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Effect of Monosodium Glutamate on Some Vitamins and the Protective role of Vitamin B12 in Adult Female Rats

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ABSTRACT: The aim of study: the current study investigated the effect of MSG and protective role of Vitamin B 12 on the Vitamin C and E concentration level in Female Albino Rats 50, because it is considered a physiological antioxidant**.** The study included Female Albino Rats 50 were divided into five equal groups n=10, each group was further divided into two groups [A and B, n=5] and treated for 30 and 60 days, respectively. The 1st group received distilled water orally by gavage, the 2nd and 3rd group received MSG in a dose of 20, and 40 mg/kg body weight orally by gavage, respectively, the 4th and 5th groups received MSG and vitamin B12 in a dose of 20 or 40 mg MSG/kg in addition to 0.2 m vitamin B12/kg, respectively. The findings found the Group 5 showed increased in Vitamin C concentration after 30 and 60 days (1.246±0.319, 1.335±0.341) respectively with significant differences with other groups same results reported in Vitamin E that showed Vitamin E concentration after 30 and 60 day(1.136±0.319, 1.109±0.019) respectively with significant differences at ($p \leq 0.05$) with other groups.the current study revealed Vitamin B12 had a protective role.

Key word: Monosodium glutamate, vitamin B12, Vitamin E, Vitamin C

1. INTRODUCTION

A common flavor enhancer, monosodium glutamate (MSG) is made from L-glutamic acid, an amino acid that may be found naturally in many different food items [1]. Umami, a flavor that is unique to MSG, was once thought to be prominent in Asian civilizations before spreading to Western ones much later [2]. Apart from its fundamental uniqueness, the umami flavor may intensify flavors overall and make food more palatable. The content of the umami molecule and the food matrix are the two most significant elements influencing this response [3]. A wide range of natural sources as well as food products such processed meat, canned vegetables, soups, sauces, dry bouillon cubes, and salty flavored snacks include them as additions [4]. Additionally, IMP is employed as a flavor

enhancer to bring out the umami flavor of MSG [5]. While several preclinical and clinical research have questioned the safety of MSG use, particularly after chronic exposure, food safety regulatory organizations still believe that MSG consumption is safe. The fact that endogenous glutamate is involved in both physiological and pathological processes is probably what fuels the debate. In addition to being an important substrate for energy production in enterocytes, glutamate also serves as an excitatory neurotransmitter in the central nervous system (CNS) and is a precursor of important metabolites like glutathione (GSH, an oxidative stress modulator) and N-acetylglutamate (a metabolic regulator) [6]. Due to their antioxidant properties, vitamins can help reduce oxidative stress [10]. Antioxidants are essential for overall health and the first line of defense against damage caused by free radicals [11]. Vitamin B12: Also referred to as

cobalamin, this vitamin is crucial for the metabolism of cells, particularly for DNA synthesis, methylation, and mitochondrial metabolism [7]. One of the most significant antioxidants found in a regular diet is vitamin E, which offers protection against a number of illnesses in humans [8]. Given that vitamins and MSG may be found in food, it is important to assess how they interact to determine whether vitamins may worsen or lessen the negative effects of MSG. When vitamin C (500 mg/kg) was given in conjunction with MSG for 45 days, rats' parenchymal architecture was protected against MSG by a decrease in cellular proliferation, as shown by a decrease in the expression of ki-67 and a mutation in a tumor suppressor gene [9].

2. MATERIAL AND METHODS

2- 1 Experimental Animals

50 female Swiss albino rats weighing between 200 and 250 grams and was three months old were used in this investigation. They were acquired from Al-Mustansiriya University and the Iraqi Center for Cancer Research and Medical Genetics Research. The animals were housed at Al-Mustafaniriya University's Research Center's Animal House. Sawdust blanketed the floor and they were housed in metal cages with metal mesh coverings. The animals were divided into 5 groups after being cleaned by replacing the sawdust in their cages once a week and sterilizing them with 70% alcohol concentration, 25 °C temperature, ventilation, and natural lighting.

1. **G1 =control:** Involved 10 adult female rats, animals in this group administered distilled water and served as control.
2. **G2 =monosodium glutamate: -**Involved 10 adult female rats, which were subdivide in two main sub groups; each group contained (5 animals), each group was given monosodium glutamate (20mg/kg B.W) orally daily by gavage for thirty and sixty days.
3. **G3 =monosodium glutamate: -**Involved 10 adult female rats. Which were subdivided in two main subgroups, each group contained (5 animals), each group was given monosodium glutamate (40mg/kg B.W) orally daily by gavage for thirty and sixty days.
4. **G4 =monosodium glutamate + B12: -**Involved 10 adult female rats which were subdivided in two main subgroups, each group contained [5animals], each group was given monosodium glutamate (20mg/kg B.W), then intubated orally B12 (0.2 mg/kg B.W) orally daily by gavage for thirty and sixty days.
5. **G5 =monosodium glutamate + B12: -**Involved 10 adult female rats which were subdivided in two main subgroups, each group contained (5 animals), each group was given monosodium glutamate (40mg/kg B.W), then intubated orally B12 (0.2 mg/kg B.W) orally daily by gavage thirty and sixty days.

All groups of 5 animals were sacrificed after 30 days of the experiment after blood collection by heart puncture and remain 5 animals sacrificed at 60 days of the experiment after blood collection by heart puncture [10]. The method of disposing of the animals after the end of the experiment was to put them in special bags and then place them in an environmentally friendly incinerator in Yarmouk Hospital. The animals were dealt with by euthanasia.

2-2 Preparation Mononsodium glutamate

Monosodium glutamate was prepared by dissolving the powder in distilled water to obtain the following 20mg and 40mg doses. The solution was kept in containers for used in experimental.

2-3 Preparation B12

Vitamin B12 has been prepared by grinding the tablets and dissolved it with sterile water and keeping them in an opaque glass container to be ready for use. B12 was prepared in doses of 0.2 mg/kg.

The doses were prepared using the following equation:
x / rat weight = dose given / 100

2-4 Measurement Vitamin C and E concertation level

All tests in the current study were determined by ELISA kits from Cusabio, USA.

Principles:

These assays use quantitative sandwich enzyme immune assay technology. A pre-selected antibody was plated onto a microplate. Standards and samples are pumped into wells and any antigen present is bound to the fixed antibody. After removing any unconjugated
substances, the biotin-conjugated antibody is placed Antigen-specific added to wells. After washing, conjugated avidin Horseradish peroxidase [HRP] is added to the wells. After washing to remove any unbound avidin enzyme reagent, a substrate solution was added to the wells and color developed in proportion to the amount of bound antigen in the initial step. Color development was stopped and color intensity was measured.

Procedure:

1- The prepared reagents samples and standards as instructed.
2- The standard or sample 100 µL added to each well then incubated 2 hr at 37°C.
3- The liquid was removed from each well, do not wash.
4- Biotin antibody (1x)100 µl was added to eachwell incubate for 1 h at 37 °C
5- pulled and washed 3times
6- HRP-avidin (1x]) 100 µl was added to each well Incubate 1 h at 37 °C.
7- Aspirate and wach 5 times
8- TMB substrate 90 µl added to each well Incubate 15-30 min at 37 °C.
9- the stop solution 50 µl added to each well Read done at 450 nm within 5 minutes.

2- 5 Statistical analysis:

To determine the influence of several variables (Groups and Treatment Time) on research parameters, the Statistical Analysis System- SAS (2012) program was utilized. In this study, the least significant difference –LSD test (ANOVA) was utilized to make a meaningful comparison between means [11].

3. RESULT AND DISCUSSION

vitamin (C) and vitamin (E) concentration

A. After 30 days

The results in table (1), (2) were showed that non -significant difference after 30 days in vitamin C, vitamin E concentration in groups G2, G3, G4, G5 respectively as compared with G1 .Also there was non- significant difference ($P \leq 0.05$) in group G4 that received MSG + B12 as compared with group G2 that received MSG and non- significant as compared with control G1.There are was non- significant difference in group G5 that received MSG +B12 as compared with group G3 that received MSG and non- significant as compared with control G1.

B. After 60 days

The results in tab (1, 2) were showed that non-significant difference after 60 daysin vitamin C, vitamin E concentration in groups G2, G3, G4, G5 respectively compared with G1. While there was non- significant difference in group G4 that received MSG + B12 as compared with group G2 that received MSG and non- significant as compared with control G1.Also there are was non- significant difference in group G5 that received MSG +B12 as compared with group G3 that received MSG and non- significant as compared with control G1.

C. Comparing between 30 days and 60 days.

Comparing the mean of vitamin C, vitamin E concentration between periods 30 days and 60 days, there was non-significant difference in vitamin concentration.

Table (1): Effect of monosodium glutamate and vitamin B12 on serum Vitamin C concentration in female rats

Groups	The first period 30 day	The second period 60 day
G1=control	0.906±0.253	0.859± 0.327

G2=MSG [20]mg	0.759 ±0.082	0.487±0.052
G3=MSG [40]mg	0.829±0.221	0.666±0.145
G4=MSG [20]mg+B12[0.2]	0.843±0.489	0.840±0.270
G5=MSG [40]mg+B12[0.2]	1.246±0.319 *	1.335±0.341 *
group [N=10] ,sub group [N=5]		

Table (2): Effect of monosodium glutamate and vitamin B12 on serum Vitamin E concentration in female rats

Groups	The first period 30 day	The second period 60 day
G1=control	0.757±0.2751	0.757±0.241
G2=MSG [20]mg	0.821 ±0.434	0.892±0.073
G3=MSG [40]mg	0.624±0.168	0.867±0.082
G4=MSG [20]mg+B12[0.2]	0.728±0.471	0.883±0.100
G5=MSG [40]mg+B12[0.2]	1.136±0.319*	1.109±0.019*
group [N=10] ,sub group [N=5]		

The findings found the Group 5 showed increased in Vitamin C concentration after 30 and 60 day (1.246±0.319, 1.335±0.341) respectively with significant differences with other groups same results reported in Vitamin E that showed Vitamin C concentration after 30 and 60 day (1.136±0.319 , 1.109±0.019) respectively with significant differences at ($p \leq 0.05$) with other groupsSince MSG may be present in the diet of humans according to various studies, it is important to assess how vitamins and MSG interact to determine whether vitamins might worsen or lessen the negative effects of MSG. When given in conjunction with MSG for 45 days, vitamin C (500 mg/kg) had a hepatoprotective impact on the parenchymal architecture of the liver in rats by lowering cellular proliferation, as shown by a mutation in a tumor suppressor gene and a decrease in ki-67 expression [9]. The primary mechanisms by which vitamin C inhibits the growth of cells are by its extracellular effect, induction of apoptosis, activation of cell cycle arrest, and suppression of the expression of genes related to protein synthesis [12]. Additionally, rats exposed to 0.6 mg/g bw of MSG had oxidative stress and hepatotoxicity due to the production of LPO, a reduction in GSH levels, and an increase in GST, SOD, and catalase activities in the liver. When vitamin E (0.2 mg/g bw) and MSG (0.6 mg/g bw) were given together, the LPO was improved, the GSH level rose, the hepatic SOD activities of GST and catalase were lowered, and the serum's ALT, AST, and GGT activities were decreased [13]. A considerable reduction in MDA levels and the number of atresia follicles, as well as an increase in FSH levels and the number of primary follicles, indicate that the treatment of combination vitamin C and E protected MSG-induced ovarian damage [14]. On the other hand, Group V's results showed an increase, which was ascribed to the vitamins' synergy, which has been linked to a number of metabolic processes. There are several ways in which these interactions occur, including the way that vitamins affect the metabolism of minerals, the way that minerals affect the metabolism of vitamins, and how these interactions work together to safeguard the body[12]. The body might benefit from several benefits due to the synergistic action of micronutrients. For example, it raises the bioavailability of nutrients; vitamin C and E work together to enhance absorption of each other, likewise, vitamin C promotes the body's absorption of iron[15]. In the context of micronutrients, the concept of dietary synergy heavily depends on bioavailability. Understanding nutrient bioavailability can aid in creating dietary suggestions for complementary foods that the broader public will find acceptable. Additionally, research is being done extensively to establish the evidence-based basis for the claims that the micronutrient synergy can help prevent and treat non-communicable chronic diseases like diabetes

mellitus, cardiovascular disease, obesity, and cancer. In this way, the body becomes trapped in a vicious cycle of malnourishment and infection. Certain meal combinations that work well together strengthen the immune system and support good health. Zinc and vitamins C and D are the micronutrients that have demonstrated the immune system's synergistic assistance [15] Unintentional gastrointestinal side effects can occur when a vitamin is taken as a supplement in a concentrated or bolus form [16] however Although the exact relationship between vitamins B12 and C is yet uncertain, Interactions may possibly happen even while taking large dosages of vitamins B12 and C together. Since B12 is not bonded to IF and is therefore more sensitive, passive diffusion accounts for the majority of B12 intake, particularly in situations when IF is scarce. Still, there aren't enough research to conclusively support this, and some even claim that vitamin C enhances the absorption of B12 [17].

Nonetheless, Zorn and Smith conducted a research [18] discovered Excessive consumption of certain vitamins seems to affect mercuric chloride methylation in vivo in guinea pigs. Methylmercury concentrations in the liver rose when megadoses of vitamin B12 were added and given either alone or in conjunction with folic acid. Furthermore, B12 administration significantly raised % methylmercury levels in the liver (B12 alone and B12/folic acid) and brain (B12/vitamin CIncluding large amounts of folic acid in the diet significantly raised the content of methyl mercury, especially in the tissues of the liver and hair. Vitamin C supplementation led to higher methylmercury levels and % methylmercury values with B12 in the muscle and brain tissue. This was especially true when combined with folic acid [muscle] or B12 [brain]. In terms of vitamin E's anti-inflammatory properties, it directly prevents NF-κB from being activated and translocated [17]. Since the inflammatory process may deplete antioxidant resources, including vitamin C, through the generation of ROS, its use in treating inflammation linked to illnesses has been investigated. Research suggests that vitamin C's ability to regulate nuclear factor kappa B's DNA binding activity accounts for both its antioxidant and anti-inflammatory qualities. By downregulating hepatic mRNA expression, vitamin C can lower the plasma levels of the inflammatory mediators TNF-α and IL-6[19]. Furthermore, vitamin C increased the antioxidants' concentration and activity [20].

4. CONCLUSION:

Monosodium glutamate at a dose of 20mg/kg, 40mg/kg has no effect on vitamin C and E concentrations. Vitamin B12 had a protective role, but it was not statistically significant**.**

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