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Pathomechanism of dental plaque and calculus formation – a literature review

Dental plaque is constituted by firmly adhering, soft, sticky, dense dental deposit, yellow-grey in colour. That deposit sticks firmly to the surface, is not removed by autocleaning or during rinsing of the oral cavity, or by the use of aerosols. Dental plaque formation is a continuous process. Its deposition begins soon after finishing hygienic procedures of the oral cavity.

Mineralized dental plaque in the form of hard concretion found on teeth surface is called dental calculus. Most often it is yellow-white substance of clay texture, which is especially deposited near teeth gingival line. The largest amounts of dental calculus are formed in the area of main salivary glands orifices, i.e. on the lingual surface of the inferior incisors and buccal surfaces of the superior molars (13). Due to its location we can distinguish supragingival and subgingival dental calculus. Subgingival dental calculus differs in location, composition and appearance from supragingival dental calculus. It is characterized by dark brown colour and hard texture (6). It was found to contain more calcium, magnesium and fluorine than supragingival dental calculus. This is due to the fact that its components mainly come from pocket fluid, and not from saliva as it is in the case of supragingival dental calculus.

Mature dental calculus is a highly mineralized substance. Its content of inorganic components is 70–85% of dry weight and is approximately equal to bone, dentine and cement (2, 7). The main inorganic components include calcium, phosphates and magnesium as well as trace quantities of sodium, zinc, strontium, bromine, copper, wolfram, gold, aluminium, silicon, iron and fluorine (13). Approximately two-thirds of the inorganic components occur in their crystalline forms. Crystals are formed by calcium phosphates of different degrees of hydration and different crystalline structure (4). Normally, in one calculus sample there are at least two crystalline forms, however, the percentage composition mainly depends on mineralization time.

The organic part of dental calculus mainly consists of glycoproteins, exfoliated epithelium cells, leukocytes and microorganisms. The largest percentage of calculus organic part is constituted by proteins of salivary and bacterial origin (about 8%). Carbohydrate component in the amount of 1.9–9.1% consists of extracellular polysaccharides produced by bacteria and from saliva glycoproteins and glycolipids. In addition, there were found smaller amounts of glucose, galactose, galactosamine, fucose, mannose and sialic acid. Lipids contained in dental calculus occur in the form of free fatty acids, cholesterol, glycolipids and phospholipids.

MECHANISM OF FORMATION AND MINERALIZATION OF DENTAL PLAQUE

The first stage of dental plaque formation is glycoprotein layer deposition on enamel surface. They stick to enamel surface due to their reactive lateral chains, creating stroma for other particles such as e.g. peptides. This is how acquired pellicle is formed. Under the influence of saliva enzymes modifications of pellicle components occur. At this stage of development dental plaque does not yet contain any bacteria. Within one or two days the plaque is colonized by Gram-positive cocci and rods. In the situation of small carbohydrate supply the plaque grows slowly. In the diet rich in carbohydrates bacteria produce extracellular polysaccharides, which constitute the plaque *matrix* and are the base of its further development. Subsequently, the plaque is colonized by Gram-negative bacteria, and then it becomes pathogenic for the parodontium (9). In the last phase dental plaque matures, contains ciliated bacteria and trepomena, and becomes visible to the naked eye.

Mineralization is the third and last stage of dental calculus formation. It begins in the early stages of dental plaque formation, and the first symptoms can already be found on the second day of its formation. That process is an example of pathological mineralization of biological base in the organism. Most researchers state that first of all the plaque *matrix* is mineralized (1). There are several theories of dental concretion mineralization. The first of them, called the active theory, assumes that degenerative changes in bacterial cells are the condition initiating that process. As a result of those changes centres with local electric charge are formed, which demonstrate potential possibilities for crystallization (14). In those centres micro currents originate, which enable ion movement independently of concentration gradient. According to Driesens et al. the speed of ions within dental plaque is about 6 mm/hr (3). According to a different theory, mineralization of soft dental concretion consists in passive deposition of ions, mainly phosphate, carbonate and calcium ones, around crystallization centres (8). In the case of Gram-positive bacteria, crystallization begins intracellularly with the participation of mesosomes. That type of mineralization rarely occurs in the case of Gram-negative bacteria, where extracellular mineralization usually occurs with the participation of the material of follicular structure produced by those bacteria. A dominant role in the process of crystallization is ascribed to filiform bacteria. It was observed that the surface of supragingival calculus is covered in majority with filiform bacteria, whose cells are positioned perpendicularly to calculus surface. Some authors claim that such spatial arrangement of bacteria slows saliva flow by calculus surface and facilitates concretion precipitation. The ability of that type of bacteria to capture calcium salt is also possible (7). The formation of dental concretion is a complex process. Apart from bacterial factor that process is also affected by various properties of saliva.

SALIVA ROLE IN DENTAL PLAQUE AND CALCULUS FORMATION

Considering the composition and salivary origin of dental calculus components it could be supposed that the factors influencing saliva composition, its secretion rate, its protein components synthesis also have an effect on dental calculus formation. The process of mineralization can be affected by pH, cations such as magnesium, zinc, copper, and anions: carbonates, pyrophosphates, citrates, oxalates, fluorides and vitamin C. Immunoglobulins A and G play a retarding role in dental calculus growth. They are adsorbed on the surface of crystals being formed, due to that their growth is inhibited (11). Crucial protective role against excessive deposition of dental calculus is performed by constant and satisfactory saliva secretion, which enables removing of bacteria from enamel surface and returning of saliva pH to appropriate values (12). In *in vitro* conditions there was revealed the negative correlation between mineralization rate and environment temperature. In the conditions

of the oral cavity temperature variations are so small that this factor has no influence on dental concretion mineralization (14).

The influence of saliva pH on dental concretion mineralization is more complex than it might result from theoretical assumptions. In aqueous solutions an increase in pH coincides with salt precipitation from the solution. In the oral cavity environment saliva pH first of all affects certain protein fractions of glycoproteins, whose isoelectric point is approximately equal to pH 4. That causes the change of cell spatial structure, which facilitates its binding with enamel surface and increases its abilities to bind calcium. As a result, a decrease in pH contributes to an increase in dental calculus deposition.

The effects which protein substances have on dental concretion formation are multidirectional, and often mutually opposing. In healthy people most proteins are synthesized in salivary gland cells, and only scarce amounts of proteins come from blood serum. Variety of particle structure and size (from a few to a few thousand kDa) determines salivary proteins function. It consists in proteins participation in acquired pellicle formation, dental plaque development and affecting the process of dental concretion crystallization. The formation of dental concretion involves glycoproteins responsible for saliva viscosity, containing N-acetylglucosamine and N-acetylgalactosamine. They cause increase in adhesion of bacteria and food debris to dental surfaces and impede removal of exfoliated gingival epithelium. A special role in the formation of dental concretion is performed by mucins classified as salivary glycoproteins (5). Mucin particles have asymmetric structure with irregular texture. The main part of the particle is constituted by polypeptides, to which carbohydrate lateral chains are attached. The lateral chains end with negatively charged groups of e.g. sialic acid, which facilitates binding with bacteria cells and enamel surface. Mucin particles are hydrophilous, they absorb large amounts of water and for that reason their structures are resistant to dehydration. That enables to perform better lubricating and moistening functions. Some oligosaccharides present in mucin particles have similar structure to glycoprotein carbohydrate components of the oral mucosa. That fact enables competitive binding and excretion of such bacteria groups which do not have abilities to bind with the mucosa (5).

Salivary mucins can be classified as belonging to one of the two groups. The group MG_1 of large molecular mass contains mucins with high content of oligosaccharide lateral chains, responsible for ramified mucin structure. MG_2 group of small molecular mass consists of single glycosylated peptide chains (10). MG_1 mucins adsorb strongly on dental surfaces and, in this way, take part in the formation of acquired pellicle. They initiate the colonization of bacterial plaque by saprophytous flora. They increase bacteria adhesion to acquired pellicle simultaneously causing dental plaque growth. They also constitute short-term source of nutrients for bacteria. MG_1 mucins create various complexes with other salivary proteins such as amylase, proline-rich proteins, staterin or histatin, causing their activation on the binding site. MG_2 mucins also have the ability to bind with enamel surface, however, such binding is weak and they can be easily disconnected (10). In salivary environment they cause bacteria aggregation and facilitate bacteria removal from the oral cavity. They cooperate with IgA creating a complex which binds pathogens with higher efficiency than each of the individual components.

The possibilities of dental plaque mineralization are largely determined by the concentration and availability of calcium in saliva. In the oral cavity there are mechanisms maintaining potential saliva supersaturation with calcium, which prevents excessive demineralization of hard dental tissues. On the other hand, that mechanism prevents precipitation of calcium salts in salivary gland outlet ducts and in the oral cavity. The role of those mechanisms is performed by precipitation inhibitors. One of the major inhibitors of calcium phosphate precipitation in saliva is staterin. It is a protein with asymmetrical particle both in terms of structure and electric charge. It has great affinity to enamel

hydroxyapatite and other bacterial strains as well. It can strongly connect with hydroxyapatite and initiate connections of other proteins with enamel surface. Staterin particle has the ability to inhibit calcium phosphate precipitation, however, inhibiting of crystal growth is controlled only by terminal hexapeptide of strong acidic properties. Apart from staterin, the major calcium salt precipitation inhibitors are proline-rich proteins (*PRPs*). They are the main component of acquired pellicle, they are also present in mature dental plaque. The protective activity consists in binding calcium ions in saliva solution (12).

The process of dental plaque mineralization still occurs despite the presence of precipitation and crystal growth inhibitors in saliva. That fact emphasizes the complexity of the problem. The examinations of numerous dental plaques failed to confirm inhibiting effects of staterin and PRPs on calcium phosphate precipitation in the very dental plaque (5). This phenomenon can be explained by impeded diffusion of precipitation inhibitors to the plaque surface, the presence and activity of proteolytic enzymes, and also bacteria's production of their own inhibitor protecting from precipitation. Some proteins, including PRPs, contained in saliva play a double role. After strong binding with enamel hydroxyapatite, apart from their role of reducing of calcium salts precipitation, they become strong promoter of bacterial adhesion to dental surface (5). In highly supersaturated solutions a protein particle, which is an inhibitor, can become the nucleus of crystallization.

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SUMMARY

The formation of dental concretions is a complex process which is determined by a number of factors such as pH, protein and mineral composition of saliva. An important role is played by the immune system and bacterial flora of the oral cavity. The study presents the pathomechanism of dental concretion formation on the basis of the available literature.

Patomechanizm powstawania płytki i kamienia nazębnego – przegląd literatury

Formowanie się złogów nazębnych jest procesem uzależnionym od wielu czynników, takich jak pH, skład proteinowy i minerałowy śliny. Istotną rolę odgrywa również układ immunologiczny oraz flora bakteryjna jamy ustnej. W oparciu o dostępną literaturę przedstawiono patomechanizm tworzenia złogów nazębnych.