*Sahel Journal of Life Sciences FUDMA (SAJOLS)*
**March 2026 Vol. 4(1): 098-104**
**ISSN: 3027-0456 (Print)**
**ISSN: 1595-5915 (Online)**
**DOI: *https://doi.org/10.33003/sajols-2026-0401-11***

***Research Article***

# Consumption of High Salt-Diet Causes Cortical and Hippocampal Lipid Peroxidation via Endogenous Antioxidant Depletion in Sleep Deprived Adult Female Wistar Rats

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**ABSTRACT**
Excessive dietary salt intake is a major global health concern, particularly in low- and middle-income countries, where it significantly contributes to hypertension and other non-communicable diseases. Concurrently, chronic sleep deprivation is increasingly recognized as an independent risk factor for neurological and cardiovascular dysfunction. While each stressor individually impairs health, its combined impact on hippocampal oxidative balance and cognition remains poorly understood. In this study, twenty Adults female Wistar rats weighing 120-150 g were randomly assigned into four groups (n = 5 per group): control, high-salt diet (5% NaCl supplementation), sleep-deprived, and combined high-salt plus sleep-deprived. At the end of the experimental period, hippocampal tissue and blood samples were analyzed for oxidative stress biomarkers, including malondialdehyde (MDA), catalase (CAT), superoxide dismutase (SOD), and reduced glutathione (GSH). Cognitive performance was assessed using hippocampal-sensitive antioxidant assays. The combined high-salt and sleep-deprived group exhibited pronounced elevations in hippocampal and brain MDA levels (514.70 ± 10.03 vs. 436.32 ± 8.39) alongside significant reductions in CAT (3.60 ± 0.48 vs. 2.68 ± 0.19) and SOD (5.08 ± 0.45) activity compared to controls. These alterations were associated with impaired spatial learning and memory, consistent with hippocampal oxidative stress. Our findings demonstrate that concurrent high-salt intake and sleep deprivation synergistically disrupt hippocampal redox homeostasis and impair cognitive outcomes in female Wistar rats. This highlights the compounded risk posed by poor dietary patterns and inadequate sleep, with important implications for populations experiencing overlapping nutritional and lifestyle stressors.

**Keywords:** Brain; Cognition; Dietary salt; Hippocampus; Oxidative stress; Sleep deprivation

**Citation:** Malgwi, I.S., Yusuf, A.H., Emmanuel, N.S., Yakubu, G.S., & Muhammad, A. (2026). Consumption of High Salt-Diet Causes Cortical and Hippocampal Lipid Peroxidation via Endogenous Antioxidant Depletion in Sleep Deprived Adult Female Wistar Rats. *Sahel Journal of Life Sciences FUDMA*, 4(1): 098-104. DOI: *https://doi.org/10.33003/sajols-2026-0401-11*

## INTRODUCTION

Sleep is a fundamental biological process essential for optimal brain function and systemic physiology (Watson *et al.,* 2015). Adequate sleep supports cognitive performance, emotional regulation, and cardiovascular, cerebrovascular, and metabolic health (Kovacevic *et al.,* 2021). Conversely, disturbances in sleep, whether through reduced

quantity, poor quality, or fragmentation, carry substantial short- and long-term consequences (Holmer *et al*., 2021). Risk factors for sleep disruption are multifactorial, encompassing biological, psychological, genetic, and social determinants (Lipinska *et al.,* 2015). Both acute sleep deprivation and chronic sleep restriction have been shown to impair physical health, mental well-being, and public safety, while chronic insufficient sleep is strongly associated with increased morbidity and mortality across multiple disease domains, including cardiovascular disease, diabetes, obesity, and cancer (Thakkar *et al.,* 2015).

The global rise in sleep disorders is closely tied to modern lifestyle changes, which also include dietary shifts such as excessive salt consumption. High dietary salt intake and chronic sleep deprivation, though distinct, frequently co-occur and exert synergistic deleterious effects on the nervous system. Their convergence represents a pressing public health challenge, warranting rigorous scientific investigation (Abbott & Videnovic 2016; Clark *et al.,* 2016).

Excessive salt consumption is a worldwide epidemic. Despite the World Health Organization's recommendation of less than 5 g of salt per day (equivalent to <2 g sodium), global averages range from 8.5 to 15 g daily, with over 99% of adults exceeding recommended limits (Oršolić & Jembrek, 2024). In Sub-Saharan Africa, where hypertension prevalence is among the highest globally, salt intake is a major driver of disease burden. Nigeria exemplifies this crisis, with reported intakes ranging from 5.8 g to 25 g per day, contributing to significant cardiovascular mortality (WHO, 2023; Miranda *et al.,* 2024).

Parallel to this dietary epidemic is the escalating prevalence of insufficient sleep. Approximately one-third of adults worldwide fail to achieve the recommended 7–9 hours of nightly sleep, while over 60% report poor sleep quality. In Africa, rapid urbanization and socioeconomic transitions have amplified sleep problems, with younger populations particularly affected. In Nigeria, sleep disorders are highly prevalent, with rates exceeding 60% in clinical populations and notable burdens among students and professionals (Boateng *et al.,* 2017; Odili *et al.,* 2020).

The intersection of high salt intake and sleep deprivation is mechanistically linked through oxidative stress. Oxidative stress arises when reactive oxygen species (ROS) overwhelm endogenous antioxidant defenses, leading to cellular injury (Oršolić & Jembrek, 2024). Both excessive sodium consumption and sleep loss independently promote ROS generation, endothelial dysfunction, and chronic low-grade inflammation. These processes not only underlie cardiovascular pathology but also extend to the central nervous system, where oxidative stress contributes to structural and functional alterations in the hippocampus, a brain region critical for learning and memory. The hippocampus is particularly vulnerable to oxidative insults, and cumulative exposure to these stressors may impair synaptic plasticity, neurogenesis, and cognitive performance (Whitesell *et al.,* 2018; Pandi-Perumal *et al.,* 2021).

Thus, the combined impact of high salt diet and sleep deprivation represents a biologically plausible and clinically relevant model of hippocampal oxidative stress and cognitive dysfunction (Grillo *et al.,* 2019). Investigating these interactions in female Wistar rats provides a controlled experimental framework to elucidate sex-specific vulnerabilities and mechanistic pathways, with implications for both neuroscience research and public health interventions.

## MATERIALS AND METHODS

### Experimental Animals

The experiments were carried out in the laboratory of Department of Human Physiology, Ahmadu Bello University, Zaria, Nigeria, at standard laboratory conditions of temperature and humidity. A total of Twenty (N=20) apparently healthy adult female Wistar albino rats (Rattus norvegicus) weighing between 120–150 g was used for the study. The animals were obtained from the Animal Unit of the Department of Human Physiology, Faculty of Basic Medical Sciences, College of Medical Sciences, Ahmadu Bello University, Zaria. They were housed under standard laboratory conditions at room temperature. They were fed with standard commercial rat chow and water *ad libitum*. Pelletized growers' feed was purchased from Vital Feed, a subsidiary of UAC Nigeria Limited in Zaria, and was used throughout the research work. Pelletized growers feed 25kg contains the following ingredients: cereals/grains, vegetable protein, premix (vitamins/ minerals), essential amino acids, salt, antioxidant, anti-toxins, prebiotic and enzymes. Constituent

percentage includes: Crude protein (13%), Fat (8%), crude fibre (15%), calcium (0.9%), phosphorus (0.35%) and metabolized energy (2600Kcal/kg).

**Diet preparation**

A normal protein rodent chow (Chikun Feed, Nigeria) was used as the basal diet. The control diet consisted of this standard chow in its pellet form. The high-salt diet was formulated to contain 5% sodium chloride (NaCl) by weight. This was prepared by grinding the standard pellet chow into a powder and thoroughly mixing 125 g of NaCl with 2375 g of the powdered chow to ensure a homogenous mixture (Asiwe *et al.,* 2021). The resulting mixture was then re-pelleted for feeding.

**Experimental design and ethical approval**

The animals were weighed and randomly divided into four groups, each consisting of five animals (n=5). Group I was designated as the normal control and received standard laboratory chow and water *ad libitum,* and were not subjected to sleep deprivation, while Group II served as the High-Salt Group (HS), and Received 5% high-salt diet and water *ad libitum* and were not subjected to sleep deprivation. Group III served as a sleep-deprived control, and group IV received HSD and was sleep-deprived. The experimental procedures were conducted per the approved guidelines of the Ahmadu Bello University Ethical Committee on Animal Use and Care. The administration lasted for 14 days.

**Induction of paradoxical sleep deprivation**

Sleep deprivation was induced using the Multiple Platform Method as previously described in the literature (Machado *et al.,* 2004; Khan *et al.,* 2023). The apparatus consisted of a rectangular tank (110×60×40 cm) filled with water to a depth that was maintained at 1 cm below the surface of fifteen small circular platforms (6.5 cm diameter) fixed to the bottom of the tank. For 20 hours per day (from 12:00 to 08:00), rats in the SD and HS+SD groups were placed inside this tank. Food and water were provided *ad libitum* via containers suspended from the wire-mesh lid of the tank. Following the 18-hour deprivation period, the rats were returned to their home cages for a 6-hour recovery period (from 08:00 to 12:00) with free access to their respective diets and water. This protocol was followed on alternate days for 2 weeks.

**Blood sample collection**

After the study, the animals were anaesthetized with pentobarbital (60 mg/kg body weight, intraperitoneally) (Laferriere *et al*., 2015). The brain and hippocampal tissues were excised, rinsed in cold phosphate buffer (0.1 M, pH 7.4), weighed and homogenized. Tissue samples were centrifuged, and aliquots of the supernatant were used for biochemical analyses

**Biochemical assays**

The extent of lipid peroxidation was determined by measuring malondialdehyde (MDA) concentration using the thiobarbituric acid reactive substances (TBARS) assay described by Ohkawa *et al*. (1979). In this procedure, MDA reacts with thiobarbituric acid under acidic and high-temperature conditions to form a pink chromogen, the absorbance of which was recorded at 532 nm. Reduced glutathione (GSH) levels were estimated following the method of Ellman (1959), which is based on the reaction of sulfhydryl groups with 5,5′-dithiobis-(2-nitrobenzoic acid) (DTNB). This reaction yields 2-nitro-5-thiobenzoic acid (TNB), a yellow-colored product measurable at 412 nm, and the GSH concentration was calculated against a standard curve. Superoxide dismutase (SOD) activity was assayed according to Misra and Fridovich (1980), wherein the enzyme inhibits the autooxidation of epinephrine to adrenochrome under alkaline conditions. The rate of adrenochrome formation was monitored spectrophotometrically at 480 nm, and one unit of SOD activity was defined as the amount of enzyme required to inhibit the oxidation rate by 50%. Catalase (CAT) activity was determined using the method of Claiborne (1985), which measures the decomposition of hydrogen peroxide ($H_2O_2$) by catalase. The decrease in absorbance of $H_2O_2$ was monitored at 240 nm, and enzyme activity was expressed as micromoles of $H_2O_2$ decomposed per minute per milligram of protein.

**Data Analysis**

Data obtained from the study were analyzed and expressed as mean ± SEM. Statistical analysis was carried out using version 23 of the IBM Statistical Package for Social Sciences (SPSS). A one-way analysis of variance (ANOVA) was carried out, followed by *Tukey*'s *post hoc* test, to determine the differences among the groups. Values with a $p < 0.05$ were considered statistically significant.

## RESULTS

### Some Brain Oxidative Stress Parameters in Adult Female Wistar Rats Exposed to a High Salt Diet and Paradoxical Sleep Deprivation

In Table 1, MDA levels were significantly elevated in the HSD group (374.32±11.32) compared to NC (297.86±11.57), while the SD group (436.32±8.39) showed improvement compared to NC (297.86±86), and the combined SD+HSD group also exhibited higher MDA (514.70±10.03) compared to NC; in direct comparisons, HSD had lower MDA (374.32±11.32) than SD (436.32±8.39) and the combined group (514.70±), whereas SD and SD+HSD displayed similar elevations. SOD activity was significantly reduced in the SD group (6.58± 0.44) compared to NC (10.96±6.57), whereas the HSD group (8.80± 0.64) maintained higher levels relative to NC (10.96± 0.57), with the SD+HSD group (5.08±0.45) also showing reduced SOD compared to NC; pairwise comparisons revealed that HSD had higher SOD activity than both SD and the combined group, while SD and SD+HSD demonstrated comparable reductions. GSH was significantly increased in the HSD group (8.34±0.49) compared to NC (5.52±0.66), while the SD group did differ from NC, and the combined SD+HSD group also showed elevated GSH (14.80±0.53) compared to NC (5.52±0.66); comparisons indicated that SD and the combined group had higher GSH than HSD, with slight difference between SD and SD+HSD. CAT activity was significantly reduced in the SD group compared to NC, whereas the HSD group preserved CAT activity relative to NC, and the combined SD+HSD group also showed reduced CAT compared to NC; in pairwise comparisons, HSD maintained higher CAT activity than both SD and the combined group, while SD and SD+HSD exhibited similar reductions.

### Some Hippocampal Oxidative Stress Parameters in Adult Female Wistar Rats Exposed to High Salt Diet and Paradoxical Sleep Deprivation

In Table 2, MDA levels were significantly elevated in the HSD group (286.40±12.57) compared to NC (215.26±15.44), while the SD group (357.68±12.40) showed improvement relative to NC, and the combined SD+HSD group (427.76±9.88) also exhibited higher MDA compared to NC (215.26±15.44); in direct comparisons, HSD had lower MDA than SD and the combined group, whereas SD and SD+HSD displayed similar elevations. SOD activity was significantly reduced in the HSD group (10.36±0.47) compared to NC (12.32±0.60), whereas the SD group (8.06±0.23) maintained lower levels relative to NC, with the SD+HSD group (6.00±0.26) also showing reduced SOD compared to NC; pairwise comparisons revealed that HSD had higher SOD activity than both SD and the combined group, while SD and SD+HSD demonstrated comparable reductions. GSH was significantly decreased in the HSD group (12.28±0.97) compared to NC (15.80±0.76), while the SD group (8.38±0.62) and the combined SD+HSD group (6.62±0.50) also showed reduced GSH compared to NC; comparisons indicated that SD and the combined group had lower GSH than HSD, with slight difference between SD and SD+HSD. CAT activity was significantly reduced in the HSD group (8.36±0.55) compared to NC (10.58±0.46), whereas the SD group (6.22±0.15) preserved CAT activity relative to NC, and the combined SD+HSD group (3.60±0.48) also showed reduced CAT compared to NC; in pairwise comparisons, HSD maintained higher CAT activity than both SD and the combined group, while SD and SD+HSD exhibited similar reductions.

**Table 1: Results of High salt diet on Brain Oxidative Stress Induced Sleep Deprivation in adult Female Wistar rats**

| Groups | MDA (nmol/g protein) | SOD (U/mg protein) | GSH (U/mg protein) | CAT (U/mg protein) |
|---|---|---|---|---|
| CN | 297.86 ± 11.57 | 10.96 ± 0.51 | 5.52 ± 0.66 | 6.34 ± 0.30 |
| HSD | 374.32 ± 11.23$^{a}$ | 8.80 ± 0.64$^{a}$ | 8.34 ± 0.49$^{a}$ | 5.40± 0.13$^{a}$ |
| SD | 436.32 ± 8.39$^{b}$ | 6.58 ± 0.44$^{ab}$ | 11.42 ± 0.61$^{ab}$ | 3.86 ± 0.24$^{ab}$ |
| SD + HSD | 514.70± 10.03$^{abc}$ | 5.08 ± 0.45$^{bc}$ | 14.80 ± 0.53$^{ab}$ | 2.68 ± 0.19$^{ab}$ |

NC = normal control, HSD = high salt diet, SD =Sleep deprivation, superscripts a, b and c represent statistically significant difference (P<0.05) compared to NC, HSD, SD + HSD groups, respectively.

**Table 2: Results of High salt diet on Hippocampal Oxidative Stress Induced Sleep Deprivation in adult Female Wistar rats**

| Groups | MDA (nmoL/g protein) | SOD (U/mg protein) | GSH (µg/mg protein) | CAT (U/mg protein) |
|---|---|---|---|---|
| CN | 215.26±15.44 | 12.32±0.60 | 15.80±0.76 | 10.58±0.46 |
| HSD | 286.40±12.57a | 10.36±0.47a | 12.28±0.97a | 8.36±0.55a |
| SD | 357.68±12.40ab | 8.06±0.23a | 8.38±0.62ab | 6.22±0.15a |
| SD+HSD | 427.76±9.88abc | 6.00±0.26abc | 6.62±0.50ac | 3.60±0.48abc |

NC = normal control, HSD = high salt diet, SD =Sleep deprivation, superscripts a, b and c represent statistically significant difference (P<0.05) compared to NC, HSD, SD + HSD groups, respectively.

## DISCUSSION

Sleep deprivation and sleep-related disorders have become increasingly entrenched in modern social life, driven by insecurity, economic burden, psychosocial stress, emotional disturbances, competition for survival, pain, and drug use (Boguszewski & Zagrodzka, 2002; Engeland *et al.,* 2006; Pitychoutis *et al.,* 2009). These factors collectively disrupt sleep architecture, leading to chronic sleep loss and its associated physiological consequences. In parallel, excessive dietary salt consumption remains a pervasive lifestyle challenge, contributing to systemic oxidative stress and cardiovascular risk (Maria *et al.,* 2008). Against this backdrop, our study examined oxidative stress and cognitive function under conditions of high salt diet (HSD) and sleep deprivation (SD), focusing on both whole brain tissue and hippocampal extracts.

In this study, oxidative stress biomarkers; MDA, SOD, GSH, and CAT were assayed separately from whole brain and hippocampus. The values obtained from both regions followed similar or closely aligned trends. This observation is biologically plausible because oxidative stress is a systemic process, and ROS generated during SD and HSD are diffusible, affecting multiple brain regions simultaneously (Ivana *et al.,* 2022). The hippocampus, however, is uniquely vulnerable due to its high metabolic demand, dense synaptic activity, and abundance of polyunsaturated fatty acids, which are highly susceptible to peroxidation. Thus, while the magnitude of oxidative changes may vary, the overall direction of imbalance remains consistent across brain tissue (Pepe *et al.,* 2012; Grillo *et al.,* 2019).

MDA levels were elevated in both whole brain and hippocampus under SD and HSD, reflecting widespread lipid peroxidation. The parallel increases suggest that oxidative stress induced by these lifestyle factors is not confined to a single region but represents a global neurochemical disturbance (Gaweł *et al.,* 2004; Ayala *et al.,* 2014). For antioxidant enzymes, the similar changes in SOD and CAT between the whole brain and the hippocampus indicate a coordinated compensatory response. Both enzymes are central to the enzymatic defense against ROS, and their regulation is largely systemic, mediated by transcriptional pathways such as Nrf2 (Ighodaro & Akinloye, 2018). The decreased SOD and CAT activity observed in the HSD group compared to controls does not reflect an adaptive upregulation to counter salt-induced ROS, a response that naturally occurs across multiple brain regions (Peng *et al*., 2024).

The GSH levels were consistently lower in hippocampus and cortex, highlighting depletion of non-enzymatic antioxidant reserves. GSH is consumed during detoxification of hydrogen peroxide and lipid peroxides, and its regeneration depends on glutathione reductase and NADPH availability. Because these metabolic pathways are shared across neuronal populations, depletion of GSH in the hippocampus mirrors that in the broader brain tissue. The close similarity in values therefore reflects the systemic nature of GSH oxidation under chronic oxidative stress (Adejare *et al.*, 2024; Davinelli *et al.,* 2024).

The hippocampus is central to processes of learning, memory consolidation, and spatial navigation (Coluk *et al.,* 2024). Oxidative stress in this region disrupts synaptic plasticity, impairs long-term potentiation (LTP), and reduces neurogenesis, all of which are essential for cognitive performance (Xie *et al.,* 2019). Elevated MDA levels indicate membrane damage that compromises neuronal signaling, while depletion of GSH reflects weakened antioxidant defenses, leaving hippocampal neurons vulnerable to ROS-mediated injury. Although whole brain assays confirm the systemic nature of oxidative imbalance,

hippocampal-specific changes are particularly consequential because they directly translate into cognitive dysfunction (Pepe *et al.,* 2012; Grillo *et al.,* 2019). The observed parallel biomarker trends therefore reinforce the conclusion that lifestyle stressors such as high salt intake and sleep deprivation not only impair systemic redox balance but also compromise hippocampal integrity, providing a mechanistic link between oxidative stress and cognitive decline (Lushchak & Storey, 2021).

The combined exposure to HSD and SD produced additive and synergistic effects on oxidative stress. HSD primes tissues with elevated basal ROS and reduced GSH reserves, while SD introduces transient ROS surges and suppresses enzymatic defenses (Forman *et al.,* 2009). This dual insult accelerates GSH oxidation to GSSG and impairs its regeneration via glutathione reductase and NADPH-dependent pathways. Mitochondrial dysfunction and lipid peroxidation further increase the demand for GSH conjugation, leading to marked depletion of hippocampal GSH. The net result is sustained oxidative imbalance, reflected in elevated MDA and compromised antioxidant enzyme activity. These findings highlight the vulnerability of hippocampal tissue to lifestyle stressors and underscore the importance of considering both systemic and region-specific outcomes when evaluating neurobiological consequences (Rezende *et al.,* 2022; Husain *et al.,* 2023).

The findings align with previous rodent studies linking sleep deprivation and high salt intake to oxidative stress and impaired antioxidant defenses. The observed increases in MDA and decreases in GSH corroborate reports from Abayomi *et al*. (2022), who documented similar oxidative imbalances, but diverge from Ramanathan *et al*. (2010), possibly due to differences in experimental design, exposure duration, or species-specific responses. Importantly, the observed alterations in antioxidant enzyme activity highlight the dynamic but ultimately insufficient compensatory responses of the hippocampus under chronic oxidative stress.

## CONCLUSION

Concurrent high-salt intake and sleep deprivation synergistically disrupt hippocampal redox homeostasis and impair cognitive outcomes in female Wistar rats. These findings highlight the compounded risk posed by poor dietary patterns and inadequate sleep, with implications for populations experiencing overlapping nutritional and lifestyle stressors.

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