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ANTIOXIDANT-PROOXIDANT SYSTEM OF THE ORAL MUCOSA IN RATS UNDER CHRONIC STRESS AGAINST THE BACKGROUND OF A THERAPEUTIC-PREVENTIVE COMPLEX IN THE CONTEXT OF PEDIATRIC DENTISTRY

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The study was devoted to evaluating the impact of a multi-component therapeutic-prophylactic complex on indices of oxidative damage and antioxidant protection in oral mucosal homogenates of rats subjected to chronic acoustic stress. Thirty-four male Wistar rats were randomly assigned to an intact control, a chronic-stress group, and a chronic-stress+therapeutic-prophylactic complex group. Acoustic stress was induced for 6 h per day¹ over 50 days using an LS-912 ultrasonic pest repeller (Leaven Enterprise, Taiwan; 30–60 kHz, 130 dB). Animals in the third group received the therapeutic-prophylactic complex daily throughout the stress period. Catalase activity and malondialdehyde content were assayed in oral-mucosal homogenates, and an antioxidant-prooxidant index was calculated. Findings substantiate further exploration of the therapeutic-prophylactic complex as a promising strategy for preventing oxidative-stress-driven complications of the oral mucosa, particularly in paediatric dentistry.

Key words: stress, oral mucosa, therapeutic-prophylactic complex, paediatric dentistry, rats, experimental study.

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АНТИОКСИДАНТНО-ПРООКСИДАНТНА СИСТЕМА СЛИЗОВОЇ ОБОЛОНКИ ПОРОЖНИНИ РОТА ЩУРІВ ПРИ ХРОНІЧНОМУ СТРЕСІ НА ТЛІ ЛІКУВАЛЬНО-ПРОФІЛАКТИЧНОГО КОМПЛЕКСУ В КОНТЕКСТІ ДИТЯЧОЇ СТОМАТОЛОГІЇ

Дослідження було присвячене оцінці впливу багатокомпонентного лікувально-профілактичного комплексу на показники окисного пошкодження та антиоксидантного захисту в гомогенатах слизової оболонки ротової порожнини щурів, які піддавалися хронічному акустичному стресу. Тридцять чотири самці щурів породи Вістар були випадковим чином розподілені на інтактну контрольну групу, групу з хронічним стресом та групу з хронічним стресом+лікувально-профілактичний комплекс. Акустичний стрес індукували протягом 6 годин на добу протягом 50 днів за допомогою ультразвукового відлякувача шкідників LS-912 (Leaven Enterprise, Taiwan; 30–60 кГц, 130 дБ). Тварини третьої групи отримували лікувально-профілактичний комплекс щодня протягом усього періоду стресу. Активність каталази та вміст малонового діальдегіду були визначені в гомогенатах слизової оболонки ротової порожнини, а також був розрахований антиоксидантний-прооксидантний індекс. Отримані результати підтверджують доцільність подальшого дослідження лікувально-профілактичного комплексу як перспективної стратегії профілактики ускладнень слизової оболонки ротової порожнини, спричинених окислювальним стресом, особливо в дитячій стоматології.

Ключові слова: стрес, слизова оболонка порожнини рота, лікувально-профілактичний комплекс, дитяча стоматологія, щури, експериментальне дослідження.

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The oral mucosa functions as a dynamic barrier equipped with innate immune components and antioxidant defenses that maintain homeostasis despite constant exposure to microbes and oxidative challenges. Under physiological conditions, a delicate balance is maintained between the production of reactive oxygen species (ROS) and their neutralization by salivary antioxidants (e.g., superoxide dismutase, catalase, glutathione systems), thereby preserving mucosal integrity. Disruption of this equilibrium due to microbial dysbiosis or systemic stressors can precipitate an excessive ROS accumulation and a shift toward oxidative stress, leading to lipid peroxidation, protein oxidation, and activation of inflammatory pathways in oral tissues [9, 10].

Chronic psychological stress is particularly detrimental to this balance. It triggers hyperactivity of the hypothalamic-pituitary-adrenal axis, leading to elevated glucocorticoid release, systemic immune dysfunction, and a heightened oxidative burden [6]. Clinically, individuals under prolonged stress exhibit worsened oral health outcomes: epidemiological studies link higher perceived stress with periodontal inflammation and impaired healing of gingival tissues [9]. In experimental models, concurrent stress exposure markedly aggravates oral inflammatory lesions. For example, rats subjected to chronic stress

show increased periodontal breakdown, accompanied by heightened local oxidative damage and NF-κB activation, indicating that stress accelerates inflammation, in part, by intensifying tissue ROS injury [7]. Biochemically, stress conditions drive a significant increase in malondialdehyde (MDH) – a reactive end-product of lipid peroxidation – along with a concomitant decline in antioxidant enzyme activities in the oral mucosa [7]. Such findings underscore that chronic stress tilts the mucosal redox system toward a pro-oxidant state, overwhelming the protective capacity of enzymes like catalase to decompose hydrogen peroxide. Notably, even in pediatric subjects, systemic stressors can elicit similar oxidative shifts: sleep deprivation, as a model of chronic stress, elevates gingival crevicular MDH levels and disrupts the normal redox balance of the oral cavity [12]. Consistently, pathologies such as oral lichen planus exemplify the consequences of redox imbalance, with patients showing significantly increased MDH and nitric oxide in saliva alongside depleted antioxidant reserves [4]. These observations support MDH's status as a key biomarker of oxidative injury in oral disease and highlight the vulnerability of oral tissues to sustained oxidative stress.

Given the central role of oxidative stress in mediating mucosal damage, there is growing interest in therapeutic strategies that bolster the oral antioxidant defense. A range of natural antioxidant compounds has demonstrated protective effects on oral tissues in both clinical and preclinical studies. For instance, polyphenolic phytochemicals such as curcumin can attenuate mucosal inflammation and oxidative injury by activating the Nrf2 pathway and upregulating cytoprotective enzymes (e.g., heme oxygenase-1, superoxide dismutase, catalase) [5]. Curcumin's multifaceted antioxidant and anti-inflammatory actions have been shown to reduce the severity of experimental oral mucositis, enhancing epithelial healing and integrity. However, while such single-agent interventions are promising, long-term evidence from clinical trials remains limited, and challenges with bioavailability and consistency of outcomes persist [5]. These considerations have spurred the development of multi-component therapeutic-prophylactic regimens that combine antioxidants with immunomodulatory and antimicrobial elements to achieve synergistic mucosal protection. Recent studies in mucosal immunology suggest that integrated protocols that simultaneously quell oxidative stress, modulate the inflammatory response, and support the oral microbiome can yield more sustained benefits than single-modal therapies [5, 11]. Such an approach is especially pertinent in pediatric dentistry, where safe and effective prophylactic measures are needed to fortify the oral cavity's defenses during growth and under stress. In fact, age-related analyses reveal that children have a distinct salivary antioxidant profile, and reinforcing the antioxidant barrier early may help mitigate oxidative damage over the lifespan [8].

Therefore, the purpose of this study was to systematically investigate the impact of chronic stress on the antioxidant-prooxidant system of the oral mucosa and to evaluate a novel multi-component therapeutic-prophylactic complex (TPC) designed for pediatric dental use.

The purpose of the study was to evaluate the effect of a therapeutic complex of drugs, intended for future implementation in pediatric dentistry, on the indicators of the antioxidant-prooxidant system in the oral mucosa of rats against the background of chronic stress modeling.

Materials and methods. Experimental studies were conducted using 34 male Wistar rats, 2 months old with an average body weight of 140±8 g. Inclusion criteria were: confirmed Wistar strain and male sex; age exactly 8±1 weeks (verified from breeding records); body-weight falling within cohort mean at baseline (140±8 g); absence of visible injury, malocclusion, or systemic/oral pathology on veterinary inspection; no previous exposure to experimental procedures or pharmacological agents. Exclusion criteria, applied immediately before random allocation, comprised: clinically detectable disease or behavioural abnormalities; weight outside the predefined range; wounds or oral lesions; failure to adapt during the 7-day acclimatisation period. Only male Wistar rats were enrolled to minimise biological variability introduced by sex-specific endocrine cycles.

The study was carried out from 24 April 2023 to 30 June 2023.

All animals were maintained in the accredited barrier-type vivarium of SE "ISMFS NAMS" under the husbandry conditions specified in the institute's standard operating procedure, which complies with both Ukrainian legislation and ARRIVE 2.0 recommendations. Briefly, rats were group-housed (4–5 per cage) in individually ventilated Euro-Type IV polycarbonate cages (floor area ≈ 1500 cm^2; internal dimensions 425 × 266 × 185 mm) fitted with stainless-steel wire lids and placed on ventilated racks delivering 12±2 air changes h^{-1}. Cages contained 3 cm of autoclaved aspen-wood shavings (LIGNOCEL® Hygienic Animal Bedding, JRS GmbH) that were replaced twice weekly. Environmental enrichment was provided ad libitum and comprised shredded paper nesting material (Nestlets), hardwood gnawing blocks (2×2×6 cm), and a cardboard tunnel (⌀ 9 cm, length 12 cm) per cage; items were refreshed at each cage change.

The room was held at 21±2 °C with 55–75 % relative humidity and a 12:12 h light/dark cycle (lights on 07:00–19:00). Noise was <50 dB SPL outside scheduled stress-exposure periods. Reverse-osmosis-filtered water was supplied in polycarbonate bottles with stainless-steel sipper tubes and replaced thrice weekly. Animals received a complete pelleted chow (PK-120-1, LabKorm, Ukraine) ad libitum; diet batches were screened for mycotoxins and heavy metals according to ISO 22005. Daily husbandry included visual health checks and removal of soiled enrichment; full cage sanitisation (hot-water wash and autoclave) was performed weekly.

Each rat was weighed, assigned an individual code, and allocated to an experimental arm using simple computer-generated randomisation in Microsoft Excel. Animals were assigned to three experimental groups:

1 – Intact control (standard vivarium diet), n=10;

2 – Chronic sound stress model, n=12;

3 – Chronic sound stress model + drug complex, n=12.

No animal met any exclusion criterion, and there were no deaths or withdrawals after group allocation. consequently, the composition of all three groups remained unchanged throughout the 50-day observation period, and data from every animal were included in the final analysis.

Experimental studies were conducted at the "Laboratory of Biochemistry and Vivarium" of the SE "The Institute of stomatology and maxilla-facial surgery National academy of medical sciences of Ukraine" (SE "ISMFS NAMS"). All experiments on rats were conducted according to standard operating procedures approved by SE "ISMFS NAMS", developed in accordance with the Guidelines of the Pharmacological Committee of the Ministry of Health of Ukraine, the "General Ethical Principles of Animal Experiments" adopted by the Seventh National Congress on Bioethics (Kyiv, 2019) and was guided by the recommendations of the European Convention for the Protection of Vertebrate Animals for Experimental and Other Scientific Purposes (Strasbourg, 1985) [2]. Studies recommended by the Commission on Bioethical Expertise (conclusion of the bioethics commission of the SE "ISMFS NAMS", protocol No. 1015 of 04/12/2023).

Stress was simulated using an LS-912 ultrasonic pest repeller (Leaven Enterprise, Taiwan), which operates in the audible and ultrasonic ranges and has a frequency of 30 to 65 kHz. Sound pressure 130 dB, power 1.5 W over an area of up to 232 m².

Simulation of sound stress with ultrasound in rats in groups 2 and 3 was carried out for 5 days, 6 hours a day, according to the following scheme: for 2 days, ultrasound with a frequency of 30 kHz was applied, the next 2 days – 40 kHz, the next 2 days – 50 kHz, the next 2 days – 60 kHz. The pattern was then repeated using ultrasound. Every day, an audible sound was added to the ultrasound for 1 hour by pressing the sound control button on the repeller. The repeller was installed at the same level as the animal cages, 3 m away.

Animals were first given melatonin (INN Melatonin; "Vita-Melatonin", PJSC Farmak, Kyiv, Ukraine): 0.7 mg per animal, dissolved in 0.3 mL of distilled water, and administered orally to support circadian antioxidative defence. Thirty minutes later, the animals were given a multivitamin/multimineral immunomodulatory complex (INN Retinol + Ascorbic acid + α-Tocopherol + B-group vitamins + Zinc + Selenium; "Orthomol Immun", Orthomol pharmazeutische Vertriebs GmbH, Langenfeld, Germany). A suspension containing 3 mg of pulverised tablet material in 0.5 mL of 0.9 % saline was administered orally to each rat to enhance systemic antioxidant capacity and immune competence. Immediately afterwards, the animals received a collagen-based nutricosmetic formulation (INN Collagen hydrolysate + Hyaluronic acid + Ascorbic acid + Zinc; "Orthomol Beauty", same manufacturer): 0.8 mL per rat of the commercial drinkable solution, delivered via gavage, to reinforce connective-tissue resilience and mucosal repair.

The experiment lasted 50 days. The experimental animals were euthanized under thiopental anesthesia (40 mg/kg, PJSC "Kievmedpreparat", Ukraine) by bloodletting from the heart. In the mucous membranes of the oral cavity (20 mg/ml of 0.05 M Tris-HCI buffer, pH 7.5; Merck, Germany). Lipid peroxidation marker – malondialdehyde content was assessed by the TBARS assay using 2-thiobarbituric acid, ≥99 % (Sigma-Aldrich, USA); the chromogen's absorbance was read at 532 nm. Catalase activity was quantified by the ammonium molybdate stop-reaction method using 30 % H_2O_2 (Sigma-Aldrich, USA) as the substrate and ammonium molybdate, ACS reagent (Sigma-Aldrich, USA) as the quencher, monitoring residual peroxide at 410 nm. The antioxidant-prooxidant index (API) was calculated as the ratio of catalase activity (μkat g^{-1} protein) to MDH level (nmol mg^{-1} tissue) [1].

Data processing was carried out with STATISTICA 6.1. Before parametric testing, the Shapiro-Wilk normality test was applied to each continuous variable; none showed significant deviation from a Gaussian distribution ($p>0.05$). Therefore, inter-group comparisons were performed with the two-tailed

Student's t-test. When pairwise contrasts were required (Control × Comparison, Control × Intervention, Comparison × Intervention), the family-wise type I error rate was controlled with the Bonferroni adjustment. Between-group differences were deemed statistically significant at $p<0.003$ [3].

Results of the study and their discussion. It is known that the balance between pro- and antioxidant systems maintains lipid peroxidation (LPO). To assess the functioning of the antioxidant link of the prooxidant-antioxidant system of the oral mucosa (OM) of experimental animals, the activity of catalase, one of the main enzymes that provides protection against oxidative damage, was determined. The generalized results of determining the indicators of the antioxidant-prooxidant system against the background of modeling chronic sound stress and correction of changes with a composition of drugs are presented in Table 1.

Table 1

Indicators of the antioxidant-prooxidant system in the oral mucosa of rats under chronic stress and under the influence of a therapeutic-prophylactic complex of drugs, M±m

Indices / Group	Catalase activity, μkat/kg	MDH content mmol/kg	API
1. Intact control, n=10	8.6±0.3	16.4±1.0	5.24±0.25
2. Chronic stress, n=12	6.0±0.1 p<0.001	27.3±1.3 p<0.001	2.19±0.16 p<0.001
3. Chronic stress + TPC, n=12	8.2±0.2 p>0.8 p_1<0.001	19.4±1.1 p>0.5 p_1<0.002	4.22±0.19 p<0.02 p_1<0.001

Note: p – significance relative to the intact group; p_1 – significance relative to the chronic stress group.

Analyzing the data obtained from the experimental study, it should be noted that under conditions of simulated stress in the oral mucosa of rats in group 2, the activity of the first link enzyme of the antioxidant system, catalase, reliably decreases by 30.2 % ($p<0.001$) and at the same time, the antioxidant-prooxidant index decreased by 2.4 times ($p<0.001$) relative to the intact group. Also, against the background of chronic stress, lipid peroxidation was intensified, with MDH content increasing by 66.6 % ($p<0.001$).

Thus, the results of the experimental study indicate that chronic stress activates lipid peroxidation processes with simultaneous insufficient antioxidant protection by catalase activity, which does not ensure hydrogen peroxide inactivation and promotes the active formation of lipid peroxidation end products – TBK reactants, which in turn cause cytotoxic effects, as well as pronounced dystrophic and necrobiotic changes in the cells of the oral mucosa.

Regular administration of a preventive complex of drugs to rats of the third group against the background of stress modeling leads to a significant increase in antioxidant enzymes – catalase activity by 36.6 % ($p_1<0.001$), antioxidant-prooxidant index by 1.93 times, ($p_1<0.001$), while the MDH content (the end product of lipid peroxidation) reliably decreases by 28.9 % ($p_1<0.002$) compared to the indicators of the 2nd group (chronic stress). Thus, daily treatment of the experimental animals in the third group for 50 days, against a background of chronic stress, prevented negative effects on the oral mucosa, inhibited the development of systemic inflammation and LPO, and significantly stimulated the antioxidant system.

The experimental data demonstrate a clear stress-induced redox disequilibrium in rat oral mucosa, manifested by a 30 % decline in catalase activity, a 66 % surge in malondialdehyde (MDH), and a greater than two-fold reduction in the antioxidant-prooxidant index (API) relative to intact controls. These findings corroborate evidence that chronic psychophysical stress amplifies local lipid peroxidation while simultaneously depleting enzymatic defenses, thereby creating a permissive environment for inflammatory degeneration of soft tissues [7]. The magnitude of oxidative disruption observed here parallels reports in periodontal and oral epithelial models in which sustained hypothalamic-pituitary-adrenal activation precipitated NF-κB-mediated tissue injury and accelerated matrix breakdown [10]. Significantly, daily administration of the multi-component therapeutic-preventive complex almost completely normalized the oxidative profile: catalase activity rebounded to near-basal values, MDH fell by roughly one-third, and API doubled relative to stressed untreated animals. This recovery underscores the benefit of simultaneously reinforcing enzymatic antioxidants and membrane-stabilizing factors, an approach increasingly advocated for mucosal protection but seldom validated in vivo [11]. While single phytochemical antioxidants have shown promise in experimental mucositis models, their clinical efficacy is often constrained by bioavailability and monotherapy. In contrast, the TPC's composite formulation appears to deliver a broader spectrum of redox modulation, aligning with calls for multi-facet prophylaxis capable of intercepting both ROS generation and downstream lipid damage [10]. From a translational standpoint, these results are especially pertinent to pediatric dentistry. Salivary surveys reveal that children possess a distinct,

developmentally regulated antioxidant architecture that may render them more susceptible to oxidative insults under environmental or psychosocial stressors [8]. Indeed, inadequate sleep – a proxy for chronic stress in school-aged cohorts – has been shown to elevate gingival crevicular MDH and disturb the salivary redox ratio in children, mirroring the biochemical derangements documented in the present rat model [12]. The TPC-mediated restoration of API therefore suggests a promising prophylactic avenue to bolster the immature antioxidant barrier during growth. Moreover, the marked catalase rebound observed here could offset the catalytic depletion implicated in stress-exacerbated periodontal inflammation, potentially curbing early-life trajectories toward chronic oral pathologies [7]. Collectively, these data provide mechanistic support for integrating multi-component antioxidant protocols into pediatric preventive regimens. By re-establishing redox homeostasis under stress, such complexes may not only safeguard oral mucosal integrity but also mitigate the systemic oxidative load increasingly linked to neuroendocrine and metabolic sequelae of childhood stress exposure [10, 11]. Future studies should verify long-term safety, optimize dosing for pediatric applications, and explore synergistic interactions with established non-pharmacologic interventions (e.g., sleep hygiene and stress management programs) to achieve holistic oral health resilience.

Conclusions

1. Chronic acoustic stress elicits a marked oxidative shift in rat oral mucosa, evidenced by a 66 % rise in MDH, a 30 % reduction in catalase activity, and a 2.4-fold fall in the antioxidant-prooxidant index.

2. Fifty-day administration of the therapeutic-prophylactic complex significantly increases catalase activity to levels statistically indistinguishable from intact controls, lowers MDH by nearly one-third, and doubles the antioxidant-prooxidant index.

3. The TPC exhibits pronounced antioxidant, membrane-stabilising, and anti-inflammatory effects, supporting its suitability for the prevention and correction of stress-induced oxidative injury of the oral mucosa.

4. These results justify further studies to optimise dosing regimens and to translate the findings into clinical paediatric-dental models aimed at enhancing redox homeostasis under psycho-emotional stress conditions.

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