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Role of the Oral Microbiome and its Dysbiosis in the Pathogenesis of Peri-Implantitis

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Abstract

Peri-implantitis is a severe infectious and inflammatory complication of dental implantation capable of undermining treatment outcomes. The aim of the work is to conduct a systematic analysis and integration of scientific data to determine the role of microbial dysbiosis in the pathogenesis of the disease, with an emphasis on taxonomic and functional shifts in the peri-implant microbiome. The methodological basis includes a systematic review and analytical synthesis of peer-reviewed studies selected from the Scopus and Web of Science databases. The body of results indicates that what matters is not so much the presence of individual periodontopathogens as a pronounced dysbiotic restructuring of the community, manifesting as increased microbial diversity and the dominance of Gram-negative obligate anaerobes. The principal pathogenetic link is the functional reorganization of the microbiocenosis: a shift from homeostasis-oriented carbohydrate metabolism to destructive amino acid catabolism. This metabolic drift is accompanied by the accumulation of proinflammatory metabolites, dysregulation of the host immune response, and subsequent activation of bone resorption. The conclusions support the concept of functional dysbiosis as the central mechanism in the development of peri-implantitis. The analysis underscores the need to shift the therapeutic focus from elimination-based interventions to strategies for restoring microbial homeostasis and opens prospects for the development of diagnostic and therapeutic approaches based on microbiome biomarkers. The information presented in the article will be of interest to dental clinicians, implantologists, periodontologists, and researchers in the fields of oral microbiology and immunology.

Keywords: *Peri-Implantitis, Oral Microbiome, Dysbiosis, Pathogenesis, Biofilm, Metagenomics, 16S rRNA, Functional Dysbiosis, Microbiome-Oriented Therapy, Dental Implantation.*

INTRODUCTION

Dental implantation is recognized as the reference method of rehabilitation for partial and complete edentulism, demonstrating consistently high success rates of over 90–95% over 5–10 years [1]. At the same time, the expansion of clinical application is inevitably accompanied by an increase in the frequency of biological complications. Peri-implantitis is coming to the forefront, posing a serious threat to the long-term stability of implants and to patient health [2, 3].

Contemporary epidemiological summaries confirm the scale of the problem. According to systematic reviews and meta-analyses, the prevalence of peri-implantitis varies widely: from 9.6% to 22% at the patient level and from 12.5% to 19.5% at the implant level [2]. Individual studies report even more unfavorable indicators, up to 26–43.9% in patients with implant function exceeding five years [6].

This diversity in estimates is due not only to the heterogeneity of the studied samples, but also to a key methodological deficit, the absence of unified, widely accepted diagnostic criteria for peri-implantitis [2]. The use by different research groups of dissimilar threshold values for critically important parameters, probing depth and the degree of bone tissue resorption, makes the results incomparable and hinders an objective assessment of the true epidemiological situation [4, 5]. As a result, methodological uncertainty slows the formation of standardized treatment and prevention protocols, as well as the rational planning of healthcare resources.

Traditionally, the pathogenesis of peri-implantitis was interpreted by analogy with periodontitis: a leading role was attributed to a narrow range of specific periodontopathogens forming an aggressive biofilm [8]. However, the introduction of high-throughput sequencing (NGS) has shown that the microbiota of the peri-implant niche, despite partial similarity to the periodontal one, has its own structural specificity and demonstrates a broader taxonomic diversity [10]. Against this background, the limitations of a purely inventory, taxonomic approach have become evident: listing the species present does not explain the mechanisms of the disease. The key gap in contemporary science lies in the necessary transition to a functional paradigm that considers the collective metabolic activity of the microbial community as the main driver of the pathological process [3].

Accordingly, the **objective** of the study is the systematic analysis and integration of scientific data to determine the

role of microbial dysbiosis in the pathogenesis of the disease, with an emphasis on taxonomic and functional shifts in the peri-implant microbiome.

The scientific novelty lies in the holistic linking of changes in species composition with metabolic reprogramming of the microbial consortium, the latter being interpreted as the central link initiating destructive inflammation.

The author's hypothesis posits that the course of the disease is determined not so much by the presence of individual pathogens as by functional dysbiosis: activation of amino acid metabolism pathways in the microbial community with subsequent production of proinflammatory metabolites, dysregulation of immune homeostasis, and bone tissue resorption induced thereby.

MATERIALS AND METHODS

The studies reviewed in the present work can be provisionally grouped into several directions:

1. Epidemiology and clinical context. Retrospective and review/meta-analytic studies define the magnitude of the problem and explain the heterogeneity in prevalence and incidence figures. Thus, in the retrospective study by Astolfi V. et al. [1], it was demonstrated that the frequency of peri-implantitis varies substantially depending on concomitant patient factors and prosthetic design, whereas the meta-analysis by Diaz P. et al. [4] shows a wide range of prevalence estimates across populations specifically due to differences in diagnostic criteria and study designs. For special clinical groups, Margvelashvili-Malament M., Eckert S. E. [13] emphasize that in completely edentulous patients with fixed full-arch prostheses on four implants, the risk of peri-implant diseases is comparable to or higher than in mixed samples, underscoring the role of prosthetic-occlusal factors. Issues of early diagnosis and conservative management are summarized in the narrative review by Kwon T. H., Yen H. H., Levin L. [2], where the need to recognize the transition from mucositis to peri-implantitis based on microbial and clinical signals before marked bone loss is emphasized.

2. Microbial communities: comparative and systematic reviews. A large body of work compares the microbiota of periodontal and peri-implant niches and refines the composition of the dysbiotic community. The systematic review and meta-analysis by Sahrmann P. et al. [7] demonstrated that the peri-implantitis microbiome is heterogeneous and is not limited to classic periodontopathogens, which is supported by NGS-centric reviews by Chun Giok K., Menon R. K. [8] and Iușan S. A. L. et al. [15], where differences at the levels of taxa and functional pathways are emphasized. The cross-sectional pilot study by Barbagallo G. et al. [3] demonstrates differences among healthy, periodontal, and peri-implant sites within a single oral cavity, which aligns well with the within-patient comparison by Yu X. L. et al. [9], where in a single patient differences in community structure between periodontitis and peri-implantitis were identified. In patients with a history of periodontitis, characteristic shifts are recorded already at the stage of mucositis: Zhou N. et al. [14] show that pre-existing periodontal disorders tune the peri-implant microbiota toward an inflammatory phenotype. At strain resolution, Ghensi P. et al. [19] identified strain-specific signatures associated with implant diseases, which is important for the transition from genus/species-level to strain-level diagnostics. In the review by Rajasekar A., Varghese S. S. [21], microbial profiles of periodontitis and peri-implantitis are compared, and the conclusion is drawn about a partial overlap of the core of pathogens alongside representatives unique to the implant-associated ecosystem.

3. Dysbiosis as a dynamic process: temporal and network characteristics. Studies of dynamics show that dysbiosis is not a static state but the result of successive community turnover. In a canine experimental model, Jiang Q. et al. [17] traced the temporal drift from early biofilm colonization to a mature dysbiotic peri-implantitis community. Zhang Y. et al. [20] linked dysbiosis to impaired local stability and changes in the network organization of the microbiocenosis: weakening of community resilience is accompanied by the growth of opportunistic taxa and functional modules that sustain inflammation.

4. Biofilm, implant material, and biocorrosion. Central to the implant-specific phenotype is the interaction of the microbiome with the implant surface and material. Conceptually, this was formulated by Kotsakis G. A., Olmedo D. G. [6] and Romanos G. E., Delgado-Ruiz R., Sculean A. [18]: peri-implantitis is not a small periodontitis but an independent phenotype in which microbiome–biomaterial interactions determine the clinical presentation. An early historical review of biofilms by Colombo A. P. V., Tanner A. C. R. [16] helps explain why surface properties and conditions shift the microbial balance. Practical implications for titanium are discussed by Costa R. C. et al. [5], analyzing microbial (tribo)corrosion: bacteria can catalyze the release of ions/particles, altering microrelief and nutritional niches. The pilot study by Ganesan S. M. et al. [24] on clinical material already links biomechanical loads, roughness, and microbial composition, proposing an integrated biome–microbiome model: mechanical microdamage and titanium wear products act as a selective force for dysbiotic consortia.

5. Patient immune response and tissue pathways. Transcriptomic analysis of tissues within the same patient by Yuan S. et al. [10] showed that inflammatory signatures in peri-implantitis and periodontitis partially overlap; however, there are specific modules of innate

immunity and bone remodeling, which is consistent with material-dependent triggers and a distinct microbial environment.

6. Diagnostics and microbiome-centered therapeutic approaches. From the clinical perspective, early diagnosis and conservative treatment are described by Kwon T. H., Yen H. H., Levin L. [2], where priority is given to biofilm control, device-based decontamination, and conservative pharmacotherapy. Randomized clinical trials demonstrate mixed yet informative effects of antimicrobial adjuvants: systemic metronidazole in the study by Blanco C. et al. [12] provided additional benefits on top of non-surgical therapy, whereas local minocycline during surgical intervention in Cha J. K., Lee J. S., Kim C. S. [23] improved short-term outcomes, supporting the concept of targeted biofilm modulation. On the horizon is microbiome therapy: work on precise selection of probiotic strains in gastroenterology (Yang J., Qin S., Zhang H. [11]) provides methodological guidance for the oral cavity (strain specificity, functional assays, colonization potential) but requires accounting for systemic consequences of antibiotics/probiotics for the ecosystem (e.g., shifts in the gut microbiome after H. pylori eradication according to Hsu P. I. et al. [22]), which is potentially relevant to the oral/general resistome.

Taken together, these works delineate the following pathogenetic scheme: primary colonization of the implant surface leads to an early biofilm community which, under the influence of materials science (roughness, corrosion microparticles), biomechanics (loads, micromovements), and the heritage of periodontal dysbiotic consortia (especially in patients with a history of periodontitis), shifts toward a stable dysbiotic state with distinctive strain signatures; subsequently, specific tissue and immune programs are initiated that differ from periodontitis, forming the clinical phenotype of peri-implantitis.

However, contradictions can also be traced in the studies:

- Some authors interpret peri-implantitis as a continuum of periodontal dysbiosis [9, 21], whereas others emphasize material dependency and specific microbial/immune signatures [6, 10, 18, 19].

- Systematic reviews document the absence of consensus regarding the list of key bacteria [7, 8, 15], pushing toward an ecological-functional rather than taxon-centric model.

- Prevalence estimates differ because of heterogeneous definitions and protocols [4, 13].

- RCTs on metronidazole and minocycline show short-term benefits [12, 23], but questions remain about long-term benefits/risks for the local and systemic microbiome [22] and about the durability of the effect without correcting biomechanics/surface.

Thus, the literature converges toward a multifactorial, ecological–materials science model of peri-implantitis; however, contradictions persist in the interpretation of the specificity of the microbiocenosis, in the assessment of disease burden, and in the evidence base for antimicrobial interventions; methodological heterogeneity remains the main obstacle to forming a definitive consensus.

RESULTS AND DISCUSSION

Understanding the pathogenesis of peri-implantitis requires analysis of the microbial communities that adhere to the implant surface and peri-implant tissues. Contemporary evidence indicates that the transition from clinical health to an inflammatory state is not a primitive invasion by individual pathogens, but a multistage ecological reorganization: a shift from a balanced symbiotic conglomerate to a dysbiotic, pathogen-oriented consortium [5, 7].

The microbiome accompanying clinically stable implants reflects a state of eubiosis/symbiosis: it is characterized by a comparatively low taxonomic diversity and the predominance of Gram-positive facultative cocci and rods [17]. Key representatives of such a community include bacteria of the genera Streptococcus, Rothia, and Actinomyces [9]; members of the genera Neisseria and Haemophilus are also detected in notable amounts [9]. As early colonizers, these microorganisms form a stable biofilm with barrier properties: they actively metabolize carbohydrates, maintain a near-neutral pH, and produce bacteriocins, thereby creating unfavorable conditions for the attachment and proliferation of opportunistic and pathogenic species. As a result, this microbial ensemble supports immune homeostasis and the integrity of peri-implant tissues.

The onset of peri-implantitis is directly linked to the disruption of this equilibrium, that is, to dysbiosis. This shift is manifested by a marked increase in overall microbial diversity and a qualitative reorganization of community structure toward the dominance of Gram-negative obligate anaerobes [13]. The key initiating factor of this cascade is the accumulation of dental plaque in the setting of inadequate oral hygiene [2].

Classical periodontopathogens: Bacteria of the red complex — Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola — are consistently detected in peri-implant pockets, and their quantitative accumulation is closely linked to the severity of clinical manifestations. These species possess a diverse set of virulence factors; in particular, proteolytic enzymes (gingipains) mediate degradation of connective tissue proteins and promote immune evasion.

Bridge species and pathogens of the orange complex: Fusobacterium nucleatum acts as a key organizer of the polymicrobial biofilm. Owing to its broad coaggregation capacity, it forms a functional bridge between early and late colonizers, facilitating the adhesion and survival of obligate anaerobic pathogens, including members of the red complex [10, 11].

Unique and opportunistic pathogens: Unlike periodontitis,

the microbiome of peri-implantitis is characterized by a substantial proportion of microorganisms atypical for classical periodontal pathology. Of particular note is Staphylococcus epidermidis, which demonstrates a high capacity for biofilm formation on titanium surfaces [18]. The pathogenesis of the disease also involves Filifactor alocis, Fretibacterium fastidiosum, Mogibacterium timidum, and Parvimonas micra, which are frequently detected in deep peri-implant pockets and are associated with active tissue destruction [9].

A comparative description of the dominant taxa is presented in Table 1.

Table 1. Comparative characteristics of dominant bacterial taxa in health and peri-implantitis (compiled by the author based on [9, 15, 16,18]).

Taxonomic group	Association with health (Genera)	Association with peri-implantitis (Genera/Species)	Key role in pathogenesis
Gram (+) aerobes/ facultatives	Streptococcus, Rothia, Actinomyces	Staphylococcus epidermidis, Parvimonas micra	Biofilm formation on titanium, induction of inflammation
Gram (-) aerobes/ facultatives	Neisseria, Haemophilus	-	Decrease in abundance under dysbiosis
Gram (-) anaerobes	Veillonella (early colonizer)	Porphyromonas, Tannerella, Treponema, Fusobacterium, Prevotella, Fretibacterium, Filifactor alocis	Production of virulence factors, tissue degradation, modulation of the immune response

A pronounced restructuring of the taxonomic profile becomes clearly apparent when the microbial community is considered at a higher level — the level of bacterial classes (see Fig. 1). In clinically intact areas, the class Bacilli predominates, including representatives of the genus Streptococcus; in contrast, in peri-implantitis their proportion decreases sharply, and the leading positions shift to the classes Bacteroidia (with the involvement of Porphyromonas and Prevotella) and Fusobacteriia [13].

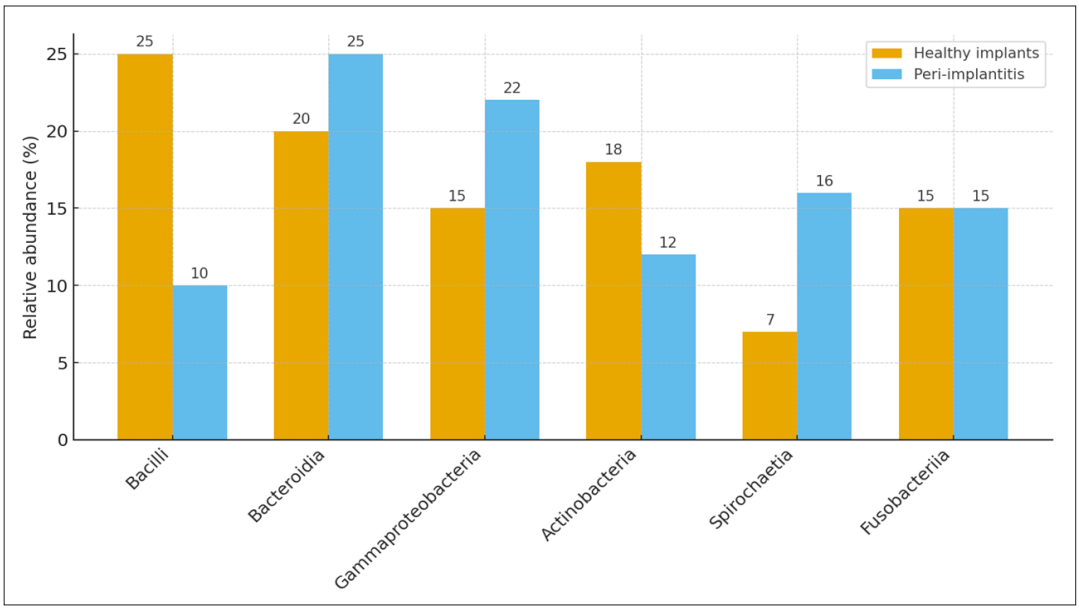

Fig. 1. Comparative number of bacterial classes in health and peri-implantitis (compiled by the author based on [12 - 14]).

The body of recent data convincingly shows: the defining factor in the pathogenesis of peri-implantitis is not so much the taxonomic composition of the biofilm as its aggregate metabolic activity. This gives rise to a paradigm shift — from the notion of a specific pathogen to the model of functional dysbiosis, in which the reconfiguration of the metabolic profile of the entire microbial community becomes pivotal [13].

This perspective reframes peri-implantitis from a purely infectious process to a metabolically mediated disease of microbial origin. As the peri-implant pocket deepens and inflammation intensifies, the local environment is transformed: oxygen availability decreases, and the primary substrates are no longer the carbohydrates of saliva but proteins and amino acids derived from gingival crevicular fluid and from disrupted host cells. This confers a selective advantage to proteolytic anaerobes. They not only adapt to the altered conditions but actively modify them, establishing a self-sustaining circuit: inflammation leads to tissue destruction and the release of amino acids; their intensive utilization by the microbiota is accompanied by the formation of toxic, proinflammatory metabolites that amplify the inflammatory response and deepen tissue destruction.

Metagenomic and metatranscriptomic approaches have

shown that specific metabolic circuits correlate strictly with tissue status — health or inflammation.

In the dysbiotic microbiocenosis in peri-implantitis, amino acid catabolism is markedly intensified [13]. The most pronounced increase is observed in the activity of enzymes involved in histidine degradation (urocanate hydratase) and other amino acids (tripeptidyl aminopeptidase) [13]. A key element is also the enhancement of the biosynthesis of arginine and polyamines (putrescine, citrulline) [3]. The products of these reactions—ammonia, hydrogen sulfide, indoles, and polyamines—exert direct cytotoxicity, undermine the epithelial barrier function, shift the local pH toward the alkaline range (inhibiting the growth of commensals), and substantially modulate the immune response, sustaining chronic inflammation [19, 20].

In the symbiotic state, the microbial community exhibits a fundamentally different profile. Carbohydrate utilization pathways (glycolysis) predominate, along with biosynthetic trajectories that support growth and homeostasis: biosynthesis of nucleotides (purines and pyrimidines) and tetrapyrroles, structural components of cytochromes [3]. This metabolic pattern reflects balanced, nonaggressive functioning aimed at maintaining ecosystem stability.

A deeper understanding of the role of functional dysbiosis in the development of peri-implantitis opens qualitatively new horizons for the design and implementation of innovative diagnostic and therapeutic solutions.

The traditional diagnostic paradigm, relying on clinical and radiological signs (bleeding on probing, increased depth of the periodontal pocket, bone resorption), registers destruction of peri-implant structures that has already occurred. In contrast, microbiome analysis enables the detection of pathological shifts at the preclinical stage. Promising biomarkers include:

Taxonomic markers: the ratio of the abundance of key pathogens to resident commensals (for example, Porphyromonas gingivalis to Streptococcus spp.) can serve as a sensitive indicator of dysbiotic changes in the ecosystem [9, 22].

Functional markers: determination of the activity of enzymes associated with pathogenic metabolism (for example, urocanate hydratase), or the direct detection of the corresponding metabolites (for example, polyamines) in gingival fluid or even saliva can form the basis of highly sensitive, noninvasive tests [13].

Integrative approaches: applying machine learning algorithms to the combination of taxonomic and functional data substantially increases recognition accuracy, providing an area under the ROC curve (AUC) at the level of 0,85, which indicates high predictive power of the model [13, 23].

Traditional approaches to the therapy of peri-implantitis — mechanical debridement of the implant surface combined with antibiotics — aim to nonspecifically reduce the overall microbial burden [11]. Despite a possible short-term clinical effect, their effectiveness is often unsustained: research data indicate rapid recolonization of the implant, often by the same pathogenic taxa [10]. The key flaw of these interventions is a focus on sterilization rather than rebalancing of the ecosystem: by eliminating both conditionally pathogenic and beneficial commensal biota, the therapy creates a vacant ecological niche that is highly likely to be repopulated by microorganisms optimally adapted to the inflammatory milieu. In this context, the magnitude of clinical improvements is associated more with qualitative reconfiguration of the community, that is, with reduction of dysbiosis, than with simple quantitative reduction of the microbial pool [24].

This implies the necessity of shifting the emphasis toward microbiome-modulating therapeutic strategies aimed at restoring a healthy microbial community (Table 2).

Table 2. Promising microbiome-oriented therapeutic strategies (compiled by the author based on [12, 21]).

Strategy	Mechanism of action	Examples of agents	Level of evidence (as of 2022)
Probiotics	Competitive inhibition of pathogens, production of antimicrobial substances, immunomodulation.	Lactobacillus reuteri, Weissella cibaria, Lactobacillus salivarius.	Conflicting clinical data; standardization of strains, doses, and application protocols is required.
Postbiotics	Direct antimicrobial and anti-biofilm action, anti-inflammatory effect.	Inactivated cells, cell lysates, culture supernatant of Lactobacillus spp.	Promising preclinical and in vitro data; high stability and safety.
Phage therapy	Specific lysis of target bacteria without affecting the commensal flora.	Bacteriophages specific to P. gingivalis, A. actinomycetemcomitans.	Early stages of research; major challenges include delivery to the target and a possible immune response.
Microbiota transplantation	Complete replacement of a dysbiotic community with a healthy one to restore ecological balance.	Oral microbiota transplant (OMT) from a healthy donor.	Conceptual and preclinical studies (in animals); issues of standardization and safety.

The emerging paradigm of clinical management for patients with peri-implantitis appears to rely on an integrated, stepwise strategy (Fig. 2), within which traditional and innovative methods are combined sequentially.

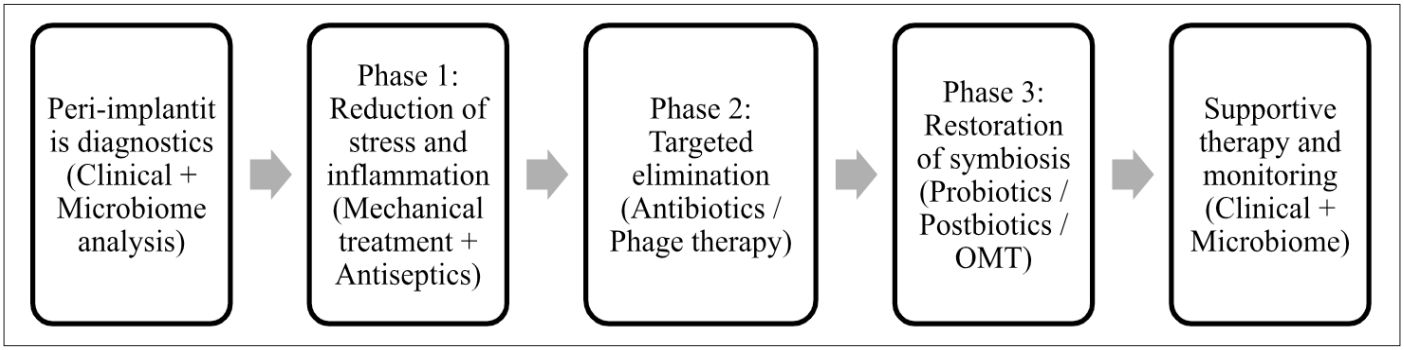

Fig. 2. Conceptual diagram of an integrated approach to the treatment of peri-implantitis (compiled by the author based on [1, 11, 12, 21]).

Thus, it can be said that different therapeutic strategies affect the key indicator of microbiome health—its diversity—in different ways. Whereas traditional regimens often induce a rapid but short-lived depletion of the community followed by a return to a dysbiotic state, an integrated approach that includes microbiota modulation is aimed at a more gradual and durable restoration of a healthy level of diversity and functional integrity.

CONCLUSION

The conducted systematic literature review identified fundamental propositions regarding the contribution of the oral microbiome to the pathogenesis of peri-implantitis. It was shown that the disease unfolds as a polyetiological process grounded not in isolated colonization by target pathogens but in pronounced dysbiosis that transforms the composition and functional organization of the peri-implant microbial community.

The principal result is the verification of the concept of functional dysbiosis as the central pathogenetic hub. The triggering event for destructive changes in peri-implant tissues is metabolic reprogramming of the consortium: a shift from homeostasis-oriented carbohydrate metabolism characteristic of health to aggressive proteolytic catabolism of amino acids. This shift is accompanied by the accumulation of toxic and pro-inflammatory metabolites, leading to activation of osteoclastogenesis and progressive bone resorption around the implant.

Consequently, the stated objective—to comprehensively characterize the role of microbial dysbiosis—has been achieved. The author's hypothesis on the central role of functional reconfiguration of the microbiome in the pathogenesis of peri-implantitis was supported by analysis of the sources.

The practical significance of the conclusions lies in the need to rethink clinical tactics for the treatment and prevention of peri-implantitis. Approaches focused on nonspecific bacterial elimination should be supplemented or replaced by strategies aimed at restoring microbial homeostasis and modulating the functional activity of the microbiota. The priority directions for further research are:

— Validation of taxonomic and functional biomarkers for the development of systems for early, preclinical diagnosis of peri-implantitis.

— Conduct of large-scale randomized clinical trials to assess the efficacy and safety of microbiome-modulating interventions (probiotics, postbiotics, phage therapy).

— Development of personalized therapeutic protocols based on the patient's individual microbial and metabolic profile.

Integration of the outlined approaches into clinical practice will not only enhance the effectiveness of treatment of manifest peri-implantitis but also establish effective preventive systems that ensure the long-term stability of dental implants.

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