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Full-Length Research Article

Protective Effect of Vitamin E Against Mobile Phone–Induced Oxidative Stress and Neuronal Injury in Rats

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Summary: Chronic exposure to radiofrequency electromagnetic radiation (RF-EMR) emitted from mobile phones has raised concerns regarding its potential biological effects on the central nervous system. The present study investigated whether prolonged RF-EMR exposure induces oxidative, neuroendocrine, and neuronal stress in rats, and whether Vitamin E supplementation offers protective effects. Male Sprague-Dawley (SD) rats were divided into control, RF-EMR–exposed, RF-EMR plus Vitamin E–treated, and vehicle control groups. Animals were exposed to mobile phone–emitted RF-EMR (0.9/1.8 GHz) for 1 hour/day over 50 days and supplemented with Vitamin E (100 mg/kg body weight, orally through oral gavage needle). Feed intake and body weight were recorded, while serum malondialdehyde (MDA), corticosterone, and neuron-specific enolase (NSE) levels were assessed as markers of oxidative stress, neuroendocrine stress, and neuronal injury, respectively. Hippocampal glial fibrillary acidic protein (GFAP) expression was also evaluated. Compared with control rats, RF-EMR exposure did not significantly alter feed intake or body weight; however, serum MDA, corticosterone, and NSE levels were significantly elevated ($p < 0.05$). Vitamin E supplementation significantly attenuated these biochemical alterations compared with the RF-EMR group ($p < 0.05$). GFAP expression in the hippocampal CA3 region showed no significant differences among groups. These findings suggest that chronic RF-EMR exposure induces biochemical and neuronal stress prior to overt behavioral changes, and that Vitamin E provides effective antioxidant and neuroprotective benefits.

Keywords*: RF-EMR, corticosterone, MDA, NSE, GFAP, mobile radiation, antioxidant protection*

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INTRODUCTION

The extensive global use of mobile phones has resulted in continuous exposure to RF-EMR, a form of non-ionising radiation emitted during communication and data transfer activities. Although RF-EMR does not directly ionize biological molecules, accumulating evidence suggests that chronic exposure may induce physiological stress and subtle neurobiological alterations (Hardell & Carlberg, 2015). Among the various organ systems, the central nervous system (CNS) is particularly vulnerable to RF-EMR-induced effects due to its high metabolic demand, lipid-rich composition, and limited antioxidant capacity (Yakymenko *et al*., 2016). Such alterations may manifest initially as biochemical and cellular stress responses that precede overt functional or behavioral disturbances.

Stress-inducing stimuli, including RF-EMR, are known to activate the hypothalamic–pituitary–adrenal (HPA) axis, leading to elevated glucocorticoid secretion and altered neuroendocrine signaling (Pall, 2016). Prolonged activation of these pathways can influence neuronal homeostasis and systemic physiology. While the hypothalamus is classically recognized as the primary center regulating hunger and satiety, feeding-related regulation extends beyond homeostatic mechanisms and involves higher brain regions associated with cognition, memory, and contextual processing (Davidson *et al*., 2009; Kanoski & Davidson, 2011). In this context, the hippocampus plays a modulatory role by integrating metabolic cues with learned experiences, environmental information, and stress-related inputs, thereby influencing decisions related to what to eat, when to eat, and how much to eat (Davidson *et al*., 2009; Tracy *et al*., 2016).

The hippocampus, particularly the CA3 region, is highly sensitive to oxidative stress and glucocorticoid exposure due to its dense excitatory circuitry and high expression of stress hormone receptors (McEwen, 2007). Alterations within this region have been reported under chronic stress conditions and may indirectly affect feeding-related decision-making through impaired cognitive and stress-regulatory processes rather than through direct appetite control (Kanoski & Davidson, 2011). Experimental studies have reported that electromagnetic radiation exposure may influence food intake patterns, circadian feeding rhythms, and metabolic balance; however, such functional changes are believed to

arise secondary to underlying oxidative stress, neuronal injury, and glial responses (Kim *et al*., 2019).

Oxidative stress is considered a central mechanism underlying RF-EMR-induced biological effects. Increased production of reactive oxygen species (ROS) can overwhelm endogenous antioxidant defenses, resulting in lipid peroxidation, membrane damage, and neuronal injury (Yakymenko *et al*., 2016). MDA, a stable end-product of lipid peroxidation, is widely used as a marker of oxidative damage. NSE serves as a sensitive indicator of neuronal injury and cellular stress and has been reported to increase following prolonged RF-EMR exposure in experimental models (Odaci *et al*., 2008; Pagadala *et al*., 2022).

In addition to neuronal injury, RF-EMR exposure has been associated with glial responses, particularly astrocytic alterations. GFAP, an intermediate filament protein expressed by astrocytes, is commonly used as a marker of astrocytic reactivity rather than definitive neuroinflammation. Changes in GFAP expression provide insight into astrocytic responses and central nervous system vulnerability under conditions of oxidative and metabolic stress. (Srivastava *et al*., 2018).

Vitamin E (α-tocopherol), a lipid-soluble antioxidant, plays a crucial role in protecting cellular membranes against oxidative damage by scavenging free radicals and inhibiting lipid peroxidation (Al-Shabanah *et al*., 1998). Its antioxidant and cytoprotective effects have been demonstrated in several experimental models of oxidative stress, where Vitamin E supplementation reduced MDA levels and attenuated markers of neuronal injury (Jelodar *et al*., 2016; Hassan *et al*., 2020). Based on these properties, Vitamin E is hypothesized to mitigate RF-EMR-induced oxidative stress, neuroendocrine activation, and neuronal injury.

In light of increasing environmental exposure to RF-EMR and limited clarity regarding its early neurobiological effects, the present study was designed to evaluate RF-EMR-induced oxidative and neuronal stress in rodents and to assess the potential protective role of Vitamin E. Feed intake and body weight were assessed as supportive physiological parameters to determine whether biochemical and neuronal alterations occur in the absence of overt functional disturbances, while oxidative stress markers (MDA), stress hormone levels (corticosterone), neuronal injury markers (NSE), and hippocampal GFAP expression were evaluated to provide mechanistic insight into RF-EMR-associated CNS effects. The objectives of this study are (1). To evaluate the effect of RF-EMR emitted from mobile phones on feed intake in rats. (2) To assess RF-EMR–induced neuronal injury by measuring NSE, MDA levels in serum blood samples, and GFAP expression in rat brain tissue and (3) To determine the protective effect of Vitamin E supplementation on RF-EMR–induced alterations in feed intake, oxidative stress biomarkers (NSE, MDA), and GFAP expression in rodents.

MATERIALS AND METHODS

The study was carried out at the Central Animal House, Sri Devaraj Urs Academy of Higher Education & Research, Kolar, Karnataka, as a prospective comparative experiment after obtaining approval from the Institutional Animal Ethics Committee (IAEC/PHARMA/SDUMC/2017–18/8a) for the use of twenty-four rats. Male Sprague Dawley rats (10–12 weeks old, weighing 180–220 g) were housed in polypropylene cages under controlled laboratory conditions (Temperature 23 ± 2 °C, relative humidity 55 ± 5%, and a 12:12 h light–dark cycle) with ad libitum access to standard pellet diet and RO water (National Research Council, 2011). Animals were acclimatized for two weeks prior to experimentation, and all experimental procedures were conducted in accordance with CPCSEA guidelines (CPCSEA, 2010).

The standard pellet diet contained 10% moisture, 18% crude protein, 4% crude fiber, 4% crude fat, 60% nitrogen-free extract, 1% calcium, and 0.5% phosphorus. Paddy husk bedding was replaced on alternate days to maintain hygienic conditions.

Experimental Design: Twenty-four rats were randomly divided into four groups (n = 6 per group), with two animals housed per cage (Ilhan *et al.*, 2004; Odaci *et al.*, 2008).

Group I – Control: Animals were not exposed to RF-EMR and had free access to food and RO water.

Group II – RF-EMR: Animals were exposed to RFEMR emitted from a commercially available 4G Android mobile phone (Lenova A6000/2015) operating at GSM frequencies (0.9/1.8 GHz) with a Specific absorption rate (SAR) of 1.4 watt/kg for 1 hour/day for 50 days. The mobile phone was placed in continuous call (answer) mode throughout the exposure period to ensure sustained RF-EMR emission, and radiation levels were verified using an Electrosmog Meter (ED-178s) prior to and during exposure sessions to ensure consistency. Rats were allowed freely in their home cages to avoid environmental stress and the mobile phone was suspended centrally with in the cage with free access to feed and water. Network-related power fluctuations were minimized by maintaining stable signal conditions throughout the exposure period. (Mal uin *et al*., 2021)

Group III – RF-EMR + Vitamin E: Animals were exposed to RF-EMR as described above and concurrently supplemented with Vitamin E at a dose of 100 mg/kg body weight, administered orally via gavage for 50 days (Hassan *et al*., 2020).

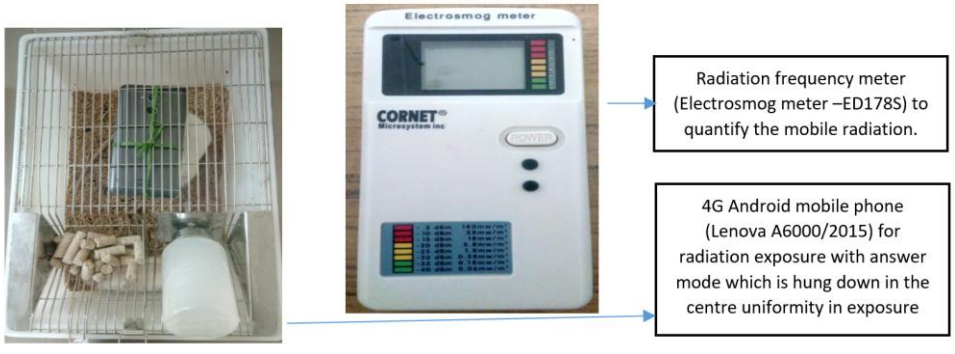

Figure1:
Image shows the cage with rats and mobile phone during radiation experience and radiation frequency meter (Electrosmog meter – ED178S) to quantify the mobile radiation.

Figure 2:
Image shows supplementation of Vitamin E as well as water orally through oral gavage needle.

Group IV – Vehicle Control: Animals received water (0.2 mL), the vehicle for Vitamin E, administered orally once daily using a calibrated gavage needle for 50 days, with free access to standard feed and RO water.

Group I served as an untreated baseline control to establish normal physiological parameters, while Group IV functioned as a vehicle control to account for handling and oral gavage–related effects. Neither group was exposed to RF-EMR, allowing separation of baseline values from procedural influences associated with oral administration.

Assessment of Body Weight and Feed intake; Body weight of the animals was recorded at 10-day intervals using an electronic balance. Feed intake was assessed by individually housing the rats and providing pre-weighed food pellets and RO water. Feed intake was calculated based on the difference in weight after 24 hours (Pagadala *et al*., 2023).

Blood Collection and Brain Harvesting; At the end of the 50-day experimental period, blood samples were collected via retro-orbital puncture under deep anesthesia using ketamine (50–80 mg/kg) and xylazine (5–10 mg/kg), performed by trained personnel in accordance with IAEC approval and CPCSEA guidelines. This method was employed to enable rapid collection of an adequate serum volume prior to immediate tissue harvesting, thereby minimizing procedural duration. Approximately 2 mL of blood was allowed to clot and centrifuged at 3000 rpm for 20 minutes. Serum was separated and stored at −20 °C until biochemical analysis. Animals were euthanized immediately after blood collection, and brains were promptly excised and fixed for subsequent GFAP immunostaining.

Biochemical Analysis: Serum corticosterone: Serum corticosterone levels were measured using a commercially available ELISA kit to confirm stress response.
MDA: Lipid peroxidation was assessed by estimating serum MDA levels using the method described previously for oxidative stress assessment (Pagadala *et al*., 2022).
NSE: Serum NSE levels were quantified using a commercial ELISA kit (Cat. No. BC-ER140914; Biocodon Technologies, USA).

GFAP Immunostaining and Quantification; Fixed brain tissues were processed for GFAP staining. Hippocampal sections were imaged at 40× magnification using a Zeiss digital microscope with ZEN software. GFAP-positive glial cells in the CA3 region were quantified using Image J software.

Statistical Analysis
Data were analyzed using GraphPad Prism 8.0.3 and expressed as mean ± SEM (n=6). Two-way ANOVA followed by Dunnett's post-hoc test was used for body weight and feeding behavior. One-way ANOVA with Dunnett's post-hoc test was applied for biochemical parameters (NSE, MDA, corticosterone, GFAP). Statistical significance was set at $p < 0.05$

RESULTS

Effect of RF-EMR and Vitamin E on Feed intake: Feed intake remained consistent and optimal in both the control and vehicle groups throughout the study. RF-EMR–exposed rats, with or without Vitamin E supplementation, did not show any statistically significant changes in feed consumption compared with the control group.

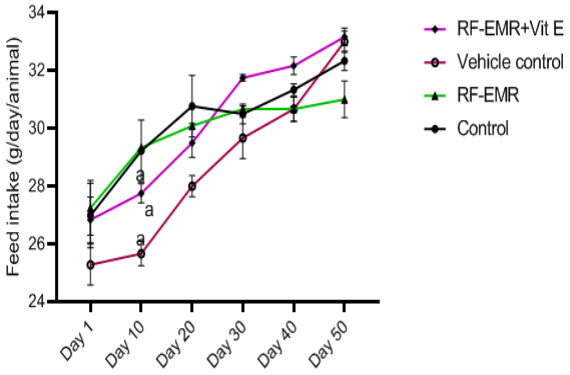

Figure 3:
Effect of RF-EMR exposure and Vitamin E supplementation on daily feed intake in rats over the experimental period. Rats were divided into control, RF-EMR–exposed (1 h/day for 50 days), RF-EMR + Vitamin E–treated (100 mg/kg/day, orally through oral gavage needle), and vehicle control groups (n = 6 per group). Data are expressed as mean ± SEM. Statistical analysis was performed by using two-way ANOVA followed by Dunnett's post-hoc test; $p < 0.05$ was considered statistically significant.

Effect of RF-EMR and Vitamin E on Body Weight: Control and vehicle groups demonstrated a normal, progressive increase in body weight over the experimental period. Rats exposed to RF-EMR showed a slight reduction in body-weight gain compared with controls; however, this difference did not reach statistical significance. Animals in the RF-EMR + Vitamin E group exhibited body-weight values comparable to those of control rats, with no statistically significant differences observed among groups (Figure 4).

Effect of RF-EMR and Vitamin E on Serum Corticosterone: RF-EMR exposure resulted in a significant elevation ($p=0.002$) in serum corticosterone levels relative to controls, confirming stress induction. Vitamin E co-treatment markedly attenuated this increase. The vehicle group showed no significant difference compared to controls (Figure 5).

Effect of RF-EMR and Vitamin E on Serum MDA Levels
Chronic RF-EMR exposure significantly increased ($p=0.004$) serum MDA levels, indicating enhanced lipid peroxidation. Vitamin E supplementation for 50 days

effectively prevented this rise, resulting in MDA levels comparable to the control group. No significant differences were observed in the vehicle group (Figure 6).

Effect of RF-EMR and Vitamin E on Serum NSE Levels: Long-term RF-EMR exposure caused a significant rise (p=0.0001) in serum NSE levels, reflecting neuronal injury. Vitamin E supplementation significantly reduced NSE levels in the RF-EMR+Vitamin E group, normalising them to values similar to controls. The vehicle group did not differ significantly from the control. (Figure 7).

Effect of RF-EMR and Vitamin E on GFAP Expression: No significant differences in GFAP expression were detected among the control, vehicle, RF-EMR, and RF-EMR+Vitamin E groups, indicating no measurable astrocytic activation under the experimental conditions. Quantification of the GFAP glial cell density in the study groups (Plate 1).

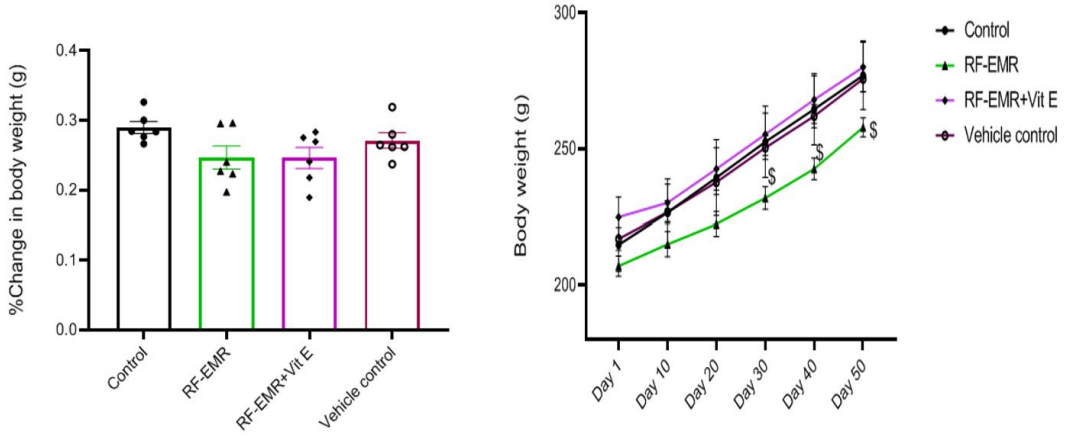

Figure 4:
Effect of RF-EMR exposure and Vitamin E supplementation on percentage change in body weight in rats over the experimental period. Rats were divided into control, RF-EMR–exposed (1 h/day for 50 days), RF-EMR + Vitamin E–treated (100 mg/kg/day, orally through oral gavage needle), and vehicle control groups (n = 6 per group). Data are expressed as mean ± SEM. Statistical analysis was performed by using two-way ANOVA followed by Dunnett's post-hoc test; $p < 0.05$ was considered statistically significant.

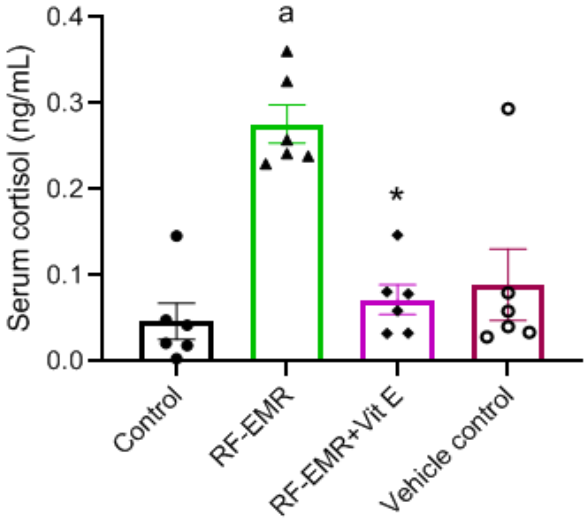

Figure 5:
Effect of RF-EMR exposure and Vitamin E supplementation on serum corticosterone levels in rats. Rats were divided into control, RF-EMR–exposed (1 h/day for 50 days), RF-EMR + Vitamin E–treated (100 mg/kg/day, orally through oral gavage needle), and vehicle control groups (n = 6 per group). Data are expressed as mean ± SEM. Statistical analysis was performed by using one-way ANOVA followed by Dunnett's post-hoc test; $p < 0.05$ was considered statistically significant. Significance labels: 'a' denotes the significance vs Control group; * denotes the significant difference between RF-EMR control and RF-EMR + Vitamin E

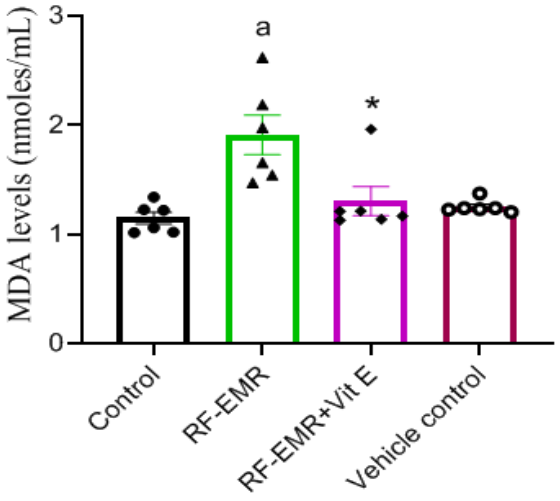

Figure 6:
Effect of RF-EMR exposure and Vitamin E supplementation on serum MDA levels in rats. Rats were divided into control, RF-EMR–exposed (1 h/day for 50 days), RF-EMR + Vitamin E–treated (100 mg/kg/day, orally through oral gavage needle), and vehicle control groups (n = 6 per group). Data are expressed as mean ± SEM. Statistical analysis was performed by using one-way ANOVA followed by Dunnett's post-hoc test; $p < 0.05$ was considered statistically significant. Significance labels: 'a' denotes the significance vs Control group; * denotes the significant difference between RF-EMR control and RF-EMR + Vitamin E

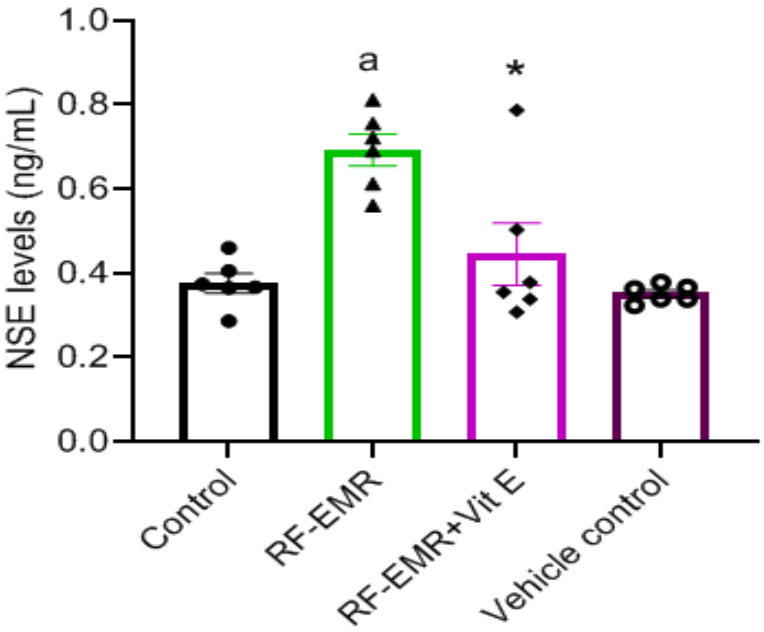

Figure 7
Effect of RF-EMR exposure and Vitamin E supplementation on serum NSE levels in rats. Rats were divided into control, RF-EMR-exposed (1 h/day for 50 days), RF-EMR + Vitamin E–treated (100 mg/kg/day, orally through oral gavage needle), and vehicle control groups (n = 6 per group). Data are expressed as mean ± SEM. Statistical analysis was performed by using one-way ANOVA followed by Dunnett's post-hoc test; $p < 0.05$ was considered statistically significant. Significance labels: 'a' denotes the significance vs Control group; * denotes the significant difference between RF-EMR control and RF-EMR + Vitamin E.

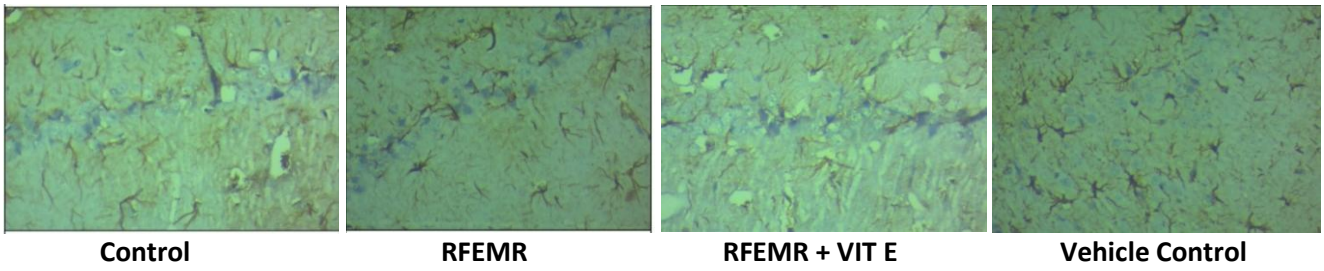

Plate 1:
Representative photomicrographs and quantitative analysis of GFAP immunostaining in the hippocampal CA3 region of rat brain. Sections from control, RF-EMR–exposed (1 h/day for 50 days), RF-EMR + Vitamin E–treated (100 mg/kg/day, orally), and vehicle control groups are shown. Representative images were captured at 40× magnification under identical imaging conditions. Quantitative data are expressed as mean ± SEM (n = 6 per group).

DISCUSSION

The exponential increase in mobile phone usage over the past two decades has raised concerns regarding the potential biological effects of chronic RF-EMR exposure. Several experimental and observational studies have associated RF-EMR exposure with physiological and neurobiological alterations, including increased stress levels, sleep disturbances, cognitive impairment, and immune dysregulation (Kim *et al*., 2021). These effects are often mediated through oxidative and neuroendocrine pathways. However, the influence of RF-EMR on food intake and related physiological parameters has not been extensively explored. Given that both chronic stress and RF-EMR exposure can induce biochemical and molecular changes, it is important to characterize these responses in order to identify potential mitigating strategies. Although the antioxidant properties of vitamin E are well established, its modulatory role under chronic RF-EMR exposure conditions remains incompletely understood.

In the present study, rats exposed to RF-EMR exhibited a trend toward reduced food intake and body-weight gain compared with control animals; however, these changes did not reach statistical significance. These findings suggest that under the present exposure conditions, RF-EMR did not produce overt alterations in food intake or growth parameters. The absence of statistically significant changes indicates that food intake should be interpreted as a supportive physiological parameter rather than a primary behavioral endpoint. It is possible that longer exposure duration, higher exposure intensity, or larger sample sizes may be required to detect subtle functional changes, warranting further investigation.

Despite the lack of significant changes in food intake, RF-EMR exposure resulted in significant biochemical alterations. Chronic RF-EMR exposure significantly elevated serum corticosterone levels, indicating activation of the stress response. Similar observations have been reported in previous studies, supporting the role of RF-EMR as a physiological stressor (Pall, 2016). However, inconsistencies exist in the literature, with some reports demonstrating no significant changes in corticosterone or cortisol levels following RF-EMR exposure (Ohtani *et al*., 2015). Such discrepancies may be attributed to differences in exposure parameters, experimental design, species variation, or duration of exposure.

Oxidative stress is one of the most consistently reported biological consequences of RF-EMR exposure. Multiple

studies have documented increased MDA levels following RF-EMR exposure, reflecting enhanced lipid peroxidation and oxidative damage (Ilhan *et al*., 2004; Oktem *et al*., 2005). RF-EMR is known to promote reactive oxygen species generation, leading to oxidative damage of cellular components and disruption of membrane integrity (Valko *et al*., 2006). Consistent with these reports, the present study demonstrated a significant elevation in serum MDA levels following chronic RF-EMR exposure, supporting the involvement of oxidative stress mechanisms.

The observed increase in serum NSE further suggests the presence of neuronal stress associated with RF-EMR exposure. NSE is widely used as a marker of neuronal stress or injury; however, when measured in serum, it is not specific to the brain and may originate from peripheral sources. Therefore, the elevated NSE levels observed in the present study should be interpreted cautiously as indicative of systemic neuronal stress rather than definitive central neuronal injury. The lack of region-specific brain tissue estimation of NSE represents an important limitation of the study.

GFAP immunohistochemical analysis of the hippocampal CA3 region did not demonstrate significant alterations in astrocytic expression. This finding indicates the absence of overt gliosis or structural astrocytic pathology under the present experimental conditions. Together with the non-significant changes in food intake, these results suggest that RF-EMR-induced oxidative and neuroendocrine stress may precede observable functional or structural alterations in the central nervous system.

Vitamin E supplementation significantly attenuated RF-EMR-induced elevations in corticosterone and MDA levels, supporting its role in mitigating oxidative and stress-related biochemical changes. The antioxidant and cytoprotective properties of vitamin E have been well documented in RF-EMR and stress-related models. Jelodar *et al*. (2016) reported restoration of antioxidant enzyme activity and reduction of lipid peroxidation following vitamin E supplementation in RF-EMR-exposed rats. Similarly, Sahin *et al*. (2001) demonstrated that vitamin E reduces stress-induced corticosteroid elevation and tissue injury in animal models. In the present study, vitamin E did not significantly influence food intake or GFAP expression but effectively modulated biochemical stress markers, indicating its protective role at the molecular level.

The present study has several limitations. RF-EMR exposure was generated using a mobile phone in answer mode, and direct estimation of the SAR was not feasible, limiting precise dosimetric characterization and comparison with standardized exposure systems. The selected Vitamin E dose was based on prior experimental literature; however, tissue or plasma Vitamin E levels were not measured to confirm bioavailability or dose–response relationships. In addition, GFAP expression was assessed only in the hippocampal CA3 region, and analysis of additional brain regions would provide a more comprehensive evaluation of region-specific central nervous system responses.

While the neuroprotective properties of Vitamin E are well established, the present study contributes by examining its modulatory influence under chronic RF-EMR exposure conditions and by integrating oxidative stress, neuroendocrine stress, and astrocytic response assessments within a single experimental framework. Nevertheless, the lack of brain tissue-specific biochemical analyses and the absence of overt functional or structural CNS alterations limit definitive conclusions regarding central neurobiological effects. Future studies incorporating standardized RF-EMR exposure systems, region-specific biochemical and inflammatory markers, and extended exposure paradigms are warranted to further elucidate RF-EMR-induced CNS effects and the mechanistic role of antioxidant interventions.

In conclusion, the present study demonstrates that chronic exposure to RF-EMR emitted from mobile phones is associated with significant oxidative and neuroendocrine stress in rodents, as evidenced by elevated serum MDA and corticosterone levels. An increase in serum NSE was also observed; however, given that NSE was measured in serum, this finding is interpreted as indicative of systemic neuronal stress rather than definitive central neuronal injury. Changes in food intake and body weight did not reach statistical significance, indicating the absence of overt functional disturbances under the present experimental conditions. Collectively, these findings suggest that RF-EMR exposure may elicit early biochemical stress responses prior to the development of measurable behavioral or structural alterations.

GFAP expression in the hippocampal CA3 region did not show significant changes, indicating the absence of overt astrocytic activation or gliosis. This finding suggests that the observed biochemical alterations were not accompanied by detectable astroglial structural responses within the exposure duration employed in this study. The lack of significant GFAP changes highlights the need for cautious interpretation of central nervous system involvement.

Vitamin E supplementation significantly attenuated RF-EMR–induced increases in oxidative and stress-related biochemical markers, consistent with its established antioxidant and cytoprotective properties. While these effects do not provide novel mechanistic insight, they support the potential of antioxidant intervention in mitigating RF-EMR–associated biological stress. Future studies incorporating extended exposure durations, region-specific brain biochemical analyses, and detailed histopathological evaluation are required to further elucidate RF-EMR–induced central nervous system effects.

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