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Clonal Diversity of *Escherichia coli* Colonizing Stools and Urinary Tracts of Young Girls

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Intestinal carriage of *Escherichia coli* in prepubertal girls without a history of urinary tract infection was examined by collecting weekly stools and periurethral and urine samples over 3 to 4 weeks of study. Dominant and minor clones were defined by grouping 28 *E. coli* isolates into clonal types. Multiple enteric clones of *E. coli*, which changed week to week, were found in the 13 girls during the study (median, 3 clones/girl; range, 1 to 16 clones/girl). Dominance of an enteric clone did not predict persistence in the stool. In only 10 (34%) of the 29 episodes in which a dominant clone present in one weekly sample could have been detected the following week did it persist as the dominant clone in the next weekly sample. In 5 (17%) of the 29 episodes, a dominant clone found in one weekly sample was classified as a minor clone the next week. Both dominant and minor clones were observed to colonize the urinary tract. However, when colonization of the periurethra or bladder urine occurred, it was brief and often did not reflect the dominant stool flora from the same week. In fact, in only 40% of episodes was a clone that was detected either on the periurethra or in the urine also recovered from the stool the same week. Our findings suggest that the intestinal flora of healthy girls is multiclonal with frequent fluctuations in composition.

Escherichia coli organisms infecting the urinary tract are thought to originate in the intestinal flora. There are two theories that attempt to explain infection of the urinary tract with *E. coli* derived from the intestine (12). The prevalence theory holds that the numerically dominant fecal strain is most likely to infect the urinary tract. The special-pathogenicity theory holds that a special subset of the intestinal microflora expressing specific virulence markers is most likely to infect the urinary tract. Virulence markers have been defined based on their higher frequency in *E. coli* isolates from patients with urinary tract infection (UTI) than among the dominant fecal strains of the UTI patient (5, 8, 13) or of healthy controls (6, 7, 18). Support for either theory relies on examination of the dominant strain in the stool at the time infection of the urinary tract occurs. Drawing conclusions from comparison of strains infecting the urinary tract to the dominant strain in stool is predicated on the notion that the dominant strain in an individual's stool is stable over time. The stability of dominant clones and the frequency of transfer of dominant clones to the urinary tract in healthy controls are therefore important background information for interpreting findings in patients with UTI.

Few studies have examined carriage of dominant clones in the intestines of healthy controls (3, 4, 16). In the present study, we examined the intestinal carriage of *E.coli* in healthy girls without the confounding variables of antibiotic pressure, estrogenization, or sexual activity. For this, stool, periurethral, and urine samples were obtained weekly from prepubertal females. Dominant and minor clones of *E. coli* were defined in each sample by grouping up to 28 *E. coli* isolates into clonal types by multilocus enzyme electrophoresis (MLEE). We examined whether dominance of an enteric clone was a stable attribute of *E. coli* in the enteric environment and whether the dominance of a clone predicted colonization of the periurethra and urine.

METHODS

Population and surveillance cultures. Seventeen 3- to 6-year-old toilet-trained girls with no history of UTI or antibiotic use within the past month were recruited from a rural community in Virginia and enrolled after informed consent from the female parent. Guidelines for human experimentation of the U.S. Department of Health and Human Services and the University of Virginia Human Investigation Committee were followed in the conduct of clinical research. Each child was visited at home once a week for three or four consecutive weeks. At each visit, a stool sample, a periurethral sample, and a urine sample were obtained. Thirteen girls completed the study from February 1994 through October 1994; four girls who were unable to provide three stool samples within the study period were excluded. To reduce the amount of contamination of the periurethral tissue with urine, the periurethral sample was obtained prior to the midstream urine sample at the time of the visit, which was early morning. At each visit, the periurethral sample was obtained without prior cleansing by separating the vaginal labia and circumferentially swabbing the periurethral mucosa one time with a single cotton-tipped swab (15). The swab was immediately placed in 2 ml of sterile saline, mixed, rolled against the side of the glass vial to remove liquid, and discarded. The second sample collected, the urine, was obtained by midstream technique in a sterile wide-mouth container with a screw lid (11). The stool specimen was collected by the mother in a sterile container; the mother then inserted a single cotton-tipped swab into the center of the stool (9), immediately placed the swab in 2 ml of sterile saline, mixed and rolled the swab against the side of the glass vial to remove liquid, and discarded the swab. If the stool specimen was not obtained on the day of the visit, a visit was made each following day until a specimen was obtained (range, 1 to 3 days). All specimens were immediately refrigerated; transported on ice to the Pediatric Microbiology Laboratory, University of Virginia; and plated within 10 h of collection.

For isolation of bacteria from the periurethral and stool specimens, 10 μl of eluate was spread onto 5% sheep blood agar plates and MacConkey agar plates, which were incubated at 37°C and examined after 24 h for the presence of colonies. The minimum density of bacteria detected by our method of sampling the periurethra and stool was estimated to be 10^4 organisms/swab (15). For bacterial isolation from urine specimens, 10^{-3} ml of urine was inoculated onto

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TABLE 1. Clonal types of *E. coli* detected by MLEE analysis of 28 isolates from each weekly stool culture from 13 girls

Patient group[a]	Patient no.	Clonal type(s)[b] present during Week:			
		1	2	3	4
A	1	**1**	**1**	**1**	
	2	**1**, 2	**1**, 2, 3	**1**	
	3	**1**, 2	**5**[c]	**5**	**5**[c]
B	4	**1**, 2, 3	**3**	**3**	
	5	**1**[c]	1, **2**[c]	**1**	
	6	**1**, 2[c]	**2**	**1**, 2	
	7	**1**	**1**[c]	1, **2**[c]	
	8	**2**, 12	1, 2, 4, 5, **6**, 7	1, 2, **11**	
	9	**1,** 4, 5, 6, 7	**1**	**7**	**7**
C	10	1, 3, 4, 5, **6**, 7, 8, 9	**1**	12, **13**, 14, 15	**1**, 10
	11	**1**, 2, 3, 4[c]	**2**, 3, 4	**8**, 11[c]	
	12	**1**, 2	3, 4, **5**, 6	7, 8, **9**, 10[c]	
	13	3, 4, 5, 6, 7[c]	8, **9**, 10, 11, 12[c]	12–18[c]	

[a] Groups: A, dominant clone present and dominant for 3 weeks; B, dominant clone present but does not remain dominant for 3 weeks; C, dominant clone changes each week or is not present.
[b] Dominant stool clone shown in boldface.
[c] < 28 isolates typed.

5% sheep blood and MacConkey agar plates. Organisms were identified by standard methods.

Bacterial isolates. Isolates of *E. coli* detected on the original plate from urine or the periurethral or stool swab were saved. For each stool specimen, 28 colonies (isolates) were randomly picked from all four quadrants of the original plate and stored in 50% glycerol in trypticase soy broth at −70°C. From the number of randomly selected colonies, we estimated a >90% chance of detecting a minor clone (i.e., a clone representing only 10% of the population of *E. coli*) from one stool culture based on the binomial formula $1 - (1 - p)^n$, where p is the frequency of the minor clone (i.e., $P = 0.10$) and n is the number of colonies picked. In some cases, fewer than 28 colonies were observed on a plate, so all suitable colonies were isolated. For each periurethral and urine specimen, 5 to 28 single colonies, when present, were selected from all four quadrants of the original plate and stored. The probability of including at least one isolate of the dominant clone in a random sample of colonies was calculated to be 99% for five colonies. This calculation is similar to previous work (10).

Enzyme polymorphisms. The degree of genetic relatedness among isolates was assessed by enzyme polymorphism determined by enzyme electrophoresis as described previously (1). Electromorphs (mobility variants) of each enzyme were equated with alleles at the corresponding chromosomal genetic locus. Distinctive combinations of alleles, marking strains with distinct multilocus genotypes, were designated as an electrophoretic type (ET) or clonal type. Isolates of the same clonal type were considered to be of the same naturally occurring clone or cell line. Because the primary focus of this study was the variation between patients, not all electromorphs were compared in side-by-side runs, so clones from different patients were not equated.

Definitions. A dominant ET was defined as a clone which represented > 50% of typed isolates in a sample. A minor ET was defined as a clone which represented <10% of typed isolates in a sample.

RESULTS

Carriage of clones in weekly stool samples. The 13 healthy girls had multiple *E. coli* clones detected in the three stool samples collected a week apart. A median of 2 clones were isolated per sample (range, 1 to 8 clones/sample); a median of 3 clones (range, 1 to 16 clones/girl) were found in the three samples from each girl (Table 1). The average duration of carriage for a stool clone was 1.6 weeks. Each girl on average gained 2.7 clones and lost 1.8 clones over the study period.

A dominant clone could be identified in 39 (93%) of 42 stool samples. Detection of a dominant clone in the stool one week did not reliably predict that the clone would be dominant the following week. In three girls (patients 1, 2, and 3), the dominant clone in the first sample persisted as the dominant clone in subsequent weeks. In six girls (patients 4, 5, 6, 7, 8, and 9), the dominant clone persisted but did not continue to be dominant during the study period. In four girls (patients 10, 11, 12, and 13), there were different dominant clones from week to week or there was no dominant clone detected. In only 10 (34%) of the 29 episodes in which a dominant clone present in one weekly sample could be detected the following week did it persist as the dominant clone in the next weekly stool sample. In 5 (17%) of the 29 episodes, a dominant clone found one week was classified as a minor clone the following week. In short, the clonal population of *E. coli* in stool is diverse and changes from week to week so that two-thirds of the time a dominant clone detected one week was not dominant the following week.

Colonization of the periurethra with stool clones. Nine of 13 girls had *E. coli* detected on their periurethras at least once (average frequency of periurethral colonization, 0.36/week/girl) (Table 2). Dominant clones were more likely than minor clones to spread to the periurethra. However, when a dominant clone was in the stool, it was also present on the periurethra during the same week less than 25% of the time. In 8 (21%) of 38 episodes when a dominant clone was present in the stool, the same clone was also detected on the periurethra during the same week. In 4 (6%) of 64 episodes when a minor clone was present in the stool, the same clone was also detected on the periurethra during the same week ($P = 0.03$; Fisher's exact test).

The spread of a dominant clone to the periurethra was not consistent. One week the dominant stool clone would colonize the periurethra, but the next week the clone was not found on the periurethra even though the clone remained dominant in the stool. Alternatively, a dominant clone would not be found on the periurethra one week, then the following week, when the same stool clone was no longer dominant, the clone was detected on the periurethra. Colonization of the periurethra

TABLE 2. Clonal types of *E. coli* detected in stools and periurethrae of nine girls and in stools and urine of eight girls from weekly cultures

Patient no.	Source	Week 1		Week 2		Week 3		Week 4	
		Clonal type(s)[a]	*E. coli* titer: (CFU/ml)	Clonal type(s)	*E. coli* titer (CFU/ml)	Clonal type(s)	*E. coli* titer (CFU/ml)	Clonal type(s)	*E. coli* titer (CFU/ml)
4	Urine	NP[b]		NP		NP			
	Periurethra	NP		3 (28)		NP			
	Stool	**1**, 2, 3		**3**		**3**			
5	Urine	NP		NP		NP			
	Periurethra	NP		NP		1 (28)			
	Stool	**1**		1, **2**		**1**			
2	Urine	NP		NP		NP			
	Periurethra	NP		1 (25), 3 (2)		NP			
	Stool	**1**, 2		**1**, 2, 3		**1**			
6	Urine	2 (28)	10^5	2 (28)	10^4	NP			
	Periurethra	2 (14)		2 (28)		2 (28)			
	Stool	**1**, 2		**2**		**1**, 2			
9	Urine	1 (28)	10^4	NP		1 (8) , 7 (20)	10^3	1 (8) , 7 (20) 1	10^3
	Periurethra	2 (2) , 3 (1)		1 (5) , 5 (2) , 7 (13)		NP		NP	
	Stool	**1**, 4, 5, 6, 7		**1**		**7**		**7**	
3	Urine	NP		5 (20) , 4 (4)	10^4	NP		NP	
	Periurethra	3 (3)		5 (3)		NP		NP	
	Stool	**1**, 2		**5**		**5**		**5**	
8	Urine	1 (4) , 2 (8) , 3 (10) , 4 (4), 5 (2)	10^5	NP		9 (18) , 10 (10)	10^4		
	Periurethra	1 (1) , 2 (14) , 7 (7) , 8 (4) , 9 (1) , 3 (1)		NP		NP			
	Stool	**2**, 12		1, 2, 4, 5, **6**, 7		1, 2, **11**			
10	Urine	1 (27) , 2 (1)	10^3	NP		18 (28)	10^4	NP	
	Periurethra	1 (1)		10 (7) , 11 (4) , 12 (1) , 13 (16)		16 (2), 17 (2)		NP	
	Stool	1, 3, 4, 5, **6**, 7, 8, 9		**1**		12, **13**, 14, 15		**1**, 10	
11	Urine	NP		NP		9 (1) , 10 (11)	10^3		
	Periurethra	NP		NP		NP			
	Stool	**1**, 2, 3, 4		**2**, 3, 4		**8**, 11			
12	Urine	1 (16) , 2 (2)	10^3	NP		NP			
	Periurethra	1 (1)		NP		NP			
	Stool	**1**, 2		3, 4, **5**, 6		7, 8, **9**, 10			
13	Urine	1 (19) , 2 (7)	10^5	NP		NP			
	Periurethra	NP		NP		NP			
	Stool	3, 4, 5, 6, 7		8, **9**, 10, 11, 12		12, 13, 14, 15, 16, 17, 18			

[a] Dominant clone shown in boldface. The numbers in parentheses are the numbers of isolates representing each clone.
[b] NP, *E. coli* not present.

with a stool clone was brief; only one girl (no. 6) carried the same clone on the periurethra for more than 1 week.

When a clone was detected on the periurethra, less than a third of the time it was the dominant clone in the stool during the same week. More commonly, a clone(s) on the periurethra was detected in the stool the week prior or the week following detection on the periurethra or not at all. Nine of the total 26 periurethral clones were never detected in the stool during the study. In summary, most *E. coli* clones from the enteric environment did not spread to the periurethra. When periurethral colonization occurred, it was brief (less than 2 weeks) and did not often reflect the dominant stool clone from the same week.

Colonization of the urine with stool clones. *E. coli* at a titer of $\geq 10^3$ CFU/ml was detected in 13 urine cultures from eight girls (average frequency of bacteriuria, 0.31/week/child) (Table 2). Twenty-eight isolates from each urine culture (when available) were characterized by MLEE for clonal type. In 4 of the 13 urine cultures, a single clone was identified; in 8 cultures, two clones were identified: and in 1 urine culture, five clones were identified. Two of the eight girls had the same clone(s)

isolated from their urine 2 weeks in a row (patients 6 and 9). All of the girls were asymptomatic and did not receive antibiotic treatment.

Dominant clones were no more likely to colonize the urine than minor clones. In 6 (16%) of 38 episodes when a dominant clone was present in the stool, the same clone was also detected in the urine during the same week. In 4 (6%) of 64 episodes when a minor clone was present in the stool, the same clone was also detected in the urine during the same week ($P = 0.16$; Fisher's exact test).

When a clone was detected in the urine, only 24% of the time was the dominant clone found in the stool the same week. More often, clones in the urine were found in the stool the week prior to or the week after detection in the urine or not at all. Nine of the total 21 clones detected in the urine were not recovered in the stool during the study. Dominance of the stool clone did not predict that colonization of the periurethra or urine would occur that week.

DISCUSSION

Studies comparing *E. coli* strains from urine in patients with UTI to *E. coli* strains in the stool have utilized the dominant stool strain to represent the enteric environment. Bettelheim et al. (2) serotyped strains of *E. coli* obtained from different sites in nine stool samples and found that a single serotype constituted more than half the colonies isolated from eight stools. In one stool, there was no dominant serotype detected, and in all samples, multiple serotypes in addition to the dominant one were distinguished. They concluded that multiple colonies from a single stool sample may need to be typed in order to accurately assess the number of *E. coli* serotypes present in the stool or the serotype responsible for extraintestinal infections. Lidin-Janson et al. (10) serotyped 10 randomly selected colonies in one fecal sample from each of 52 schoolgirls. In each specimen they found one serotype that was present in a greater number of colonies than any other, and that strain was defined as dominant. These authors calculated that, in studies of fecal flora, a random sample of three *E. coli* colonies would have a 97% chance of including one isolate of the dominant clone. However, nondominant or minor strains would be missed. In our study, we wished to identify the diversity of clones carried in the stools of healthy prepubertal girls without a history of UTI. For this, we calculated that a random sample of 28 colonies would have a greater than 90% chance of including one isolate of a nondominant or minor clone. We confirmed that a dominant strain could be detected in 93% of stool samples from healthy girls. The problem was that the dominant clone changed from week to week.

The source of *E. coli* on the periurethra and in the urine is thought to be the gastrointestinal tract (17). Our work demonstrated that dominance of one clone may not be a stable attribute of *E. coli* in the enteric environment. An *E. coli* clone(s) detected on the periurethra or in the urine did not reflect the dominant stool clone from the same sampling period. Our study is consistent with the findings of Richards and Cooke (14), who examined women with a history of UTI and serotyped five colonies of *E. coli* from each periurethral culture, one colony from each urine culture, and five colonies from each stool culture. Twelve infections occurred in five women with a history of UTI. In 11 of the 12 episodes of UTI, the urinary strain of *E. coli* was found on the periurethra during the same sampling period. However, only half the time (6 of 12) was the urinary strain found in the stool. They postulated that the infecting strain was in the feces for such a short time before the development of UTI that it was not detected.

Studies have compared the frequency of a virulence factor in urinary strains from UTI to that of one dominant fecal strain from controls. For example, Johanson et al. (6) compared the frequency of *E. coli* with P adhesin in the urinary strains from children with UTI to that of the dominant fecal strain from healthy controls. P adhesin strains were more commonly found in the urinary strains from children with UTI than in the dominant fecal strains from controls. Plos et al. (13) examined the last three colonies from each of three fecal samples obtained from children with UTI and the last colony from one fecal sample obtained from controls. Children with at least one P-fimbriated *E. coli* strain in their fecal flora were defined as "carriers." More children with UTI than controls were carriers. Plos concluded that children who develop UTI have an increased tendency to carry P-fimbriated *E. coli* in their stools. This type of comparison may be problematic, since the dominant fecal strain in the control population changes from week to week. An interesting question is whether the frequency of P-fimbriated *E. coli* strains would increase if minor fecal strains were examined in control populations.

The distribution of virulence factors in the enteric environment might be more accurately depicted if dominant and minor strains from a single stool sample were examined. Yamamoto et al. (20) examined a total of 100 *E. coli* colonies from fecal and urine samples from 9 women with acute cystitis and 30 colonies from each fecal sample from each of 30 healthy women without UTI. In contrast to previous work, *E. coli* stool strains other than the strain infecting the urinary tract carried virulence factors in patients with UTI. Furthermore, half of the 30 women with no history of UTI had *E. coli* strains with one or more virulence factors in their stools. The authors concluded that the enteric environment is not homogeneous even among dominant stool strains and that examination of minor strains in the control population increased the ability to detect virulence characteristics in the intestinal environment. Conclusions drawn regarding uropathogenic *E. coli* isolates and their origin in the intestinal flora may be dependent on the number of isolates examined and the time when the samples from the stool and urinary tract were obtained. Studies examining the frequency of virulence factors in a dominant clone at one time are viewing only a small piece of the puzzle. Inclusion of minor clones when characterizing the attributes of intestinal *E. coli* may contribute to our knowledge of infection of the urinary tract. Whether minor stool clones persist in the large intestine (19) or express P adhesin in patients with UTI remains to be determined. Our findings with healthy girls suggest that the urogenital tract is exposed to a changing variety of *E. coli* clones because the host's intestinal flora is multiclonal with frequent fluctuations in composition (3).

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