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Adhesion of Piliated *Escherichia coli* Strains to Phagocytes: Differences Between Bacteria with Mannose-Sensitive Pili and Those with Mannose-Resistant Pili

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Escherichia coli with mannose-resistant (MR) pili, in contrast to those with mannose-sensitive (MS) pili, did not adhere to rat peritoneal macrophages and human polymorphonuclear granulocytes, as measured by use of radioactive bacteria and by the chemiluminescence response induced by the cell contact. With some MS-piliated *E. coli* strains, unpiliated bacteria, obtained by growth at a pilus-restrictive temperature, did show MS adherence to phagocytes, presumably by virtue of bacterial cell wall adhesins which, like MS pili, recognize α-mannose-containing structures of the phagocyte membrane. Possible roles of MR pili, MS pili, and MS cell wall adhesins in the unspecific cellular host defense are discussed.

Bacterial pili (7) or fimbriae (11) are known to mediate adhesion of bacteria to the surface of eucaryotic cells (8, 19, 20, 34, 37, 39). The adhesion may or may not be inhibited by α-mannosides and is then termed mannose sensitive (MS) or mannose resistant (MR) (10, 11). The respective pili are called MS or MR pili. They represent groups of adhesins which have only in common that they do or do not recognize receptors containing α-mannose. *Escherichia coli* with MS pili, including the common type 1 pili (9, 15, 26, 33, 34), agglutinate yeast cells and guinea pig erythrocytes, whereas those with MR pili agglutinate human erythrocytes or erythrocytes of other species (10, 11, 15, 33). MS pili recognize receptors which contain α-mannose, presumably in the terminal position of a glycoprotein(s) in the plasma membrane of the mammalian cells. MR pili are a large group of adhesins which may vary widely with respect to composition, serological specificity, morphological appearance, genetic determination, or receptor specificity. Some MR pili of urinary tract-infective *E. coli* recognize α-galactose or α-*N*-acetylgalactosamine (9, 15, 18, 21, 28, 33). Receptors of other MR pili, such as the colonization factor antigens of enteropathogenic *E. coli* or the MR pili of some uropathogenic *E. coli*, are not known (10, 13, 14, 17, 40). Thus, it should be clear that the only common denominator of MR pili is that they are non-MS.

Generally, *E. coli* with MR pili adhere to epithelial cells but much less to tissue culture cells (17, 18, 30, 38), whereas those with MS pili adhere to many tissue culture cells and also to the urinary tract glycoprotein known as Tamm Horsfall protein (10, 17, 30, 34). It was found that MS pili mediate adhesion of *E. coli* to phagocytic cells, presumably via mannose-containing structures of the plasma membrane (4, 5, 23, 32).

Since most *E. coli* causing urinary tract infections or diarrhea have MR pili which play a role in the infective process (12–14, 19, 39, 40), we wanted to know whether phagocytic cells also have, like many epithelial cells, receptors for MR pili. To evaluate the situation in a nonimmune host, we tested the adhesion of *E. coli* with MR or MS pili to rat peritoneal macrophages (PM) and to human peripheral polymorphonuclear granulocytes (PMN) in the absence of antibody. The adhesion was monitored by using radiolabeled bacteria as well as by measuring the chemiluminescence which accompanies the adhesion and phagocytosis of inert particles and bacteria (2, 3, 6, 24, 27, 36).

MATERIALS AND METHODS

Bacteria and cultivation. The bacterial strains used are listed in Table 1. They were kept on Loeb agar and were cultivated before the tests by three daily passages in static culture in Merck standard broth and then grown in the same medium for 18 h with or without [^{14}C]acetate (10 μCi/ml), according to the test used. To repress the formation of pili, the bacteria were grown at 20°C instead of 37°C for the same period of time, as described above, except that radiolabeling of the bacteria was done at 20°C for 24 h. The final bacterial suspensions were counted in a Neubauer chamber, and the radioactivity of a sample was determined with a Packard scintillation counter.

Isolation of rat PM and human PMN. PM were obtained from 8- to 12-week-old L.WP rats which were

TABLE 1. *E. coli* strains used in adhesion to rat PM and human PMN[a]

Strain	Serotype	*E. coli* pilus type	Isolated from:	Agglutination of:			Reference
				Human RBC	Guinea pig RBC	*S. cerevisiae*	
2699	O6:K13:H$^-$	MS	Urine	MS	MS	MS	17
20043	O83:K:H$^+$	MS	Feces	MS	MS	MS	22
2131	K-12,CT1	MS	Laboratory strain		MS	MS	
20629	O78:H11,CFA/I$^-$	MS	Feces		MS	MS	13
20157	O6:K2:H	MS + MR	Urine	MR	MS	MS	17, 30
2980	O18ac:H	MR	Feces	MR			17, 40
20003	O4:K1:H$^-$	MR	Blood	MR			17
20025	O4:K12:H$^-$	MR	Urine	MR			17
20215	O2:H	MR	Urine	MR			17

[a] MS pili mediate MS agglutination of guinea pig erythrocytes (RBC) and *S. cerevisiae*; MR pili mediate MR agglutination of human RBC. CT1, Common type 1 pili; CFA/I, colonization factor antigen I (13, 14).

kept under specific-pathogen-free conditions. The animals were killed with ether, and 60 ml of ice-cold Eagle medium containing penicillin (100 IU/ml), dehydrostreptomycin (100 μg/ml), and heparin (5 IU/ml) was injected into the peritoneum. After slight massage, the contents were removed with a syringe. Generally, about 50 ml could be recovered containing 10^7 peritoneal cells (PC) per animal. The cell suspension was centrifuged at 1,000 × *g* for 10 min and suspended in Eagle medium containing penicillin and dehydrostreptomycin as above. About 90 to 95% of the PC were viable, as evidenced by staining with trypan blue. Adherent cells, which were taken as PM, were obtained from the PC by incubation in Eagle medium for 1 h at 37°C in an atmosphere of 12% carbon dioxide, using tissue culture vessels.

Human PMN were isolated from blood in a modification (H. Mossmann, personal communication; B. Schmitz, dissertation, University of Giessen, Giessen, West Germany, 1980) of elutriative centrifugation (35), using a JE-6 elutriator rotor in a Beckmann J-21B centrifuge.

Adhesion assay. Suspensions (1 ml) of 5×10^5 PC or 2.5×10^5 PMN in Eagle medium containing sodium bicarbonate, penicillin, and dehydrostreptomycin as above were placed in the wells of tissue culture plates (Costar). After 1 h at 37°C in a carbon dioxide atmosphere (12%), the medium was removed; the adherent cells were then washed twice with phosphate-buffered saline (PBS) at 37°C and incubated for 2 h (PMN) or 16 h (PM) in Eagle-sodium bicarbonate medium containing penicillin and dehydrostreptomycin in a carbon dioxide atmosphere. The monolayers were then washed with 1 ml of PBS per well, and to each well 1 ml of PBS containing 5×10^7 bacteria was added. After incubation at 37°C on a reciprocating shaker (120 rpm) for 40 min, unbound bacteria were removed by three washings with 1 ml of PBS each. The cell layers were solubilized by incubation in 0.5% sodium dodecyl sulfate and scraping with a rubber policeman. The solubilized mixtures were counted in a Packard liquid scintillation counter.

Chemiluminescence assay. To determine chemiluminescence, 2×10^6 PC or 2×10^5 PMN were cultivated in liquid scintillation vials in the same way as described for the adhesion assay. After washing of the monolayers twice with 2.5 ml of warm PBS, 2.5 ml of PBS containing 50 μl of a solution of 2 mg of luminol (5-amino-2,3-dihydro-1,4-phthalazinedione) and 8 μl of triethylamine in 1 ml of PBS was added. The background chemiluminescence of the cells (1×10^5 to 2×10^5 cpm) was then measured in the coincidence-off mode with a modified scintillation counter (Packard C2425) containing two selected photomultipliers with high sensitivity in the blue-green region of the light spectrum. After 1 h, 10^8 bacteria were added to the monolayers of PM, and 10^7 bacteria were added to the monolayers of PMN. The chemiluminescence response was then measured for 1 h.

RESULTS

Characterization of the bacteria. The bacteria listed in Table 1 were all piliated, as evidenced by electron microscopy. Their agglutination properties were designated as MS if their agglutination of guinea pig erythrocytes and *Saccharomyces cerevisiae* could be inhibited with 50 mM methyl-α-mannoside (αMM) and as MR if their agglutination of human erythrocytes could not be inhibited with αMM. With the exception of strains 20043 and 20629, the adhesive activities of these strains have recently been described (17). Strain 20629 is identical to *E. coli* H10407p of Evans et al. (13), strain 20043 corresponds to *E. coli* O83 of Lodinova et al. (22), which has been used for oral vaccination of children against enteric infections, and strain 20157 is identical to *E. coli* C1212 of Ørskov et al. (30). For maximal expression of MS pili, the respective bacteria were cultivated repeatedly in static broth culture for 24 h.

Adhesion experiments. Monolayers of rat PM and human PMN were incubated in the abscence of serum with radiolabeled *E. coli*. To monitor mannose sensitivity, the experiments were performed in the absence and in the presence of 50 mM αMM (Table 2). *E. coli* strains which caused MS agglutination adhered to both types of phagocytic cells to about the same extent. The adhesion of all strains with MS pili could be

TABLE 2. Adhesion of radiolabeled *E. coli* bacteria with MS or MR pili to rat PM and to human PMN in the absence or presence of 50 mM αMM

Phagocytic cell	*E. coli* strain	Pilus type	Adhesion of radiolabeled bacteria (%)[a]		% Inhibition
			−αMM	+αMM	
PM	2699	MS	6.0	0.6	90
	20043	MS	4.8	1.3	73
	2131	MS	3.5	1.1	70
	20629	MS	2.6	2.1	20
	20157	MS + MR	1.6	0.7	56
	2980	MR	0.2	0.2	0
	20003	MR	0.2	0.2	0
	20025	MR	0.3	0.3	0
	20215	MR	0.3	0.3	0
PMN	2699	MS	5.0	1.4	72
	20043	MS	3.6	1.6	55
	2131	MS	2.0	1.1	45
	20629	MS	3.2	2.7	13
	20157	MS + MR	1.4	0.8	41
	2980	MR	0.4	0.4	0
	20003	MR	0.3	0.3	0
	20025	MR	0.5	0.5	0
	20215	MR	0.4	0.4	0

[a] Measured after 40 min of cell contact and expressed as percentage of the bacteria added.

inhibited with αMM. The low values obtained with *E. coli* exhibiting MR pili were considered background values, since they were also obtained after growth of the bacteria at 20°C (see below), and similar values are found with bacteria which do not have MR pili (6; E. Blumenstock and K. Jann, unpublished data).

Adhesion and chemiluminescence. Adhesion of inert particles and bacteria to phagocytes results in the liberation of active oxygen, which causes an increase of chemiluminescence well above that exerted by the metabolism of the resting phagocytes (2, 3). Since chemiluminescence is an indication of particle-phagocyte contact, we monitored the adhesion of the piliated bacteria to PM and PMN by measuring the chemiluminescence elicited by cell contact in the presence of luminol (6, 24, 27, 36). In these experiments, the bacterial suspensions used were always tested in agglutination of human erythrocytes and *S. cerevisiae* (Fig. 1A). Whereas the contact with bacteria which had only MS pili and which agglutinated *S. cerevisiae* resulted in a distinct chemiluminescence response of the PM, no chemiluminescence was observed when the PM were incubated with bacteria that had only MR pili and agglutinated human erythrocytes but not *S. cerevisiae*. *E. coli* 20157 with both MS and MR pili also induced chemiluminescence in PM, which indicated that the interaction of MS pili with PM was not blocked by MR pili. Strain 20629 had a very dense peritrichial flagellation. It is possible that the flagella partially prevented the interaction of the MS pili with the phagocytes. This would explain the low adhesion and chemiluminescence exhibited by this *E. coli* strain. The same results were obtained with human PMN. Both extent and kinetics of the response were like those shown in Fig. 1A.

Since adhesion mediated by MS pili can be inhibited with αMM, we wanted to know whether the chemiluminescence response was also sensitive to this inhibitor. The experiments were therefore performed in the presence of 50 mM αMM. The chemiluminescence response was much reduced under these conditions (Table 3). Inhibitions were always more pronounced with *E. coli* 2699 and 20043 than with 2131 and 20629. The latter two strains exhibited classical type 1 pili and differed from the other two strains in their agglutination patterns (see Table 1).

Effect of piliation on adhesion. Since pili seemed to play a role in both adhesion and chemiluminescence, it was interesting to compare piliated and nonpiliated bacteria. When grown at 20°C, more than 95% of the bacteria were not piliated and about 5% were only slightly piliated, as evidenced by electron microscopy (data not shown). We therefore measured the adherence of the bacteria grown at 20°C through their capacity to elicit chemiluminescence. Figure 1B shows the chemiluminescence response of rat PM to *E. coli* with MS pili which were grown at 20°C (pilus-restrictive temperature). After suppression of pili, *E. coli* 20629 did not induce chemiluminescence at all, and *E. coli* 2131 did so only slightly. In contrast, strains 2699 and 20043 gave rise to about the same

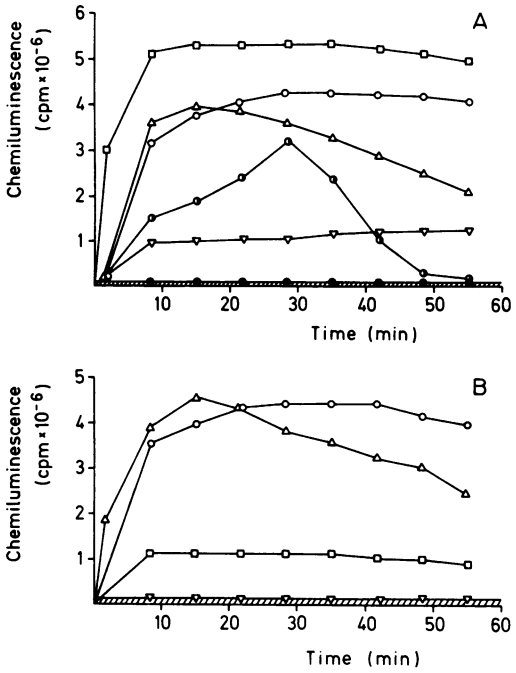

FIG. 1. (A) Chemiluminescence response of rat PM to *E. coli* with MS pili (□, strain 2131; ○, strain 2699; △, strain 20043; ▽, strain 20629) to *E. coli* with MR pili (●, strains 2980, 20003, 20025, and 20215) and to *E. coli* with MS and MR pili (◑, strain 20157). The hatched region indicates the background chemiluminescence, obtained with luminol in the absence of bacteria. The macrophages were cultivated in Packard scintillation vials. Experimental details are described in Materials and Methods. (B) Chemiluminescence response of rat PM to *E. coli* grown at the pilus-restrictive temperature of 20°C.

chemiluminescence response after growth at 20°C as after growth at 37°C. Chemiluminescence elicited by nonpiliated strain 2699 was completely inhibited by 50 mM αMM, and that obtained with strain 20043 was inhibited to about 50%. This was about the same degree of inhibition as that observed with the piliated bacteria (Table 2), indicating that the interaction of the nonpiliated bacteria via bacterial cell wall adhesins was as mannose sensitive as that via MS pili.

The validity of the chemiluminescence experiments was tested by using the same bacterial suspensions in agglutination experiments. In parallel to the results obtained with macrophages, strains 2699 and 20043, after growth at 20°C, agglutinated *S. cerevisiae*, and strains 2131 and 20629 did not. It should be mentioned that, as we have shown previously (17, 40), growth of the bacteria at 20°C affects not only the expression of MS pili, but in the same way also that of MR pili.

DISCUSSION

Although it has been reported that phagocytes have receptors for MS pili (4, 5, 23, 32), it was hitherto not known whether they also express receptors for MR pili. We therefore studied the adhesion of *E. coli* with only MS pili, only MR pili, or both, using rat PM and human PMN. To circumvent the adhesion via immune receptors, the bacteria were not opsonized; i.e., the experiments were performed in the absence of antibodies and complement.

All strains with MS pili, as checked by running agglutination of *S. cerevisiae* in parallel, exhibited MS adhesion to the phagocytes which was accompanied by luminol-dependent chemiluminescence. When bacteria which had been grown at a pilus-restrictive temperature were used, it was found that strains 2699 and 20043 exhibited MS adhesion also in the unpiliated form (and also gave MS agglutination of *S. cerevisiae*). These results show that the MS adhesion of *E. coli* does not necessarily involve pili and that it may also be mediated by MS cell wall adhesins. In this connection, it should be mentioned that we have recently demonstrated the presence of MS pili and MS cell wall adhesins in strain 2699, both of which exhibited MS agglutination of yeast cells (Eshdat, Speth, and Jann, submitted for publication).

In contrast to *E. coli* with MS pili, those with MR pili did not adhere to PM and PMN, although they agglutinated human erythrocytes in parallel experiments in an MR way. Incubation of the phagocytes with *E. coli* which had MR pili did not induce a chemiluminescence response. This was an indication that the plasma membrane of the phagocytic cells does not contain or does not express receptors for MR pili. Our results, however, do not exclude the possible existence of cryptic receptors for MR pili, which may be blocked either sterically by other com-

TABLE 3. Chemiluminescence induced by *E. coli* with MS pili in rat PM and human PMN and its inhibition by 50 mM αMM

Phagocytic cell	*E. coli* strain	Chemiluminescence induced (cpm × 10^{-6})[a]		% Inhibition
		−αMM	+αMM	
PM	2699	4.2	0.0	100
	20043	2.7	0.8	62
	2131	5.5	3.7	32
	20629	1.0	0.4	40
PMN	2699	2.3	0.0	100
	20043	1.5	0.0	100
	2131	3.3	0.5	85
	20629	1.3	0.5	60

[a] The values obtained after 25 min of cell contact are corrected for background chemiluminescence, exhibited by the phagocytes without bacteria (0.2 × 10^6 cpm).

ponents of the plasma membrane or by substituents on the molecule such as neuraminic acid. This problem is currently under study in our laboratory.

Strain 20157, suspensions of which gave both MS and MR agglutinations, also adhered to phagocytes and induced chemiluminescence. Adhesion and chemiluminescence were less pronounced than with bacteria exhibiting only MS pili. If the suspension of strain 20157 was a mixture of bacteria with only MS or MR pili, this would mean that the concentration of bacteria with only MS pili was relatively low, thus giving rise to an only moderate effect. Alternatively, *E. coli* 20157 may contain MS and MR pili on the same bacterial cell, and there is evidence in favor of such an interpretation (30). In such a case, the lower adhesion may indicate the necessity of multipoint contacts for adhesion which can be envisaged as less efficient if MS pili are interspersed on the bacterial surface with MR pili.

We found that more than 90% of the bacteria adhering to macrophages were killed in vitro after 40 min (data not shown). This was found not only with strains adhering specifically via MS pili, but also with those having only MR pili which adhered much less (5 to 10% of the MS adhesion). Since MS pili mediate adhesion of bacteria to phagocytes, more of the bacteria exhibiting MS pili are killed than of those without these adhesins. One can thus assume that *E. coli* with MS pili are more readily eliminated in vivo than are those with MR pili. This is especially so in the absence of antibody, a situation encountered at the onset of infection. *E. coli* with MR pili are often found in urinary tract infections and in diarrhea. Receptors for MR pili have been isolated from urinary tract epithelial cells (21). Intestinal epithelial cells also have receptors for MR pili, since the small intestine can be colonized with *E. coli* by virtue of MR pili (10, 12, 13). The chemical nature of these receptors is not yet known. From comparison of agglutination data with erythrocytes lacking the galactose-containing glycolipids of the blood group P system and results of adhesion experiments (17), it can be concluded that some mammalian cells (inter alia, intestinal epithelial cells) exhibit receptors for MR pili different from those isolated from uroepithelial cells. Thus, cellular receptors for MR pili may differ in occurrence and structure and, by the same token, MR pili represent a group of pili which may be structurally and functionally different. Irrespective of such differences and because of their property to mediate adhesion of *E. coli* to epithelial cells but not their lethal encounter with phagocytic cells, MR pili can be considered as bacterial assets for pathogenicity.

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