Magna Scientia Advanced Research and Reviews

eISSN: 2582-9394

Cross Ref DOI: 10.30574/msarr

Journal homepage: https://magnascientiapub.com/journals/msarr/

(RESEARCH ARTICLE)

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Effects of Consumption of Potash (*Akanwu*) on Oxidative Stress, Antioxidant Status and Liver Enzymes in Male Wistar Rats

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Magna Scientia Advanced Research and Reviews, 2026, 17(01), 253-262

Publication history: Received on 21 April 2026; revised on 29 May 2026; accepted on 01 June 2026

Article DOI: https://doi.org/10.30574/msarr.2026.17.1.0096

Abstract

Background: Oxidative stress results from an imbalance between oxidant production and antioxidant defenses, leading to cellular damage. Potash (*Akanwu*), commonly consumed in some regions, has been implicated in oxidative toxicity. This study investigated the effects of chronic potash consumption on oxidative stress, antioxidant status, and liver enzyme activity in male Wistar rats.

Methods: Thirty-five male Wistar rats were divided into five groups (n=7). Group A served as control, while Groups B–E orally received potash at 250, 500, 750, and 1000 mg/kg daily for 60 days. Serum biomarkers including malondialdehyde (MDA), superoxide dismutase (SOD), catalase (CAT), total antioxidant capacity (TAC), reactive oxygen species (ROS), hydrogen peroxide (H_2O_2), and nitric oxide (NO) were measured using spectrophotometric assays. Liver enzymes (ALT, AST, ALP) were assayed with commercial kits, and histological evaluation of liver tissue was performed.

Results: Potash administration produced a biphasic antioxidant response. At lower doses (250–500 mg/kg), SOD, CAT, TAC, and NO were elevated, while at higher doses (750–1000 mg/kg), these antioxidants declined significantly, while ROS and H_2O_2 increased, indicating oxidative stress. ALT and AST remained unchanged, but ALP was significantly reduced across treatment groups. Histological analysis revealed normal hepatocytes with mild vascular congestion at 750 mg/kg.

Conclusion: Regular consumption of potash disrupts redox balance in male Wistar rats, elevates ROS and H_2O_2 while suppressing antioxidant defenses at higher doses. The biphasic response of SOD, CAT, TAC, and NO suggests initial adaptation followed by exhaustion of antioxidant systems. Although overt hepatocellular injury was absent, reduced ALP activity and altered oxidative markers highlight potash's toxicological potential.

Keywords: Potash; Oxidative stress; Antioxidant; Biomarkers; Akanwu; Reactive oxygen species; Enzymes

1. Introduction

Natural minerals obtained through geological extradition has been known to be good food additives in different parts of the globe (Imafidon *et al.,* 2016). Potash is a vital ingredient that plays a significant role in the preparation of traditional dishes, it is made up of various mined and manufactured salts that contain potassium in water soluble form (Onuigbo, 2025). The use of inorganic salts as food preservatives or additives have gained substantial growth in Nigeria. However, potash has a low amount of potassium compared against the salt of sodium chloride, making it the second most common salt consumed in Nigeria (Aribido *et al.,* 2001; Imafidon *et al.,* 2016). In Nigeria, potash occurs in abundance and it is known by various names such as akanwu (Igbo), kanwa (Hausa), kaun (Yoruba), bettieh (Obudu).

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Most importantly, because of its popularity and usage for various domestic purposes in some part of the world, it is ranked next in importance to the common table salt in Nigeria (Bankole *et al.,* 2015).

Oxidative stress (OS) is an imbalance that occurs between production of oxidants and antioxidant defence system. It refers to the imbalance between the formation of free redicals and the capability of the biologic system to neutralize them. An imbalance in the oxidant and antioxidant systems, favours the increase in oxidants, coupled with the body's inability to salvage oxidized molecules. Oxidative stress has damaging effects on various cellular structures like proteins, lipids, and nucleic acids, which ultimately lead to various pathological conditions (Pizzino *et al*., 2017). Malondialdehyde (MDA) is a lipid peroxidative biomarker known to possess mutagenic and toxic effects (Cordiano *et al*., 2023), it is used extensively as a biomarker to measure oxidative stress in numerous biological samples affected by distinct diseases such as cancer, cardiovascular, pulmonary and neurodegenerative diseases (Cordiano *et al.,* 2023; Del Rio *et al*., 2005). Enzymatic antioxidants such as superoxide dismutase (SOD), glutathione peroxidase (GPx), and catalase (CAT), are known to scavenge reactive oxygen species (ROS) from the gonads and seminal fluids (Ahmad *et al*., 2017). For instance, superoxide dismutase (SOD), in the prostate gland and seminal vesicles is a first-line defense against oxidative stress in reproductive cells and a regulator of reactive oxygen species production (Lubos *et al*., 2011).

Reactive oxygen species (ROS) are the primary effector molecules of oxidative stress, which are produced during cell metabolism, and under pathological conditions (*Sies et al*., 2022). The endogenous sources of ROS include the mitochondria, plasma membrane, endoplasmic reticulum and peroxisomes, where enzymatic reactions and autoxidation of various compounds occur (Moldovan and Moldovan, 2004). However, plasma membrane is a major source of ROS through NAD(P)H oxidases located on either side. Enzymes of the same class displaying low activity, as well as their components, are also present free in cytoplasm, regulating the actin cytoskeleton and cell motility. Mitochondria can be a major source of ROS, mainly in processes leading to apoptosis (Xie *et al*., 2020). The protein synthetic pathway (endoplasmic reticulum and Golgi apparatus), including the nucleus, as well as protein turnover, are all exquisitely sensitive to ROS-related redox conditions. Nitric oxide (NO) and hydrogen peroxide (H_2O_2) are key oxidative stress markers; with NO produced by nitric oxide synthase (NOS) in endothelial cells, neurons, and immune cells (Andrabi *et al*., 2023). Thus, NO acts as a signalling molecule regulating vascular tone, neurotransmission, and immune responses (Lundberg and Weitzberg, 2022). H_2O_2 is an oxidative stress marker generated by SOD converting superoxide radicals. At low concentrations, H_2O_2 functions as a signaling molecule in cell proliferation and defense while at high concentrations, it contributes to oxidative stress, leading to apoptosis or necrosis (Zenin *et al*., 2022).

Liver function plays a pivotal role in the regulation of endocrine pathways that influence male reproductive physiology. The most commonly assessed liver enzymes include alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP). These enzymes serve as biomarkers for liver cell injury or cholestasis and are frequently elevated in people with liver disease, metabolic syndrome, or exposure to hepatotoxic agents. Emerging evidence suggests that abnormal levels of these enzymes may correlate with impaired reproductive hormone balance, reduced sperm quality, and overall fertility potential (Selice *et al*., 2011). Research has demonstrated that men with hepatic disorders such as non-alcoholic fatty liver disease (NAFLD) or chronic hepatitis often exhibit significant reductions in serum testosterone levels, elevated estrogen concentrations due to impaired hepatic clearance, and abnormal semen parameters (Kelishadi *et al*., 2022). A cross-sectional study by Fan *et al*. (2023) reported that elevated levels of ALT and AST were significantly associated with decreased sperm motility and concentration in men from a general population cohort. Similarly, ALP, which is expressed in both hepatic and testicular tissues, has been linked with defects in Sertoli cell function and impaired spermatogenesis when abnormally expressed (Chandra *et al*., 2020).

However, due to paucity of data, this study investigated the effects of regular potash consumption on oxidative stress markers, antioxidants, and liver enzyme activity in male Wistar rats.

2. Materials and Method

2.1. Materials Used

Thirty-five (35) male Wistar rats, Potash (Trona), Electronic weighing balance (M-Metlar M311L; China), 800D Electric Centrifuge Machine, and Plain Blood tube (Fantastik, China). Standard Cages and distilled water, Filter paper (Whatman Qualitative Filter Paper No. 1, Sigma Aldrich WHA1001042), Rats kits for MDA, SOD, ROS, Catalase (CAT), Total antioxidant capacity (TAC), Nitric oxide (NO), and Hydrogen Peroxide (H_2O_2). Randox reagent Kits for AST, ALT and ALP (Sigma Aldrich, USA).

2.2. Ethical Approval

Ethical approval letter was obtained from the Animal ethics committee, Faculty of Basic Medical Sciences, College of Health Sciences and Technology, Nnamdi Azikiwe University, Nnewi Campus.

2.3. Animal Handling and Procurement

Thirty-five male Wistar rats with weight of 115-140 gram was purchased from the Animal House, Department of Physiology, College of Health Sciences, Nnamdi Azikiwe University, Nnewi Campus, Anambra State, Nigeria. The animals were housed in standard plastic cages containing water feeder in animal house with proper ventilation. They were acclimatized for a period of two weeks and fed with normal rat chow and water *ad libitum*.

2.4. Potash Procurement and Identification

The potash was gotten from a local market (Nkwo, Nnewi) in Anambra state. The authenticity of the Potash was confirmed in Pharmacognosy Department of Nnamdi Azikiwe University, Awka by Mrs. Amaka Nworah.

2.5. Preparation of Potash

The potash was crushed into fine particles using a mortar and a pestle. Some samples of the potash were sent to the department of Pharmacognosy and Traditional Medicine, Nnamdi Azikiwe University, Agulu Campus and Springboard Laboratory, Awka for trace elemental analysis. Thereafter, 100g of Potash was weighed out using a weighing balance and was dissolved in 500ml of distilled water to give a stock solution of 200ml/ml. The mixture was then stored in an air tight container and kept in refrigerator pending usage. A fresh stock solution was prepared on weekly basis throughout the experimental period.

2.6. Experimental design

The animals were randomly divided into five groups of 7 animals each. They were labelled group A, B, C, D and E. Group A served as the control and received only feed and tap water. Groups B to E served as test groups and received 250mg/kg, 500mg/kg, 750mg/kg and 1000mg/kg of Potash respectively. Potash was orally administered daily to the animals for a period of 60days.

2.7. Blood collection and termination of Experiment

Blood was collected from the experimental rats following an overnight fast of the last dose of potash as described by Parasuraman *et al.* (2010). Blood was obtained through ocular puncture using capillary tube into a plain sample bottle and allowed to clot for 5-minutes. The clotted blood was centrifuged using an 800D Electric Centrifuge Machine for 10 minutes at 3000 RPM, the serum was retrieved using a micropipette and kept into the corresponding blood bottles for Lipid peroxidation (MDA), SOD, Total antioxidant capacity (TAC), Nitric oxide (NO) and Hydrogen Peroxide (H_2O_2). Also, AST, ALT, and ALP level were evaluated using serum. The liver were excised from the animals through intrabdominal incision and were weighed, rinsed in normal saline and fixed in 10% formo-saline for histological examination.

2.7.1. Determination of lipid peroxidation (MDA), SOD, reactive oxygen species (ROS) and Hydrogen peroxide (H_2O_2), Catalase (CAT), Nitric oxide (NO), and TAC activity

Lipid peroxidation (MDA level) was determined by measuring the formation of thiobarbituric acid reactive substances (TBARS) according to the method of Varshney and Kale (1990). However, SOD was assayed by colorimetric method of Misra and Fredovich, (1972). Catalase activity was determined by colorimetric method of Sinha, (1972), TAC was determined by the Trolox equivalent antioxidant capacity (TEAC) assay by Re and Pellegrini, (1999). The reactive oxygen species (ROS) and Hydrogen peroxide (H_2O_2) level was assayed using serum following procedure of Rubio and Cerón (2021), using spectrophotometer technique. Also, levels of Nitric oxide (NO), was assayed as described by the method of Kaur *et al*. (2022) using spectrophotometric-based Griess assay technique.

2.7.2. Determination of Liver enzymes (AST, ALT, and ALP) activity.

Serum levels of AST, ALT and ALP were measured using commercially available kits according to the manufacturer's instructions.

2.7.3. Histological Evaluation

Tissues (liver) were fixed in 5% buffered formalin and were dehydrated in four (4) concentrations of Isopropyl alcohol, i.e., 70%, 80%, 90%, 100% for 1hr each and then cleared in xylene before embedded in molten paraffin wax to remove

the isopropyl alcohol. Micro sections of 5micrometer was done using Leica RM 212 Rt. Rotary Microtome. Tissues were stained using Haematoxylin and Eosin (H and E) to demonstrate general tissue structure. Tissues sectioned were examined and interpreted using Leica DM 750 binocular microscope with photomicrographic facilities and then photomicrographed by a histopathologist (Ahmed, 2016).

2.8. Statistical Analysis

The study's results were presented as mean and standard error of mean (MEAN±SEM) and data obtained were analysed using SPSS version 26. One-Way Analysis of Variance (ANOVA) was used to analysed the serum levels of AST, ALT, ALP, MDA, SOD, ROS, H_2O_2, Catalase, Nitric oxide and TAC activity followed by post Hoc Fisher's LSD comparison. Statistical significance was set at $p \leq 0.05$.

3. Results

Table 1 Effect of Potash administration on MDA, SOD and Catalase levels

Groups	MDA level (nmol/mL)	SOD level (U/mL)	Catalase level (U/mg protein)
	MEAN±SEM	MEAN±SEM	MEAN±SEM
Group A (control)	0.79±0.03	1.08±0.00	14.50±0.13
Group B (250 mg/kg of P)	0.85±0.05	1.14±0.02	17.14±0.37*
Group C (500 mg/kg of P)	0.83±0.03	1.36±0.00*	19.83±1.19*
Group D (750 mg/kg of P)	0.76±0.02	0.97±0.02*	13.00±0.00
Group E (1000 mg/kg of P)	0.69±0.03	0.84±0.04*	8.45±0.95*

Values are expressed as mean ± SEM, n=5. Data were analysed using one way ANOVA followed by LSD post-hoc test*indicates p<0.05 compared to control.

The result (table 4.1) showed that Potash at the different doses used have no significant effect on serum MDA level when compared with control (p>0.05). Potash at a low dose of 250mg/kg had no significant effect on superoxide dismutase (SOD) when compared with control (p>0.05). At a dose of 500mg/kg, Potash significantly increased SOD compared with control (p<0.05). There was a significant reduction in SOD at doses of 750 and 1000 mg/kg of Potash compared with control(p<0.05). Administration of Potash at doses of 250mg/kg and 500mg/kg significantly increased Catalase when compared with control (p<0.05). Potash administration at a dose of 750mg/kg had no significant effect on Catalase while at an increased dose of 1000mg/kg, there was a significant reduction in Catalase (p<0.05).

Table 2 Effects of Potash administration on Total antioxidant capacity and Reactive oxygen species

Groups	TAC level (mmol Trolox equivalents/L)	ROS (Relative fluorescence units (RFU)
	MEAN±SEM	MEAN±SEM
Group A (control)	10.21±0.16	1.37±0.03
Group B (250 mg/kg of P)	11.27±0.26	1.15±0.03
Group C (500 mg/kg of P)	13.91±0.62*	1.66±0.03*
Group D (750 mg/kg of P)	8.87.±0.21*	2.05±0.09*
Group E (1000 mg/kg of P)	5.60±0.35*	3.98±0.05*

Values are expressed as mean ± SEM, n=5. Data were analysed using one way ANOVA followed by LSD post-hoc test*indicates p<0.05 compared to control.

The result (table 2) showed that administration of Potash at a dose of 250mg/kg had no significant effect on total antioxidant capacity and reactive oxygen species when compared with control (p>0.05). At doses of 500, 750 and 1000mg/kg, Potash significantly reduced total antioxidant capacity, however at same doses, there was significant increase in reactive oxygen species when compared with control (p<0.05).

Table 3 Effect of Potash administration on hydrogen peroxide and nitric oxide

Groups	H_2O_2 level (µmol/L)	NO level (µmol/L)
	MEAN±SEM	MEAN±SEM
Group A (control)	0.28±0.00	2.11±0.07
Group B (250 mg/kg of P)	0.33±0.00*	2.89±0.12*
Group C (500 mg/kg of P)	0.36±0.01*	1.86±0.03*
Group D (750 mg/kg of P)	0.39±0.00*	1.44±0.07*
Group E (1000 mg/kg of P)	0.52±0.03*	1.11±0.07*

Values are expressed as mean ± SEM, n=5. Data were analysed using one way ANOVA followed by LSD post-hoc test*indicates p<0.05 compared to control.

The result (table 3) showed a significant increase in hydrogen peroxide in all Potash administered groups when compared with control ($p<0.05$). The result also showed that Potash administration at a dose of 250mg/kg significantly increased nitric oxide when compared with control ($p<0.05$). However, Potash at doses of 500, 750 and 1000mg/kg significantly reduced nitric oxide levels when compared with control ($p<0.05$)

Table 4 Effect of potash on ALT, AST, ALP, and relative liver weight

Groups	ALT level (U/L)	AST level (U/L)	ALP level (U/L)	Relative liver weight (g)
	MEAN±SEM	MEAN±SEM	MEAN±SEM	MEAN±SEM
Group A (control)	64.86±5.53	43.37±3.61	142.23±63.62	3.64±0.25
Group B (250 mg/kg of P)	56.91±9.56	42.72±4.99	42.23±7.16*	3·93±0.32
Group C (500 mg/kg of P)	57.24±5.49	39.57±3.33	48.56±17.03*	3.69±0.12
Group D (750 mg/kg of P)	61.08±8.88	44.13±6.20	18.38±5.87*	3.76±0.34
Group E (1000 mg/kg of P)	45.58±1.51	34.72±2.97	33.46±7.47*	3.81±0.27

Values are expressed as mean ± SEM, n=5. Data were analysed using one way ANOVA followed by LSD post-hoc test*indicates p<0.05 compared to control.

Table 4 result showed that Potash at the various doses used had no significant effects on alanine aminotransferase (ALT)) and Aspartate aminotransferase (AST) when compared with control ($p>0.05$). The result also showed that Potash significantly reduced alkaline phosphatase (ALP) in all treatment groups when compared with control ($P<0.05$). However, the relative liver weight result had no significant difference in the treatment groups compared to control.

3.1. Photomicrographs section of the Liver treated with different dosage of potash

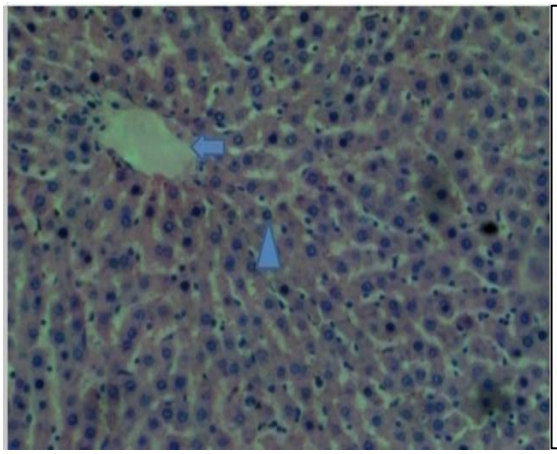

Figure 1 Group A (feed and water only)

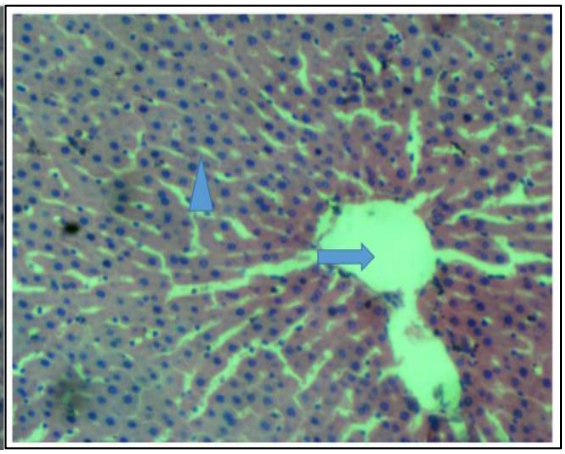

Figure 2 Group B (250 mg/kg of potash)

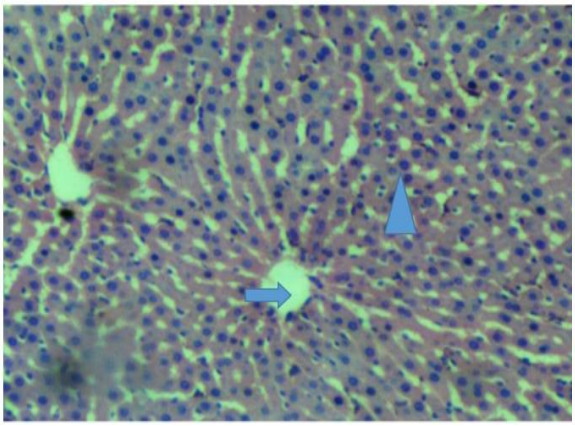

Figure 3 Group C (500 mg/kg of potash)

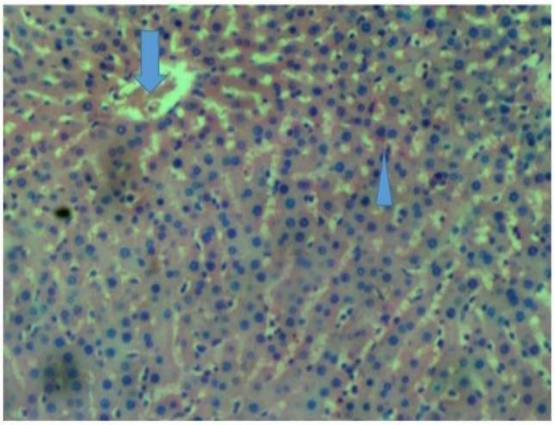

Figure 4 Group D (750 mg/kg of potash)

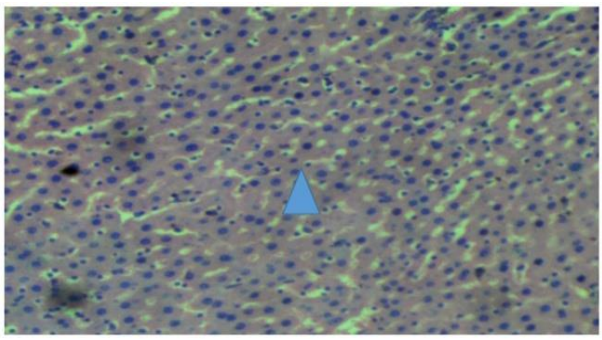

Figure 5 Group E (1000 mg/kg of potash)

Histological Interpretation: **Figure** 1: Group A (received feed and water only): Photomicrograph of liver section showing morphology consistent with normal liver histology. The hepatocytes (arrowhead) and central vein (arrow) are normal with no obvious sign of injury (H&E X 400).

Figure 2: Group B (250 mg/kg of potash): Photomicrograph of liver section showing morphology consistent with normal liver histology. The hepatocytes (arrowhead) and central vein (arrow) are normal with no obvious sign of injury (H&E X 400).

Figure 3: Photomicrograph of liver section showing morphology consistent with normal liver histology. The hepatocytes (arrowhead) and central vein (arrow) are normal with no obvious sign of injury (H&E X 400).

Figure 4: Group D (750 mg/kg of potash): Photomicrograph of liver section showing morphology consistent with normal liver histology but with mild vascular congestion. The hepatocytes are normal (arrowhead) while the central vein show mild congestion (arrow) (H&E X 400).

Figure 5: Group E (1000 mg/kg of potash): Photomicrograph of liver section showing morphology consistent with normal liver histology. The hepatocytes (arrowhead) are normal with no obvious sign of injury (H&E X 400).

4. Discussion and Conclusion

The current study evaluates the effects of chronic consumption of Potash on oxidative stress, antioxidant status, and liver enzymes in Male Wistar Rats. Oxidative stress has been identified as one of the mechanisms for idiopathic male infertility and high level of reactive oxygen species (ROS) has been reported in male population with idiopathic infertility (Ayade *et al*., 2022a; Agarwal *et al*., 2019a). This study provides compelling evidence that potash disrupts the redox balance in male rats. Potash administration resulted in a marked increase in reactive oxygen species (ROS) and hydrogen peroxide (H_2O_2). Reactive oxygen species (ROS), hydrogen peroxide (H_2O_2), were markedly elevated at higher doses, indicating increased oxidative stress. Thus, oxidative stress has been widely linked to sperm damage, impaired steroidogenesis, and Leydig/Sertoli cell dysfunction (Agarwal *et al*., 2014). Although MDA, a key marker of lipid

peroxidation, did not change significantly, the absence of a significant increase in MDA here may reflect limitations of serum MDA detection compared to tissue-specific assays.

Research has shown that TAC is very often used to assess the antioxidant status of biological samples and can evaluate the antioxidant response of organism against the free radicals produced in a given disease (Rubio *et al*., 2016). Research has demonstrated a significant correlation between low TAC levels and reduced semen quality. Agarwal *et al*. (2012) in their study found that men with reduced TAC had lower sperm concentration, motility, and morphological integrity. This study showed that total antioxidant capacity (TAC) was elevated at 500 mg/kg and significantly declined at higher doses. Enzymatic antioxidants such as superoxide dismutase (SOD), glutathione peroxidase (GPx), and catalase (CAT), are known to scavenge reactive oxygen species (ROS) from the gonads and seminal fluids (Ahmad *et al*., 2017).

This study further observed an initial increase in SOD and CAT at lower doses (250–500 mg/kg), suggesting a compensatory response to rising ROS levels. However, at higher doses of 750 and 1000 mg/kg of potash, SOD and catalase (CAT) levels declined, suggesting antioxidant system exhaustion. The suppression of SOD and CAT at higher doses confirms that potash compromises antioxidant defense. These results support the hypothesis that potash induces oxidative stress by generating excessive ROS, overwhelming the intrinsic antioxidant defenses These findings also confirm the oxidative nature of potash toxicity, as previously reported by Ezejiofor *et al*., (2013) who demonstrated that potash promotes oxidative damage by generating ROS and reduces testicular antioxidant capacity. Oludele *et al*., 2020 and Ko *et a*l., 2019 also reported similar findings on the oxidative potential of potash.

Interestingly, the observed increase in SOD and CAT at low doses and their subsequent decline at higher doses is suggestive of a biphasic response. This biphasic response pattern was not highlighted in earlier akanwu studies and provides new insights into the redox dynamics during potash exposure.

Nitric oxide (NO), a vital vasodilator and signalling molecule in male fertility, was significantly reduced at higher potash doses in contrast with findings on hydrogen peroxide. These findings reinforce the presence of oxidative imbalance and may partially explain the observed reduction in sperm parameters (Nwaefulu and Nwobodo, 2026). Reduced NO levels have been associated with erectile dysfunction, testicular ischemia, and poor sperm quality (Sikka, 2001). The biphasic effect of nitric oxide (NO), elevated at low potash dose but suppressed at higher doses of potash, is in line with findings by El-Missiry *et al*., (2015), who noted that mild oxidative stimuli initially upregulate NO as a compensatory vasodilatory response. However, excessive ROS and H_2O_2 later downregulate NO by oxidizing nitric oxide synthase (NOS), reducing its activity and availability. This antagonistic pattern suggests that high-dose potash impairs vascular signalling in the testes, possibly compromising blood-testis barrier function and Leydig cell steroidogenesis, as reported by Kostic *et al*. (2006). The steady rise in H_2O_2 also confirms oxidative accumulation, which has been implicated in peroxidation of sperm membranes and lipid-rich testicular tissues (Sikka, 2001).

Liver enzymes are routinely measured in toxicological studies as biomarkers of tissue function and integrity. Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) are predominantly cytosolic enzymes in hepatocytes; their elevation in serum usually indicates hepatocellular membrane damage or necrosis (Airadion *et al*., 2021; Giannini *et al*., 2005). Alkaline phosphatase (ALP), on the other hand, is present in multiple tissues, including the liver, bone, intestine, and testes, where it plays a role in spermatogenic cell metabolism and differentiation (Rosalki and Rao, 2000).

In this study, ALT and AST levels remained largely unaltered across potash-treated groups, suggesting that gross hepatocellular injury was absent within the exposure period. This finding contrasts with some toxicity models (e.g., Olayemi *et al*., 2017), where hepatocellular enzymes increased, possibly due to longer exposure durations or higher systemic bioavailability of toxic components. The absence of ALT/AST elevation here may reflect the liver's early-stage oxidative stress without overt necrosis. However, the significant suppression of ALP observed at higher doses is notable. Unlike transaminases, ALP reduction can occur due to enzyme inhibition, deficient synthesis, or target tissue impairment. In the testes, ALP participates in phosphate transfer processes essential for germ cell development and maturation (Setchell and Waites, 1975). Suppression of testicular ALP may, therefore, directly impair spermatogenesis, providing a plausible mechanistic link to the sperm quality decline observed in this study. Reduction in ALP has also been correlated with Leydig cell dysfunction (Rosalki and Rao, 2000). Given that ALP suppression occurred without concomitant ALT or AST elevation, it is likely that potash exerts more subtle biochemical interference rather than acute hepatocellular necrosis.

Histological evaluation of the liver showed only minimal changes. Most groups had normal hepatocyte architecture with normal cords and sinusoids. At the 750 mg/kg dose, occasional sinusoidal congestion was observed. Central veins and lobular sinusoids contained more blood than usual, but hepatocytes themselves remained healthy and no necrosis was

observed. This mild vascular congestion is a very subtle finding, without accompanying hepatocyte degeneration or inflammation.

5. Conclusion

Chronic administration of potash in male Wistar rats induced oxidative stress by elevating ROS and hydrogen peroxide while suppressing antioxidant defenses at higher doses. The biphasic response observed in SOD, CAT, TAC, and NO suggests an initial compensatory mechanism that becomes exhausted under prolonged exposure. Although ALT and AST remained stable, the significant reduction in ALP indicates subtle biochemical interference, particularly in testicular and hepatic metabolism. Histological analysis confirmed minimal liver injury, with only mild vascular congestion at higher doses. Overall, potash consumption compromises antioxidant capacity, disrupts nitric oxide signaling, and promotes oxidative imbalance, reinforcing its toxicological potential.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of ethical approval

Ethical approval was sought from ethical committee, Faculty of Basic Medical Sciences

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