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INFLUENCE OF ENERGY DRINKS ON THE MORPHOFUNCTIONAL STATE OF THE STOMACH

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Summary

The World Health Organization (WHO) has warned that «…the high consumption of energy drinks among young people and its long-term consequences are being ignored by scientists and the public,…» noting that this may lead to serious consequences potentially result in future challenges for the health care system. In this context, conducting research aimed at developing and enhancing the principles of prevention, diagnosis and treatment of diseases associated with functional and organic disorders of the stomach remains one of the urgent priorities of modern medicine.

The purpose *was to evaluate the morphofunctional changes of the stomach under the influence of energy drinks.*

Materials and methods. *The study was conducted on the stomachs of male albino rats aged 36 weeks. The experimental animals were divided into two groups. The control group comprised six male rats. The experimental (main) group included 23 animals exposed to an energy drink for periods of 4, 8, and 12 weeks. . The energy drink (ED) was administered intragastrically via a plastic tube once daily at a dose of 10 mg/kg of body weight for the specified durations..*

Results and discussions. *After 4 weeks of ED exposure, the mean gastric wall thickness was 34.89±0.52 (rms); the mucosal thickness was 17.57±0.32; the submucosal layer measured 5.83±0.11; and the combined thickness of the muscular and serous layers was 11.49±0.16. The density of glandular distribution within the mucosal layer was 12.32±0.26. Following 12 weeks of ED administration, the total thickness of the gastric wall measured 33.99±0.30; mucosal thickness was 16.47±0.29; submucosal thickness was 5.80±0.14; and the muscular and serous layers combined measured 11.12±0.32. The glandular density within the mucosa was 10.71±0.24. Thus, chronic exposure to energy drinks during 12 weeks resulted in histological features characteristic of chronic gastritis, including congestion of small blood vessels in the mucosa and submucosa of stomach, lymphocytic infiltration, and atrophy of the mucosal glands.*

Conclusions. *Chronic exposure to an energy drink over a 12-week period resulted in morphological changes consistent with chronic gastritis, including congestion of the small blood vessels of the mucosa and submucosa, lymphocytic infiltration, and atrophy of mucosal glands.*

Keywords: *Enzyme immunoassay, immunohistochemistry, morphometry, morphology, CD3, CD20, pepsinogen, serum, rat, stomach*

Introduction

Manufacturers of energy drinks claim that their products contain natural ingredients that that enhance energy levels, improve concentration and mental performance, and are harmless to health [1-3]. Simultaneously, the global medical community expresses concern regarding the adverse effects frequently associated with the consumption of such beverages. Energy drinks contain pharmacologically active ingredients, and comprehensive information regarding their composition and production is available in the literature [4-6]. The World Health Organization (2014) stated that «…the high consumption of energy drinks among young people and their long-term consequences are being ignored by scientists and the public,…» warning that this may lead to serious consequences and potentially result in future problems for health care systems [7].

In recent years, the consumption of energy drinks has increased markedly, prompting concern among medical professionals and public health authorities. These beverages, which contain elevated concentrations of caffeine, sugar, and other stimulants, are gaining popularity particularly among young and active people [8,9]. However, the lack of robust scientific data regarding their impact on human health underscores the necessity for research aimed at assessing their effects on various organ systems, including the gastrointestinal tract [10,11].

Of note, 20 of 82 calls to the National Poison Center of New Zealand between February 2005 and December 2009 were related to nausea, vomiting, and abdominal pain associated with energy drink consumption. The inhibitory effect of caffeine on gastric mucosal secretion is considered one of the key factors contributing to gastric mucosal injury [12]. In this context, studies aimed at the development and refinement of strategies for the prevention, diagnosis, and treatment of diseases associated with functional and structural disorders of the stomach represent a pressing issue in contemporary medicine.

Experimental studies in animal models, such as rats, provide essential data on morphofunctional alterations in the gastric wall resulting from exposure to energy drinks. Investigation of structural and functional changes in the stomach may aid in identifying potential pathologies associated with prolonged intake of such beverages. This is particularly relevant given increasing concerns about possible adverse effects, including gastritis and peptic ulcer disease [13,14].

Furthermore, elucidation of morphofunctional alterations in the gastric wall may contribute to the development of guidelines for the safe consumption of energy drinks and inform public perception regarding their health risks. Research in this domain may also assist health care professionals in educating patients about the potential consequences of excessive consumption of such products and contribute to the prevention of gastrointestinal disorders [15, 16, 17].

Thus, the investigation of morphofunctional changes in the gastric wall in rats subjected to experimental exposure

to energy drinks constitutes an important topic of scientific inquiry. Such studies not only expand current knowledge regarding the biological effects of these beverages but may also serve as a foundation for future clinical research and the development of effective strategies for the prevention of diseases associated with their consumption.

The purpose was to evaluate the morphofunctional changes of the stomach under the influence of energy drinks.

Materials and methods. Diagnosis at the nosologic level is complicated by the fact that the clinical manifestations of various gastrointestinal tract diseases are masked by common symptoms. As gastrointestinal diseases do not exhibit a symptom complex characteristic of a distinct clinical variant, the diagnostic process must first exclude organic pathologies with similar symptomatology. This necessitates the application of specific morphological, experimental, laboratory, and instrumental methods in accordance with established diagnostic algorithms to achieve diagnostic accuracy.

The stomachs of male rats aged 36 weeks were selected as the object of study. The experimental animals were allocated into two groups. The control group included six male albino rats. The main group consisted of 23 experimental animals exposed to energy drinks over periods of 4, 8, and 12 weeks. Animals in both groups were maintained under identical vivarium conditions. The distribution of animals by groups is presented in Table No. 1.

Table 1

Distribution of animals of control and main groups.

Age of animals animals	Control group		
36 weeks	6		
36 weeks	Main group		
	Those who received ED for 4 weeks	Those who received ED for 8 weeks	Those who received ED for 12 weeks
	8	7	8
Total	29		

To extrapolate the findings of experimental research to human physiology, the age of the animals was taken into consideration and corresponding recalculations were performed. The majority of experimental studies are conducted on small mammals, primarily laboratory albino rats, due to their favorable maintenance requirements. Additionally, these animals are characterized by a short gestation period and high reproductive capacity. The average lifespan of rats is approximately two years. In studies aimed at establishing proportional age correlations between humans and laboratory rats in postnatal ontogeny, a coefficient of 1.7 has been adopted, whereby a rat age of 120 days is considered equivalent to late adolescence in humans, i.e., 17 years (204 months): X (human age in months) = 1.7 × rat age (in days). Accordingly, the ratio of human life in months to rat life in days is 1.7 (K = 1.7). Based on the biological age correlation between humans and white laboratory rats, 1 day of rat life corresponds to 52 days of human life. The correlation of animal age with human age in this study was as follows: 36-week-old (252-day-old) rats corresponded to a human age of 36 years. Taking into account the duration of the experimental period, the age coverage within the study was broader. Specifically, considering that 36-week-old rats aged an additional 12 weeks during the experiment, the corresponding human age range extended from 36 to 48 years.

The experiment conducted on these animals was carried out in accordance with the regulations established by the Research Ethics Committee (Resolution No. 1/9-1854 of the Ethics Committee of the Ministry of Health of the Republic of Uzbekistan). The animals included in the study were housed in individual plastic cages under controlled environmental conditions with relative humidity maintained at 70% and ambient temperature at 24±1 °C. A standard 12-hour light/dark cycle was maintained throughout the experiment. All animals had unrestricted access to water and standard rodent chow in pellet form.

In this study, a widely consumed, locally manufactured energy drink available on the market of the Republic of Uzbekistan was used. The animals in the control group received an intragastric administration of 7.5 mL of saline solution once daily for 4, 8, and 12 weeks. The experimental group received the energy drink (ED) intragastrically for the same time periods via plastic gavage tubes. Each rat in the experimental group was administered 10 mL/kg of body weight of ED once daily through a probe. The dosage was determined based on the Paget and Barnes conversion table for rats and corresponds to the equivalent human dose [10]. The energy drink contained caffeine, taurine, glucuronolactone, sugars and other carbohydrates, food coloring agents, flavorings, vitamins, inositol, niacin, herbal supplements, and other ingredients [9].

On the final day of the experiment, the animals were fasted overnight. At 8:00 a.m., following anesthesia via intramuscular injection of a 2% xylazine solution at a dose of 0.2 mL/kg, blood samples were collected directly from the heart for laboratory analysis. The animals were euthanized by transection of the abdominal aorta. The abdominal cavity was opened, and internal organs were harvested in full compliance with established bioethical protocols. The stomach was excised by opening along the greater curvature, rinsed with cold physiological saline, and photographed. Macroscopic examination of the gastric mucosa was performed. Each specimen was labeled and placed in a plastic container filled with 10% neutral buffered formalin, suspended in whole form for fixation. Entire fixed specimens were embedded in paraffin in accordance with standard histological procedures.

For the purpose of evaluating general morphology and morphometric parameters of the structural components of the stomach, tissue sections obtained from paraffin blocks using a microtome were stained with hematoxylin and eosin. Each specimen was processed using the above methods.

Microscopic examination and photodocumentation were performed using a Leica light microscope equipped with a special camera. Morphometric analysis of the gastric wall structures was conducted using an ocular micrometer and a reticle eyepiece containing a grid with 256 intersection points. A magnification of ×40 was applied to assess the density of glandular distribution within the mucosa. Additionally, the thickness of the mucosal, submucosal, muscular, and serosal layers was measured separately. The collected data were subjected to statistical processing and the results were analyzed.

The immunohistochemical study initially employed tissue-specific antibodies for the determination of histogenetic type. CD3, a T-lymphocyte activation antigen, was used to assess receptor expression on the cell membrane. CD20, a B-lymphocyte antigen, is a protein functioning as a coreceptor of immunoglobulin within the cytoplasm of the cells. The results obtained were evaluated according to the ALLRED scoring system, which determines the proportion of receptor-positive cells and the intensity of receptor expression after immunostaining. These parameters are combined to assign a score ranging from 0 to 3. A score of 0 corresponds to negative expression, 1 point indicates low positive expression (10-30%), 2 points reflects medium positive expression (30-60%), and 3 points signifies high positive expression (60-100%).

In order to determine morphofunctional changes in the stomach, an enzyme-linked immunosorbent assay (ELISA) was performed on blood samples collected from the experimental animals. For this purpose, blood samples were left at room temperature for 30 minutes and then centrifuged at 4000 rpm for 15 minutes. Following centrifugation, the serum was extracted and stored at –20 °C until analysis. The immunologic evaluation included measurements of pepsinogen I (PG1), pepsinogen II (PG2), and the oncomarker CA 74-2 using commercially available enzyme immunoassay kits produced in the Russian Federation. The measurable concentration range for PG1 was 0-200 μg/L, for PG2 was 0-50 μg/L, and for CA 74-2 was 0-200 U/mL.

In recent years, a number of studies have been conducted in various countries with the aim of replacing invasive methods, such as endoscopy, with more effective and simplified noninvasive approaches for the screening of gastric diseases, including gastric and duodenal ulcers, gastroesophageal reflux disease, atrophic gastritis, and other gastric pathologies.

Pepsinogens, which are proenzymes of pepsin, mediate the initial and most critical stage of the digestive process by catalyzing the breakdown of proteins into amino acids. The normal concentration of PG1 in rat serum is generally within the range of 9-11 μg/L (ng/mL). It is known that the concentration of pepsinogens in peripheral blood is a reliable diagnostic marker reflecting the morphofunctional state of the gastric mucosa, thus enabling a noninvasive alternative to gastric mucosal biopsy. Therefore, noninvasive diagnostics play an important role in the early detection and prevention of gastric cancer. Malignant neoplasms of the gastrointestinal tract remain among the most prevalent forms of cancer worldwide. The application of specific antigen CA 74-2 for the early diagnosis of gastric cancer or precancerous alterations in the gastric mucosa under experimental exposure to energy drinks is of significant clinical relevance for timely therapeutic intervention in this pathology.

Results and discussion.

Morphometric alterations in the gastric wall of 36-week-old rats in the experimental group following administration of ED for 4, 8, and 12 weeks were as follows: after 4 weeks of ED exposure, the mean thickness of the gastric wall was 34.89±0.52 (rms units); the mucosal layer measured 17.57±0.32, the submucosal layer 5.83±0.11, and the combined thickness of the muscular and serosal layers was 11.49±0.16. The density of mucosal gland distribution was 12.32±0.26 (Figure 1).

In experimental animals receiving ED for 8 weeks, the total thickness of the gastric wall was 34.91±0.51, the mucosal layer 17.30±0.22, the submucosal layer 6.30±0.23, and the muscular-serosal layers 11.30±0.22. The density of mucosal glands was 11.19±0.26 (Figure 1). In animals exposed to ED for 12 weeks, the total gastric wall thickness was 33.99±0.30, the mucosa measured 16.47±0.29, the submucosa 5.80±0.14, and the muscular-serosal layers 11.12±0.32. The mucosal gland density was 10.71±0.24 (Figure 1).

Comparison of the morphometric parameters of the gastric wall layers between the experimental and control groups demonstrated the following: in animals receiving ED for 4 weeks, the total gastric wall thickness decreased by 33%, mucosal layer thickness by 44%, submucosal layer by 31%, and muscular-serosal layers by 8.5%, compared to controls. The density of mucosal glands decreased by 32.5% (Figure 1).

In experimental animals chronically exposed to ED for 12 weeks, the total thickness of the gastric wall showed a 36% reduction relative to controls, representing the most marked change among the groups. Mucosal thickness was reduced by 47%, submucosal thickness by 31%, and muscular-serosal layers by 11.5%. The density of mucosal glands decreased by 41%. The most significant alterations resulting from chronic ED exposure were characterized by pronounced thinning of the mucosal and submucosal layers and a marked decrease in mucosal gland density (Figure 1).

In rats exposed ED for 12 weeks, the degree of morphometric alterations in the mucosa and its glands, as well as in the submucosal and muscular-serosal layers, was greater than in those receiving ED for 4 or 8 weeks. Morphologic analysis revealed that the mucous and submucous membranes of the gastric wall exhibited fully engorged small blood vessels, inflammatory signs, and lymphocytic infiltration. Atrophy of the gastric mucosal glands was observed, with histological features consistent with chronic gastritis (Figures 2 and 3).

Analysis of morphometric indices of the gastric wall demonstrated that experimental animals receiving ED for 4 and 8 weeks exhibited significant adverse alterations, which were more pronounced with chronic exposure (12 weeks). These changes were primarily manifested by thinning of the gastric mucosa and a reduction in the density of mucosal gland distribution. Immunohistochemical examination revealed negative staining for CD3 and CD20 in the gastric mucosa of control group animals (Figures 3A and 3B).

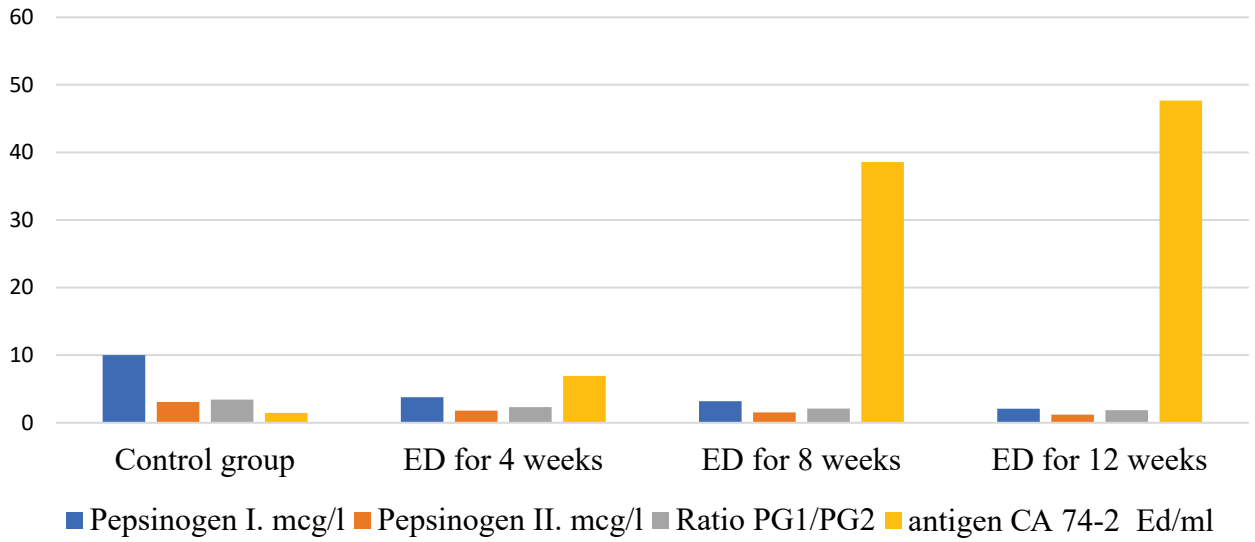

Figure 1. Comparative morphometric indices of gastric wall of 36-week experimental and control groups of animals

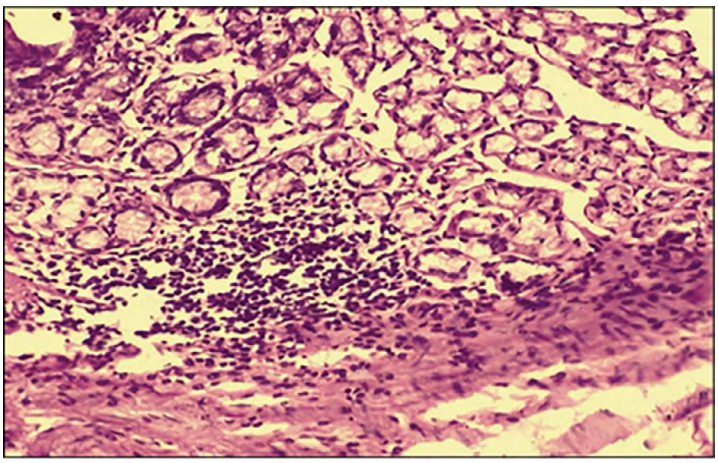

Figure 2. Histologic microdissection: foci of inflammation and lymphocytic infiltration in the gastric wall of rats treated with ED for 12 weeks. H-E staining. Col. 10. S40.

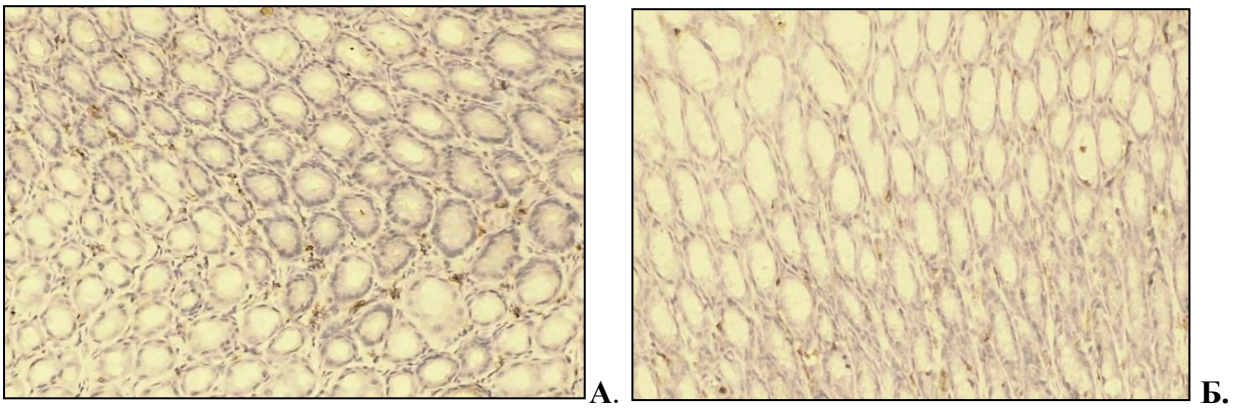

Figure 3. CD20 (A) and CD3 (B) negative reagent reactions in rat gastric mucosa. Dye: DAB-chromogen. S10 × S40.

In 36-week-old rats consuming ED for 4 weeks, CD20 expression was weakly positive, as evaluated by the ALLRED scoring system, with a score of 1 point. CD20 expression reflects the presence of lymphocytes at various stages of maturation, including lymphocytic immunoblasts, B lymphocytes, and plasma cells (Figure 4A). The gastric mucosa in rats is lined by columnar epithelium forming gastric fossae, at the base of which the gastric glands open. Immunohistochemical staining for CD3 in rats exposed to ED for 1 month revealed a weakly positive reaction localized to the lymphoid infiltrate of the gastric glands. This expression was also assigned a score of 1 point by the ALLRED method (Figure 4B).

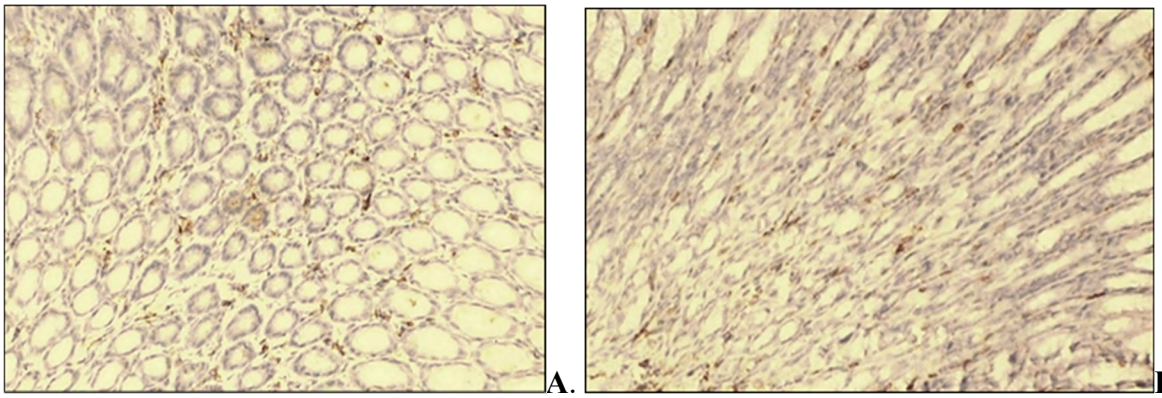

Figure 4. A. CD20 expression in the gastric mucosa in the form of small clusters; B. expression of T-lymphocytes with light CD3 marker in the gastric mucosa of 9-month-old rats treated with energy drinks for 1 month. Staining: DAB-chromogen. S. 10. S.40.

In 9-month-old rats of the main group consuming ED for 3 months, immunohistochemical examination of the gastric wall revealed foci of lymphoid tissue infiltration characterized by strong expression of the CD20 marker; however, primary follicles did not develop into secondary follicles. Moderate positivity for the CD20 marker was detected in certain areas of the gastric glands (Figure 5A). Partial expression of T lymphocytes, as identified by the CD3 marker, was noted within the lymphoid infiltrate of the gastric glands. Low positivity was observed in 30% of rats, and moderate positivity in 70% (Figure 5B). The marker expression was primarily localized to partially atrophied gastric glands, mucosal and submucosal layers, as well as to inflammatory foci.

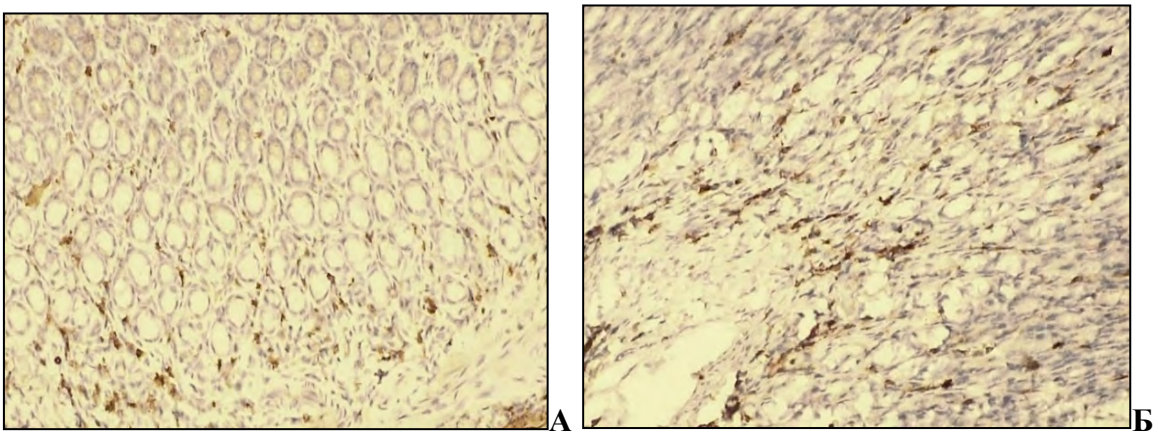

Figure 5. Moderate expression of CD20 marker (A) and CD3 marker (B) in primary lymphoid follicles of gastric mucosa in 9-month-old rats treated with energy drinks for 3 months. Staining: DAB chromogen. S10 × S 40.

Immunohistochemical analysis demonstrated that in 9-month-old rats administered energy drinks for one month, CD20 and CD3 marker expression throughout the mucous membrane exhibited a weak positive reaction, which was scored as 1 point using the ALLRED method. In rats exposed to ED for 3 months, CD20 expression showed a moderate positive reaction within atrophied mucous glands and lymphoid tissue of the submucosal layer, appearing as lymphocyte aggregates. These results corresponded to scores of 1 point in 30% and 2 points in 70% of animals, respectively, based on the ALLRED scoring system. CD3 expression was predominantly detected in lymphoid infiltrates of the gastric mucosal glands, areas of submucosal atrophy, and inflammatory sites.

To evaluate the morphofunctional status of the gastric wall under the influence of energy drink administration, blood levels of PG1, PG2, the PG1/PG2 ratio, and the concentration of the CA74-2 antigen were measured in 36-week-old rats from the experimental group. In rats exposed to ED for 4 weeks, PG1 concentration was 3.78±0.35 μg/L, PG2 concentration was 1.69±0.21 μg/L, the PG1/PG2 ratio was 2.31±0.32, and the specific antigen CA74-2 level was 6.91±4.75 IU/mL (Figure 6).

After 8 weeks of exposure, PG1 concentration was 3.19±0.33 μg/L, PG2 concentration was 1.52± 0.12 μg/L, with a PG1/PG2 ratio of 2.10±0.17. The concentration of CA74-2 antigen increased to 38.75±30.97 IU/mL (Figure 6).

In 36-week-old animals exposed for 12 weeks, PG1 concentration decreased further to 2.07 ± 0.18 μg/L, PG2 concentration to 1.18 ± 0.12 μg/L, with a PG1/PG2 ratio of 1.86 ± 0.21. The CA74-2 antigen level increased to 47.67 ± 42.81 IU/mL (Figure 6).

As a result of ED consumption in experimental animals for 4 and 8 weeks, a moderate decrease in serum levels of PG1, PG2, and the PG1/PG2 ratio was observed, while the concentration of the specific antigen CA 74-2 increased 19-fold following 8 weeks of intake. With prolonged exposure (12 weeks), a marked decrease in PG1 and PG2 levels was noted, accompanied by a 23.5-fold increase in CA 74-2 antigen concentration (Figure 6).

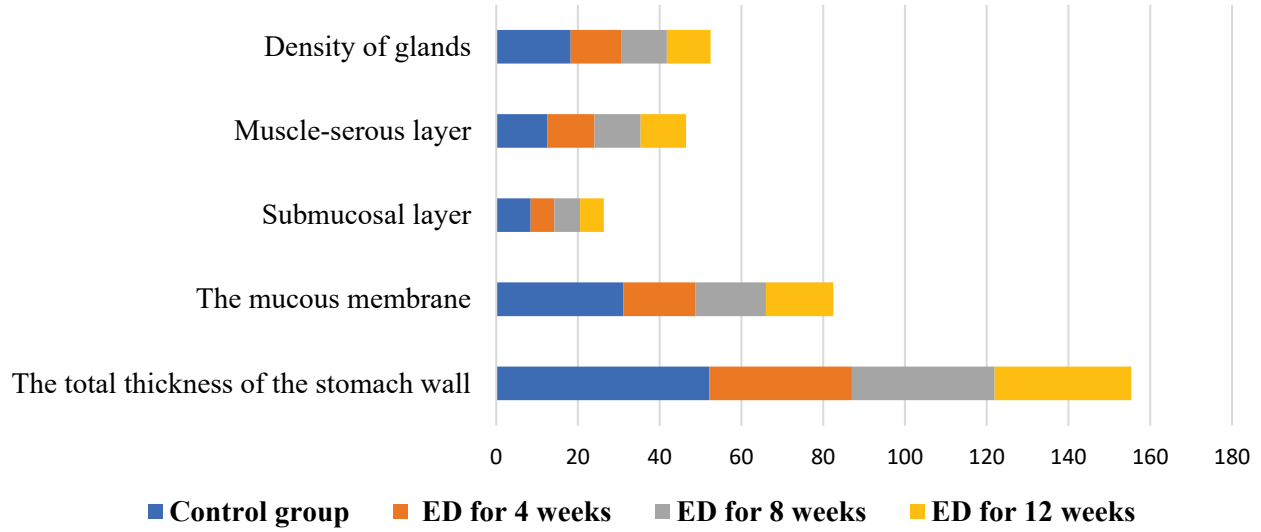

Figure 6. Comparative analysis of the ratio of PG1, PG2, PG1/PG2 and the amount of CA 74-2 in 36-week-old rats of the control and experimental groups who consumed an energy drink for different periods of time.

Assessment of the morphofunctional state of the gastric mucosa demonstrated a significant decline in PG1, PG2, and the PG1/PG2 ratio, alongside a sharp elevation in CA 74-2 levels after chronic ED consumption for 12 weeks. A progressive decrease in serum PG1 concentration is indicative of structural damage to the fundic glands, which contain a high proportion of main cells responsible for PG1 secretion. A pronounced reduction in PG2 levels suggests injury to the cervical glands of the fundus. Accordingly, following 4 and 8 weeks of exposure, PG2 production appears to be temporarily maintained in a compensatory state depending on the extent of mucosal injury. Alteration of the PG1/PG2 ratio reflects the severity of morphological disruption and the functional integrity of the gastric mucosa and its glands. As inflammation of the gastric mucosa progresses, metaplastic transformation of epithelial cells may occur, potentially leading to the development of atrophic gastritis. This is corroborated by the observed increase in CA 74-2 antigen levels in the blood following prolonged ED exposure.

The data obtained from morphometric evaluation of the gastric wall, immunohistochemical findings, and laboratory analyses collectively indicate that the deleterious effects of energy drink consumption are directly correlated with both the administered dose and duration of intake.

Conclusions. In experimental animals, exposure to the energy drink for 4 and 8 weeks resulted in pronounced alterations in the morphological and morphometric characteristics of the gastric wall. Chronic administration for 12 weeks induced more advanced morphological changes consistent with chronic gastritis and inflammation, manifested by vascular congestion in the mucosal and submucosal layers, lymphocytic infiltration, and glandular atrophy within the gastric mucosa. Immunohistochemical analysis of gastric mucosal samples from animals administered the energy drink for 3 months revealed CD20 and CD3 positivity in 70% of cases, indicating the presence of inflammatory infiltrates and glandular atrophy within the mucosa and submucosa, consistent with a chronic pathological process. A reduction in pepsinogen 1.2 amount (79%) suggests structural impairment of the fundic glands. A marked elevation in cancer marker CA 74-2 levels reflects epithelial cell metaplasia. These findings collectively represent features of gastric mucosal atrophy and chronic gastritis.

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ВПЛИВ ЕНЕРГЕТИЧНИХ НАПОЇВ НА МОРФОФУНКЦІОНАЛЬНИЙ СТАН ШЛУНКА

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Резюме.

Всесвітня організація охорони здоров'я (ВООЗ) попередила, що «…високе споживання енергетичних напоїв серед молоді та його довгострокові наслідки ігноруються вченими та громадськістю…», зазначивши, що це може призвести до серйозних наслідків, які потенційно можуть створити проблеми для системи охорони здоров'я в майбутньому. У цьому контексті проведення досліджень, спрямованих на розробку та вдосконалення принципів профілактики, діагностики та лікування захворювань, пов'язаних з функціональними та органічними розладами шлунку, залишається одним із нагальних пріоритетів сучасної медицини.

Метою дослідження було оцінити морфологічні та функціональні зміни шлунка під впливом енергетичних напоїв.

Матеріали та методи. Дослідження проводилося на шлунках самців щурів-альбіносів віком 36 тижнів. Експериментальних тварин було розділено на дві групи. Контрольна група складалася з шести самців щурів. Експериментальна (основна) група включала 23 тварин, які отримували енергетичний напій протягом 4, 8 та 12 тижнів. Енергетичний напій (ЕН) вводили внутрішньошлунково через пластикову трубку один раз на день у дозі 10 мг/кг маси тіла протягом зазначеного періоду.

Результати та обговорення. Після 4 тижнів впливу ЕД середня товщина стінки шлунка становила 34,89±0,52 (rms); товщина слизової оболонки становила 17,57±0,32; товщина підслизового шару становила 5,83±0,11; а сукупна товщина м'язового та серозного шарів становила 11,49±0,16. Щільність розподілу залоз у слизовому шарі становила 12,32±0,26. Після 12 тижнів прийому ЕД загальна товщина стінки шлунка становила 33,99±0,30; товщина слизової оболонки становила 16,47±0,29; товщина підслизової оболонки становила 5,80±0,14; а сумарна товщина м'язового та серозного шарів становила 11,12±0,32. Щільність залоз у слизовій оболонці становила 10,71±0,24. Таким чином, хронічне вживання енергетичних напоїв протягом 12 тижнів призвело до гістологічних ознак, характерних для хронічного гастриту, включаючи застій у дрібних кровоносних судинах слизової оболонки та підслизової оболонки шлунка, лімфоцитарну інфільтрацію та атрофію залоз слизової оболонки.

Висновки. Хронічне вживання енергетичного напою протягом 12 тижнів призвело до морфологічних змін, характерних для хронічного гастриту, включаючи застій у дрібних кровоносних судинах слизової оболонки та підслизової оболонки, лімфоцитарну інфільтрацію та атрофію залоз слизової оболонки.

Ключові слова: імуноферментний аналіз, імуногістохімія, морфометрія, морфологія, CD3, CD20, пепсиноген, сироватка, щур, шлунок.

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