International Journal of Sport Studies for Health

Journal Homepage

Changes in Oxidant–Antioxidant Status of Hippocampal Tissue Following Eight Weeks of Aerobic Exercise and Vitamin E Supplementation in Reserpine-Induced Parkinsonian Rats

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A r t i c l e I n f o

Article type:
Original Research

How to cite this article:
Sadeghi, F., Banai, J., Eftekhari Ghinani, E., & Keshavarz, S. (2025). Changes in Oxidant–Antioxidant Status of Hippocampal Tissue Following Eight Weeks of Aerobic Exercise and Vitamin E Supplementation in Reserpine-Induced Parkinsonian Rats. *International Journal of Sport Studies for Health*, *8*(1), 86-93.
http://dx.doi.org/10.61838/kman.intjssh.8.1.10

A B S T R A C T

Objective: The present study aimed to investigate changes in the oxidant–antioxidant status of hippocampal tissue following eight weeks of aerobic exercise (AT) and vitamin E (VE) supplementation in reserpine-induced Parkinsonian rats.

Methods and Materials: In this experimental study, 40 male Sprague-Dawley rats (aged 14–16 months, weighing 250–270 grams) were induced with Parkinson's disease using 2 mg/kg reserpine (Res) and were divided into the following groups: (1) PD, (2) AT, (3) VE, and (4) AT+VE. To evaluate the effects of Res on the variables, eight healthy control (HC) rats were included. Aerobic exercise was performed for eight weeks, five sessions per week, with each session lasting 15–48 minutes at a speed of 10–24 m/min. VE supplementation was administered orally at a dose of 30 mg/kg daily. Data analysis was performed using one-way ANOVA and Tukey's post-hoc test ($P \leq 0.05$).

Findings: The Res+AT, Res+VE, and Res+AT+VE groups showed higher levels of superoxide dismutase (SOD) and total antioxidant capacity (TAC) and lower levels of malondialdehyde (MDA) compared to the Res group ($P = 0.05$). In the Res+AT+VE group, SOD levels were higher and MDA levels were lower than in the Res+AT and Res+VE groups ($P = 0.001$). Additionally, TAC levels in the Res+AT and Res+AT+VE groups were higher than in the Res+VE group ($P = 0.001$).

Conclusion: Aerobic exercise and vitamin E supplementation appear to have antioxidant effects on brain tissue, both individually and interactively. Given the more favorable effects of combining exercise with vitamin E, the simultaneous use of these two interventions is recommended in neurodegenerative diseases.

Keywords: *Exercise, Vitamin E, Antioxidant, Hippocampus, Parkinson's Disease.*

Article history:
Received 28 September 2024
Revised 10 December 2024
Accepted 16 December 2024
Published online 01 January 2025

1. Introduction

With the aging of human societies and under the shadow of economic and social challenges, the incidence of neurodegenerative diseases has also increased. Among these diseases, Parkinson's disease (PD) is identified as the second most common disease after Alzheimer's disease. Evidence indicates that PD is characterized by the occurrence of motor and non-motor disorders, cognitive impairments, bradykinesia, tremors, and muscle rigidity (1).

Statistics show that the incidence of PD is related to various factors such as geographical region, race, age, and gender, with the highest risk of the disease reported with increasing age (2). Reports indicate that in two of the world's most populous countries, the United States currently has over one million individuals with PD, while China has 3.6 million cases, imposing an annual cost of $52 billion on these societies (3).

From a physiological perspective, current data suggest that mitochondrial dysfunction following aging leads to an increase in free radicals, which combine with nitrogen and oxygen present in the cytoplasm, resulting in the production of reactive oxygen species (ROS) (4). Subsequently, ROS interact with cellular biomolecules, leading to increased lipid peroxidation (indicated by malondialdehyde (MDA)), protein peroxidation (indicated by protein carbonyl (PC)), and DNA oxidation. Under these conditions, the balance between oxidants and antioxidants (such as superoxide dismutase (SOD) and glutathione peroxidase (GPx)) shifts in favor of oxidants, resulting in reduced total antioxidant capacity (TAC), and triggering cellular damage processes such as inflammation, neuronal fibrillation, and neuronal apoptosis (5).

Given the rising prevalence of neurodegenerative diseases, researchers believe that physical activity can play an effective role in halting disease progression. It is believed that physical exercise can improve brain metabolism, cognitive function, and neuronal plasticity, thus enhancing the quality of life both physically and psychologically in neurodegenerative diseases (6, 7).

Specifically, researchers have suggested that physical exercise, by increasing catecholamines in response to exercise-induced stress, activates phosphoinositide 3-kinase (PI3K), which subsequently activates nuclear respiratory factor 2 (NRF2). NRF2 then dissociates from its inhibitor Keap1 and translocates to the DNA, where it promotes the transcription of antioxidant enzymes such as SOD and catalase (8).

In this context, researchers have shown that both high-intensity interval training (HIIT) and continuous training improve cognitive function and increase SOD levels, although the effect of HIIT was more favorable than continuous training. However, the effect of these training types on GPx and catalase was not significant (9).

In another study, researchers demonstrated that endurance, resistance, and combined training each reduced MDA, total oxidative stress (TOS), and tumor necrosis factor-alpha (TNF-α), while increasing TAC, SOD, and glutathione in the brain tissue of morphine-addicted rats (10).

Furthermore, a study showed that four weeks of swimming exercise significantly reduced MDA and increased SOD and GPx in the brain tissue of Parkinsonian rats (11).

The study by Moradi et al. (2020) demonstrated that six weeks of interval aerobic training reduced MDA levels in PD rats, while continuous training had no significant effect. Additionally, dopamine levels improved in both types of training, with only interval aerobic training significantly increasing GPx. Despite prior studies, the antioxidant mechanism of exercise on brain tissue is not yet fully understood.

Since dietary recommendations and the use of side-effect-free antioxidants are mainstays of neurodegenerative disease management, the side effects of synthetic drugs have prompted the use of antioxidants such as vitamin E (VE) as a potentially beneficial option for neurodegenerative diseases (12).

VE, composed of α-, β-, γ-, and δ-tocopherol, is known for its antioxidant properties in neurological disease management by neutralizing free radicals and influencing antioxidant transcription pathways such as PI3K/NRF2 (13).

In this regard, daily consumption of 40 mg/kg VE has been shown to reduce MDA levels and increase catalase, SOD, glutathione reductase, and glutathione peroxidase in immobilized rats (12). Another study demonstrated that VE supplementation improved sleep disorders, increased GPx, SOD, and catalase levels, and reduced the GSH/GSSG ratio in individuals with sleep disorders (14).

Additionally, a dietary study indicated an inverse relationship between VE intake and the incidence of PD (15). Despite existing research, studies examining the simultaneous effects of aerobic training (AT) and VE on the brain's antioxidant defense system remain limited. Researchers have shown that concurrent HIIT and VE improve anxiety, depression, physical performance, and

caloric expenditure in Alzheimer's model rats (7). Moreover, a study demonstrated that exercise and VE improved cognitive function and increased antioxidant capacity in older adults (16). A recent study showed that AT and VE improved mitochondrial biogenesis in the brain tissue of reserpine-induced Parkinsonian rats (17).

Given the uncertainties surrounding the oxidant–antioxidant effects of various exercise modalities and the pressing need for non-invasive, side-effect-free strategies to manage PD progression, investigating cellular and molecular changes following exercise and antioxidant interventions seems essential. Therefore, the present study aimed to investigate changes in the oxidant–antioxidant status of hippocampal tissue following eight weeks of aerobic exercise and vitamin E supplementation in reserpine-induced Parkinsonian rats.

2. Methods and Materials

2.1 Animal Preparation and Maintenance

In this experimental and fundamental study, 48 male Sprague-Dawley rats, aged 15.12 ± 2.14 months and weighing 260.35 ± 30.20 grams, were obtained from the Animal Breeding and Research Center at Islamic Azad University, Marvdasht Branch. After procurement, the rats were housed in the university's exercise physiology laboratory for seven days to acclimate to the environment. Throughout the study, ethical principles for working with laboratory animals and optimal housing conditions were maintained, including a 12-hour light/dark cycle, moderate temperature (22–24°C), relative humidity of 55–60%, and free access to water and a specialized rat diet, in accordance with the Helsinki Treaty.

2.2 Induction of Parkinson's Disease and Study Design

Eight days after the acclimation period, 40 rats were anesthetized using ether. Once anesthesia was confirmed, the rats received an intraperitoneal injection of 2 mg/kg of reserpine neurotoxin dissolved in citrate buffer and normal saline. Clinical behaviors and Parkinson's symptoms, including periocular hemorrhage, aggression, anxiety, tail twisting, gait disturbances, and rotational tests, were assessed to ensure disease induction (18). After 14 days, the PD-induced rats were divided into four groups: (1) Res, (2) Res + vitamin E (Res+VE), (3) Res + training (Res+T), and (4) Res+T+VE. Eight healthy rats were designated as the healthy control (HC) group to assess the effects of Res on oxidant-antioxidant markers.

2.3 Exercise Protocol

Aerobic training was performed for eight weeks, with five sessions per week. Initially, the rats warmed up for 5 minutes at a speed of 8 m/min. In the first week, rats ran for 15 minutes at 10 m/min. From the second week onwards, treadmill speed increased by 2 m/min each week, and the duration increased by 4.1 minutes weekly, reaching 24 m/min and 48 minutes of exercise by the eighth week (19, 20).

2.4 Vitamin E Preparation and Administration

Vitamin E supplements were procured from SolarBio, China. Based on the number and weight of the rats in the supplement groups, 1.2 grams of vitamin E were dissolved daily in 4.8 cc of dextrose. Each rat received 0.3 cc of the solution orally, equivalent to 30 mg/kg body weight of vitamin E (21).

2.5 Dissection and Sampling

Forty-eight hours after the final training session and following a 12-hour fasting period, the rats were anesthetized with 50 mg/kg ketamine and 20 mg/kg xylazine from Alfasan, the Netherlands. Anesthesia was confirmed using pain and paw compression tests. The cranial cavity was opened with a cutter, and brain tissue was carefully extracted. The hippocampus was isolated immediately, stored in cryotubes, and transferred to –70°C for preservation.

2.6 Measurement of Variables

MDA levels were measured using an ELISA kit from ZelBio, Germany (CAT No. ZB-MDA-96A) in micromolar units. SOD levels were measured using an ELISA kit from Navand Lab Kit, Iran (Version 0.6) in U/ml or mg protein. TAC levels were measured using an ELISA kit from ZelBio, Germany (CAT No. ZB-TAC-96A) in µmol/L.

2.7 Data Analysis

The Shapiro-Wilk test was used to assess data distribution. One-way ANOVA was conducted to compare group differences, and Tukey's post-hoc test was performed

for pairwise comparisons using SPSS version 26, with significance set at $P \leq 0.05$.

3. Results

One-way ANOVA indicated significant differences in hippocampal SOD (P = 0.001, F = 98.37), MDA (P = 0.001, F = 37.10), and TAC (P = 0.001, F = 46.38) levels among the study groups.

Tukey's post-hoc test showed significantly lower SOD levels in the Res group compared to the HC group (P = 0.001), while Res+VE (P = 0.001), Res+T (P = 0.001), and Res+T+VE (P = 0.001) groups exhibited significantly higher SOD levels than the Res group. No significant difference in SOD levels was observed between the Res+VE and Res+T groups (P = 0.064), but the Res+T+VE group showed significantly higher levels than both Res+VE (P = 0.001) and Res+T (P = 0.001) groups (Figure 1).

MDA levels were significantly higher in the Res group compared to the HC group (P = 0.001). However, MDA levels in the Res+VE (P = 0.001), Res+T (P = 0.001), and Res+T+VE (P = 0.001) groups were significantly lower than in the Res group. No significant difference in MDA levels was found between the Res+VE and Res+T groups (P = 0.99), while the Res+T+VE group showed significantly lower levels than both Res+VE (P = 0.008) and Res+T (P = 0.007) groups (Figure 2).

TAC levels were significantly lower in the Res group compared to the HC group (P = 0.001). However, TAC levels in the Res+VE (P = 0.002), Res+T (P = 0.001), and Res+T+VE (P = 0.001) groups were significantly higher than in the Res group. TAC levels in the Res+T (P = 0.001) and Res+T+VE (P = 0.001) groups were significantly higher than in the Res+VE group, with no significant difference between the Res+T and Res+T+VE groups (P = 0.20) (Figure 3).

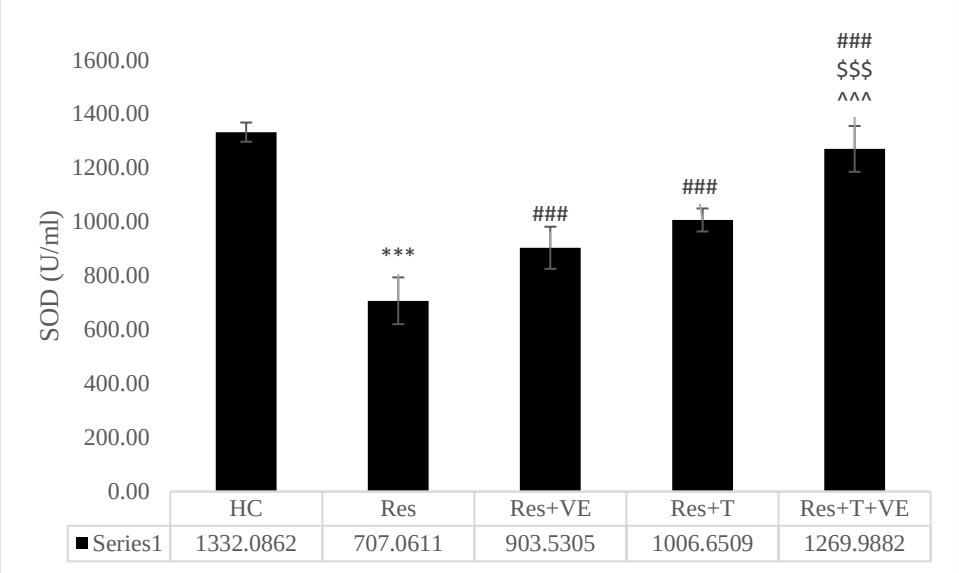

Figure 1. SOD levels in hippocampal tissue of study groups. *** (P = 0.001) Significant decrease compared to HC; ### (P = 0.001) Significant increase compared to Res; $$$ (P = 0.001) Significant increase compared to Res+VE; ^^^ (P = 0.001) Significant increase compared to Res+T.

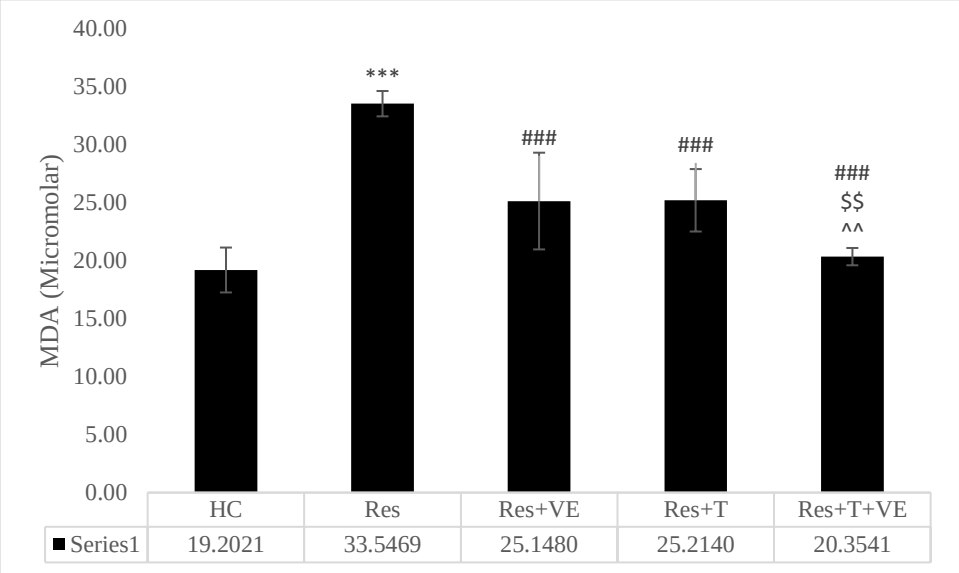

Figure 2. MDA levels in hippocampal tissue of study groups. *** (P = 0.001) Significant increase compared to HC; ### (P = 0.001) Significant decrease compared to Res; $$ (P = 0.01) Significant decrease compared to Res+VE; ^^ (P = 0.01) Significant increase compared to Res+T.

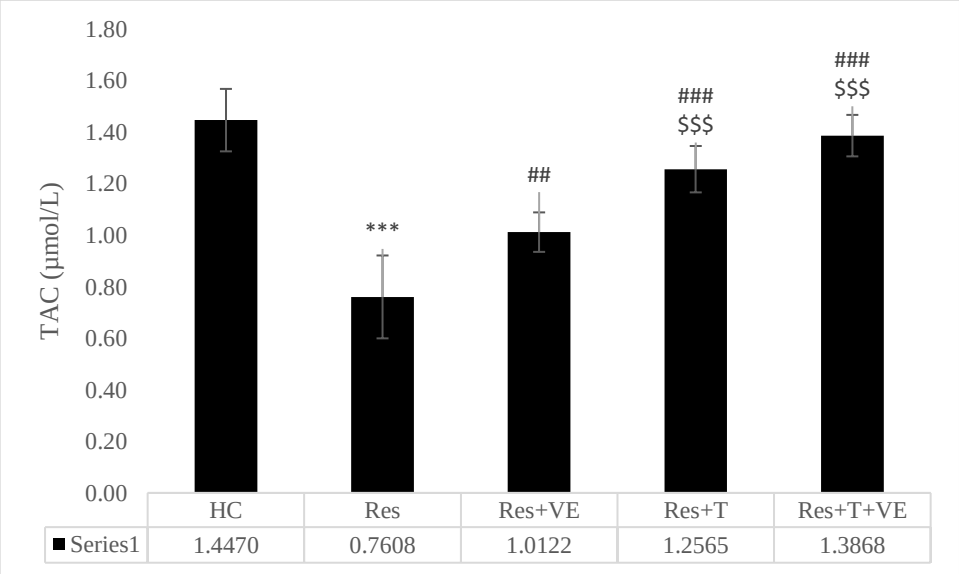

Figure 3. TAC levels in hippocampal tissue of study groups. *** (P = 0.001) Significant decrease compared to HC; ### (P = 0.001) and ## (P = 0.01) Significant increase compared to Res; $$$ (P = 0.001) Significant increase compared to Res+VE.

4. Discussion and Conclusion

The results showed that in the Res group, SOD and TAC levels were significantly lower, while MDA levels were significantly higher compared to the HC group. However, in the Res+T group, SOD and TAC levels were significantly higher and MDA levels were lower compared to the Res group. Due to the challenges associated with studying the central nervous system in human samples, researchers utilize various methods to model PD in animal models, which facilitates a better understanding of the cellular and molecular mechanisms of PD. Reserpine is a chemical agent historically used in modeling PD, as it disrupts monoamine transporter functions, including dopamine and L-dopa, leading to mitochondrial dysfunction, increased oxidative stress, elevated alpha-synuclein (α-syn) levels, apoptosis, and ultimately cognitive impairment (18).

As previously mentioned, physical activity increases AMPK, leading to PI3K phosphorylation and subsequent NRF2 activation, which then binds to the antioxidant response element (ARE) on DNA, initiating the transcription of antioxidant enzymes such as SOD and catalase. This sequence of events inactivates heme oxygenase-1 and neutralizes other ROS, ultimately reducing lipid peroxidation in neurons (8). Consistent with the present study, resistance training over six weeks has been shown to increase SOD, GPx, TAC, and decrease MDA levels in the brain tissue of stanozolol-exposed rats (22).

Additionally, another study demonstrated that both moderate- and high-intensity exercise increased SOD, GPx, TAC, and reduced MDA levels in the liver tissue of rats with autism (23). However, another study found that six weeks of HIIT reduced MDA levels, while continuous training did not produce significant changes, though both training types increased GPx and dopamine levels in the brain tissue of PD rats (23). Another study showed that while HIIT and continuous training similarly affected NGF levels, HIIT was more effective in increasing catalase levels (19). The results suggest that exercise intensity and duration may serve as stimuli for activating antioxidant mechanisms through oxidative stress generation.

Furthermore, short-term low-, moderate-, and high-intensity exercises have all been shown to significantly increase cerebral dopamine neurotrophic factor (CDNF), with high-intensity exercise having a more favorable effect. SOD levels increased in all three exercise intensities, but MDA levels showed no significant changes, except for a slight increase following an acute single-session exercise (24).

The results also indicated that in the Res+VE group, SOD and TAC levels were significantly higher and MDA levels significantly lower compared to the Res group. Al-Sowayan and Almarzougi (2024) similarly reported that 40 mg/kg VE increased SOD, GPx, GSH, and reduced MDA levels in the brain tissue of immobilized rats (12). Another study showed that VE supplementation in animal models of neurological disorders increased BDNF and reduced beta-amyloid (13). Additionally, 100 mg/kg VE supplementation increased GPx, SOD, GSH, and reduced MDA levels in the brain tissue of sleep-disorder-induced rats (14).

In the study by Hamid et al. (2011), eight weeks of 30 mg/kg VE supplementation decreased SOD and catalase activity, though DNA damage indices significantly declined post-supplementation (21). Usman et al. (2023) demonstrated that VE supplementation increased antioxidant enzymes (SOD, GPx, and TAC) and reduced MDA levels in the brain tissue of rats with traumatic brain injury (25). Most of these studies, except for Hamid et al. (2011), were consistent with the present study, and differences in baseline levels and disease models likely contributed to the discrepancies (21).

VE, with its cholesterol-binding capacity, can increase high-density lipoproteins (HDL), reduce polysaccharides, lipid peroxidation, and free radicals in brain tissue through its non-enzymatic antioxidant mechanism. Additionally, VE directly activates the PI3K pathway, facilitating antioxidant enzyme transcription via NRF2 activation, and also stimulates CaMKII, CREB, synapsin 1, and BDNF pathways in the nervous system, ultimately improving cognitive function (13).

The present study showed that in the Res+T+VE group, SOD and TAC levels were higher and MDA levels lower compared to the Res group. Furthermore, SOD levels were higher and MDA levels lower in the Res+T+VE group compared to the Res+T and Res+VE groups, while TAC levels were higher in both the Res+T and Res+T+VE groups compared to the Res+VE group. Although no studies were found examining the combined effect of exercise and VE on the brain's oxidant-antioxidant system, exercise, through AMPK elevation and PI3K/NRF2/ARE pathway activation, appears to increase SOD and catalase expression, reduce MDA, and enhance TAC (8, 22). VE supplementation, through lipid metabolism improvement, free radical neutralization, and polysaccharide reduction via non-enzymatic antioxidant pathways, along with

PI3K/NRF2/ARE activation, increases antioxidant expression and CREB/BDNF activity in brain tissue (13). Both interventions seem to share similar pathways, with their combined effect surpassing individual effects, suggesting mutual reinforcement.

VE and exercise have independently and jointly been shown to reduce SOD and catalase levels in rats, with VE providing greater protection against DNA damage (21). Additionally, Hajarian et al. (2023) demonstrated that exercise and VE together enhanced mitochondrial biogenesis in PD rat brain tissue (17). The absence of PI3K/NRF2 pathway assessment is a limitation of this study, and future studies should evaluate this pathway. Moreover, the lack of oxidative stress assessment through histopathological methods like hematoxylin-eosin staining is another limitation, which future studies should address using various methodologies.

It appears that aerobic exercise and VE supplementation have antioxidant effects on brain tissue both independently and interactively, with their combined use being more favorable for managing neurodegenerative diseases.

Authors' Contributions

F. S. designed the study, performed the experiments, and contributed to data analysis. J. B. assisted with experimental procedures, supervised the research, and provided critical revisions to the manuscript. E. E. G. contributed to data collection, statistical analysis, and manuscript drafting. S. K. conceptualized the research, coordinated the study, and provided final approval of the manuscript. All authors reviewed and approved the final manuscript and are accountable for the accuracy and integrity of the work.

Declaration

In order to correct and improve the academic writing of our paper, we have used the language model ChatGPT.

Transparency Statement

Data are available for research purposes upon reasonable request to the corresponding author.

Acknowledgments

We would like to express our gratitude to all individuals helped us to do the project.

Declaration of Interest

The authors report no conflict of interest.

Funding

According to the authors, this article has no financial support.

Ethics Considerations

Ethical standards for animal research were supervised by the Biomedical Research Ethics Committee under the approved code IR.IAU.NAJAFABAD.REC.1401.052.

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