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THE POSSIBLE AMELIORATIVE EFFECT OF VITAMIN E AGAINST TOXICITY INDUCED BY MONO SODIUM GLUTAMATE ON THE DORSAL SURFACE OF THE TONGUE IN ALBINO RATS

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Abstract

Introduction: Although monosodium glutamate (MSG) is commonly employed in the food industry, its possible toxic effects remain a concern. Vitamin E (Vit E), a strong lipid-soluble antioxidant, offers potential protection against oxidative damage caused by MSG exposure. **Aim:** To evaluate the protective effect of vitamin E against MSG-induced structural and molecular alterations in the dorsal surface of the tongue in albino rats. **Materials and Methods:** Thirty adult male albino rats were randomly assigned into three groups (n=10): Group 1 (Control) received saline for 40 days; Group 2 (MSG) received MSG (60 mg/kg/day) for 30 days; Group 3 (Vit E+MSG) received vitamin E (200 mg/kg/day) for 10 days prior to, and during, MSG administration. Tongue specimens were examined histologically, immunohistochemically for inducible nitric oxide synthase (iNOS), and by qRT-PCR for heme oxygenase-1 (HO-1) expression. **Results:** In MSG treated group, there was marked degeneration of filiform, fungiform, and circumvallate papillae with inflammatory infilteration. There was significant increase in iNOS immune reactivity ($p<0.0001$), and significant decline in HO-1 gene expression ($p<0.0001$) compared to control group. Vitamin E co-administrated group showed marked improvement in the histological damage, reducing iNOS immune reactivity by ~35% vs. MSG alone ($p<0.05$), and increasing HO-1 gene expression by ~2.4-fold compared to the MSG group ($p<0.05$). **Conclusion:** Vitamin E reinforcing its ameliorative effect against toxicity induced by MSG on the dorsal surface of the tongue in albino rats.

Keywords: Monosodium Glutamate, Vitamin E, Tongue, iNOS, HO-1 gene.

1. INTRODUCTION

Mono sodium glutamate (MSG) is the most well-known food additive that is used as a flavor enhancer. It was first extracted from the seaweed Laminaria Japonica and commonly sold under the name Ajinomoto.

The consumption of MSG has increased worldwide in the last 30 years. The savory taste of MSG increases the attraction towards junk food such as hamburgers, salad dressings and fried chicken, in which MSG is a main constituent. Moreover, MSG has been used in vaccines to increase its shelf life. [1]

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Despite being classified as safe" (GRAS) by the U.S. Food and Drug Administration (FDA), chronic or excessive consumption of MSG may cause symptoms like weakness, flushing, dizziness, sweating and headache. It may lead to further complications including asthma, urticaria, neuropathy, and abdominal discomfort. Furthermore, MSG may induce damage to cardiac tissue architecture, alteration in cardiac rhythm, liver toxicity and alteration in the rate of cell growth. [2]

MSG induces detrimental toxicity effects as reactive oxygen species (ROS) that disrupt peroxidation of mitochondrial lipid and cellular metabolism. Moreover, it promotes the intrinsic apoptotic pathway which stimulates cytochrome c release, caspase cascade activation leading to cellular death. [3]

Several studies have proved the deleterious toxic effects of MSG on different body tissues including thyroid gland [4], liver [5],placenta [6], kidneys [7]. Moreover, the impact of MSG on oral tissues in experimental rats reveals the damaging effects on the gingiva [8], buccal mucosa [9], and submandibular salivary glands. [10] To counteract the effect of MSG, natural and chemical antioxidant agents were manipulated to reduce the harmful effects of oxidative stress. [11]

Finding antioxidants of natural food sources has an obvious interest to retard the progress of many chronic diseases and inhibit the oxidation chain reaction. [12] Vitamin E is a widely applied plant derived, lipophilic, bio-available vitamin. It has high affinity to scavenge against the free radicals involved in lipid peroxidation.

It has protective ability by either reducing or preventing endotoxin induced oxidative stress in rat tissues suggests its antioxidant activity. [13] Donation of hydrogen atom to the free radicals enhances the antioxidant activity of vitamin E that leads to formation of a relatively stable substrate and breaks the propagation of radicals' chain. [14]

According to the previous studies and up to our knowledge of the toxic effects of MSG on different organs of body and the possible mitigating effect of vitamin E as a potent antioxidant, the aim of this study was to investigate the possible ameliorative effect of vitamin E against toxicity induced by MSG on the dorsal surface of the tongue in albino rats**.**

2. MATERIALS AND METHODS

2.1 Materials

MSG known commercially as Chinese salt with chemical formula ($C_5H_9NO_4$) in the form of crystal powder. It was purchased from local market licensed by manufacturer company Ajinomoto Co. INC. Tokyo, Japan. Vitamin E in the form of gelatin capsules at concentration 400 mg. It was purchased from a local pharmacy. The gelatin capsules were emptied and diluted in corn oil.

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2.2 Methods:

2.2.1 Sample size calculation

The sample size for this study was 30 samples. It was calculated according to Charan & Biswas, 2013 [15] who used the following equation: $N = \frac{(Z_\alpha)^2 * (S)^2}{(d)^2}$

N = Total sample size, $\mathbb{Z}_\alpha$= Standard normal variation and its equal 1.96 at P < 0.05, SD = Standard deviation of the variable, d = Absolute error or precision

2.2.2 Study design:

After the approval of the Research Ethics Committee, the present research was carried out on 30 adult male albino rats with an average of 150-200 gm body. Rats were acclimated 7 days before the experimentation.

The rat cages were kept in well-ventilated animal house of the Faculty of Dentistry, Suez Canal University under the supervision of specialized veterinarians since their housing till they got rid of rats' bodies. They were kept at a temperature of 27-30°C, fed with dry rat pellets and allowed drinking water.

2.2.3 Animal Grouping:

Thirty rats were randomly and equally divided into 3 groups (n=10) as follows: Group 1 served as negative control and received distilled water daily via oral gavage for 40 days. [16] Group 2 served as positive control and received 60 g/kg MSG daily for 30 days via oral gavage. [4]

Group 3 served as prophylactic group and received 200 mg/kg vitamin E for 10 days then combination of MSG and vitamin E daily for 30 days via oral gavage. [16]

After the experiment period (40 days), rats were euthanized by extra dose of anesthesia. After euthanasia, the tongue specimens were dissected. The dead hamsters were disposed of by burning in Animal Aching Unit of Medicine.

2.2.4 Histopathological Analysis

After separating the specimen, half of them were fixed in 10% buffered formalin then embedded in paraffin sections of 5μ thickness to be examined by light microscopy and photographed in Oral Biology lab.

2.2.5 Immunohistochemical Analysis

Another 5 μ sections were cut and mounted on positively charged glass slides. Inducible nitric oxide synthase (iNOS) 'rabbit polyclonal antibody' was used according to manufacturer's instructions for ROS identification with brown cytoplasmic and nuclear expression. Tissue sections were examined and analyzed at magnification 400X. The digital image acquisition was used for measuring the screen area. The optical density was quantitatively measured using an image analysis system (ImageJ, Fiji distribution) in the Pathology Laboratory.

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2.2.6 Molecular Analysis

The fresh remaining half tongue specimens were kept at –80 °C for real-time quantitative polymerase chain reaction (qRT-PCR) analysis for detection of Heme Oxygenase-1 (HO-1) gene. Tissue homogenization was performed using the Tissue Raptor II in the presence of lysis buffer, then centrifuged. Purification of total extracted RNA through using the RNeasy® Mini Kit (Cat. No. 74104), followed by synthesis of complementary DNA through using Reverse Transcription Kit, cat. No: 205310.

Amplification of the HO-1 gene by Primer Assay, SYBR Green PCR Kit and β-actin as the housekeeping gene were used (Cat. No. 249900, 204141 and 249900 respectively). All kits were purchased from Quantitech (*Qiagen, Hilden, Germany*). Consequently, qRT-PCR includes a three-step cycling program of denaturation (94 °C / 15 sec), annealing (55 °C / 30 sec), and extension (70 °C / 30 sec), repeated for 40 cycles.

2.2.7 Statistical analysis

The numerical obtained data was collected, tabulated and analyzed for normality by checking the distribution of data, calculating the mean, evaluating histograms and normality curves by using Social Science software computer program version 23 (SPSS, Inc., Chicago, IL, USA).

One-way Analysis of variance (ANOVA) and Tukey tests were used for comparing data regarding the area percentage of positive reaction to iNOS. P-value less than 0.05 was considered statistically significant.

3. RESULTS

3.1 Histopathological Results:

Examination of the hematoxylin and eosin-stained sections of the dorsal surface of the tongue in different groups showed variable results.

Group 1 (negative control) revealed normal mucous membrane of the tongue's dorsal surface. The filiform papillae appeared as well organized conical shaped and covered with keratinized stratified squamous epithelium.

The fungiform papillae had rounded mushroom shaped bulges with flattened tops with a single taste bud located centrally at its top. The circumvallate papillae (CVP) appeared as a single papilla surrounded by a trough and covered by non-keratinized stratified squamous epithelium.

Numerous taste buds appeared as normally barrel shaped with prominent nuclei, rested on basement membrane. Their taste pore opened into the trough. The lingual salivary glands preserved the normal architecture of serous and mucous acini with. The underlying connective tissue was normal with no signs of inflammatory infiltrate (Figure 1 A: E).

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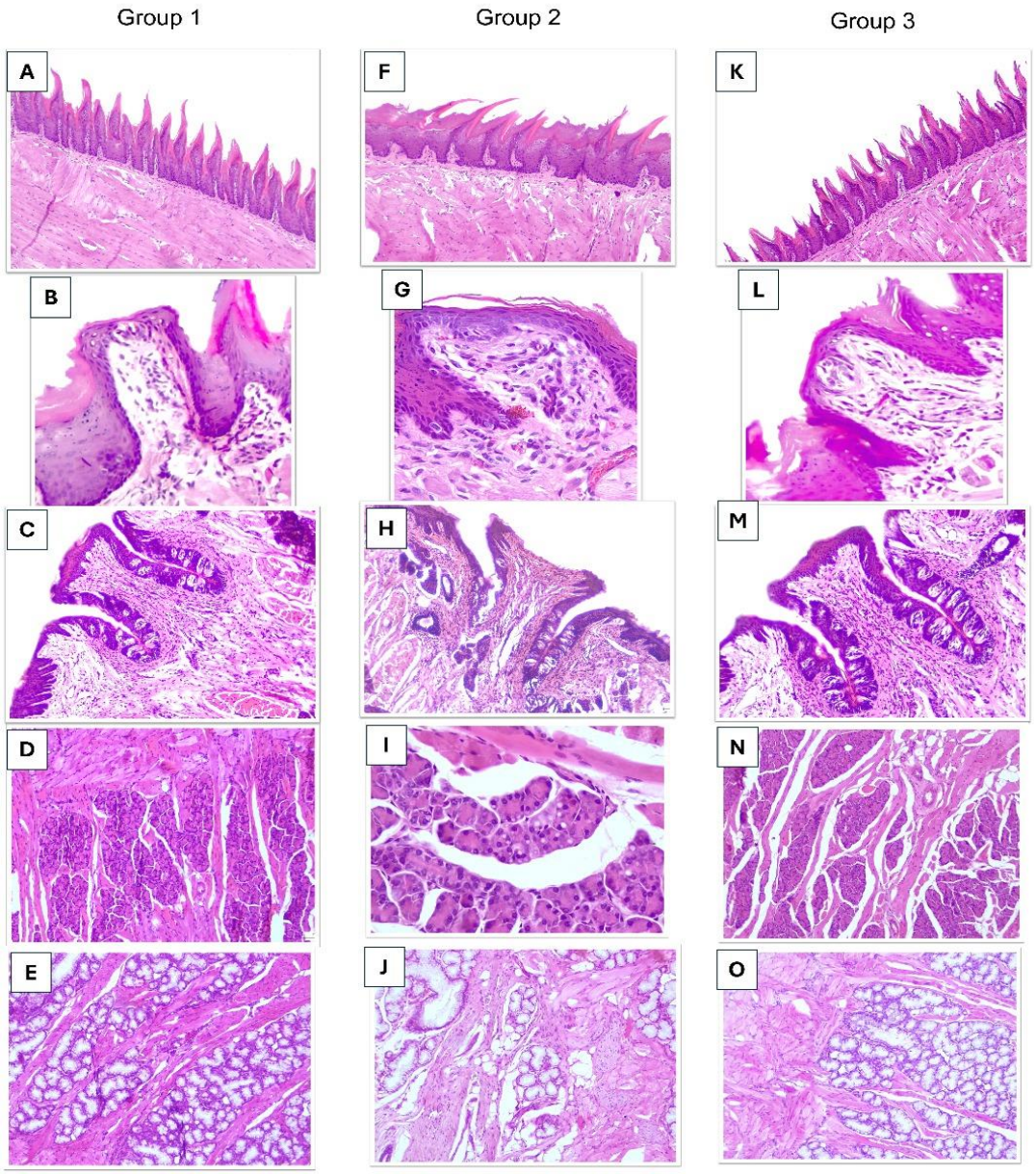

Figure 1: Hematoxylin & Eosin photomicrographs of tongue's dorsal surface in different groups. Group 1 showing normal appearances of (A) filiform, (B) fungiform, (C) circumvallate papilla and (D&E) salivary gland architecture. Group 2 showing severe degenerative changes in (F) filiform, (G) fungiform, (H) circumvallate papilla and (I&J) salivary gland architecture. Group 3 showing marked improvement within (K) filiform, (L) fungiform, (M) circumvallate papilla and (N&O) salivary gland architecture. (H&E. orig. mag. A, F, K 100X – C: E, H: J, M: O 200X - B, G, L 400X)

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Group 2 (positive control) showed severe atrophic and degenerative changes within the mucus membrane of the dorsal surface. The filiform papillae revealed marked non uniform orientation, stunted and lost their characteristic conical shape. Keratin separation from the underlying epithelium as well as areas of hyper keratinization were noticed in some areas. Vacuolated cytoplasm of the basal and supra-basal epithelial cells was seen. The fungiform papillae and its taste buds were disfigured with an ill-defined taste pore.

The CVP showed deformity in the general outline. Their taste buds revealed undetected taste pore. The epithelium at the base of the trough showed loss of its normal orientation. The lingual salivary glands revealed marked atrophic changes in their acinar cells with loss of their normal architecture with cystic degeneration and widening of CT septa. Dissociation of collagen fibers and dilated congested blood vessels within the underling lamina propria with inflammatory cell infiltration were noticed (Figure 1 F: J).

Group 3 (prophylactic group) showed marked improvement within the dorsal surface of the tongue that was covered by normal thickness of keratinized stratified squamous epithelium with minor areas of keratin separation. The filiform papillae nearly restored their pattern with tapering ends. The fungiform papillae appeared almost normal, and their taste buds revealed minimal signs of deformation or degeneration.

The CVP retained their normal outline and was surrounded by a regular narrow trough. The number and arrangement of taste buds on the sides of the trough appeared normal. Atrophy of few taste buds and minimal degeneration of some taste cells were also noticed. The acinar architecture of the lingual salivary glands showed marked improvement with no cytoplasmic vacuolations and minimal cystic degeneration. The underlying connective tissue revealed mild inflammation with small blood vessels. (Figure 1 K: O).

3.2 Immunohistochemical Results

Examination of the iNOS immunohistochemical expression in stained sections of the dorsal surface of the tongue in different groups showed variable results. Group 1 showed mild cytoplasmic immunoreactivity to iNOS (7.893 ± 0.4562) along the dorsal surface of the tongue, which slightly limited to basal and parabasal cell layer (Figure 2 A).

Group 2 revealed strong cytoplasmic immunoreactivity to iNOS (68.06 ± 4.172) all over the epithelial thickness compared to the control group (Figure 2 B). Group 3 revealed marked decrease in the expression of iNOS (14.34 ± 2.010) along the epithelial layers with moderate expression in the prickle cell layer (Figure 2 C). Tukey`s post hoc pairwise comparison confirmed ANOVA results as shown in table 1 & 2. Group 1 exhibited the lowest mean area percentage ($p < 0.05$).

Group 2 showed a significantly higher mean area percentage ($p < 0.05$) compared to other groups. Groups 3 showed moderate mean area percentages, which is significantly higher than Group 1 ($p < 0.05$) but lower than Group 2 (Figure 2 D).

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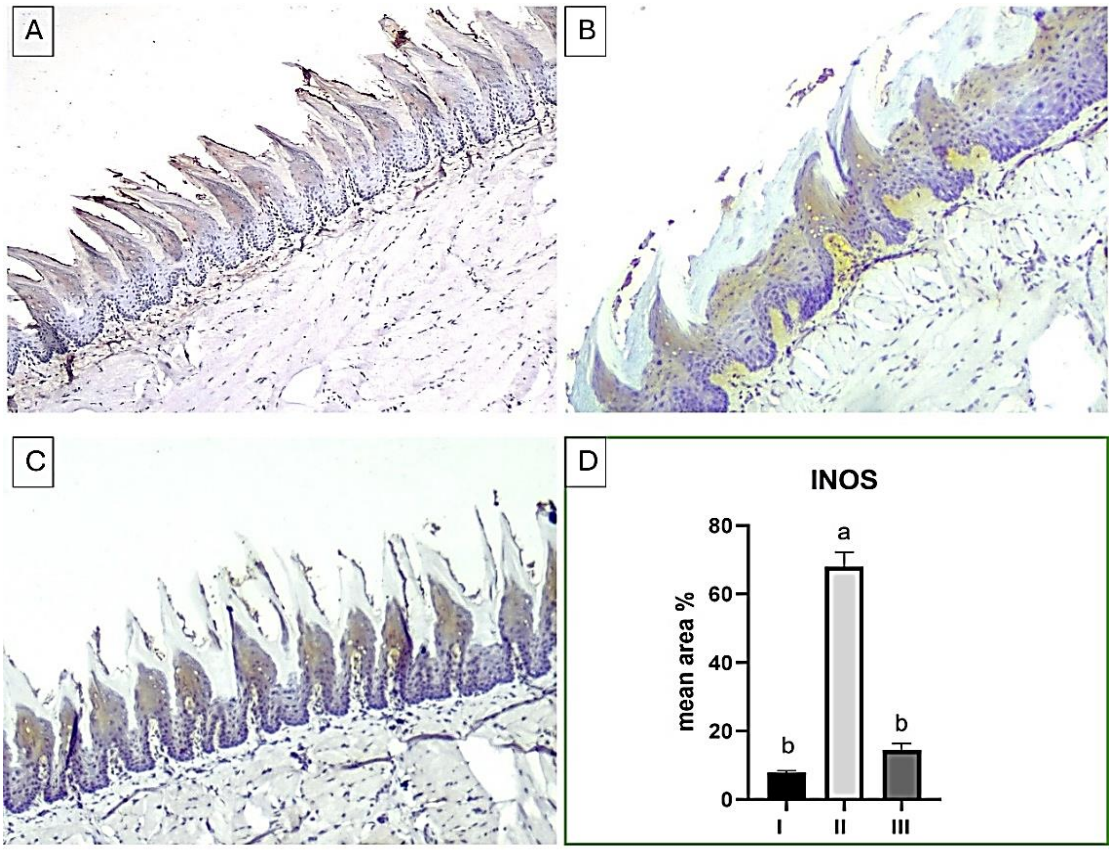

Figure 2: Photomicrographs showing iNOS immunohistochemical expression of tongue's dorsal surface in different groups. (A) Group 1 showing mild cytoplasmic immunoreactivity. (B) Group 2 showing strong cytoplasmic immunoreactivity all over the epithelial thickness. (C) Group 3 showing moderate expression within the prickle cell layer. (D) Histogram showing the mean area % of iNOS immunohistochemical expression. (IHC. Orig. mag. A&C 100X – B 200X)

Table 1: Descriptive statistics analysis via ANOVA test

	Group 1		Group 2		Group 3	
	iNOS	**HO-1**	**iNOS**	**HO-1**	**iNOS**	**HO-1**
Mean	7.893	0.9890	68.06	0.3100	14.34	0.8733
Minimum	7.400	0.8470	63.60	0.2000	12.33	0.8100
Maximum	8.300	1.100	71.87	0.4000	16.35	0.9400
Range	0.9000	0.2530	8.270	0.2000	4.020	0.1300
Std. Deviation	0.4562	0.1293	4.172	0.1015	2.010	0.06506
Std. Error of Mean	0.2634	0.07466	2.409	0.05859	1.160	0.03756
Lower 95% CI of mean	6.760	0.6678	57.69	0.05789	9.350	0.7117
Upper 95% CI of mean	9.027	1.310	78.42	0.5621	19.34	1.035

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3.2 Molecular Results

Variable results were noticed during the detection HO-1 gene in freshly specimens within different groups (Table 1 - 2). The highest mean relative fold change in HO-1 gene expression was recorded in group 1 (0.989 ± 0.253) while the lowest mean was observed in group 2 (0.310 ± 0.1015). A statistically significant difference was detected between the studied groups ($p < 0.05$). On comparing group 1 to group 2, there was a highly significant increase in the mean HO-1 gene expression ($p < 0.0001$). Moreover, on comparing group 3 (0.8733 ± 0.06506) to group 1 (0.989 ± 0.253), there was a significant decrease in mean HO-1 expression ($p < 0.05$) but a significant increase when compared to group 2 ($p < 0.05$).

Table 2: Tukey`s post hoc pairwise comparison

	Tukey's multiple comparisons test	Mean Diff.	95.00% CI of diff.	Summary	Adjusted P Value
iNOS	Group 1 vs. 2	-60.16	-65.83 to -54.49	****	<0.0001
	Group 1 vs. 3	-6.450	-12.12 to -0.7787	*	0.0230
	Group 2 vs. 3	53.71	48.04 to 59.38	****	<0.0001
HO-1	Group 1 vs. 2	0.6790	0.4125 to 0.9455	****	<0.0001
	Group 1 vs. 3	0.1157	-0.1508 to 0.3822	ns	0.6946
	Group 2 vs. 3	-0.5633	-0.8298 to -0.2968	***	0.0001

4. DISCUSSION

Mono Sodium Glutamate (MSG) can improve the deliciousness of meals and increase appetite. It is regularly consumed despite concerns regarding its safety. The intake of MSG can cause oxidative stress and consequently the production of ROS. This occurs when there is an imbalance between free radicals and body antioxidant defense. [17] As antioxidants scavenge harmful free radicals. Vitamin E was chosen in this study as ameliorating substances against MSG toxicity due to their antioxidant properties. [18]

Although MSG is widely consumed, its effect on the dorsal surface of the tongue has not been thoroughly investigated. The tongue is often regarded as a reflection of general health, with particular emphasis on the filiform papillae. For this reason, it was chosen for this study. Due to high metabolic activity of the filiform papillae, any nutritional disturbance made them more vulnerable to atrophy. Moreover, histological alterations in lingual tissues can express the adverse effects of different pathological conditions and chemical-induced toxicity. [19]

In the present study, the histological examination of the dorsal surface of the tongue in group 2 (MSG group) showed marked atrophy and degeneration of the surface papillae filiform, fungiform and circumvallate. These results were analogous with the results of Fathy et al., 2019 [9] who studied the effect of MSG on buccal mucosa of albino rats and found that it caused severe histological changes including shortening of epithelial ridges and destruction of superficial mucosa.

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The filiform papillae showed atrophy as well as areas of hyper keratinization. This may be a result of a protective response of the body to limit the penetration of harmful additives into the underlying lamina propria. Similar results were reported by Ragab et al. (2018), who demonstrated that MSG induced degenerative changes in renal tubules characterized by cytoplasmic vacuolations. [20]

Furthermore, the disfigurement of the CVP and fungiform papillae due to dissociation of MSG into glutamate and toxic ammonium ions lead to subsequently cellular damage through the release of mitochondrial apoptotic factors. This came in agreement with Atef et al., 2019 [21] who reported that MSG is considered a hepatotoxic agent and has oxidant effects through increased the generation of ROS and lipid peroxidation.

The degenerated taste buds and taste cells might be attributed to excess extracellular glutamate that had a direct impact on glutamate receptors overstimulation and intracellular calcium overload. This was demonstrated by Farhat et al., 2021 **Error! Bookmark not defined.** who studied the cytotoxic effect of glutamate on neural cells.

Abdelhamid et al., 2023 [22] stated that MSG enhanced degenerative changes, cellular necrosis and fibrous degeneration. These findings explained the fibrous dissociation within the lamina propria below different types of papillae. On contrary to our findings, Abd-Elkareem et al. (2022) [23] noticed that excessive collagen fibers were prominent in kidney rats treated with MSG.

Fibrosis might be attributed to release of multiple fibro-genic factors, such as lymphokines by the action of MSG that activate fibroblast proliferation and thick collagen fibers deposition within renal tissues. This discrepancy might be explained depending on the target tissue under investigation that affects different cellular responses.

The predominance of dilated and congested blood vessels might be due to inhibition of prostaglandins synthesis which is responsible for the blood flow regulation. This finding was in harmony with El-Imam et al., 2019 [10] who studied the effect of MSG on submandibular salivary glands and explained the blood vessels congestion to the same cause.

Vitamin E is a naturally occurring antioxidant that protects against oxidative damage. It acts as a free radical scavenger, suppresses the production of new free radicals, and delays the onset of detrimental side effects associated with oxidative injury. [24] Abdel-Daim and Abdeen (2018), [25] reported that vitamin E produced stable non-radical products that inhibiting lipid peroxidation through its integration into the cell membranes lipid bilayer. As, its hydroxyl group served as a hydrogen donor to hydroxyl radicals. Similar observations were reported by Eid et al. (2019) [26] who suggested that MSG (4 g/kg) with vitamin E (300 mg/kg) had a protective role in hepatocytes against MSG-induced ultrastructural alterations.

Histological assessment of group 3 revealed marked improvement in combination with vitamin E. These findings were in harmony with Hamza and Al-Harbi (2014) [16] who found that vitamin E substantially protected the testicular tissue against the effect of MSG

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at a comparable dose. Furthermore, it reduced the absorption of MSG in the gastrointestinal tract and preserved the cell membrane structural integrity through preventing oxidative damage to membrane lipids.

Inducible nitric oxide synthase (iNOS) is a biomarker of inflammatory activity and oxidative damage that is responsible for increasing the production of nitric oxide (NO). [27] The mean area of iNOS immune reactivity in group 2 administered with MSG showed a highly significant increase when compared to group 1. This was explained by Zhen et al., 2008 [28] who noted that iNOS expression could be triggered by oxidative stress leading to nitric oxide (NO) generation resulting in the formation of cytotoxic compounds causing tissue dysfunction. The mean area of iNOS immune reactivity of group 3 was significantly decreased in comparison to group 2 supporting the fact that vitamin E is a powerful antioxidant that had the potential to ameliorate the inflammatory effects after ROS production. This agreed with Bas et al., 2021 [29] who demonstrated the antioxidant and anti-inflammatory effect of vitamin E treatment against periodontitis through reducing the expression of iNOS.

Heme oxygenase (HO-1) expression is upregulated under oxidative stress and hypoxic conditions. However, downregulation of HO-1 gene expression induced by MSG could occur due to several factors as cellular damage beyond the adaptive threshold or impairment of stress responsive genes like carbon monoxide and bilirubin. It could downregulate HO-1 expression through a negative feedback mechanism and prevent excessive heme degradation. In the present study, the expression of HO-1 gene was down regulated upon exposure to MSG in group 2. Abu-Elfotuh et al., 2022 [30] evidenced that the decline in HO-1 gene expression because of oxidative stress induced by MSG toxicity in the form of cellular injury caused by depletion of glutathione. The qRT-PCR findings stated that the expression of HO-1 gene was upregulated in group 3 that received vitamin E concomitantly with MSG due to the antioxidant effect of vitamin E against MSG induced oxidative stresses.

Finally, regarding all the previous studies, histological, immunohistochemical and molecular assessment confirmed that vitamin E had a powerful ameliorating effect on MSG induced toxicity on the dorsal surface of the tongue.

5. CONCLUSIONS

The current investigation emphasized the protective role of vitamin E against MSG-induced toxicity on the dorsal surface of the tongue. Owing to its strong antioxidant capacity, natural origin, and favorable safety profile, vitamin E could be considered a hopeful supplement to counteract oxidative stress and suppress apoptosis, thereby contributing to better preservation of tissue architecture.

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Conflict of Interest: None declared

Conflict of interest statement: The authors declare that there is no conflict of interest.

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