WWW.IJRPR.COM

International Journal of Research Publication and Reviews

Journal homepage: www.ijrpr.com ISSN 2582-7421

Evaluation of Testicular Oxidative Stress and Reproductive Profiles in Potassium Bromate-Treated Male Rats Supplemented with *Vernonia amygdalina* and *Celosia argentea*

Ibezute Albert Chukwuemeka[1] and Kingsley, Akpeh Peter[2]

[1]Department of Environmental Management and Toxicology, College of Sciences, Federal University of Petroleum Resources, P.M.B. 1221, Effurun, Delta State, Nigeria.

[2]Department of Biochemisty, Faculty of Life Sciences, University of Benin, P.M.B. 1221, Benin City, Edo State, Nigeria.

Email: emekaibezute2@gmail.com; kingsley.akpeh@uniben.edu

ABSTRACT

This study investigates the effects of potassium bromate (PB) exposure on oxidative stress, hormonal profile, and seminiferous fluid analysis in male Wistar rats, with a focus on potential protective effects of bitter leaf (BTL) and Shoko (SHK) treatments. PB exposure significantly reduced antioxidant enzyme activities, including superoxide dismutase (SOD) from 2.10 ± 0.08 U/g in the control to 1.41 ± 0.16 U/g ($P < 0.05$), and catalase (CAT) from 1.43 ± 0.07 U/g to 1.06 ± 0.20 U/g. Hydrogen peroxide (H_2O_2) levels increased significantly from 34.30 ± 5.78 µg/mL in the control to 40.27 ± 2.5 µg/mL ($P < 0.05$). Hormonal analysis revealed significant reductions in testosterone from 3.00 ± 0.20 nmol/L to 2.20 ± 0.10 nmol/L ($P < 0.05$), and estrogen from 1.00 ± 0.10 pg/mL to 0.70 ± 0.00 pg/mL ($P < 0.05$) in PB-exposed rats, suggesting endocrine disruption. Seminiferous fluid analysis showed a decrease in sperm count from 192.00 ± 21.50 × 10^6 cells/ml in the control to 116.00 ± 14.00 × 10^6 cells/ml ($P > 0.05$), and a significant increase in progressive motility from 25.00 ± 10.00% in the control to 47.50 ± 12.50% with BTL and SHK ($P < 0.05$). Treatment with BTL and SHK improved sperm motility and partially mitigated oxidative stress but did not fully restore hormonal balance or sperm count. These findings confirm the role of oxidative stress in PB-induced reproductive toxicity and highlight the partial protective effects of BTL and SHK.

Keywords: Potassium bromate, oxidative stress, testicular tissue, antioxidant, bitter leaf, Shoko, sperm motility, hormonal profile, reproductive toxicity, Wistar rats.

1. Introduction

Reproductive health is a crucial aspect of overall well-being, yet it remains susceptible to environmental and chemical stressors. Potassium bromate ($KBrO_3$), a compound extensively utilized in the food and beverage industries as a flour enhancer and water disinfectant, has raised significant health concerns due to its potent oxidative properties. Exposure to potassium bromate is associated with the generation of reactive oxygen species (ROS), leading to oxidative stress and cellular damage, particularly in tissues with high metabolic activity, such as the testes (Kurokawa *et al*., 1990). The implications of such oxidative damage include disruptions in spermatogenesis, hormonal imbalances, and impaired fertility, thereby highlighting the need for strategies to mitigate these adverse effects (Ahmed *et al*., 2021).

Oxidative stress, characterized by an imbalance between ROS production and the antioxidant defense system, is a major factor in testicular dysfunction and male infertility. Excessive ROS can induce lipid peroxidation, protein oxidation, and DNA fragmentation in the testes, resulting in reduced sperm count, motility, and morphology (Agarwal *et al*., 2014). Moreover, oxidative stress can impair Leydig cell function, leading to alterations in testosterone synthesis and overall hormonal regulation (Desai *et al*., 2009). These detrimental effects underscore the urgency of identifying effective antioxidants to counteract the oxidative damage induced by potassium bromate exposure.

Vernonia amygdalina, commonly known as bitter leaf, has been widely recognized for its medicinal properties, particularly its antioxidant potential. Rich in bioactive compounds such as flavonoids, alkaloids, and saponins, *Vernonia amygdalina* has demonstrated the ability to scavenge free radicals, enhance endogenous antioxidant enzyme activity, and reduce lipid peroxidation (Erasto *et al*., 2007). Similarly, *Celosia argentea*, a leafy vegetable with significant medicinal value, contains phenolic compounds and flavonoids that contribute to its antioxidative capabilities. Studies have shown that *Celosia argentea* can mitigate oxidative stress by improving antioxidant enzyme levels and protecting cellular integrity (Iwalewa *et al*., 2016).

The reproductive benefits of these plants extend beyond their antioxidative properties. *Vernonia amygdalina* has been reported to improve sperm quality and boost testosterone levels, suggesting its role in enhancing male fertility (Owolabi *et al*., 2008). Likewise, *Celosia argentea* has shown

potential in maintaining testicular function and promoting healthy reproductive outcomes in animal models (Ezekiel *et al*., 2020). These findings indicate that the combined use of these plants may offer a synergistic approach to ameliorating reproductive damage induced by oxidative stress.

This study aims to evaluate the testicular oxidative stress and reproductive profiles in potassium bromate-treated male rats supplemented with *Vernonia amygdalina* and *Celosia argentea* leaf extracts. By analyzing hormonal profiles, semen quality, and oxidative stress biomarkers, the research seeks to elucidate the protective mechanisms of these plants and their potential as therapeutic agents in preserving male reproductive health.

2. Materials and Methods

2.1 Potassium bromate Toxicity and Dosage

Potassium bromate was prepared as a 100 mg/kg solution in distilled water. This dosage selection aligns with methodologies reported in earlier studies (Ibezute and Marcus-Abdul, 2025). Details on the selection of sodium nitrite dosage and preparation have been elaborated in Ibezute and Marcus-Abdul (2025) and were referenced in planning this experiment

2.2 Plant Extraction

The preparation of *Vernonia amygdalina* and *Celosia argentea* extracts was performed following protocols documented in Ibezute and Marcus-Abdul (2025), ensuring reproducibility of results. Plant extraction techniques mirrored those established in Ibezute and Marcus-Abdul (2025), incorporating validated methods for aqueous extraction.

2.3 Experimental Animal Housing and Experimental Design

The experimental subjects were male Wistar rats (6-7 weeks, weighing 125g to 150g), obtained from the University of Benin's Animal House. Following arrival, they were acclimatized in standard laboratory conditions for two weeks, with a 12-hour light/dark cycle, temperature set at 22 ± 2°C, and humidity maintained at 50 ± 5%. The rats were housed in wooden cages with wire mesh covers and provided with ad libitum access to commercial rodent chow and distilled water. Their body weight was measured at the start and end of the acclimatization period. After this period, the rats were divided into four treatment groups:

- **Group A**: Control
- **Group B**: Petassium Bromate
- **Group C**: Petassium Bromate + *Vernonia amygdalina* extract
- **Group D**: Petassium Bromate + *Celosia argentea* extract

The Petassium Bromate solution was prepared in distilled water at 90 mg/kg body weight. The *Vernonia amygdalina* and *Celosia argentea* extracts were administered at 150 mg/kg and 100 mg/kg, respectively. Treatment continued for 60 days, with each substance being administered every 48 hours. Afterward, blood was collected for biochemical analysis, and serum was separated by centrifugation at 3,000 rpm for 10 minutes.

2.4 Biochemical and Oxidative Stress Markers

Serum concentrations of testosterone, estrogen, follicle-stimulating hormone (FSH), and luteinizing hormone (LH) were evaluated as hormonal profile markers. Oxidative stress markers, including hydrogen peroxide, malondialdehyde (MDA), superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), glutathione (GSH), total antioxidant capacity (TAC), and vitamin C, were assessed in testicular tissues. The testes were homogenized in a chilled buffer, followed by centrifugation to extract the supernatant. These markers were analyzed using commercial kits from Human Diagnostics (Germany), and absorbance was measured with an OPTIMA SP-300 spectrophotometer (Japan). The values obtained were normalized against protein concentration in the tissue homogenates for consistency and accuracy. Sperm cells were isolated from the vas deferens of euthanized rats using pre-warmed normal saline (37 °C). The suspension was analyzed for motility under a microscope at x20 and x40 magnifications, categorizing cells into progressive, non-progressive, and immotile groups. Vitality was assessed via eosin-nigrosine staining, where live cells appeared white and dead cells stained red under an oil immersion lens (x100).

2.5 Statistical Methods

The data were analyzed using SPSS version 25 and Microsoft Excel software. Results were presented as mean ± standard error (SE). To assess statistical differences, one-way analysis of variance (ANOVA) was performed. Post-hoc comparisons were made using the Duncan Multiple Range Test. A significance level of $p < 0.05$ was considered statistically significant.

3. Results and Discussions

Potassium bromate exposure significantly reduced Superoxide Dismutase (SOD) activity (1.41 ± 0.16 U/g) compared to the control (2.10 ± 0.08 U/g, $P < 0.05$) (Table 1). Treatment with BTL and SHK restored SOD to 1.72 ± 0.10 U/g and 2.15 ± 0.17 U/g, respectively, with SHK showing full recovery ($P < 0.05$). Catalase (CAT) activity decreased under PB exposure (1.06 ± 0.20 U/g) compared to the control (1.43 ± 0.07 U/g), but both BTL (1.14 ± 0.01 U/g) and SHK (1.46 ± 0.04 U/g) improved CAT levels, though differences were not significant ($P > 0.05$). Glutathione Peroxidase (GPx) activity dropped with PB (4.17 ± 0.81 U/g) versus the control (5.66 ± 0.25 U/g) but was elevated by SHK (5.83 ± 0.15 U/g, $P > 0.05$). Malondialdehyde (MDA), a marker of lipid peroxidation, was lower in PB-exposed rats (0.34 ± 0.03 mol/g) than the control (0.47 ± 0.01 mol/g), yet it increased significantly with SHK (0.69 ± 0.01 mol/g, $P < 0.05$). Reduced Glutathione declined with PB exposure (37.14 ± 6.67 µg/mL) compared to the control (48.57 ± 3.33 µg/mL) but improved with BTL (40.48 ± 0.48 µg/mL) and SHK (41.43 ± 0.48 µg/mL, $P > 0.05$). Vitamin C levels decreased significantly with PB (6.57 ± 1.46 µg/mL) compared to the control (11.68 ± 1.46 µg/mL, $P < 0.05$), but BTL treatment increased it significantly (16.42 ± 0.36 µg/mL). TAC dropped with PB (43.08 ± 0.94 µg/mL) compared to the control (49.37 ± 2.67 µg/mL), but BTL (52.99 ± 0.16 µg/mL) and SHK (45.91 ± 3.77 µg/mL) improved TAC levels ($P > 0.05$). Hydrogen Peroxide (H_2O_2) levels rose significantly with PB (40.27 ± 2.5 µg/mL) compared to the control (34.30 ± 5.78 µg/mL, $P < 0.05$) and further increased with SHK (52.50 ± 4.14 µg/mL). These findings confirm oxidative stress induced by PB and the mitigating, though varied, effects of BTL and SHK.

Table 1: Oxidative stress and Reactive oxidative species in the testis tissue of wistar rats given potassium bromate food preservatives and possible abatements

	CTR	PB	PB+BTL	PB+SHK	P-Value
Superoxide Dismutase (U/g)	2.10±0.08	1.41±0.16	1.72±0.10	2.15±0.17	P<0.05
Catalase (U/g)	1.43±0.07	1.06±0.20	1.14±0.01	1.46±0.04	P>0.05
Glutathione Peroxidase (U/g)	5.66±0.25	4.17±0.81	4.49±0.04	5.83±0.15	P>0.05
Malondialdehyde (mol/g)	0.47±0.01	0.34±0.03	0.59±0.06	0.69±0.01	P<0.05
Reduced Glutathione (µg/mL)	48.57±3.33	37.14±6.67	40.48±0.48	41.43±0.48	P>0.05
Vitamin C (µg/mL)	11.68±1.46	6.57±1.46	16.42±0.36	11.68±0.00	P<0.05
Protein (g/dL)	0.66±0.03	0.93±0.18	0.83±0.01	0.64±0.02	P>0.05
Total Antioxidant Capacity (µg/mL)	49.37±2.67	43.08±0.94	52.99±0.16	45.91±3.77	P>0.05
Hydrogen Peroxide (µg/mL)	34.30±5.78	40.27±2.5	37.09±0.1	52.50±4.14	P<0.05

The results from Table 2 indicate that potassium bromate (PB) exposure significantly altered the hormonal profile of Wistar rats. Testosterone levels dropped from 3.00 ± 0.20 nmol/L in the control to 2.20 ± 0.10 nmol/L in PB-exposed rats ($P < 0.05$), with further reduction observed when PB was combined with bitter leaf (BTL, 1.55 ± 0.15 nmol/L) and Shoko (SHK, 1.90 ± 0.10 nmol/L). This suggests that PB disrupts testosterone production, and while SHK partially mitigates the effect, BTL exacerbates it. For LH, PB exposure reduced levels from 2.90 ± 0.55 IU/L in the control to 1.50 ± 0.10 IU/L, with no significant changes when combined with BTL (1.10 ± 0.10 IU/L) or SHK (1.30 ± 0.10 IU/L), indicating that the treatments had little impact on LH restoration. FSH levels decreased from 2.30 ± 0.45 IU/L in the control to 1.15 ± 0.05 IU/L in PB-exposed rats, with further reduction when combined with BTL (0.85 ± 0.05 IU/L) and a slight increase when combined with SHK (1.00 ± 0.10 IU/L). This suggests BTL worsened PB-induced suppression, while SHK provided minimal amelioration. Estrogen levels decreased from 1.00 ± 0.10 pg/ml in the control to 0.70 ± 0.00 pg/ml in PB-treated rats, with further reduction observed with BTL (0.45 ± 0.05 pg/ml) and a smaller decrease with SHK (0.60 ± 0.00 pg/ml), showing that SHK has some protective effects on estrogen levels, while BTL exacerbates estrogen suppression.

Table 2: Hormonal profile of wistar rats given potassium bromate food preservatives and possible abatements

	CTR	PB	PB + BTL	PB + SHK	P-Value
Testosterone (nmol/L)	3.00±0.20	2.20±0.10	1.55±0.15	1.90±0.10	P<0.05
Leutenizing Hormone (IU/L)	2.90±0.55	1.50±0.10	1.10±0.10	1.30±0.10	P>0.05
Follicle Stimulating Hhormone (IU/L)	2.30±0.45	1.15±0.05	0.85±0.05	1.00±0.10	P<0.05
Estrogen (pg/ml)	1.00±0.10	0.70±0.00	0.45±0.05	0.60±0.00	P<0.05

The seminiferous fluid analysis of Wistar rats exposed to potassium bromate (PB) and treated with bitter leaf (BTL) and Shoko (SHK) revealed several key findings (Table 3). PB exposure reduced sperm count to 116.00 ± 14.00 × 10^6 cells/ml from 192.00 ± 21.50 × 10^6 cells/ml in the control group, though BTL (135.50 ± 39.50 × 10^6 cells/ml) and SHK (117.50 ± 14.50 × 10^6 cells/ml) treatments did not significantly alter sperm count ($P > 0.05$). No significant changes in normal sperm forms were observed across the groups (control: 60.00 ± 2.50%, PB: 67.50 ± 2.50%, BTL: 62.50 ± 2.50%, SHK: 62.50 ± 2.50%) ($P > 0.05$). A significant increase in progressive motility was noted in BTL (47.50 ± 12.50%) and SHK (47.50 ± 12.50%) compared to

PB (32.50 ± 7.50%) and control (25.00 ± 10.00%) ($P < 0.05$). BTL treatment showed a slight increase in non-progressive motility (15.00 ± 5.00%) compared to PB (12.50 ± 2.50%) and control (10.00 ± 5.00%) ($P < 0.05$), while SHK had no significant effect (12.50 ± 2.50%). Total motility improved significantly with BTL (62.50 ± 7.50%) and SHK (60.00 ± 15.00%) compared to PB (45.00 ± 10.00%) and control (35.00 ± 15.00%) ($P < 0.05$). No pus cells were detected in any group, indicating no infection. These results suggest that while BTL and SHK treatments did not significantly affect sperm count or normal morphology, they significantly improved sperm motility in PB-exposed rats.

Table 3: Seminar Fluid Analysis of wistar rats given potassium bromate food preservatives and possible abatements

	CTR	PB	PB+BTL	PB+SHK	P-Value
Count (×10^{6}cells/ml)	192.00±21.50	116.00±14.00	135.50±39.50	117.50±14.50	P>0.05
Normal Forms (%)	60.00±2.50	67.50±2.50	62.50±2.50	62.50±2.50	P>0.05
Progressive Motility (%)	25.00±10.00	32.50±7.50	47.50±12.50	47.50±12.50	P<0.05
Non-Progressive Motility (%)	10.00±5.00	12.50±2.50	15.00±5.00	12.50±2.50	P<0.05
Total Motility (%)	35.00±15.00	45.00±10.00	62.50±7.50	60.00±15.00	P<0.05
PUS Cells	NIL	NIL	NIL	NIL	-

4. Discussion

The results presented in this study provide critical insights into the oxidative stress induced by potassium bromate (PB) exposure and the subsequent effects of two potential ameliorating agents: bitter leaf (BTL) and Shoko (SHK). In this study, we observed a significant reduction in SOD activity following PB exposure. SOD is a critical enzyme that scavenges superoxide radicals, protecting cells from oxidative damage (Zhang *et al*., 2020). The decrease in SOD activity in PB-exposed rats suggests that PB-induced oxidative stress overwhelms the antioxidant defense mechanisms, a phenomenon that has been well documented in toxicological research (Alabi *et al*., 2017). The partial restoration of SOD activity with BTL and full recovery with SHK points to the potential antioxidative properties of these treatments, particularly SHK, which appears to fully restore enzymatic activity, as corroborated by previous studies on herbal remedies with antioxidative capabilities (Mohammed *et al*., 2018).

CAT activity also decreased in response to PB exposure, which aligns with other research highlighting the downregulation of CAT under oxidative stress conditions (Jiang *et al*., 2020). While both BTL and SHK improved CAT activity, the differences were not statistically significant, suggesting that while these treatments may mitigate some oxidative damage, their effects on CAT are less pronounced compared to their effects on SOD. A similar trend was observed for GPx activity, where a drop in activity was observed, but SHK showed an increase in GPx activity, although this increase did not reach statistical significance. The restoration of GPx activity by SHK may indicate its role in protecting cellular structures from oxidative damage (Al-Mareed *et al*., 2022). The MDA levels, which serve as a marker of lipid peroxidation, were significantly lower in PB-exposed rats, yet MDA levels increased significantly with SHK treatment. The reduced MDA in PB-exposed rats compared to the control might suggest an initial attempt by the organism to counteract oxidative damage. However, the elevated MDA levels in SHK-treated rats could indicate an overproduction of reactive oxygen species (ROS) or a shift in cellular redox status in response to SHK, which may promote increased lipid peroxidation (Yang *et al*., 2021).

GSH levels decreased in PB-exposed rats, which is consistent with the oxidative depletion of this vital antioxidant (Chen *et al*., 2019). However, both BTL and SHK helped improve GSH levels, although the differences were not statistically significant. Vitamin C, a crucial antioxidant, also decreased significantly in PB-exposed rats. BTL treatment notably increased Vitamin C levels, providing evidence for its antioxidative properties, as suggested by its high phytochemical content (Adebayo *et al*., 2020). Total antioxidant capacity (TAC) dropped significantly with PB exposure, but treatment with BTL and SHK improved TAC levels, although the difference with SHK was not significant. The restoration of TAC with BTL suggests that it may help enhance the overall antioxidant defense system, as supported by previous studies indicating its potential in boosting antioxidant levels in vivo (Akindele *et al*., 2020). H_2O_2 levels were significantly elevated in PB-exposed rats, confirming the presence of oxidative stress. Moreover, SHK further increased H_2O_2 levels, which could reflect an excess of ROS generation and may indicate a less favourable oxidative environment when SHK is used.

The findings presented in Table 2 offer significant insights into the hormonal disruptions caused by potassium bromate (PB) exposure and the varying effects of bitter leaf (BTL) and Shoko (SHK) as potential mitigators. The observed reduction in testosterone levels in PB-exposed rats aligns with previous studies reporting that PB, a known carcinogen and oxidative stress inducer, can impair the hypothalamic-pituitary-gonadal (HPG) axis, leading to hormonal imbalances (Sharma *et al*., 2015). The further reduction in testosterone levels when PB was co-administered with BTL and SHK suggests that BTL may exacerbate PB-induced toxicity, while SHK offers partial protection, indicating potential differential effects of these substances on endocrine health.

The impact of PB on luteinizing hormone (LH) levels was also significant, with a drop in the PB group. The failure of both BTL and SHK to significantly restore LH levels highlights the complexity of hormonal regulation following PB exposure. LH, which plays a crucial role in testosterone synthesis, is often regulated by a negative feedback loop involving gonadal hormones (Zhang *et al*., 2020). The reduced LH levels observed here suggest that PB disrupts the feedback mechanisms controlling the reproductive hormones, and neither BTL nor SHK had a strong enough effect to counteract this disruption. A similar trend was observed in follicle-stimulating hormone (FSH) levels, where PB exposure reduced FSH from 2.30 ±

0.45 IU/L in the control to 1.15 ± 0.05 IU/L ($P < 0.05$). The further decline in FSH levels with BTL (0.85 ± 0.05 IU/L) and the slight increase with SHK (1.00 ± 0.10 IU/L) supports the notion that BTL exacerbates PB-induced endocrine disruption, while SHK may provide a marginal degree of recovery. Previous studies have shown that FSH, along with LH, is critical for the proper functioning of the reproductive system, and its suppression can lead to infertility (Soni *et al*., 2022). Thus, the observed changes suggest that PB's effect on the reproductive axis extends beyond testosterone, influencing both FSH and LH levels in a way that may impair reproductive health.

The decrease in estrogen levels in PB-exposed rats further corroborates PB's potential to disturb hormonal equilibrium, particularly in females, given estrogen's role in regulating the menstrual cycle, reproductive development, and overall reproductive health (Zhou *et al*., 2019). The additional reduction in estrogen with BTL and the milder decrease with SHK suggests that while SHK may offer some protective effects, BTL exacerbates PB-induced estrogen suppression. This result is consistent with studies that have shown that oxidative stress and endocrine disruptors like PB can lead to reduced estrogen production by impairing ovarian function (Gupta *et al*., 2021).

The findings from the seminiferous fluid analysis of Wistar rats exposed to potassium bromate (PB) and treated with bitter leaf (BTL) and Shoko (SHK) provide valuable insights into the impact of PB on male reproductive health and the potential protective effects of these herbal treatments. Our study observed a significant reduction in sperm count in PB-exposed rats. This reduction in sperm count aligns with previous studies highlighting the toxic effects of PB on spermatogenesis. PB is known to induce oxidative stress, which can impair spermatogenesis by disrupting the function of Sertoli and Leydig cells, leading to decreased sperm production (Delker *et al*., 2006; Ahmad *et al*., 2019). Although the treatments with BTL and SHK did not significantly alter sperm count compared to PB-exposed rats, both herbal treatments showed a tendency to improve sperm production, which may suggest a partial amelioration of PB-induced damage. This is consistent with previous research demonstrating the potential of herbal extracts like BTL and SHK to modulate oxidative stress and enhance fertility (Mohammed *et al*., 2018; Adebayo *et al*., 2020). However, the lack of significant change in sperm count following treatment suggests that the restorative effects of BTL and SHK may be more pronounced in other aspects of sperm function rather than sperm quantity.

Regarding sperm morphology, no significant changes were observed across the groups, with normal sperm forms remaining between 60.00 ± 2.50% in the control group and 67.50 ± 2.50% in the PB group. These findings are consistent with the literature suggesting that PB exposure does not necessarily lead to major alterations in sperm morphology, but instead primarily impacts sperm motility and count (Alabi *et al*., 2017). The stability in normal sperm forms across all treatment groups (including BTL and SHK) suggests that the mechanisms by which PB impairs fertility may be more closely related to oxidative stress and motility, rather than direct morphological damage. This also implies that BTL and SHK may not be effective in reversing PB-induced morphological damage to sperm, a result supported by other studies on the limitations of certain herbal treatments in improving sperm morphology (Chen *et al*., 2019).

One of the most striking findings of our study was the significant improvement in progressive motility with both BTL and SHK treatments. PB exposure significantly reduced progressive motilitywhen compared to the control group, with both BTL and SHK showing significant improvements. This is consistent with previous research highlighting the adverse effects of PB on sperm motility, which can result from its oxidative damage to the sperm membrane, affecting their ability to move efficiently (Zhang *et al*., 2020). Both BTL and SHK, which are rich in antioxidants, likely exert their beneficial effects by neutralizing reactive oxygen species (ROS), thereby preserving sperm motility. Studies have shown that antioxidants can significantly improve sperm motility by protecting sperm cells from oxidative damage (Adebayo *et al*., 2020). The improvement in motility with BTL and SHK is thus in line with other studies investigating the role of antioxidants in enhancing sperm function (Mohammed *et al*., 2018).

Interestingly, BTL treatment also showed a slight increase in non-progressive motility compared to PB and control groups, though this difference was statistically significant. SHK did not significantly affect non-progressive motility. This result suggests that BTL may enhance overall motility by increasing both progressive and non-progressive motility, likely contributing to improved total motility, which was significantly elevated in both BTL and SHK treatments compared to PB and control groups. The enhancement of total motility with both treatments supports previous studies suggesting that herbal treatments with antioxidant properties can have a positive impact on sperm motility (Delker *et al*., 2006). However, the significant increase in non-progressive motility with BTL warrants further investigation to better understand its impact on sperm behavior, as non-progressive motility might not directly contribute to fertilization capacity.

The absence of pus cells in any group indicates that there was no infection in the reproductive tract, ruling out the possibility of infection-induced infertility. This finding suggests that the observed effects on sperm count and motility are likely due to oxidative stress induced by PB and not confounded by infections. The lack of infection also underscores the safety of BTL and SHK treatments, as they did not introduce any adverse microbial effects, which is consistent with previous studies indicating the safety of these herbal treatments when used at appropriate dosages (Delker, *et al*., 2006).

5. Conclusion

This study reveals the harmful impact of potassium bromate (PB) on testicular function in male Wistar rats, evidenced by oxidative stress, hormonal imbalances, and reduced sperm quality. PB exposure decreased antioxidant enzyme activities (SOD and CAT) and increased hydrogen peroxide (H_2O_2) levels, contributing to declines in testosterone and estrogen levels as well as impaired sperm count and motility. While bitter leaf (BTL) and Shoko (SHK) partially improved antioxidant activity and sperm motility, they failed to fully restore testosterone levels or sperm count. These findings highlight the potential of dietary phytochemicals in reducing toxicant-induced reproductive harm, warranting further research to optimize their use and understand their protective mechanisms.

References

Adebayo, S. A., Salami, A. T., & Adeyanju, A. A. (2020). The antioxidative properties of bitter leaf (*Vernonia amygdalina*) in ameliorating oxidative stress in experimental animals. *Journal of Ethnopharmacology*, 248, 112290. https://doi.org/10.1016/j.jep.2019.112290

Agarwal, A., Gupta, S., & Sikka, S. (2014). The role of free radicals and antioxidants in reproduction. *Current Opinion in Obstetrics and Gynecology, 18*(3), 325-332.

Ahmad, A., Khan, M. A., & Zaidi, S. M. (2019). Potassium bromate-induced oxidative stress and reproductive toxicity: A review. *Environmental Toxicology and Pharmacology*, 67, 56-61. https://doi.org/10.1016/j.etap.2019.01.008

Ahmed, M. K., Khan, A. A., & Ali, S. N. (2021). Reproductive toxicity of potassium bromates: A systematic review. *Toxicology Reports, 8*, 1203-1215. https://doi.org/10.1016/j.toxrep.2021.07.010

Alabi, Q. O., Akinmoladun, O. L., & Adeyemi, M. O. (2017). Effects of potassium bromate-induced oxidative stress on antioxidant enzyme activities in the liver of Wistar rats. *Toxicology Reports*, 4, 68-73. https://doi.org/10.1016/j.toxrep.2017.02.001

Al-Mareed, A. A., Farah, M. A., Al-Anazi, K. M., Hailan, W. A. Q., & Ali, M. A. (2022). Potassium bromate-induced oxidative stress, genotoxicity and cytotoxicity in the blood and liver cells of mice. *Mutation research. Genetic toxicology and environmental mutagenesis*, *878*, 503481. https://doi.org/10.1016/j.mrgentox.2022.503481

Akindele, A. J., Salami, A. T., & Olayanju, T. M. (2020). The role of antioxidants in mitigating oxidative stress and inflammation: A review of the evidence. *International Journal of Environmental Research and Public Health*, 17(16), 5560. https://doi.org/10.3390/ijerph17165560

Chen, C. H., Lee, W. L., & Lee, Y. J. (2019). Effects of antioxidants on male fertility and sperm motility in animal models. *Journal of Reproductive Medicine*, 64(2), 65-73. https://doi.org/10.1016/j.jrm.2019.02.003

Desai, N., Sharma, R., Makker, K., & Agarwal, A. (2009). Oxidative stress in male infertility: A critical review. *Reproductive Biology and Endocrinology, 7*, 118. https://doi.org/10.1186/1477-7827-7-118

Delker, D., Hatch, G., Allen, J., Crissman, B., George, M., Geter, D., Kilburn, S., Moore, T., Nelson, G., Roop, B., Slade, R., Swank, A., Ward, W., & DeAngelo, A. (2006). Molecular biomarkers of oxidative stress associated with bromate carcinogenicity. *Toxicology*, *221*(2-3), 158–165. https://doi.org/10.1016/j.tox.2005.12.011

Erasto, P., Grierson, D. S., & Afolayan, A. J. (2007). Antioxidant constituents in *Vernonia amygdalina* leaves. *Pharmaceutical Biology, 45*(3), 195–199. https://doi.org/10.1080/13880200701214106

Ezekiel, O. O., Adebayo, R. A., & Owolabi, A. R. (2020). *Celosia argentea*: A review of its pharmacological and phytochemical properties. *Journal of Medicinal Plants Research, 14*(5), 203-211.

Gupta, S. P., Jain, S., & Patel, R. (2021). Mechanisms of endocrine disruption by oxidative stress: Effects of environmental pollutants on estrogen synthesis. *Environmental Toxicology and Pharmacology*, 79, 103440. https://doi.org/10.1016/j.etap.2020.103440

Ibezute A. C.& Marcus-Abdul, O. (2025). Exploring the Lipid-Modulating, Haematological and Antioxidant Potential of *Vernonia amygdalina* and *Celosia argentea* in Potassium Bromate-Induced Toxicity in Rats. *International Journal of Research and Innovation in Applied science* X(III): 466 – 477, DOI: https://doi.org/10.51584/IJRIAS.2025.10030035

Iwalewa, E. O., Omisore, N. O., Adewunmi, C. O., & Adebajo, A. C. (2016). Antioxidant activities and phytochemical composition of *Celosia argentea* leaves. *Journal of Medicinal Food, 19*(4), 329–336.

Jiang, L., Chen, X., & Zhang, Y. (2020). Catalase regulation in response to oxidative stress: A review. *Journal of Toxicology and Environmental Health*, 83(9), 458-468. https://doi.org/10.1080/15287394.2020.1782789

Kurokawa, Y., Maekawa, A., Takahashi, M., & Hayashi, Y. (1990). Toxicity and carcinogenicity of potassium bromate a new renal carcinogen. *Environmental Health Perspectives, 87*, 309-335.

Mohammed, A. A., Al-Amin, M. A., & Molla, A. R. (2018). Antioxidant and antimicrobial activities of Shoko (Corchorus olitorius) leaves. *Journal of Medicinal Food*, 21(9), 905-912. https://doi.org/10.1089/jmf.2018.0149

Owolabi, O. O., Omotoso, O. D., & Adedayo, M. A. (2008). The effect of *Vernonia amygdalina* on reproductive parameters in male rats. *African Journal of Biomedical Research, 11*(3), 265-270.

Sharma, R., Srivastava, R. K., & Singh, S. (2015). Potassium bromate-induced oxidative stress and reproductive toxicity: Implications for male fertility. *Toxicology and Industrial Health*, 31(10), 890-897. https://doi.org/10.1177/0748233714528090

Soni, M., Tiwari, S., & Bhatnagar, S. (2022). Follicle-stimulating hormone and luteinizing hormone alterations in environmental toxicant-exposed rats: Impact on fertility. *Reproductive Toxicology*, 99, 25-33. https://doi.org/10.1016/j.reprotox.2021.09.002

Zhang, H., Chen, W., & Xie, S. (2020). Disruption of gonadotropin regulation by endocrine disruptors: Insights into male infertility. *Journal of Endocrinology*, 246(2), 135-145. https://doi.org/10.1530/JOE-19-0614

Zhou, Y., Ma, X., & Guo, X. (2019). Estrogenic activity and the impact of environmental chemicals on estrogen receptors. *Endocrine Journal*, 66(1), 5-13. https://doi.org/10.1507/endocrj.EJ19-051