

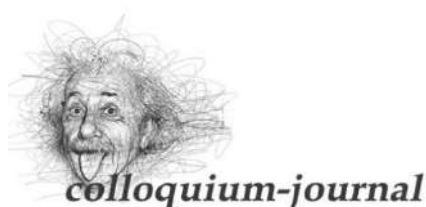


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CHANGES IN THE FUNCTIONING OF THE PERIODONTAL DEFENCE SYSTEM IN PERIODONTAL DISEASES

Abstract.

The study of lipid peroxidation processes and the antioxidant defence system in the pathogenesis of various pathological conditions, including periodontal disease, is currently receiving considerable attention, despite the fact that this topic appears to be well established.

Lipoperoxidation is recognised as a crucial factor in the pathogenesis of the inflammatory process. Reactive oxygen species, such as superoxide anion, hydrogen peroxide, hydroxyl radical, and hypochlorous acid, are by-products of normal cellular metabolism that are constantly and intentionally produced by the body. They play a vital role in the regulation of many essential processes, including phagocytosis, synthesis of group D, E and F prostaglandins, mitochondrial respiration and peroxisomal oxidation.

The same or different reactive oxygen species, depending on the conditions, can both stimulate and inhibit cell division, stimulate the expression of various genes, cell differentiation in embryogenesis and even carcinogenesis, and affect apoptosis by generating chemical signals.

However, excessive free radicals can damage cell membrane structures. The peroxidation of membrane phospholipids results in the accumulation of hydroperoxides, which leads to the formation of clusters and subsequent membrane fragmentation. This process also alters the membrane's permeability to Na and Ca ions. Inactivation of the Ca^{2+} transport enzymes ATPase leads to a slowing down of the calcium efflux from the cell and at the same time to an acceleration of its penetration into the cell, thus damaging it.

Changes in the rate of reactive oxygen species formation can affect peroxidation, which may self accelerate and lead to the complete destruction of unsaturated lipids, disruption of the structure and function of proteins and nucleic acids, and ultimately cell death. These biochemical effects manifest as exudation and proliferation reactions in the inflammatory focus. Intoxication, microcirculatory disorders, autosensitization, and immune response disorders can lead to pathological regulation and inadequate adaptive responses at the organismal level. Clinical studies have also experimentally demonstrated the key role of the antioxidant system in the metabolic correction of the redistribution of energy substrates, its impact on the structural and functional state of membranes and the receptor sensitivity of cells.

Keywords: periodontium, antioxidant protection, periodontitis.

The aim. To assess the state of the antioxidant defence system in periodontal diseases.

Materials and Methods.

During the initial phase of the study, 25 volunteers were monitored to address the issue. Of these, 17 were diagnosed with chronic generalised periodontitis at the time of the study and had no history of other infectious or inflammatory diseases. The remaining 8 subjects formed the control group and were somatically healthy at the time of examination. The study primarily focused on patients aged between 25 and 50 years, with an average age of 45.2 ± 1.4 years.

The study was not suitable for patients with generalised somatic pathology requiring current treatment: the endocrine system (such as diabetes mellitus and thyrotoxicosis), nervous system (such as cerebrovascular disorders and a history of epileptic seizures), genitourinary system, and cardiovascular system, as well as HIV-infected individuals, people with hepatitis or tuberculosis, and patients with a history of allergic reactions to drugs or drug/alcohol dependence.

Blood taken from the ulnar vein at the same time in the morning on an empty stomach was used to study

the intensity of lipid peroxidation and the level of antioxidant enzymes. Plasma was obtained using EDTA, with centrifugation of the blood tube for 10 min at 3000 rpm.

The severity of lipid peroxidation was assessed by measuring the content of hydroperoxides and malondialdehyde in the blood serum (Gavrilov V.B. et al., 1987).

The levels of superoxide dismutase and catalase in gingival blood erythrocytes were used to study antioxidant defence (Chevari S.I. et al., 1991). Additionally, the total antioxidant activity in plasma and erythrocytes was determined using the method developed by S.G. Bislorodov (1986).

The research findings and their discussion.

The analysis of the results indicates that lipid peroxidation products in the blood plasma and the ability of plasma and erythrocytes to cause peroxidation were increased in both patient groups. Additionally, the activity of the main enzymes of antiradical cell defence, catalase and superoxide dismutase, was reduced compared to the control group.

Indicators of lipid peroxidation and antioxidant system in different groups prior to treatment (M±m)

Indicators studied	Groups of researchers	
	main (n=17)	control (n=8)
MDA, mc/ml	4,9±0,2*	1,72±0,2
LHU pl., con.u.	5,6±0,24*	3,14±0,3
SOD, mg/Hb	9,4±0,4*	19,1±0,5
Catalase, mg/Hb	508,0±12,1*	705,8±13,1
AOA pl., %	16,3±1,2*	7,26±1,1
AOA er., %	31,9±1,5*	23,4±1,85

Conclusion. In patients with generalised periodontitis, an increase in lipoperoxidation occurs alongside a decrease in antioxidant defence. This is supported by the results of determining the levels of important antioxidant enzymes, such as superoxide dismutase and catalase. Based on the obtained data, it is noteworthy that patients with purulent exudation from periodontal pockets exhibit a significant increase in lipid peroxidation products in their blood plasma, as well as a decrease in superoxide dismutase, catalase, and antioxidant activity in both serum and erythrocytes.

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