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THE EFFECT OF PROBIOTIC TREATMENT ON OBESITY-RELATED CHANGES IN THE PERIODONTIUM OF OBESE RATS

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Obesity is a significant global health concern associated with various systemic diseases, including periodontitis. Probiotics have shown promising outcomes as an adjunctive therapy in both clinical and microbiological parameters; however, their impact on obesity-induced periodontal alterations remains unclear.

This study aimed to evaluate the biochemical changes in soft periodontal tissues of obese rats and assess the potential protective effects of probiotic supplementation.

Methods. A total of 46 rats were divided into three groups: (1) control, (2) obesity, and (3) obesity with probiotic treatment. Obesity was induced via neonatal monosodium glutamate administration, and probiotic supplementation was provided for three months. Biochemical analyses of periodontal tissues included oxidative stress markers, enzymatic activity, and inflammatory mediators.

The results showed that obesity significantly decreased markers of oxidative stress and reduced the activity of antioxidant enzymes. Probiotic treatment effectively normalized BMI, reduced oxidative damage, and restored enzymatic balance in periodontal tissues. Additionally, probiotic supplementation modulated nitric oxide synthase activity, suggesting a potential regulatory effect on inflammatory and vascular responses.

Conclusion. This study highlights the detrimental effects of obesity on periodontal health and suggests that probiotics may serve as a protective intervention against obesity-induced oxidative and inflammatory damage.

Keywords: obesity, periodontitis, probiotic, oxidative stress.

Obesity is a growing global health concern that has been linked to various systemic diseases, including periodontitis [1]. Periodontal tissues are particularly susceptible to oxidative stress and enzymatic imbalances associated with obesity, which may exacerbate tissue degradation and inflammatory responses. Probiotics are live microorganisms that provide health benefits to the host by influencing the composition and activity of the microbiota [2]. The mechanisms by which probiotics aid in the prevention and treatment of periodontal disease include inhibiting the growth and virulence of pathogenic species through competition for resources and the production of antimicrobial compounds, modulating the immune response to reduce inflammation and promote healing, and restoring microbial balance within the oral microbiome [3, 4]. Studies in probiotics as an adjunctive therapy showed a promising outcome in both clinical and microbiological parameters [5]. However, the impact of probiotics on obesity-induced periodontal tissue alterations remains unclear.

Aim. This study aims to evaluate the biochemical changes in soft periodontal tissues of obese rats and assess the potential protective effects of probiotic supplementation.

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Methods. Experiments were conducted on 46 rats of both genders, divided into three groups: Group I (Control): Intact newborn rats received a subcutaneous injection of 8 μL/g saline ($n = 18$). Group II (MSG-Induced Obesity): Rats received subcutaneous injections of monosodium glutamate (MSG) at a dose of 4 mg/g on days 2, 4, 6, 8, and 10 of life ($n = 16$) [6]. Group III (Obesity + Probiotic Treatment): Rats with MSG-induced obesity received the dry bacterial concentrate of probiotic microorganisms at a dose of 14 mg/g for 3 months, following a regimen of 2 weeks of administration followed by a 2-week break ($n = 12$). The distribution of animals into groups and the corresponding interventions are illustrated in Fig. A.

As a probiotic, the dry bacterial concentrate of probiotic microorganisms was used, developed under project TU 21.1.-2960512097-005:2015, which has undergone State Sanitary and Epidemiological. The concentrate is a powder of a dried suspension containing live probiotic Registration microorganisms of the genera *Lactobacillus, Bifidobacterium,* and *Bacillus* in a physiological sodium chloride solution. The microbial strains are deposited in the Microorganism Depository of the D.K. Zabolotny Institute of Microbiology and Virology of the National Academy of Sciences of Ukraine. The concentrate includes monocultures of: *Lactobacillus casei* IMB B-7280, *Lactobacillus acidophilus* IMB B-7279, *Lactobacillus delbrueckii* subsp. *bulgaricus* IMB B-7281, *Bifidobacterium animalis* VKL and/or *B. animalis* VKB, *Bacillus subtilis* IMB B-7393 and/or *B. subtilis* IMB B-7392. The concentration of viable microorganisms is no less than 4×10^{11} CFU/g.

After four months, body weight changes and body mass index (BMI) were analyzed in all groups. Soft periodontal tissues were excised, minced, treated with collagenase, homogenized, and centrifuged. The following parameters were assessed in the periodontal tissue homogenate: total proteolytic activity, antitrypsin activity, free fucose content, nitrite anion levels, total NO-synthase (NOS) activity, TBA-reactive substances, oxidatively modified proteins (OMP), and catalase activity.

Statistical analysis was performed using the non-parametric Mann–Whitney U test to compare two independent groups. Results are expressed as M ± SD, with significance set at $P < 0.05$.

Results and Discussion. We observed the development of obesity in 4-month-old MSG-treated rats, confirmed by a significant increase in BMI compared to the control group ($P < 0.05$). In the probiotic-treated group, BMI was normalized ($P < 0.01$) compared to untreated obese rats (Fig. *B*). MSG-induced obesity contributed to the activation of free radical oxidation in soft periodontal tissues, as evidenced by a significant increase in TBA-reactive substances, nearly 1.92-fold in both male and female rats ($P < 0.05$, Fig. *D*), and a 1.67-fold increase in oxidatively modified proteins (OMP) compared to controls (Fig. *C*). Under these conditions, catalase activity significantly decreased by 1.52-fold in the periodontal tissues of female rats compared to control animals ($P < 0.05$, Fig. *E*). In probiotic-treated rats, OMP and TBA levels were significantly lower than in untreated obese rats (Fig. *C* and *D*), while catalase activity in periodontal tissue was significantly higher (Fig. *E*).

In homogenates of periodontal tissues of obese rats, an imbalance between proteinases and their inhibitors' activity was observed (Fig. F and G), and treatment with probiotics restored it. The free fucose concentration was not affected significantly by probiotic treatment (Fig. *H*). NOS activity in periodontal tissues was significantly affected by obesity. Treatment with probiotics significantly decreased it. Our study demonstrated that MSG-induced obesity in rats led to significant metabolic and biochemical alterations in soft periodontal tissues, characterized by increased oxidative stress, enzymatic imbalance, and inflammatory changes. The observed increase in BMI in the MSG-treated group confirms the successful induction of obesity, which was significantly normalized following probiotic treatment. These findings suggest a potential modulatory effect of probiotics on obesity-related metabolic dysregulation.

Oxidative stress plays a crucial role in obesity-related pathologies, including periodontal tissue damage. The significant elevation of TBA-reactive substances and OMP in obese rats suggests increased lipid peroxidation and protein oxidation, indicative of heightened free radical activity, which aligns with previous findings [7]. The observed decline in catalase activity further supports an impaired antioxidant defense mechanism in periodontal tissues. Probiotic treatment effectively reduced oxidative damage by lowering OMP and TBA levels while restoring catalase activity, suggesting its protective role in mitigating obesity-induced oxidative stress. The antioxidant effect of probiotic supplementation in the treatment of periodontitis was confirmed by clinical studies [8].

Furthermore, we identified an imbalance in proteinase and inhibitor activity in the periodontal tissues of obese rats, which was restored with probiotic supplementation. This imbalance may contribute to tissue degradation and inflammation, further exacerbating periodontal damage [9]. Interestingly, free fucose levels remained largely unaffected by probiotic treatment, indicating that the intervention may not significantly alter carbohydrate metabolism in periodontal tissues.

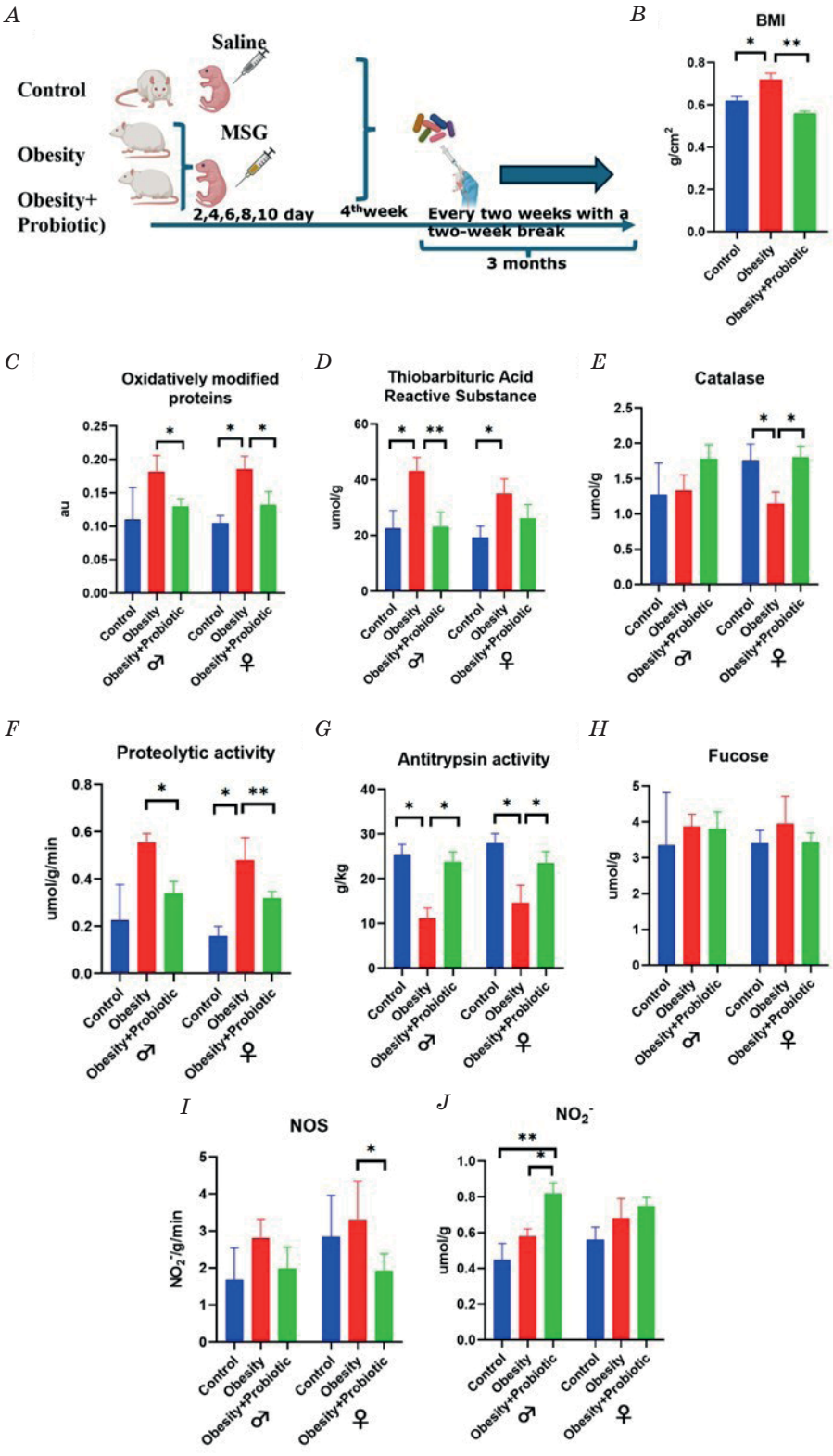

Figure. **Changes in biochemical parameters in the soft periodontal tissues of obese rats treated with probiotics:**

A — Flowchart illustrating the sequence of events in the experiment; *B* — Changes in body mass index (BMI) of rats throughout the experiment timeline; *C* — Concentration of oxidatively modified proteins in soft periodontal tissues; *D* — Levels of thiobarbituric acid reactive substances; *E* — Catalase activity; *F* — Proteolytic activity; *G* — Antitrypsin activity; *H* — Fucose concentration; *I* — NO-synthase activity; *J* — Nitrite anion levels

Conclusions. Overall, our findings underscore the detrimental effects of obesity on periodontal health and highlight the therapeutic potential of probiotics in mitigating obesity-induced oxidative and inflammatory damage. Further studies are needed to elucidate the precise molecular mechanisms underlying these protective effects and to evaluate the long-term benefits of probiotic interventions in the management of periodontitis in obese individuals.

Authors' contribution

MSk and KN conducted the experiment, measured biochemical parameters, and performed data analysis and presentation. MSp provided the necessary resources for this study. All authors contributed to the thesis writing and revision.

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