Klebsiella pneumoniae is a non-motile, Gram-negative bacterium that typically exists as a commensal resident of either the human gastrointestinal tract or the nasopharynx (1). K. pneumoniae is also a significant cause of human disease, particularly in immunocompromised individuals and patients in long-term-care facilities (2). Primarily an opportunistic pathogen, K. pneumoniae can cause a variety of infections, including ventilator-associated pneumonias, catheter-associated urinary tract infections, wound infections, septicaemia, and meningitis (3). Of particular concern is the observation that K. pneumoniae clinical isolates are increasingly resistant to multiple antibiotics. Acquisition of plasmids encoding extended-spectrum beta-lactamases has resulted in resistance to many commonly used antibiotics (4). In addition, the emergence of carbapenem-resistant strains has been observed worldwide. Carbapenem-resistant K. pneumoniae strains are resistant to nearly all available antimicrobial agents, and infections result in high rates of morbidity and mortality (5).

K. pneumoniae is the second most common cause of urinary tract infections after Escherichia coli, often due to the use of indwelling catheters (6). K. pneumoniae strain TOP52 #1721, hereafter referred to simply as Top52, was isolated from the urine of a 26-year-old woman with acute cystitis (7). After successful completion of antibiotic therapy and negative follow-up urine cultures, the patient developed recurrent cystitis with the same strain of K. pneumoniae as determined by restriction fragment length polymorphism (RFLP) analysis (8). Subsequent characterization of Top52 found that the organism is able to form intracellular bacterial communities and colonize the urinary tracts of mice, much like uropathogenic E. coli, but at lower rates (8). This is likely due to both decreased expression of the type 1 pili and variations in the fimbrial adhesion, FimH, that alter protein function (8). More recently, Top52 has also been used in a catheter-associated urinary tract infection (UTI) model of K. pneumoniae infection where type 1 and type 3 fimbrial mutants were found to be at a disadvantage for colonizing the urinary tracts of catheterized mice (9).

K. pneumoniae Top52 was grown overnight at 37°C on Luria broth (LB) agar, and genomic DNA was isolated using the Qiagen DNeasy Blood and Tissue kit (Qiagen, Valencia, CA). Genomic DNA was then fragmented to an average size of 500 bp using a Covaris M220 (Covaris, Woburn, MA). Fragments were subsequently size selected by Pippin Prep (Sage Science, Beverly, MA), and whole-genome libraries were made using the Accel-NGS 2S DNA Library Kit (Swift Biosciences, Ann Arbor, MI). These libraries were then sequenced on the Illumina MiSeq (Illumina, San Diego, CA) using MiSeq Reagent Kit v2. Approximately 4 million FASTQ reads were used for de novo genome assembly using MIRA 4 (10), which resulted in 2,959,766 reads assembled into 85 contigs with a total consensus of 5.4 Mb (average total coverage of 83×, largest contig of 482 kb and N50 of 299 kb). A BLAST search confirmed that 45 contigs map to the chromosome and 40 are plasmid contigs.

**Nucleotide sequence accession numbers.** This whole-genome shotgun project has been deposited at DDBJ/EMBL/GenBank under the accession no. JNFE00000000. The version described in this paper is version JNFE01000000.

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**REFERENCES**


