ные составляющие. Для каждого из них расчитан % выхода по отношению к сухому сырью. Качественно обнаружены входящие в них классы липидов. Оценены физико-химические показатели. Методами газовой хроматографии и массспектрометрии проведена качественная и количественная идентификация содержащихся в нейтральных липидах ненасыщенных и насыщенных жирных кислот и их процентный состав. В суммах полярных липидов качественно идентифицированы и количественно оценены входящие в их состав фосфолипиды. Показано наличие каротиноидов и аминокислот.

Согласно полученным данным, липидный состав (неполярные, полярные компоненты) исследованных масел содержит значительный ряд биологически активных компонентов, что придает исследуемым маслам определённую ценность в вопросах фармакологической конкурентности и нуждается в дальнейших научно-практических исследованиях.

რეზიუმე

საქართველოში მოზარდი ზოგიერთი მცენარის შესწავლა ლიპიდების და თანმხლები აქტიური ნაერთების შემცველობაზე

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თბილისის სახელმწიფო სამედიცინო უნივერსიტეტი, იოველ ქუთათელაძის ფარმაკოქიმიის ინსტიტუტი, საქართველო

კვლევის მიზანს წარმოადგენდა საქართველოში მოზარდი ზოგიერთი მცენარის: კრაზანას თესლის Hyperium perforatum L., ოჯახი კრაზანასებრნი, სიმინდის თესლის Zea mays L., ოჯახი მარცვლოვნები და შვიტას Equisetum arvenses L. ოჯახი შვიტასებრნი მიწისზედა ნაწილების შესწავლა ლიპიდების და თანმხლები ბიოლოგიურად აქტიური ნაერთების შემცველობაზე. საკვლევი ობიექტებიდან მიღებულია ნეიტრალური და პოლარული ლიპიდების ჯამები სხვადასხვა პროცენტული გამოსავლით, დადგენილია მათში შემავალი მირითადი კლასები, განსაზღვრულია ზოგიერთი ფიზიკო-ქიმიური მახასიათებელი, გაზურ ქრომატოგრაფიული მეთოდით ნ/ლ ჯამებში თვისობრივად და რაოფენობრივად იფენტიფიცირებულია ნაჯერი, უჯერი და პოლიუჯერი ცხიმოვანი მჟავები, ხოგიერთი მათგანის მაღალი პროცენტული შემცველობით. პ/ლ ჯამებში თვისობრივად დადგენილია და რაოდენობრივად განსაზღვრულია ფოსფოლიპიდები. კვლევის საფუძველზე აღნიშნულ ობიექტებში დადგენილია ზოგიერთი ბიოლოგიურად აქტიური ნაერთების კაროტინოიდების, ამინომჟავების არსებობა. მიღებული შედეგების საფუძველზე, საკვლევი ობიექტებიდან გამოყოფილი მცენარეული ზეთები მდიდარია სხვადასხვა ბიოლოგიურად აქტიური ნაერთებით, რომელიც გვაძლევს აღნიშნული ზეთების გამოყენების შესაძლებლობას კოსმეტოლოგიასა და პრაქტიკულ მედიცინაში.

MONOSODIUM GLUTAMATE (E621) AND ITS EFFECT ON THE GASTROINTESTINAL ORGANS (REVIEW)

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Currently, synthetic food additives are considered the most hazardous since they are xenobiotics that are unusual for the human body from the time of its evolutionary development and, therefore, it lacks enzymes that can convert them into non-toxic metabolites [1].

Monosodium glutamate (E621) is widely used in the marketing as a taste enhancer and is added to many processed foods. Monosodium glutamate, added to food products (≤ 10 g/kg), enhances their natural flavor that weakened in the course of processing and storage, and disguises certain negative components of the flavor and smell. Currently, about 50% of on-the-shelf products contain the above additive, with the average daily human consumption of about 0.3-1.0 g in European highly developed countries [2]. Although food safety regulatory authority considers the consumption of monosodium glutamate to be safe, some preclinical and clinical studies have questioned its safety, © *GMN* especially after chronic exposure. The controversy is probably caused by the involvement of endogenous glutamate in both physiological and pathological processes [3].

The Joint FAO/WHO Expert Committee on Food Additives (JECFA), the US Food and Drug Administration (FDA) and the European Food Safety Association (EFSA) considered monosodium glutamate to be a safe substance (GRAS). The food additive is included in the GRAS list if it was widely used in food products before 1958 (approval is based on the experience) or when its safety has been confirmed by scientific toxicological reports based on expected food consumption. However, currently, some authors state that the GRAS inclusion criteria, both for science-based and experience-based procedures, need to be updated based on the events conducted in toxicity testing [4].

Currently, the European Commission is considering the revision of the current standards for toxic elements in the EU specifications for monosodium glutamate (E621) to ensure that they are not a significant source of exposure to the toxic elements in food, particularly in the food categories most conducive to overall exposure to glutamic acid and its salts: small bakery products, soups and broths, sauces, meat and meat products, spices and food additives [5].

The paper was aimed at the analysis of the literature data on the effect of monosodium glutamate on various organs and systems of the human body.

Material and methods. In the course of research, the following techniques have been used: bibliosemantic method for the analysis of scientific publications. The paper provides assessment of 40 literary sources. Particular attention is paid to sources over the last 5 years (2016-2021), but some earlier publications that have not lost their relevance are also included in the review. The sources were taken from scientific metric databases Scopus, Web of Science, PubMed, Medline, Google Scholar and the portal of scientific periodicals of V.I. Vernadsky National Library of Ukraine.

Results and discussion. Monosodium glutamate (MSG) or E621 is a widely used flavor enhancer and salt substitute derived from L-glutamic acid, an amino acid of natural origin found in various foods. Common synonyms for sodium glutamate are Monosodium L-glutamate monohydrate; sodium glutamate monohydrate; L-glutamic acid, sodium salt, monohydrate (1: 1: 1); L-glutamic acid monosodium salt monohydrate, Natrium-glutaminat, Glutamate sodium, Sodium L-glutamate. MSG was discovered by Rithhausen in 1866. The stimulating effect of L-glutamic acid was studied in the 50s of the last century, though only in the 70s it was proven to be excitatory mediator for the CNS of vertebrates [6].

MSG has a special umami taste, which was initially considered the predominant taste in Asia, and then in Western cultures. This molecule was identified about 100 years ago by Kikunae Ikeda as the fifth main taste, apart from sweet, sour, salty and bitter [7]. MSG is found in foods high in protein, such as meat or fish, as well as in some types of cheese (Roquefort and Parmesan) or vegetables (tomatoes, mushrooms, broccoli). In addition to its main specificity, the umami taste can enhance the overall flavor intensity and improve the food taste. This effect depends on many factors, the most important of which are the concentration of the umami molecule and the food matrix [8].

In recent years, many scientific studies have been conducted to study several effects that affect the umami mechanism, which is detected and enhanced by certain concentrations of MSG and umami compounds [9]. Previous behavioral studies have shown that L-glutamate, an umami substance, is found in the intestine and glutamate-related information is transmitted from the intestine to the tonsils and lateral hypothalamus (LH) through the vagus nerve to establish predominance of glutamate [10]. There is a complex bidirectional communication system between the gastrointestinal tract and the brain. Originally called the "intestinal-brain axis", it has been currently renamed to "microbiotaintestinal-brain axis", given the key role of the intestinal microbiota in regulation of the local and systemic homeostasis [11]. This explains the physiological role of the dietary signal of glutamate through the intestine and brain axis due to efficient digestion and absorption through the innervation of the duodenum by vagus nerve [12].

There is a concept of monosodium glutamate dependence. For an individual who often uses the taste enhancer, regular food seems to be "flat" and tasteless. Over time, lingual taste buds fail to percept a variety of tastes. The observed effect of

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monosodium glutamate deprivation may indicate the formation of a pathological urge to consume it [13]. It is not surprising that monosodium glutamate is called a contemporary legal drug [14]. Sociological consumer survey reports that 53% of respondents have no idea what kind of substance it is, 16% never thought about its harmfulness, and 31% who have long known it, do not look at the composition and disregard its content in food products [15].

Currently, no reliable data, showing at what doses and under what conditions monosodium glutamate, consumed regularly, is harmful to health, have been found. There are studies showing that consumption of 3 g/day monosodium glutamate is already harmful to human health. According to the updated food safety information on monosodium L-glutamate, high quality monosodium glutamate is safe at all stages of the life cycle, regardless of the ethnicity or culinary preferences. MSG researchers are encouraged to use appropriate scientific methodologies, to consider the glutamate metabolism and its normal consumption in food before extrapolating pharmacological studies in rodents to humans [16]. The investigations report that daily administration of monosodium glutamate to rats, even in safe human health doses (15 and 30 mg/kg, corresponding to 1 and 2 g per average person) has a toxic effect [14, 17].

In addition to the well-known effect on the food flavor, glutamate performs various physiological functions: monosodium glutamate enhances saliva secretion and disrupts carbohydrate metabolism, as well as affects the feeling of satiety and recovery after eating [18]. It is the main substrate for energy production in enterocytes, an intermediate in protein metabolism, a precursor of the essential metabolites such as glutathione (GSH, oxidative stress modulator) or N-acetylglutamate (regulator of metabolism), and excites the central nervous system neurotransmitter [19].

After oral administration, glutamate is oxidized in enterocytes in the small intestine [20]. Subsequently, only a very small amount of it is detected in the portal blood and, most likely, this is due to glutamine catabolism as a result of glutaminase activity in the intestine, rather than the absorption of dietary glutamate [21]. After oxidation, glutamate is further converted to other amino acids or used as a precursor for the synthesis of various bioactive compounds [22].

Consumption of monosodium glutamate also correlates with changes in the homeostasis of antioxidant protection, secondary to the loss of integrity and functionality of neuronal membranes, with increased nonspecific permeability for several ions and pathological changes in the intracellular metabolic processes [23].

Monosodium glutamate in high doses has an unpleasant taste and can cause discomfort in the gastrointestinal tract, indicating its harmful effect and signaling to stop its consumption immediately [24]. As for the constant use in acceptable, almost imperceptible doses, most researchers emphasize the prolonged effect of monosodium glutamate in its long-time use, which leads to the development of pathological effects [25]. It has been reported about the significant changes in neuronal redox homeostasis (increased levels of lipid peroxidation, nitrite concentrations, decreased levels of antioxidants) and histology of hippocampal neurons, along with increased levels of cholinesterase in the brain and serum [26].

Studies have shown that excess monosodium glutamate can provoke the development of hypertension and stroke, diabetes, Alzheimer's disease and the nervous system disorders. Studies associate its consumption with neurotoxicity, cardiotoxicity, fibrosis and neoplastic changes, liver and kidney dysfunction, and metabolic and weight gain disorders [3, 27]. The following behavioral and physiological changes were observed: increased aggression; decreased motor activity and loss of muscle strength [28]. Diet with excess glutamate led to vision loss in rats caused by acute neuronal degeneration of retinal ganglion cells and its thinning [29].

Currently, more and more researchers are studying the ways monosodium glutamate affects the physiology of the gastrointestinal tract. However, the mechanism of absorption and subsequent transfer of dietary lipids into the lymph is unknown to date. There is still little information on how the consumed monosodium glutamate affects lipid lipolysis, absorption, intracellular etherification, and chylomicron formation and secretion. One of the studies has shown that monosodium glutamate causes a significant decrease in the secretion of triglycerides and cholesterol into the lymph of rats, which were administered with 2% monosodium glutamate solution. This is the first demonstration of the effect of monosodium glutamate on the lymphatic transport of lipids in the intestine [30].

Most authors emphasize that the prolonged consumption of the above food additive affects eating behavior, motility of the gastrointestinal tract, the structure and functional state of the stomach [31]. It also affects the body weight of rats, causes metabolic disorders and weight gain and leads to obesity [32]. Obesity is also promoted by the ability of monosodium glutamate to increase the distension of the antrum and the level of amino acids in plasma, even after a standard meal [33].

The effect of long-term administration of monosodium glutamate on the rats' gastric mucosa and basal secretion of gastric juice acid has also been demonstrated. It has been found that 10-, 20-, 30-day feeding of rats with monosodium glutamate at the doses of 15 to 30 mg/kg (equivalent to 1 and 2 g per person) leads to erosive and ulcerative lesions of the gastric mucosa and increased secretion of hydrochloric acid and weight gain. Excessive consumption of monosodium glutamate can cause the "Chinese restaurant syndrome" and gastritis, gastric and duodenal ulcers [34].

The analysis of many literature sources revealed that in high doses, monosodium glutamate has a local pathogenic effect on gastric tissue, revealed by thinning of all layers of the gastric wall, desquamation of the mucous membrane and its disorganization by reducing the size of gastric glands, increasing the number of vessels and their plethora. One of the mechanisms of pathogenic effect of monosodium glutamate is the contact local and free radical oxidizing effect on gastric tissues. It is caused by the stimulating effect on parietal cells, i.e., systemic consumption of monosodium glutamate pathologically excessively increases the secretion of hydrochloric acid in the stomach. Consequently, monosodium glutamate becomes a pathogenetic factor in the formation of erosive-ulcerative lesions in the gastric mucosa and hyperphagia, which is the cause of obesity [35]. In addition, the long-term administration of monosodium glutamate is associated with a significant reduction and contraction of the rough endoplasmic reticulum in the epithelial cells of the small intestine, which is also characteristic of obesity [36]. In turn, the functional deterioration of the adhesion structures between the epithelial cells of the small intestine causes dysfunction of the gastrointestinal barrier, which leads to increased intestinal permeability of blood vessels and, consequently, systemic inflammation, characterized by macrophage infiltration. Thus, in animals with chronic obesity, induced by administration of monosodium glutamate, numerous gaps between the epithelial cells of the small intestine were found, and the levels of both desmosomal and dense proteins were significantly lower in the epithelial cells of the small intestine. Moreover, there was a significant increase in the number of inflammatory intestinal cells, especially macrophages, and blood samples showed an increase in markers of inflammation, tumor necrosis factor-alpha and interleukin-1-beta [37].

It has been found that consumption of MSG for 1 month also leads to structural reorganization of the mucous membrane of the rats' colon, disruption of mucus production by goblet cells due to their hypertrophy and hyperplasia, increased content of sialo- and fucoglycoproteins and decreased lysozyme activity [38].

Reports on the impact of monosodium glutamate on gastrointestinal motility are quite inconsistent. The umami taste amino acid, glutamate, acts as a signaling molecule in many cellular systems of the body, including the brain and gastrointestinal tract. Consequently, glutamate, influencing the appetite, can regulate the motility of the gastrointestinal tract, thus affecting gastric emptying (promotes emptying) and peristalsis of the duodenum [39].

The study of the impact of complex food additives (sodium nitrite, monosodium glutamate and Ponceau 4R) on the adaptive responses of rats, even at the doses twice less than the permissible norm in food products, has established the effect on the behavioral responses of experimental animals. The "open field" test has shown that from the first week of observation, rats experienced increased anxiety, fear, blunting of adaptive responses, decreased activity and disturbance of the emotional state, which were intensified up to week 16 of the experiment. It is also believed that excessive intake of complex food additives is a direct threat of stomach damage, namely, the development of peptic ulcer disease, which is preceded by the development of acute and chronic gastritis [40].

Conclusions. The study of the mechanisms of influence of various food additives on the human body and animals is one of the most pressing problems to date. Physicians, toxicologists, physiologists are interested in the mechanisms of their toxic effect, as well as the study of compensatory-adaptive reactions in response to entry into the body.

The analysis of the publications has shown that the views on the effect of monosodium glutamate on the human body are quite contradictory: from the complete safety of the above additive to the confirmation of its negative effect on various organs and systems.

The present scientific literature review proves the importance of further study of the food additives and their effect to develop a scientifically grounded strategy to increase tolerance of humans and animals to xenobiotics by activating genetically fixed mechanisms, as well as by creating new perfect adaptogens.

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SUMMARY

MONOSODIUM GLUTAMATE (E621) AND ITS EFFECT ON THE GASTROINTESTINAL ORGANS (REVIEW)

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The study of the mechanisms of the effect of various food additives on the human and animal organism is one of the most pressing problems today. The work of physicians, toxicologists, physiologists is aimed at studying the mechanisms of the toxic effect of food additives, as well as studying compensatory-adaptive reactions in response to their ingestion. Monosodium glutamate (E621) is widely used in marketing as a flavor enhancer and is added to many processed foods. Today, about 50% of store products contain this additive, while the average daily human consumption in industrialized European countries is approximately 0.3-1.0 g.

The purpose of this work is to analyze the literature data on the effect of monosodium glutamate on various organs and systems of the human body. The research used the bibliosemantic method of analyzing scientific publications. The article assesses 40 literary sources. Special attention is paid to the sources for the last 5 years (2016-2021).

This review of the scientific literature proves the importance of further study of food additives and their effect for the development of a scientifically based strategy for increasing the tolerance of humans and animals to xenobiotics by activating genetically fixed mechanisms, as well as by creating new perfect adaptogens.

Keywords: monosodium glutamate, rats, gastrointestinal organs.

РЕЗЮМЕ

ГЛУТАМАТ НАТРИЯ (Е621) И ЕГО ВЛИЯНИЕ НА ОРГАНЫ ЖЕЛУДОЧНО-КИШЕЧНОГО ТРАКТА (ОБ-ЗОР)

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Изучение механизмов воздействия различных пищевых добавок на организм человека и животных - актуальная проблема современности. Работа медиков, токсикологов, физиологов направлена на изучение механизмов токсического действия пищевых добавок и изучение компенсаторно-приспособительных реакций в ответ на попадание их в организм. Глутамат натрия (E621) широко используется в маркетинге как усилитель вкуса и добавляется во многие обработанные пищевые продукты. На сегодняшний день около 50% магазинных продуктов содержат эту добавку. Средняя дневная норма потребления глутамата натрия в европейских промышленно развитых странах составляет примерно 0,3-1,0 г.

Цель исследования - анализ литературных данных о влиянии глутамата натрия на различные органы и системы организма человека. В ходе исследования использовался библиосемантический метод анализа научных публикаций. В статье представлена оценка 40 литературных источников. Особое внимание уделяется источникам за последние 5 лет (2016-2021 гг.).

Настоящий обзор научной литературы доказывает значимость дальнейшего изучения пищевых добавок и их влияния для разработки научно обоснованной стратегии повышения толерантности человека и животных к ксенобиотикам путем активации генетически фиксированных механизмов и создания новых совершенных адаптогенов.

რეზიუმე

ნატრიუმის გლუტამატი (E621) და მისი გავლენა საჭმლის მომნელებელი ტრაქტის ორგანოებზე (მიმოხილვა)

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პოლტავის სახელმწიფო სამეიდიცინო უნივერსიტეტი, უკრაინა

ადამიანის და ცხოველების ორგანიზმზე სხვადასხვა კვებითი დანამატის გავლენის კვლევა თანამედროვეობის აქტუალურ პრობლემას წარმოადგენს. მედიკოსების, ტოქსიკოლოგების, ფიზიოლოგების მუშაობა მიმართულია საკვები დანამატების ტოქსიკური მოქმედების მექანიზმებისა და ორგანიზმში მათი მოხვედრის საპასუხოდ განვითარებული კომპენსაციურშემგუებლობითი რეაქციების შესწავლაზე. ნატრიუმის გლუტამატი (E621) ფართოდ გამოიყენება მარკეტინგში, როგორც გემოს გამაძლიერებელი; იგი ემატება ბევრ გადამუშავებულ საკვებ პროდუქტს. დღეს მაღაზიის პროდუქტების თითქმის 50% შეიცავს ამ დანამატს. ნატრიუმის გლუტამატის მოხმარების დღიური ნორმა ევროპის განვითარებულ ქვეყნებში შეადგენს დაახლიებით 0,3-1,0 გრამს.

კვლევის მიზანს წარმოადგენდა ლიტერატურის მონაცემების ანალიზი ნატრიუმის გლუტამატის გავლენის შესახებ ადამიანის ორგანიზმის სხვადასხვა ორგანოსა და სისტემაზე. კვლევის დროს გამოყენებული იყო სამეცნიერო პუბლიკაციების ანალიზის ბიბლიოსემანტიკური მეთოდი. სტატიაში მოცემულია ლიტერატურის 40 წყაროს შეფასება. განსაკუთრებული ყურადღება დათმობილია ბოლო 5 წლის (2016-2021 წწ.) წყაროებისათვის.

წარმოდგენილი სამეცნიერო მიმოხილვა ადასტურებს საკვები დანამატების და მათი გავლენის შემდგომი კვლევის მნიშვნელობას ადამიანისა და ცხოველების ტოლერანტობის მომატების სტრატეგიის შემუშავების მიზნით ქსენობიოტიკების მიმართ გენეტიკურად ფიქსირებული მექანიზმების აქტივაციის და ახალი, სრულყოფილი ადაპტოგენების შექმნის გზით.