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SUBCHRONIC EXPOSURE TO NACL AND PLANT-DERIVED LOW-SODIUM SALTS DIFFERENTIALLY AFFECTS OXIDATIVE STRESS MARKERS IN RATS

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ABSTRACT

Excessive dietary sodium intake is widely recognized as a contributor to oxidative stress and metabolic imbalance, whereas plant-derived low-sodium salts have emerged as promising alternatives for mitigating these adverse effects. This study assessed the impact of subchronic exposure to NaCl and plant-derived low-sodium salts on oxidative stress biomarkers in rats. Thirty-six Wistar rats (18 males and 18 females) were randomly allocated into six groups (three male and three female groups; n = 6 per group) and administered either distilled water (control), NaCl, or SHOV solutions at doses of 70 or 210 mg/kg body weight. Plasma levels of malondialdehyde (MDA), reduced glutathione (GSH), catalase (CAT) activity, and total antioxidant capacity (FRAP) were determined using standard analytical methods. The results demonstrated that NaCl administration significantly increased MDA levels while decreasing GSH, CAT, and FRAP, indicating impaired antioxidant defenses and enhanced oxidative stress. In contrast, plant-derived low-sodium salts treatment reduced MDA levels and increased antioxidant biomarkers, with more pronounced effects observed at the 210 mg/kg dose. These beneficial effects are attributed to the partial replacement of sodium with potassium, as well as to the presence of other minerals with antioxidant potential. The sex-related differences observed, although moderate, suggest a slightly greater antioxidant response in females. Overall, plant-derived low-sodium salts exert a protective effect by enhancing both enzymatic and non-enzymatic antioxidant defense systems. These findings support the nutritional and health-promoting value of plant-derived low-sodium salts as a sustainable alternative to conventional salt for the prevention of oxidative imbalances associated with excessive sodium intake.

KEYWORDS: Plant-derived low-sodium salts; NaCl; Oxidative stress; Antioxidant defenses; Wistar rats.

INTRODUCTION

Table salt, or sodium chloride (NaCl), is among the most commonly used condiments in human diets worldwide. However, excessive sodium intake is now widely acknowledged as a major public health concern, as it significantly contributes to the development of hypertension and cardiovascular, renal, and metabolic disorders (WHO, 2021). The World Health Organization recommends a maximum daily salt intake of 5 g, yet average global consumption ranges between 9 and 12 g per day - nearly double the recommended limit. Beyond its well-established hemodynamic effects, excessive sodium intake disrupts redox homeostasis and promotes the overproduction of reactive oxygen species (ROS), leading to oxidative tissue damage (Krajina et al., 2022).

Experimental studies in animal models have consistently demonstrated that high-NaCl diets induce systemic oxidative stress, characterized by increased levels of malondialdehyde (MDA) and a concomitant reduction in antioxidant defenses. These alterations include decreased concentrations or activities of reduced glutathione

(GSH), glutathione S-transferase (GST), catalase (CAT), and total antioxidant capacity (FRAP) (Krajina et al., 2022; Liu et al., 2024). At the mechanistic level, sodium-induced oxidative stress has been associated with the activation of NADPH oxidase, mitochondrial dysfunction, stimulation of the renin-angiotensin system, and inhibition of the nuclear factor erythroid 2 - related factor 2 (Nrf2)/antioxidant response element (ARE) signaling pathway, which plays a central role in the regulation of antioxidant gene expression (Wang et al., 2022).

In this context, the development of alternative strategies to reduce dietary sodium intake without compromising the sensory qualities of table salt represents a major nutritional and technological challenge. Low-sodium salts, produced through the partial substitution of NaCl with potassium chloride (KCl) or plant-derived mineral-rich matrices, have emerged as promising solutions (Neal et al., 2021). Beyond sodium reduction, these alternatives provide essential micronutrients, such as potassium (K^+) and magnesium (Mg^{2+}), as well as bioactive compounds with antioxidant properties, including polyphenols, flavonoids, and phenolic acids. These constituents are known to modulate redox homeostasis by activating the Nrf2 signaling pathway, promoting glutathione (GSH) regeneration, and enhancing endogenous enzymatic antioxidant defenses (Wang et al., 2022; Kiss et al., 2025).

Recent studies have demonstrated that potassium supplementation and the consumption of plant-based, polyphenol-rich low-sodium salts contribute to blood pressure reduction, attenuation of lipid peroxidation, and restoration of antioxidant capacity (Granato et al., 2022; Wang et al., 2022 ; Zietara et al., 2025). Notably, these protective effects are particularly evident under conditions of chronic or subchronic NaCl exposure, underscoring the complementary physiological roles of potassium and plant-derived antioxidants in preserving cellular redox balance and mitigating sodium-induced oxidative stress. With this perspective, the present study aimed to comparatively assess the effects of sodium chloride and plant-derived low-sodium salts on key plasma biomarkers of oxidative stress-including malondialdehyde (MDA), reduced glutathione (GSH), catalase (CAT) activity, and total antioxidant capacity (FRAP) - in male and female rats following 28 days of subchronic exposure. By examining sex-specific responses, this work seeks to clarify the physiological and nutritional relevance of plant - based low-sodium salts as a strategy for mitigating oxidative imbalances associated with excessive sodium intake.

MATERIAL AND METHODS

1. Biological Material and Experimental Conditions

The experiment was carried out using sixty (60) adult albino Wistar rats, comprising 30 males and 30 females, aged between 8 and 10 weeks and weighing 180 - 220 g. The animals were obtained from the animal facility of the Department of Animal Physiology, Faculty of Sciences, University of Lomé. Prior to experimentation, the rats were acclimatized for 14 days in well-ventilated metal cages under standard laboratory conditions : ambient temperature of 22 ± 2 °C, a 12 h light/12 h dark cycle, and free access to drinking water.

Throughout the study, the animals were fed a standardized diet composed of corn, soybean, fish, and wheat meal in a ratio of 4 : 2 : 2 : 1 - 1. The ingredients were thoroughly mixed, shaped into patties, and oven-dried at 45 °C for 24 h before use.

All experimental procedures were conducted in compliance with the ethical guidelines of Directive 2010/63/EU on the protection of animals used for scientific purposes. The study protocol was approved by the Bioethics Committee for Health Research of the Togolese Ministry of Health 032/2023/CBRS Starting from September 30, 2022.

2. Distribution of groups and treatments

The rats were randomly divided into 6 groups, consisting of 3 groups of female rats and 3 groups of male rats (n = 6 per group).

Table 1: Distribution of groups and treatments.

Lots formed	Administered treatment	Dosage	Oral administration	Duration
Control group (males and females)	Distilled water	10 mL/kg	Administration by oral gavage	28 days
NaCl 70 (males and females)	Sodium chloride solution	70 mg/kg	Administration by oral gavage	28 days
NaCl 210 (males and females)	Sodium chloride solution	210 mg/kg	Administration by oral gavage	28 days
Plant-derived low-sodium salts 70 (males and females)	Low-sodium salts of plant origin	70 mg/kg	Administration by oral gavage	28 days
Plant-derived low-sodium salts 210 (males and females)	Low-sodium salts of plant origin	210 mg/kg	Administration by oral gavage	28 days

The test solutions were freshly prepared each morning using distilled water. Oral administration was performed by gavage with an appropriately sized gastric tube, ensuring that the administered volume did not exceed 10 mL/kg body weight.

3. Blood Sampling and Sample Preparation

At the end of the 28 - day treatment period, the animals were lightly anesthetized with diethyl ether and euthanized by retro - orbital blood collection. Blood samples were collected into heparinized tubes and immediately centrifuged at 3000 rpm for 10 min at 4 °C. Plasma was carefully separated and stored at -20 °C until analysis of oxidative stress biomarkers.

4. Measurement of oxidative stress biomarkers

4.1. Malondialdehyde (MDA)

Lipid peroxidation was evaluated by quantifying thiobarbituric acid reactive substances (TBARS) following the method described by Ohkawa et al. (1979). Briefly, an aliquot of plasma was mixed with a reaction solution containing 0.375% thiobarbituric acid (TBA), 15% trichloroacetic acid (TCA), and 0.25 N hydrochloric acid (HCl). The mixture was heated at 95 °C for 15 min, then cooled to room temperature and centrifuged to remove precipitated proteins. The absorbance of the resulting supernatant was measured spectrophotometrically at 532 nm against a reagent blank. Malondialdehyde concentrations were calculated using the molar extinction coefficient of the MDA - TBA complex ($\varepsilon = 1.56 \times 10^5\ M^{-1}\cdot cm^{-1}$) and expressed as nmol/mL of plasma.

4.2. Reduced glutathione (GSH)

Plasma reduced glutathione (GSH) levels were quantified using Ellman's colorimetric method (1959), which is based on the reaction between thiol groups and 5,5′ - dithiobis - (2-nitrobenzoic acid) (DTNB), resulting in the formation of a yellow chromophore. Absorbance was measured spectrophotometrically at 412 nm. GSH concentrations were calculated from a standard calibration curve generated with reduced glutathione and expressed as µmol/mL of plasma.

4.3. Catalase (CAT) activity

Catalase activity was determined following the method described by Aebi (1984), based on the rate of hydrogen peroxide (H_2O_2) decomposition. The reaction mixture consisted of 50 mM phosphate buffer (pH 7.0) and 10 mM H_2O_2. The decrease in absorbance was monitored at 240 nm. Catalase activity was expressed as enzyme units per milliliter of plasma (U/mL), where one unit corresponds to the degradation of 1 µmol of H_2O_2 per minute under the assay conditions.

4.4. Total antioxidant capacity (FRAP)

Total antioxidant capacity was assessed using the ferric reducing antioxidant power (FRAP) assay as described by Benzie and Strain (1996). The FRAP reagent comprised 300 mM sodium acetate buffer (pH 3.6), 10 mM 2,4,6 - tris (2 - pyridyl) – s - triazine (TPTZ), and 20 mM ferric chloride hexahydrate ($FeCl_3\cdot 6H_2O$). A 100 µL plasma sample was incubated with the FRAP reagent at 37 °C for 10 min, after which absorbance was measured at 593 nm. Results were expressed as µmol Fe^{2+}/L based on a calibration curve prepared using ferrous sulfate heptahydrate ($FeSO_4\cdot 7H_2O$).

5. Statistical analysis

Data are presented as mean ± standard error of the mean (SEM). Statistical comparisons were performed using one-way analysis of variance (ANOVA), followed by Welch's *t*-test to compare each treated group with the corresponding sex-matched control group. Differences were considered statistically significant at $p < 0.05$. All statistical analyses were conducted using GraphPad Prism software (version 9.0).

RESULTS

1. Changes in malondialdehyde (MDA) levels

In both male and female rats, plasma malondialdehyde (MDA) concentrations increased progressively and in a dose-dependent manner following NaCl administration. In males, MDA levels rose from 2.47 ± 0.09 nmol/mL in the control group to 2.88 ± 0.10 nmol/mL and 3.46 ± 0.13 nmol/mL at doses of 70 and 210 mg/kg, respectively. Similarly, in females, MDA concentrations increased from 2.33 ± 0.10 nmol/mL in controls to 2.62 ± 0.09 nmol/mL and 3.14 ± 0.12 nmol/mL at the corresponding doses.

In contrast, treatment with plant-derived low-sodium salts resulted in a reduction of plasma MDA levels compared with controls in both sexes. In males, MDA concentrations decreased to 2.26 ± 0.09 nmol/mL at 70 mg/kg and 2.08 ± 0.08 nmol/mL at 210 mg/kg, while in females, values declined to 2.15 ± 0.08 nmol/mL and 1.99 ± 0.07 nmol/mL, respectively.

Overall, NaCl administration markedly increased lipid peroxidation, whereas plant-derived low-sodium salts supplementation induced a dose-dependent reduction in MDA levels in both sexes (Table 2). Across all treatment groups, females consistently exhibited lower MDA values than males, suggesting a slightly greater resistance to oxidative stress.

Table 2: Plasma MDA Levels (nmol/mL) after Subchronic NaCl or plant-derived low-sodium salts exposure in rats.

Treatment groups	Dose (mg/kg bw)	Male rats : MDA (nmol/mL)	*p*-value (control *vs.* treatment)	Female rats : MDA (nmol/mL)	*p*-value (control *vs.* treatment)
Control group	-	2,47 ± 0,09	-	2,33 ± 0,10	-
NaCl 70	70	2,88 ± 0,10	*	2,20 ± 0,09	*

NaCl 210	210	3,46 ± 0,13	**	3,14 ± 0,12	**
Plant-derived low-sodium salts 70	70	2,26 ± 0,09	*	2,15 ± 0,08	*
Plant-derived low-sodium salts 210	210	2,08 ± 0,08	**	1,99 ± 0,07	*

Values are mean ± SEM (n = 6). *p < 0.05 ; **p < 0.01 vs. Control

2. Changes in reduced glutathione (GSH) content

- Average GSH concentrations decreased following NaCl administration and increased under plant-derived low-sodium salts treatment, showing a similar trend in both sexes. In male rats, GSH levels gradually decreased from 6.05 ± 0.24 µmol/mL in the control group to 5.48 ± 0.21 µmol/mL at 70 mg/kg NaCl, and further to 4.67 ± 0.19 µmol/mL at 210 mg/kg. In females, the decrease followed the same pattern, with values of 6.39 ± 0.25 µmol/mL in the control group, 5.88 ± 0.22 µmol/mL at 70 mg/kg NaCl, and 5.01 ± 0.20 µmol/mL at 210 mg/kg NaCl. Conversely, plant-derived low-sodium salts administration induced a dose - dependent increase in GSH content. In males, values rose to 6.51 ± 0.23 µmol/mL at 70 mg/kg and 6.87 ± 0.24 µmol/mL at 210 mg/kg, whereas in females, they reached 6.84 ± 0.25 µmol/mL and 7.29 ± 0.26 µmol/mL, respectively.

Overall, the trend in GSH levels was opposite to that of MDA, with higher concentrations in females, suggesting a more efficient antioxidant response (Table 3).

Table 3: Changes in GSH content (µmol/mL) in male and female rats.

Treatment groups	Dose (mg/kg bw)	Male rats : GSH (nmol/mL)	*p*-value (control *vs*. treatment)	Female rats : GSH (nmol/mL)	*p*-value (control *vs*. treatment)
Control group	-	6,05 ± 0,24		6,39 ± 0,25	
NaCl 70	70	5,48 ± 0,21	*	5,88 ± 0,22	*
NaCl 210	210	4,67 ± 0,19	**	5,01 ± 0,20	**
Plant-derived low-sodium salts 70	70	6,51 ± 0,23	*	6,84 ± 0,25	*
Plant-derived low-sodium salts 210	210	6,87 ± 0,24	**	7,29 ± 0,26	**

Values are mean ± SEM (n = 6). *p < 0.05 ; **p < 0.01 vs. Control

3. Changes in total antioxidant capacity, FRAP (Ferric Reducing Antioxidant Power)

Total antioxidant capacity, measured as FRAP, exhibited trends opposite to those observed for lipid peroxidation (MDA). In male rats, FRAP values gradually decreased from 798.41 ± 25.02 µmol Fe^{2+}/L in the control group to 723.57 ± 24.38 µmol Fe^{2+}/L at 70 mg/kg NaCl, and further to 685.24 ± 23.82 µmol Fe^{2+}/L at 210 mg/kg NaCl. In contrast, plant-derived low-sodium salts administration induced a dose-dependent increase in FRAP, reaching 840.35 ± 26.10 µmol Fe^{2+}/L at 70 mg/kg and 839.08 ± 27.29 µmol Fe^{2+}/L at 210 mg/kg.

A similar pattern was observed in females, with FRAP values gradually decreasing from 851.68 ± 27.33 µmol Fe^{2+}/L in the control group to 806.46 ± 25.12 µmol Fe^{2+}/L and 794.23 ± 24.42 µmol Fe^{2+}/L at 70 and 210 mg/kg NaCl, respectively. plant-derived low-sodium salts treatment increased FRAP to 875.06 ± 27.09 µmol Fe^{2+}/L at 70 mg/kg and 935.17 ± 28.67 µmol Fe^{2+}/L at 210 mg/kg.

Overall, these results indicate a reduction in total antioxidant potential under NaCl treatment and a progressive improvement with plant-derived low-sodium salts administration. Additionally, females exhibited higher antioxidant capacity than males (Table 4).

Table 4: Changes in total antioxidant capacity (FRAP, µmol Fe^{2+}/L) in male and female rats.

Treatment groups	Dose (mg/kg bw)	Fe^{2+} content in male rats	*p*-value (control *vs*. treatment)	Fe^{2+} content in Female rats	*p*-value (control *vs*. treatment)
Control group	-	798,41 ± 25,02		851,68 ± 27,33	
NaCl 70	70	723,57 ± 24,38	*	806,46 ± 25,12	-
NaCl 210	210	685,24 ± 23,82	**	794,23 ± 24,42	*
Plant-derived low-sodium salts 70	70	840,35 ± 26,10	*	875,06 ± 27,09	*
Plant-derived low-sodium salts 210	210	839,08 ± 27,29	*	935,17 ± 28,67	**

Values are mean ± SEM (n = 6). *p < 0.05 ; **p < 0.01 vs. Control

2. Changes in catalase enzyme activity (U/mL plasma)
The mean catalase activity followed a trend similar to that observed for GSH content. In male rats, catalase activity decreased from 49.53 ± 1.81 U/mL in the control group to 45.24 ± 1.72 U/mL at 70 mg/kg NaCl and further to 40.81 ± 1.60 U/mL at 210 mg/kg NaCl. Conversely, in the plant-derived low-sodium salts-treated groups, activity gradually increased to 52.87 ± 1.91 U/mL at 70 mg/kg and 56.78 ± 2.07 U/mL at 210 mg/kg.

A similar pattern was observed in females, with mean values of 51.72 ± 1.90 U/mL in the control group, 47.48 ± 1.80 U/mL at 70 mg/kg NaCl, and 43.27 ± 1.71 U/mL at 210 mg/kg NaCl, followed by increases to 52.17 ± 2.10 U/mL and 59.53 ± 2.10 U/mL at 70 and 210 mg/kg plant-derived low-sodium salts, respectively.

Overall, catalase activity decreased under NaCl treatment and increased with plant-derived low-sodium salts administration, with the effect slightly more pronounced in females (Tables 5 and 6).

Table 5: Changes in catalase activity (U/mL plasma) in male and female rats.

Treatment groups	Dose (mg/kg bw)	Catalase Activity in Male Rats (U/mL plama)	*p*-value (control *vs.* treatment)	Catalase Activity in Female Rats (U/mL Plasma)	*p*-value (control *vs.* treatment)
Control group	-	49,53 ± 1,81		51,72 ± 1,90	
NaCl 70	70	45,24 ± 1,72	*	47,48 ± 1,80	*
NaCl 210	210	40,81 ± 1,60	**	43,27 ± 1,71	**
Plant-derived low-sodium salts 70	70	52,87 ± 1,91	*	52,17 ± 2,01	-
Plant-derived low-sodium salts 210	210	56,78 ± 2,07	**	59,53 ± 2,10	**

U = enzyme units. Values are presented as mean ± SEM (n = 6 rats). Significant differences from control are indicated as *p < 0.05 and **p < 0.01.

Table 6: Summary of oxidative stress biomarkers.

Analyzed Biomarker	NaCl Effect	SHOV Effect	Sex Differences
MDA content	Dose-related enhancement	Dose-related reduction	Females exhibited slightly lower levels than males
GSH content	Dose-related reduction	Dose-related enhancement	Females exhibited higher levels than males
Total antioxidant capacity (FRAP)	Dose-related reduction	Dose-related enhancement	Females exhibited higher levels than males
Catalase activity	Dose-related reduction	Dose-related enhancement	Females exhibited a stronger response

DISCUSSION

1. Effect of NaCl
Chronic sodium overload promotes the generation of reactive oxygen species (ROS) through multiple mechanisms, including activation of vascular and renal NADPH oxidase, mitochondrial dysfunction, stimulation of the angiotensin II/AT1R signaling pathway, and endothelial inflammation. The resulting oxidative stress leads to enhanced lipid peroxidation, as reflected by elevated MDA levels, and to a depletion of both enzymatic and non-enzymatic antioxidant defenses, such as GSH, catalase, and total antioxidant capacity (measured by FRAP).

These findings are consistent with recent studies reporting that a high-salt diet increases ROS production and lipid peroxidation while reducing antioxidant enzyme activity in various rat tissues. Key mediators, including NOX and the NF-κB/TGF-β1 signaling pathway, have been implicated in the propagation of inflammation and fibrosis under high-salt conditions (Krajina et al., 2022).

In a nephropathy model exacerbated by a high-salt diet, systemic oxidative stress is elevated, as indicated by increased levels of 8-OHdG and oxidized albumin. Treatment with the antioxidant Tempol, a radical scavenger, partially mitigates these effects, highlighting the role of salt overload as an upstream trigger of the oxidative and inflammatory cascade (Liu et al., 2024).

At the molecular level, the observed reduction in GSH and GSH-dependent enzymes (e.g., GST) aligns with functional inhibition of the Nrf2-ARE pathway, which regulates the expression of key antioxidant genes, including GCL/GSS (for GSH synthesis), GST, NQO1, HO-1, and CAT. When oxidative stress exceeds the capacity of cellular defenses, Nrf2 activation becomes insufficient, leading to depletion of GSH pools, reduced GST and CAT activity, and lower total antioxidant capacity as measured by FRAP (Wang et al., 2022).

2. Effect of plant-derived low-sodium salts

Low-sodium salts of plant origin provide potassium (K^+) as a partial substitute for sodium while also supplying essential minerals that contribute to antioxidant defense mechanisms. These minerals act either as structural components of antioxidant enzymes (e.g., Zn, Cu, Mn, Fe) or exert protective effects by functioning as redox-active elements or membrane stabilizers (e.g., Zn, Mg, Ca).

Two complementary mechanisms likely explain the normalization - or even improvement - of GSH, catalase activity, and total antioxidant capacity (FRAP), concomitant with the reduction in lipid peroxidation (MDA). First, the reduction in the Na^+/K^+ ratio plays a central role. Increased dietary K^+ intake is known to attenuate salt-sensitive hypertension and reduce ROS generation through direct vascular effects, enhanced natriuresis, and inhibition of NADPH oxidase (NOX) activation. Indeed, recent experimental and clinical studies demonstrate that K^+ enrichment mitigates the deleterious effects of high salt intake on blood pressure and oxidative stress (Zietara et al., 2025).

Second, the intake of minerals with redox-modulating properties promotes activation of the Nrf2 signaling pathway, leading to restoration of intracellular GSH levels and increased activity of antioxidant enzymes such as GST and catalase. This coordinated antioxidant response contributes to reduced MDA levels and enhanced FRAP, reflecting improved global antioxidant capacity (Wang et al., 2022).

At the population level, the use of salt substitutes - where NaCl is partially replaced by KCl -has been shown to lower blood pressure and reduce cardiovascular events. These epidemiological findings support the pathophysiological relevance of this nutritional strategy, even though the present study focuses primarily on biochemical markers of oxidative stress (Neal et al., 2021).

3. Dose, tissue, and sex-dependent effects

With respect to dose and tissue dependence, the magnitude of lipid peroxidation (MDA increase) and the reduction in antioxidant defenses (GSH, catalase, and FRAP) generally intensifies with increasing sodium load and duration of exposure. However, tissue-specific enzymatic adaptations may transiently compensate for oxidative stress, partially masking clear decreases in certain organs. This adaptive response can lead to moderate inter-tissue or inter-group variability without calling into question the overall oxidative trend induced by sodium overload (Krajina et al., 2022)..

Regarding sex differences, experimental studies in rats have demonstrated differential sensitivity to the Na^+/K^+ balance between males and females. In salt-sensitive Dahl rat models, potassium supplementation attenuates hypertension more markedly in males than in females, an effect associated with distinct renal molecular signatures. Such sex-specific responses may be reflected in the present study by modest differences in oxidative and antioxidant biomarkers, with slightly more pronounced antioxidant responses observed in one sex depending on hormonal status, aldosterone signaling, and renal handling of electrolytes (Zietara et al., 2025).

These findings emphasize the importance of considering dose, tissue specificity, and biological sex when interpreting oxidative stress biomarkers in response to dietary sodium and potassium modulation.

CONCLUSION

This study evaluated the effects of subchronic (28-day) ingestion of sodium chloride (NaCl) and low-sodium salts of plant origin on multiple oxidative stress biomarkers in male and female rats. The results demonstrate markedly opposite effects between the two types of salts. Repeated NaCl administration, particularly at the highest dose tested (210 mg/kg body weight), led to a significant increase in malondialdehyde (MDA), indicative of enhanced lipid peroxidation, along with a pronounced reduction in reduced glutathione (GSH) levels, catalase activity, and total antioxidant capacity (FRAP). Collectively, these alterations reflect a disruption of redox homeostasis characterized by excessive reactive oxygen species production and depletion of endogenous antioxidant defenses.

In contrast, treatment with low-sodium salts of plant origin induced the opposite response, characterized by a reduction in MDA levels and a significant, dose-dependent improvement in antioxidant parameters (GSH, catalase, and FRAP). These findings suggest that plant-derived low-sodium salts exert a protective effect against oxidative stress, likely attributable to their reduced sodium content, enrichment in potassium, and the presence of essential minerals that support cellular antioxidant defense mechanisms. The observed sex-related differences, although moderate, suggest a slightly more favorable antioxidant response in females, potentially attributable to hormonal factors such as estrogen or a higher basal antioxidant capacity. Overall, the findings of this study indicate that excessive NaCl intake acts as a driver of oxidative stress, whereas partial substitution of sodium with low-sodium salts of plant origin represents a promising strategy to prevent or attenuate oxidative imbalances associated with high-salt diets..

These results highlight important perspectives for both nutritional interventions and food technology, particularly in the development of alternative table salts designed to reduce sodium intake while preserving antioxidant balance, thereby contributing to the reduction of cardiovascular and metabolic risk.

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