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STUDY OF MOLECULAR GENETIC CRITERIA FOR THE OCCURRENCE OF GUM RECESSION (LITERATURE REVIEW)

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ANNOTATION

Nowadays a huge variety of polymorphic genes associated with a particular pathology has been identified. Patterns of distribution of a number of genotypes of the same polymorphic genes and their associations with various diseases are revealed in different pathologies. The individual characteristics of the immune system are determined by the genetic characteristics of the organism mediated by polymorphism of the genes that are in charge of formation of the immune response. In this regard, the study of gene polymorphism would allow to determine susceptibility to gum recession, establish the relationship between genetic factors and causes, and develop individual programs for diagnosis, prevention and improvement of complex treatment of the disease in future.

Key words: *gum recession, multifactorial pathology, gene polymorphism, disease.*

The results of modern studies of the human genome and the identification of genes whose polymorphism predisposes to the most common MDs make it possible to determine with a high degree of probability a person's predisposition to a particular disease. Of greatest practical importance is the analysis of polymorphisms of candidate genes, affecting the function of encoded proteins and contributing to the development of the pathological process under the influence of external factors. Compilation of a

gene network for each MD based on knowledge of its etiology and pathogenesis, identification of central genes and modifier genes in it, study of intergenic and gene-environmental interactions, the development on this basis of a set of preventive and therapeutic measures for each patient constitutes the strategic basis of a new, rapidly developing direction - predictive medicine [2]. Moreover, in different pathologies, patterns of distribution of a number of genotypes of the same polymorphic genes and their association with various diseases are revealed. This is due to the fact that the product of virtually every gene is involved, as a rule, in several, and sometimes in very many processes that form the metabolic network of the body, realizing the pleiotropic properties of genes. In other words, each individual gene polymorphism can be associated with several different pathologies, as proven by large-scale analysis of SNP genes associated with a number of MDs. There is no doubt that pleiotropy is natural for genes and is associated with the characteristics of the disease. Analysis of gene pleiotropy is an important step towards understanding the mechanisms of development of pathologies, classification of diseases and identification of molecular targets for the development of new drugs. However, in addition to pleiotropy, when analyzing genetic association with a disease, it is necessary to take into accountthe concept of polygenicity – the combination of the influence of products of different genes and their network interactions on the development of the disease. Individual genetic polymorphisms are a weak risk factor for the development of the disease and cannot be used as a prognostic model for the development of MD, especially in cases of rare alleles. But it is well known that a combination of unfavorable alleles is dangerous for the occurrence of many MDs several genes with an additive effect, so the identification of such polymorphisms is of great importance. Any single gene polymorphism explains 1–8% of the total disease risk in population, which may seem insignificant, but the additive effect of several such risk factors can account for up to 20–70% of the total risk 25 due to genetic factors. This is important to consider when the stage of assessing the complex influence of polymorphic gene products on pathologies with the aim of creating a panel of molecular markers for prognosis, early diagnosis and clinical course

of diseases, especially those of a multifactorial nature [3]. In addition, when analyzing the MFZ one cannot ignore external factors. From the point of view of genetic analysis, most of the most common human diseases and traits of medical significance are extremely inconvenient because they do not follow a simple Mendelian pattern the principle of monogenic inheritance. The most common human diseases are the result of the action of many genetic factors in combination with environmental factors and random causes, i.e. they have a multifactorial nature. All main causes fall into the category of multifactorial signs morbidity and mortality in modern human populations: atherosclerosis, hypertension, many forms of cancer, mental illness, diabetes, bronchial asthma, rheumatoid arthritis, a hereditary component of susceptibility to infectious diseases and, probably, a significant component of the general aging process [4,5]. It is in this aspect that various research teams, both in our country and abroad, are currently implementing a complex molecular genetic studies aimed at identifying a panel of specific genotypes associated with various MDs, and also with the nature of their course and the risk of complications. Systematization of the results of studying the genetic basis of widespread diseases brings researchers ever more convincingly closer to the validity of the assumption that often clinically different diseases may be controlled by a common set of susceptibility genes. Thus, over the past decade Numerous foreign and domestic studies have accumulated a significant amount of data on the involvement of various polymorphic genes in the formation of predisposition to multifactorial pathology; it has been shown that complex interactions of genetic and environmental factors underlie the occurrence of MD. Gum recession also has a multifactorial nature and develops against the background of predisposing anatomical features, such as a thin gingival biotype, in the presence of trauma, with aggressive teeth cleaning techniques, functional trauma, and can be the result of progressive inflammatory-destructive processes in periodontal tissues, as well as as a result genetically determined incorrect ratio of the size, shape (signs of curvature) of the roots in relation to the thickness of the bone of the alveolar process of the jaw. Etiopathogenetic basis of diseases periodontal disease, including gum recession,

constitute functional weakened allelic variants of certain genes. This genetic effect can be realized against the background of unfavorable factors external environment. In children of primary school age (6–11 years), localized gum recession in the area of the anterior group of teeth with root exposure up to 3–5 mm is very often observed. The main causes Gum recession in children was previously considered to be trauma to the gingival margin during improper brushing of teeth and bad habits (biting nails and pens). But this is not entirely true; these reasons can only aggravate the process, they are superimposed on the true causes of gum recession, which are anatomical and physiological features (small vestibule of the mouth, massive muco-epithelial cords and frenulums, dentofacial anomalies, thin gum biotype), bruxism (muscle hypertonicity) and iatrogenic factors (chemical burn) [6]. Thus, the manifestation and progression of signs of gum recession depends on many factors, including the individual characteristics of the subject, social, behavioral, systemic, and genetic factors. In the presence of gum recession, patients are concerned about impaired aesthetics, increased sensitivity, cervical defects and inflammation. A large number of extra- and intracellular proteins are involved in the regulation of inflammation. It is known that the genes encoding the structure of these proteins are polymorphic in various regions, which affects the functional activity of the encoded proteins and the level of their production inflammatory cells processes. The study of polymorphism of protein genes involved in the regulation of inflammation processes will identify genetic risk factors for the development of gum recession. In recent years, the interest of researchers has been aimed at analyzing the role of genes that regulate inflammation, primarily the genes of cytokines - interleukins (IL) with anti- and pro-inflammatory activity, and genes of matrix metalloproteinases [7,8]. Cytokines are a group of non-enzymatic hormone-like proteins and peptides involved in the induction of inflammation and immune response. They represent a group of polypeptide mediators of intercellular interaction; they participate in the regulation of various physiological functions and processes of tissue regeneration when their integrity is violated [9]. IL-1β is a secretory cytokine that acts locally and at the systemic level. When tissue is

damaged, IL-1β causes activation of all cell types, participating in the formation of a local inflammatory reaction (fibroblasts, macrophages and blood leukocytes). IL-1β has stimulating effect on connective tissue metabolism by stimulating fibroblast proliferation. Cytokine genes have a high degree of polymorphism. Since cytokines are mediators of inflammation, the study of genes that control their activity is a promising task in studying the mechanisms of development, the course of many diseases, and identifying predisposition to them. It is known that the 511C/T polymorphism of the IL-1β gene (rs16944) plays an important role in the functioning ofimmune system and may be one of the main genetically due to the causes of pronounced dysregulation of inflammation, it has a significant impact on the general features of the course of the inflammatory response in periodontal tissues in patients [10].

CONCLUSION. The problem of studying molecular genetic criteria leading to gum recession in children as a multifactorial pathology is a very urgent task. In this regard, the study of gene polymorphism would make it possible to clarify the predisposition to gum recession, establish a connection between genetic factors and the causes of its occurrence, and also, in the future, develop individual programs for diagnosis, prevention and improvement of methods for complex treatment of this disease.

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