

GENETIC PREDISPOSITION TO PERIODONTITIS EXTENDS FROM INDIVIDUAL POLYMORPHISMS TO POLYGENIC RISK MODELS

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ABSTRACT

Genetic predisposition interacts with immune, microbial, behavioral factors in periodontitis, a common multifactorial disease. Variations in diagnostic criteria, study population characteristics, approaches to phenotyping the disease have complicated comparisons of genetic markers. The study aimed to systematize, critically evaluate current data on genetic predisposition to periodontitis, trace the shift from studying individual candidate gene polymorphisms to polygenic risk models, assess the prospects of using these models to predict disease onset, progression. An analytical literature review formed the basis of this work, incorporating a systematic search, critical qualitative assessment of relevant scientific publications. The analysis revealed that individual candidate gene polymorphisms—including variants associated with cytokines, innate immunity, antimicrobial defense, and bone remodeling—failed to show consistent reproducibility across different populations. While genome-wide association studies (GWAS) identified new genetic loci, no single variant common to all studied cohorts was found to serve as a universal risk marker. Polygenic risk scores emerged as a more promising approach by accounting for the cumulative contribution of multiple genetic variants; however, their clinical utility remained dependent on population ethnicity, the characteristics of the original GWAS, and the quality of external validation. Limitations in the evidence base included phenotypic heterogeneity, variations in sample size and composition, and differences in genotyping methods and statistical approaches. The study concludes that polygenic models should not be used as standalone diagnostic tools but rather as part of a comprehensive assessment integrating both genetic and clinical risk factors.

KEYWORDS: *periodontitis; genetic predisposition; genetic polymorphism; candidate genes; genome-wide association studies; polygenic risk score; genetic factors.*

Introduction

PERIODONTITIS is one of the most common chronic diseases of the oral cavity and is characterized by inflammatory damage to the tissues holding the tooth. Its severe forms lead to progressive loss of gingival attachment and alveolar bone, and in the absence of effective disease control, they can cause tooth loss. According to the World Health Organization, about 1 billion people suffer from severe periodontitis, which determines its importance not only for dentistry, but also for public health. Modern concepts of the disease associate its development not with the action of a single causal factor, but with a combination of microbial, immune, behavioral, environmental and genetic characteristics of the body [3, pp. 32-59; 28].

Recent Russian studies confirm the complex nature of the interaction of factors determining the development and course of periodontitis. E.A. Tikhomirova, who systematized data on genetic predictors of the disease, showed that the research covers several functionally different groups of genes, including genes for interleukins, defensins, Toll-like receptors and proteins involved in tissue remodeling. At the same time, the presence of a certain allele is not considered as an independent diagnostic sign: it only changes the likelihood of developing the disease [3, pp. 32-59]. M.A. Gross also notes the association of polymorphisms of proinflammatory cytokine genes with the risk and severity of inflammatory and destructive periodontal diseases, however, the results of studies of various genetic variants remain heterogeneous [2]. Some domestic works in recent years have complemented this picture by studying the molecular and immunological mechanisms of the disease, which allows us to consider the genetic predisposition in close connection with the state of local immunity, microbiota and tissue metabolism [4,5]. A comparison of these studies shows that the expansion of the list of studied genes is not accompanied by the same consistent confirmation of their prognostic value. The biological relationship of the genetic variant with the inflammatory process explains the possibility of its involvement in the pathogenesis,

but does not prove its suitability for assessing individual risk. Therefore, further analysis requires distinguishing the molecular mechanisms of the disease and the clinical significance of the identified associations.

Individual polymorphisms of candidate genes were initially studied most actively in studies of genetic susceptibility to periodontitis. The focus was on variants of the genes IL1, IL6, IL10, TNF, IL1RN, TLR, DEFB, VDR, MMP and a number of other regulators of the inflammatory response and bone remodeling [3, pp. 32-59; 10; 26]. The alleged mechanism of their influence is associated with changes in the intensity of the immune response, cytokine production, activity of proteolytic enzymes, or bone resorption processes. However, the presence of a statistical association between a specific polymorphism and periodontitis is not always reproduced in other populations. The result may be influenced by the ethnic origin of the subjects, diagnostic criteria, severity of the disease, sample size, concomitant diseases, and the interaction of genetic variants with smoking, oral hygiene, and other environmental factors [3, pp. 32-59; 10; 26].

This problem is especially noticeable when comparing the results of modern genome-wide studies. C. Gao, M. Iles, H. Larvin et al. In a systematic review of 15 GWAS published before 2023, 11 variants were identified that reached the generally accepted threshold of genome-wide significance, but no common SNPs were found reproducibly in all studies [18]. The authors attribute the discrepancies primarily to differences in sample sizes, population composition, and the criteria used to determine periodontitis. In the work of S. Schaefer, L. Nibali et al. It has also been shown that modern genetic research is gradually shifting from the search for individual candidate genes to a comprehensive study of genomic variants and related biological pathways [37]. A larger study based on the UK Biobank conducted by A. et al., revealed new genetic loci associated with periodontitis, however, the authors separately point out the limitation associated with the use of self-reported signs of the disease [19].

Consequently, the accumulation of data on individual polymorphisms did not in itself lead to the emergence of a universal genetic marker of periodontitis. A review by E.A. Tikhomirova back in 2022 indicated the lack of an accurate genetic predictor of the disease [3, pp. 32-59], and a more recent systematic analysis by H. Dommisch, D. Hoedke, E.M.-C. Lu et al. He has shown that existing genetic markers do not yet provide clinically reliable prediction of the development, progression or stabilization of periodontitis [13, pp. 182-210]. At the same time, modern data create prerequisites for a different approach: to evaluate not the isolated effect of a single SNP, but the cumulative effect of a large number of genetic variants with a small individual contribution to the risk of the disease.

In recent years, there has been renewed interest in polygenic risk scores (PRS), which combine multiple genetic variants into a single quantitative assessment of a person's hereditary predisposition. The approach corresponds to the polygenic nature of periodontitis, allows taking into account the cumulative contribution of variants, each of which individually may have a weak association with the disease [13, pp. 182-210; 18; 37]. At the same time, the use of PRS in periodontology is still at the stage of forming an evidence base. They determine how stable the models are in different populations, whether the estimates obtained are reproduced using standardized criteria for periodontitis, and whether adding a genetic indicator increases the accuracy of the prognosis compared to traditional clinical factors [13, pp. 182-210; 28; 34].

In this regard, a consistent transition from the study of individual polymorphisms to polygenic risk assessment models is of interest. Consideration of this evolution makes it possible not only to summarize the accumulated information about the genetic predisposition to periodontitis, but also to identify the causes of inconsistency in the results of individual studies, evaluate the possibilities of GWAS and PRS, and establish prospects for integrating genetic, clinical, and environmental factors into individual disease prognosis.

The aim of the study is to systematize and critically evaluate current data on the genetic predisposition to periodontitis, to trace the transition from the study of individual polymorphisms of candidate genes to polygenic risk models and to determine the prospects for their application to predict the development and progression of the disease.

MATERIALS AND METHODS

The present study is performed as an analytical review of the literature, a systematic search for publications.

The search was conducted in PubMed/MEDLINE, Web of Science, Scopus, eLibrary and CyberLeninka. The literature lists of the included systematic reviews and relevant studies were also reviewed. The main period is 2021-2026. Earlier publications were included with their fundamental importance. This concerned the assessment of the heritability of periodontitis and ideas about its genetic architecture.

The queries were formulated in Russian and English: "periodontitis", "periodontal diseases", "genetic predisposition", "genetic polymorphism", "candidate gene", "periodontitis", "periodontal disease", "genetic susceptibility", "genetic polymorphism", "candidate gene", "SNP", "GWAS", "polygenic risk score", "PRS", "genetic risk", "gene-environment interaction" – with logical operators AND/OR.

Human studies on genetic susceptibility to periodontitis met the selection criteria. This included work on candidate gene polymorphisms, as well as genome-wide association studies (GWAS), genetic biomarkers, polygenic risk indicators (PRS), and the interaction of genetic and clinical factors. In addition, there are systematic reviews and meta-analyses. They contained generalized data on genetic associations and their reproducibility.

Publications with clearly defined diagnostic criteria for periodontitis were appreciated. The characterized samples and the described genetic variants were appreciated. For PRS work, information about model validation was required. Also – about their prognostic characteristics. Animal and in vitro studies, isolated clinical observations, non-peer-reviewed publications, and duplicate work were excluded. Studies with a vaguely defined phenotype were also excluded. Or without data to assess the relationship of genetic factors to the risk, severity, or progression of the disease.

Conducting our own meta-analysis is considered impractical. The studies differ too much from each other: according to diagnostic criteria and severity of periodontitis, according to the ethnic composition of the samples, according to the set of studied genetic variants, methods of genotyping, controlled factors and endpoints [13, pp. 182-210; 18]. The inexpediency of conducting our own meta-analysis with such a heterogeneity of the initial work was recognized in advance.

The publications were evaluated critically. According to the study design, sample size, quality of phenotype determination, characteristics of the control group, correctness of statistical analysis, consideration of age, smoking and concomitant diseases, reproducibility of the associations obtained and the presence of independent validation.

Heterogeneity was also observed within individual types of work, and in different ways. GWAS also had different sample sizes and approaches to phenotyping. The set of discrepancies for PRS was different: the initial GWAS, model construction algorithms, characteristics of target populations, and moreover, the discriminatory ability, calibration, and predictive value of the model compared to clinical risk factors were evaluated.

The genetic component of periodontitis susceptibility

The periodontopathogenic microbiota does not act on its own. Tissue damage is determined by the interaction of microbial load and individual body response. The same level of microbial exposure in different patients causes disparate damage. Genetic factors are responsible for the features of the innate and adaptive immune response [3, pp. 32-59; 37]. Responsible for the regulation of inflammation, remodeling of the extracellular matrix and bone metabolism.

The scale of this genetic component was estimated by S. Haworth, A. Esberg, P. Lundberg et al. Based on a cohort of 11,974 adult twins. The heritability of quantitative signs of periodontal disease was moderate. According to some indicators, 41.5–48.3%. For the categorical definition of the disease – 42.6–45.2%. These data support the idea of the genetic component of the risk, but at the same time show that heritability is not identical to direct transmission of the disease.

Heritability is the proportion of variation in a trait. Related to genetic differences in the studied population. It is not the probability of a disease in a particular patient. Values around 40-50% cannot be directly used to calculate individual risk. This requires models that take into account both genetic and clinical characteristics.

The genetic profile sets a certain level of susceptibility. It is realized in combination with age, microbial stress, smoking, metabolic disorders and other external influences. Russian data also consider periodontitis as a disease with a polygenic nature. Predisposition here is not an independent cause, but a change in the likelihood and severity of the disease when exposed to external and microbial factors. Therefore, further study focused on identifying specific genetic variants. Able to explain the differences in individual susceptibility.

This corresponds to the modern understanding of the disease. Genetic predisposition does not replace clinical assessment, but represents one of the components of a multifactorial risk model.

Polymorphisms of candidate genes and their relation to the risk and severity of the disease

The first stage of the search for a genetic predisposition to periodontitis was based on the analysis of candidate genes. These were genes whose function indicated involvement in inflammation and tissue destruction. The focus was on polymorphisms of pro-inflammatory and anti-inflammatory cytokine genes. According to M.A. Gross, the genes of regulators of innate immunity, antimicrobial protection, and bone metabolism were also considered in early publications [2 3 10]. The approach had a biological rationale. The products of these genes are involved in the regulation of the inflammatory response, osteoclast activity, and the destruction of the extracellular matrix.

E.A. Tikhomirova adds to the list the genes of defensins, Toll-like receptors, and type I collagen $\alpha 1$ chain. The genetic model goes beyond the cytokine response [3, pp. 32-59]. The list of studied variants has significantly expanded beyond the classical cytokine genes. The polymorphisms ICAM1, CCL2, and SRXN1 have already been included in the analysis, as have the genes of the complement system, matrix metalloproteinases, regulators of epigenetic processes, and genes associated with metabolic disorders [5, p. 1324-1337; 7; 11; 23; 30, pp. 363-368; 32; 36, pp. 1626-1634; 38].

At the same time, the stability of associations varies. The same SNP may show an association with periodontitis in one population and not be confirmed in another. The result is influenced by ethnic origin, sample size, clinical definition of the disease, its stage and severity, as well as the presence of smoking and systemic diseases [3, pp. 32-59; 28]. Direct comparison of the results is difficult due to these differences. Individual SNPs cannot be considered as universal risk indicators.

To clarify the nature of these limitations, it is advisable to compare the main groups of candidate genes according to their intended functions, studied clinical parameters, and reasons for ambiguous interpretation. This comparison is presented in table 1.

Table 1 - The main groups of genetic variants studied in periodontitis

A group of genes	Examples of genes	Intended role	The indicator under study	The main problem of interpretation
Pro-inflammatory cytokines	IL1A, IL1B, IL6, TNF	Regulation of inflammation and bone resorption	The risk and severity of periodontitis	Variability of associations between populations
Anti-inflammatory mediators	IL10, IL1RN, TGF β	Limiting the inflammatory response	Susceptibility and flow	Effect dependence on genotype and phenotype
Innate immunity receptors	TLR2, TLR4	Recognition of microbial components	Inflammatory reaction	Influence of microbial composition
Antimicrobial protection	DEFB1	Mucosal protection and microbiota control	Disease risk	Limited number of replication studies
Tissue remodeling	MMP	Degradation of matrix components	Loss of attachment and bone tissue	Dependence on the stage of the disease
Bone metabolism	VDR and related paths	Regulation of bone metabolism and immune response	Severity and progression	Multifactorial phenotype

A comparison of the presented groups shows that the gene may actually be involved in inflammation or tissue remodeling. Nevertheless, the relationship of its polymorphisms with the clinical picture of periodontitis is not always reproduced. The comparison of the presented groups clearly shows this. The main limitation here is the dependence of the detected associations on the population. It is also important how the disease itself is defined. Change the sample or the criteria for diagnosis, and the association is not repeated in other material. Although it is stable in one thing.

Hence the practical conclusion. The pathogenetic significance of the candidate gene must be distinguished. And its suitability for risk forecasting. These are different estimates, and the first one cannot be mechanically transferred to the second one.

Why have individual polymorphisms not become universal markers of periodontitis?

The problem with the candidate-gene approach lies not so much in the absence of biologically plausible associations as in their insufficient reproducibility. There is no exact genetic marker of periodontitis. E.A. Tikhomirova came to this conclusion in 2022. There were already many studied variants by that time [3, pp. 32-59]. Four years later, the picture has not changed. H. Dommisch, D. Hedke, E.M.-K. Lu, A. Schaefer, G. Richter, J. Kang and L. Nibali analyzed 607 works in a systematic review of 2025. And we have not found a single genetic test with proven clinical value: neither for diagnosis, nor for predicting progression, nor for assessing the resolution of the disease [13, pp. 182-210].

By itself, the statistical association between SNP and disease does not provide sufficient sensitivity and specificity – for an individual prognosis, it is impossible to classify a patient as a high-risk group based on it. However, the absence of a reproducible association does not always mean that there is no biological effect of the variant. Discrepancies between studies arise due to insufficient statistical power, differences in diagnostic criteria, and unequal consideration of related factors: a negative result of an individual work is evaluated based on design, and is not read as a definitive refutation of a genetic link.

The inconsistency of the results has a more general explanation – the polygenic nature of the disease. In common forms of periodontitis, a single variant usually has a small effect: the final phenotype consists of the action of many variants. Genetic predisposition is realized in a certain environment [28; 37]. Smoking, diabetes, age, the state of the microbiota, oral hygiene, and other factors can change the likelihood of a genetic risk. In the review of 2025, A. Schaefer, L. Nibali emphasizes that common forms of periodontitis are characterized by numerous variants with small effects. Rare variants with a strong effect are more often associated with early or monogenic forms [37]. Hence the limited prospect of further searching for one "main" polymorphism. A more productive direction is the assessment of the total genetic contribution.

Genome-wide studies and the transition from candidate genes to a comprehensive assessment of genetic risk

Genome-wide association studies (GWAS) have changed the principle of searching for genetic factors of periodontitis. Unlike pre-selected candidate genes, this approach simultaneously checks a large number of variants, and the association can be identified without any prior assumption about the mechanism of the disease. Tsch. Gao, M. Isles, H. Larvin, D.T. Bishop, D. Bunce et al. In a systematic review in 2024, 15 GWAS of periodontitis were collected. Eleven variants have reached the generally accepted level of genome-wide significance [18]. There were no common SNPs reproducible in all studies. At the same time, the studies themselves differed markedly in phenotyping, sample structure, and analysis methodology [37]. GWAS has expanded the list of potential genetic factors. And at the same time, they showed how difficult it is to identify a stable single marker suitable for the clinic. The result obtained is crucial for the development of the topic in this dual sense.

A statistically significant locus is not the cause of the disease. This option can work as a risk indicator, a statistical marker. And at the same time not to participate in the pathological process. The distinction here is not a formality, but a practical issue. Functional studies of the associations found are needed to explain the mechanism of the disease. The prognosis is different: the suitability of the marker is tested separately on independent clinical samples, and its reproduction in other populations is required [13, pp. 182-210; 18].

The polygenic forms of the disease in Schaefer and Nibali received specific gene content: SIGLEC5, PLG, ROBO2, ABCA1, PF4, CTSC. All of them are associated with the maintenance and repair of periodontal tissues, fibrinolysis, removal of cellular detritus, remodeling of the extracellular matrix, and angiogenesis [37]. These are no longer individual SNPs, but functional genetic pathways. It is in this vein that the interpretation of GWAS is gradually shifting.

Another direction is the British cohort of UK Biobank. The expansion of genomic analysis has yielded new loci. But the phenotype here is self-reported, and this remains a significant limitation. This is essential when interpreting the results [19]. Actually, the problem statement grows out of these two lines. The sequential search for single loci has exhausted itself. The accuracy of the genetic assessment requires consideration of the combined action of multiple variants. The answer was polygenic models. They allow us to quantify the total hereditary predisposition.

Polygenic risk models for periodontitis

The polygenic risk score (PRS) summarizes the effects of multiple genetic variants – the contribution of each of them to the development of the disease individually may be small.

H. Dommisch, D. Hedke, E.M.-K. Lu et al. They see the options as the basis for future PRS construction. Variants have been identified in GWAS. At the same time, there is currently no genetic test. A test suitable for routine diagnostics [13, pp. 182-210]. Hence the limitation: PRS is not a substitute for a clinical diagnosis. This is a quantitative indicator of the hereditary component of risk. And that's exactly how it should be viewed, nothing more. It is no longer technically possible to calculate polygenic risk itself – the question today is whether the indicator is transferred between populations and adds something to the forecast against the background of traditional risk factors.

The clinical implementation is not limited to the statistical relationship of PRS with the disease. It is required that the inclusion of a polygenic indicator in the model increases its discriminative ability. An improvement in calibration and a clinically significant change in risk stratification are also required compared to a model based only on clinical and behavioral features [28; 34].

Statistically significant improvement in the prognostic model, however, does not necessarily change clinical decisions. The real value of the indicator is checked in another way: whether its inclusion allows you to more accurately identify patients who need more intensive monitoring or prevention. Without such a comparison, the complication of the model may remain without practical effect, and this concerns precisely the practical effect, and not the ability of the model to separate groups from each other. The search for a single marker is replaced by the construction of an integrated sensitivity model. And it's not just a change of method. This is a change in the object of research itself.

G. Richter et al. in the work of 2026, we checked this area directly. A polygenic model based on GWAS data for severe periodontitis. The European sample. 4243 cases. The authors attribute the limitations of existing models to two things – small samples and heterogeneity of phenotypes in the GWAS themselves [28]. However, the problem is broader than a single "sample-phenotype" pair. The indicators calculated mainly on European material are not always directly transferred to other populations. This is also indicated by modern reviews [13, pp. 182-210, 28, 34]. A full-fledged polygenic model must undergo external validation, take into account the origin of patients, and be evaluated along with clinical and behavioral factors.

The differences between the considered approaches are not limited to the number of options analyzed. The nature of the information received, the requirements for the initial data, and the possibilities of clinical application are changing. These features are systematized in table 2.

Table 2 - Evolution of approaches to assessing genetic risk in periodontitis

Approach	What is being evaluated	Advantage	The main limitation	Modern meaning
One SNP	A separate genetic variant	Simplicity	Very little effect	Limited
The SNP Panel	Multiple variants of Pre-selected genes	Takes into account several paths	There is no single standard	Research
Candidate genes	The genome as a whole	Biological validity	The risk of missing unknown loci	Historically significant
GWAS	The cumulative effect of multiple options	Unbiased search	Large samples and heterogeneity of phenotypes	The basic research approach
PRS	Corresponds to a polygenic nature	External validation is needed	A promising direction	
PRS + Genetic and Multifactorial risk			Complexity of	The most

clinical factors	environmental factors	assessment	construction and standardization	promising model
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The table shows that the transition to more complex models consistently expands the possibilities of genetic analysis, but at the same time increases the requirements for the quality of the source data, population characteristics and external validation. Therefore, the prospects of PRS are determined not by the number of options included, but by its ability to improve risk assessment compared to clinical models. The next stage of research involves combining a genetic indicator with clinical and modifiable risk factors.

Clinical significance and prospects of integration of genetic, clinical and environmental factors

The purpose of genetic risk assessment is to identify patients. In such patients, prevention should be initiated before pronounced clinical manifestations or conducted more intensively [1]. Russian works are already showing something important. Molecular genetic and clinical characteristics can be analyzed together.

The main expected result is not an independent diagnosis of periodontitis. If the clinical picture is the same, the genetic assessment clarifies the individual risk. This is important primarily for individual patients, namely for patients with varying degrees of modifiable risk factors and with a family history of the disease. Hence the model with its own device. In it, PRS is only one of the components of a multifactorial assessment [28; 34]. It may include age, smoking, diabetes, and family history. Also, the periodontal condition, microbiological characteristics, and other clinically significant signs.

The immediate task is not to increase the number of SNPs in the model. The objective is to prove its additional prognostic value, reproducibility, and clinical feasibility. Implementation is justified on one condition: only if the genetic information changes the risk assessment compared to the available clinical indicators. If genotyping does not affect the choice of preventive measures or the frequency of follow-up, its additional diagnostic value is limited. The evaluation of polygenic models should take into account more than just accuracy. It should also take into account the implications for clinical decisions.

Limitations of the current evidence base and the present review

The discrepancy in the diagnostic criteria of periodontitis is the main thing. This is what hinders the evidence base. Some works record the very fact of the disease. Others are its severity, progression, or individual clinical signs, to which is added the heterogeneity of the characteristics of the samples and methods of genetic analysis [13, pp. 182-210; 18; 34]. It is impossible to compare such results directly. And the associations found in one model are not automatically transferred to the models of another predictive task. For polygenic risk indicators, the issue price is particularly high. The quality of the initial phenotyping directly determines both the construction of the model and its subsequent verification.

There are limitations to the review itself. Studies of different designs are combined. It is impossible to quantify all the effects obtained. The conclusions are therefore based on a qualitative assessment of the reproducibility of associations, methodological characteristics of publications, and the availability of independent validation. This is enough to outline the directions of development of genetic forecasting, but not to assert the superiority of any particular polygenic model.

A separate touch is the state of Russian literature in recent years. It is dominated by works on individual genetic variants and molecular genetic characteristics. Whereas large GWAS and PRS studies are mainly represented by foreign publications. [1-3; 13; 18; 34]. Until the advent of large multicenter studies with uniform phenotyping and independent external validation, the conclusions remain preliminary. Conclusions about the clinical applicability of polygenic models.

CONCLUSION

A comparison of literature data showed that the genetic component of periodontitis was confirmed at the level of population and family studies, but its implementation was not limited to the action of individual polymorphisms. Studies on candidate genes have revealed links with variants of cytokine genes, factors of innate immunity, antimicrobial protection, and bone remodeling. At the same time, the reproducibility of associations significantly differed between populations. The result was influenced not only by the characteristics of the genotype, but also by age, smoking, microbial load, concomitant diseases and differences in diagnostic criteria.

The transition to genome-wide studies has expanded the understanding of the genetic architecture of the disease, but has not solved the problem of a universal marker. A systematic analysis of GWAS revealed a number of variants that reached genome-wide significance, but no common SNP was found to be reproducible in all the studied samples [18]. The model based on a single genetic variant proved insufficient to predict a complex multifactorial phenotype. Polygenic risk indicators, which take into account the cumulative contribution of a large number of variants, have become a more promising direction [13, pp. 182-210; 34].

PRS is not an independent clinical tool. Prognostic characteristics were determined based on the origin of the initial GWAS, the ethnic composition of the target population, the features of phenotyping, and the quality of external validation. Genetic risk is not a substitute for clinical assessment. The likelihood of periodontitis development and progression was simultaneously influenced by smoking, age, metabolic disorders, family history, and periodontal tissue condition. Therefore, the model in which genetic information was used as an additional component of multifactorial risk stratification, rather than as an independent diagnostic criterion, looked the most reasonable.

The conclusions reached remained limited by the heterogeneity of research protocols, differences in phenotype determination, sample size and composition, genotyping methods, and statistical data processing. For PRS, an additional problem was the limited portability of models between populations. Therefore, further development of the field required multicenter studies with unified criteria for periodontitis, comparable phenotyping, independent external validation of polygenic models, and mandatory comparison of their prognostic value with traditional clinical risk factors. The prospects of polygenic models are determined not by the number of included genetic variants, but by the proven ability to improve the accuracy of individual prognosis and the validity of clinical decisions. Therefore, the transition to a personalized assessment of periodontitis risk requires a comparison of genetic and clinical models based on independent samples with uniform diagnostic criteria.

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