

OXIDATIVE STRESS IN THE PATHOGENESIS OF PERIODONTAL DISEASE

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Periodontal disease is a chronic inflammatory disease caused by gram-negative bacteria, characterized by gingival inflammation and alveolar bone resorption. In the pathogenesis of periodontal disease free radicals and oxidative stress play a significant role. Free radicals are frequently formed as metabolic by-products and their overproduction leads to cell damage and the development of oxidative stress. Antioxidants are substances that reduce the effects of free radicals and represent a specific defense that protects the organism from their harmful effects.

Polymorphonuclear leukocytes (PMNL) are the main immune cells in oral tissue, protecting it from the damaging effects of bacteria. Interactions of leukocytes with bacteria initiate various defensive biochemical and physiological processes that lead to the destruction of the pathogen, but also leading to the respiratory burst in PMNL, with consequential production of free radicals and local tissues damage. Free radicals cause lipid peroxidation in a tissue, DNA and protein damage, enzyme oxidation, stimulation of pro-inflammatory cytokines.

Antioxidants play an important role in the protection of oral tissues from the damaging effects of free radicals. The group of enzymatic antioxidants includes superoxide dismutase, oral peroxidase, catalase and glutathione peroxidase; while nonenzymic antioxidants include uric acid, albumin, vitamin C and glutathione.

Free radicals play an important role in the pathogenesis of systemic diseases and diseases localized in the oral tissues as well. An imbalance between the production of free radicals and salivary antioxidants may trigger oxidative stress the onset of which is suggested as a basis for the development of periodontal disease. *Acta Medica Medianae* 2016; 55(4): 66-72.

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