

## Dental Analysis of Immunological and Microbiological Aspects of the Oral Cavity in Pregnant Women

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**Annotation.** Currently, there is a need for a comprehensive assessment of the impact of hygiene, social, somatic factors, the state of reproductive health and factors occurring during pregnancy on the degree of parodont disease of pregnant women, the strength of their impact on the integrated risk of disease, as well as the determination of the “impact weight” of factors. The initial reason was the existence of a correlation between the development of the disease and the factors that functionally affect its development. To determine the risk factors for the formation of Parodont disease, various reasons were allocated that determine the pathogenetic mechanisms of the development of parodont disease, dividing the authors into the following groups: local; social; factors reflecting the state of reproductive health; somatic health status; obstetrics and neonatal pathology. The initial reason was the existence of a correlation between the development of the disease and the factors that functionally affect its development. Based on the proposed method lies a forecasting model based on The Baes formula for independent characters, Wald sequential analysis as well as a method for evaluating information based on the Kulbach information measure. Predictive coefficients and informativeness of indicators are presented by the authors.

**Keywords:** Immunological, Microbiological, Women

### **Introduction**

Currently, there is a need for a comprehensive assessment of the impact of hygiene, social, somatic factors, the state of reproductive health and factors occurring during pregnancy on the degree of parodont disease of pregnant women, the strength of their impact on the integrated risk of disease, as well as the determination of the “impact weight” of factors. The initial reason was the existence of a correlation between the development of the disease and the factors that functionally affect its development. To determine the risk factors for the formation of Parodont disease, various reasons were allocated that determine the pathogenetic mechanisms of the development of parodont disease, dividing the authors into the following groups: local; social; factors reflecting the state of reproductive health; somatic health status; obstetrics and neonatal pathology. The initial reason was the existence of a correlation between the development of the disease and the factors that functionally affect its development. Based on the proposed method lies a forecasting model based on The Baes formula for independent characters, Wald sequential analysis as well as a method for evaluating information based on the Kulbach information measure. Predictive coefficients and informativeness of indicators are presented by the authors.

The role of microflora in the pathogenesis of Parodont inflammatory diseases is obvious. But the nature, expression and direction of the inflammatory reaction is largely determined by the body's ability to resist the pathogenic microflora of the oral cavity using non-specialized and specialized immune defense factors. The direction of local reactions in inflammatory diseases of parodont, the effectiveness of anti-infection protection depends on the balance between inflammatory and anti-inflammatory cytokine secretion

[2.4.6.8]. In the dynamics of the gestational period, chronic general periodontitis has studied the cytokine profile in the oral fluid of pregnant women, as well as the effect of cytokine dysbalance on the ratio of T-cell subpopulation in the blood. A cytokine profile dysbalance in oral fluid when the number of inflammatory cytokines increases in pregnant women with chronic general periodontitis in the middle weight level has been found to trigger formation in the 2nd trimester of pregnancy and to escalate in the 3rd trimester of the gestational age.

Two pathogenetic mechanisms can explain the potential effects of periodont diseases on the end of pregnancy – periodont bacteria form a bioplyonka and directly affect the fetoplasental system, and inflammatory mediators lead to the development of an inflammatory response. The placenta was studied and the placental microbiome was noted to be in many ways compatible with the oral microbiome, as opposed to the microbiomes of the gut, nasal cavity, skin, urogenital tract. Although bacteria in the oral cavity do not fall permanently into the circulation of the system, daily cleaning of teeth, the use of dental floss, as well as dental practices can lead to transitor bacteremia. Therefore, the "mother-fetus" system is prone to the risk of bacterial invasion, in which not only with the saprophytic flora of the oral cavity, but also with pathogenic, bacteria capable of translocating to the placenta. High DNA levels of pathogenic flora detected in the oral cavity of patients with preeclampsia have been reported. P in placental tissue. the presence of antigens to gingivalis is established and P. it has been suggested that gingivalis colonization leads to placental dysfunction. P. the presence of gingivalis has been associated with preeclampsia and other complications during pregnancy. Due to the high concentration of glucose in the saliva (0.44 to 6.33 mg of glucose in 100 ml of saliva, 0.24-3.33 mg in moderation), rapid bacterial growth as well as tartar formation is noted. In the periodontal pockets *Staphylococcus aureus*, *Enterobacter* spp., *Pseudomonas aeruginosa*, *Fusobacterium necrophorum*, *Peptostreptococcus micros*, *Streptococcus intermedius*, *Streptococcus haemolyticus*, *Candida albicans*, and intact cause an aggressive course in parodont where microflora is difficult to recover. Bacteria, their metabolic products, and a large proportion of inflammatory mediators enter the systemic bloodstream. The infection present in the periodont can affect the course of pregnancy not only through the direct entry of microorganisms into the placenta, but also through the endotoxins (lipopolysaccharides) of the bacterial cell [1.3.5.7.9.11].

The pathogenetic mechanism of this effect consists in an increase in the formation of prostaglandins and cytokines (PGE<sub>2</sub>, IL-1 $\beta$ , TNF $\alpha$ , IL-6) in the inflamed periodont, which leads to an increase in the concentration of these substances in the blood plasma. Scientific studies confirm a correlation between increased levels of IL-6 in the maternal blood and in women at risk of premature birth in amniotic fluid, which makes it possible to consider IL-6 as an indicator of preterm birth risk. It is hypothesized that the synthesis of inflammatory mediators in areas where infections accumulate in the oral cavity as well as their release into the bloodstream and then into the amniotic fluid can lead to premature birth. In addition, these substances can be synthesized directly in the placenta after being triggered by endotoxins [10.12.13]. It is believed that the highest nonpersonalization of pH in the oral cavity is caused by the metabolic breakdown of microflora products containing carbon – a metabolic explosion. The peak of this explosion corresponds to the places where microorganisms accumulate – tooth and tongue Carache. In addition to food products and microflora, there is a pH-size salivation effect in the oral cavity, ion exchange in the "oral fluid – enamel" and "oral fluid – tooth decay" systems, and the functional activity of the salivary glands;glazes. Chronic general periodontitis during pregnancy in the III trimester, the disease is exacerbated by an increase in the level of severity, a deterioration in the state of hygiene in the oral cavity, an increase in indices reflecting the condition of gums and periodont hard tissues. Increased levels of IL-1 $\beta$ , IL-6, and FNO- $\alpha$  in the oral fluid of pregnant women activate the inflammatory-destructive processes that disrupt parodont and create conditions for an outbreak of dental disease. This pathology exists the determination of the concentration of cytokines in the oral fluid of pregnant women makes it possible to have additional evaluation criteria to distinguish between patients at high risk of disease outbreaks.

Foci of infection in the oral cavity are a risk for pregnant women who have TEA, in which a decrease in the body's colonizing resistance to infection factors occurs. But microorganisms themselves are not considered the cause of the disease, but only in the case of exposure to macroorganisms, when certain negative conditions are harmonized, become the cause. Lactoferrin concentrations were found in oral fluid of 35 pregnant women with dental caries. Lactoferrin levels in oral fluid in the absence of dental diseases in patients with physiological pregnancy are higher than in healthy donors ( $1.06 \pm 0.08$  mcg/ml) and increase from I ( $2.31 \pm 0.11$  mcg/ml) to II ( $2.79 \pm 0.13$  mcg/ml) and III trimester ( $3.11 \pm 0.24$  mcg/ml). In pregnant women suffering from dental caries, lactoferrin levels in oral fluid compared to the control group are 27.6; 37.4 in the I, II and III trimesters, respectively; 19.2% higher. Lactoferrin levels in saliva increase in the presence of dental caries as well as in relation to the duration of the gestational period [11.12.13]. Harmonious increase in lactoferrin and C-reactive protein levels in the saliva of patients with mild to moderate periodontitis has been an indicator of the high pathogenetic importance of dental disease during pregnancy. In pregnant women suffering from dental caries, the marker of the activity of this process was lactoferrin. CRO levels in saliva became associated with dental pathology.

**Conclusion.** Lactoferrin and defenzins and cathelicidins in oral fluid of 207 pregnant women with dental caries at different gestational periods provided results of measuring levels of antibacterial peptides. During the gestational period in pregnant women, there was a sequential increase in LL 37 lactoferrin as well as cathelicidine in the oral fluid, which prevented the development of dental caries. The development of dental caries during pregnancy was observed in the 2nd trimester with an increase in the level of all three factors of congenital immunity in the oral cavity, followed by a decrease in 1-3 defenzins and LL 37 cathelicidin in the oral fluid. It follows that the completion of 1-3 defenzins as well as LL 37 catalycidine secretions into the oral cavity by epithelial cells and neutrophils caused the development of dental caries.

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