тически немодифицированного соевого шрота. Животных I группы (I опытная) скармливали аналогичным рационом, но соевый шрот заменяли на генетически модифицированный. Животных II группы (II опытная) кормили только генетически модифицированным шротом.

Определение наличия генетически модифицированных источников, ГМО в пищевых и кормовых продуктах осуществлялось с использованием полимеразной цепной реакции. Проведены морфометрическое исследование полученного материала и биометрическая обработка данных.

Выявлено, что поедание животными кормов, приготовленных на основе генетически модифицированных растений, не влияет на репродуктивные функции родительского поколения; однако происходит торможение скорости роста и процесса формирования половых желез потомков первого и, особенно, второго поколения; у второго поколения потомков, получающих только генетически модифицированный соевый шрот, отмечалась высокая смертность и развитие дефектных репродуктивных качеств.

რეზიუმე

რეპროდუქციული სისტემის განვითარების თავისებურებანი გენეტიკურად მოდიფიცირებული წყაროების გამოყენების პირობებში (ექსპერიმენტული კვლევა)

[1]ნ.ხარისოვა, [2]ლ.სმირნოვა, [3]ა.კუზმინი, [1]გ.რისპაევა, [1]გ.ლეპესბაევა

[1]ყარაგანდის სამედიცინო უნივერსიტეტი, მორფოლოგიისა და ფიზიოლოგიის კათედრა, ყარაგანდა, ყაზახეთი; კოსტრომის ნ.ნეკრასოვის სახ. სახელმწიფო უნივერსიტეტი, [2]ფიზიკური კულტურისა და სპორტის კათედრა; [3]ბიოლოგიისა და ეკოლოგიის კათედრა, რუსეთი

მოლეკულური ბიოლოგიის და გენეტიკური ინჟინერიის ელვისებური განვითარება ხელს უწყობს გარკვეული თვისებების მქონე მცენარეების შექმნას საკმაოდ მოკლე დროში. გენმოდიფიცირებული ორგანიზმების კვლევის ერთ-ერთ ასპექტს წარმოადგენს მათი გავლენის შესწავლა ადამიანზე, ცხოველებსა და გარემოზე.

კვლევის მიზანს წარმოადგენდა თაგვების რეპროდუქციული სისტემის მორფოფუნქციური მაჩვენებლების ცვლილებების და რეპროდუქციულ სისტემაზე გენმოდიფიცირებული ორგანიზმების გავლენის შეფასება.

კვლევის ობიექტს წარმოადგენდა ლაბორატორიული 40 გ მასის, 20 დღის თაგვები. გამოიყო სამი ჯგუფი, თითოეულში – 10 ცხოველი (7 მდედრი, 3 მამრი). III (საკონტროლო) ჯგუფის ცხოველები იღებდნენ სტანდარტულ რაციონს გენეტიკურად არამოდიფიცირებული სოიოს 20%-იანი შემცველობით. I ჯგუფის (I საცდელი) ცხოველებს კვებავდნენ ანალოგიური რაციონით, ხოლო სოიო ჩანაცვლებული იყო გენმოდიფიცირებულით. II ჯგუფის (II საცდელი) ცხოველებს კვებავდნენ მხოლოდ გენეტიკურად მოდიფიცირებული მასალით.

გენეტიკურად მოდიფიცირებული წყაროების და გენეტიკურად მოდიფიცირებული ორგანიზმების არსებობა განისაზღვრა პოლიმერაზულ-ჯაჭვური რეაქციის გამოყენებით. ჩატარდა მიღებული მასალის მორფომეტრიული კვლევა და მონაცემების ბიომეტრიული დამუშავება.

დადგენილია, რომ ცხოველების მიერ გენეტიკურად მოდიფიცირებული მცენარეების ბაზაზე მომზადებული საკვების მიღება არ მოქმედებს მშობლების თაობის რეპროდუქციულ ფუნქციაზე; თუმცა, აღინიშნება პირველი და, განსაკუთრებით, მეორე მემკვიდრე თაობის სასქესო ჯირკვლების ზრდის და ფორმირების სიჩქარის შენელება; შთამომავლობის მეორე თაობას, რომელიც მხოლოდ გენეტიკურად მოდიფიცრებული სოიოს შროტით იკვებებოდა, აღენიშნებოდა რეპროდუქციული თვისებების დეფექტები და მაღალი სიკვდილობა.

RADON HORMESIS IN EPILEPTIC PATHOGENESIS AND PREDICTORS OF OXIDATIVE STRESS

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Radon is a radioactive element and therefore has radiant properties. Under the influence of radiation it is possible to change the vitality of the organism, so because of these characteristics, it is also used in medicine, for example: radiation therapy is used to prevent hypertension, age-related changes in the brain and to treat or prevent other diseases [1]. It is known that during radon spa therapy there is an increase in adrenaline in plasma [2,3] and these properties are used in medicine. Nevertheless, radon in high concentration conditions can harm the body and cause irreversible changes [4,5], for example, radon has been named the most common cause of lung cancer in non-smokers [6].

Resort Tskaltubo has long been used for improving health in various disorders. It is located on 70 km from Black sea, altitude 90-120 meter from sea level, in western Georgia and is regarded as chloride-hydrocarbonate-sulfate-magnesium-calcium-sodium water. It is characterized as stable and unchangeable by time.

In the 70th of the 20th century important microelements such as iodine, bromine, magnesium, lithium, zinc, strontium and copper were detected in Tskaltubo mineral water. Analysis also showed that springs contain low concentration of radioactive radon [40 to 100 Becquerel (Bq)] that could be considered as one of the cause of healing effect (Khazaradze et al, 2001, Gelashvili et al, 2001). It should be noted that the action of radon, as well as other radioactive elements, is characterized by the activation of the phenomenon of Hormesis in the body which is characterized by induction of biopositive responses such as increases in immunity and antioxidants by low-dose radiation [9].

Nowadays radon therapeutic spas are used for treating various inflammatory rheumatic diseases such as ankylosing spondylitis, chronic polyarthritis, fibromyalgia, scleroderma, rheumatoid arthritis, as well as in some neurological condition (chronic neuralgia) and respiratory diseases (bronchial asthma, chronic bronchitis) [10]. Radon therapy appears to aid in the recovery of the immune system. This is the case with bronchial asthma and even in the treatment of atopic asthma [11] where long-lasting effect on the immune system has been demonstrated.

Because radon has a positive effect on pain, the immune system, its analgesic, reparative-regenerative, immunomodulative, stress-inductive action is used in medicine. Radon influences the concentration of amino acids and neurotransmitters involved in excitation and inhibition at the cellular level, as confirmed in our previous studies. Studies in aggressive rats have shown that short-term (7 days) inhalation of radon alters the concentration of amino acids involved in excitation and inhibition in the animal brain, namely a decrease in glutamic acid and GABA concentrations and a significant decrease in noradrenalin concentration (Nikolaishvili, 2006). These changes formed the basis for a change in the behavior of an aggressive animal when, after inhaling radon, the aggressive animal lost its aggression (aggressive traits) and became non-aggressive. Based on the results of this study, we can assume that inhalation of radon may be an activator or inhibitor of the activity of some neurotransmitters acting on physiological processes. Accordingly, the present study makes it possible to shed light and clearly indicate the possible benefit of therapeutic use of radon on brain functioning.

Radon Hormesis (R-Ho) could have significant beneficial effect in patients with epilepsy that is one of the most common chronic neurological diseases. Epilepsy affects more than 50 million people in the world (1% of general population) [13] and there are leaving more than 35 000 persons with active epilepsy in Georgia (8.8 per 1000) [14]. Epilepsy is more often revealed among children and in aging people.

Epilepsy has numerous causes, each reflecting brain dysfunction, but the main symptom of the disease is recurrent epileptic seizures, caused by deterioration of inhibitory and excitatory balance in the brain cortex.

Despite the multitude of causes, the exact cause that led to the development of epilepsy has finally been explored. Some forms of epilepsy are accompanied by an increase in arousal and some by a decrease in retention.

Numerous experimental and clinical observations have shown that oxidative stress (OS) plays a major role in the development of epilepsy. The epileptic seizure itself is caused by OS and in the background of the hypermetabolic state has an intense production of reactive oxygen species (ROS) in the brain. The reason for this is called a sharp change in energy metabolism. ROS and other markers of oxidative stress occur in the brain after epileptic seizures. These changes have been observed in animal models of epilepsy as well as in patients with epilepsy.

The most important result of the accumulation of ROS in epilepsy is the activation of excessive and uncontrolled lipid peroxidation (LPO) processes in the brain tissues. The intensification of LPO processes leads to changes in the structural organization of the membrane (phospholipid composition, microviscosity and ion permeability), as well as the rheological properties of the blood deteriorate as a result of peroxidation reactions.

Based on all the above, we decided to study the effect of radon inhalation on oxidative stress, namely changes in oxidative markers when animals were exposed to small doses of radon short-term (5 days) inhalation.

Material and methods. For our experiment, we used 24 month the Krushinsky-Molodkina (KM) line male rats. They are predisposed to audiogenic epilepsy (seizures in response to a strong sound). Rapid (5-7 sec.) development of clonic-tonic seizures and the development of postictal catalepsy are characteristic of KM rats (Semiok hina A.F, et.all. 1996, Inna S.Midzyanovskaya 2004)**.**

For induction of epileptic seizures, we used an audiogenic signal before the study to which the experimental animal responded with cramps. In particular, the trigger caused the development of myoclonic seizures with "limbic" localization. Long-term (15 min) exposure of KM rats to the action of sound according to a special scheme with alternating 10 s periods of strong and weak sound causes cerebral circulation disorders in them, externally manifested in the form of paresis and paralysis of the limbs (Feodorova I.B 2005, Fadiukova O.E. 2013, Kapanadze A.P. 2013, Poletova 2017). On the 5th days assessment of epileptic seizure with trigger - sound in BK rats was performed.

Radon measurement: in Tskaltubo spa center, were natural mineral water is used, we measured Radon`s radioactivity in water. The radioactivity of Radon was 37 becquerel (bk) in 1 m3 (37 bk/m3).

Radon inhalations procedure: we placed 10 experimental animals (KM rats) in Tskaltubo mineral water spa`s sauna (experimental group). Mineral water temperature was $36^0$C , Humidity 90%. Control group 10 KM rats was placed in another spa center`s sauna, were 36^0C mineral water (without radon) was delivered via inhalation. Humidity in this spa center`s experimental room was 90%. None (experimental and control group of rats) of the animals took a bath, they were just in two different saunas and living in the same conditions. Inhalation was taken through the nose, for 5 minutes, once a day, in conditions of high humidity (about 90%) during 5 days. After each procedure of inhalation, the rats were placed in a vivarium and given food and water.

For study the physiological changes caused by inhalation of Tskaltubo water on oxidative level, which prevents the development of brain disorders associated with peroxidation reactions, we measured the free radicals concentrations (d-ROM) - reactive oxygen metabolites in the blood plasma of rats, using a photometric test, measured the concentration of hydroperoxides (ROOH) in the brain tissue, which gives us a pro-oxidant status of the tissue. Hydroperoxides, also called Reactive Oxygen Metabolites (ROM), are formed during an oxidative attack when Reactive Oxygen Species (ROS) react with various organic substrates (e.g. carbohydrates, lipids, amino acids, proteins, nucleotides, etc.).

To assess the antioxidant capacity of plasma, we used the PAT (Antioxidant Concentration Test) by measuring ferric reducing ability and to evaluate the effectiveness of antioxidants, we determined the OSI (Oxidative Stress Index) and the OBRI (Oxidation Balance Status).

All named measurements were provided by means of Photometric Analytical System FRAS5 (H&D, Parma, Italy).

Table 1. The effect of Radon inhalation on the epileptic seizure in rats.

	Hidden period (sec.)	The first wild jogging duration (sec.)	Pause (min.)	The duration of the second wild jogging (sec.)	Duration of tonic-clonic seizures (sec.)
Before exposure to radon (p<0.05)	11±1.1	10±1.1	5±0.3	58±1.7	78±1.9
3 days after radon exposure p<0.05)	23±2.1	6±0.7	11±1.1	37±1.3	0
5 days after radon exposure p<0.05)	16±1.4	3±0.1	32±2.2	39±1.1	52±1.3

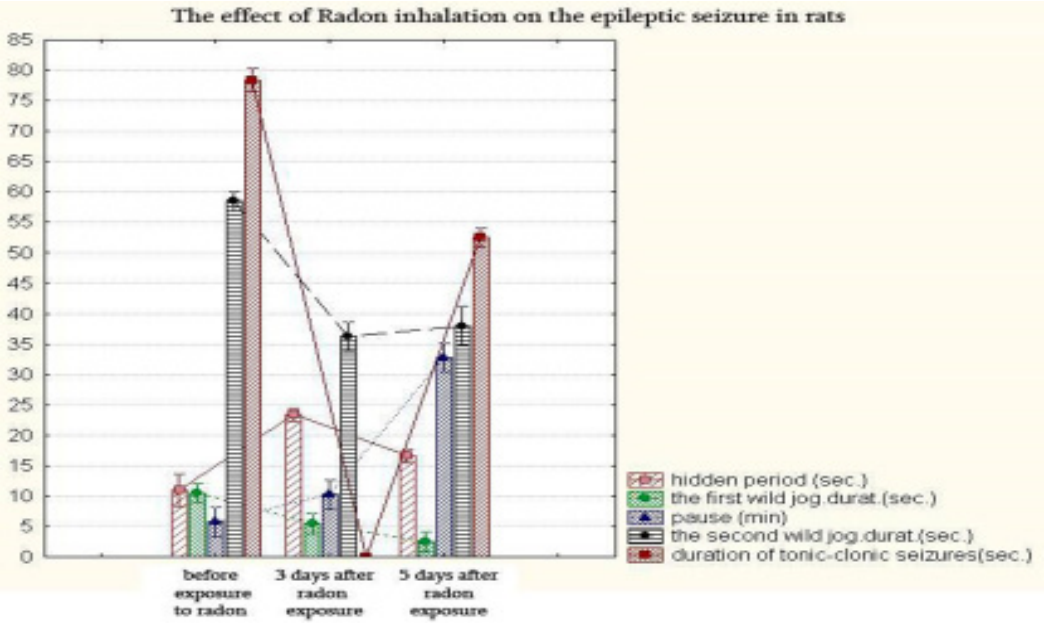

Fig.1. The effect of Radon inhalation on the epileptic seizure in rats.

Results and duscussion. *Epileptic seizure.* After completion of the inhalation course, the research and control group of experimental animals were examined on the 3rd and 5th day after completion of the inhalation course.

On the 3rd and 5th day after low doses (37 3700 Bq/m^3) Radon inhalation hidden period before seizures and pauses between seizures were significantly increased (p<0.05) in Radon exposure group, then in control. The latent period before radon inhalation was 11±1.1 s. On the 3rd and 5th day after inhalation, the latent period increased to 23±2.1 and 16±1.4, respectively.

Duration of first and second wild jumping after trigger was decreased in the Radon inhalation group of rats (p<0.05). In all groups, wild jogging started immediately after receiving the audio signal (call), but the first wild jogging duration was reduced from 10±1.1sec., (Control group) to 6±0.7 on the third day after radon inhalation and even longer on the fifth days (3 ± 0.1 sec.). After receiving the audio signal the duration in second wild jogging was reduced from 58±1.7 seconds to 37±1.3 on the third day after radon inhalation and 39±1.1 seconds on the 5th day after inhalation. No generalized audiogenic tonic-clonic seizure was not observed in the Radon inhalation group on the day 3, whereas the duration of seizures in the control group was 78±1.9 seconds, while on the 5th day after inhalation it was 52±1.3 seconds.

As can be seen from the table, the effect of radon causes an increase in the latent period on days 3 and 5 after inhalation. The duration of the first wild jog was reduced by epilepsy in rats on the third day after exposure to radon (6±0.7 sec), but the decrease in this parameter became even more noticeable on day 5 (3±0.1 sec) which was also statistically significant (p<0.05). As for the duration of the second wild jog, on the third day was 37 seconds and on the fifth day 39 seconds. No changes were observed in the data of the control group of rats. There was also no generalized audiogenic tonic-clonic seizure in the radon inhalation group on day 3 and it was equal to 0, while on day 5 it was to the control (78±1.9 seconds) was reduced to 52±1.3 seconds. Which is statistically significant (p<0.05).

Oxidative stress. From the data presented in Table 2 we can see the following. Study of dROM in genetically epileptic Krushinsky-Molodkina rats before exposure to radon showed very high oxidation status (521±3.67), and on the 5th day after exposure to radon the level of oxidative stress was 381±2.95 which is statistically significant (p<0.05). The hematological concentration of antioxidants (PAT plasma with antioxidant fragments) was at the edge before being taken to Tskaltubo in epileptic rats (2091±3.95), and as a result of inhalation of radon became (2763±5.85) – statistically it is reliable (p<0.05). OBRI (Oxidation Balance Risk Index): if it was before radon inhalation 1.95± 0.3 - dangerously high for the organism, after inhalation it was recorded as high, but this data was not dangerous for the organism (1.6±0.2,). The difference between them is statistically significant as well (p<0.05). OSI - The correlation of total oxidative status with the total antioxidant status used to determine the Global Redox status index in epileptic rats was 142± 2.3 before radon inhalation, above the critical situation, and after inhalation it was halved to 111±2.3. From the above mentioned we can conclude that a positive result was obtained when inhaling radon.

Table 2. Oxidative stress in epileptic rats

Epileptic rats	D-ROMs FAST Ucarr.	PAT	OBRI	OSI REDOX
Control	521±3.67 Free radicals, very high	2763±5.85 Antioxidants There is a deficit	1.950±0.3 Oxidative status is at a dangerous level in relation to cholesterol	142±2.3 Oxidative status index is on the critical edge
Experiment	381±2.95 Is average ($p<0.05$).	2091±3.95 Average level	1,6±0.2 High	111±2.3 The body is on alert to protect itself

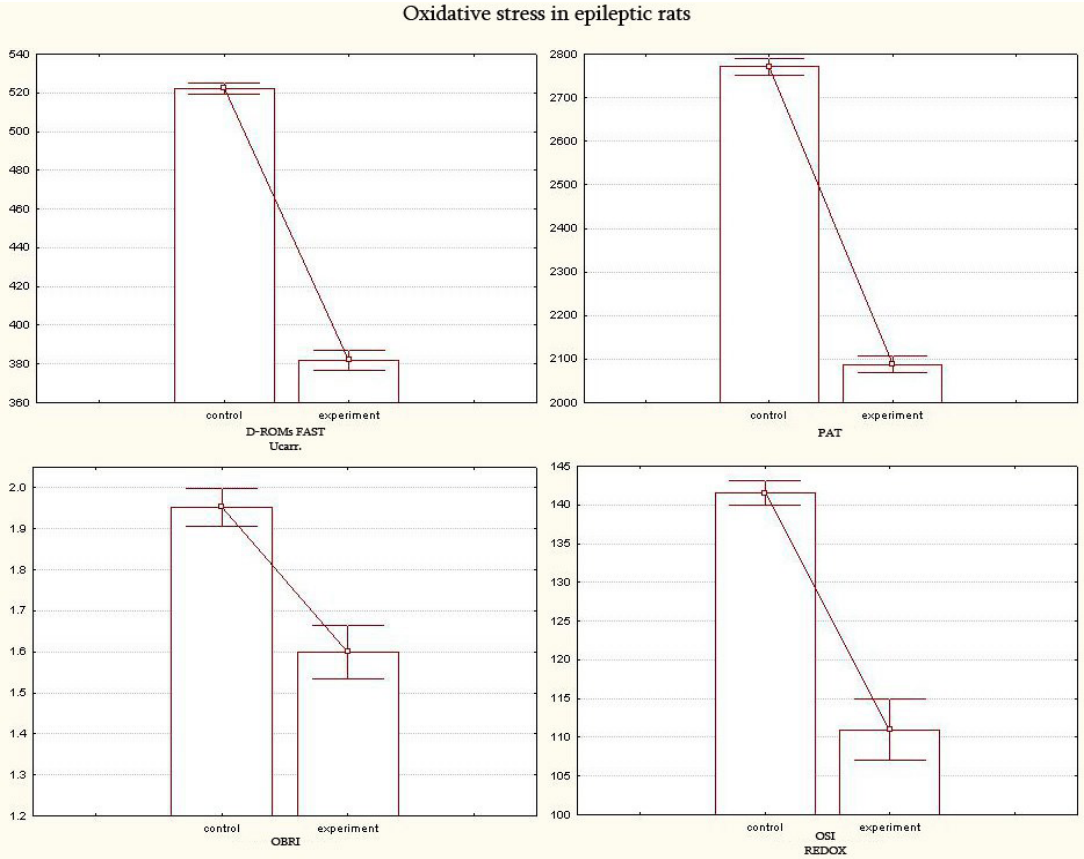

Fig.2 Oxidative stress in epileptic rats

Numerous experimental studies have been provided in animal models to prove the positive effect of radioactive radon on various disorders. In recent previous experimental studies, they were performed in rabbits to investigate the curative effects of radon hormesis the changes of levels the lipid peroxide, the oxidative stress's formative Nitric Oxide (NO) and the indicators of membrane conductivity of different organs were performed [15]. The data of this studies indicate that the effect of radon hormesis (R-Ho) happens by the activation of NO and formation of hydrogen peroxide (H_2O_2) that activate of excessive NO production in microphages under the effect of interferon gamma and beta (INF-γβ) [16]. As a result the increased production of NO activates peripheral microcirculation and central hemodynamic. In this reason NO can be considered as an autocrinic homeostatic modulator as well.

It is established that macrophage-killers are the important source of NO. By NO activation they suppress DNA synthesis of tumor cells and suppression of new tumor cells, anti-inflammatory, desensibility and sedative mechanisms. NO effects on activation of DOFA, DOFA-cines and DOFA-amino formation in blood; it is involved in regulation of the Na^+, K^+ and Ca^{++} ion changes, and play one of the main role in suppression of specific autoimmune and in activation of non-specific immune systems of the body.

In our case the reduction in oxidative stress is seen as early as the 5th day after exposure to radon inhalation due to NO activation, NO can be considered as an important neurotransmitter that can participate in synaptic transmission as classical, from presynaptic to postsynaptic neuron [17] and retrograde, lso indirectly by acting on glial cells or surrounding neurons. NO protects the brain from ischemic and neurotoxic stroke, controls the oscillatory activity of neurons [18], nitric oxide synthesis is often considered as a protective mechanism against the cytotoxic action of phagocytes, since NO inhibits the activation of the neutrophil [19] and NADPH oxidase activity, reduces xanthine oxidase activity, decreases AFK products [20]. At the same time, it should be noted that NO is involved in the development of the inflammatory process and its effect on the functional state of phagocytes can be modulated and changed over time. Therefore, the protective or cytotoxic action of NO is also characteristic of certain cells and tissues.

Experimental models of epilepsy show that increased oxidative stress (increased oxidation of lipids and proteins) is accompanied by the development of seizures [21]. Focal or first generalized epileptic activity in animals is followed by activation of LPO in the field of hyperactivity. At the same time, elevated LPO levels were observed in the blood of animals. The introduction of antioxidants into animals slowed the development of epilepsy and reduced its intensity. In-vitro experiments have shown that anticonvulsants can inhibit LPO in the brain membrane. The most pronounced changes in LPO product content are experienced by patients with prolonged epilepsy, generalized seizures, and profound personality changes.

M.K. Pandey et al. conducted studies (2012) show that MDA levels were significantly higher in the group of patients with epilepsy who had symptoms of mental disorders associated with the disease (psychosis and depression) [22]. The study determined the content of malonic dialdehyde (MDA), which is one of the final products of LPO. Thus, the severity of oxidative stress was significantly higher in patients with epilepsy who had mental disorders associated with this disease.

During seizures, there is an increase in the oxidation of free radicals, which in turn is accompanied by a decrease in the activity of the brain's antioxidant defense system: both enzymatic and non-enzymatic parts. High levels of LPO and decreased activity of major antioxidant enzymes - SOD and GPO - have been identified in the blood of patients with various forms of epilepsy [23].

Studies by E. Ben-Menachem (2000) have shown that erythrocyte SOD1 activity is significantly lower in patients with progressive myoclonic epilepsy than in healthy individuals. Decreased SOD1 activity also occurs in the cerebrospinal fluid of patients with epilepsy, especially in the disease-resistant group compared with patients with curable forms of epilepsy and in the control group of healthy individuals. The authors believe that decreased SOD1 activity is associated with recurrent seizures, and that SOD1 deficiency in cerebrospinal fluid may be a predictor of drug-resistant epilepsy [24].

In addition to the above, the convulsive state is characterized by anomalous Na + and K + metabolism. Accumulation of ammonia also occurs at this time, which is associated with the intensification of deamination reactions; All of this leads to depolarization of cell membrane shells, lowering of the excitability threshold, and a new series of seizures. In the first minutes of seizures, neurotransmitters are released, accompanied by a change in the level of secondary messengers, which is reflected in the activity of metabotropic receptors. Activation of adrenoceptors even causes an increase in cyclic adenosine monophosphate.

Excretion of excitatory amino acids (mainly glutamate) is known to increase during seizures. At this time, neuronal activity increases and intracellular calcium levels increase, which is associated with the formation of ROS [32]. Disruption of calcium metabolism is associated with a sharp increase in cell cytosol and leads to the activation of Ca^{++}-dependent enzymes. The formation of ROS is accompanied by the synthesis of prostaglandins from arachidonic acid. This in turn enhances free radical reactions that ultimately lead to membrane destruction.

Thus, numerous experimental data and individual clinical observations indicate the pathogenic role of OS in epilepsy, which is associated with disruption of the structural, hematoencephalic barrier integrity of the cell membrane of neurons, oxidative destruction of nucleic acids and radon exposure regulates oxidative stress, the clinical manifestation of which may be expressed by a reduction in epileptic seizures, and which has also occurred in the results of our study.

According to our research, the use of radon inhalation in experimental animals, particularly in genetically determined rats with epileptic seizures, altered all parameters of the epileptic seizure development picture, namely the hidden period, the first and second wild jog duration after the audiogenic signal. On the third day, no response to the audiogenic signal was observed at all, and not even a single episode of tonic-clonic seizures. All mentioned suggests that radon inhalation could be used to treat epilepsy.

According to the International Classification of Epileptic Seizures, we are talking about focal seizures and in our experiment, the effectiveness of Tskaltubo water has been confirmed in the case of focal epileptic seizures. As for generalized epilepsy, the impact of radon hormone on these types of seizures is still unclear, which requires additional scientific studies.

The impact of radon inhalation on seizures of brainstem epilepsy models is particularly important because mechanisms of prolonged bilateral (formerly generalized) seizures in humans are considered to be erased/included in brainstem structures.

Presented study is the first precedent of attempt R-Ho trough inhalation for treatment of epileptic seizures in animal models with further translation to clinical study in humans through pilot phase II study. More profound and scientifically systematized approach is needed to determine uniqueness of Tskaltubo water springs, investigation the mechanisms of radon effects on the excitatory and inhibitory functioning of CNS and further clinical studies to establish its effectiveness on humans.

Conclusion. To clarify the mechanism of radon`s action on antioxidative processes, future research is required, but based on the results of the experiment we can conclude that:

Studies in experimental animals have shown that inhalation of Tskaltubo water develops a hormesis that regulates oxidative processes in the brain by activating antioxidants, which is reflected in the reduction of existing epileptic convulsions.

Inhalation of Tskaltubo water may be considered as a method of treatment with anticonvulsant effect confirmed by experimental studies.

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SUMMARY

RADON HORMESIS IN EPILEPTIC PATHOGENESIS AND PREDICTORS OF OXIDATIVE STRESS

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Numerous experimental and clinical studies have shown that oxidative stress (OS) plays an important role in the development of epilepsy. The epileptic seizures themselves are caused by OS and, under conditions of a hypermetabolic state, cause an intensive production of reactive oxygen species (ROS) in the brain. The reason for this is called a change in the energy balance in the brain. According to the literature, ROS and other markers of oxidative stress were observed in the brain after seizures. Based on the above data, the aim of our study was to study a short-term 5-day (5 min) inhalation of radonized water from Tskhaltubo using an epileptic model of the Krushinsky-Molotkin line in rats and to identify changes in markers of oxidative stress in rats. Predictors of oxidative stress were studied, PAT, D-ROM (reactive oxygen metabolite index), OBRI (oxidative stress balance risk index) and OSI (oxidative stress index) were evaluated to assess the antioxidant capacity of plasma. Based on the data obtained, it can be concluded that: inhalation of Tskhaltubinsk water develops the effect of hormesis, which causes positive changes in all of the above markers of oxidative stress in the brain. Based on the data presented, inhalation of Tskhaltubo water can be considered as one of the methods for removing and treating convulsive phenomena, which is confirmed by experimental studies.

Keywords: epileptic rats, radon hormesis, oxidative stress.

РЕЗЮМЕ

РАДОНОВЫЙ ГОРМЕЗИС В ОКИСЛИТЕЛЬНОМ СТРЕССЕ И ПАТОГЕНЕЗЕ ЭПИЛЕПСИИ

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Многочисленные экспериментальные и клинические исследования показали, что окислительный стресс (ОС) играет значимую роль в развитии эпилепсии. Эпилептические припадки развиваются в результате ОС и в условиях гиперметаболического состояния вызывают интенсивное производство реактивного кислорода (ROS) в головном мозге. Причиной этого является изменение энергетического баланса в головном мозге. Согласно литературным данным, после судорог в головном мозге наблюдались ROS и другие маркеры окислительного стресса. С учетом вышеизложенного, целью исследования явилось определить влияние кратковременных (5 дней) ингаляций радонизированной водой Цхалтубо (5 минут) на течение эпилепсии и выявить изменения маркеров окислительного стресса у крыс.

Посредством фотометрического анализа FRAS-5 изучены предикторы ОС в плазме крови; для оценки антиоксидантной способности плазмы определяли PAT, индекс реактивного метаболита кислорода, индекс риска баланса окислительного стресса и индекс окислительного стресса.

На основании полученных данных авторами делается вывод, что при ингаляциях водой Цхалтубо происходит эффект гормезиса, который вызывает положительные изменения вышеперечисленных маркеров окислительного стресса в головном мозге, которые участвуют в регуляции окислительно-дегенеративных процессов, что клинически проявляется в уменьшении частоты эпилептических припадков. Исходя из вышеизложенного, ингаляции водой Цхалтубо следует рассматривать как один из методов снятия судорожных явлений и их лечения, что подтверждено экспериментальными исследованиями.

რეზიუმე

რადონის ჰორმეზისი ჟანგვითი სტრესის პრედიქტორებსა და ეპილეფსიურ პათოგენეზში

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მრავალმა ექსპერიმენტულმა და კლინიკურმა კვლევამ აჩვენა, რომ ოქსიდაციური სტრესი (OS) მნიშვნელოვან როლს ასრულებს ეპილეფსიის განვითარებაში. თავად ეპილეფსიური გულყრა გამოწვეულია OS-ით და ჰიპერმეტაბოლური მდგომარეობის ფონზე თავის ტვინში იწვევს რეაქტიული ჟანგბადის (ROS) ინტენსიურ წარმოქმნას. ამის მიზეზად თავის ტვინში ენერგეტიკული ბალანსის ცვლილება სახელდება. ლიტერატურულ მონაცემებზე დაყრდნობით ROS და ჟანგვითი სტრესის სხვა მარკერები თავის ტვინში კრუნჩხვების შემდეგ ფიქსირდება.

კვლევის მიზანს წარმოადგენდა რადონიზირებული წყალტუბოს წყლის ხანმოკლე 5-დღიანი ინგალაციის (5 წუთი) გავლენა კრუშინსკი-მოლოტკინას ხაზის ვირთაგვების ეპილეფსიურ მოდელზე და ოქსიდაციური სტრესის მარკერების ცვლილებების განსაზღვრა.

ექსპერიმენტულ შედეგებზე დაყრდნობით, რომლებიც მიღებულია იყო ფოტომეტრიული ანალიტიკური სისტემის FRAS-5 საშუალებით, სისხლის პლაზმაში შესწავლილია ოქსიდაციური სტრესის პრედიქტორები; პლაზმის ანტიოქსიდანტური სიმძლავრის შესაფასებლად შესწავლილია PAT-ი, რეაქტიული ჟანგბადის მეტაბოლიტების ინდექსი, ოქსიდაციური სტრესის ბალანსის რისკის ინდექსი და ოქსიდაციური სტრესის ინდექსი. მიღებულ მონაცემებზე დაყრდნობით ავტორები დაასკვნიან, რომ წყალტუბოს წყლის ინჰალაციით ვითარდება ჰორმეზისის ეფექტი, რომელიც იწვევს თავის ტვინში ყველა ზემოჩამოთვლილი ოქსიდაციური სტრესის მარკერების დადებით ცვლილებებს, რომლებიც მონაწილეობენ ოქსიდაციურ-დეგენერაციული პროცესების რეგულირებაში, რაც კლინიკურად ვლინდება ეპილეფსიური კრუნჩხვების შემცირებით. მოცემულ მონაცემებზე დაყრდნობით წყალტუბოს წყლის ინჰალაცია შეიძლება ჩაითვალოს კრუნჩხვითი ეფექტების მოხსნისა და მისი მკურნალობის ერთ-ერთ მეთოდად, რაც დასტურდება ექსპერიმენტული კვლევებით.

СОВЕРШЕНСТВОВАНИЕ ПЕРФУЗИОННОГО ПОТОКА НАСОСОВ КРОВИ

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Со дня внедрения в кардиохирургию (середина прошлого столетия) метода искусственного кровообращения, по сей день не прекращаются дискуссии о характере и значении искусственного потока крови. Приверженцы постоянного потока в качестве основного аргумента выдвигают наличие непрерывного, постоянного тока крови в микроциркуляторном русле, доказывая этим достаточность такого же потока в магистральных сосудах и указывая на простоту и дешевизну аппаратуры, создающей постоянный поток. Поэтому, во время перфузий отрицается необходимость обеспечения более сложной для воспроизведения и управления пульсовой волны искусственного потока [1,2,17,18]. Аргументы приверженцев пульсирующего потока основаны на филогенетически укоренившейся модуляции потока в крупных магистральных и преорганных артериях, создаваемого работой желудочков сердца и сохраняемого, хоть и с угасанием пульсовой волны, вплоть до артериол. Такая модуляция, раздражая сосудистые барорецепторы, поддерживает тонус магистральных и органных артерий во всем сосудистом древе и является предпосылкой поддержания нормального системного давления [3,4,7,9,11].

Наш подход к данному вопросу основывается на особенностях анатомии и физиологии сердечно-сосудистой системы и довольно просто интерпретируется. В организме генерацию кинетической энергии, придаваемой кровотоку, осуществляет миокард, функционирующий фазово в систоло-диастолическом цикле. Исходящие из сердца магистральные и проксимально расположенные артерии филогенетически адаптированы к характеру кровотока, создаваемому желудочками сердца, т.е. к циклическому функционированию. Многочисленными исследованиями доказана эволюционно сформированная нейро-гуморальная связь между сердцем и магистральными сосудами [10,12,13]. Данное обстоятельство при проведении искусственного кровообращения указывает на предпочтительность сохранения физиологического пульсирующего потока, создаваемого искусственным кровяным насосом в крупных ветвях артериального русла. Естественно, что в такой ситуации артерии будут получать адекватное барорецепторное раздражение, что теоретически должно исключать с их стороны развитие запредельных гемодинамических, а затем и метаболических ответов, влияющих на гомеостаз, таких как централизация кровообращения, нарушение органного кровотока, тканевая гипоперфузия, застойные процессы в микроциркуляторном русле, клеточное «голодание», накопление недоокисленных продуктов, буферные сдвиги.

Однако, создание насосов, конструктивно предназначенных для реализации пульсирующего потока, является технологически относительно сложной задачей и соответственно, более дорогой. Поэтому, идя по пути