

Adhesion in Bacteria

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Abstract: *Adhesins are cell surface component or appendages in bacteria that facilitate bacterial adhesion or adherence to other cell or to inanimate surface. Adhesions are a type of virulence factor. There are various types of adhesion that are seen in bacteria which enhances the capacity to produce diseases in man and animals, in this review we shall see the molecular basis of adhesion of various bacterial to cell surfaces*

Keywords: bacteria, adhesion, immunology, cell, immunity

1. Introduction

The majority of bacterial pathogens exploit specific adhesion to host cells as their main virulence factor. "A large number of bacterial adhesins with individual receptor specificities have been identified." Many bacterial pathogens are able to express an array of different adhesins. Expression of these adhesins at different phases during infection play the most important role in adhesion based virulence.[2] Numerous studies have shown that inhibiting a single adhesin in this coordinated effort can often be enough to make a pathogenic bacterium non-virulent. This has led to the exploration of adhesin activity interruption as a method of bacterial infection treatment

2. Mechanism of Bacterial Adhesion

Bacterial infections are responsible for a broad spectrum of human illnesses and medical device complications. For example, urinary tract infections caused by *Escherichia coli* affect over 7 million people annually and are among the most common infectious diseases acquired by humans.(1) Enteropathogenic *E. coli* and shiga toxin-producing *E. coli* are diarrhoeagenic pathogens causing serious health problems in both industrialized and developing countries. (2,3) *Helicobacter pylori* have been found to be a main factor in the development of gastric and duodenal ulcers and are believed to be a causative factor of gastric cancer. (4) *Staphylococcus aureus* and *Staphylococcus epidermidis* are major causes of infections associated with wounds, indwelling catheters, and cardio-vascular and orthopedic implant devices.(5,6)

Bacteria have a strong tendency to attach to surfaces. Attached cells will form a colony (biofilm) consisting of prokaryotic cells, surrounded by a matrix of biomolecules secreted by the cells. Although the structure and functions of biofilms are as varying as the type of bacteria, the same four step process is always followed in the creation of the biofilm. (7,8)

During the first step, a series of small molecules (initially water and salt ions) will adsorb to the surface. Subsequently, the substrate surface will be covered with a single layer of small organic molecules or proteins that are present in the medium. The mixture of water, ions and proteins is called conditioning film and is always present before the first microorganisms arrive at the surface.(9)

The second step is characterised by the initially reversible adsorption of micro-organisms to the conditioning film. The microbes arrive by Brownian motion, gravitation, diffusion, or intrinsic motility. They may also adhere to each other forming microbial aggregates before adsorbing to the conditioning film. Since the microorganisms adhere to the conditioning film and not the surface itself, the strength of the initial biofilm depends on the structure of the conditioning film.(10)

The initially reversible adsorption becomes irreversible, mainly through the secretion of exopolymeric substances by the adsorbed microorganisms in step three.(11) These substances will incorporate in the conditioning film and strengthen its cohesiveness. Finally, the number of microorganisms in the biofilm accumulates mainly through in situ cell growth. The final structure and composition of the biofilm is determined by these initial events. Other aspects such as the influence of surface active compounds secreted by the microorganisms(12), the hydrodynamic environment.

Bacterial adhesion leading to infection can be divided into three distinct categories: specific adhesion to host cell surface molecules, specific adhesion to extracellular matrix and blood plasma derived molecules, and adhesion to biomaterial surfaces of medical devices. In this chapter, an overview of the current understanding of the molecular basis of bacterial adhesion as it pertains to each of the three categories of bacterial adhesion is presented, followed by modeling of bacterial adhesion based upon the general principles governing molecular adhesion. Particular emphasis is given to interactions between the initially arriving microorganisms

3. Microbial Adhesion to Extra Cellular Matrix

The interactions of arriving microorganisms with a conditioning film on a surface are usually mediated by specific binding events between adhesins on the microbe surface and receptors of the extracellular matrix (ECM). Receptor binding may subsequently activate a series of complex signal transduction cascades in the host cell, which may be either inhibitive or beneficial to bacterial invasion. In several bacterial species, including *E. coli*, *Pseudomonas aeruginosa*, *Vibrio cholerae*, and *Salmonella enteritidis*, adhesins are presented at the tips of complex cell-surface

structures which extend from the outer cell membrane called pili or fimbriae. (13)

3.1 Host Cell Adhesion Mechanism

Bacterial adhesion to host cells of the urinary tract has been found to occur by specific molecular interactions between adhesins located on the distal tip of pili extending from the bacterial outer membrane and receptor molecular structures present on the host cell outer surface. Although the exact molecular structures involved for many of these interaction are yet to be determined, the specific binding mechanisms involved in a few systems have been relatively well characterised.

A different bacterial mechanism has been found to occur for the mucosal lining of the intestine leading to microvilli effacement and diarrhoea. A four stage infection process has been suggested involving initial attachment of enteropathogenic *E. coli* bacteria to the microvilli enterocyte cell surface:

- 1) A non piliated adhesin mechanism;
- 2) Type III bacterial secretion of 80kDa proteins (*E. coli* secreted protein, EspE) which mediate cytoskeleton disruption and the formation of tyrosine-phosphorylated trans-located in intimin receptors (TIR) on the host-cell surface for intimin binding;
- 3) Intimin-binding mediated bacterial attachment to the intestinal mucosa; and
- 4) Bundle-forming pili (BFP) mediated bacterial colonization.

Others have suggested that BFP serves as the adhesin controlling initial host cell contact as well as bacterial colonization. (14, 15)

S. aureus can bind to endothelial cells through its fibrinogen binding clumping factors ClfA and ClfB. Adhesion studies found that the preferential attachment of *S. aureus* to umbilical vein endothelial cells is mediated by fibrinogen adsorbed from plasma. Antifibrinogen antibodies could block the binding, indicating the specificity. Cheung et al. found that fibrinogen acts as a bridging molecule, attaching to both endothelial and *S. aureus* cell-wall integrins with each of its two γ -chains. (16)

Finally, some bacteria use the integrin on endothelial cells to invade the host. Filamentous hemagglutinin (FHA), an adhesin formed as a 50 nm monomeric rigid rod of *Bordetella pertussis*, interacts with two classes of molecules on macrophages, galactose containing glycoconjugates and the α M β 2 integrin which binds to the Arg-Gly-Asp (RGD) sequence in FHA. (17) Intimin, the outer membrane protein of *E. coli* also binds specifically to α M β 2 integrins and is inhibited by RGD containing peptides(18)

3.2 Agents that Inhibits Bacterial Adhesion:

Host-derived anti-adhesins in innate immunity: the hydrophobic molecules phinganine, a component of sphingolipids, decreases adhesion *S. mitistobuccale* epithelial cells and *Staphylococcus aureus* on salmucosal cells(19)

3.3 Adhesin-based vaccines

The inactivated *Bordetella* vaccine contains the hemagglutinin in adhesin. Vaccinated individuals developed anti-hemagglutinin antibodies which were shown to inhibit adhesion of *Bordetella* to epithelial cells (20)

4. Conclusion

As mentioned bacterial adhesion to the host is the first critical step in producing infection. These binding are generally submolecular and not completely understood. Agents which prevent the binding of the bacteria to the host or other medical devices have a great therapeutic value hence research on the molecular basis of bacterial adhesion is of great importance for both management and to prevent various bacterial diseases and infections.

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