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, Słotwińska 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 10 years. In older cats, periodontal disease is common and the prevalence is high, even in young animals. The diagnosis of periodontal disease is often difficult due to the fact that some signs may mimic other diseases. Some studies have indicated that the prevalence of periodontal disease is lower in the breeds with a lower predisposition for developing periodontal disease. This may be due to the fact that the predisposition for developing periodontal disease is related to the natural resistance of the host to bacterial adhesion. The natural resistance to bacterial adhesion is related to the unique structures present on the bacterial cell surface. 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.
8 years and in 96% of dogs aged 12 to 14 years (Lar- 
sen 2010).

The adhesion mechanisms are related to the spe-
cific 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 ep-
ithelium 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 vis-
cosus, Fusobacterium nucleatum, Prevotella loescheii, and in dogs Porphyromonas gulae, Porphyromonas 
macacae, Fusobacterium canifelimum, and other spe-
cies (Hamada et al. 2008, Riggio et al. 2011, Dahlen 
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 re-
ticulum, 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. Co-
aggregation does of course require adhesins and 
their receptors, just like adhesion. An example of 
coaggregation use is Fusobacterium nucleatum bac-
terium. Their protein adhesins bind to the galactosyl 
residues on the Porphyromonas gingivalis surface. 
But coaggregation is not always such a straightfor-
ward process. It often requires mediators. The ex-
tracellular capsules of Porphyromonas gingivalis en-
able 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 be-
tween bacteria. It is especially important for dental 
plaque formation, as well as for the adherence of 
bacteria to the smooth surfaces, such as dental en-
amel. In the process of tooth surface colonization 
dependent on bacterial adhesion a crucial role is 
played by acquired pellicle, consisting of salivary pro-
teins and mucins. The free polysaccharide groups of 
glycoproteins forming the pellicle make perfect recep-
tors 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 struc-
tures of bacterial cells and the tooth surface. Thus 
a microbial biofilm is formed – an organic, limited 
substance, comprised of a matrix of bacterial popula-
tion, where microorganisms stick to each other, form-
ing one entity. Such a structure of dental plaque en-
ables 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 tooth-
brushing cannot lead to the permanent destruction of 
biomfilm. This microbiological coat, covering the gingi-
va 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 
pecies and strains then appear. Dental plaque devel-
opment 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 me-
tabolism and growth is the gingival fluid. The gingival 
fluid also contains the elements of antibacterial de-

defensive system which can effectively disturb or inhibit 
development, where microorganisms stick to each other, form-
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 significant in-
teractions take place between Staphylococcus aureus 
surface protein and collagen or fibrinogen, between 
vasive Yersinia enterocolitica and VLA-4 or VLA-5 inte-
grins, between the adhesine of Helicobacter pylori 
and Lewis b antigen, between the LPS of Helicobacter 
pylori and laminin, or between the fibrilar hemag-
glutinin of Bordetella pertussis and CD11b/CD18 in-
tegrin or sulfatide. An important example of non-spe-
cific interaction is the adherence of Streptococcus mu-
tans to the enamel surface by means of extracellular 
glucans and fructans. The alginan present on the sur-
face of Pseudomonas aeruginosa enables colonization of 
different cell membranes. Similarly the exopolysac-
charide 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 Szkarłat 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.

References


