Published in final edited form as:

Urology. 2013 March ; 81(3): 548–555. doi:10.1016/j.urology.2012.10.056.

Prevalence of ST131 Among Fluoroquinolone-resistant *Escherichia coli* Obtained From Rectal Swabs Before Transrectal Prostate Biopsy

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Abstract

OBJECTIVE—To identify the prevalence and characteristics of fluoroquinolone-resistant (FQ-R) *Escherichia coli* ST131 isolates in men undergoing ultrasound-guided transrectal prostate biopsy (TPB).

MATERIALS AND METHODS—Twenty-seven FQ-R *E coli* isolates from rectal swabs from 136 men undergoing TPB at 3 institutions in southern California (January 2009 to March 2010), with a focus on repeat biopsy patients, were assessed for *E coli* phylogenetic group, sequence type ST131 status, extended virulence genotype, pulsed-field gel electrophoresis profile, and antimicrobial susceptibility profile.

RESULTS—ST131 accounted for 70% of the 27 FQ-R pre-TPB *E coli* rectal isolates, including 82% of those from non-Asians vs 20% from Asians ($P = .017$). ST131 was associated negatively with prebiopsy enemas and positively with previous TPB. Compared with non-ST131 isolates, the ST131 isolates had a significantly higher prevalence of 4 virulence genes (*sat*, *usp*, *ompT*, and *malX*), distinctive virulence profiles, and numerically higher virulence scores (median, 12 vs 8), but similar antimicrobial resistance scores. Most rectal ST131 isolates exhibited pulsed-field gel electrophoresis profiles typical of clinical ST131 isolates.

CONCLUSION—In our locale, the epidemic multidrug-resistant ST131 clonal group accounts for 70% of FQ-R rectal *E coli* isolates among men undergoing TPB. Such ST131 isolates have distinctive virulence profiles, are extensively antimicrobial-resistant, and are negatively associated with Asian race. Further investigation is needed regarding risk factors for and clinical consequences of colonization with such strains among men undergoing TPB.

Prostate cancer is one of the most common cancers in men, with an estimated 240,890 new cases in the United States in 2011, most of which are diagnosed by ultrasound-guided transrectal prostate biopsy (TPB).¹ TPB has traditionally had a low morbidity profile; therefore, physicians have tolerated the associated 20% to 30% cancer detection rate.² Indeed, TPB has become even more common since the incorporation of repeat biopsy into active surveillance protocols and will likely increase.³

The American Urological Association Best Practice Statement regarding antibacterial prophylaxis recommends a fluoroquinolone before TPB.⁴ Unfortunately, the incidence of postbiopsy infection has increased markedly during the past decade, which has been attributed to fluoroquinolone-resistance (FQ-R).^{5–8} Paralleling this overall increase in infectious complications is a worrisome increased rate of sepsis.⁹ Several recent studies have identified patient-related risk factors for post-TPB infection, including previous fluoroquinolone exposure and travel to areas of higher endemic FQ-R.^{10–12} Until recently, little has been known regarding the causative bacteria in this population other than their propensity to be FQ-R.

Most post-TPB infections are caused by *Escherichia coli*, with the responsible strains most likely originating from the patient's rectum at the time of the biopsy.¹³ Of concern in this context is the recently described pandemic of a particular emergent *E coli* clonal group, sequence type ST131 (serotype O25b:H4), most members of which are FQ-R; some also express extended-spectrum β -lactamases (ESBLs).¹⁴

ST131 was recently implicated as the major culprit in post-TPB bloodstream infections in New Zealand (January 2006 to December 2010), which necessitated intensive care unit admission for 25% of patients.¹⁵ Previous studies of bacteria isolated from the intestinal tract before TPB by various techniques of rectal culture have identified a 10% to 20% prevalence of intestinal colonization with FQ-R *E coli*.^{13,16} Here, we sought to characterize recent FQ-R rectal *E coli* isolates from men about to undergo TPB for phylogenetic background, ST131 status, virulence genotypes, and coresistance profiles, and to identify epidemiologic and bacteriologic correlates of *E coli* ST131.

MATERIALS AND METHODS

Subjects

The present study population of men with pre-TPB FQ-R rectal *E coli* was identified during a previous screening study that assessed the prevalence of FQ-R *E coli* among pre-TPB patients.¹⁶ The clinical protocol, inclusion and exclusion criteria, and patient characteristics were reported elsewhere.¹⁶ In brief, after Institutional Review Board approval at each institution and patient informed consent, rectal swabs were obtained from 136 men scheduled to undergo a TPB at 1 of 3 institutions (Long Beach Veterans Affairs Medical Center, Southern California Kaiser Permanente, and University of California Irvine Medical Center) during a 15-month period (January 2009 to March 2010). Patients were enrolled targeting (approximately 2:1) repeat biopsy patients and a smaller number of controls who had not had previous biopsies. All patients received ciprofloxacin as their prophylaxis; however, physicians used their typical antibiotic regimen. Some patients received a 1-day

regimen and others a 3-day regimen. Patients were only enrolled once, and the number of biopsies was obtained during the medical history (Table 1).

Selective cultures using ciprofloxacin-supplemented broth and agar plates (as described below) identified 29 men as carrying FQ-R rectal *E coli*. Of these, 27 FQ-R *E coli* isolates were available for molecular analysis and constituted the present study population. Patient demographics were obtained by medical record review and patient interview.

Detection of FQ-R *E coli*

As described elsewhere, pre-TPB rectal swabs were placed directly into 5 mL of brain–heart infusion broth containing 10 mg/mL ciprofloxacin, incubated overnight, and then subcultured onto MacConkey agar containing 10 µg/mL ciprofloxacin.¹⁶ One representative of the dominant colony morphotype was selected per plate and was characterized on the VITEK 1 or VITEK 2 instrument (bioMerieux, Durham, NC), using Gram-negative (GN) identification cards and GNS-140 and GN-30 susceptibility cards, respectively. For data analysis, resistant isolates were those intermediate or resistant according to Clinical and Laboratory Standards Institute interpretative criteria.¹⁷ The resistance score was calculated by the number of antimicrobial agents (including ciprofloxacin) to which an isolate was resistant.

Molecular Typing

FQ-R *E coli* isolates were assessed for major *E coli* phylogenetic group (A, B1, B2, D) and extended virulence genotype for 50 genes associated with extraintestinal pathogenic *E coli* by using established polymerase chain reaction (PCR)–based assays.¹⁸ For group B2 isolates, ST131 status was defined by PCR-based detection of ST131-specific single-nucleotide polymorphisms in housekeeping genes *gyrB* and *mdh*. The virulence score was the number of virulence genes detected. Isolates were defined as extraintestinal pathogenic *E coli* (ExPEC) if they contained 2 of the following: *papAH* or *papC* (counted as 1; P fimbriae), or both, *sfa/foc* (S and F1C fimbriae), *afa/dra* (Dr-binding adhesins), *iutA* (aerobactin system), and *kpsM II* (group 2 capsules).¹⁹

Pulsed-field Gel Electrophoresis Analysis

Isolates underwent *XbaI* pulsed-field gel electrophoresis analysis (PFGE) analysis according to a standardized protocol.²⁰ Using BioNumerics (Applied Maths, Austin, TX), the PFGE profiles were compared with more than 1000 previously encountered ST131 and non-ST131-associated PFGE profiles within a private library (J.R.J), using a Dice similarity value of 94% relative to index strains to classify profiles into discrete pulsotypes.²⁰

Statistical Methods

Between-group comparisons were analyzed using the Student *t* test or Fisher exact test. Paired comparisons involving the prevalence of different traits within the same population were tested using the McNemar test. The criterion for statistical significance was $P < .05$ for unadjusted comparisons. Dendrograms based on virulence genotypes, antimicrobial susceptibility profiles, and PFGE profiles were inferred according to the unweighted pair group method within Bionumerics.

RESULTS

Patient Characteristics and ST131 Status

Characteristics of the 27 men whose FQ-R pre-TPB rectal *E coli* isolates were available for analysis are reported in relation to ST131 colonization status in Table 1. The number of FQ-R isolates per subject per institution (% of present study population) was 13 (48%) for Long Beach Veterans Affairs Medical Center, 5 (19%) for Southern California Kaiser Permanente, and 9 (33%) for University of California Irvine Medical Center. There was no difference in whether a 1-day vs 3-day regimen was chosen before biopsy ($P = .75$). In the initial screening population of 136 patients, 76% had undergone one or more prior TPBs, whereas in the present study, 86% (23 of 27) represented repeat biopsy patients (Table 1).

Phylogenetic group B2 predominated (70%), followed by group D (22%), then groups A and B1 (4% each). According to PCR-based detection of ST131-specific single nucleotide polymorphisms in *mdh* and *gyrB*, the 19 group B2 isolates represented ST131. ST131 thus accounted for 70% of the FQ-R *E coli* isolates overall and was significantly more prevalent than non-ST131 FQ-R *E coli* ($P = .008$ by McNemar test).

Race and ethnicity differed significantly in relation to colonization status with ST131 vs non-ST131 FQ-R *E coli*. That is, whites were comparatively under-represented and Asians over-represented among patients with non-ST131 FQ-R *E coli* (Table 1), with Asian race being negatively associated with ST131 ($P = .02$).

ST131 colonization was associated positively with repeat prostate biopsy and negatively with prebiopsy sodium phosphate enema use ($P = .032$ for both comparisons; Table 1). In addition, ST131 exhibited a borderline significant trend toward an association with larger prostate size (mean, 57 vs 41 cm³, $P = .09$). In contrast, age, body mass index, prostate-specific antigen level, age-adjusted Charlson comorbidity index, and number of prebiopsy ciprofloxacin doses did not differ significantly between the ST131 and non-ST131-colonized subjects (Table 1).

Virulence Genotypes

Among the 27 FQ-R *E coli* study isolates, 6 of the 50 virulence genes sought exhibited >80% prevalence, including (overall prevalence; definition) *fimH* (100%; type 1 fimbriae), *fyuA* (93%; yersiniabactin system), *iutA* (93%; aerobactin system), *iha* (85%; adhesion-siderophore), *sat* (82%; secreted autotransporter toxin), and *ompT* (82%; outer membrane protease). Four virulence genes differed significantly in prevalence between ST131 and non-ST131 isolates, all being more prevalent among ST131 isolates. These included *sat* ($P = .007$), *usp* (uropathogenic-specific protein; $P < .001$), *ompT* ($P < .001$), and *malX* (pathogenicity island-associated marker) ($P < .001$; Table 1). As a group, the ST131 isolates had numerically higher aggregate virulence scores (median score, 12; range, 9–15) than the non-ST131 isolates (median score 8; range, 4–14; $P > .10$) and were slightly more likely to fulfill the molecular criteria for ExPEC; that is, 13 of 19 ST131 (68%) vs 5 of 8 non-ST131 (63%; Table 1). A similarity dendrogram showed the aggregate virulence gene profiles of the 19 ST131 isolates were much more homogeneous than and highly distinct from those of the 8 non-ST131 isolates, with minimal overlap between the 2 groups (Fig. 1).

Comparisons of host characteristics with virulence factors showed that isolates from Asian men were negatively associated with *iha* ($P = .02$), *afa/draBC* ($P = .046$), *sat* ($P = .002$), and *ompT* ($P = .04$). In contrast, ExPEC status was not significantly associated with any host characteristic.

Antimicrobial Resistance

Antimicrobial resistance was quite extensive, with 25 of 27 FQ-R isolates (93%) exhibiting coresistance to at least 1 non-FQ agent; likewise, 9 of the 12 non-FQ antimicrobials encountered resistance in 1 isolate each (Table 1). The median aggregate resistance score was 5 (range, 1–10). The only statistically significant coresistance prevalence differences according to ST131 status involved cefazolin resistance (12% for ST131 vs 50% for non-ST131; $P = .04$) and ESBL phenotype (5% for ST131 vs 50% for non-ST131; $P = .017$). Accordingly, aggregate resistance scores were slightly lower among ST131 isolates (median, 4; range, 1–8) than non-ST131 isolates (median, 6; range, 1–10; $P = 0.12$ by Mann-Whitney U test). A dendrogram based on aggregate resistance profiles showed the ST131 and non-ST131 FQ-R isolates were extensively intermingled, without discernable by-group clustering (Fig. 2).

Pulsed-field Gel Electrophoresis

PFGE analysis of *kbaI* resolved 15 distinct pulsotypes among the 27 FQ-R *E coli* isolates (Fig. 3). The 4 pulsotypes with multiple isolates each, comprising pulsotypes 968 ($n = 7$ isolates), 800 ($n = 5$), 942 ($n = 2$), and 1260 ($n = 2$), together accounted for 16 isolates. Three of the 4 multiple-isolate pulsotypes, including the 2 most frequent (pulsotypes 968 and 800), were ST131-associated, accounting collectively for 74% of the present 19 ST131 isolates. In the PFGE dendrogram, the ST131 isolates clustered together at approximately the 67% similarity level, well separated from the non-ST131 isolates, which were more distantly related to one another (Fig. 3).

COMMENT

The rectal reservoir is probably the “staging ground” for most urinary tract infections, including those that occur after TPB, in which context the biopsy needle provides rectal microorganisms with direct access to the urinary tract. Unfortunately, intestinal FQ-R *E coli* have been detected in the pre-TPB population at rates of 10% to 30%, which predictably would predispose TPB recipients to FQ-R *E coli* infections.^{6,13,16,21} Indeed, despite fluoroquinolone prophylaxis, the overall post-TPB infection rate is now approximately 6%, with most episodes caused by FQ-R *E coli*.^{5–7}

We previously identified a 21% prevalence of FQ-R rectal *E coli* in men about to undergo TBP at 1 of 4 institutions in our locale.¹⁶ In the current study we determined that 70% of these FQ-R isolates represent *E coli* ST131, a clonal group documented to be a major cause of antimicrobial-resistant urinary tract infections and bacteremia across the globe.²² Importantly, a study in New Zealand (enrolled from January 2006 to December 2010) found ST131 *E coli* was the cause of 41% (18 of 44) of post-TPB bloodstream infections, a significantly larger proportion compared with a random sampling of non-TPB *E coli*

bloodstream infections (16% [6 of 45]; $P = .004$).¹⁵ This is consistent with introduction of intestinal FQ-R ST131 isolates from the rectal reservoir into the prostate gland during TPB, leading to bacteremia in a subset of patients.

The abundance of ST131 in our study aligns with previous studies implicating ST131 as an emerging, disseminated, multidrug-resistant pathogen.^{14,23} However, our findings show a considerably larger proportion of ST131 compared with a study of 332 healthy volunteers from Paris, France, (enrolled in 2006) that showed only 7% (4 of 51) of FQ-R *E coli* were ST131.²⁴

Among the multiple demographic characteristics studied, we found a statistically significant association of ethnic and racial background with bacterial group, in that ST131 isolates were significantly less likely to be from Asians than were non-ST131 isolates (5% vs 81%; $P = .02$). Corresponding with this, FQ-R *E coli* isolates from Asians had a significantly lower prevalence of 5 ST131-associated virulence genes compared with FQ-R *E coli* from subjects of other ethnic and racial backgrounds.

These associations with Asian race are of unclear biological validity and clinical significance and may represent chance findings. However, previous studies have identified associations of race and ethnicity with resistant organisms generally and also with specific ExPEC clonal groups.^{23,25} Notably, a 2011 study using the Surveillance, Epidemiology and End Results-Medicare data of >17,000 men compared with noninfected controls showed the highest odds of post-TPB infection were associated with nonwhite race (odds ratio, 2.13; 95% confidence interval, 1.28–3.57; $P = .004$).⁷ More studies are needed regarding intestinal carriage of ST131 or other antimicrobial-resistant *E coli* and its clinical correlates in relation to race and ethnicity, country of origin, and international travel.

Another group of interest were men undergoing repeat TPBs. Previous studies have shown that such patients are at higher risk for infectious complications.¹¹ Here, we found that ST131-colonized patients were significantly more likely to have had a prior TPB (94%) than were patients colonized with non-ST131 FQ-R *E coli* (63%; Table 1). This conceivably could result from more extensive prior fluoroquinolone use or health care contact, or both, among the repeat biopsy patients, if such exposures predispose to rectal colonization with ST131. In addition, our study was biased because we had enrolled a considerably higher number of repeat biopsy patients (2:1 ratio) to capture what was thought to be the high-risk group.

Analysis of prebiopsy interventions showed ST131 was less common among patients with FQ-R *E coli* who self-administered a prebiopsy sodium phosphate enema ($P = .032$). This may recommend performance of such an enema before TPB to decrease colonization with ST131, although a prospective, randomized trial would be needed to test this hypothesis. Notably, however, enema use was not associated with a reduced risk of having FQ-R *E coli* per se, only a shift of FQ-R *E coli* from ST131 to non-ST131 (Table 1).¹⁶ In contrast, ST131 status was not significantly associated with number of prebiopsy ciprofloxacin doses.

The pre-TPB ST131 rectal isolates exhibited typical ST131-associated virulence profiles, as observed among ST131 clinical isolates.¹⁸ Indeed, the number of virulence factors among

the ST131 isolates on average was approximately 50% greater than among the non-ST131 FQ-R isolates (median, 12 vs 8), with small numbers precluding statistical significance. Some of these ST131-associated virulence factors have been significantly associated with invasive disease in previous comparisons of fecal isolates vs blood, prostatitis, or pyelonephritis isolates.^{23,26–28} For example, Sannes et al²⁸ identified *ompT* as an independent predictor of *E coli* bacteremia among veterans, and 86% of our ST131 study isolates were *ompT*-positive, a significantly greater proportion than of the non-ST131 FQ-R isolates (14%). A study of men with febrile urinary tract infection compared with noninfected men enrolled from 1993 to 1996 similarly found that *iha*, *fyuA*, and *ompT* were associated with clinical infection; these traits were also common in our FQ-R *E coli* population (85%, 93%, and 82%, respectively).²⁹ In addition, 70% of our FQ-R *E coli* fulfilled molecular criteria for ExPEC, implying an enhanced ability to cause extraintestinal infections.¹⁹ Therefore, our prebiopsy study subjects had virulent-appearing FQ-R *E coli* (mostly ST131; also non-ST131) in the rectal reservoir, with the potential to cause infection if introduced iatrogenically into the prostate.

The present ST131 and non-ST131 FQ-R rectal isolates were extensively resistant to antimicrobials overall, with only minor between-group variation. Paradoxically, despite ST131 being known for ESBL production (particularly of the CTX-M-15 enzyme), the ESBL phenotype in our FQ-R *E coli* population was actually more common among non-ST131 isolates.³⁰ The overall prevalence of the ESBL phenotype was 19%, which is concerning because combined resistance to FQs and extended-spectrum cephalosporins severely limits treatment options.

According to PFGE analysis, the 2 main pulsotypes in the present population (types 968 and 800) were ST131-associated. These 2 pulsotypes, which accounted for 44% of study isolates overall and for 74% of ST131 isolates, were also the leading pulsotypes in a recent global survey of historical and current ST131 isolates, predominantly human clinical isolates.²⁰ Thus, the present rectal surveillance ST131 isolates represent largely the same prominent globally distributed ST131 clones that cause most ST131-associated human infections. This further supports the likely pathogenic potential of these isolates in men undergoing TPB.

Study limitations include the small population size, the single geographic locale (albeit with 3 participating institutions), and that only colonization, not clinical infection, was studied. In addition, all study isolates were FQ-R, precluding comparisons with FQ-susceptible isolates. Furthermore, whether the selective culturing protocol may have selected for or against ST131 compared with other FQ-R *E coli* is unknown. Finally, the use of multiple comparisons risked false detection of significant associations by chance alone.

The major study strength is the extensive molecular evaluation of FQ-R *E coli* in the pre-TPB population, including identification of virulence genes, phylogenetic group, ST131 status, and PFGE profiles. Future studies should include larger populations from multiple institutions in different locales, with and without post-TRB infections.

CONCLUSIONS

Our study found 70% of 27 FQ-R fecal *E coli* isolates from men undergoing TPB in our locale represented the epidemic multidrug-resistant ST131 clonal group. Compared with non-ST131 isolates, the ST131 isolates exhibited highly distinctive aggregate virulence profiles, a significantly higher prevalence of 4 specific virulence genes (*sat*, *usp*, *ompT*, and *malX*), and a lower prevalence of cefazolin resistance and ESBL phenotype. ST131 was associated negatively with Asians and prebiopsy sodium phosphate enemas and positively with repeat TPB. These findings identify a substantial rectal reservoir of *E coli* ST131 among men undergoing TPB. They thus indicate a need for additional studies to better define risk factors for and clinical consequences of colonization with FQ-R *E coli* in this setting, particularly with what appears to be the dominant lineage, *E coli* ST131.

Acknowledgments

The manuscript is dedicated to Amy Nakama-Peebles, who performed most of the isolation and sensitivity analysis in the microbiology lab and tragically passed away; she will be missed dearly. Drs. Richard Szabo (Southern California Kaiser Permanente) and Atreya Dash (Long Beach Veterans Affairs Medical Center and University of California-Irvine Medical Center) helped obtain Institutional Review Board approval and participated in initial patient enrollment at the respective institutions.

Funding Support: This material is partly based on work supported by the Office of Research and Development, Medical Research Service, Department of Veterans Affairs, grant #I01 CX000192 01 (J.R.J.). Hardy Diagnostics provided the quality-controlled culture media, and Copan Diagnostics provided the culture swabs.

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Figure 1.

Dendrogram shows virulence genotypes of 27 FQ-R *Escherichia coli* rectal isolates obtained before transrectal prostate biopsy. Colored squares indicate the presence of the gene. Only those virulence genes detected in 1 isolate are shown. Color codes: red, adhesins; green, toxins; blue, siderophore receptors; yellow, protectins; purple, miscellaneous virulence genes. Sequence type is shown for ST131 isolates only. Phylo group, major *E coli* phylogenetic group (A, B1, B2, D). ESBL (extended-spectrum β -lactamase production) status is indicated as POS if positive, blank if negative. ST131 isolates (group B2) are clustered tightly toward the top of the tree, with only one interposed non-ST131 isolate.

Figure 2. Dendrogram shows antimicrobial resistance profiles of 27 FQ-R *Escherichia coli* rectal isolates obtained before transrectal prostate biopsy. Colored squares indicate resistance to indicated antimicrobial agent. Only agents to which resistance was detected are shown. (No isolate was resistant to imipenem.) Drug abbreviations: AMP, ampicillin; SAM, ampicillin-sulbactam; KZ, cefazolin; CTX, ceftriaxone; CAZ, ceftazidime; FEP, cefepime; CN, gentamicin; TOB, tobramycin; CIP, ciprofloxacin; SXT, trimethoprim-sulfamethoxazole. Color code: green, penicillins; orange, cephalosporins; blue, amino-glycosides; yellow, fluoroquinolones; purple, folate antagonists. Phylo group, major *E. coli* phylogenetic group. ESBL, extended-spectrum β -lactamase; POS, positive.

Figure 3.

Dendrogram shows *Xba*I pulsed-field gel electrophoresis (PFGE) profiles of 27 FQ-R *Escherichia coli* rectal isolates obtained before transrectal prostate biopsy. PFGE, pulsotype (as defined by 94% profile similarity to the index isolate for a given pulsotype, within a large private PFGE profile database: JRJ). Phylo group, major *E. coli* phylogenetic group (A, B1, B2, D). Isolate ID# indicates the hospital of origin: VA, Veterans Affairs Medical Center; KP, Kaiser Permanente; UC, University of California, Irvine Medical Center. Pulsotype numbers <1000 were already defined in the reference PFGE library; numbers >1000 are novel to the present study.

Table 1

Characteristics of 27 men with fluoroquinolone-resistant (FQ-R) rectal *Escherichia coli* before transrectal prostate biopsy (January 2009 to March 2010)

Characteristic	Distribution of Characteristics by <i>E coli</i> Group		<i>P</i> Value
	ST131 FQ-R	Non-ST131 FQ-R	
	(n = 19) Mean (SE)	(n = 8) Mean (SE)	
Age, years	65.5 (1.3)	66.6 (1.8)	.64
Body mass index, kg/m ²	28.2 (0.8)	28.9 (1.90)	.72
Prostate size, cm ³	57.3 (5.6)	41.3 (3.7)	.09
Age-adjusted Charlson score	3.6 (0.5)	5.1 (0.7)	.32
PSA level,* ng/mL	6.2 (1.7)	5.6 (2.6)	.63
	No. (%)	No. (%)	
Race/ethnicity			.06
White	14 (74)	3 (38)	
African American	2 (11)	1 (13)	
Hispanic	2 (11)	0 (0)	
Asian (<i>P</i> = Asian vs other)	1 (5)	4 (50)	.02
Population			.19
University medical center	6 (32)	3 (38)	
VA Medical Center	11 (58)	2 (25)	
Large HMO medical center	2 (11)	3 (38)	
Prebiopsy sodium phosphate enema	2 (11)	4 (57)	.03
Previous prostate biopsy (1)	18 (94)	5 (62.5)	.03
Diabetes	4 (21)	4 (50)	.18
Family member(s) in health care	2 (11)	2 (25)	.62
Hospitalization (past 12 mos)	2 (11)	2 (25)	.56
	Mean (SE)	Mean (SE)	
Virulence gene (significant only)			
<i>sat</i>	19 (86)	3 (14)	.007
<i>usp</i>	19 (95)	1 (5)	<.001
<i>ompT</i>	19 (86)	3 (14)	<.001
<i>malX</i>	19 (90)	2 (10)	<.001
Coreistance profile			
Ampicillin	1 (5)	1 (13)	.51
Ampicillin/sulbactam	5 (26)	1 (13)	.63
Piperacillin/tazobactam	1 (5)	2 (25)	.2
Cefazolin	2 (7)	4 (50)	.04
Ceftazidime	1 (5)	2 (25)	.2
Gentamicin	7 (37)	4 (50)	.67
Tobramycin	9 (47)	4 (50)	>.99
Trimethoprim-sulfamethoxazole	6 (32)	5 (63)	.21

<u>Distribution of Characteristics by <i>E coli</i> Group</u>			
Characteristic	<u>ST131 FQ-R</u>	<u>Non-ST131 FQ-R</u>	<i>P</i> Value
	(n = 19)	(n = 8)	
	Mean (SE)	Mean (SE)	
ESBL phenotype	1 (5)	4 (50)	.02

ESBL, extended-spectrum β -lactamase; FQ-R, fluoroquinolone-resistant; HMO, health maintenance organization; PSA, prostate-specific antigen; SE, standard error; VA, Veterans Affairs.