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Epidemiology and Characteristics of *Escherichia coli* Sequence Type 131 (ST131) from Long-Term Care Facility Residents Colonized Intestinally with Fluoroquinolone-Resistant *Escherichia coli*

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Abstract

The objective of this study was to evaluate molecular and epidemiologic factors associated with *Escherichia coli* sequence type 131 (ST131) among long-term care facility (LTCF) residents who acquired gastrointestinal tract colonization with fluoroquinolone-resistant *E. coli* (FQREC). Colonizing isolates from 37 residents who newly developed FQREC colonization at three LTCFs from 2006–2008 were evaluated. Twenty-nine (78%) of 37 total FQREC colonizing isolates were

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ST131. Most ST131 isolates had a distinctive combination of *gyrA* and *parC* replacement mutations. The ST131 and non-ST131 isolates differed significantly for the prevalence of many individual virulence factors but not for the proportion that qualified molecularly as extraintestinal pathogenic *E. coli* (ExPEC) or aggregate virulence factor scores. *E. coli* ST131 was highly prevalent among LTCF residents with FQREC colonization. Future studies should determine the risk factors for infection among ST131-colonized residents, and assess the potential for increased transmissibility of ST131 in the long-term care setting.

Keywords

Escherichia coli; ST131; Long-term care

INTRODUCTION

The increasing prevalence of multidrug-resistant *Escherichia coli* has been driven largely by the widespread emergence of a single clonal group, sequence type 131 (ST131) [1–3]. *E. coli* ST131 is characterized by multidrug resistance, including resistance to fluoroquinolones (FQs) and extended-spectrum cephalosporins, the latter usually mediated by CTX-M-15 extended-spectrum beta-lactamase (ESBL)-production [4–7]. In addition, ST131 has been associated with expression of multiple virulence factors, including various toxins, adhesins, and siderophores [1, 7, 8]. More recently, *E. coli* ST131 has been shown to be associated with older age and long-term care facility (LTCF) residence [5].

The recent rapid increase in the elderly population has led to a corresponding rise in LTCF utilization in the United States [9]. Due to certain host and healthcare factors (e.g., immune senescence, comorbidities, and antibiotic use), LTCF residents are at significant risk for colonization and/or infection with antibiotic-resistant organisms [10–12]. In particular, fluoroquinolone-resistant *Escherichia coli* (FQREC) has dramatically increased in prevalence in LTCFs over the past decade, representing a major cause of infections in this vulnerable population [13–15].

FQREC infections are most likely preceded by gastrointestinal tract colonization with the causative strain [16, 17]. *E. coli* isolates within phylogenetic group B2, including specifically *E. coli* ST131, may have a greater capacity for successful and persistent colonization of the gastrointestinal tract [18, 19]. Although FQ resistance mechanisms and virulence factors associated with ST131 status have been described in clinical isolates [6], very little is known about these traits in colonizing isolates. Furthermore, despite the increasing burden of FQREC in LTCFs, the molecular and clinical epidemiology of *E. coli* ST131 colonization has only recently been characterized in the long-term care setting in the United States [20].

To improve understanding of *E. coli* ST131 in the long-term care setting, we assessed the molecular and clinical epidemiology of ST131 and non-ST131 *E. coli* colonizing isolates from residents who acquired FQREC colonization during LTCF stay. In addition, within individual residents we evaluated clonal relatedness between newly acquired FQREC isolates and preceding FQ-susceptible *E. coli* isolates to assess for possible in situ

development of resistance (i.e., conversion of a FQ-susceptible clone to a FQ-resistant clone). We also compared the present LTCF isolates with a large library of clinical ST131 isolates.

SUBJECTS AND METHODS

Study design and setting

This study was conducted from 2006 to 2008 at three LTCFs within the University of Pennsylvania: 1) LTCF #1, a 124-bed facility; 2) LTCF #2, a 240-bed facility; and 3) LTCF #3, a 200-bed facility. As previously described [21], LTCF residents who were colonized initially with FQ-susceptible *E. coli* (FQSEC) were followed longitudinally for up to 12 months. Serial fecal samples were obtained approximately every 14 days until recovery of FQREC, discharge, or death. The study was approved by the institutional review board of the University of Pennsylvania.

Study population

For the present study, we selected for further characterization the initial FQREC *E. coli* isolate from each of the 37 LTCF residents in the parent study who were documented previously to have developed incident FQREC colonization while under surveillance, i.e., who were colonized initially only with FQSEC and were found subsequently to be colonized with FQREC, +/− FQSEC (21). Additionally, to assess whether the subject's first detected FQREC isolate arose within the patient from a preceding colonizing FQ-susceptible isolate (i.e., conversion of a clone from FQSEC to FQREC), for each subject the most recent FQ-susceptible *E. coli* isolate prior to the index FQREC isolate was included for pulsed-field gel electrophoresis (PFGE) analysis if the sampling date was ≤ 30 days prior to the first fecal swab with FQREC.

Microbiologic methods

Given the multi-step nature of the development of FQ resistance (e.g., accumulation of mutations in the quinolone resistance-determining regions [QRDR] of the *gyrA* and *parC* genes), isolates with elevated FQ minimum inhibitory concentrations (MICs) may exhibit mutations in FQ target genes [22–25]. Therefore, isolates with elevated FQ MICs are important in evaluating development of FQ resistance. Accordingly, as described previously [21], FQ resistance was defined operationally as a levofloxacin MIC ≥ 0.25 μg/mL. FQ susceptibility was therefore defined as a levofloxacin MIC < 0.25 μg/mL.

Furthermore, LTCF residents have previously been shown to have high rates of gastrointestinal colonization with multiple distinct strains of *E. coli* [26]. To identify, isolate, and characterize multiple *E. coli* colonies with diverse FQ susceptibility profiles from a given patient, stool samples were inoculated and replica-plated, as follows: a perirectal swab with freshly (≤ 24 hours) collected stool sample was inoculated by triple-streaking onto a MacConkey agar plate and incubated at 37°C overnight. Single colonies suspected of being *E. coli* (up to 25 per plate) were replica-plated onto 4 MacConkey agar plates, each supplemented with a different concentration of levofloxacin (8 μg/mL, 1 μg/mL, 0.25μg/mL, no antibiotic). Each colony underwent PYR and indole testing. Indole-positive and PYR-

negative colonies were preliminarily considered *E. coli* and were saved in 30% glycerol stock at −80°C for further analyses.

From each specimen, up to six each presumed FQ-susceptible (levofloxacin MIC <0.25 μg/mL) and FQ-resistant (levofloxacin MIC ≥0.25 μg/mL) *E. coli* isolates, as available, were analyzed further. Isolates were identified definitively as *E. coli* and underwent standardized susceptibility testing using the semi-automated Vitek 2 identification and susceptibility system (bioMérieux, Durham, NC). A patient was considered to be colonized with FQREC if at least one stool isolate was confirmed as FQREC.

Evaluation for specific mechanisms of FQ resistance was performed as described previously [27–29]. Overexpression of AcrAB was measured using the organic solvent tolerance (OST) assay [30, 31]. The genetic relatedness of *E. coli* isolates was determined by PFGE [27], with all profiles analyzed using the Bionumerics v6.6 (Applied Maths, Austin, TX) and interpreted according to established criteria [32]. Strains were defined as those isolates sharing a pulsotype. Pulsotypes were assigned based on > 94% similarity to reference strains within an established PFGE library [33]. Susceptibility testing was performed using the semiautomated Vitek 2 system and interpreted according to Clinical and Laboratory Standards Institute criteria.

Major *E. coli* phylogenetic group (A, B1, B2, D) was determined by triplex PCR [34]. ST131 status was determined by detection of ST131-specific single-nucleotide polymorphisms (SNPs) in *mdh* and *gyrB* [35]. ST131 isolates were screened by PCR for presence of the O25b *rfb* variant [8] and for membership in the *H*30 ST131 subclone [6, 36]. The prevalence of the (CTX-M-15-associated) *H*30Rx subclone [37] was assessed by PCR-based detection of a subclone-specific SNP (G723) within the allantoin protein-encoding *gene, ybbW* [38].

Fifty extraintestinal virulence-associated genes were detected by multiplex PCR [35, 39]. Virulence scores were calculated as the total number of virulence genes detected, adjusted for multiple detection *of the pap* (P fimbriae), *sfa/foc* (S and F1C fimbriae), *kpsM* II (group 2 capsule), and *clb* (colibactin) operons. Isolates were regarded as extraintestinal pathogenic *E. coli* (ExPEC) if positive for ≥ 2 of the following genes: *papA* and/or *papC* (P fimbriae); *afa/draBC* (Dr-family adhesins); *sfa/focDE; iutA* (aerobactin receptor); and *kpsM* II (group 2 capsule synthesis) [36].

Data collection

Baseline demographic and clinical characteristics were extracted from the LTCF medical record using a standardized data abstraction form [21]. Clinical variables included comorbidities, fecal incontinence, bed-bound status, and presence of a urinary or central venous catheter at the time of study enrollment. All antimicrobial therapy received within 30 days of initial detection of FQREC colonization, was documented, as was receipt of immunosuppressive agents.

Statistical analysis

Among FQREC isolates, associations were explored between *E. coli* ST131 and 1) mechanisms of FQ resistance, 2) antibiotic susceptibility profiles, and 3) virulence factors. Clinical characteristics were compared between subjects with and without an ST131 FQREC colonizing isolate. Categorical variables were compared using the Fisher exact test, and continuous variables were compared using the Wilcoxon rank-sum test. Odds ratios (OR) and 95% confidence intervals (CI) were calculated to evaluate the strength of associations between ST131 status and clinical variables. All statistical calculations were performed using commercially available software (STATA v13.0; StataCorp LP, College Station, Texas).

RESULTS

Prevalence of ST131

E. coli isolates were analyzed from all 37 LTCF residents who were identified as having incident FQREC colonization (levofloxacin MIC ≥0.25 μg/mL) during the study period. Most (n = 24; 65%) subjects had multiple strains of *E. coli* detected. The median number of distinct *E. coli* strains identified on testing from each initial FQREC-containing fecal sample for these 24 residents was 2 (interquartile range [IQR], 2, 3), most commonly one FQ-susceptible and one FQ-resistant strain.

The first-identified FQREC isolate was characterized for each of the 37 residents; of these, 29 (78%) were identified as ST131 by dual-SNP PCR (Table 1). The only other ST131 isolate identified among the study subjects was a FQSEC isolate (levofloxacin MIC of 0.047 μg/mL) from a multiply-colonized patient who had one non-ST131 FQ-resistant strain and two FQ-susceptible strains, including this ST131 isolate. Thus, 29 (97%) of the study's 30 total ST131 isolates were FQREC.

All 29 ST131 FQREC isolates represented the *H*30 subclone within ST131, and 9 (31%) belonged specifically to the *H*30Rx subgroup within the *H*30 subclone. In contrast, the single ST131 FQSEC isolate did not belong to the *H*30 ST131 subclone.

Fluoroquinolone resistance mechanisms

The 29 ST131 and 8 non-ST131 FQREC isolates did not differ significantly for levofloxacin MIC (median value, 24 μg/mL versus 20 μg/mL, respectively, $P = 0.58$) (Table 1). ST131 FQREC isolates had a greater number of replacement *gyrA* mutations compared to non-ST131 FQREC isolates, with a median of 2 (interquartile range [IQR], 2, 2), versus 2 (IQR, 1, 2) ($P = 0.01$). All isolates with two *gyrA* mutations exhibited Ser83Leu and Asp87Asn, regardless of ST131 status. Similarly, ST131 isolates had a greater number of replacement *parC* mutations compared to non-ST131 isolates, with a median of 2 (IQR, 2, 2), versus 1 (IQR, 0, 1) ($P < 0.001$). The most common combination of replacement *parC* mutations among ST131 FQREC isolates, present in all but two of the characterized isolates, was Ser80Ile and Glu84Val.

Virulence genes

Differences in virulence gene profiles between ST131 and non-ST131 FQREC isolates are shown in Table 2. Virulence genes significantly associated with ST131 included *fimH*, *sat*, *usp*, *ompT*, and *malX*. A similar proportion of ST131 and non-ST131 isolates qualified as ExPEC (45% and 50%, respectively; $P > 0.99$). There was no significant difference in virulence gene scores between ST131 isolates and non-ST131 isolates, with median scores of 9 (IQR, 8, 10) and 8 (IQR, 4, 12), respectively ($P = 0.64$). The virulence gene profile of the one FQ-susceptible ST131 *E. coli* isolate included adhesins (*iha*, *fimH*, *afa/draBC*), toxins (*hylD*, *sat*, *tsh*), siderophore receptors (*iroN*, *fyuA*, *iutA*), and other factors (*kpsM II*, *K5*, *iss*, *ompT*, *ibeA*, *usp*, *traT*, *rfc*, *cvaC*), qualifying this isolate molecularly as ExPEC.

Antibiotic susceptibility

Overall, ST131 and non-ST131 FQREC isolates (as defined by a levofloxacin MIC ≥0.25 μg/mL) exhibited no significant differences in antibiotic susceptibility profiles (Table 3). Eight (28%) ST131 isolates were ceftazidime-resistant; four (50%) of these belonged to the *H*30Rx subgroup within the H30 subclone. The prevalence of ceftazidime resistance was numerically but not statistically significantly higher among *H30Rx* isolates compared to the other ST131 isolates (44% versus 20%; $P = 0.20$).

PFGE analysis

PFGE was performed for 28 of the 29 FQREC ST131 isolates, and pulsotypes were compared to those of previously characterized clinical ST131 isolates from a large, established library [33]. Among the present ST131 colonizing isolates the predominant pulsotypes (defined based on ≥ 94% similarity to an index isolate) were 968 and 808, with 7 and 4 isolates respectively (Figure 1), which also are the leading pulsotypes among the diverse-source clinical ST131 isolates in the reference library [33].

In assessing longitudinal development of resistance, 26 (70%) of 37 subjects had a fecal swab that yielded FQSEC (levofloxacin MIC <0.25 μg/mL) within 30 days prior to the first fecal swab that yielded FQREC (levofloxacin MIC ≥0.25 μg/mL). On PFGE analysis, none of these FQSEC isolates exhibited ≥ 76% profile similarity to the corresponding patient's subsequent FQREC isolate. Furthermore, none of the FQSEC isolates were ST131.

Risk factors for ST131

Among the 37 subjects with incident FQREC colonization, clinical characteristics were compared between the 29 subjects whose new FQREC strain was ST131 and the 8 for whom it was not (Table 4). These two subgroups did not differ significantly for LTCF of residence, presence of various comorbidities, device use, or receipt of various antibiotic agents or classes ($P > 0.10$ for all comparisons). There was a trend approaching statistical significance toward an association between female gender and having an ST131 incident colonizing FQREC strain (OR, 0.12; 95% CI, 0.01–1.03; $P = 0.05$).

DISCUSSION

During a 3-year period of screening for colonization with FQREC among residents of 3 LTCFs in Pennsylvania (2006–2008) we evaluated the molecular and clinical epidemiology of ST131 and non-ST131 *E. coli* colonizing isolates from residents who acquired FQREC colonization during their LTCF stay. We found a strikingly high prevalence of *E. coli* ST131 (nearly 80%) among the incident FQREC isolates (. Although ST131 and non-ST131 isolates differed significantly for the prevalence of many individual virulence genes, they did not differ for the proportion of isolates that qualified molecularly as ExPEC or for aggregate VF scores. In addition, there were no significant differences in clinical risk factors, including recent antibiotic use, for recovery of ST131 versus non-ST131 FQREC. Lastly, the PFGE pulsotypes for the ST131 colonizing isolates from residents in this study corresponded to those that were common among previously characterized clinical ST131 isolates.

The prevalence of antibiotic-resistant organisms in LTCFs is high, likely due in part to high rates of antibiotic use, elderly hosts, and significant opportunities for person-to-person transmission [40]. In a recent study, LTCF residence and older age were predictors of infection with *E. coli* ST131 [5]. Similarly, the prevalence of *E. coli* ST131 in our study cohort of LTCF residents was notably high, with nearly 80% of residents who developed new colonization with FQREC during longitudinal sampling acquiring *E. coli* ST131. Clinical infection with FQREC is most likely preceded by gastrointestinal tract colonization [16, 17]. Therefore, this finding is particularly concerning given the elderly, vulnerable population, and suggests that LTCFs may serve as an important reservoir of *E. coli* ST131.

The high prevalence of *E. coli* ST131 among incident colonizing FQREC isolates in our study may have been due in part to a greater propensity for colonization of the gastrointestinal tract by *E. coli* isolates within phylogenetic group B2 [18, 19] and ST131 specifically, in combination with other risk factors present in the LTCF setting (e.g., antibiotic selection pressure). Notably, a significant proportion (~40%) of LTCF residents who acquired ST131 FQREC colonization received at least one antibiotic prior to ST131 *E. coli* recovery. Lastly, increased colonization pressure with *E. coli* ST131 may have increased the risk of subsequent acquisition of ST131 FQREC by LTCF residents.

As shown previously for clinical ST131 FQREC isolates [6], the majority of fecal ST131 FQREC isolates in our study demonstrated a distinct *gyrA/parC* allele combination. In addition, common ST131-associated pulsotypes from a large reference library were the predominant pulsotypes (968 and 808) represented in our LTCF cohort. These findings support the likely clinical relevance of the present colonization isolates.

Notably, among the 26 study subjects who had an available FQ-susceptible fecal *E. coli* isolate from within 30 days prior the subject's incident FQREC isolate, in no instance was the antecedent FQSEC isolate genetically related to the subject's later incident FQREC isolate. These findings establish that person-to-person or environmental transmission of already-resistant strains was a more important determinant of FQREC acquisition in these LTCFs than in situ development of de novo FQ resistance (e.g., by stepwise acquisition of *gyrA* and *parC* mutations in a given strain during sustained colonization). In this regard,

ST131 strains, or the particular ST131 lineages identified here, may be characterized by increased transmissibility, compared with other FQREC. This potentially greater risk of dissemination should be further evaluated in future studies, focusing particularly on the LTCF setting, which is notable for increased opportunity for person-to-person contact.

A recent study demonstrated that, among clinical FQREC isolates, a significantly greater proportion of ST131 compared to non-ST131 isolates were classified as ExPEC [36]. In contrast, among the present fecal FQREC isolates, although the ST131 and non-ST131 isolates differed significantly for the distribution of specific virulence genes, they did not differ for the proportion that qualified molecularly as ExPEC. This finding may be explained by differences in virulence potential between colonizing isolates versus those causing clinical infections. Furthermore, it is possible that the rapid dissemination of ST131 may be a result more of ST131's ability for successful and persistent intestinal colonization than for greater virulence. Nevertheless, data are needed from ST131-colonized LTCF residents regarding the incidence of and clinical and molecular characteristics associated with progression to clinical infection.

Finally, both ST131 and non-ST131 FQREC isolates exhibited resistance to multiple non-fluoroquinolone antibiotics, including aminoglycosides, ampicillin-sulbactam, and trimethoprim-sulfamethoxazole. The multidrug resistance seen in the present ST131 isolates confirms high rates of colonization with multidrug-resistant gram-negative pathogens seen in the long-term care population [14, 40], and has important implications for selection of empiric therapy for infections in LTCF residents.

Potential limitations of our study must be considered. First, the small sample size, especially for the non-ST131 group, limited statistical power for identification of risk factors for ST131 colonization. Second, since the present study was conducted in LTCFs that were part of a single healthcare system, the results may not be generalizable to other LTCFs with differing characteristics. Third, since our study assessed only incident FQREC isolates, it is uninformative the distribution of ST131 vs. non-ST131 *E. coli* in prevalent FQREC colonization, which might favor ST131 even more disproportionately than seen here if ST131 strains tend to out-persist other FQREC in the gut.

In conclusion, we found a high prevalence of ST131 among FQREC strains newly acquired by LTCF residents during their LTCF stay. Given the predominantly elderly, vulnerable population in the long-term care setting, future studies are needed to evaluate potentially modifiable risk factors for such acquisition and for subsequent infection in ST131 colonized residents. Furthermore, elucidating the potential for increased transmissibility of ST131 isolates, particularly in long-term care settings characterized by frequent patient-to-patient contact, will be particularly important for development of effective infection control strategies to limit the spread of this emerging multidrug-resistant pathogen.

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- A prevalence of *E. coli* ST131 of nearly 80% was found among incident FQREC isolates in long-term care facility residents.
- ST131 and non-ST131 isolates differed significantly in the prevalence of virulence factors.
- ST131 and non-ST131 isolates did not differ in the proportion of isolates that qualified molecularly as ExPEC.
- PFGE pulsotypes for colonizing ST131 isolates were common among previously characterized clinical ST131 isolates.

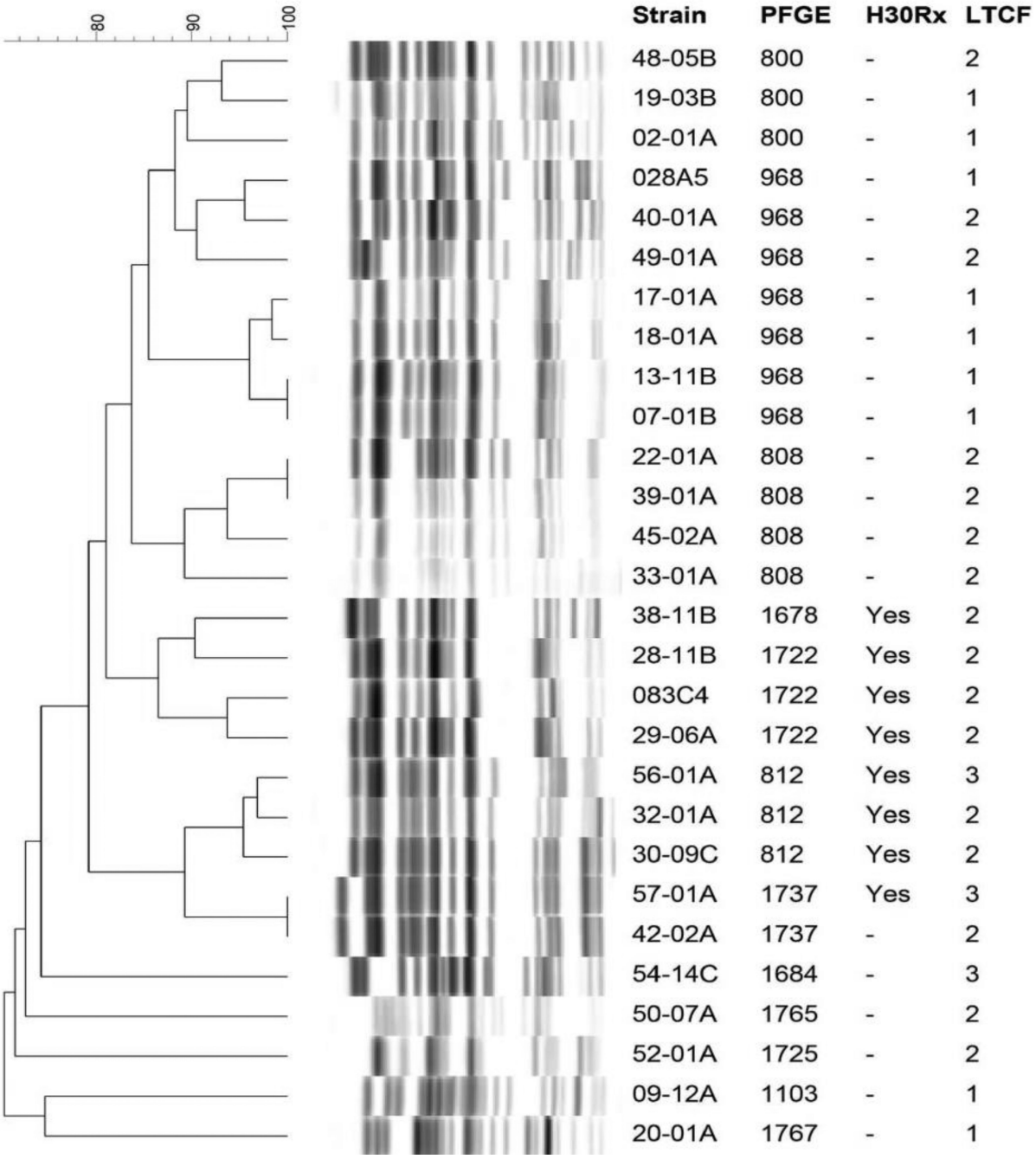

Figure 1. Pulsed field gel electrophoresis patterns of 28 fluoroquinolone-resistant ST131 fecal *Escherichia coli* isolates from long-term care facility residents

Data columns to the right of the PFGE profiles are the strain number, pulsotype, *H*30Rx subclone status, and long-term care facility of origin (1, 2, or 3). Pulsotypes were assigned by comparison to an existing large private PFGE profile reference library (33). All isolates represented the *H*30 subclone within ST131.

Table 1

Microbiologic characteristics of ST131 and non-ST131 *E. coli* fecal isolates from long-term care facility residents colonized with fluoroquinolone-resistant *Escherichia coli*.

Variable	ST131 (n = 29)	Non-ST131 (n = 8)	*P* value
Median levofloxacin MIC (IQR)	24 (16, 32)	20 (8, 32)	0.58
Median no. of *gyrA* mutations (IQR)[a]	2 (2, 2)	2 (1, 2)	0.01
Median no. of *parC* mutations (IQR)[b]	2 (2, 2)	1 (0, 1)	< 0.001
OST-positive status, no. (%)	6 (21)	4 (50)	0.17

NOTE. IQR, interquartile range; OST, organic solvent tolerance.

[a]One ST131 isolate could not be characterized in regard to *gyrA* mutations.

[b]One ST131 isolate could not be characterized in regard to *parC* mutations.

Table 2

Distribution of virulence-associated traits among ST131 and non-ST131 *E. coli* fecal isolates from long-term care facility residents colonized with fluoroquinolone-resistant *Escherichia coli*.

Trait		Prevalence of trait, no. (%)		*P* value[a,b]
		ST131 (n = 29)	Non-ST131 (n = 8)	
Adhesins	*papAH*[c]	0 (0)	1 (13)	0.22
	iha	21 (72)	3 (38)	0.10
	fimH	29 (100)	6 (75)	0.04
Toxins	*sat*	23 (79)	3 (38)	0.04
	vat	0 (0)	5 (63)	< 0.001
	hlyF[d]	0 (0)	1 (13)	0.22
Siderophores	*iroN*	0 (0)	1 (13)	0.22
	fyuA	29 (100)	7 (88)	0.22
Miscellaneous	*usp*	27 (93)	4 (50)	0.01
	traT	27 (93)	5 (63)	0.06
	c*vaC*	9 (0)	1 (13)	0.22
	K1	29 (100)	1 (13)	0.22
	ompT	29 (100)	5 (63)	0.007
	iss	0 (0.0)	1 (13)	0.22
	H7 *fliC*	0 (0)	2 (25)	0.04
	malX	49 (100)	6 (75)	0.04
	clbB/N	0 (0)	1 (13)	0.22

[a] Only traits that yielded a *P* value < 0.30 (by Fisher exact test) are shown. *papAH* (P fimbria structural subunit), *iha* (adhesin-siderophore), *fimH* (type 1 fimbria adhesin), *sat* (secreted autotransporter toxin), *vat* (vacuolating toxin), *hlyF* (hemolysin F), *iroN* (catecholate siderophore receptor), *fyuA* (yersiniabactin receptor), *usp* (uropathogenic-specific protein), *traT* (serum resistance outer membrane protein), *cvaC* (colicin V precursor), K1 (group 2 capsule variant), *ompT* (outer membrane protease), *iss* (increased serum survival protein), H7 (flagellar variant), *malX* (pathogenicity island marker), and *clbB/N* (colibactin synthesis system).

[b] The presence of the following traits were evaluated but not detected in any isolate: *papG* allele I (P adhesin variant), *papG* allele III (P adhesin variant), *sfa/focDE* (S and F1C fimbriae), *sfaS* (S fimbriae), *focG* (F1C fimbriae), *afa/draBC* (Dr-binding adhesins), *afaE8* (variant afimbrial adhesin), *bmaE* (M-agglutinin subunit), *gafD* (G fimbriae), F17 (fimbrial adhesin), *clpG* (CS31A surface antigen), *hlyD* (hemolysin D), *cnf1* (cytotoxic-necrotizing factor 1), *cdtB* (cytolethal distending toxin), *tsh* (temperature sensitive hemagglutinin), *ireA* (siderophore receptor), *iutA* (aerobactin receptor), *kpsM* II (group 2 capsule), *kpsM* III (group 3 capsule), K5 (group 2 capsule variant), K15 (group 2 capsule variant), K2/K100 (group 2 capsule variants), *rfc* (O4 antigen polymerase), and *ibeA* (invasion of brain endothelium).

[c] *papC* (P fimbria assembly), *papEG* (P fimbria tip pilins), *papG* allele II (P adhesin variant), and *hra* (heat-resistant agglutinin) demonstrated similar results to those for *papAH*.

[d] *pic* (protein associated with intestinal colonization) and *astA* (enteroaggregative *E. coli* toxin) demonstrated similar results to those for *hlyF*.

Table 3

Antibiotic resistance by ST131 status among fluoroquinolone-resistant fecal *Escherichia coli* isolates from long-term care facility residents.

Antibiotic[a]	Prevalence of resistance, no. (%)			*P* value
	All isolates (n = 37)	ST131 (n = 29)	Non-ST131 (n = 8)	
Ampicillin-sulbactam	31 (84)	25 (86)	6 (75)	0.59
Cefazolin	13 (35)	10 (35)	3 (38)	> 0.99
Ceftazidime	10 (27)	8 (28)	2 (25)	> 0.99
Ceftriaxone	9 (24)	7 (24)	2 (25)	> 0.99
Gentamicin	13 (35)	8 (28)	5 (63)	0.10
Tobramycin	16 (43)	11 (38)	5 (63)	0.25
Piperacillin-tazobactam	4 (11)	2 (7)	2 (25)	0.20
Trimethoprim-sulfamethoxazole	17 (46)	14 (48)	3 (38)	0.70

[a] Antibiotics listed are those to which ≥ 1 isolate was resistant. No isolate was resistant to imipenem.

Table 4

Bivariable analyses of risk factors for ST131 *Escherichia coli* among long-term care facility residents colonized with fluoroquinolone-resistant *E. coli*.

Variable	ST131 (n = 29)[a]	Non-ST131 (n = 8)[a]	OR (95% CI)	*P* value
Year of culture				
2006	11 (38)	4 (50)		
2007	8 (28)	3 (38)		0.61
2008	10 (34)	1 (13)		
Mean age (SD)	75 (12)	70 (15)	--------	0.28
Female sex	3 (10)	4 (50)	0.12 (0.01–1.03)	0.05
Non-white race	19 (66)	4 (50)	1.90 (0.28–12.5)	0.45
Site				
LTCF #1	9 (31)	4 (50)		
LTCF #2	16 (55)	4 (50)		0.62
LTCF #3	4 (14)	0 (0)		
Hospitalization in previous year	22 (76)	5 (63)	1.89 (0.23–12.8)	0.67
Comorbidities and medications[b]				
Chronic pulmonary disease	11 (38)	1 (13)	4.28 (0.43–210)	0.23
Malignancy	12 (41)	1 (13)	4.94 (0.50–241)	0.22
Any antibiotic[c]	11 (38)	1 (13)	4.28 (0.43–210)	0.23

OR, odds ratio; CI, confidence interval; SD, standard deviation; LTCF, long-term care facility.

[a] Data are presented as numbers (percentages) except for age.

[b] Only variables with $P < 0.30$ are shown.

[c] ≤ 30 days prior to recovery of FQREC.