

The Toxic Impact of Monosodium Glutamate in Rats

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Abstract

Background. Nowadays, more than 2500 additives are intentionally added to food in order to keep certain properties or to extend shelf life. One of the most common food additives in Ukraine and in Europe is monosodium glutamate. Potentially negative health effects of monosodium glutamate prompt us to question safety of its wide-spread use.

Objectives. In this study we defined the effect of monosodium glutamate (E 621) on the main markers of endogenous intoxication in rats.

Materials and Methods. Experimental studies were conducted on 72 nonlinear male white rats weighing 150-180 g. The experimental animals were administered monosodium glutamate at a dose of 30 mg/kg body weight (corresponds dose 2 g per day in humans) for 7, 14 and 30 days. The control group of animals was given normal saline. Syndrome of endogenous intoxication was evaluated using measurements of low, medium, and high molecular weight substances in blood plasma, red blood cell suspension, and urine.

Results. Our results indicated a shift of the markers of intoxication syndrome towards mainly catabolic substances. The results obtained after one week of the experiment correspond with phase of partial compensation, characterized by increased concentrations of low and middle molecular weight substances in red blood cells and plasma. After two weeks and up to one month of the experiment, the predominantly catabolic markers of endogenous intoxication continue to increase in erythrocytes and plasma, indicating a shift to the phase of partial decompensation to systems and organs of detoxification.

Conclusion. The administration of monosodium glutamate at a dose of 30 mg/kg body weight was associated with development of excessive contents of low and middle molecular weight substances with reduced ability of kidneys to excrete toxic products.

Keywords: monosodium glutamate, endogenous intoxication, indices, rats.

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Introduction

Since the dawn of man, our species searches for better ways to feed itself, by developing more efficient methods of hunting,

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animal/vegetable domestication, food preservation by physical methods, and finally, by adding molecules to food in order to enhance flavors or to preserve it. Today, more than 2500 additives are intentionally added to food in order to keep certain properties or to extend shelf life, while many others were banned throughout the years, some of them at a global level and others only in specific countries ¹.

One of the most common food additives in Ukraine and in the world is monosodium glutamate (MSG, $C_5H_8NO_4NaH_2O$). It is the sodium salt of the non-essential amino acid glutamic acid, which is one of the most abundant amino acids found in nature and exists both as free glutamate and bound with other amino acids in protein encoded E621 ², it is a food additive from a group of flavor enhancers, used in a wide range of foods, such as soups, sauces, mixed condiments, chips, meat products, and puddings ³.

The estimated average daily MSG intake per person in industrialized countries is 0.3– 1.0 g, but it depends on the MSG content in foods and an individuals' taste preferences ⁴. Despite its widespread use as food flavor and its generally considered safety, some questions regarding the impact of its use on general health have arisen ⁵. There are reports which indicate that MSG is toxic to humans and laboratory animals especially at high doses ⁶⁻⁹. Also, MSG allergy been known to cause asthma, urticaria, atopic dermatitis, ventricular arrhythmia, neuropathy, and abdominal discomfort. It has also been implicated in male infertility by causing testicular damage and abnormal sperm cell morphology ⁴. Moreover, it has been reported that MSG is neurotoxic, capable of producing degeneration of population of neurons, accompanied by pathological conditions, such as stroke, epilepsy, schizophrenia, anxiety,

depression, Parkinson's disease, Alzheimer's disease, Huntington's disease, and amyotrophic lateral sclerosis ¹⁰.

There are studies indicated that MSG intake at a dose of 3 g per day is dangerous to human health ¹¹. However, some scientists argue that daily administration of MSG to rats even at low doses is toxic. The results of the M.S. Tawfik and N. Al-Badr investigation have shown that MSG at low doses (0.6 and 1.6 mg/g body weight for 14 days) is capable of producing alterations in the body weight, liver and kidney functions ¹².

The integral marker of endotoxemia is the content of middle molecular weight substances (MMWS) — heterogeneous group of substances of various structures with molecular weight from 300 to 5000 Da ¹³⁻¹⁵. MMWS include substances of low and medium molecular weight, containing catabolic and anabolic pools and oligopeptides, having a molecular weight less than 10 kDa. Substances of low and medium molecular weight contain creatinine, urea, oligosaccharides, lactic acid, bilirubin, amino acids, cholesterol, lipid peroxidation products and other compounds. They are distributed in the blood between plasma proteins, micelles of different classes of lipoproteins and erythrocyte glycocalyx, capable of transporting these substances. Oligopeptides contain regulatory peptides (neurotensins, somatostatin, vasoactive intestinal peptide, enkephalins and other biologically active substances) and unregulatory peptides (products of proteolytic degradation of plasma and tissue proteins that enter the blood as a result of autolysis, ischemia, organ hypoxia and proteolysis processes) ¹⁶.

According to M.Ya. Malakhova ¹⁷, the catabolic pool of the MMWS which is detected at wavelengths ranges of 242 to 258 nm consist of protein catabolism products and low

molecular weight metabolites such as urea, creatinine, uric acid, purine metabolism products, as well as nucleotides and their derivatives, nucleoprotein metabolites. A significant increase in the number of catabolic products is one of the stages in endogenous intoxication syndrome development. Anabolic pool of the MMWS is detected at wavelength ranges of 258 - 298 nm. The group includes mainly fragments of protein molecules containing aromatic amino acids, metabolites of urea cycle, purine and pyrimidine, and their derivatives.

MMWS as markers of endogenous intoxication adequately reflect, on the one hand, the metabolic shifts, and on other, the intensity of protein catabolism, which is the main source of endogenous toxins¹⁶.

This paper will discuss the effect of monosodium glutamate (E 621) at "safe" (allowed) dose on the main markers of endogenous intoxication in rats.

Materials and Methods

Experimental studies were conducted on 72 nonlinear male white rats weighing 150-180 g, that were housed at 25±3°C and a humidity of 55±2%, under a 12 hours light and dark cycle. Water was available *ad libitum*. Our study and manipulations complied with the requirements of the Law of Ukraine "On the protection of animals against cruel treatment" (No. 1759-VI from 15.12.2009) and the international principles of the European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes.

The experimental study was approved by the Ethics Committee of I. Horbachevsky Ternopil State Medical University. MSG was purchased from Sigma-Aldrich (USA).

Laboratory animals were divided into 4 groups. The first group was administered MSG at a dose of 30 mg/kg body weight (corresponds dose 2 g per day in humans) for 14 days. The second group was administered MSG at a dose of 30 mg/kg body weight for 30 days. The third group was administered MSG at a dose of 30 mg/kg body weight for 60 days. The control group of animals was given normal saline.

Animal euthanasia was carried out by cardiac puncture under deep anaesthesia, in accordance with the requirements of the Animal Care Committee¹⁸. Urine was extracted from the bladder by needle aspiration (No 147-TSMU/2016).

Syndrome of endogenous intoxication was evaluated using measurements of low and medium molecular weight substances contents in blood plasma, red blood cell suspension and urine quantified by extraction-spectrophotometric method¹⁹. High molecular weight substances of blood plasma, erythrocytes, and urine were precipitated in 15% solution of trichloroacetic acid. Trichloroacetic extracts of blood plasma and red blood cells were measured by spectrophotometer SF-200 at wavelengths of 242, 254 and 282 nm, trichloroacetic extracts of urine - at wavelengths of 236, 254 and 282 nm. The obtained data are expressed in standard units of optical density (U). Using the data, the following indices were calculated to evaluate the intensity of endogenous intoxication¹⁵:

1. Ct – total contents of low and medium molecular weight substances in plasma:

$$Ct = (E242 + E254 + E282) \times 40;$$

2. Cc - the value of catabolic pool of low and medium molecular weight substances in plasma:

$$Cc = (E242 + E254) \times 12;$$

3. Pc% - catabolic pool of plasma:

$$Pc\% = Cc / Ct \times 100 \%;$$

4. ICP - intensity of catabolic processes in plasma:

$$ICP = (E242 + E254) / (E254 + E282);$$

5. K_1 - distribution rate of low and medium molecular weight substances between blood plasma proteins, and erythrocyte glycocalyx:

$$K_1 = (E242 + E254 + E282) \text{ of plasma} / (E242 + E254 + E282) \text{ of erythrocytes};$$

6. K_2 - elimination process condition, indicating the ability of kidneys to excrete endotoxemia products:

$$K_2 = (E236 + E254 + E282) \text{ of urine} / ((E242 + E254 + E282) \text{ of plasma} + (E242 + E254 + E282) \text{ of erythrocytes}).$$

The results were analyzed using Statistica 6.0 software and presented as mean with standard deviations, and the minimum and maximum values of ranges. To evaluate the distribution of the character together by sampling data we have used Lilliefors and Kolmogorov - Smirnov tests. Statistical significance was determined by the student's t-test or nonparametric Mann-Whitney criterion. A p-value of <0.05 was considered statistically significant.

Results

Analysis of (242-282) nm range wavelength spectrograms for low and medium molecular weight substances in plasma and red blood cells glycocalyx indicates significant difference between experimental and control groups. In the range of 242 nm in plasma of portal vein and inferior vena cava of animals administrated with MSG, the values of extinction increased, peaking after two months of MSG intake. This indicates the high contents of catabolic substances in the blood of these rats.

In ranges of 242 and 280 nm spectrograms of red blood cells showed displacement of spectral curves and higher optical density values. This suggests strain of detoxification reserves in the portal vein and inferior vena cava of the animals and increased contents of catabolic substances.

These results demonstrate that contents of low and medium molecular weight substances increased after two months of MSG intake, mainly in the blood plasma.

The pool of low and medium molecular weight substances form a marker group of the metabolic status of animals used to analyze the possible impact of the exposition to toxic substances. This is due to the fact that red blood cells bind and transport endogenous toxic components adsorbing them on their surface. We have found an increase in the contents of medium molecular weight fraction in red blood cells at 254 nm of (portal vein) and (inferior vena cava) cava after 14-day experiment, which then with further increased after one month. This increasing was in comparison to control as well as to the 1st experimental group (p<0.05). It should be noted, that after two months of MSG intake the average molecular weight contents was higher than in control animals (in Portal vein by 40 %, and in inferior v. cava by 43%) but did not significantly differ from that of the two experimental groups. We have found increase in the level of medium molecular weight substances detected at 254 nm in portal vein plasma starting at the 2nd week of the experiment, and after one month of MSG intake this marker not only in Portal vein, but also in inferior v. cava with an upward trend in the experimental group 3 (Table 1).

Table 1. Low and medium molecular weight substances contents in erythrocyte glycocalyx, blood plasma and urine of rats administrated with monosodium glutamate

Index		Control group	1 experimental group (14 days of MSG intake), U	2 experimental group (30 days of MSG intake), U	3 experimental group (60 days of MSG intake), U
Blood plasma					
E ₂₄₂	Portal vein	0.13±0.01 (0.09;0.18)	0.16±0.01 (0.11;0.26)	0.24±0.01; (0.19;0.28) p _{1,2} <0.001	0.26±0.01 (0.23;0.30)
	inferior v. cava	0.09±0.01 (0.06;0.16)	0.10±0.01 (0.11;0.24)	0.16±0.01; (0.11;0.21) p _{1,2} <0.001	0.22±0.01 (0.17;0.25)
E ₂₅₄	Portal vein	0.31±0.01 (0.22;0.37)	0.37±0.02 (0.27;0.42)	0.46±0.02; (0.34;0.53) p _{1,2} <0.001	0.51±0.02; (0.40;0.60) p _{1,2} <0.001
	inferior v. cava	0.26±0.02 (0.15;0.36)	0.28±0.01 (0.21;0.33)	0.35±0.01; (0.29;0.40) p _{1,2} <0.001	0.38±0.01; (0.33;0.44) p _{1,2} <0.001
E ₂₈₂	Portal vein	0.24±0.02 (0.13;0.31)	0.35±0.02 (0.28;0.46) p ₁ <0.001	0.38±0.01; (0.29;0.46) p ₁ <0.001	0.39±0.01; (0.33;0.45) p ₁ <0.001; p ₂ =0.03
	inferior v. cava	0.24±0.01 (0.12;0.32)	0.34±0.01 (0.29;0.42) p ₁ <0.001	0.39±0.01; (0.29;0.46) p ₁ <0.001; p ₂ =0.01	0.37±0.01 (0.33;0.42) p ₁ <0.05
Red blood cell suspension					
E ₂₄₂	Portal vein	0.21±0.01 (0.16;0.27)	0.26±0.01; (0.17;0.31) p ₁ =0.004	0.32±0.01; (0.25;0.35) p _{1,2} <0.001	0.36±0.01; (0.27;0.42) p _{1,2} <0.001; p ₃ =0.01
	inferior v. cava	0.18±0.01 (0.12;0.26)	0.21±0.01 (0.15;0.25)	0.30±0.01; (0.23;0.33) p _{1,2} <0.001	0.33±0.01; (0.29;0.41) p _{1,2} <0.001; p ₃ =0.01
E ₂₅₄	Portal vein	0.46±0.02 (0.32;0.52)	0.54±0.02; (0.43;0.60) p ₁ <0.001	0.60±0.01; (0.53;0.67) p ₁ <0.001; p ₂ =0.003	0.64±0.02; (0.55;0.76) p _{1,2} <0.001

Index		Control group	1 experimental group (14 days of MSG intake), U	2 experimental group (30 days of MSG intake), U	3 experimental group (60 days of MSG intake), U
	inferior v. cava	0.41±0.02 (0.31;0.50)	0.52±0.01; (0.44;0.60) p ₁ <0.001	0.58±0.01; (0.51;0.65) p ₁ <0.001; p ₂ =0.002	0.60±0.02; (0.47;0.66) p _{1,2} <0.001
E ₂₈₂	Portal vein	0.22±0.01 (0.12;0.31)	0.29±0.02; (0.21;0.40) p ₁ <0.002	0.34±0.01; (0.25;0.41) p ₁ <0.001; p ₂ =0.01	0.39±0.01; (0.34;0.45) p _{1,2} <0.001; p ₃ =0.01
	inferior v. cava	0.18±0.02 (0.10;0.29)	0.21±0.01 (0.15;0.29)	0.31±0.02; (0.22;0.40) p _{1,2} <0.001	0.37±0.01; (0.33;0.42) p _{1,2} <0.001; p ₃ =0.002
Urine					
E ₂₃₆		0.47±0.02 (0.27;0.58)	0.49±0.02 (0.31;0.60)	0.48±0.01 (0.26;0.56)	0.47±0.01 (0.35;0.46)
E ₂₅₄		0.45±0.02 (0.36;0.58)	0.44±0.01 (0.40;0.56)	0.43±0.01 (0.40;0.53)	0.42±0.01 (0.38;0.53)
E ₂₈₂		0.41±0.03 (0.34;0.52)	0.42±0.02 (0.39;0.53)	0.43±0.02 (0.40;0.47)	0.41±0.01 (0.39;0.46)

Notes: p₁ – significant difference compared to the control group; p₂ – significant difference compared to the 1st experimental group; p₃ – significant difference compared to the 2nd experimental group.

The levels of substances detected at E₂₈₀ increased in red blood cells and portal vein plasma of the 1st experimental group. After one month of the experiment, the medium molecular weight content at 280 nm in red blood cells of Portal vein and inferior v. cava steadily increased up to two months of the experiment. In blood plasma of Portal vein this parameter remained practically unchanged during the study, while in the inferior v. cava, it was the highest at one month of the experiment. Low molecular weight substances content increased in red blood cells of Portal vein after two weeks of the experiment, in red blood cells and plasma of Portal vein and inferior v. cava

after one month, and then remained virtually unchanged until the end of the experiment.

One of the most informative signs of adaptive reaction to stress exposition was the change in marker K₁, which increased by 13% after one month of the experiment in red blood cells of Portal vein compared to the control values (p < 0.05). Against this background, the values of K₂ marker; which are used for comprehensive assessment of endotoxemia on the body, showed gradual decline. This decline corresponded to the duration of MSG intake and indicated the reduced ability of kidneys to excrete toxic products (Table 2).

Table 2. Indices of endogenous intoxication in rats administrated with monosodium glutamate

Index		Control group	1 experimental group (14 days of MSG intake), U	2 experimental group (30 days of MSG intake), U	3 experimental group (60 days of MSG intake), U
Ct	Portal vein	25.5±0.92	33.86±0.93; p ₁ <0.001	41.60±0.74; p _{1,2} <0.001	45.49±0.82; p _{1,2} <0.001; p ₃ =0.002
	inferior v. cava	23.12±1.11	28.83±0.82; p ₁ <0.001	36.07±0.76; p _{1,2} <0.001	38.74±0.65; p _{1,2} <0.001; p ₃ =0.01
Cc	Portal vein	4.96±0.18	6.12±0.15; p ₁ <0.001	8.08±0.20; p _{1,2} <0.001	9.05±0.20; p _{1,2,3} <0.001
	inferior v. cava	4.23±0.28	4.65±0.19	6.27±0.16; p _{1,2} <0.001	7.28±0.49; p _{1,2,3} <0.001
Pc%	Portal vein	19.49±0.55	18.16±0.41; p ₁ =0.04	19.41±0.31; p ₂ =0.01	19.86±0.21; p ₂ <0.001
	inferior v. cava	18.27±0.72	16.15±0.40; p ₁ <0.001	17.44±0.31; p ₂ =0.008	18.76±0.19; p _{2,3} <0.001
ICP	Portal vein	0.80±0.04	0.75±0.02	0.84±0.02; p ₂ <0.001	0.85±0.01; p ₂ <0.001
	inferior v. cava	0.72±0.02	0.61±0.02; p ₁ <0.001	0.72±0.02; p ₂ <0.001	0.83±0.03; p _{1,2,3} <0.001
K ₁	Portal vein	0.75±0.03	0.79±0.04	0.85±0.01; p ₁ <0.001	0.85±0.02; p ₁ <0.001
	inferior v. cava	0.73±0.02	0.75±0.02	0.76±0.02	0.74±0.02
K ₂	Portal vein	0.89±0.03	0.72±0.02; p ₁ <0.001	0.56±0.02; p _{1,2} <0.001	0.53±0.01; p _{1,2} <0.001; p ₃ =0.01
	inferior v. cava	0.99±0.04	0.82±0.02; p ₁ <0.001	0.62±0.01; p _{1,2} <0.001	0.56±0.01; p _{1,2,3} <0.001

Notes: p₁ – significant difference compared to the control group; p₂ – significant difference compared to the 1st experimental group; p₃ – significant difference compared to the 2nd experimental group.

Discussion

Endogenous intoxication syndrome is characterized by metabolic, morphological and

functional disorders of various organs and systems and occurs in response to various factors of the external and internal

environment, as a result of toxic substances accumulation in tissues and biological fluids - an excess of normal and impaired metabolism products or cellular response^{15,17}. Today, the endogenous intoxication syndrome concept is widespread as a process within the syndrome of a systemic inflammatory response²⁰. It is known that the overstrain of adaptation mechanisms, the breakdown of compensation, the imbalance of reactions at the biomolecular level lead to structural and metabolic changes that cause the development of homeostasis disorders. However, many substances in this case may acquire the properties of endotoxins, not being such in the physiological conditions. The significance of this concept is relevant, since the clinical picture of metabolic disorders does not manifest itself clearly in the early stages of its development. Syndromic diagnosis of impaired metabolism, as a rule, lags behind the events of developing pathological processes at the cell-biochemical level²¹.

Among a wide range of metabolites that have the ability to demonstrate a toxic effect, a special place is occupied by a pool of middle molecular weight substances, the dynamics of which reflect the severity of endogenous intoxication and correlate with main clinical and laboratory prognostic criteria of metabolic disorders²²⁻²³.

A significant feature of MMWS is their high biological activity. They have neurotoxic activity, inhibit protein synthesis, promote hemolysis of erythrocytes, inhibit erythropoiesis and enzymatic activity, and cause a state of secondary immunosuppression¹³. Also MMWS are capable of blocking cell receptors, binding to the active centres of the albumin molecule, competing with regulatory peptides, and thus disrupting the process of humoral regulation²⁴. The accumulation of

MMWS and the violation of their distribution between plasma and erythrocytes, as well as the violation of their excretion by the kidneys, caused by various etiological factors, lead to the development of endogenous intoxication¹⁶.

Our results indicate shift of the markers of endogenous intoxication syndrome towards mainly catabolic substances. The results obtained after two weeks of the experiment correspond with phase of partial compensation, characterized by increased concentrations of low and middle molecular weight substances in red blood cells and plasma. After one month and up to two months of the experiment, the predominantly catabolic markers of endogenous intoxication continue to increase in erythrocytes and plasma, indicating a shift to the phase of partial decompensation of the detoxification systems.

Thus, even the minimal intake of exogenous toxic substances, such as MSG in the blood, triggers endogenous intoxication. Metabolic products, which are usually removed from the body under normal metabolic processes, can cause intoxication if over-produced, if their elimination from the blood circulation is insufficiently effective, or if both of these events occur simultaneously²⁵. We suggest that two months of MSG administration at a dose of 30 mg/kg body weight causes overproduction of toxic metabolites and disrupted elimination process resulting in endogenous intoxication syndrome development.

One of the mechanisms through which MSG causes overproduction of toxic metabolites is its possibility dissociate easily to release free glutamate. The diminution of glutamate produces ammonium ion (NH_4^+) that could be toxic unless detoxified in the liver via the reactions of the urea cycle¹². The possible ammonium ion overload that can cause damage

to the liver is the possible reason to the violation of detoxification processes. In addition, it is known that the liver is a place for the serum albumins synthesis, which is the main transport link of the body's physiological detoxification system. On the other hand, there are the data demonstrated that MSG administration increased oxidative stress in the liver and kidney of rats ³. In case of sodium glutamate administration mitochondrial respiratory chain is the main source of reactive oxygen species (ROS). In addition, the increase in extracellular glutamate levels increases production of hydroxyl radicals. Research of Sharma A. showed increased of α -ketoglutarate dehydrogenase activity, which can activate oxygen and formation of superoxide anion and hydrogen peroxide ²⁶. However, most scientists associate the occurrence of oxidative stress with glutamate receptors ²⁷⁻²⁸. According to Payenok O.S. study ²⁹, the activation of lipid peroxidation processes is an important pathophysiological mechanism for the

development of endogenous intoxication. Excessive lipoperoxidation is accompanied by the accumulation of peroxide oxidation products and the depletion of antioxidant system reserves, causing hyperenzymemia and accumulation of toxic substances ³⁰⁻³¹.

Conclusion

Our results indicate that administration of MSG at a dose of 30 mg/kg body weight was associated with development of excessive contents of low and middle molecular weight substances with reduced ability of kidneys to excrete toxic products.

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التأثير السام للغلوتامات أحادية الصوديوم في الجرذان

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الملخص

الخلفية: أكثر من 2500 من الإضافات عن قصد إلى الغذاء من أجل الاحتفاظ ببعض الخصائص أو تمديد فترة الصلاحية. واحدة من الإضافات الغذائية الأكثر شيوعاً في أوكرانيا وأوروبا هي الغلوتامات أحادية الصوديوم. الآثار الصحية السلبية المحتملة من الغلوتامات أحادية الصوديوم تدفعنا إلى التشكيك في سلامة استعمالها الواسعة الانتشار.

الأهداف: في هذه الدراسة حددنا تأثير الغلوتامات أحادية الصوديوم (E621) على العلامات الرئيسية للتسمم الداخلي في الفئران. المواد والأساليب: أجريت دراسات تجريبية على 72 من الفئران البيضاء (غير الخطية، ذكور) التي تزن 150-180 جم. الحيوانات التجريبية قد تم إعطاؤها الغلوتامات أحادية الصوديوم بجرعة 30 جم / كجم من وزن الجسم (تتجاوب مع الجرعة 2 غرام في اليوم لدى البشر) لمدة 7 ، 14 و 30 يوماً. أعطيت مجموعة المرجعية محلول الماء والملح المعياري (متلازمة داخلية) (تم تقييم متلازمة التسمم الداخلي) تم تقييم التسمم باستخدام قياسات للوزن الجزيئي المنخفض والمتوسط والعالي المواد في بلازما الدم وتعليق خلايا الدم الحمراء والبول. النتائج: تشير النتائج إلى تحول من علامات متلازمة التسمم إلى مواد مسببة للتآكل. النتائج التي تم الحصول عليها بعد أسبوع من التجربة تتوافق مع مرحلة التعويض الجزئي ، تتميز بزيادة تركيزات منخفضة ومتوسطة.

المواد: الوزن الجزيئي في خلايا الدم الحمراء والبلازما. بعد أسبوعين وتصل إلى شهر واحد. التجربة: العلامات التي تسبب التآكل في الغالب من التسمم الذاتي. تستمر في زيادة كريات الدم الحمراء والبلازما ، مما يشير إلى تحول إلى مرحلة من عدم التعويض الجزئي لنظم وأجهزة إزالة السموم. إعطاء الغلوتامات أحادية الصوديوم بجرعة 30 جم / كجم من وزن الجسم وارتباط الوزن مع تطوير محتويات مفرطة من الجزيئات المنخفضة والمتوسطة الوزن مع انخفاض قدرة الكلى لإفراز المنتجات السامة.

الكلمات الدالة: الغلوتامات أحادية الصوديوم ، التسمم الداخلي ، المؤشرات ، الفئران.