Diversity of the *Escherichia coli* Type 1 Fimbrial Lectin

Differential Binding to Mannosides and Uroepithelial Cells*

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Type 1 fimbriae are the most common adhesive organelles of *Escherichia coli*. Because of their virtual ubiquity, previous epidemiological studies have not found a correlation between the presence of type 1 fimbriae and urinary tract infections (UTIs). Recently it has become clear that type 1 fimbriae exhibit several different phenotypes, due to allelic variation of the gene for the lectin subunit, FimH, and that these phenotypes are differentially distributed among fecal and UTI isolates. In this study, we have analyzed in more detail the ability of isogenic, recombinant strains of *E. coli* expressing fimH genes of the predominant fecal and UTI phenotypes to adhere to glycoproteins and to uroepithelial cells. Evidence was obtained to indicate that type 1 fimbriae differ in their ability to recognize various mannoses, utilizing at least two different mechanisms. All FimH subunits studied to date are capable of mediating adhesion via trimannosyl residues, but only certain variants are capable of mediating high levels of adhesion via monomannosyl residues. The ability of the FimH lectins to interact with monomannosyl residues strongly correlates with their ability to mediate *E. coli* adhesion to uroepithelial cells. In this way, it would be possible for certain phenotypic variants of type 1 fimbriae to contribute more than others to virulence of *E. coli* in the urinary tract.

*Escherichia coli* is a commensal inhabitant of the mammalian large intestine and the most common cause of urinary tract infections (UTIs)\(^1\) in humans (1, 2). A variety of the so-called urovirulence factors may be important in enabling *E. coli* to become established in a urinary tract niche (1, 2), but the array of specific genetic factors found in urinary tract isolates of *E. coli* vary such that no single factor can be considered essential. Fimbriae (3), or pili (4), of *E. coli* enable the bacteria to attach to mucosal surfaces and have long been considered to be primary urovirulence factor candidates (1, 2). The role of P fimbriae in pyelonephritis is well-established, due in large measure to accumulated epidemiological evidence showing that approximately 70% of *E. coli* strains from pyelonephritis, but less than 20% of normal intestinal isolates, produce P fimbriae (1, 2, 5). Still, P fimbriae are not required for UTIs, because significant numbers of isolates from asymptomatic bacteriuria, cystitis, and even pyelonephritis do not express P fimbriae (1).

A considerable body of evidence from *in vitro* and animal studies indicates a role for type 1 fimbriae in the virulence of *E. coli* in the urinary tract (6–12). Type 1 fimbriae, the most common adhesive organelles of *E. coli*, are the prototypical examples of adhesins containing lectins, their adhesive function being inhibited by D-mannose and its derivatives (3, 13–15). Despite relatively abundant evidence, significant controversy exists concerning the role of type 1 fimbriae as a virulence factor because up to 95% of all *E. coli* isolates, irrespective of origin, express type 1 fimbriae, and epidemiological studies do not show differential distributions of type 1 fimbriated *E. coli* between uropathogenic isolates and fecal isolates of healthy individuals (1, 2, 5, 16).

Type 1 fimbriae are encoded by the fim gene cluster (17) and are composed primarily of the structural subunit, FimA. Small amounts of the adhesin subunit, FimH (18), are found at intervals along the fimbrial shaft (19) and also at the tips (20). It was recently demonstrated that type 1 fimbriae exhibit a remarkable phenotypic variation not previously appreciated (21–23). Allelic variants of the fimH gene confer distinctly different receptor specificities not limited to oligomannose structures previously thought to be the primary receptor (22, 23). Adhesion of wild and recombinant strains to three model substrata revealed at least three phenotypic classes of FimH. The M phenotype mediates adhesion only to substrates rich in exposed mannose residues, such as yeast mannan (MN). The MF phenotype mediates adhesion not only to MN, but also to complex-type oligosaccharides, such as in human plasma fibronectin. The MFP phenotype mediates adhesion to MN and fibronectin and also to synthetic peptides completely devoid of saccharide moieties (22). This functional diversity was not considered in the previous epidemiological studies.

Further studies of the predominant M phenotype surprisingly revealed that adhesion to MN could vary by up to 10-fold or more among *E. coli* isolates, even though the morphologies of their fimbriae were indistinguishable (23). In fact, no effective substratum was found in this previous study for the low MN-adhesive FimH phenotype predominant among fecal strains. The potential relevance of the variations in the magnitude of MN adhesion to UTIs was highlighted by the finding that type 1 fimbriated *E. coli* isolates obtained from UTIs exhibited an average of a 3-fold greater ability to adhere to immobilized MN than type 1 fimbriated *E. coli* isolated from the feces of healthy

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\(^1\) The abbreviations used are: UTI, urinary tract infection; MN, yeast mannan; ManBSA, mannosylated bovine serum albumin; (Man)\(^3\)BSA, trimannosyl-BSA; αMM, methyl-α-D-mannopyranoside; BRB, bovine RNase B; PBS, phosphate-buffered saline.
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EXPERIMENTAL PROCEDURES

Reagents—Salivary mucin was purified from whole human saliva, as described previously (24). Monomannosylated BSA (ManBSA) was obtained from EY Laboratories, Inc. (San Mateo, CA). a1–3, a1–6-mannosetol-BSA (Man,BSA) was obtained from V-Labs, Inc. (Covington, LA). Human laminin was purchased from Life Technologies, Inc. Purified Tamm-Horsfall protein was generously provided by Dr. I. Ofek (Tel-Aviv University, Tel-Aviv, Israel). All other reagents were obtained from Sigma.

Bacterial Strains and Plasmids—Most of the recombinant strains utilized here were constructed using a Δfim K-12 derivative constructed by Blomfield et al. (25) and were described previously (23). Briefly, the fim gene cluster was deleted from E. coli MG1655 to create AAEC191A (25). AAEC191A was transformed with the recombinant plasmid pPKL114 to create strain KB18. Plasmid pPKL114 is a pH5R22 derivative containing the entire fim gene cluster from the E. coli K-12 strain, PC31, but with a translational stop-linker inserted into the unique Kpn site of the fimH gene. Strain KB18 cells express no fimbrae or very few numbers of long, nonadhesive fimbrae. For the studies reported here, strain KB18 was co-transformed with a series of isogenic pGB2-24-based plasmids. Plasmid pGB2-24 is a previously constructed pACYC184 derivative that is convenient for expression of fimH genes polymerase chain reaction-cloned from different E. coli (22). Recombinant strains created using these plasmids express large numbers of fully functional, type 1 fimbrae (22, 23). In most of the experiments, we employed a recombinant strain, KB91 (KB18(pGBB17)), expressing the fimH1–36 gene and an isogenic strain, KB54 (KB18(pGB6)), expressing the fimH2–131 gene. E. coli F-18 is a normal intestinal isolate (26), and E. coli CI129 is a UTI isolate (22, 23). Five other isogenic recombinant strains (KB21, KB23, KB59, KB92, and KB96) differed from KB91 and KB54 only in the allelic variant of the fimH gene that was present on the pGB2-24-based plasmid. The abilities of these recombinant strains to adhere to MN were variable and correspond to the wild strain phenotypes. The phenotypes and deduced amino acid sequences of each of these FimH subunits were described previously (23).

Another set of strains was based on a FimH2–131 derivative of E. coli F-18 (27; gift of Dr. P. Cohen). E. coli F-18 FimH was transformed with plasmid pGB6 (harboring fimH1–36) to create strain KBF109 or with plasmid pGB17 (harboring fimH2–131) to create strain KBF110.

Adhesion Assays—Assays of bacterial adhesion to glycoproteins bound to 96-well plates or to epithelial cells in 8-well tissue culture chamber slides (Nunc, Naperville, IL) were carried out as described previously (22, 23, 28). Briefly, glycoproteins were dissolved at 20 mg/ml in 0.02 M bicarbonate buffer, and 100-μl aliquots were incubated in microtiter wells for 1 h at 37°C. The wells were then washed three times with PBS and quenched with 0.1% BSA in PBS. Bacteria were added in 0.1% BSA in PBS and incubated for 40 min at 37°C without shaking to achieve saturation, and the wells were then washed with PBS. The number of bound bacteria was determined by a growth assay (28) or by using 3H-thymidine-labeled bacteria, as described previously (23). The density of bacteria used in all assays was 5 × 107 colony-forming units per 100 μl except for the equilibrium binding experiments in which 12 to 16 serial dilutions of bacteria covering the densities 2.3 × 108 to 8 × 105 colony-forming units per 100 μl were utilized. Equilibrium measurements and other comparative studies were performed in parallel. Adhesion to epithelial cells was determined as described previously, enumerating bound bacteria by light microscopic examination of stained samples (23).

Electron Microscopy—Suspensions of bacteria in PBS were adsorbed to Formvar-coated grids for 2 min, followed by staining on drops of 0.5% phosphotungstic acid (pH 4) for 2 min. After drying, bacteria were examined using a JEOL1200EX electron microscope.

Correlation of the abilities of seven recombinant strains to bind to MN with their abilities to adhere to J82 human bladder epithelial cells. Strain numbers are shown. Statistical analyses of the data are provided in the text.

Statistics—Correlation coefficient, r, was calculated using Crick Graph (Crickt Software, Philadelphia, PA), where applicable. The significance of r was determined according to Fisher.

RESULTS

Adhesion to MN Correlates Directly with Adhesion to Uroepithelial Cells—We found a highly significant, direct correlation between the magnitude of adhesion to MN and the level of adhesion to either J82 human bladder epithelial cells (Fig. 1; r = 0.97, p > 0.995) or A498 human kidney epithelial cells (r = 0.93, p > 0.995; data not presented in figure) using 7 isogenic recombinant strains expressing subunits encoded by fimH genes derived from various E. coli strains. The results suggest that the differences in the magnitude of adhesion among the FimH-expressing recombinants are not restricted to immobilized MN and thus may represent fundamental differences in the fine sugar specificity of the allelic variants of FimH. To examine this possibility, the adhesion of two strains, KB91 and KB54, was studied in more detail. Strain KB91 represents the low MN-adhesive phenotype typical of normal intestinal isolates, whereas strain KB54 represents the high MN-adhesive phenotype typical of UTI isolates (23).

Scatchard Plot Analyses of Adhesion to MN—The adhesion of strains KB91 and KB54 to MN-coated wells was analyzed by equilibrium measurements. Scatchard plot analyses showed that at saturation, KB54 could utilize a maximum of 22.5 × 106 combining sites per well with a Kd ~5.0 × 10−7 and 4.4 × 106 combining sites with a Kd ~6.1 × 10−8 (Fig. 2). The analyses also revealed that the FimH of KB91 mediated adhesion through two apparent combining sites. There were approximately 4.1 × 105 low affinity combining sites per well for KB91, with a Kd ~7.1 × 10−7. Adhesion to one type of site exhibited a relatively high affinity (Kd ~1.1 × 10−7), but the number of such sites was limited to approximately 1.0 × 106 per well.

Adhesion to a Spectrum of Glycoproteins—Detection of a relatively high affinity site for strain KB91 on the MN substra-

um prompted us to compare the patterns of the adhesion of strains KB91 and KB54 to a variety of immobilized glycoprotein substrates (Fig. 3). As expected, neither of these M phenotype strains bound to the glycoproteins exhibiting exclusively complex type N-linked glycans that have no terminally exposed mannosyl residues or O-linked glycans that have no mannose: human serum apotransferrin, human α-acid glycoprotein, and bovine milk casein. Strain KB54 adhered in large numbers to each of the other glycoproteins that are known to possess at least a
certain fraction of either hybrid or high mannose type oligosaccharide moieties, both of which have terminal mannose residues. In contrast, the adhesion of strain KB91 to these substrata varied dramatically. Adhesion to one group of glycoproteins (Tamm-Horsfall protein, human amylase, salivary mucin, intestinal mucin, and mouse IgA) exhibited a clear MN-like pattern, in that KB91 adhered at a much lower level than did KB54. Adhesion of KB91 to a second group of glycoproteins (porcine thyroglobulin, chicken egg albumin, human laminin, horseradish peroxidase, and mouse IgA) was increased, but still much below adhesion of KB54. Adhesion of strain KB91 to a third group of glycoproteins (bovine lactoferrin, human secretory IgA, and bovine RNase B) was roughly equal to that of KB54, in distinct contrast to the MN-like pattern.
strains to bind to ManBSA and their ability to bind to MN (Fig. 6A; \( r = 0.98, p > 0.995 \)). All of the strains adhered relatively well to the \((\text{Man})_3\)BSA substratum, and there was a strong positive correlation between their abilities to bind to \((\text{Man})_3\)BSA and to bRB (Fig. 6B; \( r = 0.77, p > 0.95 \)). There was no positive correlation between either the ability of the strains to bind to \((\text{Man})_3\)BSA and MN (\( r = 0.0 \)) or between the ability of strains to bind to ManBSA and bRB (\( r = -0.3 \)).

Interestingly, while the levels of adhesion of strain KB54 to ManBSA and \((\text{Man})_3\)BSA substrata were quantitatively similar, the \(\alpha\)MM IC\(_{50}\) for the adhesion to ManBSA was approximately 50-fold less than the \(\alpha\)MM IC\(_{50}\) for adhesion to \((\text{Man})_3\)BSA, similar to the differential inhibition observed for adhesion to MN and bRB (see Fig. 5). Binding of strain KB91 to \((\text{Man})_3\)BSA was approximately 2-fold less sensitive to inhibition by \(\alpha\)MM than was the adhesion of KB54. Thus, regarding the levels of both adhesion and sensitivity to \(\alpha\)MM inhibition, the reactions of \(E. \ coli\) with MN and ManBSA were similar, and the reactions of \(E. \ coli\) with bRB and \((\text{Man})_3\)BSA were similar. These observations are consistent with the foregoing results suggesting that high MN-adhesive subunits, but not low MN-adhesive subunits, are able to mediate adhesion effectively via individual mannose residues terminally exposed in high mannose-type or hybrid-type oligosaccharide structures. At the same time, all FimH subunits are capable of mediating strong adhesion via interaction with unsubstituted trimannosyl groups.

**Adhesion of Recombinant Strains Constructed in an F-18 Background to MN, bRB, ManBSA, and \((\text{Man})_3\)BSA**—Our observations in previous publications have indicated that the MS-adhesive phenotype of the two-plasmid recombinant strains used here corresponded to the MS-adhesive phenotype of wild strains and is dependent on the \(\text{fimH}_{\text{CI12}}\) and \(\text{fimH}_{\text{F-18}}\) genes expressed on plasmids pGB6 and pGB17 and is basically the same as that of the wild strains. Values are means ± S.E. (\( n = 3 \)).

To determine whether the MS-adhesive phenotype of the wild strains is also determined by the \(\text{fimH}\) allele, we tested the binding of a FimH\(_{\text{2}}\) derivative of \(E. \ coli\) F-18. This strain did not bind to any of the tested substrates (Fig. 7). However, when this strain was transformed with plasmids containing \(\text{fimH}_{\text{F-18}}\) or \(\text{fimH}_{\text{CI12}}\) genes, creating KBF110 and KBF109, respectively, the transformants adhered in the same pattern as did the corresponding wild strains (Fig. 7). These observations reinforce the concept that MS-adhesive phenotype is dependent primarily on the \(\text{fimH}\) allele and not the host background strain.

Electron microscopic examination demonstrated that the FimH\(^-\) derivative of F-18 expressed fewer numbers of fimbriae.

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**FIG. 5.** Inhibition of adhesion of strains KB54 (circles) and KB91 (squares) to MN (open symbols) and bRB (filled symbols) by \(\alpha\)MM. Data obtained were converted to percent inhibition using the number of bacteria binding in the absence of inhibitor as control. The dotted line indicates the IC\(_{50}\). The x axis is on a log scale, and concentrations of \(\alpha\)MM are given in mM.

**FIG. 6.** Correlation of the levels of adhesion of seven recombinant strains to ManBSA with their adhesion to MN (A) and correlation of the adhesion of the same strains to \((\text{Man})_3\)BSA with their adhesion to bRB (B). Strain numbers are shown. Analyses of the data are provided in the text.

**FIG. 7.** Adhesion of wild \(E. \ coli\) strains CI12-7 and F-18 (A) and the recombinant \(E. \ coli\) strains F-18 (FimH\(^-\)), KBF109, and KBF110 to bRB ( ), MN ( ), \((\text{Man})_3\)BSA ( ), and ManBSA ( ) (B). The patterns of adhesion of recombinant strains to these glycoproteins is dependent upon the \(\text{fimH}_{\text{CI12}}\) and \(\text{fimH}_{\text{F-18}}\) genes expressed on plasmids pGB6 and pGB17 and is basically the same as that of the wild strains. Values are means ± S.E. (\( n = 3 \)).
per cell, essentially the same as the K-12 derivative, KB18. The fimH transformants KBF109 and KBF110, however, expressed large numbers of fimbriae typical of type 1-fimbriated wild strains and with essentially identical morphology (Fig. 8).

Adhesion of Recombinant Strains to Uroepithelial Cells—To determine whether adhesion to human uroepithelial cells corresponds more closely to the monomannoside-type binding (MN- and ManBSA-like) or the trimannoside-type binding (bRB- and (Man)3BSA-like), a quantitative comparison was performed using all seven recombinant strains. There was a significant correlation ($r = 0.98, p > 0.995$) between the level of bacterial adhesion to J82 human bladder epithelial cells and the ability to bind to monomannosyl receptors (Fig. 9A). Results were similar when adhesion to A498 human kidney epithelial cells was tested ($r = 1.0, p > 0.995$). In contrast, there was no correlation ($r = 0.05–0.08$) between the adhesion of the recombinant strains to epithelial cells and their ability to bind to trimannosyl receptor structures (Fig. 9B).

**DISCUSSION**

Although data can be found in the literature that point toward the phenotypic diversity of type 1 fimbriae (e.g. Refs. 30–32), very little, if any, attention was previously given to this phenomenon until recently (21–23). In a previous publication, we reported that different alleles of the lectin-like subunit, FimH, mediate very different levels of adhesion of type 1-fimbriated wild strains and with essentially identical morphology (Fig. 8).

Based on the data presented here and discussed in more detail below, it can be proposed: 1) that allelic variants of the FimH lectin of *E. coli* type 1 fimbriae are not all alike in their ability to recognize terminal mannosyl structures and exhibit at least two distinct mechanisms of ligand-receptor interaction; 2) that all FimH subunits studied to date are capable of mediating adhesion via trimannosyl residues, but only certain variants are capable of mediating adhesion via monomannosyl residues; and 3) that the ability of the FimH lectins to interact with monomannosyl residues strongly correlates with their ability to mediate *E. coli* adhesion to uroepithelial cells. Whether these phenotypic differences result in differences in tissue tropism in a human or animal host remains to be determined.

The hypothesis that different receptor specificities are responsible for the apparent magnitude of adhesion to MN was prompted by equilibrium binding measurements of adhesion of strains KB91 and KB54 to MN. Scatchard plot analyses of bacterial adhesion data can provide important information regarding receptor specificity, giving an indication of both affinity and the heterogeneity of binding sites (33). Scatchard plot analyses indicated the possibility that strain KB91 reacts relatively weakly with the high affinity MN receptors recognized by strain KB54 and either does not react or reacts at undetectable levels with the low affinity MN sites of strain KB54. However, a number of high affinity binding sites for strain KB91 in MN were detected, indicating the ability of this FimH adhesin to interact strongly with certain receptor structures which were exposed poorly in immobilized MN. Indeed, while KB91 exhibited low levels of adhesion to MN, it adhered avidly to several glycoprotein substrata, with IgA, lactoferrin, and bRB being the most prominent of those tested thus far. The equilibrium analyses of adhesion to bRB indicated that the FimH lectins of both strains probably interact with the same structural element on the bRB-coated surface and with the same effectiveness. Thus, it is likely that separate mechanisms of ligand-receptor interactions are responsible for the differential adhesion of these two strains to MN and bRB.

Inhibition of ligand-receptor interactions by receptor analogs is an important adjunct to direct adhesion studies. The more
effective inhibition of bacterial adhesion to MN by soluble monomannosides than adhesion to bRB prompts us to suggest that the mechanism of adhesion to MN is primarily via interaction with single terminal mannose residues, whereas adhesion to bRB involves a more complex interaction with multiple mannose residues. Indeed, strain KB54 was able to adhere much better than strain KB91 to ManBSA, whereas both strains adhered well to (Man)₃BSA. Interestingly, although the N-linked carbohydrate moieties of both MN and bRB do provide terminal mannosyl residues, terminal mannotriose structures are abundant in the Man₃ and Man₄ oligomannose units which constitute almost 90% of bRB oligosaccharide units, but not in the mannotriose structures of *Saccharomyces cerevisiae* (34). Also, it is known that human IgAgs contain hybrid-type oligosaccharides which have terminal mannotriose structures (35), whereas ovalbumin contains a mixture of oligomannose, hybrid, and multiantennary complex N-linked glycans. Whether other oligomannose structures would provide increased or decreased levels of adhesion in comparison to the trimannose remains to be determined.

It has been proposed previously that the combining site of the type 1 fimbrial lectin is in the form of a complex, trisaccharide-sized pocket that has three adjacent subsites, each of which accommodates one residue of the trisaccharide (36). Many previous studies of the fine sugar specificity of type 1 fimbriae called attention to oligomannose structures as the primary, if not exclusive, receptors. However, the precise nature of the ligand-receptor interactions was not fully developed (14, 15). It is now reasonable to speculate that the strong binding of KB91 and KB54 FimH subunits to trimannosyl structures occurs via the interaction of the subsites of the combining pockets of both adhesins with multiple mannosyl residues. The ability of KB54 subunits to mediate adhesion to monomannosides could be due to the ability of a single subsite to react with sufficient affinity to accomplish adhesion. The requirement of the KB91 FimH for trimannose units could be Because none of the subsites has a structure that allows high affinity interaction with a single mannosyl residue. The hypothesis that FimH can mediate adhesion via binding to monomannoside residues is quite novel for type 1 fimbriae and could have significant physiological implications.

Neither MN nor bRB are likely to be important receptors for *E. coli* on host mucosal surfaces. However, their use as model substrata helped to identify two mechanisms of interaction of the FimH lectins which could help to dissect the mechanism of binding to physiologically relevant glycoproteins (e.g., salivary and intestinal mucins (37), IgA (35), lactoferrin (38), uroplakins (39), leukocyte integrins (40), etc.). The divergent mechanisms of interaction of FimH with terminal mannosyl residues described here imply that FimH subunits recognizing monomannoside residues, as represented by the KB54 FimH, should mediate a broader spectrum of bacterial adhesion than FimH subunits recognizing only trimannoside residues, as represented by the KB91 FimH. The stricter selectivity of the trimannose-specific FimH subunits may allow targeting of the strains to surfaces that are physiologically important for normal intestinal *E. coli* and may help to avoid or reduce the impact of nonspecific host defense barriers, such as lectino-phagocytosis (41). The reduced sensitivity of the trimannose-specific FimH subunits to the inhibition by soluble compounds containing exposed mannose could be another advantage of this phenotype. Thus, the trimannose-specific phenotype could provide more efficient adhesion for *E. coli* cells in an environment where mucosal surfaces are bathed with mannose-rich glycoproteins. On the other hand, strains bearing the monomannose-specific FimH subunits may have an increased chance to find a substratum containing an acceptable mannosylated compound. Such an expanded receptor specificity for the monomannose-specific FimH might provide a selective advantage for type 1-fimbriated *E. coli* in the colonization of certain ecological niches (42) and, for example, may be of great importance in the contribution these organelles make to the virulence of *E. coli* in the urinary tract (12). Although the exact structure of the oligosaccharides exposed on the uroepithelial surface is not yet defined (38), the strong correlation between the ability to bind to ManBSA and the ability to bind to uroepithelial cells among the FimH-expressing strains suggests that bacterial attachment is accomplished primarily via the monomannoside binding mechanism. Thus, the enhanced ability to bind to uroepithelial cells may explain why UTI isolates express predominantly fimbriae bearing monomannose-specific FimH.

It is not yet clear precisely how uropathogenic *E. coli* orchestrates the contributions of type 1 fimbriae and other virulence factors in the complex process that results in cystitis or pyelonephritis. The observations reported here strongly suggest that some phenotypic variants of FimH contribute much more to UTI than do others by increasing the ability of *E. coli* to adhere better to uroepithelial cells. It may be of interest in this regard to consider the recent observations reported by Connell et al. (12). Clonally related uropathogenic strains of *E. coli* O1:K1:H7 were tested for virulence in a mouse UTI model. The strains expressed type 1 and P fimbriae and were shown to be members of the same clone by serotyping and by multilocus enzyme electrophoresis. However, they were recovered in dramatically different numbers from kidneys and bladders after experimental UTI in mice. We have recently found that the more highly virulent strain recovered from kidneys and bladders in high numbers expresses monomannose-specific FimH, while the strain exhibiting relatively low virulence and recovered from kidneys and bladders in much lower numbers expresses trimannose-specific FimH.

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