Genome Sequence of *Stenotrophomonas maltophilia* S028, an Isolate Harboring the AmpR-L2 Resistance Module

Shipping Song,^a Xitong Yuan,^b Shiwei Liu,^c Ning Zhang,^c Yufei Wang,^b Yuehua Ke,^b Jie Xu,^b Liuyu Huang,^b Zeliang Chen,^b and Yan Li^a

PLA 307 Hospital, Academy of Military Medical Science, Beijing, People's Republic of China^a; Department of Infectious Disease Control, Beijing Institute of Disease Control and Prevention, Beijing, People's Republic of China^b; and Wangjing Hospital, Chinese Academy of Traditional Medicine, Beijing, People's Republic of China^c

Multidrug-resistant *Stenotrophomonas maltophilia* has emerged as an important cause of nosocomial infections, which is attributable mainly to the production of diverse β -lactamases by *S. maltophilia*. The L2 β -lactamase mediated by the AmpR-L2 module is the most represented lactamase. Here, we announce the genome sequence of S028, an isolate harboring the AmpR-L2 module.

Stenotrophomonas maltophilia is a common commensal that is readily isolated from water, soil, and sewage. *S. maltophilia* is an important opportunistic pathogen in immunocompromised hosts and causes hospital-acquired as well as community-acquired infections (6). The respiratory tract is the most common site of *S. maltophilia* infection, especially in patients with cystic fibrosis and those with compromised lung function (3). Clinical investigations showed that the increase in the incidence of *S. maltophilia* infections might be a consequence of selective pressure caused by overuse of broad-spectrum β -lactams (9). Similar to many other nonfermentative bacteria, *S. maltophilia* isolates are intrinsically resistant to many commonly used antimicrobial agents. Over the past 2 decades, infections due to multidrug-resistant *S. maltophilia* have emerged as important nosocomial infections worldwide (2). This is in part attributable to the increasing number of immunocompromised patients as well as the increasing use of broad-spectrum β -lactam agents. The β -lactamase production is a major mechanism causing bacterial resistance to β -lactam antibiotics. *S. maltophilia* produces two types of β -lactamase, L1, a zinc-dependent metalloenzyme, and L2, a cephalosporinase of class A lactamase. Of the two β -lactamases, L2 is the most represented one and exists in most clinical resistant isolates. The L2 β -lactamase is chromosomally encoded and located adjacent to AmpR, a regulator of the LysR regulator family, forming the AmpR-L2 module (5, 8). The promoters of the AmpR and L2 β -lactamase genes exhibit a face-to-face architecture. The intergenic region is diverse in sequence, which might result in differential regulation of the resistance. In an AmpR-L2 intergenic polymorphism screening, we found that the polymorphism is closely related with resistance profiles. Here, we announce a genome sequence of a multidrug-resistant *S. maltophilia* isolate of S028, a representative strain with an AmpR-L2 module.

The genome of *S. maltophilia* S028 was sequenced with a HighSeq 2000 Sequencer with paired-end protocol. The low-quality reads were filtered, and the remaining reads were assembled with Clc bio genomics workbench version 5.5 by the *de novo* assembly method. After filtering of low-quality reads, the remaining reads were assembled into contigs. A total of 297 contigs covering a total of 3,754,755 bp were generated. The average length of contigs was 12.6 kb. The final approximate coverage for these contigs was about 110 $\times$.

Then, the genome sequence was annotated. Open reading frames (ORFs) were predicted by the RAST (1). The rRNA was predicted by using RNAmmer (4), and tRNA were identified with tRNAscan-SE 1.21 (7). The total genome has a G+C content of

67.07%. AmpR-L2 was identified in one of the contigs. The genome sequence of S028 makes it possible for comparative and functional genomics analysis of resistant *S. maltophilia* isolates. Further detailed analysis, including functional annotations, comparative genomics, and a regulation mechanism at the genome scale, are in process and will be included in our future publication.

Nucleotide sequence accession numbers. This whole-genome shotgun project has been deposited at DDBJ/EMBL/GenBank under the accession number [ALYK00000000](#). The version described in this paper is the second version, ALYK02000000.

ACKNOWLEDGMENTS

This work was supported by the National Natural Science Foundation of China (81071399, 31000548, 31000041, 81071320), the National Basic Research Program of China (grant no. 2009CB522602), and the National Key Program for Infectious Diseases of China (2008ZX10004-015, 2009ZX10004-103, 2008ZX10004-008).

REFERENCES

1. Aziz RK, et al. 2008. The RAST Server: rapid annotations using subsystems technology. *BMC Genomics* 9:75.
2. Brooke JS. 2012. *Stenotrophomonas maltophilia*: an emerging global opportunistic pathogen. *Clin. Microbiol. Rev.* 25:2–41.
3. Denton M, Kerr KG. 1998. Microbiological and clinical aspects of infection associated with *Stenotrophomonas maltophilia*. *Clin. Microbiol. Rev.* 11:57–80.
4. Lagesen K, et al. 2007. RNAmmer: consistent and rapid annotation of ribosomal RNA genes. *Nucleic Acids Res.* 35:3100–3108.
5. Lin CW, et al. 2009. The role of AmpR in regulation of L1 and L2 β -lactamases in *Stenotrophomonas maltophilia*. *Res. Microbiol.* 160:152–158.
6. Looney WJ, et al. 2009. *Stenotrophomonas maltophilia*: an emerging opportunist human pathogen. *Lancet Infect. Dis.* 9:312–323.
7. Lowe TM, Eddy SR. 1997. tRNAscan-SE: a program for improved detection of transfer RNA genes in genomic sequence. *Nucleic Acids Res.* 25:955–964.
8. Okazaki A, Avison MB. 2008. Induction of L1 and L2 β -lactamase production in *Stenotrophomonas maltophilia* is dependent on an AmpR-type regulator. *Antimicrob. Agents Chemother.* 52:1525–1528.
9. Safdar A, Rolston KV. 2007. *Stenotrophomonas maltophilia*: changing spectrum of a serious bacterial pathogen in patients with cancer. *Clin. Infect. Dis.* 45:1602–1609.

Received 23 September 2012 Accepted 1 October 2012

Address correspondence to Zeliang Chen, zeliangchen@yahoo.com, and Yan Li, liyanmdhuxi@163.com.

S.S., X.Y., S.L., N.Z., and Y.W. contributed equally to this work.

Copyright © 2012, American Society for Microbiology. All Rights Reserved.

[doi:10.1128/JB.01809-12](https://doi.org/10.1128/JB.01809-12)