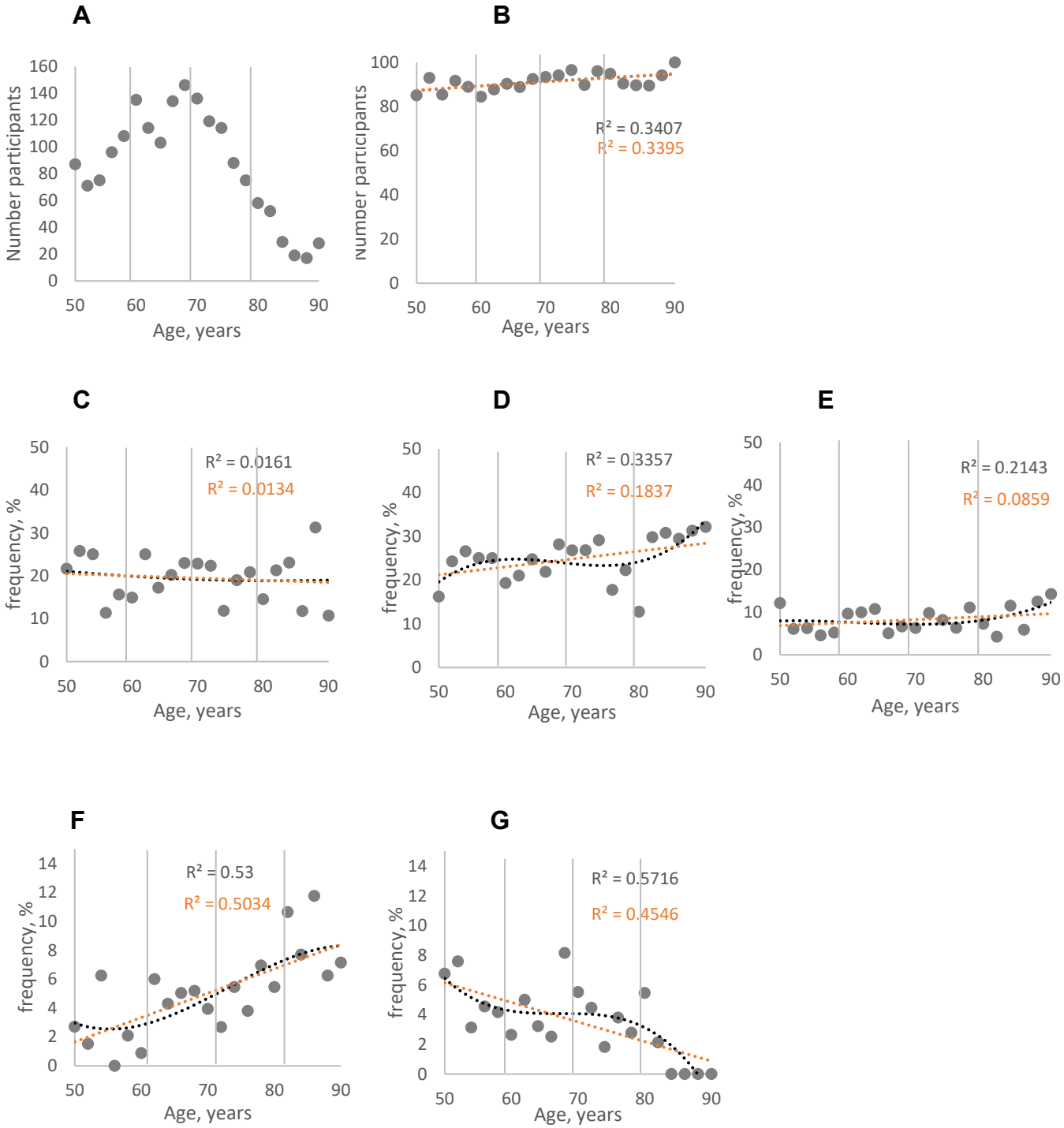
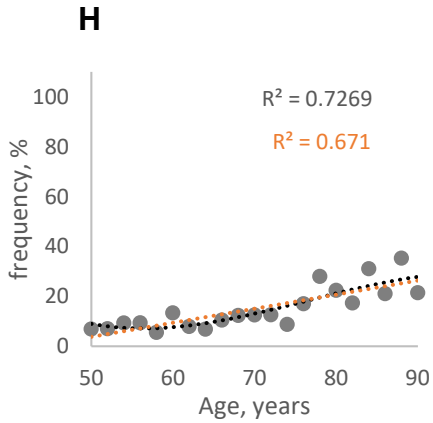
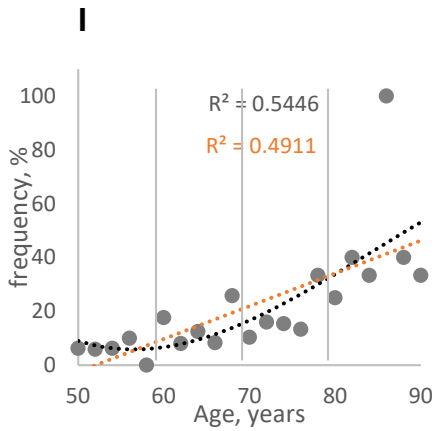
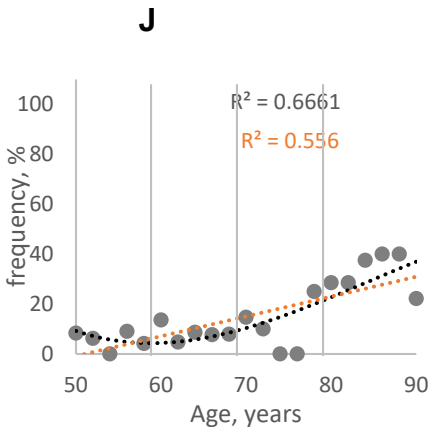
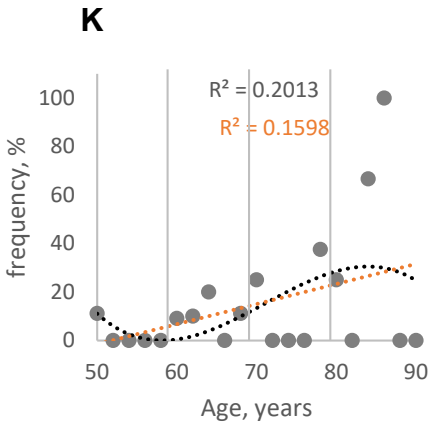


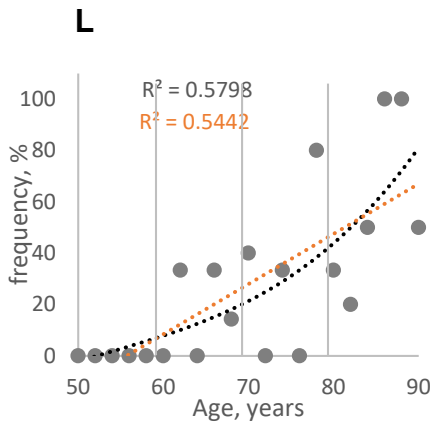
Figure S1. Analysis of gut *E. coli* carriage and UTI incidence by age. Number of participants, carriage rates and UTI incidence rates were aggregated for every two-year age bracket, and the frequency was calculated as a percentage of the total number of participants within each two-year age range. For participants 90-98 years of age numbers were aggregated into one bin. Plotted frequencies were used to calculate either the best-fit trend (grey dotted line), or linear trend (orange line), with R^2 value shown on the graph in grey and orange, respectively. (A) Overall age distribution among study participants. (B) Prevalence of fecal samples with E. coli. (C) Prevalence of fecal samples with *E. coli* resistant to CIP. (D) Prevalence of fecal samples with *E. coli* resistant to TS. (E) Prevalence of fecal samples with *E. coli* resistant to 3GC. (F) Prevalence of fecal samples with *E. coli* from ST131-H30. (G) Prevalence of fecal samples with *E. coli* from ST1193.

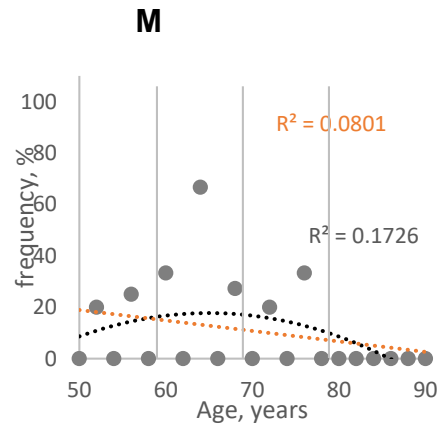












Supplemental Figure S1. Analysis of gut *E. coli* carriage and UTI incidence by age22 (continued). (H) Overall UTI incidence rate distribution by age. UTI incidence rates in resistant *E. coli* carriers: FQREC carriers (I), TSREC carriers (J), 3GCREC carriers (K), H30 carriers (L) and ST1193 carriers (M).

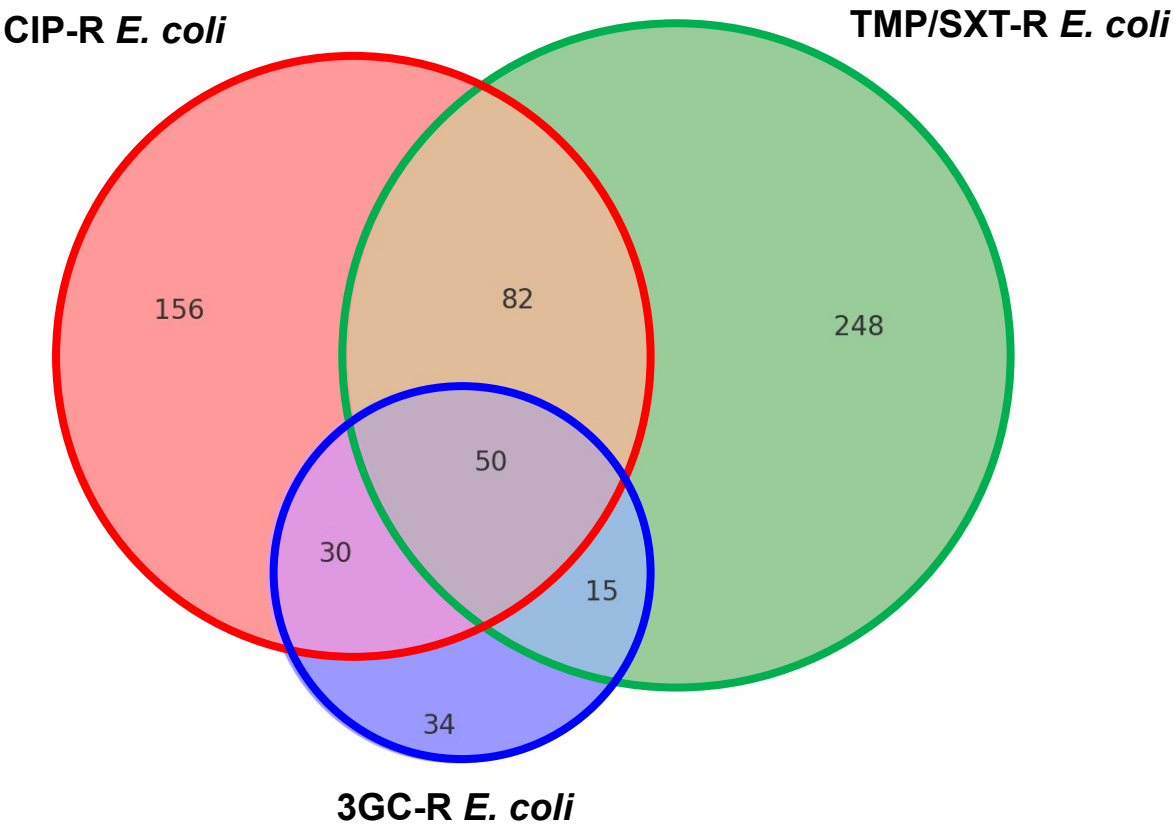
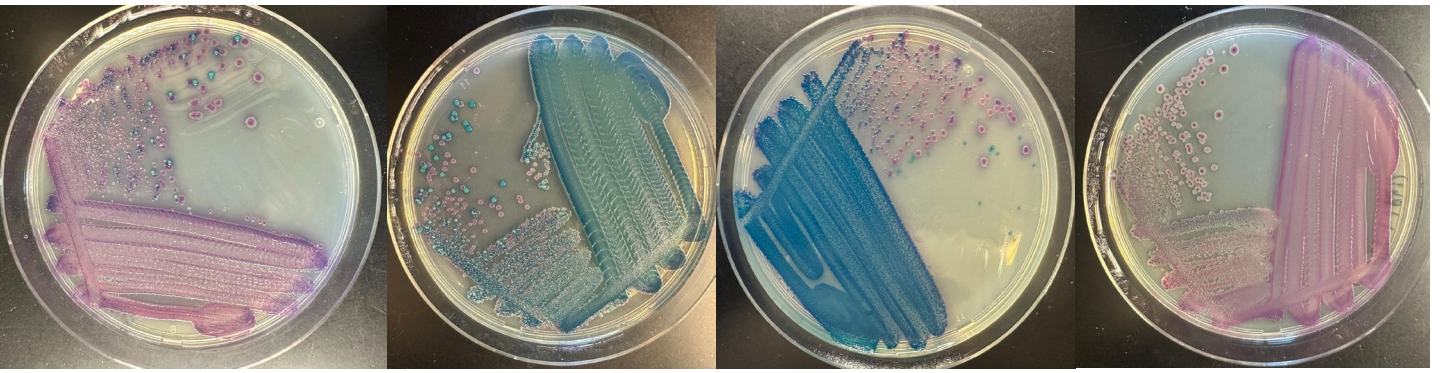


Figure S2. Venn diagram showing the distribution of CIP-R, TMP/STX-R, and 3GC-R *E. coli* in fecal samples. Overlaps indicate the presence of *E. coli* resistant to multiple antibiotics within the same fecal sample, regardless of whether the resistances are carried by the same or different clones.

A



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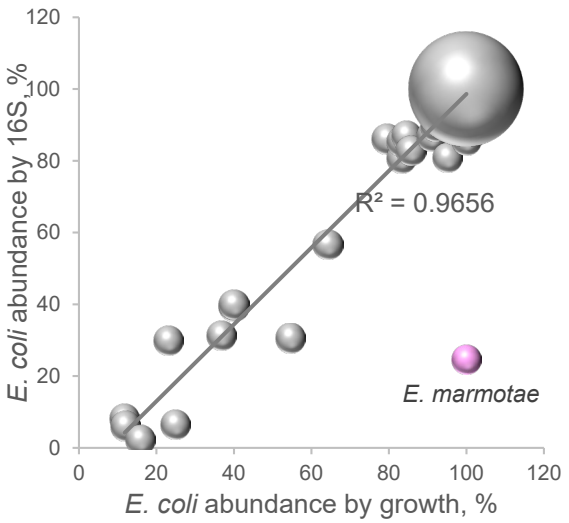
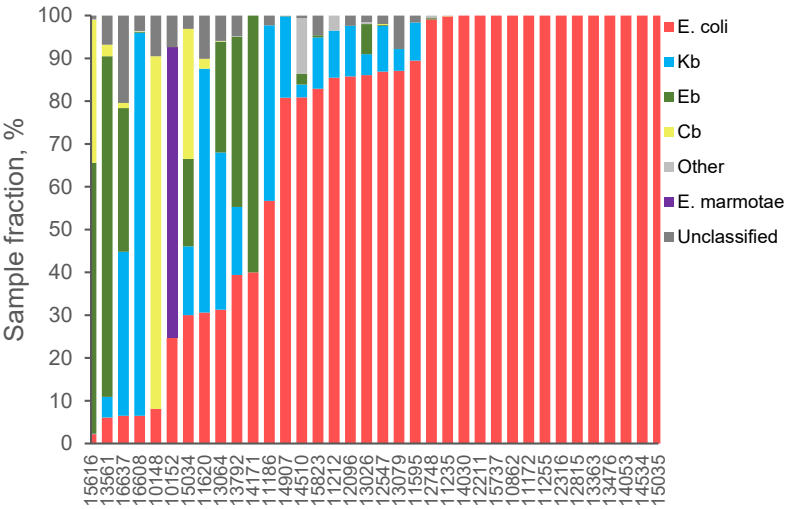
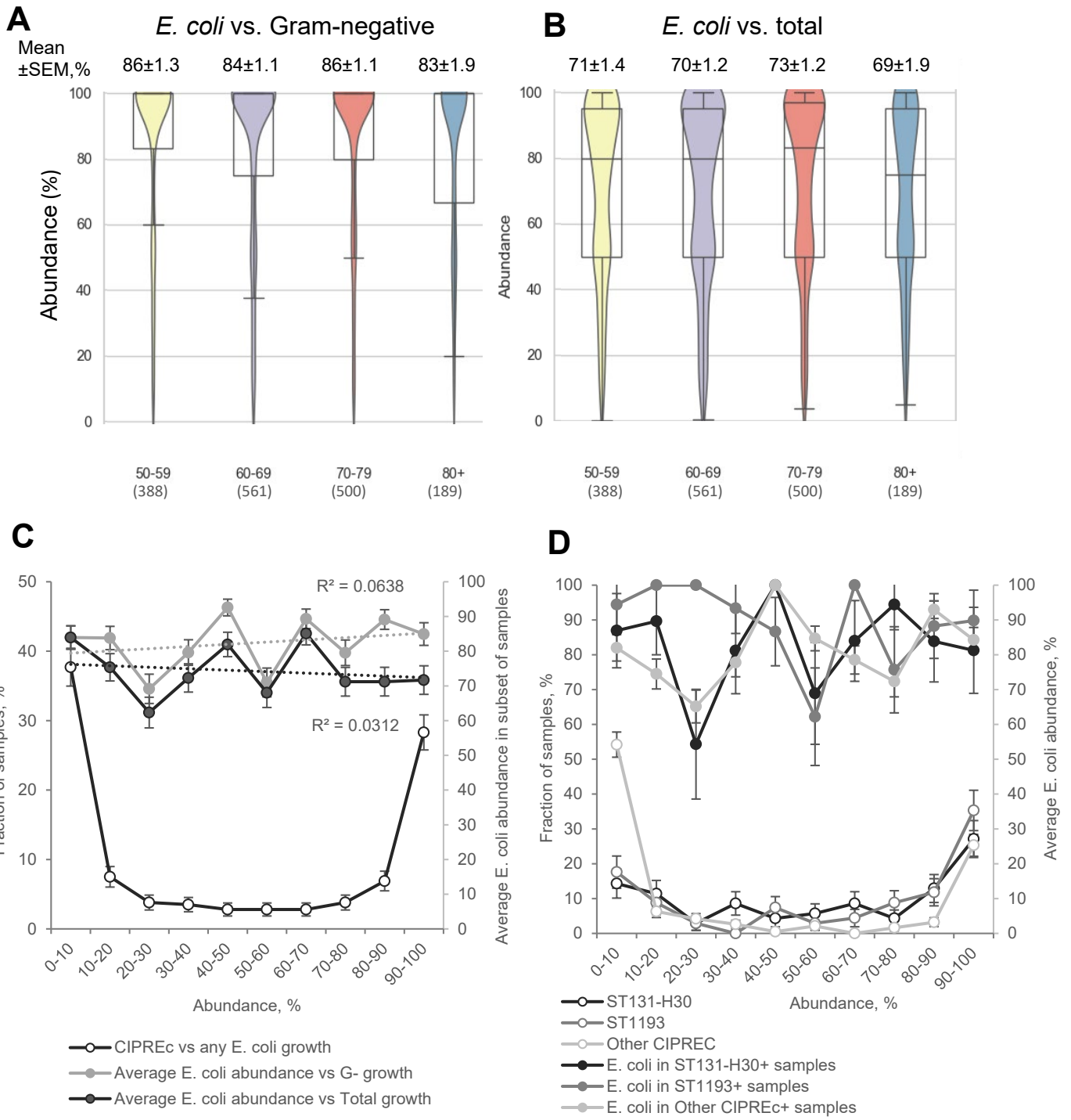


C



Supplemental Figure S3. Comparative analysis of relative abundance of E. coli in fecal sample versus other bacteria. (A) Various patterns of relative species abundance based on the four-quadrant streaking of the fecal samples on UTI agar. *E. coli* produce pink colonies. (B) Correlation between *E. coli* abundance in fecal samples estimated by growth on chromogenic UTI agar and determined by 16S sequencing. The size of a bubble reflects the number of samples. A linear trendline was plotted for all samples except one marked in color (*E. marmotae)*, with R^2 value indicated on the graph. (C) Relative abundance of *E. coli* and other bacterial species in fecal samples by 16S. The X-axis represents individual fecal samples, while the Y-axis shows the relative abundance of bacterial species as a percentage of the total bacterial composition in each sample.



Supplemental Figure S4. Distribution of different *E. coli* abundancies in fecal samples. (A-B) Combined violin/box plot distribution of *E. coli* abundances in fecal sample relatively to Gram-negative (A) or total (B) bacterial grown on UTI agar, split by the different age groups. Sample sizes for each age group are shown in parentheses. Mean abundances (Mean ± SEM %) are displayed above the plots, with no significant difference between the groups. (C-D) Distribution of CIP-R *E. coli* abundance compared to average *E. coli* abundance. (C) All CIP-R growth level relative to any *E. coli* growth is plotted on primary vertical axis, whereas average *E. coli* growth relative to Gram-negative (G-) or overall (Total) bacterial growth in these subsets of samples is plotted on a secondary vertical axis, with trendlines and R^2 values representing linear fit of the data. Error bars represent standard error. (D) Same as (C) for different CIPREc clonal groups, with average *E. coli* growth relative to Gram-negative growth only.

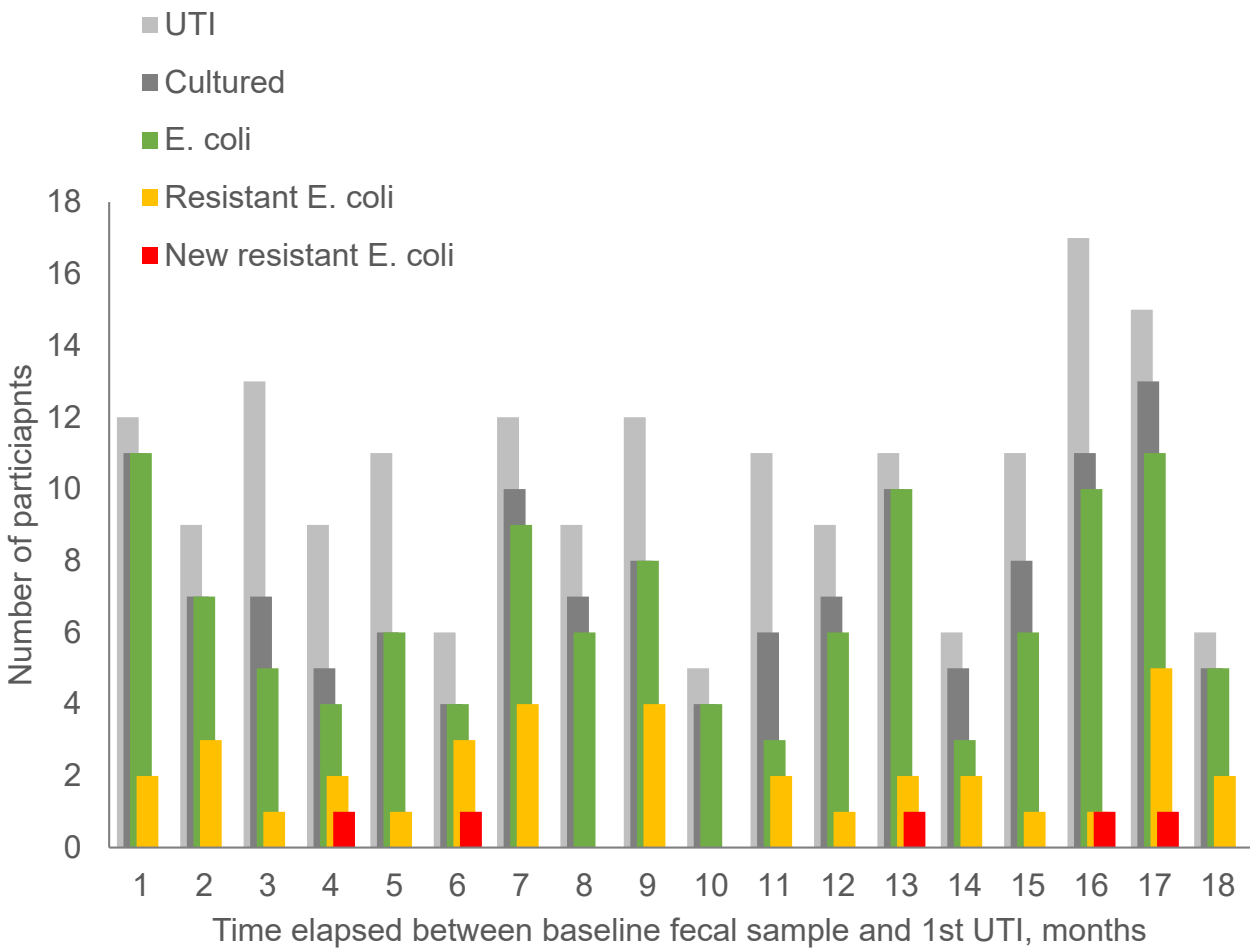
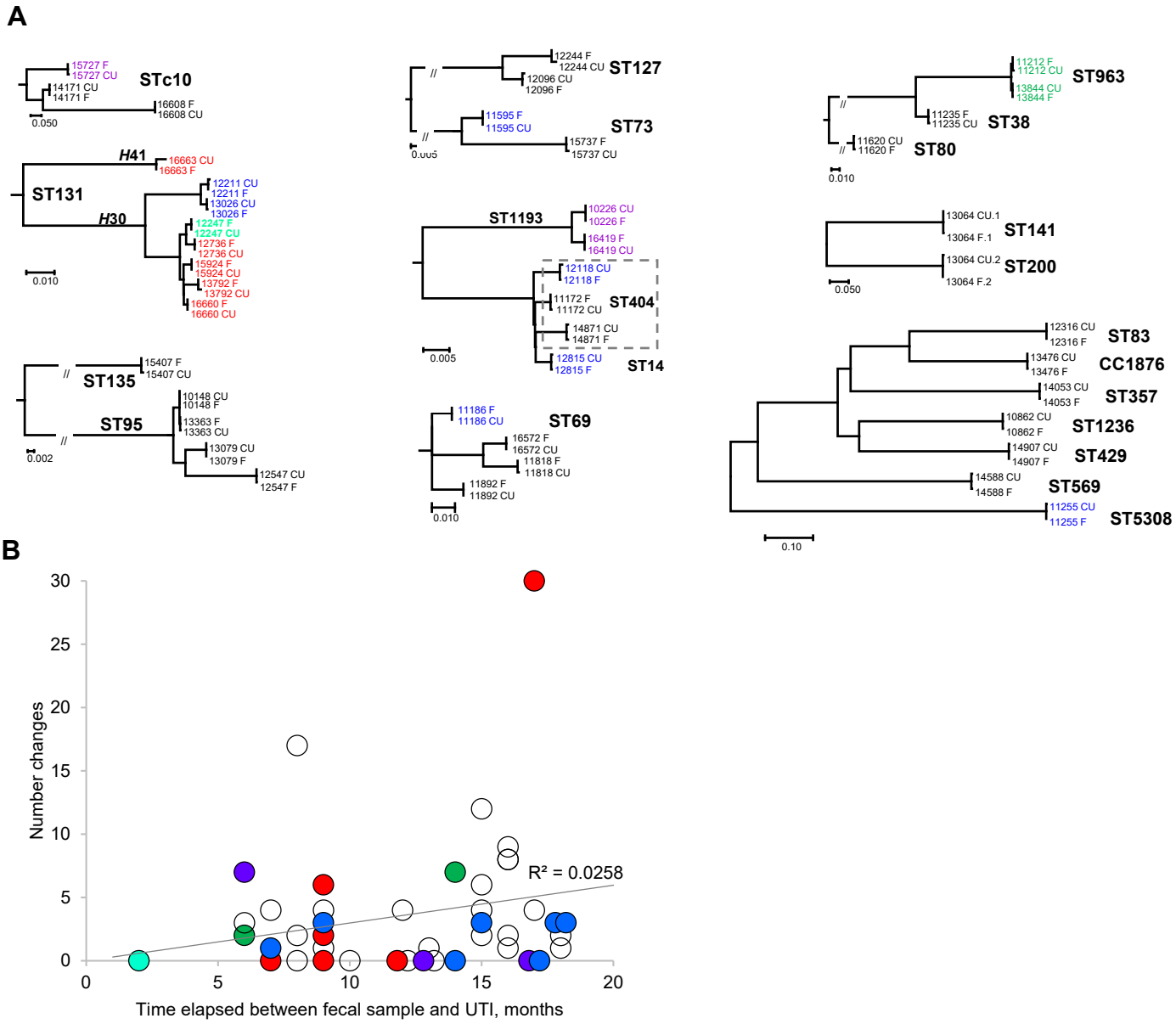
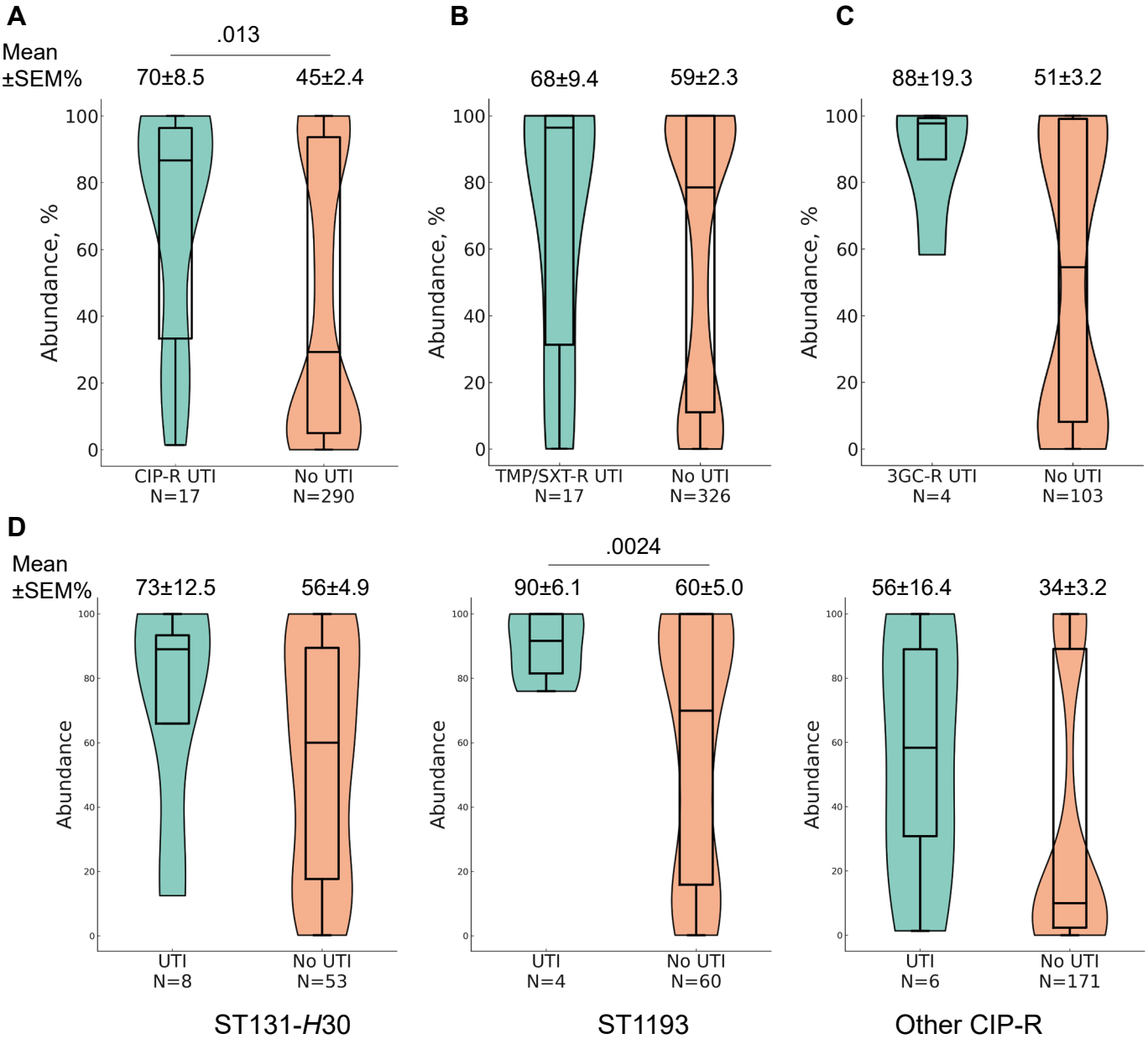


Figure S5. UTI incidence rates and uropathogens' characteristics. Plotted here are overlaying frequencies as follows: total number of UTIs occurring within 1-18 months from submitting fecal samples in grey; positive urine culture in dark-grey; *E. coli* in urine green; *E. coli* resistant to FQ, TS and/or 3GC in orange; *E. coli* resistant to FQ, TS and/or 3GC and not found in fecal sample in red.



Supplemental Figure S6. Analysis of core-genome sequences for paired fecal (F) and corresponding clinical urine (CU) isolates. (A) Molecular phylogenetic analyses were performed using the Maximum Likelihood method implemented in MEGA7, generating SNP-based phylogenies for *E. coli* fecal-urine pairs with closely related sequence types (STs). Separate phylogenetic trees were constructed for each group of closely related pairs. Nine pairs that lacked a closely related counterpart within the dataset were placed on two trees: one for sample ID_13064, which contained *E. coli* belonging to two distinct sequence types, and one for the remaining seven STs. **(B)** Total numbers of core-gene changes (SNPs and indels) between F and CU isolates plotted against the time interval between baseline fecal sampling and clinical urine collection. A linear trendline is shown with the R^2 value indicated. Isolates resistant only to CIP are shown in red, only to TMP/SXT in blue, only to 3GC in green, to both CIP and TMP/SXT in purple, and to all three antibiotics in bold turquoise.



Supplemental Figure S7. Violin/box plot of abundancies of different resistant *E. coli* vs. total *E. coli* in fecal samples of women with and without UTI. A) Among CIP-R *E.* coli carriers; B) Among TMP/SXT-R *E.* coli carriers; C) Among 3GC-R *E.* coli carriers; D) Among carriers of different clonal groups of CIP-R *E. coli*. Sample size is shown in parenthesis. Mean abundance (Mean ± SEM%) is displayed above the plots. *P* values for statistically significant differences are indicated above the solid lines.

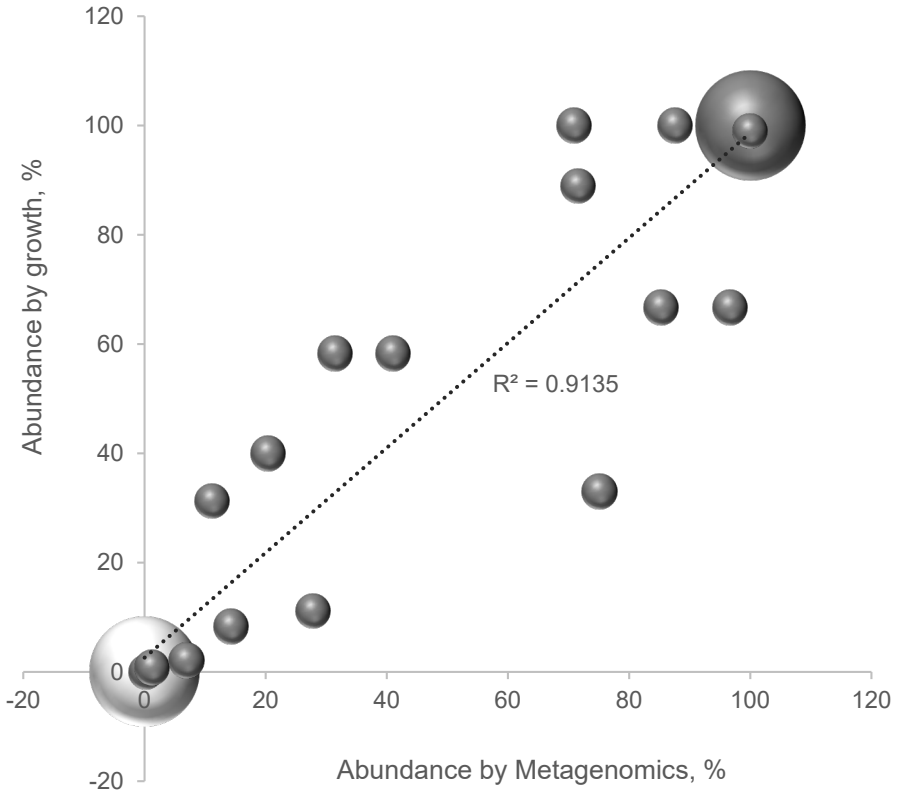
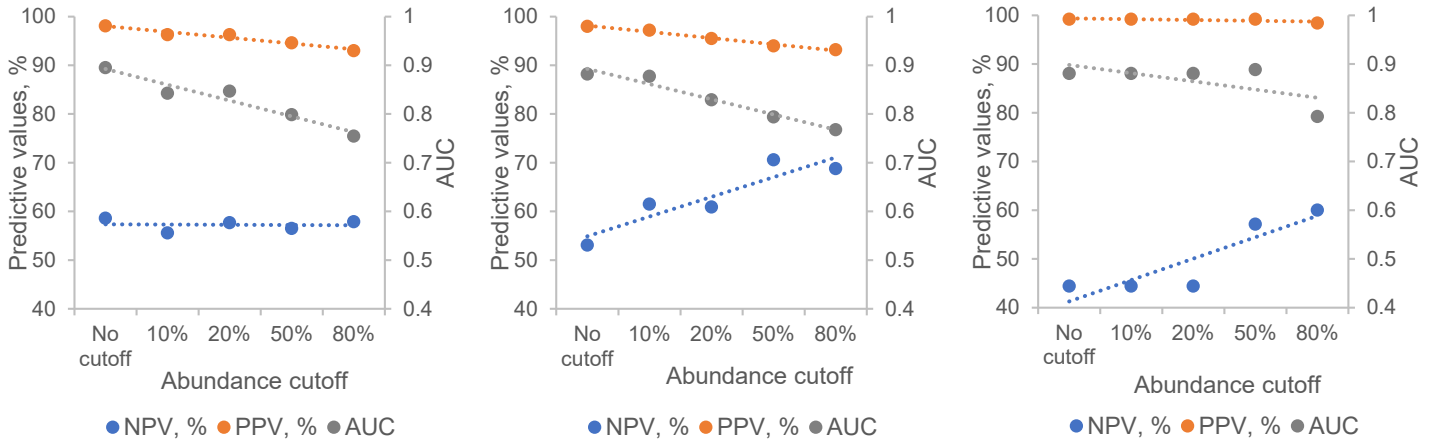
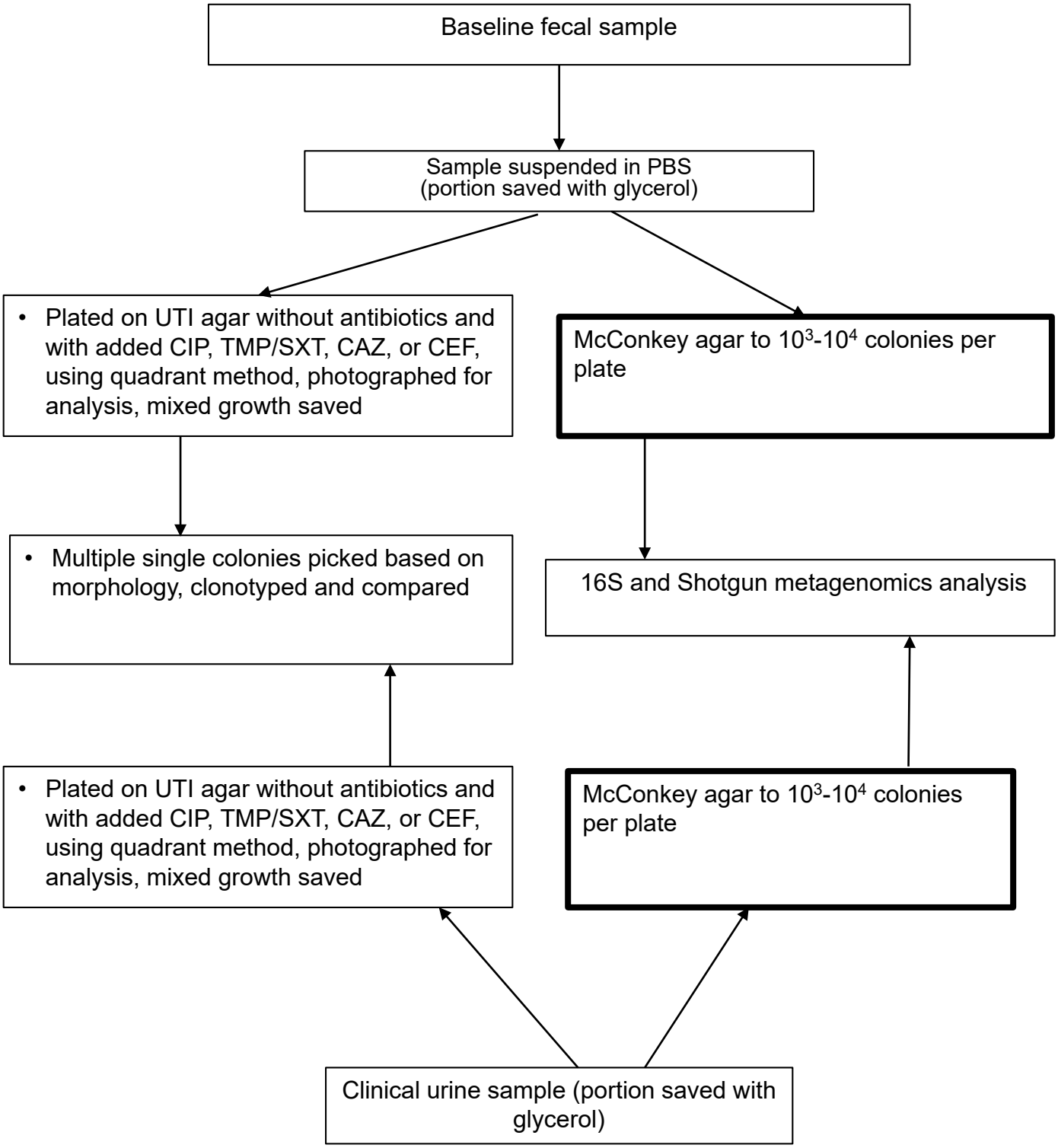


Figure S8. Correlation between prevalence of clones identified by growth analysis and Metagenomics analysis. For 36 fecal samples shotgun metagenomics was performed for pooled 1,000-10,000 single colonies grown on McConkey agar. UTI *E. coli* clone's abundance among all fecal *E. coli* within each sample was determined both from metagenomic analysis and culture data, and compared on the graph, with the bubble size reflecting number of samples. Light grey bubble indicates cases where urinary *E. coli* was not found either by metagenomic analysis or by culture. Trendline and R^2 value indicated the linear fit.



Supplemental Figure S9. Effect of abundance of resistant *E. coli* within fecal sample on prediction of uropathogen's antibiotic susceptibility. PPV (prediction of susceptibility to antibiotic), NPV (prediction of resistance to antibiotic) and AUC (overall test performance) was calculated for different cutoff of resistant *E. coli* prevalence within fecal samples (aka, abundance).



Supplemental Figure S10. Flowchart outlining the workflow for processing baseline fecal samples and subsequent clinical urine isolates, including sample collection, culturing, sequencing, and downstream genomic analyses.

Supplemental Methods

Fecal sample collection

Fecal samples were collected via self-collection kits. The kits mailed to potential enrollees included a culture swab and tube, biohazard bag with absorbent material inside, a piece of bubble padding, a shipping box, invitation letter, consent information sheet and detailed instructions. Instructions specified the importance of labeling the collected sample with the date of collection and mailing the sample as soon as possible. Samples were received by KPRWHI survey team and stored at 4^{o}C until picked up by UW processing team. For all samples the time lapse between collection date and processing date was on average 5.3±2.0 days, with 90% samples processed within a week from recorded sample collection.

Sample processing

The flow of sample processing steps is illustrated in Supplemental Figure S10. Fecal samples were self-collected using the FecalSwab™ Sample Collection and Preservation System for Enteric Bacteria by Copan Diagnostic Inc. (Carlsbad, CA, USA). At the processing start samples were visually assessed for the quality of fecal matter before being plated on four types of agar as described in our previous manuscript[1]. Pre-poured HardyCHROM™ UTI agar plates (Hardy Diagnostic, USA) were used for non-antibiotic plating of *E. coli*. The proprietary composition of these plates allows for the differential detection of uropathogenic microorganisms. For plating on ciprofloxacin, plates containing ciprofloxacin at 0.5, 2 or 10 mg/L were prepared using HiChromeTM UTI Agar (HiMedia Laboratories Pvt, Ltd., India). Sample was plated using standard quadrant plating technique[2]. The rest of the sample was split into two tubes (with and without 10% glycerol) and stored at -80^{o}C. The plates were incubated at 37°C for 16-20 hours, inspected visually, and the growth characteristics were documented including the quantitative growth level of potential *E. coli*, Gram-negative and total bacteria. Single colonies with *E. coli*-like morphology were isolated from all growth-positive CIP plates and a random selection of UTI plates for further analysis. The

mixed cultures were then preserved in 10% glycerol-containing freezer medium and stored at -80°C.

Quantification of relative *E. coli* and CIP-resistant *E. coli* abundance in fecal samples

As described above, the semi-quantitative quadrant streaking was performed on chromogenic UTI agar to estimate the relative abundance of *E. coli* (on plain agar plates) and CIP-resistant *E. coli* (on ciprofloxacin-supplemented plates) in fecal samples. Colony counts were taken from the final growth quadrant, where individual colonies could be clearly distinguished. Morphological characteristics provided by the manufacturer were used to differentiate *E. coli* from other Gram-negative and Gram-positive bacteria (see **Supplemental Figure S3A** for example). This approach allowed calculation of the proportion of *E. coli* colonies relative to the total Gram-negative or overall bacterial growth. The abundance of CIP-resistant *E. coli* relative to total *E. coli* was determined by comparing colony counts on ciprofloxacin-containing versus plain agar.

Identification of TMP/STX-R and 3GC-R *E. coli* in fecal samples

An aliquot of every fecal sample stored in glycerol was resuspended in 100 µL of Mueller-Hinton (MH) broth, incubated at 37°C for 2 hours, plated on UTI agar supplemented with ciprofloxacin (CIP, 0.5 mg/L), trimethoprim/sulfamethoxazole (TMP/STX, 4/76 mg/L), ceftazidime (CAZ, 8 mg/L), cefotaxime (CTX, 2 mg/L), and without antibiotics. After overnight incubation plates were processed same way as described above. Growth on UTI-CIP plate served as data reproducibility control. All potentially resistant *E. coli* colonies were subcultured on MH-agar supplemented with respective antibiotic to confirm their non-susceptibility status.

Clinical urine sample collection and processing

If a study participant submitted a urine sample to KPWA clinical laboratory, a routine urinalysis test was performed, followed by culture and sensitivity testing if required. Starting February 2022, the UW laboratory was provided an aliquot of the urine sample,

which was processed using the same protocol as fecal sample described above, with initial plating on plain and CIP-UTI plates. Potential *E. coli* were saved as at least 4-5 individual colonies and used for antibiotic susceptibility testing and identification of *E. coli* clonality (see below). The time lapse between the beginning of the study (May 2021) and the start of availability of clinical urine samples to the UW lab accounts for the lack of clonal information for some *E. coli*-caused UTIs.

Identification of *E. coli* clonality

E. coli clonality was determined by CH typing based on *fumC*/*fimH* sequencing[3], presence of QRDR mutations was determined by sequencing of *gyrA* and *parC*[4]. All reactions were carried out by 2-step colony PCR. Briefly, a single *E. coli* colony was resuspended in 50 µL of sterile water and heated at 98°C for 10 min. Primary PCR reactions were set up in a 15 µL volume using DreamTaq Mastermix (Thermofisher, USA), supplemented with 0.5 µM forward and reverse primers, and 1.5 µL of the boiled colony template. Primary PCR was run for 30 cycles under the manufacturer's recommended conditions. Subsequently, 1 µL of the PCR1 product was used for an additional PCR reaction, using nested forward and reverse primers supplemented with T7 and T7-Term tails, respectively. The nested PCR was run for 15 cycles under the same conditions, aiming to obtain a highly specific single band with T7-tailed primers suitable for downstream sequencing. The primer sequences can be found in **Supplemental Table S4**.

Testing antibiotic resistance of *E. coli* isolates

Resistance of fecal *E. coli* isolates to antibiotics of interest was performed using agar dilution method as described in CLSI manual [5]. Resistance of urinary *E. coli* isolates to a panel of 12 antibiotics was tested using Kirby-Bauer disk diffusion method as described in CLSI manual [5].

Metagenomic analysis

Sequencing.

A subset of 36 pairs of fecal and clinical urine samples was analyzed to assess the relative abundance of *E. coli* among enterobacteria, as well as the presence and prevalence of specific *E. coli* clones. Different dilutions of samples were plated on MacConkey agar to obtain 10^3-10^4 colonies per plate. Total growth was pooled and DNA was extracted from pooled colonies. DNA was used for shotgun metagenomic sequencing on an Illumina MiSeq platform using the MiSeq 600 cycle v3 kit, following the manufacturer's guidelines. Genomic DNA libraries were prepared with the Nextera XT Library Prep Kit (Illumina, CA). The raw reads were analyzed for species composition and urinary *E. coli* clone abundance as follows.

Determining species composition in sample.

Shotgun metagenomic reads were analyzed using the PATRIC Taxonomic Profiling Tool (https://patricbrc.org/). Raw reads were quality-checked with FastQC and trimmed using Trim Galore (Phred < 20). High-quality reads were processed through PATRIC, which employs Kraken2 for taxonomic classification and Bracken for abundance estimation. Taxonomic profiles, including relative species abundances, were generated and exported for downstream analysis.

Detection and quantification of urinary E. coli clone in samples.

The presence and abundance of the urinary *E. coli* clone in fecal samples were determined through comparative genomics and targeted read alignment. First, the sequenced urinary clone was compared to genomes of the same sequence type (ST) and different STs using both in-house sequenced isolates and publicly available genomes from EnteroBase (https://enterobase.warwick.ac.uk/). This analysis identified ST-specific alleles and isolate-specific SNPs unique to the clone of interest.

Raw sequencing reads were aligned to the identified alleles using BWA-MEM with default parameters. The resulting SAM file was converted to a sorted and indexed BAM file using SAMtools for efficient variant calling and read depth analysis. Variant calling was performed using BCFtools mpileup, with a maximum depth (-d 250), a minimum mapping quality (-q 60), and a minimum base quality (-Q 30), ensuring high-confidence variant detection. The bcftools call function was used in multiallelic (-m) and variant-only

(-v) mode, with a ploidy setting of 1 (haploid), to generate a compressed VCF file containing identified variants. The VCF file was then indexed and queried to extract depth of coverage (%DP) and allele frequencies (%AD) for each position of interest.

The output was manually examined for the presence of alleles unique to the strain of interest. The relative abundance of the strain was calculated as the proportion of sequencing reads supporting the unique SNPs, using the following formula:

$$Abundance\ (\%) = \sum RefReads\ (unique\ SNPs) / \sum (RefReads\ (unique\ SNPs) + AltReads\ (unique\ SNPs)) \times 100$$

If no unique SNPs were detected, the alignment process was repeated for other identified alleles to confirm the absence of the strain in the sample.

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1. Tchesnokova, V.*, et al.* Increase in the community circulation of ciprofloxacin-resistant Escherichia coli despite reduction in antibiotic prescriptions. *Commun Med (Lond)* **3**, 110 (2023).
2. Cappuccino, J.G.S., Natalie. *Microbiology: A Laboratory Manual*, (Pearson, New York, NY, 2021).
3. Weissman, S.J.*, et al.* High-resolution two-locus clonal typing of extraintestinal pathogenic *Escherichia coli*. *Appl Environ Microbiol* **78**, 1353-1360 (2012).
4. Johnson, J.R.*, et al.* Abrupt emergence of a single dominant multidrug-resistant strain of *Escherichia coli*. *J Infect Dis* **207**, 919-928 (2013).
5. CLSI. Performance standards for antimicrobial susceptibility testing; twentieth information supplement. Vol. 30 (2010).

Supplemental Tables

Table S1. Clonotype distribution of multidrug-resistant CIP-R *E. coli* isolated from fecal samples. ST, sequence type, CC, clonal complex, H, *fimH* allele, Q, number of QRDR mutations in *gyrA* and/or *parC* known to confer CIP-resistance; for ST69-H27(2Q) and ST38-H5(1Q) and ST38-H65(3Q) *gyrA* and *parC* allele numbers are listed after comma to indicate different CIP-R clones.

CIP-R E. coli Clone	No. CIPR-SC (%Total)	No. CIPR MDR SC (%Total)
H30	70 (20.3)	27 (18.4)
ST1193	68 (19.7)	29 (19.7)
ST131-H41(1Q)	15 (4.3)	8 (5.4)
ST69-H27, 14-76(2Q)	15 (4.3)	4 (2.7)
ST69-H27, 14-13(2Q)	12 (3.5)	6 (4.1)
ST648-H0(3Q)	9 (2.6)	5 (3.4)
ST69-H27(0Q)	8 (2.3)	3 (2)
ST69-H27(1Q)	6 (1.7)	3 (2)
ST69-H27(3Q)	5 (1.4)	3 (2)
ST38-H5(3Q)	4 (1.2)	3 (2)
ST10-H27(1Q)	4 (1.2)	2 (1.4)
ST354-H58(4Q)	3 (0.9)	3 (2)
ST636-H0(1Q)	3 (0.9)	3 (2)
ST394-H30(1Q)	3 (0.9)	2 (1.4)
ST95-H41(1Q)	3 (0.9)	1 (0.7)
ST38-H65, 11-14(3Q)	3 (0.9)	0 (0)
ST405-H27(3Q)	2 (0.6)	2 (1.4)
ST44(CC10)-H54(3Q)	2 (0.6)	2 (1.4)
ST6151(CC21)-H0(3Q)	2 (0.6)	2 (1.4)
ST773(CC10)-H0(3Q)	2 (0.6)	2 (1.4)
ST10-H54(4Q)	2 (0.6)	1 (0.7)
ST38-H65, 11-11(3Q)	2 (0.6)	1 (0.7)
ST457-H145(3Q)	2 (0.6)	1 (0.7)
ST648-H30(3Q)	2 (0.6)	1 (0.7)
ST6754(CC101)-H0(1Q)	2 (0.6)	1 (0.7)
ST69(3Q)	2 (0.6)	1 (0.7)
CC10-H27(3Q)	2 (0.6)	0 (0)
ST10-H215(3Q)	2 (0.6)	0 (0)
ST1177(CC38)-H65(2Q)	2 (0.6)	0 (0)
ST224(CC58)-H61(3Q)	2 (0.6)	0 (0)

ST38-H5, 14-36(1Q)	2 (0.6)	0 (0)
ST48(CC10)-H34(0Q)	2 (0.6)	0 (0)
ST93(CC10)-H30(3Q)	2 (0.6)	0 (0)
CC10-H54(3Q)	1 (0.3)	1 (0.7)
CC349-H54(1Q)	1 (0.3)	1 (0.7)
CH12-342(0Q)	1 (0.3)	1 (0.7)
ST10-H27(0Q)	1 (0.3)	1 (0.7)
ST10-H28(1Q)	1 (0.3)	1 (0.7)
ST117-H97(3Q)	1 (0.3)	1 (0.7)
ST127-H2(1Q)	1 (0.3)	1 (0.7)
ST12-H7(0Q)	1 (0.3)	1 (0.7)
ST131-H41(4Q)	1 (0.3)	1 (0.7)
ST1380(CC394)-H1057(0Q)	1 (0.3)	1 (0.7)
ST14-H27(1Q)	1 (0.3)	1 (0.7)
ST1844(CC111)-H38(0Q)	1 (0.3)	1 (0.7)
ST2006(CC58)-H61(3Q)	1 (0.3)	1 (0.7)
ST2197(CC10)-H23(3Q)	1 (0.3)	1 (0.7)
ST224(CC58)-H54(3Q)	1 (0.3)	1 (0.7)
ST362-H96(0Q)	1 (0.3)	1 (0.7)
ST38-H5, 294-36(1Q)	1 (0.3)	1 (0.7)
ST38-H54(1Q)	1 (0.3)	1 (0.7)
ST38-H65, 11-13(3Q)	1 (0.3)	1 (0.7)
ST393(CC69)-H54(3Q)	1 (0.3)	1 (0.7)
ST457-H145(0Q)	1 (0.3)	1 (0.7)
ST58-H1325(1Q)	1 (0.3)	1 (0.7)
ST617(CC10)-H29(3Q)	1 (0.3)	1 (0.7)
ST62-H44(0Q)	1 (0.3)	1 (0.7)
ST636-H27(1Q)	1 (0.3)	1 (0.7)
ST69-H25(1Q)	1 (0.3)	1 (0.7)
ST69-H27, 14-2(2Q)	1 (0.3)	1 (0.7)
ST73-H10(1Q)	1 (0.3)	1 (0.7)
ST95-H41(0Q)	1 (0.3)	1 (0.7)
ST95-H468(1Q)	1 (0.3)	1 (0.7)
ST95-H99(1Q)	1 (0.3)	1 (0.7)
CC10-H34(3Q)	1 (0.3)	0 (0)
CC2797-H1637(0Q)	1 (0.3)	0 (0)
CC349-H54(3Q)	1 (0.3)	0 (0)
CC58-H121(1Q)	1 (0.3)	0 (0)
CH12-1580(1Q)	1 (0.3)	0 (0)
CH14-27(0Q)	1 (0.3)	0 (0)
ST10-H0(0Q)	1 (0.3)	0 (0)
ST10-H28(0Q)	1 (0.3)	0 (0)
ST10-H30	1 (0.3)	0 (0)

ST10-H54(0Q)	1 (0.3)	0 (0)
ST10-H54(3Q)	1 (0.3)	0 (0)
ST1163	1 (0.3)	0 (0)
ST131-H41(3Q)	1 (0.3)	0 (0)
ST140(CC95)-H15(1Q)	1 (0.3)	0 (0)
ST1431(CC58)-H32(3Q)	1 (0.3)	0 (0)
ST155(CC58)-H0(0Q)	1 (0.3)	0 (0)
ST1723-H38(3Q)	1 (0.3)	0 (0)
ST206(CC77)-H0(0Q)	1 (0.3)	0 (0)
ST206(CC77)-H23(3Q)	1 (0.3)	0 (0)
ST216-H69(1Q)	1 (0.3)	0 (0)
ST2772(CC58)-H26(0Q)	1 (0.3)	0 (0)
ST2973-H31(2Q)	1 (0.3)	0 (0)
ST349-H54(0Q)	1 (0.3)	0 (0)
ST372-H1639(1Q)	1 (0.3)	0 (0)
ST3863(CC21)-H1632(0Q)	1 (0.3)	0 (0)
ST38-H0(3Q)	1 (0.3)	0 (0)
ST38-H30(3Q)	1 (0.3)	0 (0)
ST38-H5(0Q)	1 (0.3)	0 (0)
ST405-H29(3Q)	1 (0.3)	0 (0)
ST4267(CC58)-H54(0Q)	1 (0.3)	0 (0)
ST4305(CC10)-H0(2Q)	1 (0.3)	0 (0)
ST450-H34(3Q)	1 (0.3)	0 (0)
ST452-H0(0Q)	1 (0.3)	0 (0)
ST5150(CC38)-H65(2Q)	1 (0.3)	0 (0)
ST569-H5(1Q)	1 (0.3)	0 (0)
ST5869(CC3945)-H31(3Q)	1 (0.3)	0 (0)
ST58-H30(0Q)	1 (0.3)	0 (0)
ST58-H32(0Q)	1 (0.3)	0 (0)
ST59-H34(0Q)	1 (0.3)	0 (0)
ST68-H382(1Q)	1 (0.3)	0 (0)
ST69(0Q)	1 (0.3)	0 (0)
ST744(CC10)-H54(3Q)	1 (0.3)	0 (0)
ST744-H54(3Q)	1 (0.3)	0 (0)
ST80-H1(0Q)	1 (0.3)	0 (0)
ST8492(CC58)-H38(3Q)	1 (0.3)	0 (0)
ST906(CC58)-H32(0Q)	1 (0.3)	0 (0)
ST93(CC10)-H0(0Q)	1 (0.3)	0 (0)
ST969(CC625)-H115(1Q)	1 (0.3)	0 (0)
not determined	1 (0.3)	0 (0)
Total CIP-R SC	345	147

Table S2. UTI *E. coli* resistant to antibiotics.

ID	ET [a]	UTI *E. coli* clone [b]	Resistance [c]	CU=F [d]
10094	7	sample unavailable	TS	YES
10152	17	ST131-H30	CIP	**no**
10226	13	ST1193	CIP TS	YES
10273	2	sample unavailable	TS	YES
10571	5	sample unavailable	TS 3GC	YES
10860	6	sample unavailable	TS	**no**
10891	2	sample unavailable	CIP	YES
11186	14	ST69-H27	TS	YES
11212	14	ST963(CC38)-H26	3GC	YES
11255	9	ST5308(CC674)-H233	TS	YES
11403	4	sample unavailable	3GC	**no**
11595	17	ST73-H10	TS	YES
11921	7	sample unavailable	TS	YES
12118	18	ST404(CC14)-H27	TS	YES
12211	15	ST131-H30 (0 QRDR)	TS	YES
12247	2	ST131-H30	CIP TS 3GC	YES
12450	1	sample unavailable	CIP	YES
12736	9	ST131-H30	CIP	YES
12815	18	ST404(CC14)-H27	TS	YES
13026	7	ST131-H30	TS	YES
13168	11	ST69-H27(2Q)	CIP TS	YES
13561	16	ST131-H30	CIP	**no**
13792	9	ST131-H30	CIP	YES
13844	6	ST963(CC38)-H26	3GC	YES
14315	1	sample unavailable	CIP	YES
14440	4	sample unavailable	CIP	YES
14509	11	ST131-H30	CIP	YES
14737	3	sample unavailable	CIP	YES
15034	17	ST131-H41	TS	YES
15727	6	CC10-H54	CIP TS	YES
15823	13	ST69-H27	TS	**no**
15924	7	ST131-H30	CIP	YES
16419	17	ST1193	CIP TS	YES
16572	9	ST69-H27(2Q)	CIP	YES
16660	12	ST131-H30	CIP	YES
16663	17	ST131-H41	CIP	YES

[a] ET, time elapsed between UTI and fecal sample, in months.

[b] No sample – sample not available in UW laboratory, clonal typing not performed.

[c] Resistance of uropathogenic *E. coli* was determined in KPWA clinical laboratory and confirmed in UW laboratory if sample was available. CIP, ciprofloxacin, TS, trimethoprim/sulfamethoxazole, 3GC, 3rd generation cephalosporins (ceftazidime and/or ceftriaxone)

[d] CU=F, clinical uropathogenic *E. coli* clone was identified among fecal *E. coli* from the same participant

Table S3. Non-*E. coli* UTI uropathogens.

ID	ET [a]	Urinary pathogen [b]	Resistance [b]	Fecal *E. coli* Resistance
11653	3	*C. koseri* (sample unavailable)	none	CIP
12140	3	*K. pneumoniae* (sample unavailable)	none	none
10700	4	*K. pneumoniae* (sample unavailable)	none	none
12218	7	*E. faecalis*	none	none
11544	8	*K. pneumoniae* (sample unavailable)	none	none
15712	11	*C. freundii*	none	TMP/SXT
10430	11	*Klebsiella sp.*	none	none
13734	11	*K. pneumoniae*	none	none
15321	12	*P. mirabilis*	none	none
14115	14	*K. pneumoniae*	none	none
11764	14	*P. mirabilis*	none	none
12805	15	*K. pneumoniae*	none	none
13446	15	*P. mirabilis*	none	none
15189	16	*Klebsiella sp.*	none	TMP/SXT
12041	17	*K. pneumoniae* (sample unavailable)	none	none
10944	17	*Salmonella sp.*	none	none

[a] ET, time elapsed between UTI and fecal sample, in months.

[b] Both uropathogen's species and resistance were determined in KPWA clinical laboratory and confirmed in UW laboratory if sample was available.

Supplemental Table S4. Primers used for CH typing, *gyrA-parC* sequencing.

Test	Target	Primer name	Sequence	Ref
CH typing	**fumC: PCR1**	fumC-F	GCATCACAGGTCGCCAGCG	This study
		fumC-R	GTACGCAGCGAAAAAGATTC	
	fumC: Nested	fumC-F'-T7Pro	TAATACGACTCACTATAGGGGCGCTTCAAATTTGTTCGG	This study
		fumC-R'-T7Term	GCTAGTTATTGCTCAGCGGGTACGCAGCGAAAAAGATTC	
	fimH: PCR1	fimH-F	CTGTTTGCTGTACTGCTGATG	This study
		fimH-R	CCACAATAAACGGTAAGAGGAAT	
	fimH: Nested	fimH-F'-T7Pro	TAATACGACTCACTATAGGGACTGCTGATGGGCTGGTC	This study
		fimH-R'-T7Term	GCTAGTTATTGCTCAGCGGAGGAATTGGCACTGAACC	
Detection of QRDR SNPs	**gyrA: PCR1**	gyrA-F	CGACCTTGCGAGAGAAAT	This study
		gyrA-R	GTTCCATCAGCCCTTCAA	
	gyrA: Nested	gyrA-F'-T7Pro	TAATACGACTCACTATAGGGCGAGAGAAATTACACCG	This study
		gyrA-R'-T7Term	GCTAGTTATTGCTCAGCGGAGCCCTTCAATGCT	
	parC: PCR1	parC-F	CGATTGCCGCCTGAGCCACTT	This study
		parC-R	GCGAATAAGTTGAGGAATCAG	
	parC: Nested	parC-F'-T7Pro	TAATACGACTCACTATAGGGTGAGCCACTTCACGCA	This study
		parC-R'-T7Term	GCTAGTTATTGCTCAGCGGGAGGAATCAGAATTAA	