

Available online at: <u>http://www.iajps.com</u>

Research Article

THE REGULARITY OF PERIODONTAL INFECTION AS A RESULT OF HYDRODYNAMIC BIOTA MOVEMENT

Alexander V. Tscymbalystov*, Alexander A. Kopytov, Irina V. Voytyatskaya, Gurgen G. Pahlevanyan, Elena A. Bulycheva

Belgorod National Research University, 85, Pobedy Str., Belgorod, 308015, Russia

Abstract:

The issue of optimization of power consumption and time on the satisfaction of somatic needs and implementation of reproductive tactics is fundamental to the animal world. If the representatives of the microflora of the oral cavity do not find an adequate response to the changing environment, it becomes problematic for their survival and evolution. On the contrary, the set of optimal solutions leads to the possibility of success, reproductive behavior and adaptability. Modern dentistry, for osnovnu the theory of the existence of microorganisms in the biofilm. For this reason, the implementation of therapies aimed at combating microorganisms, existing in the form of plankton is considered to be losing relevance. This approach is not promising because it is known therapy does not offer ways of elimination of biofilms. Taking as the object of therapy, biofilm, fixed up on the surfaces of the periodontium, the fading opportunity to discuss preventive measures aimed at the development of algorithms prevents movement of biofilm in deep periodontal departments.

Key words: Microorganisms, biofilm, periodontitis, hydrodynamics, biological fluid.

Corresponding author:

Alexander A. Kopytov Belgorod National Research University, 85, Pobedy Str., Belgorod, 308015, Russia Email id: kopitov.aleks@yandex.ru



Please cite this article in press as Alexander A. Kopytov et al, **The Regularity of Periodontal Infection as a Result of Hydrodynamic Biota Movement**, Indo Am. J. P. Sci, 2017; 4(10).

INTRODUCTION:

At present, there is no clear understanding of how microorganisms in the oral cavity reach the deep periodontal regions. A number of studies prove that microorganisms in the oral cavity exist in the form of films covering all hard and elastic surfaces [1]. Comparison of young and mature biofilms revealed that the latter contained more Enterococcus faecalis and Enterococcus faecium, and the sensitivity of microorganisms to antibiotics was significantly less in mature biofilms than in young biofilms [2]. In the infection chronicization, a significant role belongs to biofilms, which is a problem for the clinician. The possibility of preserving biofilms on the background of antibiotic therapy leads to the idea of expediency of carrying out therapy excluding the use of antibiotics [3]. The effect of sodium hypochlorite reduces the biofilm mass while the living cells of Staphylococcus aureus remain in it, which leads to the search for additional ways to increase the effectiveness of the fight against infectious diseases [4].

The **work purpose** is to find mechanisms demonstrating the possibility of reaching deep periodontal organs by microorganisms.

MATERIALS AND METHODS:

Three groups of arguments offered for discussion disclose the aspects of periodontitis development demonstrating the irrationality of close study of the biofilm dynamics, against the background of diminishing interest in the plankton form of the existence of microorganisms.

RESULTS AND DISCUSSION:

To discuss the first group of arguments, it is advisable to analyze the stages of biofilm development. At the first stage of biofilm formation, a monomolecular layer of molecules is formed on the parodontium surfaces. It is proved that the possibility of molecule orientation is provided by interaction of Coulomb forces, dipole intermolecular forces, valence forces, Vander-Waals attraction forces, repulsion forces and electron pressure, balancing the forces of Coulomb interaction [5].

In the second stage of film formation, the adsorption of microorganisms is provided by the ready-made adhesins, or adhesins adapting to the substrate [6]. In microorganisms that have passed into a stationary state, the probability of detachment from the surface by the liquid current decreases and the resistance to toxins and poisons increases [7].

There is an increase in the biofilm survival during the third stage due to the transition of adsorption attachment of microorganisms to the adhesion one by the activation of synthesis of matrix biopolymers, which makes it possible to firmly attach to the surface [8]. Specifying the nomenclature of the biofilm formation, it is advisable to use the terms "adsorption", "attachment" describing the early stages of film formation. If the study is conducted within 6-24 hours, it is preferable to describe adhesion using the terms "colonization", "biofilm formation" or "matrix formation" [9].

At the fourth stage of the cycle, an increase in the film volume is ensured by the multiplication of the expediency primary colonizers. The of microorganism growth is explained by the fact that as the population increases, the adaptation and reproductive potential of individuals increases as well. In addition, the protection level of common resources. the degree of the interaction environment modification also increases [10].

The fifth stage is characterized by the expansion of biofilm species composition by attaching microorganisms that are not capable of primary colonization [11]. One of the regulatory factors allowing the biota responding to changes in the population, habitat and response of neighbors to the changes occurred is the Quorum sensing (QS) systems [12]. The signal molecules of QS are accumulated in a bacterial population and autocatalytically affect bacteria without taking direct participation in the bacterial growth [13].

During the sixth stage, the membranes providing mechanical strength are formed in the biofilm volume [14]. There are membrane vesicles in some bacteria that are designed to transport these bacteria to the film surface [15]. The biofilm array is permeated with channels that provide trophic, removing metabolic products and creating the possibility of information exchange between microorganisms [16].

Seventh stage. Achieving a critical number of microorganisms in the biofilm volume leads to its local destruction, dispersion and planktonization of some part of the biota [17]. The destruction of the biofilm matrix can occur due to the endogenous and exogenous effects. The endogenous destruction is due to increased biota activity. An increase in the number of population individuals resulting in dispersion is possible under favorable environmental conditions [18]. Exogenous infringement of a biofilm matrix is connected with physical influence; lack of nutrients; phagocytic cells; an increase in the concentration of chelating agents, biogenic and abiogenic detergents, as well as enzymes that break down the molecular basis of the biofilm matrix [19].

In other words, the biota increases its survival by increasing the number at stages 2, 5 and 7. The changes taking place are quantitative and expedient in a favorable ecological environment.

In the process of implementation of 3, 4, 6 stages, the biota activates the developed mechanisms, increasing the group security due to the matrix generation. The idea of the existence of microorganism community

as a sequence of stages of increasing the number of microorganisms and the stages of increasing the reliability of protective extracellular matrix forms the boundaries in which it is possible to discuss the issues of reproductive biota tactics. The result of this is the development of new territories, in our case - the deep periodontium segments.

The population does not have enough time and energy to reproduce and form a response to changes in the habitat. To describe such situations, we introduced the term of reproductive compromise. This term explains the inverse correlations between the provision of the population with the possibility of reproduction and its response to changes in the habitat, as well as the number and quality of descendants according to the criterion of ability to master the habitat [20].

The logic of the first group of arguments. Previously, the periodontitis therapy involved "fighting plankton microorganisms". The biofilm discovery led to the idea of the advisability of combating periodontitis by influencing the film form of the biota existence. However, biofilm represents an evolutionarily developed form of the microorganism existence. So there is a question: If the medicine could not reduce the incidence of periodontitis, affecting the planktonic, unprotected form of the microorganism existence, can we expect success from the influence on the film form of the microorganism existence formed for the purpose of survival in adverse environmental conditions?

Discussing the second group of arguments, let us turn to the Lotka-Volterra law (1925-1926), which describes the principles of self-regulation and mutual influence of the populations. Based on this law, we created the mathematical models of the species interaction characterized as: "predatorprey", "host-pathogen", "host - parasite", "resourceconsumer" [21]. To understand the nature of the species interaction, the microorganisms are recognized as victims and leukocytes - as predators by analogy with the studies carried out. It is known that the dentogingival space (periodontal pocket) is the main supplier of leukocytes to the oral cavity. If the periodontal tissue is damaged, the number of leukocytes increases 2-4 times. If the microorganism concentration in the area of dentogingival space (periodontal pocket) increases, the corresponding number of leukocytes will increase accordingly [22]. That is, the number of microorganisms (preys) should be the least dense in the region with the maximum density of leukocytes (predators) [23]. For this reason, an inverse correlation between the highest concentration of leukocytes and microorganisms should be observed in the area of dentogingival space (periodontal pocket).

The second group of arguments should include the possibility of satisfying the trophic requests made

by the microorganisms. The crevicular fluid contains fewer substances suitable for trophic microorganisms than oral fluid. Increasing the crevicular fluid filtration appears to be a factor of the habitat degradation, which reduces the concentration of nutrients, which should lead to a local reduction in the number of plankton microorganisms and biofilm volumes [24]. The biota reduction in the area of dentogingival space (periodontal pocket) is possible due to a decrease in the intensity of microorganism reproduction or their migration to more favorable areas of the oral cavity.

The logic of the second group of arguments. As we move from the mouth of the dentogingival space (periodontal pocket), there should be a decrease in the density of leukocytes and an increase in the concentration of substances suitable for satisfying trophic needs by the microorganisms in the oral fluid. According to the Lotka-Volterra law, this regularity can be considered an argument linking the distance from the mouth of the dentogingival space (periodontal pocket) and the quality of habitats of the microorganisms. However, the amount of biota in the area of dentogingival space (periodontal pocket) exceeds the amount of biota localized in other volumes of the oral cavity, which makes one think that the law of interspecific competition does not work in the area of dentogingival space (periodontal pocket), or its action is blocked by another law, which acts more intensively in this place.

The third group of arguments. Considering the biota vital activity as a sequence of the microorganism existence in the mobile - planktonic form and immobile biofilm form, it is necessary to clarify which of the laws of natural science determines the achievement of deep periodontal segments by the microorganisms. To facilitate the search, we should formally formalize the periodontitis genesis as follows: there are hydrodynamic mechanisms in the oral cavity, the action of which increases the kinetics of microorganisms in such a way that they can withstand the crevicular fluid flow and penetrate into the deep parts of periodont, overcoming the force of surface tension in the cells formed by the periodontal fibers.

In healthy people, 0.5 to 2.4 ml of crevicular fluid is filtered daily into the oral cavity. In pathology, the filtration volume of the crevicular fluid can increase more than 10 times [25]. Discussing the distribution of microorganisms in the oral cavity, one should take into account their passive movement determined by the laws of molecular and microturbulent diffusion of the leveling concentrations of microorganisms throughout the oral cavity [26, 27]. It is established that the microturbulent diffusion plays an important role in determining the concentration of inclusions in a given volume. This is explained by the fact that the ratios of turbulent diffusion are several orders of magnitude greater than the molecular one [28]. In our problem, the microorganisms are expediently considered as particles moving in accordance with the principles of molecular diffusion and striving to equalize the concentration in the dentogingival space (periodontal pocket) by moving apically. In turn, the crevicular fluid turbulent flow tends to level the concentration, moving coronarily. Since the kinetics of turbulent diffusion exceeds the kinetics of molecular diffusion, therefore, the diffusion laws do not explain the large concentration of microorganisms in the region of the dentogingival space (periodontal pocket) as compared with other volumes of the oral cavity.

In describing hydrodynamic processes, it is used the concept of hydraulic size, which facilitates the apical movement of microorganisms into the periodontal space of the teeth located only on the lower jaw. On the upper jaw, this hydrodynamic mechanism must be considered as interfering with the apical migration of microorganisms [29].

The logic of the third group of arguments. Summarizing the literature review, it should be noted that we have not been able to identify the hydrodynamic mechanisms that explain the possibility of reaching the deep periodontal segments of the oral cavity by the microorganisms. **SUMMARY:**

In our opinion, the wide spread of periodontitis can be explained by the interaction of two streams: crevicular and oral fluid. The kinetics of crevicular fluid flow is provided by the heart muscle, which generates a pressure exceeding atmospheric pressure by 20-30 mm of water column in the terminal sections of the circulatory system. The pressure of the oral fluid flow is equal to atmospheric. There are microorganisms in the array of this flow. Under normal conditions, the crevicular fluid flow is pressing and the oral fluid flow perceives pressure. Their interaction causes the interface localization and depends on the clinical picture. The flows can form the interface in the mouth of the dentogingival space or at any distance from the enamel-cement border. In order the microorganisms could reach the periodontal lumen and solidify on its internal surface, we need a significant increase in the pressure of the oral fluid flow. Such pressure increase can provide a jumping distance of the tooth root and in the case of compliance with the condition:

P of the atmosphere + P of the tooth root = P of the oral fluid> σ + P of the capillary pressure,

where σ is the surface tension ratio, i.e. the ratio of the work required to increase the surface area to the magnitude of this area increment.

$$\begin{array}{c} \sigma & \Delta \\ = & \frac{W}{\Delta s} \end{array}$$

In this case, the oral fluid containing microorganisms will acquire sufficient energy to move the boundary with the crevicular fluid into the deep periodontal regions. Multiple occlusal loading will lead to the movement of the border of crevicular and oral fluid flows [30]. At the same time, the microorganisms, under the influence of increased pressure, penetrate into deeper periodontal segments, infect them, involving bone tissue in the inflammatory process (31).

CONCLUSIONS:

Many years of clinical practice, including antibiotic therapy, which affects the planktonic form of microorganisms, have not brought the desired success. Evidence of the biofilm existence, the impact on which has not previously been carried out, leads to the idea of expediency of finding the ways to minimize the survival of microorganisms in the form of biofilm. In our opinion, this is not very promising, since the evolutionary expediency of biofilm formation is determined by multiple environmental loads that make microorganisms covered by a matrix unreceptive to the effects of unfavorable factors. For this reason, it seems expedient to firstly influence the possibility of organisms existing in plankton form to expand the habitat range, i.e. change the hydrodynamic conditions that allow the apical movement of microorganisms in the periodontal gap. Secondly, in our opinion, the studies aimed at reducing the possibility of adsorption of primary colonizers on the periodontal surface have some prospects.

REFERENCES

1. Stafeev A.A., Zinoviev G.I., Biofilm on the Boundary of Structural Material - Fixing Material - Tooth Tissue. Institute of Stomatology, 2012; 2 (55): 76-77.

2. Holmberg A., Rasmussen M. Mature biofilms of Enterococcus faecalis and Enterococcus faecium are highly resistant to antibiotics. Diagnostic Microbiology and Infectious Disease, 2015; 84 (1): 19–21.

3.Blasi F., Page C., Rossolini G.M., Pallecchi L., Matera M.G., Rogliani P., Cazzola M., The effect of N-acetylcysteine on biofilms: Implications for the treatment of respiratory tract infections. Respiratory. Medicine, 2016; 117: 190–197.

4. Almatroudi A., Gosbell I.B., Hu H., Jensen S.O., Espedido B.A., Tahir S., Glasbey T.O., Legge P., and others, Staphylococcus aureus dry-surface biofilms are not killed by sodium hypochlorite: implications for infection control. Journal of Hospital Infection, 2016; 93 (3): 263–270.

5. Rajvanshi A.K., 2008. Irving Langmuir - A Pioneering Industrial Physical Chemist. Resonance, 13 (7): 619–626, doi: 10.1007/s12045-008-0068-z 6. Mayansky A.N., Chebotar I.V., Strategy of control for bacterial biofilm processes. Journal of Infectology, 2012; 4 (3): 5-15.

7. Rodionova T.A., Nikolaev Yu.A., Protective Effect of Reversible Adhesion of the Thermophilic Bacterium Bacillus Licheniformis 603 from N-Ethylmaleimide. Microbiology, 2004; 73 (1): 133-134.

8. Maksimova Yu.G., 2015. Heterogeneous Biocatalysts Based on Cells of Nitrile Hydrolyzing Bacteria and their Enzymes for the Transformation of Nitriles and Amides of Carboxylic Acids: Thesis of the Doctor of Biological Sciences, Perm: 21-27.

9. An Y.H., Friedman R.J., 2000. Handbook of bacterial adhesion: Principles, methods, and applications. Totowa, N.J.: Humana Press Inc.: 644.

10. Allee W.C., Animal aggregations, a study in general sociology. Chicago: The University of Chicago Press 1931 456.

11. Smirnova T.A., Didenko L.V., Azizbekyan R.R., Romanova Yu.M., Structural and Functional Characteristics of Bacterial Biofilms. Microbiology, 2010; 79 (4): 435-446.

12. Fuqua W.C., Winans S.C., Greenberg E.P., Quorum sensing in bacteria: the LuxR-LuxI family of cell density-responsive transcriptional regulators. J. Bacteriol, 1994; 176: 269-275.

13. De Kievit T.R., Quorum sensing in Pseudomonas auruginosa biofilms. Environ. Microbiol, 2009; 1 (2): 279–288.

14.Romanova Yu.M., Smirnova T.A., Andreev A.L., Ilyina T.S., Didenko L.V., Ginzburg A.L., The Biofilms Formation - an Example of the Bacteria "Social Behavior". Microbiology, 2006; 75 (4): 481-485.

15. Beveridge T.J., Structures of gramnegative cell walls and their derived membrane vesicles. J. Bacteriol, 1999; 181: 4725–4733.

16. Bulantsev A.L., New Ideas about the Ecology of Bacterial Populations with a Communicative Alarm System. Problems of Especially Dangerous Infections, 2006; 91: 11-14.

17. Stoodely P., Wilson S., HallStoodley S., Boyle J.D., LappinScott H.M., Costerton J.W., Growth and detachment of cell cluster from mature mixedspecies biofilm. Appl. Environ. Microbiol, 2001; 67: 5608–5613.

18. Dynamic Models in Biology. Basic Models of Mathematical Biophysics. Limited Growth. The Verhulst Equation.

www.dmb.biophys.msu.ru/registry?article=32

19. Mayanskiy A.N., Chebotar I.V. Strategy of Bacterial Biofilm Process Management. Journal of Infectology, 2012; 4 (3): 5-15.

20. Bennett A.F. and Lenski R.E., An experimental test of evolutionary trade-offs during temperature

adaptation. Proc. Natl. Acad. Sci. USA, 2007; 104: 8649–8654.

21. Rusakov S.V., Chirkov M.V., Control with Feedback in a Classic "Predator-Prey" System. Russian Journal of Biomechanics2015; 19 (1): 65-72.

22. Polishchuk L.V., Romanovsky Yu.E. The Relative Importance of Food Insufficiency, Invertebrate and Vertebrate Predator Pressure in the Dynamics of Fertility and Maintenance of the Stability of Populations of Planktonic Crustaceans. Report on Research Work No. 95-04-11741 (Russian Foundation for Basic Research).

23.Kiseleva E.I., Chemosensory Directed Behavioral Reactions of Tadpoles of the Pond Frog RANA LESSONAE (cam.) to the Excreta of Adult Con- and Heterospecific Tailless Amphibians of Sympatric Species. Sensor Systems. 2009; 23 (2): 137-144.

24.Lukyanov O.A., Berdyugin K.I., Vasiliev A.G., Research of the Role of Migrations in Repairation, Regulation and Structurally-Functional Organization of Populations (on the Example of Model Species of Small Mammals), Report on Research Work No. 97-04-48082 (Russian Foundation for Basic Research)

25. Kopytov A.A., 2007. Dynamics of Crevicular Fluid Parameters in the Rehabilitation Process of Patients with Bridge Works with Different Inclinations of Supporting Teeth: Abstract Thesis of the Candidate of Medical Sciences / Moscow State Medical and Dental University. Moscow, 22 p.

26. URL: <u>http://www.enc-</u> <u>dic.com<microbiology/Diffuzija-2446/</u>. (Access date 19 05.2017)

27. Abakumov A.I, Israilsky Yu.G., Phytoplankton and Minerals Move Passively due to Molecular and Microturbulent Diffusion. System Modeling. 2013; 2 (36): 14 - 23.

28. Ozmidov R.V., 1986. Diffusion of Impurities in the Ocean. Leningrad: Gidrometeoizdat.

29.Matveev I.A., Filippov V.E., Matveev A.I., Eremeeva N.G., Behavioral Features of Mineral Particles of a Flattened Form in the Water Flow. Non-Ferrous Metals.2017; 1: 8-13.

30. Kopytov A.A., Meirmanov A.M., Galtsev O.V., Hydropreparation as an Etiological Factor of Alveolar Bone Atrophy. Periodontology. 15. (4):. 32-36.

31. Kopytov A.A., Meirmanov2010 A.M., Galtsev O.V., Bellows, Calculation of the Flow Boundary Movement. Scientific Bulletin of the Belgorod State University. Series: Medicine. Pharmacy. 2011; 14 (10): 218-222.