The Use of Biochemical Markers, Serotype and Fimbriation in the Detection of *Escherichia coli* Clones

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Biochemical reactions, O and K serotypes and presence of P-fimbriae were analysed in 116 *Escherichia coli* strains isolated in blood cultures from patients with bacteraemia and in 99 faecal strains isolated from healthy individuals. By using biochemical typing, the strains could be grouped into six main clusters with similarity index <0.8 (Gower, 1971) and altogether 16 subclusters with similarity index 0.82–0.89. The most discriminating tests between the clusters were fermentation of D-tagatose, saccharose, salicin and sorbose. No single biochemical property could differentiate bacteraemic isolates from faecal strains, although strains isolated from blood were significantly more often found in certain subclusters, whereas other subclusters contained mainly control strains. Bacteraemic strains possessed P-fimbriae more often, especially strains isolated from patients with *E. coli* in the urine concomitantly with bacteraemia. Equally, no single reaction could separate P-fimbriated from non-P-fimbriated strains. D-Tagatose was fermented more often by the P-fimbriated strains; on the other hand, melibiose and lactose fermentation tests were less often positive. Certain O serotypes (O1, O4, O6, O7, O18 and O25) were more common among bacteraemic isolates than controls. K serotypes such as K1, K5 and K52 were also more frequent among blood isolates. We conclude that a combination of biochemical tests, fimbriation and serotyping might be used to identify potentially pathogenic clusters of *E. coli*.

INTRODUCTION

Escherichia coli is one of the major micro-organisms causing non-obstructive urinary tract infections in children (Winberg *et al.*, 1974) as well as in adults (Jacobson *et al.*, 1985). It is also one of the most common micro-organisms found in blood cultures (McGowan *et al.*, 1979). Bacteraemia caused by *E. coli* is generally associated with a high mortality rate (Weinstein *et al.*, 1983). When the portal of entry is the non-obstructed urinary tract, however, the mortality rate of *E. coli* bacteraemia is lower (Brauner *et al.*, 1985).

The identification of *E. coli* seldom causes problems in the clinical laboratory. Many studies have shown that *E. coli* is a species consisting of phenotypically variable strains (Orskov, 1984).

Abbreviations: PPA, P-fimbriae particle agglutination test; DAC, diagnostic ability coefficient.

Certain properties of *E. coli* strains are associated with specific virulence attributes e.g. certain O-serotypes are associated with urinary tract infection (O1, O2, O4, O6, O7, O8, O9, O11, O22, O25, O62 and O75) (Orskov, 1978), enteropathogenic strains (EPEC), enterotoxinogenic strains (ETEC) or enteroinvasive strains (EIEC) (Orskov, 1984). On the other hand, neonatal septicaemia and meningitis are often associated with capsular serotype K1 (McCracken *et al.*, 1974).

Adhesion of *E. coli* to uroepithelial cells is a virulence attribute of strains causing human urinary tract infections. Most *E. coli* strains causing acute pyelonephritis show mannose-resistant agglutination of human erythrocytes (Källénus & Möllby, 1979; Green & Thomas, 1981; Hagberg *et al.*, 1981). The adhesion of pyelonephritogenic *E. coli* is, in the majority of cases, mediated by fimbriae with specificity for glycosphingolipids corresponding to antigens of the P blood group system (Källénus *et al.*, 1981), accordingly named P-fimbriae. The minimal active receptor structure is the α -D-Galp-(1 $\rightarrow$ 4)- β -D-Galp disaccharide sequence (Källénus *et al.*, 1980). A high incidence of P-fimbriated *E. coli* is normally found in the urine from children (Källénus *et al.*, 1981) as well as from adults (Jacobson *et al.*, 1985) with the first attack of acute pyelonephritis.

Biotyping appears to be a useful method to identify isolates of *E. coli* (Gargan *et al.*, 1982). By examining a number of biochemical reactions, especially those which have around 50% positive reactions, a number of profiles are created which allow a precise identification of various strains. However, there seem to be few distinctive biochemical characters associated with virulence although enterotoxinogenic strains, for example, are associated with certain phenotypes (Kühn & Möllby, 1986). Colonization of the intestinal tract of infants with *E. coli* does not seem to occur at random, and one investigation indicated that certain phenotypes or clones have a greater tendency to establish themselves in the gastrointestinal tract (Kühn *et al.*, 1986).

In order to study the occurrence of O and K serotypes and P-fimbriae and to correlate them with biochemical characteristics we have by multivariate analysis investigated 116 *E. coli* strains isolated from blood cultures and compared them with *E. coli* strains isolated from faeces of healthy individuals.

METHODS

Patients. During a 30 month period (January 1, 1983 to June 30, 1985), 113866 patients were hospitalized at the Karolinska Hospital, Stockholm, Sweden. Blood cultures were taken from 3356 patients and a significant growth was observed in 542 of these. *E. coli* was found in 104 patients with a total of 116 bacteraemic episodes. The cases were retrospectively evaluated by review of hospital charts: 54 were women, median age 64 years, and 50 were men, median age 63 years (ranges 12–93 and 4–89 years, respectively). The controls used were 99 *E. coli* strains from faecal swabs obtained from 94 healthy individuals (37 women and 57 men) visiting a health care centre.

Microbiological investigation. Blood cultures were done using a biphasic medium (Bio-Test, Bio-Hospital, Sweden). The agar slant contained brain heart infusion agar (Difco) with *p*-aminobenzoic acid (PABA) and sodium polyanethole sulphonate (SPS). The liquid phase contained meat infusion broth (Oxoid) with penicillinase. About 5 ml blood was injected into each of two vials, one for aerobic and one for anaerobic culture. The blood cultures were subcultured after 2 d and inspected daily for growth for 7 d.

Biochemical testing. All Gram-negative bacteria from blood isolates were identified by the API 20E system and the accompanying code book. *E. coli* strains were further analysed with the API 50 CHE system. A conventional lactose test on CLED agar (Oxoid) was done on all strains.

Taxonomic study. The following tests were taken into account for the classification process: conventional lactose fermentation test, API 20 E (first 11 tests) and API 50 CHE tests except for lactose fermentation.

A similarity matrix was established using Gower's generalized coefficient (Gower, 1971), without taking into account negative matching. A dendrogram was then constructed using the UPGMA method (Sneath & Sokal, 1973). The clusters were determined empirically. The set of most efficient tests for differentiation of the clusters was determined using a method based on Descamps's Diagnostic Ability Coefficient INF (Descamps & Veron, 1981).

Test for P-fimbriation. Briefly, bacteria were grown overnight at 37 °C on colonization factor antigen (CFA) agar (Evans *et al.*, 1977) and subsequently tested for P-fimbriation using the P-fimbriae specific particle agglutination (PPA) test as described by Svenson *et al.* (1982). This test is based on the specific agglutination of particles to

which the minimal P-fimbriae receptor active carbohydrate structure, α -D-Galp-(1 $\rightarrow$ 4)- β -D-Galp, is attached (Källénus *et al.*, 1980).

Determination of O antigens. Serotyping was done in plastic microtitre plates according to Lidin-Janson *et al.* (1977). *E. coli* strains were tested for the presence of 68 antigens selected because of their occurrence among *E. coli* strains isolated from patients with urinary tract infection. Some strains (designated non-typable) could not be typed with the antisera available.

Determination of K antigen. This was done using agar plates containing antiserum, as described by Kaijser (1977); 23 different antisera were used. Some strains (designated non-typable) could not be typed with the antisera available.

Statistical methods. Fisher's exact two-tailed test and the Chi square test were used (Sokal & Rohlf, 1981).

RESULTS

Patients

A total of 116 *E. coli* strains were isolated from 104 patients. There was an equal distribution among men and women. The majority (96) of the patients had only one bacteraemic episode, whereas seven patients had two bacteraemic episodes during the observation time. Four of these reinfections occurred within 3 months, the others after 8, 23 and 27 months. One patient had three bacteraemic episodes at 2 and 3 month intervals. Three patients had two different *E. coli* strains in the same blood culture. Five more patients had mixed infections with other bacteria, two with other *Enterobacteriaceae* and the other three with *Streptococcus* spp. non-pyogenes.

Biochemical markers

The analysis of the biochemical markers is given in Fig. 1. A primary cutting at a similarity coefficient of 0.8 allowed us to define six main clusters, numbered from A to F on the Figure. Cluster C could be subdivided into three subclusters (C1, C2 and C3) at a similarity coefficient of 0.85 and C2 and C3 into two further clusters each (C2a, C2b and C3a, C3b respectively) at 0.88 similarity. Cluster D was subdivided into three clusters at 0.86 similarity and D1 into two clusters (D1a and D1b) at 0.89 similarity. Cluster E was divided into two subclusters (E1 and E2) at 0.87 similarity. Finally, cluster F could be subdivided into two clusters (F1 and F2) at 0.82 similarity, and F2 into two more subclusters (F2a and F2b) at 0.85 similarity. A triangle representation has been drawn to show the relations between these clusters (Fig. 1).

The tests were ranked by using the decreasing diagnostic ability coefficient (DAC) (Descamps & Veron, 1981). The overall most discriminating tests were D-tagatose (0.89), saccharose (0.75), salicin (0.70) and sorbose (0.69). Among these tests, a set was chosen so that each pair of clusters was separated by at least one test at a DAC greater than or equal to 0.75, except for cluster C2b for 5-oxogluconate (DAC = 0.66). Table 1 shows the percentage of positive reactions for the different clusters for the 11 tests selected. The percentages of positive reactions among bacteraemic and control strains were also calculated although no single test had a DAC high enough to permit discrimination.

Occurrence of P-fimbriated *E. coli*

Almost half the strains isolated in blood cultures were P-fimbriated (47%). Four strains could not be evaluated with the PPA test. Strains from bacteraemic patients showed a significantly higher incidence of P-fimbriation (53/112) than *E. coli* isolated from the faecal flora in healthy controls (10/98, one strain was non-typable, $P < 0.001$). Among the bacteraemic patients there was a higher incidence of P-fimbriated strains in patients who also had a positive urine culture (35/58, 60%), than in patients with negative urine culture or where no urine culture was taken (18/54, 33%, $P < 0.01$).

In cluster B all seven strains came from patients with bacteraemia. All but one were P-fimbriated and six of them belonged to the K1 serotype. Of three O16 serotypes in the material, all were found in cluster B. In cluster D3 all seven strains also originated from patients with bacteraemia and all were P-fimbriated.

No single biochemical test could distinguish between P-fimbriated and non-P-fimbriated strains. D-Tagatose was fermented in 46/51 (90%) of the P-fimbriated strains, but only in 37/55

Fig. 1. Dendrogram showing clustering of 225 strains of *E. coli* based on the results of 61 biochemical tests. Abbreviations: n, No. of isolates in a cluster; bact, number of isolates from blood culture; P⁺, number of P-fimbriated strains; O, K, predominating serotype in a cluster. When non-typable (N) strains are in a majority, but a typable serotype is of interest, this figure is given as well. * No dominating O-serotype.

Table 1. *Biochemical tests on strains in different clusters*

The values given are the percentage of positive reactions for 11 selected tests on isolates in the clusters shown in Fig. 1. Sixteen bacteraemic isolates and three control strains could not be grouped into any cluster and were therefore excluded from this Table.

		Cluster														Total		
		A	B	C				D			E		F					
				C1	C2	C3		D1	D2	D3	E1	E2	F1	F2				
				C2a	C2b	C3a	C3b	D1a	D1b					F2a	F2b			
Total no. of strains	...	9	7	5	25	20	14	6	27	24	4	7	3	16	5	17	7	196
Bacteraemic strains	...	2	7	4	18	12	8	1	17	8	0	7	3	3	5	3	3	100
Control strains	...	7	0	1	7	8	6	5	10	16	4	0	0	13	0	14	4	96
Adonitol		100	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
Sorbose		0	86	100	100	100	100	17	93	100	0	100	33	0	100	0	71	
Aesculin		67	43	100	100	45	100	100	19	0	25	0	100	100	0	6	0	
Salicin		67	0	100	100	0	100	100	0	4	0	0	33	81	0	6	0	
Sucrose		11	14	20	4	0	100	100	100	100	100	0	0	0	0	0	14	
Raffinose		33	0	0	8	0	86	100	96	100	75	57	100	13	0	0	0	
D-Tagatose		0	100	100	96	95	100	0	100	4	0	100	0	0	0	0	100	
5-Oxogluconate		22	86	40	80	85	57	100	67	58	0	100	33	25	60	18	0	
Lysine decarboxylase		56	100	100	92	80	86	67	85	96	100	0	100	94	20	100	100	
Ornithine decarboxylase		11	100	100	100	95	100	100	93	54	100	0	100	6	0	24	14	
Lactose		100	0	0	100	100	100	83	100	96	100	86	67	100	100	100	100	

(67%) of the non-P-fimbriated strains ($P < 0.01$). On the other hand, melibiose and lactose were less often fermented by the P-fimbriated strains (41/51, 80% and 39/51, 77%) than by the non-P-fimbriated strains (54/55, 98% and 53/55, 96%, $P < 0.01$ in both cases).

E. coli serotypes

The distribution of O and K antigen serotypes of the *E. coli* strains from bacteraemic patients is shown in Table 2. Ninety of the 116 (78%) *E. coli* strains isolated from patients with bacteraemia were O-typable. *E. coli* serotype O18 was most frequently found (16% of the typable isolates). *E. coli* serotype O4 was found in 12% of the isolates and serotypes, O1, O6, O7 and O25 occurred in 8% each. With the available K antisera 70/116 (60%) *E. coli* strains were typable. K1 was the most frequently found K serotype (35/70, 50%) of the K-typable strains. K5 was found in 17% and K52 in 7% of the typable bacteraemic *E. coli* isolates.

The serotypes of *E. coli* strains from bacteraemic patients were compared to those isolated from faeces in healthy controls. Among the control strains, 57% were O-non-typable and 62% K-non-typable with the available antisera. The main typable serotypes were O1, O2 and O75 with seven, six and four strains respectively in each group, and K1 and K5 with 22 and six strains in each group. Strains from patients with bacteraemia showed a significantly higher incidence (53/116) of certain O serotypes (O1, O4, O6, O7, O18 and O25) than did the strains from the controls (15/99, $P < 0.001$ Chi square test). *E. coli* from bacteraemic patients also had a significantly higher incidence (52/116) of certain K antigens (K1, K5 and K52) than in isolates from controls (28/99, $P < 0.01$, Chi square test).

The analysis of the clusters revealed a tendency to unequal distribution among the serotypes. Seven of eight strains in cluster B belonged to serotype K1, which is significantly higher than in the rest of the bacteraemic strains ($P < 0.002$, Fischer's exact test). The O-serotypes showed a similar tendency to unequal distribution. The numbers in the different clusters were, however, too small to permit any further conclusions.

DISCUSSION

Analysis of biochemical data indicated that certain clusters were associated with pathogenic properties. Statistical analysis of the numbers of bacterial and control strains among the various subclusters showed a highly significant deviation from random distribution ($P < 0.001$, Chi square test).

In cluster D3, where all seven isolates were from patients with bacteraemia, four strains belonged to the K1 serotype and the remaining strains were K-non-typable. Of four O7 serotypes in the whole material, all were found in cluster D3; the remaining strains in that cluster were O-non-typable. Two more clusters, namely E1 and F1, containing isolates from three and five patients respectively, also had strains only from patients with bacteraemia. However, in one cluster, D2, all four strains originated from healthy individuals. Cluster E2 with 16 strains had only three strains originating from patients with bacteraemia. The same relationship was found in cluster F2a, where 3/17 strains originated from bacteraemic patients.

The incidence of P-fimbriated *E. coli* isolated from patients with bacteraemia was 47%. In an earlier study (Brauner *et al.*, 1985) 63% of *E. coli* causing bacteraemia were P-fimbriated. In the present study, a high percentage, 47/112 (42%) (four strains non-typable with the available PPA test) of the patients had compromising diseases, such as malignancies; acute colitis, cholecystitis or pancreatitis; debilitating drug abuse or chronic corticosteroid treatment. This distribution among the patients was found in our earlier study and can probably explain the differences in P-fimbriation of the blood isolates. In both studies the highest incidence of P-fimbriated strains was found in bacteraemic patients who previously had been apparently healthy and in whom the portal of entry was the urinary tract.

K antigens generally associated with urinary tract infection (K1, K2, K3, K12, K13, K52, K53 and K54) (Kajser, 1973; Kajser *et al.*, 1977; Svanborg-Eden *et al.*, 1978) were found in 48/70 K-typable strains (69%). Of 90 O-groupable *E. coli* strains 58 belonged to O serotypes associated with urinary tract infection (Orskov, 1978). These *E. coli* strains revealed a

Table 2. *O* and *K* serotypes in *E. coli* strains from blood cultures

(a) *P*-fimbriated strains

Four *P*-fimbriae non-typable strains have been included.

K Antigenic type	O-Antigenic type																				5 23 42 59	Total O- typable strains	Total non-O- typable strains	Total
	1	2	4	6	7	8	10	11	15	16	17	18	22	25	75	83	101	119	120	22 83	19 23			
K1	4	1	-	-	1	-	-	-	-	3	-	1	-	1	-	-	-	-	-	-	-	11	6	17
K2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	1
K3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
K5	-	1	-	1	-	-	-	-	-	-	-	5	-	2	-	-	-	-	-	-	-	9	-	9
K7	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
K10	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
K12	-	-	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	-	2
K13	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	1
K16	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	1	-	1
K22	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
K24	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
K51	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
K52	-	-	3	-	-	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	-	5	-	5
K53	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
K95	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total, K- typable strains	4	2	5	2	1	-	-	-	2	3	-	7	-	3	-	-	-	-	-	-	-	29	7	36
Non-K- typable strains	-	1	3	3	3	-	-	1	-	-	-	2	-	-	2	-	-	-	-	2	-	17	-	17
Total	4	3	8	5	4	-	-	1	2	3	-	9	-	3	2	-	-	-	-	2	-	46	7	53

Table 2. *continued*(b) *Non-P-fimbriated strains*

	O-Antigenic type																							Total O- typable strains	Total non-O- typable strains	Total
	1	2	4	6	7	8	10	11	15	16	17	18	22	25	75	83	101	119	120	22	19	23	59			
K Antigenic type	1	2	4	6	7	8	10	11	15	16	17	18	22	25	75	83	101	119	120	83	19	23	59	23	42	59
K1	1	1	-	-	1	-	-	-	-	-	-	2	-	-	2	-	-	-	-	1	-	-	-	8	9	17
K2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
K3	-	-	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	-	2
K5	-	-	-	1	-	-	-	-	-	-	-	1	-	1	-	-	-	-	-	-	-	-	-	3	-	3
K7	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	1	-	1
K10	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	1
K12	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
K13	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	1	-	1
K16	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	1	-	-	-	-	1	1	2
K22	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	1	-	1
K24	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	1	-	1
K51	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	1
K52	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
K53	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	1
K95	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	1	-	1
Total, K- typable strains	2	1	2	2	1	-	-	1	-	-	1	3	1	2	3	1	-	-	1	1	-	-	-	22	10	32
Non-K- typable strains	-	-	1	-	2	1	1	-	2	-	1	2	1	2	-	-	1	1	-	-	1	2	2	18	9	27
Total	2	1	3	2	3	1	1	1	2	-	2	5	2	4	3	1	1	1	1	1	1	2	40	19	59	59
P-fimbriae non-typable strains	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	1
K1	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	1
K51	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	1
Non-K- typable strains	-	-	-	-	-	1	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	-	2
Total	1	1	-	-	-	1	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	4	-	4
Total, all strains	7	5	11	7	7	2	1	2	5	3	2	14	2	7	5	1	1	1	1	3	1	2	90	26	116	116

significantly higher incidence (32/55, three strains non-typable with the PPA test) of P-fimbriation than strains belonging to other serotypes (21/57, one strain non-typable with the PPA test, $P < 0.05$ Fisher's exact test). Orskov & Orskov (1975) showed that more than half of the O-groupable *E. coli* isolates from blood cultures were O2, O4, O6 or O75. Among *E. coli* strains isolated at the Karolinska Hospital only 31% belonged to any of these groups. This low incidence might be due to the large number of patients with compromising diseases which enable even *E. coli* strains with a normally low virulence to become pathogenic.

Certain clones of a limited number of *E. coli* strains (Väisänen-Rhen *et al.*, 1984) have been shown to be associated with pyelonephritis. These strains possess certain homogeneous characteristics such as P-fimbriae and special O and K antigens. Tullus *et al.* (1984) reported epidemic outbreaks of first-time pyelonephritis and bacteraemia caused by particular *E. coli* clones in children previously being cared for in a particular neonatal ward. Neonates and staff of this ward showed a high incidence of the same *E. coli* strain in their faecal flora, indicating nosocomial spread. Pyelonephritis and septicaemia induced by *E. coli* has also been reported from an infant nursery (Sweet & Wolinsky, 1964).

In this study, we had few indications of nosocomial spread of *E. coli* strains. Only two patients, both heavily compromised, and treated at the oncological department, had strains belonging to the same biochemical cluster.

Other methods based on characterization of outer membrane proteins and plasmid content have been used (Achtman *et al.*, 1983; Achtman & Pluschke, 1986) in differentiating *E. coli* strains. These methods, although effective, seem less suitable for screening large numbers of strains.

We conclude that *E. coli* strains causing bacteraemia were P-fimbriated and that a higher percentage belonged to certain O and K serotypes compared with the control strains. We also found that a few biochemical markers were associated with bacteraemia and P-fimbriation. A combination of biochemical markers, serotypes and fimbriation might therefore be helpful in the detection of potentially virulent *E. coli* clones. However, the clones found in this study had properties characteristic of our hospital. At other times and in other hospitals the patterns might vary.

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