Markov A. V. Influence of a high-fat diet on inflammation and dysbiotic processes in the gums and blood serum of rats with lincomycin dysbiosis. Journal of Education, Health and Sport. 2020;10(1):128-135. eISSN 2391-8306. DOI http://dx.doi.org/10.12775/JEHS.2020.10.01.014
https://apcz.umk.pl/czasopisma/index.php/JEHS/article/view/JEHS.2020.10.01.014
http://dx.doi.org/10.5281/zenodo.3630655

The journal has had 5 points in Ministry of Science and Higher Education parametric evaluation. § 8. 2) and § 12. 1. 2) 22.02.2019.

Received: 20.12.2019. Revised: 25.12.2019. Accepted: 29.01.2020.

UDK 612.397:577.16:613.2

INFLUENCE OF A HIGH-FAT DIET ON INFLAMMATION AND DYSBIOTIC PROCESSES IN THE GUMS AND BLOOD SERUM OF RATS WITH LINCOMYCIN DYSBIOSIS

A. V. Markov

Lviv National Medical University named after Danylo Galytskij

Abstract

Background. To determine the effect on the periodontium and the whole organism of excessive consumption of palm oil, against the background of lincomycin dysbiosis.

Methods. The experiments were carried out on rats, divided into 3 groups: 1st – control, 2nd and 3rd with dysbiosis, which was reproduced using lincomycin administered with drinking water at a dose of 60 mg / kg for 5 days. Rats of the 3rd group additionally received palm oil at a dose of 16 g / kg with food. Rats were fed for 21 days. The activity of elastase, urease, lysozyme, catalase, and the content of malondialdehyde (MDA) and hyaluronic acid were determined in the blood serum and in the gums of rats.

Results. In rats, the administration of lincomycin caused an increase in the serum and gum activity of elastase and MDA content, an increase in urease activity and the degree of dysbiosis, but a significant decrease in lysozyme activity.

Conclusion. The introduction of lincomycin causes the development of systemic inflammation and generalized dysbiosis, the development of gingivitis and dysbiosis in the gums, especially when combining the administration of lincomycin with the consumption of palm oil.

Key words: **periodontal; blood serum; dysbiosis; inflammation; antioxidants; fat nutrition; antibiotic.**

INTRODUCTION

It is known that the introduction of antibiotics into the body causes the development of dysbiosis (disturbance of the balance of probiotic and conditionally pathogenic bacteria) and even dysbiosis (when the level of nonspecific immunity of the macroorganism is reduced against dysbiosis) [1, 2].

Of the majority of antibiotics studied, lincomycin was the most effective, most inhibiting the growth of probiotic bifidum and lactobacilli [3]. Lincomycin is used to reproduce intestinal and oral dysbiosis [4, 5].

It is established that high-fat diets also cause the development of dysbiosis [6, 7]. This is especially true in view of the increase in human consumption of fats, especially with high palmitic acid content [8, 9].

The aim of this work was to determine the effect on the condition of the gums and blood serum of rats of the connective action of a high-fat diet (HFD) and lincomycin dysbiosis.

MATERIAL AND RESEARCH METHODS

The high-fat diet was obtained by adding 3.2 g (16 g / kg) of palm oil to the standard compound feed for rats, calculated on each rat daily for 21 days. Used palm oil produced by “Dukess RBD” (Malaysia) with a content of 42% palmitic acid.

For the reproduction of dysbiosis used drug lincomycin production «Pharmacevtic firm «Darnitsa», Ukraine»,which was administered from the first day of the experiment for 5 days with drinking water at a dose of 60 mg/kg [10].

The experiments were performed on 24 white Wistar rats (males, 8 months old, mean live weight 200 ± 15 g), which were divided into 3 equal groups: 1-a control (intact rats), 2-rats from the first day experiment received lincomycin (5 days) and 3rd rats, which received from the first day of lincomycin (5 days) and HFD (21 days). After euthanasia of animals on the 22nd day under thiopental anesthesia (20 mg / kg) by total bleeding from the heart received blood serum

and secreted gums. The serum was determined by the level of biochemical markers of inflammation [11], namely the activity of the proteolytic enzyme elastase [12] and the content of malondialdehyde (MDA) [13], as well as the activity of urease (indicator of bacteremia) [14], the activity of lysozyme [15] and by the ratio of the relative activities of urease and lysozyme – the degree of dysbiosis according to A. P. Levitsky [15].

The gum homogenate (20 mg/ml 0.05 M Tris-HCl buffer pH 7.5) determined the level of inflammatory markers (elastase activity and MDA content), the activity of the antioxidant enzyme catalase [16], and the antioxidant-prooxidant index (API) [11] was calculated by the ratio of catalase activity and MDA content. The gum homogenate determined the content of hyaluronic acid [17], as well as the activity of urease, lysozyme and the degree of dysbiosis according to A. P. Levitsky [15].

The results of the experimental studies were subjected standard processing [18].

RESULTS AND DISCUSSION

In Fig. 1 presents the results of determination in the blood serum of biochemical markers of inflammation, which indicate a significant increase in both indicators, both in the conditions of dysbiosis, and in the conditions of the connecting action of dysbiosis and HFD.

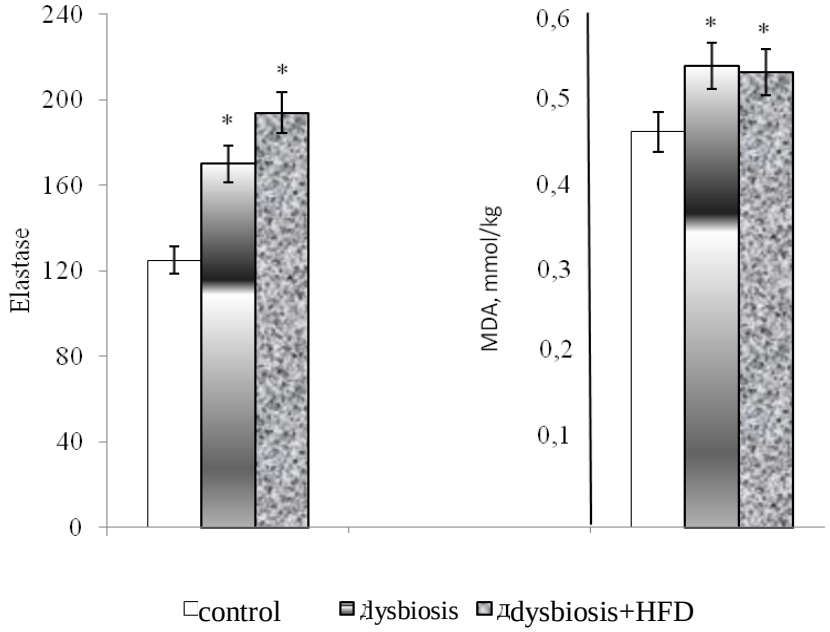

Fig. 1. The level of inflammatory markers (elastase, MDA) in the serum of rats treated with HFD against the background of dysbiosis

In Fig. 2 presents the results of determination in the blood serum of urease, lysozyme and the degree of dysbiosis. From these data it is seen that in rats with dysbiosis and rats with connective action of dysbiosis and HFD significantly (5-6 times) increases both the activity of urease (indicating the development of bacteremia), and the degree of dysbiosis. The activity of lysozyme, on the contrary, is reduced by the action of dysbiosis and, in particular, by the combined action of dysbiosis and HFD.

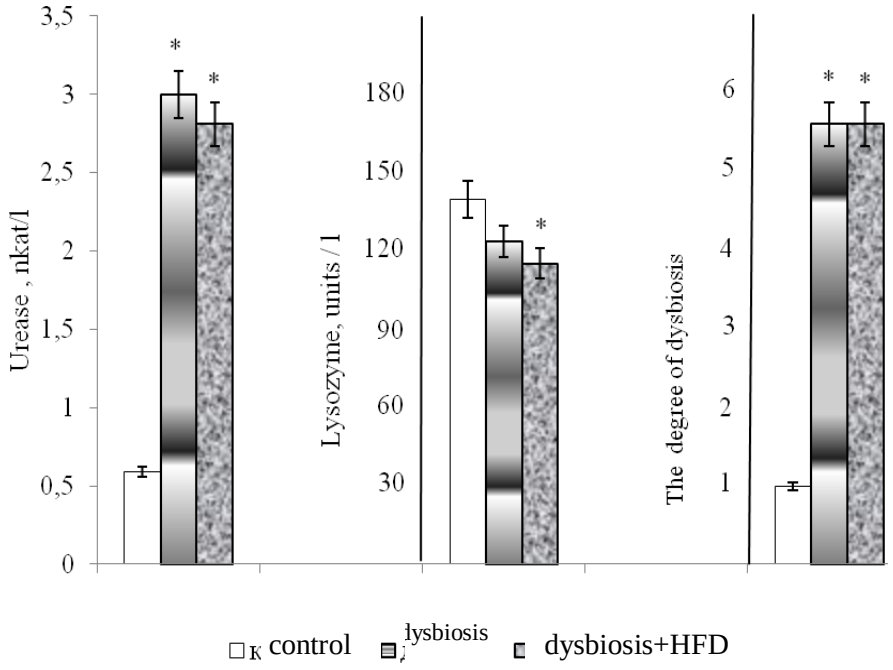

Fig. 2. The activity of urease, lysozyme and the degree of dysbiosis in the serum of rats treated with HFD against the background of dysbiosis

Obtained data on changes in biochemical markers of blood serum give every reason to claim that rats develop systemic inflammation and generalized dysbiosis, which are signs of dysbiotic syndrome.

Table 1 presents the results of determining biochemical markers of inflammation in the gums of rats. These data show that in rats with dysbiosis the level of elastase increases by 23% and the level of MDA by 33%. However, with the combined action of dysbiosis and HFD, the activity of elastase increases significantly, namely by 59%, which indicates an increase in the inflammatory process under the influence of excessive fat intake.

Table 1. The level of inflammatory markers in the gums of rats treated with HFD against the background of dysbiosis (n = 8 in all groups)

Number of group	Groups	Elastasa, mc-kat/kg	MDA, mmol/kg
1	Control	22±4	16,1±1,2
2	Lincomycin dysbiosis (LD)	27±3 p>0,3	21,4±1,9 p<0,05
3	LD + HFD	35±2 p<0,01; p_1<0,05	21,6±1,4 p<0,05; p_1>0,8

Notes: p – in comparison with gr. № 1; p_1 – in comparison with gr. № 2.

Table 2 presents the results of the determination in the gums of the activity of catalase and API index. From these data it is clear that in rats with dysbiosis, as in rats treated with HFD against dysbiosis, only a tendency to decrease in the level of catalase was detected, whereas the level of the API index decreased significantly in both groups. These data indicate disturbances in the gums of the balance of antioxidant and prooxidant systems in favor of the latter.

Table 2. Catalase activity and the antioxidant-prooxidant index of API in the gums of rats treated with HFD against the background of dysbiosis (n = 8 in all groups)

Number of group	Groups	Catalase, mc-kat/kg	API
1	Control	4,84±0,66	3,01±0,45
2	Lincomycin dysbiosis (LD)	4,25±0,64 p>0,3	1,93±0,32 p<0,05
3	JILD+ HFD	4,20±0,54 p>0,3; p_1>0,7	1,94±0,31 p<0,05; p_1>0,9

Notes: see tab. 1.

Table 3 presents the results of determining the content of hyaluronic acid in the gums of rats. As can be seen from these data, under the conditions of dysbiosis or dysbiosis + HFD, there is only a slight tendency to decrease the content of hyaluronic acid. As is known, the latter is an important component of the system that determines the permeability of histo-hematical barriers. A slight decrease in hyaluronic acid may have been associated with a short study period (21 days total).

Table 4 presents the results of determination in the gums of the activity of urease, lysozyme and the degree of dysbiosis. As can be seen from these data, in rats with dysbiosis

urease activity increased by 23.6% (however, p> 0.05), but in rats treated with HFD against dysbiosis, urease activity increased by 39.3% (p <0, 05). These data indicate an increase in bacterial insemination of gums. The activity of lysozyme, in contrast, is significantly (2-fold) reduced in the gums of rats with dysbiosis and even more so in rats treated with HFD against dysbiosis, namely 3.75 times. The decrease in lysozyme activity indicates a significant decrease in nonspecific immunity in periodontium under conditions of dysbiosis and, especially, when consuming HFD against dysbiosis. As a result of these changes, we have a significant increase in the degree of dysbiosis in the gums: 2.22 times in the case of dysbiosis and 5.15 times in terms of consumption of HFD against dysbiosis.

Table 3. The content of hyaluronic acid in the gums of rats treated with HFD against the background of dysbiosis (n = 8 in all groups)

Number of group	Groups	Hyaluronic acid, mg/kg
1	Control	680±51
2	Lincomycin dysbiosis (LD)	615±54 p>0,3
3	LD + HFD	617±52 p>0,3; p_1>0,8

Notes: see tab. 1.

Table 4. The activity of urease, lysozym and the degree of dysbiosis in the gums of rats treated with HFD against the background of dysbiosis (n = 8 in all groups)

Number of group	Groups	Urease, mc-kat/kg	Lysozim units/kg	The degree of dysbiosis
1	Control	0,89±0,10	300±30	1,00±0,17
2	Lincomycin dysbiosis (LD)	1,10±0,10 p>0,05	150±50 p<0,05	2,22±0,26 p<0,05
3	LD + HFD	1,24±0,13 p<0,05; p_1>0,3	80±6 p<0,001; p_1<0,05	5,15±0,58 p<0,001 p_1<0,01

Notes: see tab. 1.

Thus, our research revealed the negative impact of excessive fat intake (in our case, it is palm oil) on the periodontal condition, especially in the presence of dysbiotic effects in the organism.

CONCLUSIONS

1. The use of lincomycin antibiotic causes the development of systemic inflammation and generalized dysbiosis, especially when consuming palm oil against a background of dysbiosis.

2. The consumption of palm oil causes to a greater extent the development of inflammatory processes and dysbiosis in the gums of rats treated with lincomycin, due to a significant decrease in the level of nonspecific immunity and the balance of antioxidant and prooxidant systems in favor of the latter.

REFERENCES

1. Levitsky AP. Lysozyme instead of antibiotics. Odessa, KP OGT, 2005: 74. (in Russian)

2. Polunina TYe, Mayev IV. Clinic, diagnosis and correction of acute drug hepatitis. Therapist. 2007; 1: 88-89. (in Russian)

3. Novik GI, Astapovich NI, Ryabaya NE. The production of hydrolases and antibiotic resistance of lactobacilli and bifidobacilli. Applied Biochemistry and Microbiology. 2007; 43 (2): 184-192. (in Russian)

4. Levitsky AP, Makarenko OA, Tomilina TV [and others]. The experimental methods of reproduction ob immunodeficite conditions: method guidelines. Odessa, KP OGT, 2016: 20. (in Russian)

5. Levitsky AP, Makarenko OA, Demyanenko SA. Methods of experimental dentistry (teaching aid). Simferopol, Tarpan, 2018: 78. (in Russian)

6. Van der Kleij D, Yazdanbakhsh M. Control of inflamatory diseases by pathogens: lipids and the immune system. Eur. J. Immunol. 2003; 33: 2953-2963.

7. Velichko VI, Tkachuk VV, Levitsky AP. Development of dysbiosis in tissues of rats fed with a high fat food. Journal of Health Sciences. 2014; 4(12): 84-92. (in Russian)

8. Khodakov IV, Levitsky AP, Tkachuk VV [and others]. Prodysbiotic action of food fats with high content of palmitic acid. Byulleten' XIV chteniy im. V. V. Podvysotskogo. Odessa, 2015: 200-201. (in Russian)

9. Titov VN. Excess palmitic fatty acid in food is the main cause of lipidosis of insulin-dependent cells: skeletal myocytes, cardiomyocytes, pediportal hepatocytes, Kupffer macrophages and pancreatic β-cells. Clinical laboratory diagnosis. 2016; 61(2): 68-77. (in Russian)

10. Levitsky AP, Makarenko OA, Denga OV [and others]. The experimental methods of restoration and estimation of the degree of dysbiosis in oral tissues. Announcer of stomatology. 2010; 2: 22-23. (in Russian)

11. Levitsky AP, Denga OV, Makarenko OA. [and others]. Biochemical markers of inflammation of oral cavity tissue: method guidelines. Odessa, KP OGT, 2010: 16. (in Russian)

12. Levitsky AP, Stefanov AV. The methods of the determination of the activity of elastase and its inhibitors: method guidelines. Kiev, GFK, 2002:15. (in Russian)

13. Stalnaya ID, Garishvili TG. The method of revelation of malonic dialdehyde with thiobarbituric acid. Moskva, Meditsina, 1977: 66-68. (in Russian)

14. Gavrikova LM, Segen IT. Urease activity of oral liquid in patients with acute odontogenic infection of maxillo-facial part. Stomatology. 1996; The extra issue :49-50. (in Russian)

15. Levitsky AP, Makarenko OA, Selivanskaya IA [and others]. Enzymatic methods for determination of oral dysbiosis for screening pro- and prebiotics: method guidelines. Kiev, GFC, 2007: 22. (in Russian)

16. Girin SV. The modification of the method of the determination of catalase activity in biological substrates. Laboratory diagnostics. 1999; 4: 45-46. (in Russian)

17. Asatiani VS. The new methods in biochemical photometry. Moskva, Nauka, 1965: 543. (in Russian)

18. Truhacheva NV. Mathematical Statistics in biomedical research using application package Statistica. Moscow, GJeOTAR-Media, 2012: 379. (in Russian)