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Review

Host and bacterial adhesion

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Abstract

Bacterial adhesion is an important step in tissue colonization and depends extensively on the surface properties of a bacterial cell. For many microorganisms the prerequisite for host body occupancy is a break in tissue continuity. The next step is ongoing tissue destruction by products of bacterial metabolism: microbial enzymes and toxins. This happens, for example, in the initial phase of periodontitis. The mechanisms of adhesion are related to the specific structures present on the bacterial cell surface. This article summarizes recent data about bacterial attachment to host cells.

Key words: bacterial adhesion, mechanisms

Bacterial attachment to host cells – called adhesion – is an important step in tissue colonization, usually equivalent to the initiation of a disease process. The effectiveness of adhesion depends significantly on the surface properties of the bacterial cell. For many microorganisms the prerequisite for host body occupancy is a break in tissue continuity. Tissue destruction leads to the exposure of the applicable receptors, usually the extracellular matrix proteins, thus enabling microorganisms to adhere and colonize the body. The next step is ongoing tissue destruction by the products of bacterial metabolism: microbial enzymes and toxins. This happens, for example, in the initial phase of periodontitis.

Periodontal disease (PD) occurs in a wide range of species from rodents to humans (Hennet and Harvey 1992, Slotwinska 2011a,b). Periodontitis is one of the most common diseases of adult dogs with up to 80% of animals affected. The canine oral subgingival flora is highly diverse and shows great similarities to the subgingival bacteria from humans at the genus level (Riggio et al. 2011, Dahlen et al. 2012). PD belongs to a group of inflammatory diseases. It is caused by bacterial plaque in the periodontium and results from the interaction of the host defence mechanisms with the plaque microorganisms. Early diagnosis and treatment are very important both for human and veterinary medicine, due to the high prevalence of PD (Oz and Puleo 2011, Albuquerque et al. 2012). Study of plasminogen activator activity (PAA) and tissue-type plasminogen activator (t-PA) antigen level has revealed that these markers may be used to evaluate the evolution of periodontal disease in the dog (Papadimitriou et al. 2006). The significance of gingival stippling in the diagnosis of PD in dogs is limited (Kyllar et al. 2010). Periodontal disease is common in beagle dogs and its prevalence is high already at the age of two (Kortegaard et al. 2008). PD is also common in cats (Girard et al 2009). A study of North American pets showed a 20% – 24% incidence of calculus and/or gingivitis in dogs and cats of all ages. Dental and periodontal diseases in older pets are especially common. Some authors identified periodontitis in 82% of dogs aged 6 to

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8 years and in 96% of dogs aged 12 to 14 years (Larsen 2010).

The adhesion mechanisms are related to the specific structures present on the bacterial cell surface, called adhesins. Adhesins recognize receptors on the host cell surface (Bank et al. 2011, Senevirante et al. 2011, Umeda et al. 2012). Within the oral cavity these include mucous membrane cells, sulcular epithelium cells, as well as the teeth surface. In many Gram-positive and Gram-negative bacteria the role of adhesins is played by fimbriae (fimbrilin protein), also known as colonization factors. These bacteria include *Porphyromonas gingivalis, Actinomyces viscosus, Fusobacterium nucleatum, Prevotella loescheii,* and in dogs *Porphyromonas gulae*, *Porphyromonas macacae*, *Fusobacterium canifelinum*, and other species (Hamada et al. 2008, Riggio et al. 2011, Dahlen et al. 2012, Nomura et al. 2012, Senhorinho et al. 2012). Moreover, the role of adhesins can also be played by other protein structures on the bacterial cell surface, such as pili (pilin protein), flagelli (flagellin protein), specific capsules and fibrinous reticulum, as well as polysaccharides and envelope polypeptides, exopolisaccharides forming mucus, lipopolysaccharides, lipooligosaccharides and many other surface proteins. The receptors for bacterial adhesins include various structures on the surface of epithelial cells, fibroblasts, and neutrophils, as well as blood proteins and extracellular matrix proteins, such as galactosyl and mannose residues, collagen type I and V, laminin, fibronectin and other proteins. Some bacterial species do not exhibit direct adhesive capacity. Yet they make use of the phenomenon of adhesion too, by means of coaggregation – cell-to-cell recognition of specific bacteria. Coaggregation does of course require adhesins and their receptors, just like adhesion. An example of coaggregation use is *Fusobacterium nucleatum* bacterium. Their protein adhesins bind to the galactosyl residues on the *Porphyromonas gingivalis* surface. But coaggregation is not always such a straightforward process. It often requires mediators. The extracellular capsules of *Porphyromonas gingivalis* enable the adherence of these bacteria to the surface receptors of *Eubacterium saburreum*. The crucial role in bacterial adhesion is played by a microbial polysaccharide, glycocalyx. Glycocalyx, present on the surface of bacterial cells, enables aggregation between bacteria. It is especially important for dental plaque formation, as well as for the adherence of bacteria to the smooth surfaces, such as dental enamel. In the process of tooth surface colonization dependent on bacterial adhesion a crucial role is played by acquired pellicle, consisting of salivary proteins and mucins. The free polyssacharide groups of glycoproteins forming the pellicle make perfect receptors for the adhesins of bacteria colonizing dental plaque. Adhesion is one of the first stages of dental plaque formation. It leads to the creation of a very strong and durable bond between the surface structures of bacterial cells and the tooth surface. Thus a microbial biofilm is formed – an organic, limited substance, comprised of a matrix of bacterial population, where microorganisms stick to each other, forming one entity. Such a structure of dental plaque enables the durable and safe existence of bacteria within the oral cavity, in spite of daily hygienic routine (Dunne 2002, Rickard et al. 2003, Choj et al. 2011). In the human, even a very precise and systematic toothbrushing cannot lead to the permanent destruction of biofilm. This microbiological coat, covering the gingiva and teeth, redevelops quickly, just in a few hours after professional plaque removal (Costerton et al. 1994, Marsh and Bradshaw 1995, Darveau et al. 1997). The first ones are proteins and glycoproteins from the saliva and gingival fluid. Successive bacterial species and strains then appear. Dental plaque development depends mostly on the adherence capabilities of bacteria. The most important process is obviously the above – mentioned bacterial coaggregation and adherence between specific species. A very important point in dental plaque development is the cooperation between the individual bacterial species and strains, involving their metabolism and life functions. The main source of nutritional factors for microbial metabolism and growth is the gingival fluid. The gingival fluid also contains the elements of antibacterial defensive system which can effectively disturb or inhibit the process of dental plaque development.

The bacterial cells interact with macroorganisms or another substrate. These interactions include specific and non-specific ones. Some of the siginificant interactions take place between *Staphylococcus aureus* surface protein and collagen or fibrinogen, between invasive *Yersinia enterocolitica* and VLA-4 or VLA-5 integrins, between the adhesine of *Helicobacter pylori* and Lewis b antigen, between the LPS of *Helicobacter pylori* and laminin, or between the fibrilar hemagglutinin of *Bordetella pertussis* and CD11b/CD18 integrin or sulfatide. An important example of non-specific interaction is the adherence of *Streptococcus mutans* to the enamel surface by means of extracellular glucans and fructans. The alginian present on the surface of *Pseudomonas aeruginosa* enables colonization of different cell membranes. Similarly the exopolysaccharide of *Staphylococcus epidermidis* facilitates the colonization of biopolymers.

The scientific research on bacterial adhesion is multidirectional. In the case of *Pseudomonas aeruginosa* the proteolytic activity of microorganisms

has been found to be inversely proportional to the adhesive capacity of bacteria (Pajdak and Szkarlat 1993). Adhesion of *Helicobacter pylori* species is a very complex process. Between the surface of stomach epithelial cells and bacterial cells there is always a gap of a few nanometers, free of any structures. It is an atypical adhesion, comprising several steps, with many bacterial substances, reacting with specific stomach cell receptors, being involved in this process (Janas and Bartel 1997). The adhesive molecules present on the surface of *Candida albicans* cells are capable of reacting with the membrane receptors of the host cells, as well as with the receptors of the extracellular matrix. Such an antigenic resemblance of distinct receptors is known as molecular mimicry. Proteolytic enzymes, secreted by yeasts, degrade protein matrix components, thus facilitating host tissue penetration. A decrease of immunity can lead to systemic candidiasis (Macura-Biegun and Macura 1997). The extracellular polysaccharides of coagulase-negative staphylococci play an instrumental role in the adhesion of these bacteria to the human tissues, and thus in staphylococcal infections (Kubler 1998). Staphylococci in the hospital environment exhibit a high potential for adhesion to human epithelial cells (Waldon and Szewczyk 2002). It should be noted that the heterogeneity of the types of enteropathogenic *Escherichia coli* adhesion to human cells in the course of diarrhoea has led to the reclassification of this group of intestinal pathogens (Sobieszczanska and Gryko 2001, Edwards et al. 2011, Yu et al. 2012).

In conclusion, the process of adhesion (adherence) is the main factor enabling the bacterial cells to colonize the macroorganism. It is irreversible. Adhesion is also one of the first stages of bacterial infection. Thus a significant role is played by the constant readiness of animal and human immune systems to fight against bacterial invasion or bacterial metabolites.

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