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Surface Structures in Bacteria; Resistance to Environmental, Chemical and Biological Agents: Review Article

Shila Jalalpour

Molecular Medicine Research Center, Rafsanjan University of Medical Sciences, Rafsanjan, Iran.

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ABSTRACT

Surfaces structure in bacteria have important role in mechanical stabilization, protection against bacteriophages and phagocytosis, resistance bacteria in change of pH, inhibit introduce some of biomolecules, adhesion and stabilization of the membrane in bacteria and Archaea. In this present study the role of surface structures in increase of pathogenesis in pathogen bacteria was determined. Related original and review (Available freely to download) papers to role of surfaces structure in increase of bacterial pathogenesis were extracted from original and review articles published in Pubmed and Elsivier Science during the period of 1995 to 2013. For this study key words which were search include "surfaces structure, pathogenesis, pathogen bacteria and resistance". About the surfaces structure in all of similar original and review articles, there is consensus that the existence of surface structures in bacteria lead to increased pathogenesity in bacteria. Surface structures are virulence agents in bacteria and preset this structure in pathogen bacteria due to increase of pathogenesis and infection resistant in host.

INTRODUCTION

All of the various surface components of a bacterial cell are important in its ecology since they mediate the contact of the bacterium with its environment. The only senses that a bacterium possesses result from its immediate contact with its environment [2,6,22,32]. It must use its surface components to assess the environment and respond in a way that supports its own existence and survival in that environment. The surface properties of a bacterium are determined by the exact molecular composition of its membrane and cell envelope, including capsules, glycocalyx, S layers, peptidoglycan and LPS, and the other surface structures, such as flagella and pili or fimbriae. Bacterial surface components may have a primary biological function that has nothing to do with pathogenicity. Thus, the function of the LPS in the outer membrane of Gram-negative bacteria has to do with its permeability characteristics, rather than its toxicity for animals [2,6,22,32]. However, there are endless examples wherein a bacterial surface component plays an indispensable role in the pathogenesis of infectious disease. Bacterial surface structures may act as (1) permeability barriers that allow selective passage of nutrients and exclusion of harmful substances (e.g. antimicrobial agents); (3) adhesins used to attach or adhere to specific surfaces or tissues; (3) enzymes to mediate specific reactions on the cell surface important in the survival of the organism; (4) protective structures against phagocytic engulfment or killing; (5) antigenic disguises to bypass activation of host immune defenses; (6) endotoxins, generally cell wall components, that cause an inflammatory response in the host; (7) "sensing proteins" that can respond to temperature, osmolarity, salinity, light, oxygen, nutrients, etc., resulting in a molecular signal to the genome of the cell that will cause expression of some determinant of virulence (e.g. an exotoxin) [2,6,22,32] In medical situations, the surface components of bacterial cells are major determinants of virulence for many pathogens. In animals, they may be used to colonize tissues, resist phagocytosis and immune responses, and to induce inflammation, complement activation and harmful immune responses [2,6,22,32] Structurally, a bacterial cell has three architectural regions: appendages (proteins attached to the cell surface) in the form of flagella and fimbriae; a cell envelope consisting of a capsule, cell wall and plasma membrane; and a cytoplasmic region that contains the cell genome (DNA) and ribosomes and various sorts of inclusions. The surface components of a bacterium are the constituents of its cell envelope and appendages [2,6,22,32]The cell wall of a bacterium is an essential structure that protects the delicate cell protoplast from osmotic lysis. The cell wall of Bacteria consists of a polymer of disaccharides cross-linked by short chains of amino acids (peptides). This molecule is a type of peptidoglycan called murein. Murein is unique to the Domain, Bacteria. In the Gram-positive bacteria, the cell

Corresponding Author: Shila Jalalpour, Molecular Medicine Research Center, Rafsanjan University of Medical Sciences, Rafsanjan, Iran.
E-mail:shilla.jalalpoor@yahoo.com

wall is thick (15-80 nanometers), consisting of several layers of peptidoglycan complexed with molecules called teichoic acids. In the Gram-negative bacteria, the cell wall is relatively thin (10 nanometers) and is composed of a single layer of peptidoglycan surrounded by a membranous structure called the outer membrane [2,6,22,32]. The cell wall, more properly the cell envelope, is a complicated structure, fundamentally different in Gram-positive and Gram-negative bacteria. Cell wall components are major determinants of virulence in both groups of bacteria. Endotoxin, inherent to all Gram-negative bacteria, is toxic to animals in a variety of ways. Peptidoglycan and LPS, as well as some teichoic acids, induce the alternate complement pathway leading to inflammation. Teichoic acids and O-specific polysaccharides may be used as adhesins by Gram-positive and Gram-negative bacteria, respectively. Some cell wall components protect against phagocytic engulfment or digestion. Variations in the macromolecular structure of cell wall components may be at the basis of antigenic variation as well as specific host resistance to pathogens [2,6,22,32]. The essential outer membrane of Gram-negative bacteria is the target for attack by complement, hydrophobic agents and certain antibiotics. Murein (peptidoglycan) is dismantled by a host enzyme, lysozyme, found in most body fluids. Several antibiotics, mainly the beta lactams, exert their antimicrobial effect by blocking the synthesis and assembly of peptidoglyca [2,6,11,22,32]The membranes of bacteria are structurally similar to the cell membranes of eucaryotes, except that bacterial membranes consist of saturated or monounsaturated fatty acids (rarely polyunsaturated fatty acids) and do not normally contain sterols. The plasma membrane is an exceptionally dynamic structure in bacteria which mediates permeability, transport, secretion and energy generation. In terms of pathogenesis of a bacterium, it is often dependent upon the integrity and function of its plasma membrane. The membrane might be responsible for secretion of toxins, resistance to antimicrobial agents, tactic responses or sensing other environmental signals to turn on genes for virulence [2,6,22,32]. Flagella are filamentous protein structures attached to the cell surface that provide swimming movement for most motile bacterial cells. The diameter of a bacterial flagellum is about 20 nanometers, well-below the resolving power of the light microscope. The flagellar filament is rotated by a motor apparatus in the plasma membrane allowing the cell to swim in fluid environments. Bacterial flagella are powered by proton motive force (chemiosmotic potential) established on the bacterial membrane.

Bacteria are known to exhibit a variety of types of tactic behavior, i.e., the ability to move (swim) in response to environmental stimuli. For example, during chemotaxis a bacterium can sense the quality and quantity of certain chemicals in its environment and swim towards them (if they are useful nutrients) or away from them (if they are harmful substances). During aerotaxis, bacteria swim toward or away from O2. For a few pathogens motility is known to be a determinant of virulence. In the case of Vibrio cholerae, the vibrios apparently swim (laterally) into the intestinal mucosa to avoid being flushed out by the peristaltic action of the gut. Flagella are antigenic, and therefore, vulnerable to attack by host antibody molecules. Antibody molecules directed against flagellar antigens can agglutinate and/or immobilize bacterial cells, or possibly opsonize them from phagocytosis, which presumably would aid in host defense [2,6,22,32]. Fimbriae and Pili are interchangeable terms used to designate short, hair-like structures on the surfaces of bacterial cells. Fimbriae are shorter and stiffer than flagella, and slightly smaller in diameter. Like flagella, they are composed of protein. A specialized type of pilus (always called a pilus), the F or sex pilus, mediates the transfer of DNA between mating bacteria, but the function of the smaller, more numerous common pili is quite different. Inasmuch as many bacteria are able to exchange genes for virulence by means of conjugation, the sex pilus which confers the ability to conjugate, may well play a role in the their assembly of virulence determinants [2,6,22,32].Common pili or fimbriae are often involved in adherence (attachment) of bacterial cells to surfaces in nature. In medical situations, they are major determinants of bacterial virulence because they allow pathogens to attach to (colonize) tissues and, sometimes, to resist attack by phagocytic white blood cells. As surface structures on the bacterial cell, the functions of fimbriae overlap with those of capsules discussed below. Fimbriae are also antigenic and secretory antibodies (IgA) will often block bacterial colonization, while circulating antibodies (IgG or IgM) will opsonize bacterial cells for phagocytosis [2,6,22,32]. Most bacteria contain some sort of a polysaccharide layer outside of the cell wall or outer membrane. In a general sense, this layer is called a capsule. A true capsule is a discrete detectable layer of polysaccharides deposited outside the cell wall. A less discrete structure or matrix which embeds the cells is a called a slime layer. Slime layers are equivalent to biofilms (below) A type of capsule found in bacteria called a glycocalyx is a thin layer of tangled polysaccharide fibers which is a observed on the surface of cells growing in nature [2,6,22,32]. Some microbiologists consider all types of exopolysaccharides to be glycocalyx. Capsules, slime layers, and glycocalyx are known to mediate specific or non specific adherence of bacteria to particular surfaces. The also protect bacteria from engulfment by predatory phagocytes and from attack by antimicrobial agents [2,6,22,32]. In nature, and in many medical situations, colonies of bacteria construct and live in a biofilm, made up principally of capsule material. A biofilm usually consists of a consortium (mixture) of bacteria living in a matrix of slime which is secreted by one of the bacterial members. Dental plaque is an example of a natural biofilm, as is a slimy mass of bacteria attached to a rock in a mountain stream. In medical situations, bacteria in a biofilm may have certain advantages over planktonic counterparts. For example, biofilm bacteria may be less susceptible to phagocytosis, drugs, or

neutralizing antibodies [2,6,22,32]. Many polysaccharide capsules possess an antigenic epitope so they will induce and react with host antibodies. Where the capsule is a main determinant of virulence of a pathogen (e.g. *Streptococcus pneumoniae*) antibodies against the bacterium neutralize its virulence.

S-layers are proteins in the outermost cell envelope of a broad range of bacteria. S-layers can function as adhesins, enabling the bacterium to adhere to host cell membranes and tissue surfaces in order to colonize. Many of the cell-associated protein adhesins used by pathogens are components of the S-layer. The S-layer may protect bacteria from harmful enzymes or changes in pH. Like many other surface components, S-layers contribute to virulence by protecting the bacterium against complement and attack by phagocytes [2].The surface properties of a bacterium are determined by the exact molecular composition of its membrane and cell envelope, including capsules, glycocalyx, S-layers, peptidoglycan and LPS, and the other surface structures, such as flagella and pili or fimbriae [2]. the past 3 decades of research, it has become apparent that one of the most common surface structures on bacteria are monomolecular crystalline arrays of proteinaceous subunits termed surface layer or S-layer. S-layer subunits can recrystallization at interfaces [28]. S-layers have now been identified in hundreds of different species belonging to all major phylogenetic groups of bacteria, and they represent a feature common to almost all Archaea. This widespread occurrence on prokaryotic organisms has not always been appreciated, since S-layers are often lost during prolonged cultivation under laboratory conditions, consequently, fresh isolates should be examined by electron microscopic techniques as soon as possible, preferably by freeze-etching of pellets of unwashed cells in complete medium [25-29,31]. For high-resolution studies on the mass distribution of S-layer lattices, negatively stained preparations of isolated or recrystallized S-layers have been used .More recently, high-resolution images of S-layers were also obtained by applying underwater atomic-force microscopy [25-29,31].The S-layer lattices can have oblique (p1, p2) square (p4), or hexagonal (p3, p6) symmetry. The data now available show that hexagonal symmetry is predominant among archaea .Depending on the lattice type, one morphological unit consists of one, two, four, three, or six identical (glyco) protein subunits, respectively, and they exhibit center-to-center spacings of approximately 2.5 to 35 nm. S-layers are 5 to 25 nm thick, and they reveal a rather smooth [25-29,31].

Cell surface layers are common structures of the bacterial cell envelope with a lattice-like appearance that are formed by a self-assembly process. Frequently, the constituting S-layer proteins are modified with covalently linked glycan chains facing the extracellular environment. S-layer glycoproteins from organisms of the Bacillaceae family possess long, O-glycosidically linked glycans that are composed of a great variety of sugar constituents. The observed variations already exceed the display found in eukaryotic glycoproteins. Recent investigations of the S-layer protein glycosylation process at the molecular level, which has lagged behind the structural studies due to the lack of suitable molecular tools, indicated that the S-layer glycoprotein glycan biosynthesis pathway utilizes different modules of the well-known biosynthesis routes of lipopolysaccharide O-antigens (1,23,24,30).

The subject of this study was study role of surface structures in increase of pathogenesis in pathogen Bacteria.

Discussion:

About the surface structure in all of similar original and review articles, there is consensus that the existence of this surface structure in bacteria lead to increased resistance into hard physical and chemical agent and pathogenesity in bacteria [2,7-9].

Result of previous study show high frequency of antibiotic resistant and S-layer producer in pathogen bacteria [3-6,17]. According previously result frequency of S-layer in *B.cereus* st. isolated from hospital biotic condition was 84/6% and frequency of S-layer in *B.cereus* st. isolated from hospital abiotic condition was 7/7% [7.10]. According to antibiogram result, S-layer non producer strain, in comparative S-layer producer strain, were sensitive to antibiotics [7,12,18-21].

Surface structures in bacteria such as cell wall, cell membrane, flagella, pilus, glycocalyx, capsules, slime layers and S-layers are virulence agent in bacteria and preset this structure in pathogen bacteria due to increase of pathogenesis and infection resistant in host.

The Surface structures in bacteria has been associated with a number of possible functions, these include the following:

1- The Surface structures protect bacteria from harmful enzymes (S-layers from Bacillaceae were found to function as adhesion sites for cell-associated exoenzymes) and antimicrobial agents; 2- The Surfaces structures protect bacteria from changes in pH; 3- The Surface structures protect bacteria from attack by bacterial parasites such as *Bdellovibrio bacteriovorus*, and from bacteriophages; 4- The Surface structures can function as an adhesin, enabling the bacterium to adhere to host cells and environmental surfaces, colonize, and resist flushing; 5- The Surfaces structures may contribute to virulence by protecting the bacterium against complement attack and phagocytosis, and 6- The Surface structures may act as a as a coarse molecular sieve.

The Surfaces structures can contribute to virulence when they are present as a structural component of the cell envelope of pathogens [13-16].

Regarding this point that the surface structures can protect pathogen bacteria into some of environmental hard condition and chemical and bimolecular, present this structure in bacteria, lead to nosocomial antibiotic resistance in human.

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