http://www.pjbs.org

PJBS

ISSN 1028-8880

Pakistan Journal of Biological Sciences

ANSInet
Asian Network for Scientific Information
308 Lasani Town, Sargodha Road, Faisalabad - Pakistan

OPEN ACCESS Pakistan Journal of Biological Sciences

ISSN 1028-8880
DOI: 10.3923/pjbs.2020.1146.1153

Research Article
How Pectin Play a Role in Histological Changes by Monosodium Glutamate (MSG) in the Ovary of Mice?

Sara A.l. Othman and Rania Suliman

Department of Biology, Faculty of Science, Princess Nourah Bint Abdulrahman University, Saudi Arabia

Abstract

Background and Objective: The effects of pectin from the natural vitamins and herbs on the ovary of mice induced by monosodium glutamate (MSG) leads to over accumulations in living cells and finally produces cellular toxicity and damage, pectin helps to rapidly reduce this changes. **Materials and Methods:** Cytotoxicity of monosodium glutamate was investigated histologically by using hematoxylin and eosin (H and E) stains. The animals received (MSG) in drinking water at a dose of 3 g kg^{-1} b.wt., in drinking water for three weeks. The ovary tissues were subjected to histological and morphological analysis. **Results:** In female rats treated with a dose of MSG of 3 g kg^{-1} daily in drinking water clear toxicological effects on the ovary tissue were significantly obtained. The mice were then anesthetized, dissected the ovary samples were taken from female mice and kept in a 10% neutral formalin solution to make tissue slides after that examined under the microscope to see the differences. Sections showed the occurrence of several histopathological changes in the ovary. **Conclusion:** This study concluded that the effectiveness of pectin therapy on ovarian cells destroyed by the effect of monosodium glutamate, which has proven to be very effective in treating all affected and restoring tissue to normal.

Key words: Monosodium glutamate, pectin, ovary, food additives, pollution

Citation: Sara A.l. Othman and Rania Suliman, 2020. How pectin play a role in histological changes by monosodium glutamate (MSG) in the ovary of mice? Pak. J. Biol. Sci., 23: 1146-1153.

Corresponding Author: Rania Suliman, Department of Biology, Faculty of Science, Princess Nourah Bint Abdulrahman University, Saudi Arabia

Competing Interest: The authors have declared that no competing interest exists.

Data Availability: All relevant data are within the paper and its supporting information files.

INTRODUCTION

Monosodium glutamate (MSG), the sodium salt of glutamate is a prototypical umami ligand used as a flavour enhancer in human diets[1]. The umami taste is the 5th basic taste, after sweet, sour, salty and bitter[2]. It is highly addictive, causing consumers to continue to return to eating more excessively. In this process, brain cells are destroyed because MSG destroys the brain and changes its ability to respond to the signal from leptin that satiety has occurred[3,4]. Furthermore, the researchers reported that cellular damage can result from oxidative stress in tissues[5] and therefore, it has been confirmed that monosodium glutamate oxidative stress causes the generation of reactive oxygen species by reducing antioxidants in the kidneys, leading to cellular injury and oxidation of proteins and fats[6]. Treatment with MSG also causes depletion of glutathione in tissues[7]. The current study showed that postpartum MSG administration to male rats caused mitochondrial dysfunction, accompanied by oxidative stress and an increase in ADP levels, in adult rat liver[8]. Pectin is a type of structural fiber found in the primary cell wall and intracellular layer of plant cells mainly in fruits such as; apples, oranges, lemons and so on. Modern pharmacological remedies, intended for treatment of acute and chronic intoxications with MSG, produce a series of undesirable effects that limit their application considerably[9]. In recent years, new data have appeared indicating the possibility of using alternative preparations of plant origin belonging to the group of non-starch polysaccharides that possess a metal-binding activity. This group of promising compounds also includes pectin obtained from the eelgrass from the Natural Vitamins and Herbs. Experiments *in vitro* have shown that this pectin surpass many other sorbents in its metal binding ability and is very comparable with complexing agents[10]. As well, pectin promotes the excretion of cytotoxic effects like MSG from animals tissue[11]. This work is aimed at evaluation of the antitoxic effect of pectin extracted from the Natural Vitamins and Herbs on the ovary state of rats with experimental process. The aim of this study was to identify the potential protective role of pectin against ovarian toxicity caused by monosodium glutamate in female rats. It shows its harmful effect on human and due to the widespread ingestion of this substance as food flavorings as found in all foods.

MATERIALS AND METHODS

Monosodium glutamate: Monosodium glutamate will be use and obtain from one of the commercial centers to sell food. It will be in the form of white powder packed in bags. It will be dissolve in drinking water.

Pectin: In this research, apple pectin will be obtained from the Natural Vitamins and Herbs Center (GNC). It is a tablet containing 300 mg tablets. Aqueous solution was prepared. It is 300 mg/70 kg of body weight.

Experimental animals: This research was conducted from December, 2018-September, 2019. Female albino mice were obtained from the animal house of the College of Pharmacy, King Saud University, Riyadh. The animals will select with an average age of 12-15 weeks and an average body weight of 60 g. The animals was distribute in special cages with feeding bottles to drink water in their ventilate rooms subject to suitable natural factors of humidity, light and temperature between 25 and 35°C.

The experimental animals were divided into the following groups:

First group (Experimental control group):

- The group of animals that was given natural water and this group include 5 female mice
- The group of animals was given pectin at a dose of 300 mg/70 kg of body weight in drinking water for 3 weeks

Second group (Group treatment)

Group of animals that were treated with monosodium glutamate: It contains 10 female rats where experimental animals were give monosodium glutamate at a dose of 3 g kg^{-1} b.wt., in drinking water for three weeks.

Animals group treated with monosodium glutamate and pectin: It contains 10 female rats were experimental animals which give monosodium glutamate at a dose of 3 g kg^{-1} b.wt., in drinking water for 3 weeks. Then give pectin directly for three weeks after the ingestion of monosodium glutamate at a dose of 300 mg/70 kg of body weight.

Histological technique: Liver and kidney mice were taken and preserved in a 10% neutral formalin solution to prepare histological segments by using the hematoxylin-eosin dye.

RESULTS

Histological structure of the normal ovary in mice as a control group: The ovaries are surrounded by a capsule and have an outer cortex and an inner medulla. The capsule is of

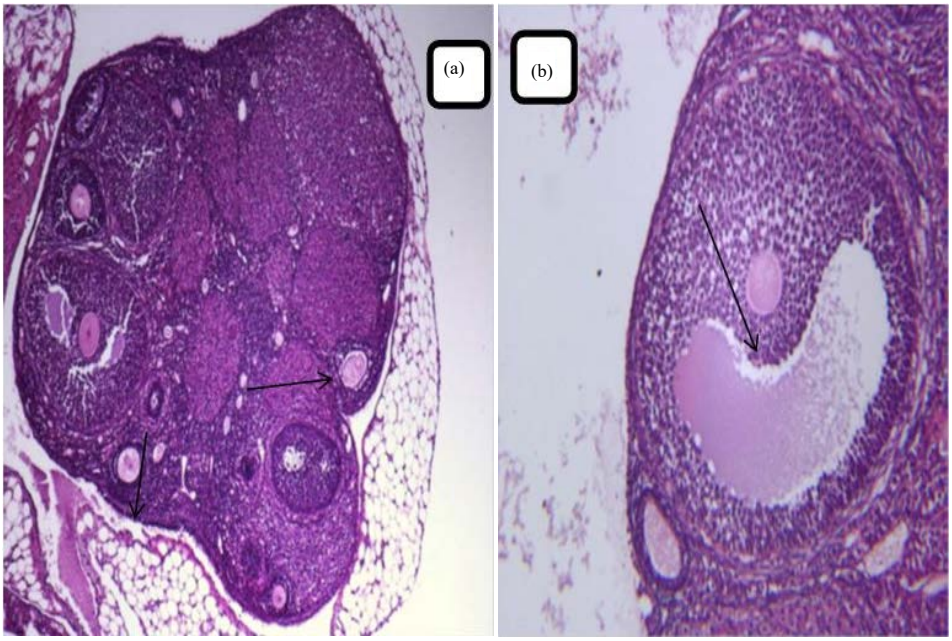

Fig. 1(a-b): Mice ovary of control group (a) Germinal Epithelium (GE) and Primary Follicle (PF) and (b) Follicular Cells (FC)
(H and E stain: x400)

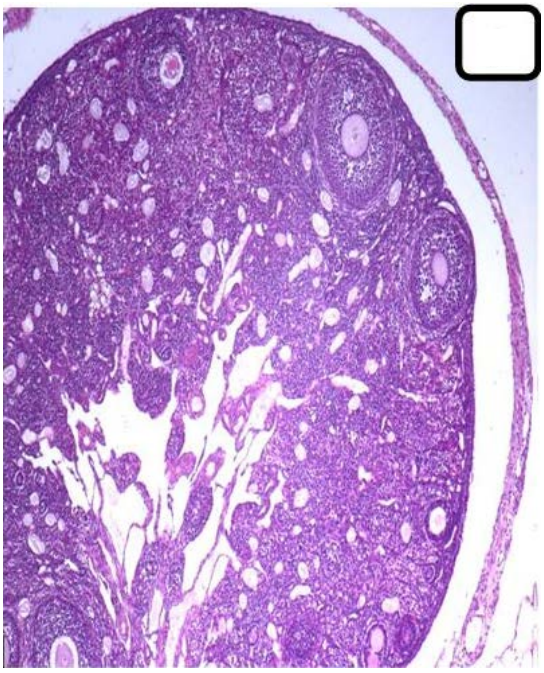

Fig. 2: Normal ovary tissues
(H and E stain: x400)

dense connective tissue and is known as the *Tunica albuginea*. The surface of the ovaries is covered with a membrane consisting of a lining of simple cuboidal-to-columnar shaped mesothelium called the germinal epithelium.

The outer layer is the ovarian cortex consisting of ovarian follicles and stroma in between them. Included in the follicles are the cumulus oophorus, membrane granulosa (and the granulosa cells inside it), corona radiate, zona pellucid and primary oocyte. Theca of follicle, antrum and liquor follicle are also contained in the follicle. Also, in the cortex is the corpus luteum derived from the follicles. The innermost layer is the ovarian medulla. It can be hard to distinguish between the cortex and medulla, but follicles are usually not found in the medulla.

Follicular cells are flat epithelial cells that originate from surface epithelium covering the ovary are surrounded by granulosa cells that have changed from flat to cuboidal and proliferated to produce a stratified epithelium as shown in Fig. 1a-b.

Pectin group: Ovarian segments appear in the pectin group similar to the natural composition as shown in Fig. 2.

Monosodium glutamate treatment group: The effect of monosodium glutamate was evident in the ovarian tissue where an increase in the *Tunica albuginea* thickness and also a decrease in the population of the ovarian follicles in the cortical part. *Tunica Albuginea*, great defects in follicles, atretic follicles and corpus luteum in developmental stages with high congestion compared to the control group as shown in Fig. 3a and b.

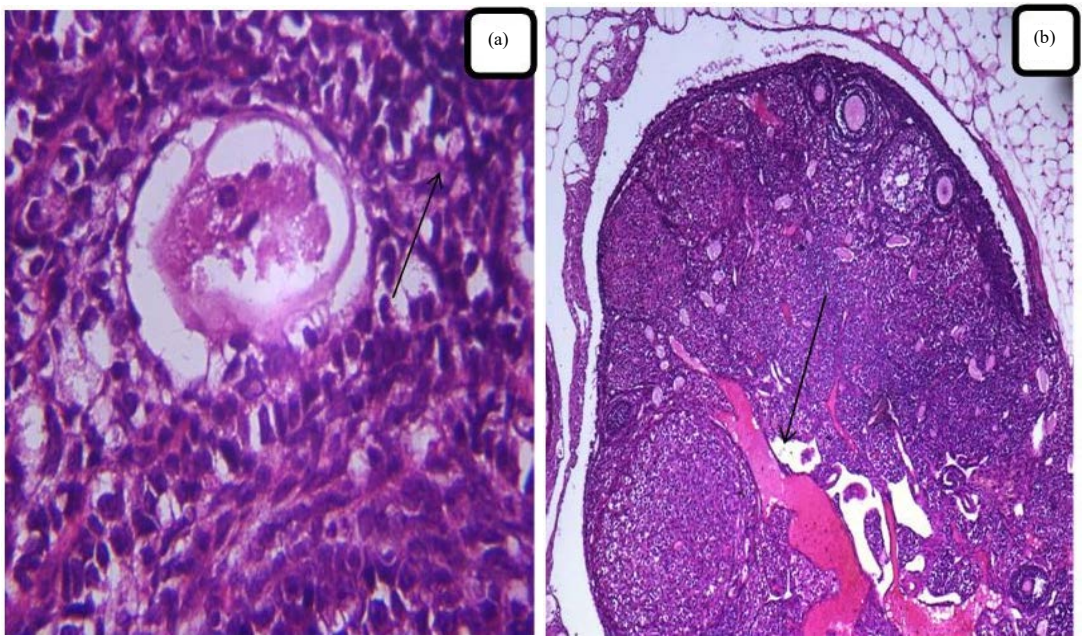

Fig. 3(a-b): Mice ovary of test group (a) *Tunica albuginea* and Corpus Luteum (CL) and (b) Congestion and Atretic Follicles (AF) (arrow)
(H and E stain: x400)

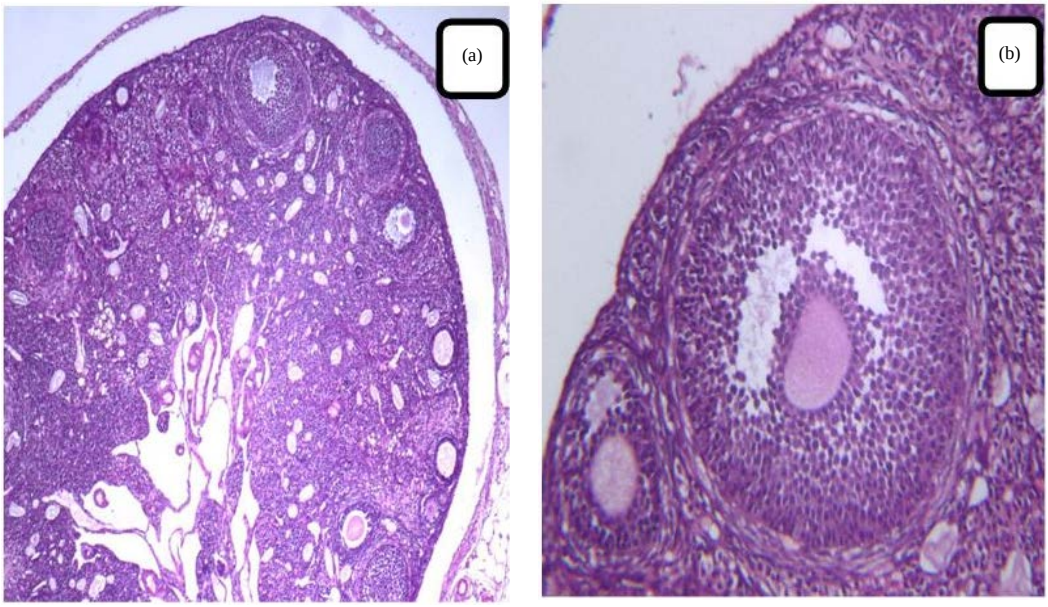

Fig. 4(a-b): Mice ovary of treated group (a) Normal ovary tissues with no evidence of congestion and (b) Degenerative change
(H and E stain: x400)

Pectin treated group: There was a significant improvement in the composition of the ovary as a result of eating pectin, where the tissue appeared almost similar to normal features of the ovary with no visible degenerative changes as shown in Fig. 4a and b.

DISCUSSION

Several pathological changes in the ovary were observed for mice exposed to monosodium glutamate where there was degenerative and multiple congestions of

many blood vessels which caused immature graafian follicle and harm of the corpus luteum with a central cavity.

Our results are consistent with several studies where histological studies in adult Wistar mice have shown that administration of monosodium glutamate at a dose 2 g kg^{-1} for 7 days has several adverse effects on the liver[12]. There was a significant increase in liver weight in mice treated with 70 mg kg^{-1} monosodium glutamate for 30 days which could be attributed to increased activity of inflammatory agents and oxidative damage that may lead to inflammation of the liver tissue after administration of MSG to mice (40 mg kg^{-1}, 9 days). There was a significant increase in body weight that may be due to obesity. The MSG-link obesity led to a change in the regulatory mechanism that affects fat metabolism[13,14], discovered many histological changes in liver cells and nuclei with the presence of inflammatory cells around the central veins in adult rats after treatment with MSG 3 and 6 mg kg^{-1} of body per day for 45 days. Harmful liver tissue changes also occurred in mice treated with MSG (3 and 6 mg kg^{-1} b.wt./day for 45 days)[13]. The results of another study showed that short-term exposure to MSG in pregnancy caused many histological changes in the tissues of the mother and fetal liver were evacuated in the liver cells and degenerative and necrotic areas and atrophy in hepatocytes with nuclei condensation (pyknotic nuclei).These changes were observed in the central and portal areas at a dose of 7 g 10 mL kg^{-1} b.wt., given from the 9th-14th day of pregnancy[14]. Histological examination of mice treated with MSG at a dose of 0.6 and 1.6 mg g^{-1} for 28 days also showed variable liver structure, central vein congestion and hypertrophy of the sinuses. The liver also invades many inflammatory cells, fluid retention, bleeding, congestion and cellular necrosis[15]. Biochemical and histological effects of low-dose monosodium glutamate (60 and 120 mg kg^{-1}) were determined for 28 consecutive days on the liver of male Sprague-Dawley mice of adults. The results showed that the total protein decreased significantly ($p<0.05$) and the antioxidant levels of superoxide dismutase (SOD) significantly ($p<0.05$) histological results revealed changes in normal liver structure and accumulation of red blood cells in central veins in both MSG groups. Histological findings in the study showed changes such as; central venous dilatation, which contained undistorted red blood cells, cellular abnormalities of liver cells and atrophic and degenerative changes in the liver of animals receiving feed containing monosodium glutamate[16] at a dose of 0.08 mg kg^{-1}. Sudden degeneration also appeared in hepatocyte straps and it was scalded that the blood vessels. Necrosis in hepatocytes, a blood disorder in the sinuses, decreased Kupffer cell count. Liver cells and vascular fibrosis were analyzed in rats that were administered at a single daily oral dose of 30 mg/kg/day (MSG) for 4 weeks continuously. Accordingly, it can concluded that MSG causes changes in the histological structure of the liver[17]. In mice treated with a dose of 1.6 mg g^{-1} b.wt., of dissolved MSG in 2 mL of oral distilled water, the samples examined showed the histological structure of the liver, where congestion in the central vein, an expansion of the sinuses was observed, reduced size of the nucleus of liver cells[18]. Bloody sinuses of the effect of monosodium glutamate on kidney tissue was evident in this study, where a change in histological structure was observed. Necrosis of most of them is the decomposition of some cells in addition to the condensation of nuclear chromatin in others and shrinkage and disappearance in renal globules. Shrinkage of renal glomeruli and the emergence of bleeding as was laceration in the walls of renal tubules and congestion of blood and bleeding apparent in some areas of renal tissue. Our results are consistent with several types of research where a group of mice received a daily dose of MSG (4 g kg^{-1}) for 7 days. Pyknotic nuclei, swollen mitochondria, margins of damaged frayed edge, elongated tubes, expand in Bowman governor with shrinkage and deformation of some glomeruli[19]. The study was conducted on male Wistar rats (5 weeks) fed a supplementary diet with 3 g of MSG kg/b.wt.,/day, 5 days a week. The excretion of sodium, potassium, calcium, phosphorus, creatinine, protein and nitric oxide in the urine was analyzed. It used clearance techniques to examine the glomerular filtration rate and cortical renal plasma flow. The oxidative state and pathological changes of renal tissue were examined. After the treatment of MSG, the excretion of sodium and potassium decreased despite the presence of hyperfiltration. The MSG group showed an increase in blood pressure, but the secretion of nitric oxide was significantly reduced and in the reduced glutathione /oxidative cycle and GPx and GR enzymes[20]. In this study, the effect of MSG was investigated at a dose of 4 mg MSG/g intraperitoneal body weight on the renal cortex of Worcester-type white mice, evolving pathological changes in the renal cortex, there was a pronounced influence of the tissue structure of the cauterization. Many glomeruli (66.4%) showed cell hyperparathyroidism, i.e., cellular proliferation of mesangial or endothelial cells and inflammatory cell infiltration[21]. Biochemical and tissue parameters in mice treated by MSG showed liver/kidney function tests, fasting cholesterol, triglycerides, uric acid in the blood significantly increased. Tissue showed inflammation in the portal areas with

peripheral fibrosis and interstitial nephritis in mice. Some studies have reported the oxidative effects of the use of this substance on different tissues and to increase changes in apoptosis in the testicular bacterial epithelium[22].

Furthermore, the researchers reported that cellular damage can result from oxidative stress in tissues . Thus, it has been confirmed that monosodium glutamate causes oxidative stress and generates reactive oxygen species by reducing antioxidants in the tissues, leading to cellular injury and oxidation of proteins and fats. Treatment with MSG also causes depletion of glutathione in tissues[23]. A study showed that postpartum MSG administration to male mice caused dysfunction of the mitochondria, accompanied by oxidative stress and an increase in ADP levels, in adult liver of mice. Some studies suggested that ingestion of monosodium glutamate at a dose of 4 mg g^{-1} b.wt. and above, causes induced oxidative stress in liver tissue in adult male rats[24]. Monosodium glutamate at all doses causes oxidative stress in the epididymis and prostate gland of experimental mice. Excessive consumption of monosodium glutamate has been reported to cause oxidative stress on the brain, liver and kidneys resulting in increased production of Reactive Oxygen Species (ROS)[25].

Pectin treatment has led to a marked improvement in the structure of the ovary where the tissue appeared almost similar to normal.

Our findings are consistent with several researches that have addressed the prevention of associated oxidative disorders and organ toxicity by taking antioxidants. In addition, scientists[26] have suggested that many natural and synthetic antioxidants can be used to prevent fat oxidation. Dietary fiber is one of the topics in which scientific research continues. The importance of research in the field of dietary fiber lies in the search for human sources of natural resources to maintain health after it was confirmed that modern technology has brought him diseases and illnesses unless his parents and grandparents knew. Pectin is characterized by that it is not affected by digestive enzymes in the human body, so when ingested and enter the digestive system it found that it absorbed part of the secretions of the gut and water, pectin goes into the colon and then expels out of the body[26]. A 20% complementary diet of pectin resulted in a significant reduction in the number of cases of colon tumors in mice and lower levels of prostaglandin E2 (PGE2) in the colon mucosa. The ability of pectin to decrease PGE2 was dependent, these results indicated an anti-inflammatory effect of pectin. Diets contained pectin and fish oil, which protected against colon cancer by programmed cell death and suppression of proliferation during the cancerous process[27]. Pectin demonstrated the dual advantages as a drug and therapeutic carrier for use in the treatment of colon cancer, where pectin is suitable for use as a drug delivery tool for the colon is selectively digested by microflora to release the drug with minimal degradation in the upper digestive tract[28]. Natural pectin for colon cancer prevention has been reported as a Dietary Fiber (DF) to enhance bioavailability and bio-efficacy. They have been reported to inhibit tumor growth, stimulate apoptosis, suppress malignant tumors and modify immune responses. In addition, pectin is an appropriate means of anti-cancer drug delivery systems, due to its adjustable functional groups and its special physical chemical properties[29]. In addition, pectin showed antioxidant activity of about 161.94 indicated that pectin has potential properties such as; biopolymers for biomedical applications and moderate antioxidant activity. It has been suggested that pectin directly affects immune cells to regulate inflammatory responses. The inflammatory cell expression caused by toll receptors (TLR) in spinal cord cells is inhibited by inhibiting the TLR signal by the lateral chains of the pectin. In a study of the effect of pectin on cancer patients, it was reported to inhibit cancer growth and improve the condition of patients with severe malignancy cancer hematologic acute, adding that it has a role in the treatment of prostate cancer and a role in inhibiting the growth and spread of cancerous tumors in the colon. Pectin also has a role in the resistance, protection and increased activity of Cirrhosis in the liver to manufacture antioxidant enzymes[30]. It also increases antioxidant activity, leading to regeneration of antioxidant balance, which is confirmed by the high coefficient of integrated antioxidant defense[31,32] 0.09-1.

CONCLUSION

From this study, we conclude that the exposure to monosodium glutamate has led to many damages in the ovary and the risk of this substance in its consumption of antioxidants leading to serious diseases and also named this substance. Pectin intake has reduced the toxicity of monosodium glutamate since, it is a powerful antioxidant that suppresses and disrupts the effect of this substance on different tissues.

ACKNOWLEDGMENTS

We thank Princess Noura Bint Abdul Rahman University for funding this research by the Deanship of Scientific Research at Princess Noura Bint Abdul Rahman University through the Rapid Research Funding Program.

This study discover the protective role of pectin against ovarian toxicity caused by monosodium glutamate in female rats. That can be beneficial for shows its harmful effect on human and due to the widespread ingestion of this substance as food flavorings as found in all foods.

REFERENCES

1. Beyreuther, K., H.K. Biesalski, J.D. Fernstrom, P. Grimm and W.P. Hammes and *et al*., 2007. Consensus meeting: Monosodium glutamate-An update. Eur. J. Clin. Nutr., 61: 304-313.
2. Ninomiya K., 2002. Umami: a universal taste. Food Rev. Int., 18: 23-38.
3. Campos, S., J. Monteiro, L. Antunes, P.S. Branco, L.M. Ferreira, L. Félix and P.G. de Pinho, 2014. Simultaneous quantification of propofol and its non-conjugated metabolites in several biological matrices using gas chromatography/ion trap–mass spectrometry method. J. Anal. Bioanal. Tech., 10.4172/2155-9872.1000195
4. Pyo J.S., 2016. Selective and Accurate Determination Method of Propofol in Human Plasma by Mixed-Mode Cation Exchange Cartridge and GC-MS. J. Anal. Methods Chem., 10.1155/2016/9531769.
5. He, K., S. Du, P. Xun, S. Sharma, H. Wang, F. Zhai and B. Popkin, 2011. Consumption of monosodium glutamate in relation to incidence of overweight in Chinese adults: China Health and Nutrition Survey (CHNS). Am. J. Clin. Nutr., 93: 1328-1336.
6. Vitor-de-Lima, S.M., L.B. Medeiros, R.D.L. Benevides, C.N. Dos Santos, N.O.L. da Silva and R.C.A. Guedes, 2019. Monosodium glutamate and treadmill exercise: Anxiety-like behavior and spreading depression features in young adult rats. Nutr. Neurosci., 22: 435-443.
7. Diniz, Y.S., A.A.H. Fernandes, K.E. Campos, F. Mani, B.O. Ribas and E.L.B. Novelli, 2004. Toxicity of hypercaloric diet and monosodium glutamate: Oxidative stress and metabolic shifting in hepatic tissue. Food Chem. Toxicol., 42: 313-319.
8. Shivasharan, B.D., P. Nagakannan, B.S. Thippeswamy and V.P. Veerapur, 2013. Protective effect of and *Calendula officinalis* andL. flowers against monosodium glutamate induced oxidative stress and excitotoxic brain damage in rats. Indian J. Clin. Biochem., 28: 292-298.
9. Rogan, W.J., 2000. Safety and efficacy of succimer in toddlers with blood lead level of 22-44 Mg/dL. Pediatr. Rev., 48: 593-599.
10. Khotimchenko, M., I. Serguschenko and Y. Khotimchenko, 2006. Lead absorption and excretion in rats given insoluble salts of pectin and alginate. Int. J. Toxicol., 25: 195-203.
11. Food Standards Agency, 2010. Current EU approved additives and their E numbers. Food Standards Agency, November 11, 2010. https://www.food.gov.uk/
12. Nnadozie J.O., U.O. Chijioke, O.C. Okafor, D.B. Olusina and A.N. Oli and *et al*., 2019. Chronic toxicity of low dose monosodium glutamate in albino Wistar rats BMC Res. Notes, 10.1186/s13104-019-4611-7.
13. Abu hanipah, E.N., N.J. Yahya, E.M. Ajik and N.A. Yusoff, 2018. Monosodium Glutamate Induced Oxidative Stress in Accessory Reproductive Organs of Male Sprague-Dawley Rats. J. Health Sci. Malaysian, 16: 67-73.
14. Anbarkeh F.R., R. Baradaran, N. Ghandy, M. Jalali and M.R. Nikravesh and *et al*., 2019. Effects of monosodium glutamate on apoptosis of germ cells in testicular tissue of adult rat: An experimental study Int. J. Reprod. Biomed., 17: 261-270.
15. Singh, K. and A. Pushpa, 2005. Alteration in some antioxidant enzymes in cardiac tissue upon monosodium glutamate (MSG) administration to adult male mice. Indian J. Clin. Biochem., 20: 43-46.
16. Ortiz, G.G., O.K. Bitzer-Quintero, C. Beas-Zarate, S. Rodriguez-Reynoso and F. Larios-Arceo and *et al*., 2006. Monosodium glutamate-induced damage in liver and kidney: A morphological and biochemical approach. Biomed. Pharmacother., 60: 86-91.
17. Havsteen, B.H., 2002. The biochemistry and medical significance of the flavonoids. Pharmacol. Therapeut., 96: 67-202.
18. Kamalakkannan, N., R. Rukkumani, K. Aruna, S.P. Varma, P. Viswanathan and V.P. Menon, 2005. Protective effect of N-acetyl cysteine in carbon tetrachloride induced liver injury in rats. Iran. J. Pharmacol. Ther., 4: 118-123.
19. Pirman T., M.C. Ribeyre, L. Mosoni, D. Rémond and M. Vrecl and *et al*., 2007. Dietary pectin stimulates protein metabolism in the digestive tract. Nutrition 23: 69-75.
20. Aozasa O., S. Ohta, T. Nakao, H. Miyata and T. Nomura, 2001. Enhancement in fecal excretion of dioxin isomer in mice by several dietary fibers. Chemosphere, 45: 195-200.
21. Licht, T.R., M. Hansen, A. Bergström, M. Poulsen and B.N. Krath, 2010. Effects of apples and specific apple components on the cecal environment of conventional rats: role of apple pectin. BMC Microbiol., 10.1186/1471-2180-10-13.
22. Cho Y., H. Kim, N.D. Turner, J.C. Mann and J. Wei and *et al*., 2011. A chemoprotective fish oil- and pectin-containing diet temporally alters gene expression profiles in exfoliated rat colonocytes throughout oncogenesis. J. Nutr., 141: 1029-1035.
23. Wong T.W., G. Colombo and F. Sonvico, 2011. Pectin Matrix as Oral Drug Delivery Vehicle for Colon Cancer Treatment. AAPS Pharm. Sci. Tech., 12: 201-214.
24. Li W., K. Zhang and H. Yang, 2018. Pectin alleviates high fat (lard) diet-induced nonalcoholic fatty liver disease in mice: possible role of short-chain fatty acids and gut microbiota regulated by pectin. J. Agric. Food Chem., 66: 8015-8025.

25. Zhang W., P. Xu and H. Zhang, 2015. Pectin in cancer therapy: A review. Trends Food Sci. Technol., 44: 258-271.
26. Ribichini E., S. Stigliano, S. Rossi, P. Zaccari and M.C. Sacchi and *et al*., 2019. Role of Fibre in Nutritional Management of Pancreatic Diseases. Nutrients, 10.3390/nu11092219.
27. Wathoni N., C.Y. Shan, W.Y. Shan, T. Rostinawati and R.B. Indradi and *et al*., 2019. Characterization and antioxidant activity of pectin from Indonesian mangosteen (*Garcinia mangostana* andL.) rind. Heliyon 10.1016/j.heliyon. 2019.e02299.
28. Ishisono K., T. Yabe and K. Kitaguchi, 2017. Citrus pectin attenuates endotoxin shock via suppression of Toll-like receptor signaling in Peyer's patch myeloid cells. J. Nutr. Biochem., 50: 38-45.
29. Hzhehots'kyĭ, M.R. and L.V. Fedorenko, 2006. State of the adaptation reactions in the correction process of the negative effect of the stress-factors of chemical nature. Fiziol. Zh., 52: 47-54.
30. Sudheesh, S. and N.R. Vijayalakshmi 2007. Role of pectin from Cucumber (*Cucumis sativus*) in modulation of protein kinase C activity and regulation of glycogen metabolism in rats. Indian J. Biochem. Biophys., 44: 183-185.
31. Anbarkeh F.R., R. Baradaran, N. Ghandy, M. Jalali and M.R. Nikravesh and *et al*., 2019. Effects of monosodium glutamate on apoptosis of germ cells in testicular tissue of adult rat: An experimental study Int. J. Reprod. Biomed., 17: 261-270.
32. Aprikian, O., V. Duclos, S. Guyot, C. Besson and C. Manach et al., 2003. Apple pectin and a polyphenol-rich apple concentrate are more effective together than separately on cecal fermentations and plasma lipids in rats. J. Nutr., 133: 1860-1865.