Influence of Monosodium Glutamate on Radiation-Induced Biochemical Alterations in Male Albino Rats

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ABSTRACT

The consumption of foods and beverages containing additives has intensely increased over the past decades. Monosodium glutamate (MSG) is one of the main flavor enhancer that can be consumed in high concentrations. Also, human exposure to ionizing radiation (RAD) has become inevitable with its vast application in diagnosis and industry. Humans are frequently exposed to RAD and MSG from various food additives, therapeutic treatments and the environment. Although the use of additives and exposure to RAD in therapeutic treatments are believed to be relatively safe, their combined effects remain unclear. The present study proposed to investigate neurotoxic potentials of exposure to MSG and/or RAD on oxidative stress, neurotransmitters disturbance and metabolic disorders in the rat's brain tissue. MSG was supplemented daily by gavages to rats at a dose of 450mg/Kg bwt/day (equivalent to 5g/day human consumption) for 7 days pre- and 21 days post-exposure to whole body gamma rays at doses of 2Gy/week up to a total dose of 8Gy. Exposure to MSG and/or RAD -induced oxidative stress, neurotransmitters disturbance and metabolic disorders. Oxidative stress was manifested by a significant increase in lipid peroxidation product malondialdehyde (MDA) and decrease in the activity of antioxidant enzymes, superoxide dismutase, catalase and glutathione content. The administration of MSG daily during exposure to gamma radiation has potentiated oxidative stress regarding each single treatment. MSG-exposure induced a highly significant decrease of serotonin (P<0.01) and a slight non significant increase (P>0.05) of aspartic and glutamic acids levels while in RAD-group the decrease of serotonin and the increase of amino acids were very highly significant (P<0.001). MSG+RAD-exposure had potentiated the decrease of serotonin and produced an additive effect on the increase of neurotransmitters amino acids. MSG as well as RAD-exposure increased

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(P<0.05) glucose and insulin levels with no effect on insulin resistance and their co-administration produces an additive effect compared to each single treatment. Regarding lipid profile, MSG as well as RAD-exposure induced hyperlipidemia more noticeable in case of irradiation. Their co-administration had potentiated hyperlipidemia compared to each single treatment. It is concluded that exposure to MSG together with RAD increased oxidative stress and neurotransmitter alteration in the brain and the risk of metabolic syndrome. It is thus recommended to limit the intake of MSG when human are at risk of overexposure to ionizing radiation.

INTRODUCTION

There is a large body of evidence that exposure to nutritional and environmental challenges can markedly influence health outcomes. Monosodium L-glutamate (MSG), the sodium salt of glutamic acid is one of the main flavor enhancer used in various food and beverages and thus could be consumed in high concentrations. Estimated average daily dietary MSG exposures in developed countries were reported to be in the range of 0.3-1.0 g/day **(Geha *et al.,* 2000),** while in UK, exposure reaches approximately 0.6 g/day, with more than 2 g/day for extreme consumers **(Rhodes *et al.*, 1991).**

Since the first description of the 'Monosodium glutamate symptom complex', originally described in 1968 as the 'Chinese restaurant syndrome', a number of anecdotal reports and small clinical studies of variable quality have attributed a variety of symptoms to the dietary ingestion of MSG **(Williams and Woessner, 2009).** In 1995**,** MSG was classified by the U.S. Food and Drug Administration (FDA) as generally recognized as safe (GRAS), and has not been allocated an adequate daily intake (ADI) **(Walker, 1999).** Still, there have been much skepticism and controversy over its potential effects.

Conventional toxicity studies using dietary administration of MSG in several species did not re-

veal any specific toxic or carcinogenic effects nor were there any adverse outcomes in reproduction and teratology studies **(Walker and Lupien, 2000).** On the other hand, glutamic acid is an "excitotoxin" which stimulates brain cells far beyond their normal state triggering brain cell death **(Nakamura *et al.*, 2010 and Dutta and Trapp, 2011).**

Conversely, **Insawang *et al.* (2012)** considered a person with a daily consumption of MSG exceeding 5g at risk for metabolic disorders. Furthermore, MSG was reported to induce genotoxicity **(Farombi and Onyema, 2006)**, oxidative stress **(Onyema *et al.*, 2013)** dyslipidemia **(Kumar and Bhandari, 2013**), alteration in lipid and nitrogen metabolism **(Chen *et al.*, 2014),** and to have deleterious effects on spleen **(Ebaid and Tag, 2012)** and kidney architecture **(Dixit *et al.*, 2014).**

Radiation is one of the most widespread sources of environmental stress in living environment. Human exposure to ionizing radiation is growing rapidly view its vast application in new technology besides exposure to radiation from the natural background. Ionizing radiation is known to induce various physiological, and biochemical changes in humans and animals. Several molecular mechanisms of ionizing-radiation have been proposed, including cumulative damage by Reactive Oxygen Species (ROS), dislocation in replicative cells, genome instability, mutation or altered expression of specific enzymes and cell death. Oxidative stress refers to the cytotoxic consequence of oxygen free radicals: superoxide anions, hydroxyl radicals and hydrogen peroxide, which are generated as by-products of normal and abnormal metabolic processes induced by irradiation and may lead to DNA damage and mutagenesis, protein and carbohydrate oxidation and metabolic disorders (**Said *et al.*, 2012 and Saada *et al.*, 2010**).

Among consumers that might ingest MSG in excess, are people at high risk of radiation over exposure. The present study would provide more information about the combined effects of MSG with

radiation.

MATERIALS AND METHODS

Experimental Animals

Male albino rats *Rattus rattus* (10 ± 2 weeks old; 120 ± 10 g) purchased from the Egyptian Holding Company for Biological Products and Vaccines (Cairo, Egypt) were used as experimental animals. Animals were maintained in conditions of good ventilation, normal temperatures and humidity ranges and kept under observation for one week prior to experimentation. The rats were fed on standard pellets. Drinking water and food were provided *ad libitum* throughout the study. Experimental analyses were performed in the morning at 11:00 ± 1 h to avoid circadian variations. All animal procedures were performed in accordance with the Ethics Committee of the National Research Centre conformed to the "Guide for the care and use of Laboratory Animals" published by the National Institutes of Health (NIH publication No. 85–23, revised 1996).

Radiation Facility

Whole body gamma irradiation of rats was carried out with a ventilated Canadian Gamma Cell-40 (^{137}Cs) (Atomic Energy of Canada Ltd, Ottawa, Ontario, Canada), located at the National Center for Radiation Research and Technology (NCRRT), Cairo, Egypt. The dose rate was 0.5Gy/minute during the experimental periods.

Monosodium glutamate preparation

Monosodium L-glutamate (MSG) was purchased from Sigma Chemical Company. The dose was calculated by extrapolating a human dose (5g/day) to rat dose on the basis of body surface area ratio by referring to the table of **Paget and Barnes (1964).** Human dose x conversion factor (0.018) for rat = "X"/200g. "X" x 5= "Y"/Kg dose. So by calculating this way, the rat dose of MSG was 450mg/Kg body weight. MSG was dissolved in distilled water and administered orally to rats with the help of gastric tube of suitable size during a period of 4 weeks.

The animals of control and irradiated group were given equal volume of distilled water

Animal groups

Experimental animals were randomly divided into four groups of 10 rats. Control: Rats received distilled water via gavages daily during 28 days; MSG: Rats received MSG (450mg/Kg/day) via gavages daily during 28 days. RAD: Rat's whole body exposed to gamma rays at doses of 2Gy/week up to a total dose of 8Gy and received distilled water during the irradiation period. MSG + RAD: Rats received MSG daily during the irradiation period.

Preparation of Samples and Biochemical Analysis

All chemicals and reagents used were pure chemical materials from Sigma-Aldrich, St Louis, MO, USA. Rats were sacrificed by decapitation on the 28th day after a fasting period of 12 hours. The cerebral hemispheres were rapidly excised and 10% (w/v) tissue homogenate was prepared in normal 0.9% saline using Teflon homogenizer (Glass-Col, Terre Haute, Ind., USA). The homogenates were centrifuged at 10,000 g for 15 min using refrigerated centrifuge (K3 Centurion Scientific, Ltd, London, UK) and aliquots of the supernatant were separated and used for the further analysis.

Assessment of oxidative stress

Lipid peroxidation was assayed according to **Yoshioka *et al.* (1979)** based on the determination of malondialdehyde (MDA) an end product of lipid peroxidation, which react with thiobarbituric acid in acidic medium to yield a pink colored trimethine complex exhibiting an absorption maximum at 532 nm (T60 UV/VIS spectrophotometer, PG instruments, London, UK). Superoxide dismutase activity (SOD) was determined according to the method of **Nishikimi *et al.* (1972)** based on the ability of the enzyme to inhibit the phenazine methosulfate-mediated reduction of nitroblue tetrazolium (NBT) dyes. The increase in absorbance at 560 nm due to the formation of reduced NBT was recorded in a spectrophotometer. Catalase activity was determined ac-

cording to **Aebi (1984)** in which the disappearance of peroxide is followed spectrophotometrically at 240 nm. The method is based on the catalytic function of the enzyme where it catalyzes the decomposition of H_2O_2 into water and oxygen. Reduced glutathione (GSH) content was determined according to the method of **Beutler et al. (1963),** which depends on the reaction of GSH with 5, 5'-dithiobis 2-nitrobenzoic acid (DTNB) to yield GSSG and 2-nitro-5-thiobenzoic acid. Absorbance of the yellow color was measured at 412 nm.

Estimation of serotonin (5-HT) level

A portion of the cerebral hemispheres was homogenized in ice-cold n-butanol solution (5ml/g tissue) according to **Miller et al. (1970),** centrifuged at 3000 rpm (1560g) for 5 min then 2.5ml of the supernatant were transferred to a tube containing 1.6ml of 0.2N acetic acid and 5ml n-heptane. After mixing on a vortex mixer for 30s, the tubes were centrifuged at 3000 rpm (1560 g) for 5min and the aqueous phases were used for the estimation of serotonin according to **Curzon and Green (1970),** based on its fluorescence behavior when heated with o-phthalaldehyde in the presence of strong acid. The intensity of fluorescence measured through a fluorospectrophotometer, Kontron Co., Milano, Italy, is proportional to the concentration.

Measurement of neurotransmitters amino acids

Deproteinized tissue extracts were prepared by homogenizing a portion of cerebral hemispheres with 5% trichloroacetic acid **(Hsu et al., 1975)**. After centrifugation (at 4°C, for 20 min,at 8000g) the clear supernatant layer was decanted and washed several times with ether to remove any trace of lipids. The clean supernatant was dried and the residue was washed several times with distilled water to remove any trace of ether. Then to the dried residue a suitable volume of sodium citrate buffer was added and the sample was analyzed using high performance Amino Acid Analyzer, Biochom 20 (Auto Sampler Version) Pharmacia Biotech England. Data analysis of chromatogram was done by Ezchem™ Chromatography Data system Tutorial and user´s Guide-version 6.7 and the concentration was expressed in ug/g tissue.

Determination of serum metabolite levels

Commercial kits from bio-diagnostic Egyptian Company were used for the determination of glucose **(Trinder, 1969),** triglycerides **(Fossati and Prencipe, 1982),** total cholesterol, and HDL-cholesterol **(Richmond, 1973).** Low density lipoprotein cholesterol (LDL-c) concentrations were calculated according to Friedewald's formula **Friedewald (1972)**. LDL-c (mg/dl) = total cholesterol - (triglycerides/5)- HDL-c. Insulin level was determined by UBI MAGIWEL assay using enzyme immunoassay kits according to **Clark and Hales (1994).**

Estimation of insulin resistance

Homeostasis Model Assessment for insulin resistance (HOMA-IR) **(Matthews et al., 1985)** was calculated using the US formula: [fasting serum insulin (µIU/ml) × fasting plasma glucose (mg/dl) / 405]. Insulin resistance was defined as HOMA-IR >3.

Statistical analysis

The data were expressed as mean ± standard deviation. The Statistical Package for the Social Sciences (SPSS/PC) computer program was used for statistical analysis of the results. Data were analyzed using one way analysis of variance (ANOVA) followed by Duncan test as a Post Hoc ANOVA test to determine significant differences between means. Differences were considered significant at $P < 0.05$, highly significant at $P < 0.01$ and very highly significant at $P < 0.001$.

RESULTS

In the current study, the administration of MSG to rats (MSG group) via gavages at a dose of 450mg/Kg body weight (approximates a human dose of 5g/day) during four weeks has induced oxidative stress in the brain exhibited by a significant increase ($P < 0.05$) of MDA level and significant decreases ($P < 0.05$) of SOD and catalase activities and GSH

content compared to their respective levels in the control group. In case of whole body gamma irradiated rats with 2Gy/week up to 8Gy (RAD group), the increase of MDA was highly significant (P<0.01) and associated to very highly significant decreases (P<0.001) of SOD and catalase activities and GSH content compared to their respective levels in the control group. The administration of MSG to rats during the period of radiation exposure (MSG + RAD group) has potentiated oxidative stress evidenced by a significantly higher increase of MDA associated to a significantly lower level of antioxidants compared to their respective levels after each treatment given alone. Nevertheless, these variations appear more noticeable when compared with their respective levels in the MSG group than the RAD group (Table 1).

As shown in Table 2, following consumption of MSG during 28 days (MSG group), serotonin recorded highly significant diminution (P<0.01), with slight non-significant increase in aspartic and glutamic acids compared to their corresponding levels in the control group. On the other hand, rats exposed to gamma radiation 2Gy/week up to 8Gy (RAD group) displayed a very highly significant decrease (P<0.001) of serotonin and a very highly significant increase (P<0.001) of aspartic and glutamic acids compared to their respective levels in the control group. The administration of MSG to rats daily during the period of exposure to gamma radiation (MSG + RAD group) has potentiated the decrease of serotonin compared to each treatment given alone while it produced an additive effect on the increase of neurotransmitters amino acids where the increase reaches the sum of both treatment. However, these variations appear more important when compared with their respective levels recorded after exposure to MSG (MSG group) than after exposure to gamma radiation (RAD group).

Table (1) *Outcome of exposure to monosodium glutamate (MSG) and/or gamma radiation (RAD) on some oxidative stress markers in rats brain cerebral hemispheres.*

	Control	MSG	RAD	MSG+RAD
Malondialdehyde (nmol/g tissue)	453 ± 85	544 ± 92 (+20) <0.05[a]	573 ± 75 (+26) <0.01[a]	640 ± 50 (+41) <0.001[a] <0.01[b] <0.05[c]
Superoxide dismutase (U/g tissue)	186 ± 30	152 ± 24 (-18) <0.05[a]	125 ± 20 (-33) <0.001[a]	112 ± 21 (-40) <0.001[a] <0.001[b] <0.05[c]
Catalase (U/g tissue)	83 ± 14	66 ± 12 (-20) <0.05[a]	57 ± 10 (-31) <0.001[a]	46 ± 11 (-44) <0.001[a] <0.001[b] <0.05[c]
Glutathione (mg/g tissue)	1.08±0.18	0.89 ± 0.12 (-18) <0.05[a]	0.78 ± 0.15 (-28) <0.001[a]	0.66 ± 0.06 (-39) <0.001[a] <0.001[b] <0.05[c]

Values are expressed Means ± Standard deviation (n=10). [a] significance vs control; [b] significance vs MSG group. [c] significance vs RAD group. Values between brackets show percentage of change from Control.

Table (2) *Outcome of exposure to monosodium glutamate (MSG) and/or gamma radiation (RAD) on neurotransmitters in rats brain cerebral hemispheres.*

	Control	MSG	RAD	MSG+RAD
Serotonin (mg/g tissue)	254 ± 42	205 ± 24 (-19) <0.01[a]	170 ± 45 (-33) <0.001[a]	137 ± 20 (-46) <0.001[a] <0.001[b] <0.05[c]
Aspartic acid (mg/g tissue)	408 ± 36	445 ± 60 (+9) >0.05[a]	520 ± 51 (+27) <0.001[a]	572 ± 50 (+40) <0.001[a] >0.001[b] <0.05[c]
Glutamic acid (mg/g tissue)	1450 ± 240	1591 ± 250 (+10) >0.05[a]	1842 ± 170 (+27) <0.001[a]	1980 ± 120 (+37) <0.001[a] >0.001[b] <0.05[c]

Legends as for Table 1

Exposure to MSG (MSG group) as well as gamma irradiation (RAD group) produced a significant increase (P<0.05) in glucose and insulin levels with no effect on insulin resistance compared to control. Exposure to the combined effect of MSG and gamma radiation (MSG+RAD group) exhibit an additive effect on both levels and induced insulin resistance (Table 3).

Alteration of lipid profile indicated by a significant increase (P<0.05) of triglycerides, and total cholesterol, a very highly significant increase (P<0.001) of LDL-c and no significant change of HDL-c level was observed after MSG treatment (MSG group). In irradiated rats (RAD group) the changes were more significant and HDL-c showed a very highly significant (P<0.001) decrease. Exposure to the combined effect of MSG and irradiation (MSG+RAD) has potentiated the increase of triglycerides, total cholesterol, and LDL-c, compared to each single treatment. However, regarding the level of HDL-c the decrease was nearly similar to that recorded for irradiated rats (RAD group) yet compared with its level in the MSG group it was significantly decreased (Table 3).

DISCUSSION

The results obtained in the current study revealed that exposure to MSG (450mg/Kg body weight/day) for four weeks induced oxidative stress in the brain of rats noted by a significant increase of MDA associated with significant decreases of SOD and catalase activities and GSH content which agrees with earlier results involving other organs (**Kuldip and Pushpa, 2012; Onyema *et al.,* 2013 & Kumar and Bhandari, 2013**).

The increase in lipid peroxidation was accompanied by hyperglycemia, thus the possible autoxidation of glucose and increased reactive oxygen species (ROS) will eventually cause oxidative stress. In irradiated rats the alteration in the oxidant/antioxidant status was more noticeable which might be related to the excess of ROS, superoxide anion ($O_2^\cdot$), hydroxyl radical ($^\cdot OH$), and hydrogen peroxide (H_2O_2) resulting as bi-products of water radiolysis. Accordingly the interaction of $^\cdot OH$ with polyunsaturated fatty acids elevates the level of MDA (**Spitz *et al.*, 2004).** In addition, the decrease of antioxidants

Table (3) *Outcome of exposure to monosodium glutamate (MSG) and/or gamma radiation (RAD) on some serum metabolites and insulin resistance (HOMA-IR).*

	Control	MSG	RAD	MSG+RAD
Glucose (mg/dL)	70 ± 13	83 ± 12 (+19) <0.05[a]	85 ± 12 (+21) <0.05[a]	96 ± 11 (+37) <0.001[a] <0.05[b] <0.05[c]
Insulin(μIU/ ml)	13.0 ±1.2	14.3 ± 1.2 (+10) <0.05[a]	14.7 ± 1.4 (+13) <0.05[a]	15.9 ± 1.2 (+22) <0.001[a] <0.01[b] <0.05[c]
HOMA-IR>3	2.25	2.93	3.08	3.77
Triglycerides (mg/dL)	100 ± 15	114 ± 9 (+14) <0.05[a]	130 ± 11 (+30) <0.001[a]	140 ±9 (+40) <0.001[a] <0.01[b] <0.05[c]
Total cholesterol (mg/dL)	110 ± 14	124 ± 13 (+13) <0.05[a]	128 ± 12 (+16) <0.01[a]	138 ±19 (+25) <0.001[a] <0.05[b] <0.05[c]
HDL-c (mg/dL)	54 ± 6	48 ± 7 (-11) >0.05 [a]	36 ± 6 (-33) <0.001[a]	35 ± 4 (-35) <0.001[a] <0.001[b] >0.05[c]
LDL-c (mg/dL)	36 ± 6	53 ± 6 (+47) <0.001[a]	66 ± 7 (+83) <0.001[a]	75 ± 7 (+108) <0.001[a] <0.001[b] <0.01[c]

Legends as for Table 1

enhances oxidative damage in view of the fact that SOD catalyzes the reduction of $O_2^{\cdot-}$ to H_2O_2, rapidly removed by catalase and GSH-Px in presence of GSH **(Sun *et al.,* 1998).** As it could be expected exposure to MSG +RAD has potentiated oxidative stress when compared to the administration of each treatment suggesting that the consumption of MSG during exposure to gamma radiation would increase the risk of oxidative stress in the brain.

Data obtained in the present study showed that following consumption of MSG during 28 days (MSG group), serotonin recorded a highly significant diminution, with slight none significant increases in aspartic and glutamic acids compared to normal levels while exposure to ionizing radiation (RAD group) produced a very highly significant decrease of serotonin and a very highly significant increase of amino acids. The exposure to MSG+RAD exhib-

ited an additive effect on the decrease of serotonin and the increase of amino acids where their level reaches the sum of each treatment given alone. To interpret the non-significant changes in aspartic and glutamic acids recorded in the MSG group and the highly significant increase recorded in MSG+RAD group we have to refer that the blood brain barrier (BBB) is impermeable to glutamate, even at high concentrations, except in a few small areas that have fenestrated capillaries **(Hawkins, 2009).** Molecules entering or leaving the brain must pass 2 membranes (luminal and abluminal domains), and each membrane has distinct properties. Na-dependent glutamate cotransporters (excitatory amino acid transporters) exist exclusively in abluminal membranes. This organization does not allow net glutamate entry to the brain; rather, it promotes the removal of glutamate and the maintenance of low glutamate concentrations **(Hawkins, 2009)**. Based on this concept, the increase of aspartic and glutamic acids level recorded in the brain of rats exposed to MSG+RAD may be linked to modifications of the blood brain barrier permeability induced by exposure to ionizing radiation **(Diserbo *et al.*, 2002 and Fauquette *et al.*, 2012).**

The metabolic syndrome is a cluster of cardiovascular risk factors including insulin resistance and alteration in serum lipid profile **(Grundy *et al.*, 2005).** The incidence and prevalence of the metabolic syndrome are rapidly growing causing a significant burden for health systems worldwide **(Runge, 2007)**. In the current study exposure to MSG (MSG group) induced a significant increase of glucose and insulin levels with no insulin resistance detected which agrees with the findings of **Macho *et al.* (2000)** that MSG induces hyperinsulinemia and hyperglycemia. Hyperglycemia is probably the results of rise of portal glutamate levels which increased hepatic metabolism of glutamate, leading to release of glucose into systemic circulation **(Stegink *et al.*, 1983)**, while hyperinsulinemia might be the consequence of the action of MSG on glutamate receptors and the stimulation of insulin secretion **(Chevassus *et al.*, 2002).** However, conversely, to our results **Kondoh and Torii (2008)** revealed that oral ingestion of 2400

mg/kg/day MSG did not influence plasma glucose, and insulin. Results obtained in the current study revealed also that whole body gamma irradiation of rats induced hyperglycemia and hyperinsulinemia with no insulin resistance detected. The increase of glucose might result from its diminished utilization by irradiated tissues **(Ahlersova *et al.*, 1988)** and the consequence of protein catabolism and accelerated gluconeogenesis **(Kilberg and Neuhaus, 1976).** Accordingly, as it could be expected the exposure to MSG +RAD had an additive effect on the increase of glucose and insulin and induced insulin resistance.

The results recorded for serum lipid profile showed that exposure to MSG (MSG group) induced hyperlipidemia manifested by a significant increase of triglycerides, total cholesterol, and LDL-c levels whereas no significant change in HDL-c level compared to control levels. The result support previous works that dietary MSG enhances lipogenesis **(Bueno *et al.*, 2005)** increases serum triglycerides**(Collison *et al.*, 2010)**, fasting glucose and insulin levels **(Diniz *et al.*, 2005)**, and is associated with the prevalence of metabolic syndrome **(Insawang *et al.,* 2012)**. Conversely, **Kondoh and Torii, 2008** found that in rats the oral ingestion of 2,400 mg/kg/day MSG did not influence plasma glucose, insulin, triglycerides, or total cholesterol. In the current study, hyperlipidemia was more pronounced after whole body gamma irradiation and a significant decrease was recorded in HDL-c level. Hyperlipidemia might result from radiation-induced injury to cellular membranes **(Saada *et al.,* 2001)**; consequently fats are released in the circulation, by intestinal mucosa, adipose tissues, and liver. The elevated serum LDL-c levels might result from radiation-induced damage to the receptors on the surface of many cells in the body that prevents the ingestion of LDL-C by endocytosis **(Gent and Braakman, 2004)**. As it could be expected the consumption of MSG combined with irradiation (MSG+RAD group) potentiated hyperlipidemia compared to each single treatment.

According to the results obtained in the current study it could be concluded that consumption of

MSG associated to radiation exposure increase the possibility of oxidative stress and neurotransmitters alteration in the brain and the risk of metabolic syndrome.

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تاثير جلوتاميت الصوديوم على ما يحدثة الاشعاع من تغيرات بيوكيميائية فى ذكور الجرذان

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الجلوتامات أحادية الصوديوم هو عبارة عن ملح مكون من الصوديوم والجلوتامات وتعرف هذه المادة أيضاً بمسميات أخرى مثل : (الملح الصينى – فيتزين– أجينوموتو–أو E621) وتستخدم هذه المادة منذ القدم فى المطبخ الآسيوى وتضاف إلى الطعام المصنع والمعلب ووجبات المطاعم لكي تعزز النكهة وتفتح الشهية. وبالرغم من ارتباط استخدامها بالأكلات الآسيوية، إلا أنها الآن تستخدم على نطاق واسع فى الكثير من الأطعمة وفى وجبات المطاعم السريعة فى معظم دول العالم. انتشرت هذه المادة بشكل هائل فى عدد من المنتجات الغذائية كالمعلبات وحليب الاطفال واغذية الاطفال ومكعبات النكهة التي تضاف للمرق والارز والماكولات السريعة حيث تستخدمها هذه المطاعم لاخفاء تدنى جودة مكونات اغذيتها واعطائها طعما طازجا.

كما اصبح الانسان اكثر عرضه للتلوث الإشعاعى نتيجة لكثرة إستخدام المواد ذات النشاط الإشعاعى فى المجالات المختلفه كالطب العلاجى والتشخيص والصناعه. يتناول هذا البحث تقييم دور جلوتامات أحادية الصوديوم المحتمل فى احداث بعض التغيرات البيوكيميائيه فى أنسجة مخ الجرذان المعرضه للإشعاع الجامى. وقد تم حقن الجرذان بجلوتامات أحادية الصوديوم عن طريق الفم بتركيز قدرة 450 مليجرام/كجم من وزن الجسم يوميا لمدة 4 اسابيع وفى نهايه كل اسبوع تتعرض الجرذان لجرعه إشعاعية 2 جراى .

وتوضح نتائج هذه الدراسة أن تعرض الجرذان لجلوتامات أحادية الصوديوم او للاشعاع الجامى ادى الى حدوث إجهاد تاكسدى- إضطرابات إيضية وتغيرات فى مستوى الناقلات العصبية. ويتمثل الاجهاد التاكسدى فى حدوث زياده فى محتوى الدهون فوق المؤكسدة مع نقص شديد فى مستوى الجلوتاثيون وبعض أنزيمات مضادات الاكسدة مثل (إنزيمى السوبر اكسيد ديسميوتيز والكاتاليز). اما تغيرات مستوى الناقلات العصبية تتمثل فى حدوث زياده معنوية فى مستوى الاحماض الامينيه مثل حمض الاسبرتك وحمض الجلوتاميك مع نقص فى مستوى السيروتونين فى أنسجة مخ الجرذان. اما الإضطرابات الأيضية تتمثل فى حدوث ارتفاع معنوى فى مستوى الجلوكوز ومستوى الانسولين يصاحبه ارتفاع معنوى فى صوره الدهون. وتخلص النتائج الى ضرورة التوقف اوالحد من استخدام الجلوتامات أحادية الصوديوم عند العلاج بالاشعاع.

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