

## Vital Surveillances

## Prevalence and Genomic Characterization of Multidrug-Resistant *Salmonella enterica* Serovar Kentucky Sequence Type 198 Circulating — Beijing Municipality, China, 2016–2023

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### ABSTRACT

**Introduction:** Highly fluoroquinolone-resistant *Salmonella enterica* serovar Kentucky (*S. Kentucky*) of sequence type (ST) 198 has emerged as a global multidrug-resistant (MDR) clone, posing a threat to public health.

**Methods:** Whole genome sequencing and antibiotic susceptibility testing was used to characterize the population structure and evolutionary history of 54 *S. Kentucky* isolates recovered from food and human clinical cases in Beijing from 2016 to 2023.

**Results:** All 54 *S. Kentucky* ST198 isolates exhibited resistance to quinolones, carrying point mutations in the quinolone resistance-determining regions (*gyrA\_S83F* and *parC\_S80I*). Resistance to other antibiotics (folate pathway inhibitors, cepheims, aminoglycosides, phenicol, rifamycin, fosfomycin, macrolides, and tetracyclines), mediated by the *sul1*, *sul2*, *dfxA14*, *bla*<sub>CTX-M</sub>, *bla*<sub>TEM-1B</sub>, *aac(3)-Id*, *aadA2*, *aadA7*, *aph(3')-I*, *aph(3'')-Ib*, *rmtB*, *floR*, *arr-2*, *fosA*, *mph(A)*, and *tet(A)* genes, was also observed in different combinations. The Beijing *S. Kentucky* ST198 evolutionary tree was divided into clades 198.2-1 and 198.2-2, which were further differentiated into three subclades: 198.2-2A, 198.2-2B, and 198.2-2C. Compared with the extended-spectrum  $\beta$ -lactamase-encoding gene *bla*<sub>CTX-M-14b</sub> in 198.2-1, the co-existence of *bla*<sub>CTX-M-55</sub> and *bla*<sub>TEM-1B</sub>, as well as chromosomally located *qnrS1*, was detected in most 198.2-2 isolates, which showed more complex MDR phenotypes. *S. Kentucky* ST198 outbreak isolates derived from two predominant clonal sources: 198.2-1 with cgST236434 and 198.2-2A with cgST296405.

**Conclusions:** The *S. Kentucky* population in Beijing is genetically diverse, consisting of multiple co-circulating lineages that have persisted since 2016. Strengthening surveillance of food and humans will aid in implementing measures to prevent and control the spread of AMR.

Nontyphoidal *Salmonella enterica* infections, particularly those caused by antimicrobial-resistant strains, are a major public health concern (1). Notably, fluoroquinolone (FQ)-resistant *Salmonella* spp. and extended-spectrum  $\beta$ -lactamase (ESBL)-producing Enterobacteriaceae were listed by the World Health Organization in 2017 as high-priority pathogens posing the greatest threat to human health (2).

Resistance to these antimicrobials has been observed in *S. enterica* serotype Kentucky since the 2000s (3–5). Highly FQ-resistant *S. Kentucky* has become widespread in recent years (6), largely associated with the spread of ST198, which is the most common ST leading to human infections worldwide. Recently, a high prevalence of *S. Kentucky* ST198 with resistance to CIP and extended-spectrum cephalosporins (ESCs) was detected in human, environmental, and chicken samples in China (7–8).

In this study, we generated a genomic collection of 54 *S. Kentucky* ST198 strains recovered from an active surveillance system in Beijing over an 8-year period from 2016 to 2023. Our aim was to characterize the genetic features and antimicrobial resistance (AMR) profiles of *S. Kentucky* ST198 in Beijing.

### METHODS

#### Sample Collection and *Salmonella* Identification

Hospital-based active surveillance was conducted from 2010 onward in Beijing, China. From January 2016 to December 2023, a total of 41,742 diarrheal samples were collected from outpatients with gastroenteritis. Various food samples of animal origin were collected from retail outlets and supermarkets to screen for *Salmonella* spp. for food safety risk surveillance. All specimens were processed by routine

microbiologic and biochemical tests to identify *Salmonella*. The *Salmonella* Kentucky serotype was detected by a slide agglutination test using commercially available antisera (SSI Diagnostica, Denmark) according to the White-Kauffmann-Le Minor scheme.

### Antimicrobial Susceptibility Testing

Antimicrobial susceptibility testing of *Salmonella* strains was conducted using the broth microdilution method following the guidelines of the Clinical and Laboratory Standards Institute document (CLSI M100-S29:2019). Twenty-eight antimicrobial agents belonging to 12 categories were tested: ampicillin (AMP), ampicillin-sulbactam (AMS), amoxicillin-clavulanic acid (AMC), cefazolin (CFZ), cefoxitin (CFX), cefotaxime (CTX), ceftazidime (CAZ), cefepime (FEP), kanamycin (KAN), gentamicin (GEN), streptomycin (STR), amikacin (AMI), tetracycline (TET), doxycycline (DOX), minocycline (MIN), nalidixic acid (NAL), ciprofloxacin (CIP), levofloxacin (LEV), gemifloxacin (GMI), sulfisoxazole (Sul), trimethoprim-sulfamethoxazole (SXT), chloramphenicol (CHL), azithromycin (AZI), aztreonam (AZM), colistin (CT), polymyxin B (PB), imipenem (IMP) and meropenem (MEM). An MDR phenotype was defined as resistant to at least three classes of antibiotics.

### Whole-Genome Sequencing (WGS) and Genomic Analysis

DNA was extracted using a QIAamp DNA Mini Kit (Qiagen, Hilden, Germany). Genomic DNA concentration was determined by agarose gel electrophoresis and fluorometric analysis (Qubit 2.0). WGS was conducted using an Illumina PE150 platform with 100× coverage (Novogene Technology Co., Ltd, Beijing, China). Raw sequencing data were quality-checked, trimmed, and assembled *de novo* into a draft genome sequence using SPAdes 3.13.

Core genome multi-locus sequence typing (cgMLST) was performed using the BacWGSTdb service. AMR genes were screened using the NCBI AMRFinderPlus tool 3.1.1b ([https://ftp.ncbi.nlm.nih.gov/pathogen/Antimicrobial\\_resistance/AMRFinder/](https://ftp.ncbi.nlm.nih.gov/pathogen/Antimicrobial_resistance/AMRFinder/)). Whole genome single nucleotide polymorphism (wgSNP) analysis for all draft genomes was conducted using snippy pipeline v.4.4.5 with the reference strain *S. Kentucky* ST198 PU131 (GenBank ID: CP026327). Single nucleotide polymorphism (SNP)

distance matrices were obtained for all isolates using snp-dist v.0.6.3. The phylogenetic tree and heatmap of resistance genes were visualized using ChiPlot (<https://www.chiplot.online/>).

## Results

### Epidemiological Information for *S. Kentucky* ST198 Isolates from Beijing

Between 2016 and 2023, an active surveillance system collected 1,838 *S. enterica* strains, of which 54 (2.9%) were *S. Kentucky* isolates. All 54 *S. Kentucky* isolates were assigned to ST198. Forty-nine were collected from patients with clinical diarrhea, and five were isolated from chicken meat. Eight and 23 of the 54 strains were from two outbreaks in 2016 and 2020, respectively, while the remaining isolates were from sporadic cases. These 54 strains were isolated between 2016 and 2023, with the following annual distribution: eight in 2016, two in 2018, two in 2019, 23 in 2020, three in 2021, seven in 2022, and nine in 2023. The strains were distributed across 12 districts.

### Antibiotic Resistance and MDR Profiles

All 54 *S. Kentucky* isolates were resistant to quinolones (NAL, CIP, LEV, and GMI), tetracyclines (TET and DOX), folate pathway inhibitors (Sul), and susceptible to carbapenems (IMP and MEM). Regarding cepheims, *S. Kentucky* isolates showed the highest resistance to CFZ (90.7%), followed by CTX (87.0%), FEP (85.2%), and CAZ (63.0%). However, resistance to AMC, FOX, CT, and PB was relatively low, at only 1.9%. The frequency of AMR is presented in Figure 1. In addition, 85.2% (46/54) of ST198 isolates were ESBL-producing strains.

All 54 isolates (100%) were resistant to at least four antimicrobial classes. Resistance to six, eight, and 10 classes was observed in 94.4%, 83.3%, and 53.7% of isolates, respectively.

### Phylogenetic Analysis of Global *S. Kentucky* ST198 Isolates

To reveal the genetic relatedness of *S. Kentucky* ST198 isolates, a global phylogenetic tree was constructed using 121 isolates, including 15 global strains, 52 Chinese isolates obtained from 11 other Provincial-level administrative divisions (PLADs), and the 54 isolates from this study (Figure 2). As previously reported (9), the *S. Kentucky* ST198 isolates were

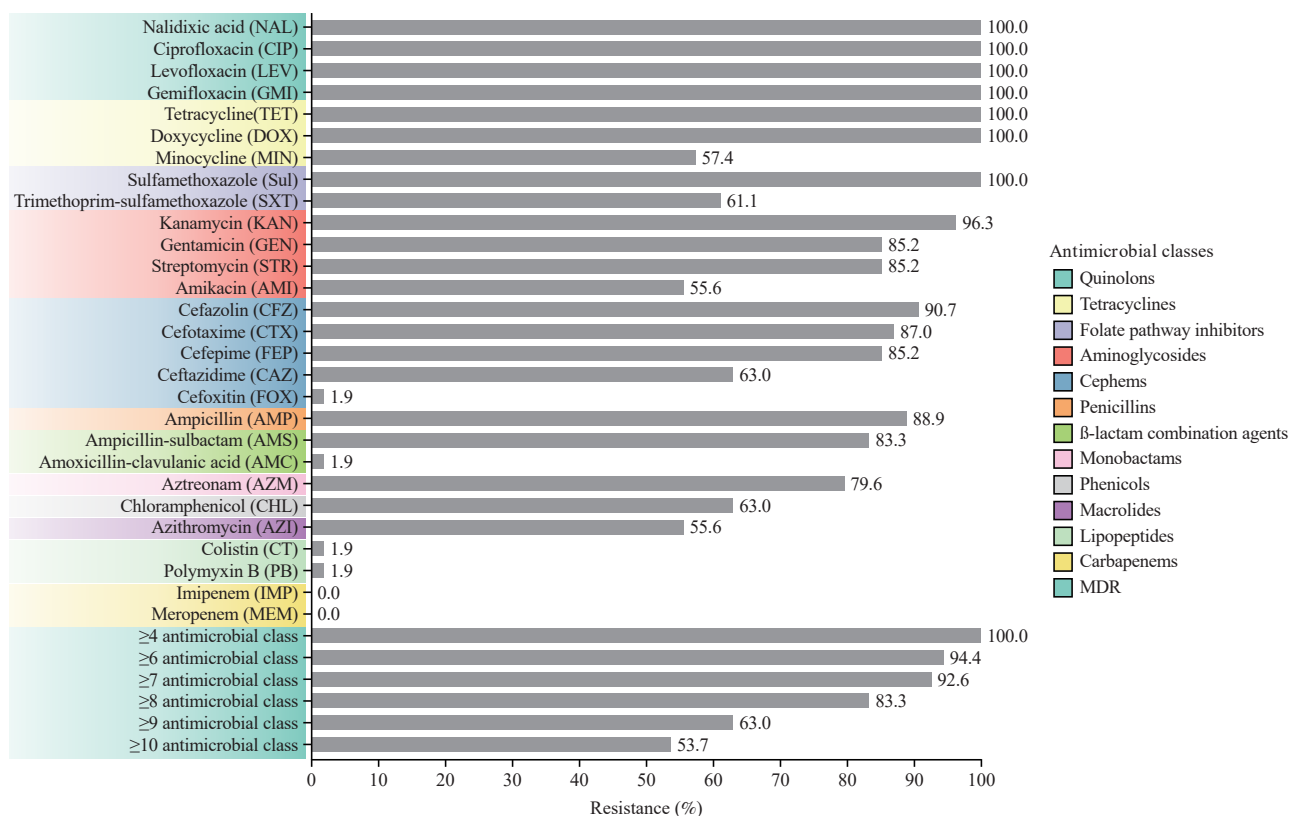


FIGURE 1. Antimicrobial resistance of 54 *S. Kentucky* isolates against 28 antimicrobial agents belonging to 12 categories. Abbreviation: *S. Kentucky*=*Salmonella Kentucky*; MDR=multidrug resistant.

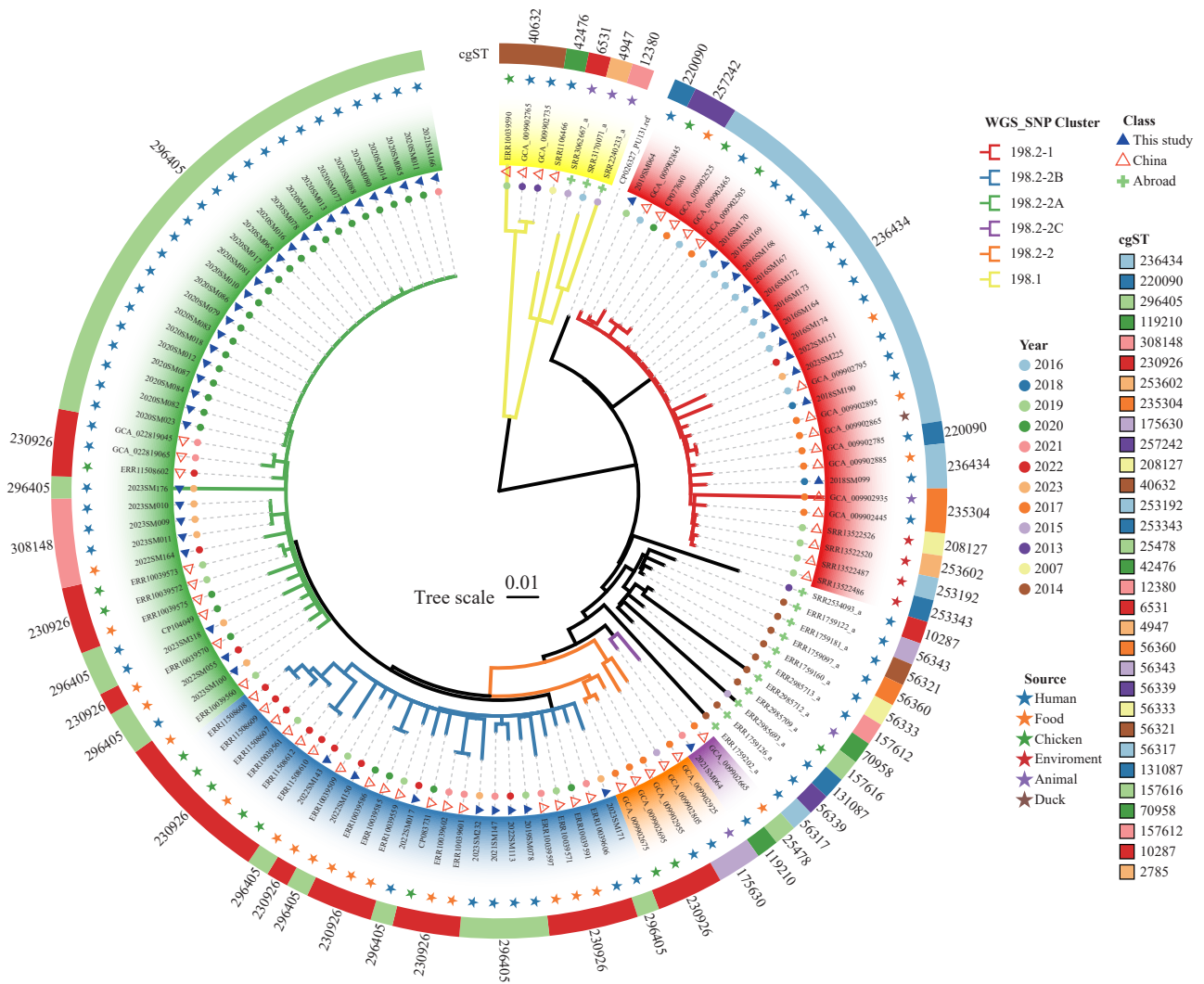
divided into two clades, 198.1 and 198.2. Clade 198.2 was further subdivided into two subclades, 198.2-1 and 198.2-2. The 15 global strains were mainly distributed in clades 198.1, 198.2-1, and 198.2-2. Although the Chinese isolates showed significant diversity, they were concentrated in clades 198.1 (3.3%, 4/121), 198.2-1 (24.0%, 29/121), and 198.2-2 (60.3%, 73/121). Furthermore, 30 subclade 198.2-1 isolates obtained from five provincial-level administrative divisions (PLADs), including Xichuan, Guangxi, Hunan, Jiangsu, and Zhejiang, were detected between 2013 and 2023, while 73 subclade 198.2-2 isolates obtained from nine PLADs (Anhui, Fujian, Guangdong, Guangxi, Guizhou, Jiangsu, Liaoning, Shandong, and Zhejiang) were collected from 2015 to 2022. Strains from various sources (animal, chicken, duck, environment, food, and human) clustered together, implying cross-transmission between hosts.

To reveal the molecular characteristics of *S. Kentucky* ST198 isolates, we performed cgMLST on the whole-genome sequences of the 121 global isolates and identified 31 cgSTs (Figure 2). The most common cgSTs were cgST296405 (30.6%), followed by cgST230926 (23.1%) and cgST236434 (15.7%).

Twenty-two cgSTs were represented by only one isolate, and cgST40632 was detected only in three clade 198.1 isolates. Among the 30 subclade 198.2-1 isolates, nine cgSTs were detected, with cgST236434 being the predominant type, accounting for 63.3% (19/30). By contrast, only six cgSTs were identified in 73 subclade 198.2-2 isolates, with cgST296405 accounting for 50.7% (37/73) and cgST230926 accounting for 38.4% (28/73).

### Diversity and Discrepancy of *S. Kentucky* ST198 Isolates from Beijing

Phylogenetic analysis of the 54 Beijing isolates was performed (Figure 3A), and a heatmap of SNPs was generated (Figure 3B). The number of SNPs ranged from 15 to 100 between food-derived isolates, from 0 to 103 between human-derived isolates, and from 9 to 102 between human- and food-derived isolates. Of the 54 Beijing isolates, 13 (24.1%), 32 (59.3%), 8 (14.8%), and 1 (1.9%) belonged to subclades 198.2-1, 198.2-2A, 198.2-2B, and 198.2-2C, respectively (Figure 3A). Thirteen isolates were distributed in subclade 198.2-1: one clustered with eight strains from outbreak 1 in 2016 (0 SNPs) in one branch, and the

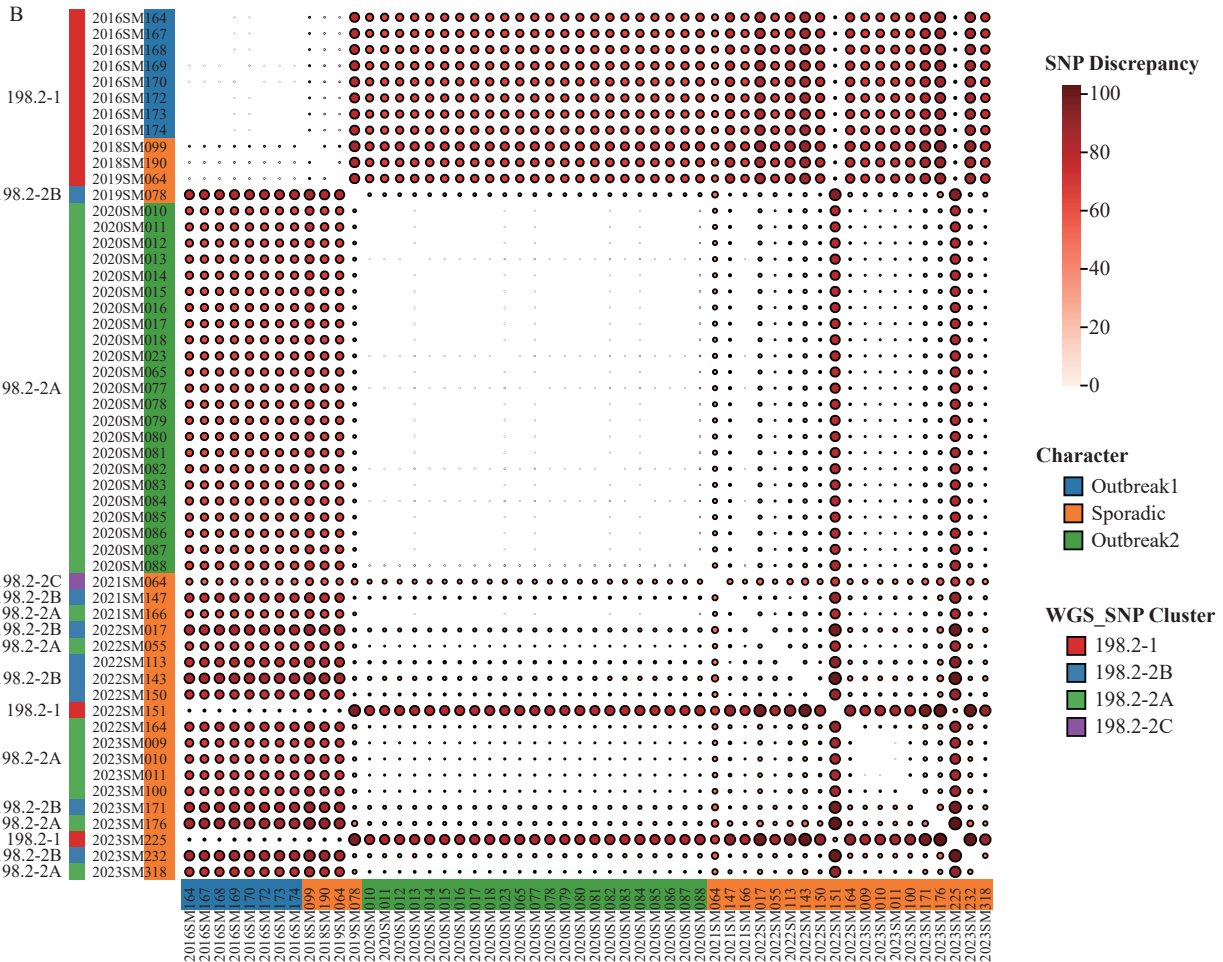
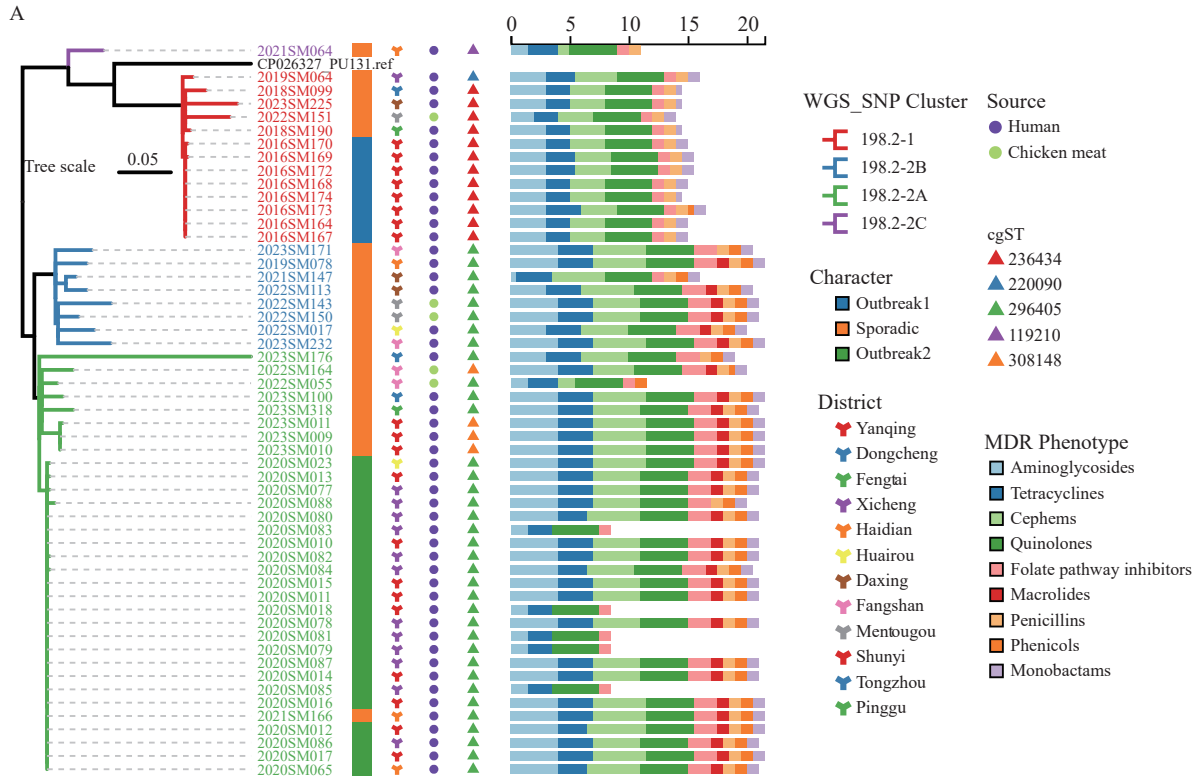


**FIGURE 2.** The phylogenetic relationship of the 121 global *S. Kentucky* ST198 isolates based on wgSNPs. Note: In the two outer circles, the cgSTs are shown in colored rings, and the sources of strains are shown in colored pentagrams. In the two inner circles, the isolation year of strains is indicated by colored dots, and strains from this study are shown as filled blue triangles, strains from China are shown as hollow red triangles, and global strains are shown as filled green crosses. The tree branches are color-coded to highlight *S. Kentucky* strains from clades 198.1, 198.2-1, 198.2-2, and sub-cluster 198.2-2 (2A-2C).

Abbreviation: *S. Kentucky*=*Salmonella Kentucky*; ST=sequence type; wgSNPs=whole genomic single nucleotide polymorphisms; cgST=core genomic sequence type.

other clustered with five sporadic strains from 2018 and 2019 (1–4 SNPs). In subclade 198.2-2A, 23 strains from outbreak 2 in 2020 clustered together (0–1 SNP), along with nine sporadic strains, including seven human isolates from 2021 and 2023, and two food isolates (chicken meat) from 2022 (1–5 SNPs). In subclade 198.2-2B, the majority of isolates ( $n=8$ ) clustered with six strains from humans (2019–2023) and two strains from food (2022) (1–4 SNPs). The remaining human isolate (2021SM064) in subclade 198.2-2C was distantly related to other Beijing isolates (22–31 SNPs).

The 54 Beijing isolates yielded five cgSTs: cgST296405 (66.7%), cgST236434 (22.2%), cgST308148 (7.4%), cgST220090 (1.9%), and cgST119210 (1.9%). The cgST296405 genotype was most prevalent, comprising 23 outbreak strains from 2020 and 13 sporadic strains from 2019 ( $n=1$ ), 2021 ( $n=2$ ), 2022 ( $n=5$ ), and 2023 ( $n=5$ ). The cgST236434 genotype included eight outbreak isolates from 2016 and four sporadic isolates from 2018 ( $n=2$ ), 2022 ( $n=1$ ), and 2023 ( $n=1$ ). These findings were consistent with SNP analysis, suggesting that cgST296405 and cgST236434 represent two major endemic genotypes in Beijing.



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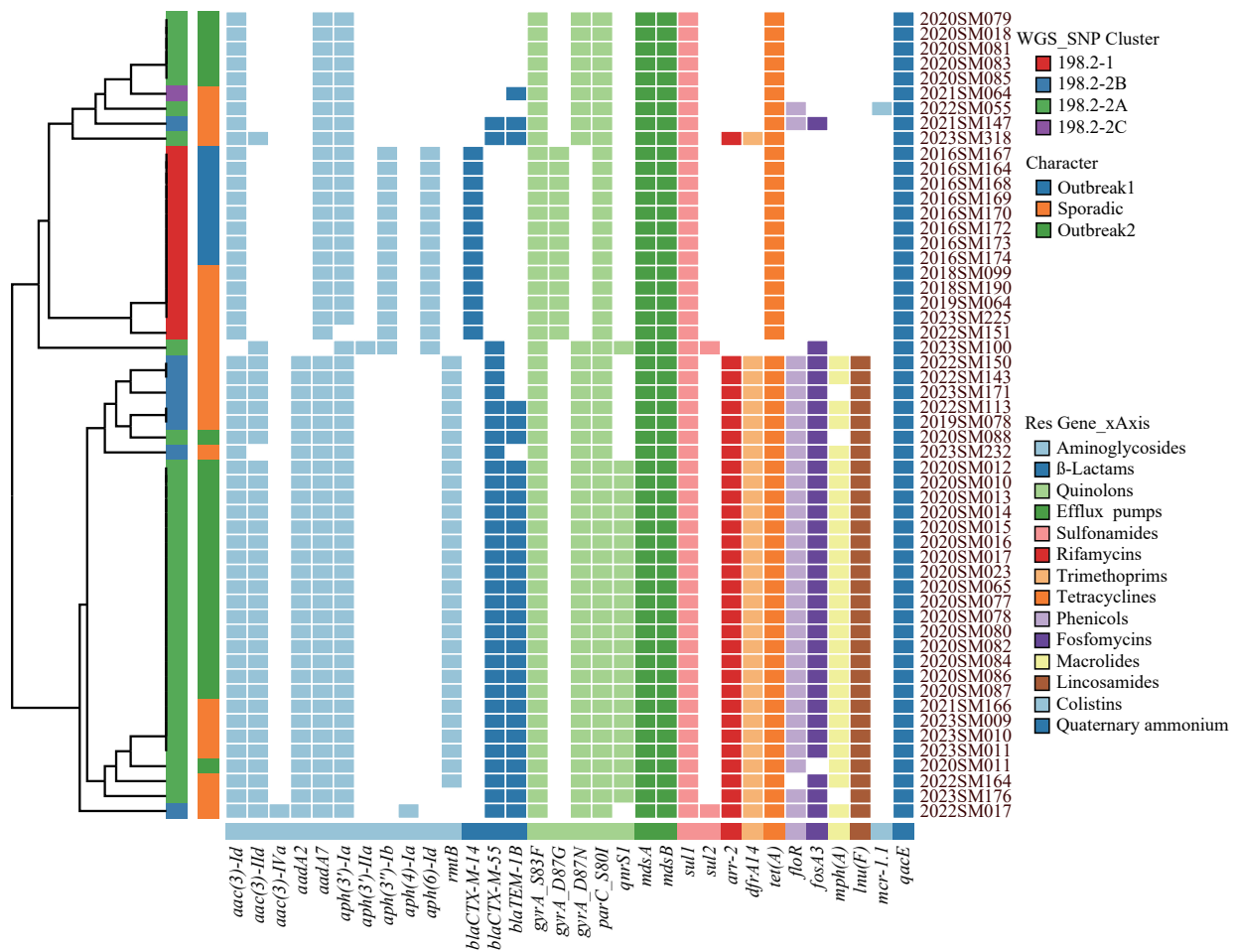


FIGURE 3. Phylogenetic analysis and the AMR phenotypic and genotypic characteristics of the 54 *S. Kentucky* isolates from Beijing. (A) The phylogenetic relationships, epidemiological and molecular features, and the MDR phenotypes of the 54 *S. Kentucky* isolates. (B) The matrix of SNPs for the 54 *S. Kentucky* isolates shows all correlations between outbreak and sporadic strains, as well as the various sources. (C) A heatmap of AMR genes. The color blocks on the X-axis represent the categories of genes. Colored cells represent the presence of genes, and white cells represent the absence of genes.

Note: The size of the circle represents the number of SNP differences between two isolates: the larger the circle, the greater the number of SNP differences, and no circle indicates no difference (0 SNPs).

Abbreviation: AMR=antimicrobial resistance; *S. Kentucky*=*Salmonella Kentucky*; MDR=multidrug resistant; SNP=single nucleotide polymorphism.

### Resistomes of *S. Kentucky* ST198 Isolates from Beijing

To characterize the AMR profiles of the *S. Kentucky* ST198 isolates from Beijing, we scanned the genomes to identify AMR-related genes and mutations. We detected 32 AMR genes in 14 classes, including those involved in resistance to aminoglycosides (11 genes),  $\beta$ -lactams (3 genes), quinolones (1 gene and 4 point mutations), efflux pumps (2 genes), sulfonamides (2 genes), rifamycins (1 gene), trimethoprim (1 gene), tetracycline (1 gene), phenicol (1 gene), fosfomycin (1 gene), macrolides (1 gene), lincosamides (1 gene),

colistins (1 gene), and quaternary ammonium (1 gene) (Figure 3C). Resistance genes from seven classes were found in strains within clade 198.2-1, while 14 classes were found in strains within clade 198.2-2.

The AMR gene profiles and phenotypes differed between the subclade 198.2-1 and 198.2-2 strains (Figure 3A and 3C). For  $\beta$ -lactamase resistance genes, *bla*<sub>CTX-M-14b</sub> was the only gene detected among the 13 subclade 198.2-1 isolates. However, in subclade 198.2-2, *bla*<sub>CTX-M-55</sub> and *bla*<sub>TEM-1B</sub> were detected in 82.9% (34/41) and 73.2% (30/41) of isolates, respectively. For the aminoglycoside resistance gene profiles, *aac(3)-Id*, *aadA7*, and *aph(3')-Ia* genes were



involved eight human-derived strains belonging to phylogenetic lineage 198.2-1 and cgST236434 (Figure 2, Figure 4). The second outbreak, occurring in four districts (Xicheng, Yanqing, Haidian, and Huairou) in 2020, involved 23 human-derived strains grouped in lineage 198.2-2A and cgST296405, suggesting cross-regional transmission and clonal expansion during this outbreak. The remaining 23 sporadic cases, distributed across 11 districts (Daxing, Docheng, Fangshan, Fengtai, Haidian, Huairou, Mentougou, Pinggu, Shunyi, Tongzhou, and Xicheng) between 2018 and 2023, included 18 human-derived and five chicken meat-derived strains. These sporadic strains derived from a polyclonal evolutionary source (198.2-1, 198.2-2A, 198.2-2B, and 198.2-2C) and formed five cgMLST groups. The five chicken meat isolates, distributed across two districts in 2022, were mainly from lineages 198.2-1, 198.2-2A, and 198.2-2B and were closely related to human isolates. These results suggest that the *S. Kentucky* ST198 outbreak isolates have two predominant clonal sources: 198.2-1 with cgST236434 before 2019 and 198.2-2A with cgST296405 after 2019.

## DISCUSSION

*S. Kentucky* ST198 has been increasingly reported in chickens and can cause human infections in China (8–9). In this study, all 54 *S. Kentucky* isolates were assigned to ST198. Our findings showed that the positive ratio for *S. Kentucky* ST198 among *S. enterica* isolates was 2.9% (54/1,838) in Beijing, which is approximately fourfold higher than that reported in a previous study (0.39%, 40/16,247) (9) and much higher than that reported in Shenzhen (0.7%, 57/8,559) (10) and among patients (0.33%, 40/12,011) in China between 2010 and 2016 (8).

In the present study, genomic analyses (cgMLST and wgSNP) were conducted to characterize the *S. Kentucky* ST198 strains. Five cgSTs were identified, with cgST296405 and cgST236434 being the predominant genotypes causing outbreaks. Phylogenetic analysis indicated that the 54 *S. Kentucky* strains clustered into lineages 198.2-1 and 198.2-2, then further divided into three sublineages, 198.2-2A, 198.2-2B, and 198.2-2C, revealing the existence of multiple lineages circulating in Beijing. Additionally, many sublineages were shared by strains from this study and strains from other PLADs, such as Guangxi, Shandong, Fujian, and Zhejiang, indicating a potential phylogenetic relationship between these strains.

In view of the increasing public health threat posed by the global emergence and dissemination of antimicrobial-resistant *S. Kentucky*, this investigation revealed a high level of both phenotypic and genotypic resistance to different classes of antimicrobials among 54 *S. Kentucky* isolates. The isolates were resistant to ESCs as follows: CTX (87.0%, 47/54), CAZ (63.0%, 34/54), and FEP (85.2%, 46/54). Resistance to ESCs is normally mediated by the production of ESBLs, and CTX-M-type ESBLs pose a particularly serious public health threat worldwide (11). In this study, all Beijing isolates in subclade 198.2-1 carried the gene *bla*<sub>CTX-M-14b</sub>, consistent with previous studies in China (6–7,9,12), indicating the formation of a clade carrying *bla*<sub>CTX-M-14b</sub> in China. In addition, compared with *bla*<sub>CTX-M-14b</sub> in 198.2-1, the co-existence of *bla*<sub>CTX-M-55</sub> and *bla*<sub>TEM-1B</sub> was detected in the majority of 198.2-2 isolates. It is worth mentioning that all *S. Kentucky* isolates in this study were resistant not only to CIP but also to other FQ antibiotics (LEV, GMI), showing substantially higher levels of resistance than previously reported for *S. Kentucky* in China or many other countries (6,8,13). Moreover, all strains carried two quinolone resistance-determining region mutations (*gyrA*\_S83F and *parC*\_S80I), but *qnrS1* and *gyrA*\_D87N were only detected in 198.2-2 isolates. Several studies reported the presence of the *qnrS1* gene on the bacterial chromosome, along with a large number of chromosomally-located resistance genes, after 2017. The emergence of *qnrS1* in *S. Kentucky* isolates with *gyrA* and *parC* mutations increased bacterial resistance to CIP (10,12). In the present study, up to 85.2% of isolates were both FQ-resistant and ESBL-producing, confirming these as common features of *S. Kentucky*. Our study further revealed that the acquisition of an MDR phenotype by 198.2-2 has potentially contributed to its higher prevalence compared with 198.2-1, making it the most prevalent subclade for clonal transmission in Beijing.

In conclusion, the human salmonellosis epidemic caused by *S. Kentucky* involved multiple native circulating lineages that have become widely distributed across Beijing districts since 2016. Many clinical isolates were genetically clonal to strains isolated from food, suggesting possible cross-host transmission. Increased genome sequencing of strains from various host sources will facilitate the identification of transmission routes and determine potential ongoing outbreaks, which is vital for formulating targeted surveillance and countermeasures.

**Conflicts of interest:** No conflicts of interest.



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