cross River State (UNICROSS), Okuku Campus. 35 rats were used to test for lipid profile, while 50 rats were used to test some parameters of gastrointestinal function. The animals were housed in well-ventilated iron cages lined with sawdust, which was changed daily. They were maintained under standard laboratory conditions (room temperature: 27 ± 2°C; relative humidity: 50–60%; 12-hour light/dark cycle) and given free access to standard feed and water ad libitum. The animals were allowed to acclimatize for 7 days before the commencement of the experiment.

Induction of Toxicity

Potassium bromate (KBr) was used to induce oxidative stress and potential cellular damage. It was administered orally at a dose of 50 mg/kg body weight once daily for two weeks prior to the commencement of treatment.

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Experimental Design and Animal Grouping

After the induction period, the animals were randomly divided into five (5) groups, each containing six (6) rats, as shown below:

Group Treatment	Description
1. Control	Received only standard feed and water.
2. KBr	Received potassium bromate only (50 mg/kg) to induce toxicity.
3. GEL LD	Received potassium bromate (50 mg/kg) and low-dose *Solanum aethiopicum* ethanolic extract (300 mg/kg).
4. GEL HD	Received potassium bromate (50 mg/kg) and high-dose *Solanum aethiopicum* ethanolic extract (600 mg/kg).
5. Vit C	Received potassium bromate (50 mg/kg) and vitamin C (100 mg/kg) as a standard antioxidant.

Groups 3,4,5 were pre-treated with potassium bromate for 2 weeks before administration of the drugs for another two weeks using an oral cannula.

Collection of Blood Samples

On the day of sacrifice, the animals were anesthetized using chloroform-soaked cotton wool placed in a desiccator. Blood samples were collected via cardiac puncture using a sterile 5 mL syringe. The samples were transferred into plain sample bottles, allowed to clot at room temperature, and then centrifuged at 3000 rpm for 10 minutes to obtain the serum. The serum aliquots were carefully separated and stored at −20°C until required for biochemical analysis.

Determination of Lipid Profile

Serum lipid profile was analyzed to evaluate the effect of treatments on lipid metabolism and oxidative stress. The parameters assessed included total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C).

Total Cholesterol (TC): TC was measured using the enzymatic colorimetric method based on the cholesterol oxidase–peroxidase (CHOD-PAP) reaction. In this assay, cholesterol esters are hydrolyzed by cholesterol esterase, followed by oxidation with cholesterol oxidase to yield hydrogen peroxide. The peroxide reacts with phenol and 4-aminoantipyrine in the presence of peroxidase to produce a colored quinoneimine complex, which was read spectrophotometrically at 500 nm.

Triglycerides (TG): TG levels were determined using the glycerol-3-phosphate oxidase–peroxidase (GPO-PAP) method. Triglycerides are enzymatically hydrolyzed by lipase to release glycerol, which is phosphorylated and oxidized to yield hydrogen peroxide. The peroxide reacts with a chromogen in the presence of peroxidase to form a stable colored complex, measured at 520 nm.

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High-Density Lipoprotein Cholesterol (HDL-C): HDL-C was quantified after selective precipitation of very low-density lipoproteins (VLDL) and low-density lipoproteins (LDL) using a phosphotungstic acid-magnesium chloride solution. The HDL fraction remaining in the supernatant was measured enzymatically by the CHOD-PAP method.

Low-Density Lipoprotein Cholesterol (LDL-C): LDL-C concentration was calculated using the Friedewald equation:

LDL-C = TC - \left(HDL-C + \frac{TG}{5} \right)

(applicable when TG values are < 400 mg/dL).

All determinations were carried out using commercially available diagnostic kits according to the manufacturer's instructions. The values obtained were expressed in mg/dL

Measurement of Mucus Gastric Output

Gastric mucus output was determined by the method described by Obembe[4]. The animals were fasted for a period of 18 hours prior to the commencement of the experiment procedure, after which they were sacrificed and their stomachs removed. The stomachs were then opened along the greater curvature and spread out on the flat board using pins to hold the edge fast. With the sue of spatula, the gastric mucus was scraped off the surface of the mucosa and introduced into a pre-weighed sterilized sample bottle containing 4 ml of normal saline. The sample bottle containing the normal saline was collected and weighed on a sensitive electronic weighing balance. Mucus output was calculated as the difference in weight of the sample bottle containing water and the sample bottle containing the normal saline and the sample bottle containing normal saline and mucus.

Gastric juice collection for pepsin activity determination

The gastric juice analysis of pepsin was collected according to the method of Okon [5]. The animals were starved for 18 hours to ensure that their stomachs were completely empty. Water was, however, allowed. The rats were then anesthetized using chloroform, and their abdomens were shaved. A midline incision was made through the linea alba to minimize bleeding, extending 2 cm downward from the xyphoid to the pyloric junction. It was picked gently with a curved probe; the stomach was not disturbed. Proper care was taken to ensure no damage is done to blood vessels and the stomach. The abdomen was then closed with an uninterrupted structure. The animal's wounds were cleaned thoroughly and recovered after 10 minutes. 4 hours later, the animals were anesthetized with chloroform. The abdomen was opened, the duodenum and appropriate peritoneal ligature were damped, an opening was made along the greater curvature, and gastric juice was drained into a tube.

Measurement of pepsin activity and total acidity

Gastric juice (1 ml) was taken into a 100 ml conical flask; to this 2-3 drops of Topfer's reagent were added and titrated with 0.01 N NaOH until all traces of red color disappeared and the color of the solution turned yellow-orange (end point). The volume of alkali added was noted. This volume corresponds to free acidity. 2-3 drops of phenolphthalein solution were added, and titration was continued until a definite red tinge reappeared. The volume of alkali was noted, which corresponds to total acidity.

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Calculations:

Acidity was calculated by using the formula:

$$\text{Acidity (mEq/litre)} = \frac{Volume\ fo\ NaOH\ x\ Normality\ of\ NaOH\ x\ 100}{0.1}$$

Estimation of pepsin activity

For pepsin estimation, 4 placed test tubes were used: (1) and (2) containing 5 ml of 1% bovine albumin in 0.01 M HCl, and (3) and (4) containing 10 ml of 0.35 M trichloroacetic acid. The gastric juice was mixed with an equal volume of 0.01 M HCl warmed to 37°C. 1 ml of this mixture was added to each of the test tubes of (1) and (4) and incubated for 15 minutes. At the end, the mixed content of tubes (1) and (3) was allowed to stand for about 4 minutes. (1) + 3 in the test, and (2) + (4) gives blank. The mixture was filtered. To 2 ml of filtrate, 10 ml of NaOH was added. Then 1 ml of phenol reagent was added and mixed by gentle rotation. After 30 minutes, the absorbance was measured at 680 nm. The difference between the test and the blank gives the measures of peptic acidity.

Measurement of Intestinal Transit

The rate of intestinal transit was measured by using the Charcoal Meal Test. A dose of activated charcoal mixed with 10% suspension of gum acacia was administered orally, and the distance travelled by the charcoal was recorded after 30 minutes. The percentage of total length of the small intestine traveled by the charcoal was calculated as an indicator of motility. Loperamide was used as a standard drug since it is used medically to treat diarrhea (0.4 mg/kg body weight).

All treatments were oral and once daily using an oral gavage and lasted for 14 consecutive days before the ulcer and extractable mucus studies were carried out.

Acute Toxicity Study (LD50)

Method [6] was adopted for the acute toxicity testing in albino Wistar rats used for the study. Five groups of 5 rats, each weighing 100-190 g of body weight, were orally dosed in different gradual doses of 10-10,000 mg/kg of body weight. The animals were observed for 24 hours, after which the numbers of the dead rats were counted in each group and the percentage mortality calculated. The LD50 was used to determine the dosage of the drugs.

Statistical Analysis

All data obtained were expressed as mean ± standard deviation (SD). Statistical analyses were performed using one-way analysis of variance (ANOVA), followed by Tukey's post hoc test to compare differences between groups. Differences were considered statistically significant at $p < 0.05$.

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RESULTS

This study assessed the effects of *Solanum aethiopicum* ethanolic leaf extract (GEL) on lipid profile parameters in potassium bromate (KBr)-induced toxicity in Wistar rats. The lipid parameters evaluated were total cholesterol, high-density lipoprotein cholesterol (HDL-C), triglycerides (TAG), low-density lipoprotein cholesterol (LDL-C), and very-low-density lipoprotein cholesterol (VLDL-C).

The results are summarized below:

Table 3.1: Effects of treatments on lipid profile parameters in potassium bromate-induced rats

Groups	Treatment	Cholesterol (mg/dl)	HDL-C (mg/dl)	TAG (mg/dl)	LDL-C (mg/dl)	VLDL-C (mg/dl)
1	Control	81.12 ± 0.51^{b}	65.75 ± 0.14^{b}	72.43 ± 0.69^{a}	0.89 ± 0.59^{b}	14.49 ± 0.14^{a}
2	KBr	115.51 ± 0.27^{d}	65.39 ± 0.43^{b}	91.27 ± 0.29^{c}	31.87 ± 0.13^{c}	18.25 ± 0.06^{c}
3	KBr + GEL LD	69.06 ± 1.82^{a}	68.38 ± 0.12^{d}	79.80 ± 0.36^{b}	-15.28 ± 1.99^{a}	15.96 ± 0.07^{b}
4	KBr + GEL HD	86.83 ± 0.85^{c}	67.01 ± 0.21^{c}	79.84 ± 0.42^{b}	3.85 ± 0.95^{b}	15.97 ± 0.09^{b}
5	KBr + VIT C	124.57 ± 1.03^{c}	63.68 ± 0.52^{a}	97.86 ± 0.66^{d}	41.31 ± 1.10^{d}	19.57 ± 0.13^{d}

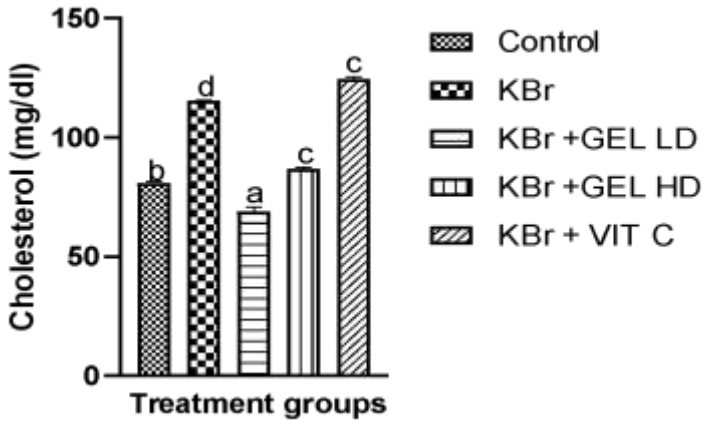
$$\text{VLDL-C} = \frac{\text{TAG}}{5}$$

LDL-C = Total cholesterol – (HDL-C + VLDL-C)

Total Cholesterol Levels Across Groups

The control group recorded 81.12 ± 0.51 mg/dl of cholesterol. KBr administration significantly ($P<0.05$) increased cholesterol to 115.51 ± 0.27 mg/dl, confirming hypercholesterolemia. Treatment with GEL reduced cholesterol levels to 69.06 ± 1.82 mg/dl (LD) and 86.83 ± 0.85 mg/dl (HD). However, Vitamin C treatment further increased cholesterol to 124.57 ± 1.03 mg/dl, higher than all groups

Figure 1: Effect of Solanum aethiopicum on total cholesterol (TC) in potassium bromate-induced toxicity in wistar rats . Values are expressed as Mean ± SEM (n=7).



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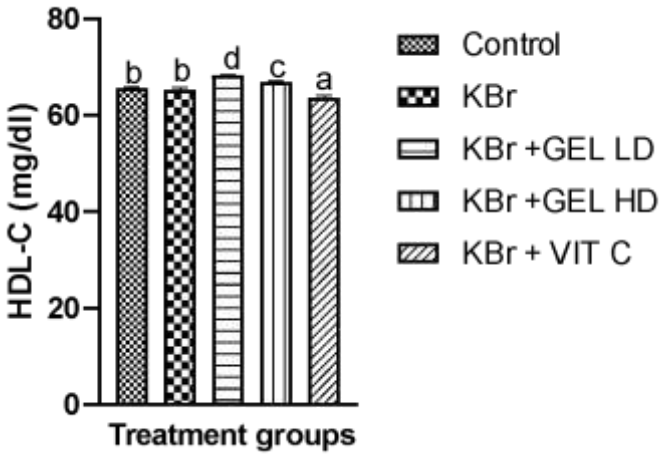
HDL-C Levels Across Groups

The control group had 65.75 ± 0.14 mg/dl of HDL-C, while KBr slightly reduced it to 65.39 ± 0.43 mg/dl. GEL treatment markedly elevated ($p<0.05$) HDL-C: 68.38 ± 0.12 mg/dl (LD) and 67.01 ± 0.21 mg/dl (HD). In contrast, Vitamin C reduced HDL-C to 63.68 ± 0.52 mg/dl, the lowest among all groups.

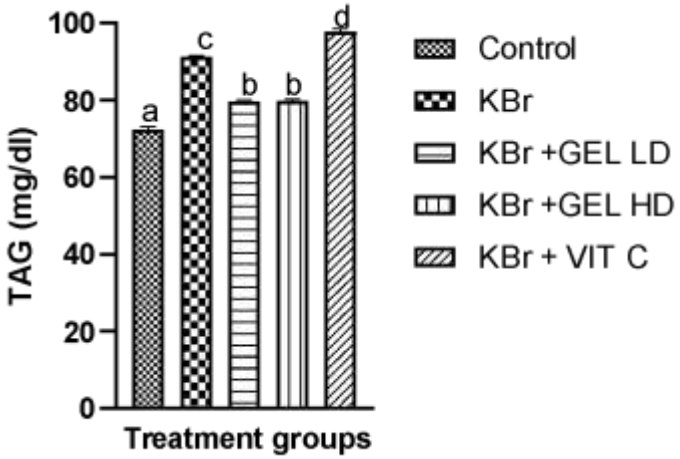
Figure 2: Effect of Solanum aethiopicum on HDL-C in potassium bromate-induced toxicity in Wistar rats. Values are expressed as mean ± SEM (n=7).



Triglyceride (TAG) Levels Across Groups

The control group recorded 72.43 ± 0.69 mg/dl TAG, whereas KBr elevated it to 91.27 ± 0.29 mg/dl. GEL treatment reduced TAG levels to 79.80 ± 0.36 mg/dl (LD) and 79.84 ± 0.42 mg/dl (HD). Vitamin C treatment increased TAG further to 97.86 ± 0.66 mg/dl, the highest value observed.

Figure 3: Effect of treatments of Solanum aethiopicum on triglycerides (TAG) in potassium-induced toxicity in Wistar rats.



Values are expressed as mean ± SEM (n=7).

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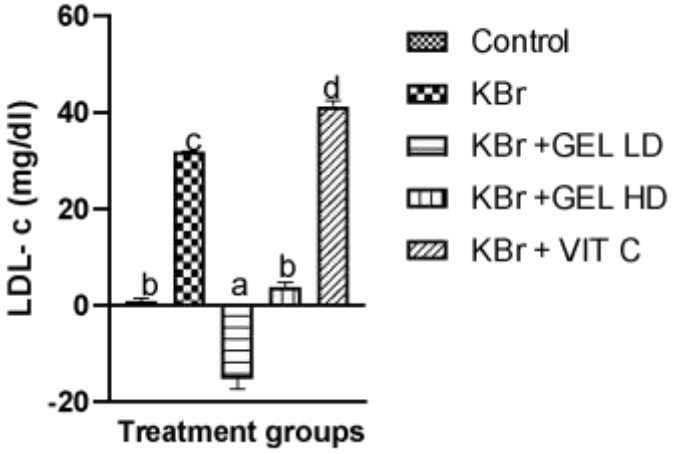
LDL-C Levels Across Groups

LDL-C in the control group was 0.89 ± 0.59 mg/dl but rose significantly to 31.87 ± 0.13 mg/dl in the KBr group. GEL treatment drastically reduced (P<0.05) LDL-C to -15.28 ± 1.99 mg/dl (LD) and 3.85 ± 0.95 mg/dl (HD). Vitamin C treatment resulted in the highest LDL-C level of 41.31 ± 1.10 mg/dl.

Figure 4: Effect of treatments with Solanum aethiopicum on LDL-C in potassium bromate-induced toxicity in Wistar rats. Values are expressed as mean ± SEM (n=7).



VLDL-C Levels Across Groups

VLDL-C was 14.49 ± 0.14 mg/dl in the control group. KBr elevated it to 18.25 ± 0.06 mg/dl. GEL treatment normalized VLDL-C to 15.96 ± 0.07 mg/dl (LD) and 15.97 ± 0.09 mg/dl (HD). Vitamin C treatment increased VLDL-C to 19.57 ± 0.13 mg/dl, the highest recorded.

Figure 5: Effect of Solanum aethiopicum VLDL-C in potassium bromate-induced toxicity in Wistar rats. Values are expressed as mean ± SEM (n=7).

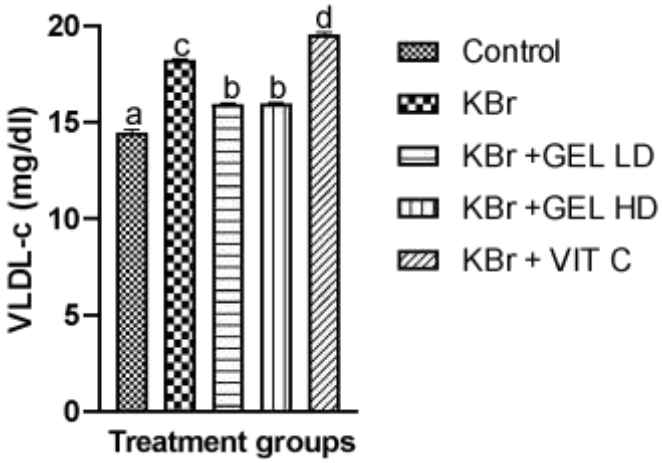


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Table 3.2 Potential effect of aqueous extract of *Solanum aethiopicum* on pepsin, total acidity and mucus secretion *in potassium bromate-induced toxicity in Wistar rats.*

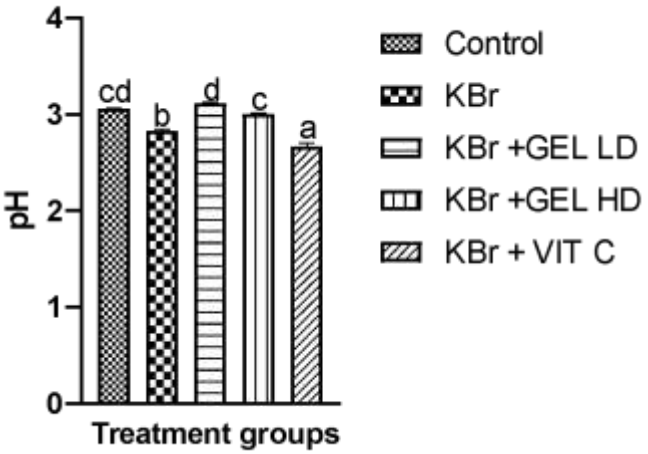
Groups	Treatment	pH	Pepsin activity (µmol tyrosine/ml)	Total acidity (mmol/L)	Extractible mucus weight (mg)
1	Control	3.06±0.01^{cd}	14.19±0.07^{a}	73.18±0.33^{a}	41.20±0.21^{d}
2	KBr	2.83±0.02^{b}	14.74±0.06^{b}	75.77±1.10^{b}	37.33±0.35^{b}
3	KBr + GEL LD	3.12±0.01^{d}	14.17±0.09^{a}	73.02±0.08^{a}	41.43±0.08^{d}
4	KBr + GEL HD	3.01±0.01^{c}	14.49±0.05^{ab}	72.63±0.14^{a}	40.20±0.11^{c}
5	KBr + VIT C	2.67±0.04^{a}	17.79±0.25^{c}	82.76±0.89^{c}	35.10±0.24^{a}

Values are mean ± SEM (n=10). Those with different superscripts (a, b, c) along the columns are statistically significant at P<0.05. While those with the same superscripts are not significant at P>0.05.

Effect of *Solanum aethiopicum* Extract on pH Level *in Potassium Bromate-Induced Toxicity in Wistar Rats*

The mean ± SEM values for control, potassium bromate, LD, HD, and Vit. C were 3.06±0.01, 2.83±0.02, 3.12±0.01, 3.01±0.01 and 2.67±0.04, respectively. The test result showed that there was a significant (P<0.05) decrease in the test group (KBrO) compared to the control group. In the test groups, LD and HD, there was a significant (P<0.05) increase in pH level. However, in the test group (Vit. C), there was a significant decrease when compared to the control. The result showed that there was a significant (P<0.05) increase in the test group in control, KBr, LD and HD when compared to the Vit. C group.

Figure 6: Effect of Solanum aethiopicum Extract on pH Level in Potassium Bromate-Induced Toxicity in Wistar Rats. Values are mean ± SEM (n=10). Those with different superscripts (a, b, c) along the columns are statistically significant at P<0.05. While those with the same superscripts are not significant at P>0.05.



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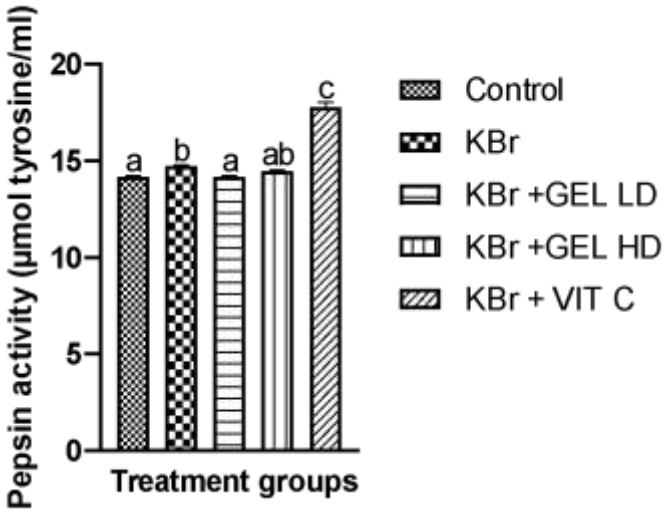
Effect of *Solanum aethiopicum* leaf extract on pepsin activity *in potassium bromate-induced toxicity in Wistar rats.*

The mean ± SEM values for control, potassium bromate, LD, HD and Vit. C of GEL extract were 14.19±0.7, 14.74±0.06, 14.17±0.09, 14.49±0.5 and 17.79±0.25, respectively. The result showed that there was a significant (P<0.05) increase in the test groups (KBr and Vit. C) when compared to the control. There was also a slight significant (P<0.05) increase in the test group HD when compared to the control. In the test group, LD of gel extract showed no significant (P<0.05) increase when compared to the control. The result showed that there was a significant (P<0.05) decrease in the test groups KBr and HD when compared to the Vit. C group. In the test groups, control and LD showed no significance.

Figure 7: Effect of Solanum aethiopicum leaf extract on pepsin activity in potassium bromate-induced toxicity in Wistar rats



Values are mean ± SEM (n=10). Those with different superscripts (a, b, c) along the columns are statistically significant at P<0.05. While those with the same superscripts are not significant at P>0.05.

Effect of *Solanum aethiopicum* leaf extract on Total Acidity (mmol/L) *in potassium bromate-induced toxicity in Wistar rats*

The mean ± SEM values for control, KBr, LD, HD and Vit. C of gel extract were 73.18±0.33, 75.77±1.10, 73.02±0.08, 72.63±0.14 and 82.76±0.89, respectively. The result showed that there was an increase in the test group (KBr) when compared to the control. However, the test group (Vit. C) showed a significant (P<0.05) increase when compared to KBr. The test groups LD and HD showed no significant difference (P<0.05) when compared to the control. The test result showed that there was a significant (P<0.05) decrease in the test group KBr when compared to the Vit. C group. However, in the test groups' control, LD and HD showed no significance when compared to Vit. C.

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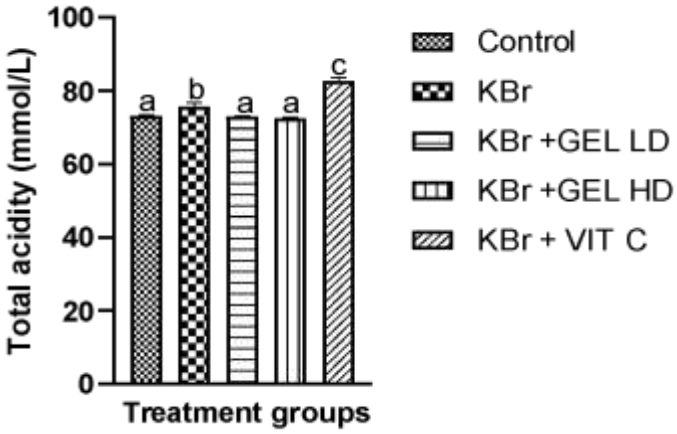
Figure 8: Effect of Solanum aethiopicum leaf extract on Total Acidity (mmol/L) in potassium bromate-induced toxicity in Wistar rats

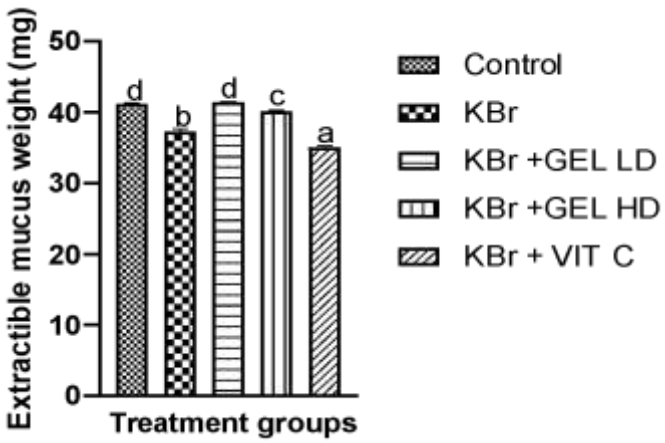


Values are mean ± SEM (n=10). Those with different superscripts (a, b, c) along the columns are statistically significant at P<0.05. While those with the same superscripts are not significant at P>0.05.

Effect of *Solanum aethiopicum* on Extractable Mucus Weight (mg) *in Potassium Bromate-Induced Toxicity in Wistar Rats*

The mean ± SEM values for control, KBr, LD, HD and Vit. C of gel extract were 41.20±0.21, 37.33±0.35, 41.43±0.68, 40.20±0.11 and 35.10±0.24, respectively. The test result showed a significant (P<0.05) decrease in the test groups (KBr and Vit C) when compared to the control. The test group (low dose) showed no significance compared to the control group, while a slight increase was observed in the high dose. The test result showed that there was a significant (P<0.05) increase in the test group's HD when compared to the Vit. C group. A slight significant increase in the KBr group. The test groups, control and LD, showed no significance when compared to Vit. C.

Figure 9: Effect of Solanum aethiopicum leaf extract on Extractable Mucus Weight (mg) in potassium bromate-induced toxicity in Wistar rats. Values are mean ± SEM (n=10). Those with different superscripts (a, b, c) along the columns are statistically significant at P<0.05. While those with the same superscripts are not significant at P>0.05.



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Effect of Solanum aethiopicum leaf extract on intestinal motility *in potassium bromate-induced toxicity in Wistar rats*

The mean ± SEM values for control, KBr, loperamide, LD and HD of gel extract were 70.87±0.28, 90.17±0.62, 56.42±0.85, 86.46±1.04 and 87.15±0.28, respectively. The test result showed that there was a significant increase ($P<0.05$) in the test group (KBr) when compared to the control. In the test (Loperamide + KBr), there was a significant ($P<0.05$) decrease when compared to the KBr group. The test groups LD and HD showed a slight increase when compared to the control. The test result showed that there was a significant ($P<0.05$) increase in the test groups' control and KBr when compared to Vit. C. The test groups LD and HD showed no significance. But in the test group (Loperamide), a significant ($P<0.05$) decrease was observed when compared with Vit. C.

Intestinal transit

Figure 10: Effect of Solanum aethiopicum leaf extract on intestinal motility in potassium bromate-induced toxicity in Wistar rats.

Groups	Treatment	Length of small intestine (cm)	Distance traveled by charcoal meal (cm)	Percentage intestinal transit
1	Normal control	92.67 ± 1.43^{b}	65.67 ± 0.97^{cd}	70.87 ± 0.28^{cd}
2	$KBrO_3$ (50 mg/kg) only	93.33 ± 0.97^{bc}	84.00 ± 0.63^{g}	90.17 ± 0.62^{g}
3	Loperamide (0.5 mg/kg) + $KBrO_3$ (50 mg/kg)	88.67 ± 1.02^{a}	50.00 ± 0.84^{a}	56.42 ± 0.85^{a}
4	Garden egg leaf extract (300 mg/kg) + $KBrO_3$ (50 mg/kg)	93.33 ± 0.48^{bc}	80.67 ± 0.66^{f}	86.46 ± 1.04^{f}
5	Garden egg leaf extract (600 mg/kg) + $KBrO_3$ (50 mg/kg)	93.33 ± 0.66^{bc}	81.33 ± 0.48^{f}	87.15 ± 0.28^{f}

Values are mean ± SEM (n=10). Those with different superscripts (a, b, c) along the columns are statistically significant at $P<0.05$. While those with the same superscripts are not significant at $P>0.05$.

DISCUSSION AND CONCLUSION

Discussion

This study was aimed at investigating the effect of *Solanum aethiopicum* on lipid profile and gastro-protection. The present results show that potassium bromate ($KBrO_3$) administration profoundly altered serum lipid parameters. Animals treated with $KBrO_3$ alone displayed significant increases in total cholesterol, triglycerides, very-low-density lipoprotein cholesterol (VLDL-C), and low-density lipoprotein cholesterol (LDL-C), accompanied by a reduction in high-density lipoprotein cholesterol (HDL-C). These changes are consistent with earlier reports that $KBrO_3$ disrupts hepatic lipid homeostasis and promotes dyslipidemia through oxidative stress–mediated mechanisms [7, 8].

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Oxidative stress is central to these alterations. $KBrO_3$ readily generates reactive oxygen species (ROS), which attack membrane phospholipids and circulating lipoproteins, initiate lipid peroxidation, and impair key enzymes of cholesterol and triglyceride metabolism. Such ROS-driven injury favors accumulation of atherogenic fractions—LDL-C and VLDL-C—while lowering the protective HDL-C fraction [9]. The lipid profile observed in the $KBrO_3$-only group therefore reflects a shift toward increased cardiovascular risk

Administration of the *Solanum aethiopicum* produced a marked, dose-dependent improvement. High-dose treatment restored total cholesterol and triglyceride concentrations close to control values, significantly lowered LDL-C and VLDL-C, and elevated HDL-C. These effects suggest that the *Solanum aethiopicum* contains bioactive constituents capable of either enhancing antioxidant defenses or modulating lipid regulatory pathways. Comparable improvements in serum lipids have been reported for plant-derived polyphenols and other natural antioxidants, which can inhibit lipid peroxidation, up-regulate hepatic LDL receptors, and improve lipoprotein clearance **[10, 11].**

Vitamin C supplementation, by contrast, failed to prevent the $KBrO_3$-induced dyslipidemia and in some measures coincided with further lipid elevation. Although vitamin C is a recognized antioxidant, its effect on serum lipids is inconsistent and can depend on dosage and the oxidative environment **[12].** Under severe ROS burden, vitamin C may even exhibit pro-oxidant behavior [13], which could explain its limited efficacy in the current context.

Taken together, these findings indicate that $KBrO_3$ promotes a pro-atherogenic lipid profile primarily through oxidative mechanisms, whereas the experimental gel exerts a significant corrective influence, particularly at higher doses. The lack of benefit from vitamin C highlights the need for more robust or multi-target antioxidant strategies to counteract bromate-induced dyslipidemia.

The result of this study also showed that *Solanum aethiopicum* possessed a significant protective effect against the gastrointestinal toxicity induced by potassium bromate (KBrO). It significantly increased pH level and mucus secretion while reducing the acidity and secretion of pepsin in the stomach, especially at low doses. The high dose was more effective in lowering total acidity. In the intestine, garden egg leaf extract produced only a modest reduction in hypermotility, with values remaining higher than those of the normal control, indicating a partial but not complete normalization of intestinal transit. Normal transit was restored by loperamide, which is a standard drug for antimotility and antidiarrheal effects.

These results findings are consistent with the previous studies that have reported antioxidant and gastroprotective effects of *Solanum aethiopicum*[14]. The extract restores pH level to its normal range of 1.5-3.5, which is suitable for antimicrobial effect, pepsin activation and nutrient absorption in the stomach. For pepsin secretion, the effect of KBrO is that it damages the gastric fundic mucosa, which houses the chief cells that secrete pepsin, and it also promotes gastric ulcers [15]. KBr also damages the small intestine, as seen from the above histology results, where it destroys and sloughs off the villi, resulting in diarrhea. Garden egg leaf extract reduces gastric acid volume and pepsin output in ulcer models and also promotes ulcer healing, contributing to its antiulcerogenic activity. Hypersecretion of stomach acid can lead to peptic ulcer disease (PUD), gastritis, GERD, bleeding and also delayed healing. For mucus secretion, a high dose of vitamin C reduced mucus secretion, which may result in the loss of the protective barrier that shields the stomach lining from acid (hydrochloric acid) and pepsin, increasing the

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risk of gastric injury resulting in ulcer formation and also greater oxidative stress as well as reduced wound healing. Whereas gel extract increases its output at low doses, which may help to maintain the integrity of the gastrointestinal mucosa and potentially support mucus production as a protective barrier against irritants and pathogens.

Report [14] about the antiulcer activity of *Solanum aethiopicum* correlates with our results. The extract has shown antidiarrheal effects, majorly due to its phytochemical composition of phenols, saponins, tannins, and alkaloids [16]. Flavonoids and phenols enhance prostaglandin-mediated cytoprotection, scavenge ROS, and suppress acid/pepsin, while tannins form a protective protein layer over lesions and tighten mucosal barrier function. These routes predict reduced ulcer index and secretion aggressors with increased mucus and pH [16]. In comparison to the ameliorative effect of vitamin C, garden egg leaf extract is clearly superior as an ameliorative agent to vitamin C. It improves protective factors (pH, mucus) and reduces aggressive ones (pepsin and total acidity). Vitamin C in contrast, worsened acidity, increased pepsin, and depleted mucus, thereby aggravating gastric damage under potassium bromate-induced stress. While loperamide, which is an antidiarrheal drug, decreases the motility rate and fosters better absorption in the GI tract. Loperamide is an over-the-counter antidiarrheal medication commonly used to manage acute or chronic diarrhea, including traveler's diarrhea and diarrhea associated with inflammatory bowel diseases [17]. It works by slowing down intestinal motility by acting on opioid receptors in the gut wall, increasing absorption of water and electrolytes, reducing stool frequency and improving stool consistency.

Conclusion

Potassium bromate caused marked dyslipidemia, raising total cholesterol, triglycerides, LDL-C, and VLDL-C while lowering HDL-C. High-dose *Solanum aethiopicum* treatment significantly reversed these changes, whereas vitamin C showed no benefit, indicating the strong protective effect of Solanum aethiopicum on lipid metabolism. *Solanum aethiopicum* (garden egg leaf) extract may have an ameliorative effect, which is beneficial in cases of gastric ulcers, particularly at low doses. At high doses, it may have a weaker or even opposing effect. In contrast, Vitamin C co-treatment failed to protect against potassium bromate-induced gastric injury and instead aggravated gastric acidity Therefore, its use should be in moderation.

Declaration of conflict of interest

Authors hereby declare that there is no conflict of interest.

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