

Chapter 10

Significance of Hydrophobicity in the Adhesiveness of Pathogenic Gram-Negative Bacteria

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Adhesiveness as a common attribute of pathogenic bacteria is well established (5, 23). This property is generally accepted as a requirement for pathogens to counter the host mechanisms that are designed to cleanse the surfaces physically of unwanted particles, including microorganisms. These mechanisms include peristalsis in the small intestine, micturition in the urinary tract, and the mucociliary escalator of the respiratory tract. After attachment, some microorganisms proliferate at and remain on the surface of the host, whereas others proceed to penetrate to deeper tissue or other locations.

Considerable effort has been directed at defining the role of the nonflagellar filamentous surface appendages, termed fimbriae or pili, as adhesins in the specific interaction with epithelial cell surface receptors (13, 60). In addition, hydrophobic interaction is believed to play a role in overcoming the repulsive forces between the bacterial cell and the host cell (5, 93). This chapter provides a brief survey of the latest developments in the adhesion capabilities of certain pathogenic gram-negative bacteria. Current knowledge about the role of cell surface hydrophobicity in the initial binding of these bacteria to host cells is evaluated.

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ESCHERICHIA COLI

Members of the species *Escherichia coli* consist of innocuous strains that reside in the intestines of humans and animals as well as those that cause urinary tract infection and distinct syndromes of diarrheal disease. The diarrheagenic strains include (i) enterotoxigenic *E. coli* (ETEC), (ii) enteroinvasive *E. coli* (EIEC), (iii) enteropathogenic *E. coli* (EPEC), and more recently (iv) enterohemorrhagic *E. coli* (EHEC) (90). The uropathogenic and ETEC strains provide a clear correlation between the presence of a specific fimbrial component and the ability of bacteria harboring it to attach to a given host epithelium (13, 60).

ETEC

Strains belonging to the ETEC group are associated with diarrheal disease in pigs, calves, lambs, and humans. Two general requirements are involved for the onset of disease: (i) the ability to attach to and colonize the mucosal epithelium and (ii) the elaboration of heat-labile or heat-stable enterotoxins that produce the diarrheal response. Both properties are plasmid encoded; a plasmid may simultaneously carry genes for both properties (29).

K88 antigen was the first fimbrial adhesin to be described as a virulence factor (51). It is present on many ETEC isolates from piglets with severe diarrhea (13, 60) and mediates the specific attachment of the bacteria to the porcine intestinal epithelium. Other recognized adhesins include K99 (associated with diarrhea in calves, lambs, and piglets), 987p (associated with diarrhea in piglets), and F41 (associated with diarrhea in calves) (13, 60). Adhesins associated with diarrhea in humans include CFA/I, CFA/II, CFA/III, and CFA/IV (13, 37, 60, 62). CFA/II consists of three fimbrial antigens (CS1, CS2, and CS3). CFA/IV also consists of three antigens, two of which (CR4 and CR5) are fimbrial antigens; CS6 has no demonstrable fimbrial structure (62).

Smyth et al. (103) first reported the association of K88 antigen with high cell surface hydrophobicity. Subsequently, similar observations were made with strains possessing antigens K99 (12) and F41 (75). However, strains carrying 987p fimbriae expressed relatively low cell surface hydrophobicity (113).

Strains with CFA/I and CFA/II adhesins expressed higher surface hydrophobicity than did the animal ETEC strains (21, 69). The screening of human ETEC strains for cell surface hydrophobicity led to the recognition of the CFA/III fimbriae (36, 37). All the strains with CFA/IV adhesin were highly hydrophobic (73) except those possessing only the CS6 antigen. The latter strains were subsequently shown to be nonadher-

ent to human small intestinal antigens and cultured human intestinal mucosa (62).

Uropathogenic *E. coli*

Uropathogenic *E. coli* resides harmlessly in the colon but is the important pathogen in urinary tract infections (57, 106). Attachment to the human urinary tract epithelium is mediated by a variety of adhesins termed P fimbriae (63, 106). Successful infection of the urinary tract definitely requires the presence of such adhesins (106), which are not affected by D-mannose and are correlated with mannose-resistant agglutination of human erythrocytes.

There is general agreement that most uropathogenic *E. coli* strains express cell surface hydrophobicity. However, there is much disagreement over which fimbriae (P or type 1) are associated with elevated surface hydrophobicity (20, 40, 44, 70, 79, 80). Sherman et al. (101) observed differences in the cell surface hydrophobicity and binding capability of a series of *E. coli* expressing type 1 fimbriae. There was a close correlation between increased cell surface hydrophobicity and *in vitro* intestinal membrane binding mediated by type 1 fimbriae. The isolation of a few strains of uropathogenic strains (40) that do not express high cell surface hydrophobicity indicates that this surface property may not always be critical in the initiation of urinary tract infection.

EIEC and *Shigella* spp.

Like shigellae, strains of EIEC cause a dysenterylike disease in humans. The two groups of organisms exhibit similar pathogenesis, and there is considerable homology among the virulence plasmids (94). Virulence is dependent on the ability of these pathogens to invade and multiply in intestinal cells (32). Their invasiveness is mediated by a high-molecular-weight plasmid (1). Stugard et al. (105) recently reported that this plasmid encodes for a 101-kilodalton protein responsible for Congo red or heme uptake. They suggested that binding of heme compounds by the pathogen *in vivo* may disguise it as a desirable molecule for the intestinal epithelial cell. The consequence would be for the host cells to bind the heme-coated bacteria through heme receptors and then actively endocytose the bacterial cells. The possibility exists that the adhesion phase is aided by an increase in bacterial surface hydrophobicity. *Shigella flexneri* has been shown to express plasmid-associated hydrophobicity (87, 99), a property that was correlated with the ability of pathogen to bind to HeLa cells (84). The capability of EIEC

strains to express high cell surface hydrophobicity has not been determined.

EPEC

Strains belonging to the EPEC group cause diarrhea but do not produce heat-labile or heat-stable enterotoxin or exhibit *Shigella*-like invasiveness (18). Cravioto et al. (12) reported the ability of EPEC strains to attach to HEP-2 cells. This attachment capability subsequently was demonstrated to be encoded on a 60-megadalton plasmid (2, 61, 78). Scaletsky et al. (97) observed two distinct patterns of attachment of EPEC strains to HeLa cells. The group exhibiting the localized adhesion (LA) pattern formed microcolonies on the HeLa cell surface, whereas the group exhibiting the diffused adhesion (DA) covered the HeLa cell uniformly. Nataro et al. (78) provided evidence that these adhesion patterns were due to at least two genetically distinct adhesins. Moreover, they observed that the EPEC strains with the DA pattern were more hydrophobic than those that exhibited the LA pattern. The EPEC strains that Wadström et al. (113) examined recently apparently consisted of both groups, since 63 of the 157 strains were hydrophobic. Whereas the LA adhesin remains largely uncharacterized, the DA adhesin was shown to be fimbrial in nature (61).

RDEC-1 is an EPEC strain that causes diarrhea specifically in rabbits, with lesions morphologically indistinguishable from those caused by human EPEC strains (7). This rabbit pathogen expresses a mannose-resistant fimbrial adhesin (designated AF/RI) that mediates species-specific adherence to the glycoproteins of the rabbit mucosa (9). Mutants lacking the ability to express the fimbriae produced less severe disease (115). Type 1 mannose-sensitive fimbriae which are also expressed by RDEC-1 may promote bacterial attachment to the mucosa to some extent (8, 101). Sherman et al. (101) reported that both fimbrial adhesins were associated with increase in the hydrophobicity of RDEC-1. All 32 strains of *E. coli* isolated from rabbits with diarrhea were recently shown to exhibit high cell surface hydrophobicity (4).

EHEC

EHEC is a recently recognized group of pathogenic *E. coli* strains that cause hemorrhagic colitis in humans characterized by abdominal cramping and bloody diarrhea but little or no fever (90). Although the disease has been attributed to a particular serotype (O157:H7), strains from other serogroups also fulfill the same pathological criteria (68).

The mechanism of virulence of EHEC strains is unknown. The

bacteriophage-mediated cytotoxins that are neutralized by Shiga toxin antiserum are putative virulence factors (104). These strains have been shown to express a plasmid-encoded fimbrial adhesin that mediates bacterial attachment to cultured Henle 407 intestinal cells (56). Unfortunately, subsequent studies did not lend support to the notion that these factors play significant roles in the virulence of EHEC strains. When infected with plasmid-bearing and cytotoxin-positive EHEC strains and their plasmidless or cytotoxin-negative derivatives, all gnotobiotic piglets were shown to be affected with equal severity of diarrhea and characteristic mucosal lesions (111). EHEC strains did not appear to express high cell surface hydrophobicity (52, 100).

SALMONELLAE

Salmonellae are enteroinvasive pathogens that require attachment to the luminal surface to counter the peristaltic cleansing motion of the intestine. However, little is known about the role hydrophobic interaction plays in the initial binding of the pathogens to the host cell. Recently, Baloda et al. (3) observed no association between the formation of mannose-sensitive fimbriae and cell surface hydrophobicity among strains of *Salmonella typhimurium* and *S. enteritidis*.

Jones (47) suggested that a plasmid-mediated mannose-resistant hemagglutinin (48, 49) may provide the means for *S. typhimurium* cells to attach to the intestinal wall. The chromosomally encoded mannose-sensitive type 1 fimbrial adhesin was much less effective than the plasmid-mediated mannose-resistant hemagglutinin in promoting adhesion or internalization of HeLa cells (49, 50). There was a reduced ability of the pathogen to colonize the mouse intestine, with the accompanying loss of virulence upon curing of the adhesin plasmid (48). Although a plasmid was indeed required for the virulence of the pathogen, Hackett et al. (31) observed no change in the ability of *S. typhimurium* to bind to HeLa cells with the loss of the plasmid. Moreover, both plasmid-bearing and plasmid-free strains invaded the Peyer's patches of the small intestine to the same extent. In a study of six classes of *TnphoA* mutants of *S. choleraesuis* that were unable to bind or enter epithelial cells, none of the insertions were in the genes encoding type 1 fimbriae or mannose-resistant hemagglutinin (23).

Recently, Finlay et al. (24) provided evidence that the binding and invasive capabilities of *S. typhimurium* and *S. choleraesuis* toward epithelial cells required de novo synthesis of several new bacterial proteins. Induction of these proteins required specific glycoproteinlike

surface receptors. Transposon mutants that were unable to synthesize these proteins became nonadherent and noninvasive to eucaryotic cells and were avirulent to mice. Whether de novo synthesis of the proteins has any effect on the surface hydrophobicity of the pathogens was not investigated.

YERSINIA ENTEROCOLITICA

The invasive enteric pathogen *Yersinia enterocolitica* requires the presence of a 42- to 48-megadalton plasmid for the pathogenesis of disease (11, 28, 118). This plasmid, whose precise role is unclear, encodes for 16 to 20 proteins (86, 102). A number of these proteins are located on the outer membrane of the bacteria, one of which is a 225-kilodalton protein (YOP1).

The ability of *Y. enterocolitica* grown at 37°C but not at 25°C to express plasmid-mediated cell surface hydrophobicity (65, 98) was an early indication of attachment to the host cell surface as a critical step in *Yersinia* infection. Heesemann et al. (33) provided evidence that the virulence plasmid directs the capability of *Y. enterocolitica* to bind to HEp-2 cells. Mantle et al. (71) recently demonstrated that the binding of *Y. enterocolitica* to brush border membranes isolated from a rabbit intestine is associated with the virulence plasmid. Moreover, they showed that attachment was greater in those regions of the gut most affected during yersiniosis, namely, the distal small intestine and the proximal colon (82, 83).

Heesemann and Grüter (34) provided evidence that the binding capability of *Y. enterocolitica* is mediated by YOP1. They succeeded in mobilizing a fragment of the virulence plasmid that encodes YOP1 into a nonadherent *Y. enterocolitica* strain, transforming it into a transconjugant that is adherent to HEp-2 cells. Kapperud et al. (55) demonstrated in orally infected mice that excretion of YOP1-positive cells was prolonged compared with excretion of YOP1-negative cells. YOP1 appears to be a structural component of a matrix of fibrillae that cover the bacterial surface (54, 66). The fibrillae impart some rather striking properties to the pathogen which significantly influence their binding potential. This surface structure appears to behave as an adhesin, enabling the bacteria to attach to each other (autoagglutination) and to guinea pig erythrocytes (hemagglutination) (54, 55). Fibrillae formation is associated with increased cell surface charge and hydrophobicity (66), forces that may play a decisive role in the initial phase of *Yersinia* infection.

Certain strains of *Y. enterocolitica* have also been shown to elaborate

chromosomally mediated fimbriae at low temperatures but not at 37°C (21, 81). Their formation is associated with increased cell surface hydrophobicity and agglutination of various animal erythrocytes (21, 58). There was no correlation between the presence of the fimbriae and ability to bind to human epithelial cells (81). This surface structure does not appear to have any significance in the intestinal colonization by *Y. enterocolitica*.

VIBRIO CHOLERAE

Cholera is an infectious diarrheal disease caused by *Vibrio cholerae*, a noninvasive bacterium that colonizes the small intestinal epithelium and subsequently secretes a protein toxin. Whereas the action of cholera toxin is well understood (26, 35), the colonization phase of cholera is far from clear.

By analogy to the adhesin-receptor interactions observed with ETEC strains, the adherence capability of *V. cholerae* has been suggested to be associated with hemagglutinating activity (46, 67). Several cell-bound and soluble hemagglutinins have been reported (22, 46, 67).

Recently, Taylor et al. (107) identified a 20.5-kilodalton protein that is a major subunit of a *V. cholerae* fimbria. Its expression was associated with increased mouse intestine colonization, an enhanced cell-cell interaction (autoagglutination), hemagglutination, and a dramatic increase in the surface hydrophobicity of *V. cholerae*. Expression of the fimbrial protein was shown to be coordinately regulated with the formation of the cholera toxin. On the other hand, another hemagglutinating fimbrial structure consisting of a 16-kilodalton protein had no effect in the binding capacity of *V. cholerae* (39).

Kabir and Ali (53) suggested that nonspecific hydrophobic interaction mediated by outer membrane proteins may play a major role in the binding of *V. cholerae* to the intestinal mucosa. They noted no close correlation in the effect of growth conditions on the emergence of hydrophobic cells of *V. cholerae* and their hemagglutinating activity. This observation is consistent with a recent report by Teppema et al. (108) that strains of *V. cholerae* with or without hemagglutinating activity in vitro were equally pathogenic as determined in an adult rabbit ligated-gut model. Further studies are needed to determine what surface component(s) promotes increased hydrophobicity of *V. cholerae* and subsequent attachment to the intestinal wall.

MESOPHILIC AEROMONADS

Besides being an important cause of disease in fish, mesophilic aeromonads have been implicated in recent years as one of the major

agents of gastroenteritis (1, 42). They also cause severe bacteremia and wound infection. In mouse studies (41), *Aeromonas sobria* strains tended to be the most virulent, whereas strains of *A. caviae* tended to be the least virulent; the category of *A. hydrophila* tended to fall between these two species. A similar virulence pattern was observed with rainbow trout (85). Although the virulence mechanism of the pathogens has not been established, a number of potential virulence factors such as hemagglutinins, enterotoxins, and invasiveness have been described (1, 42, 43).

The binding of mesophilic aeromonads to the intestinal wall would be considered a critical initial step in the development of gastroenteritis. It is therefore significant that Burke et al. (6) observed a strong association between certain hemagglutinating patterns and diarrhea-producing capability of these organisms. Recently, Clark et al. (10) reported the binding capability of these aeromonads to mouse adrenal cells in association with their piliated and nonpiliated attachment mechanisms. The more virulent species (*A. sobria* and *A. hydrophila*) tended to be more adherent than *A. caviae*. However, there was no correlation between adherence capability and hydrophobicity. In fact, avirulent *A. caviae* strains were shown recently to be more hydrophobic than the strains of *A. sobria* and *A. hydrophila*, which displayed a high degree of virulence in fish (85).

BORDETELLA PERTUSSIS

Bordetella pertussis is a respiratory tract pathogen that is the causative agent of pertussis, or whooping cough, in children. Components of *B. pertussis* that warrant consideration as possible virulence determinants include adenylate cyclase toxin, agglutinogens, dermonecrotic toxin, filamentous hemagglutinin, pertussis toxin, and tracheal cytotoxin (77, 114). The genes mediating these factors are coordinately and reversibly regulated by growth conditions (phenotypic modulation). Thus, the pathogen is in the so-called avirulent phase when none of these factors are produced at growth conditions such as incubation temperatures much less than 37°C or high concentrations of MgSO₄ (64) or nicotinic acid (74) in growth media. Under permissive conditions, *B. pertussis* reverts to the virulent phase when expression of the virulence factors resumes.

To counter the mucociliary clearance mechanism of the host, the pathogen needs to bind to ciliated epithelial cells. The agglutinogens (serotype 2 and 6 fimbriae), pertussis toxin, and filamentous hemagglutinin (FHA) have been implicated as mediators of attachment in assays with tissue culture cells (30, 88). However, only the last two hemagglutinating proteins look promising as attachment factors (109, 112). They

have been shown to act in concert as adhesins to human ciliated epithelial cells (109). Evidence from in vitro studies as well as in vivo in animal models showed that FHA was essential for initial colonization of the upper respiratory tract (89, 109, 110). However, this surface structure was not found to be essential for colonization of the lower respiratory tract of mice (59, 110). Systemic immunization of mice with FHA provided significant protection against aerosol challenge of *B. pertussis* cell suspension (59).

Robinson et al. (92) reported high cell surface hydrophobicity among the virulent-phase cells of *B. pertussis* and a marked reduction when the culture was chemically induced to revert to the avirulent phase. Fish et al. (25) suggested FHA as the likely component responsible for the cell surface hydrophobicity of the pathogen. Their conclusion was based on the examination of a series of spontaneous mutants characterized with reference to the expression of hemolysin, pertussis toxin, adenylate cyclase, and FHA. Indeed, FHA has been characterized as a highly hydrophobic, self-aggregating protein (109).

PSEUDOMONAS AERUGINOSA

Pseudomonas aeruginosa is considered as a major respiratory pathogen of immunocompromised and immunosuppressed patients (91). Successful colonization of the respiratory system of a patient by this pathogen appears to be initiated upon attachment of the organism to the respiratory epithelium (45, 116). Several studies have indicated that the pilus adhesin provides the initial adhesion of *P. aeruginosa* to the respiratory epithelial surface (16, 17, 117). It has also been shown to be a virulence factor in burn wound infection (96). This adhesin is composed of a single monomer protein, called pilin (95), whose epithelial cell-binding domain was recently located at the highly conserved C-terminal region of the protein (15, 38).

Studies indicated that hydrophobic interaction does not seem to play a major role in the binding capability of *P. aeruginosa* (19, 27, 72). However, Garber et al. (27) cautioned that the procedures used for measuring the surface hydrophobicity of this organism may not be sensitive enough to detect hydrophobic areas of the pilus adhesin.

CONCLUSION

On the basis of the possibility that hydrophobic interaction plays a major role in the initial phase of pathogenesis, a survey (summarized in

Table 1. Association of Hydrophobicity and Adhesiveness with the Virulence of Pathogenic Gram-Negative Bacteria

Bacteria	Hydrophobicity	Adhesin	Reference(s)
<i>Escherichia coli</i>			
ETEC	+	Fimbriae	13, 37, 73, 113
Uropathogenic	+	Fimbriae	70, 101, 106
EPEC			
LA	-	?	78, 97
DA	+	Fimbriae	61, 78, 97, 101
EHEC	-	Fimbriae (?)	52, 56, 100, 111
EIEC	?	Surface protein	105
<i>Shigella</i> spp.	+	Surface protein	84, 87, 99, 105
Salmonellae	?	Surface protein(s)	24
<i>Yersinia enterocolitica</i>	+	YOP1 (fibrillae)	34, 55, 66, 71
<i>Vibrio cholerae</i>	+	Fimbriae (?)	53, 107
Mesophilic aeromonads	-	?	6, 10, 43, 85
<i>Bordetella pertussis</i>	+	FHA	25, 59, 109, 110
<i>Pseudomonas aeruginosa</i>	-	Fimbriae	17, 19, 72, 96

Table 1) was made of pathogenic gram-negative bacteria that affect the enteric, urinary, and respiratory tracts. Six of the eleven pathogenic species or groups were reported to express high cell surface hydrophobicity that is associated with bacterial binding to the host cell. High cell surface hydrophobicity was not expressed by a subgroup of EPEC strains.

It may be premature to conclude at this time that hydrophobic interaction is not a universal phenomenon in bacterium-host interaction. As pointed out by Rosenberg and Kjelleberg (93), the participation of hydrophobic interaction in adhesion phenomenon is often overlooked in various studies. This is certainly true for salmonellae, which have been shown recently to require de novo synthesis of several proteins to bind to cultured epithelial cells (24). Further studies along this line should provide some indication as to whether cell surface hydrophobicity of bacteria in situ differs greatly from results obtained from growth media. Moreover, the methods used in measuring cell surface hydrophobicity may be inadequate. Hydrophobic interaction chromatography (103) and salt aggregation (69) have been the methods of choice for most studies on gram-negative bacteria. Recent studies reported a general lack of correlation of results from methods for measuring bacterial hydrophobicity (14, 76). Consequently, reliance on a single method may not indicate the true extent of hydrophobicity of the bacterial surface (see also Rosenberg and Doyle, this volume).

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