Genome Sequence of *Staphylococcus epidermidis* Strain AU12-03, Isolated from an Intravascular Catheter

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In recent years, *Staphylococcus epidermidis* has become a major nosocomial pathogen and the most common cause of intravascular catheter-related bacteremia, which can increase morbidity and mortality and significantly affect patient recovery. We report a draft genome sequence of *Staphylococcus epidermidis* AU12-03, isolated from an intravascular catheter tip.

The staphylococci are able to cause a variety of infections ranging from minor skin infections to life-threatening bacteremia, and in spite of great efforts to control their spread, they persist as a major cause of hospital-acquired infections worldwide (4). *Staphylococcus epidermidis* is a member of the staphylococci and has become the leading cause of infection related to indwelling medical devices, such as intravascular catheters, prosthetic joints, and artificial heart valves (2). Previous reports have identified *S. epidermidis* as having the ability to colonize medical device surfaces, produce antimicrobials, and form biofilms (2, 8).

S. epidermidis AU12-03 was isolated from an intravascular catheter tip by rolling a catheter tip back and forth on the surface of a Columbia agar plate supplemented with 5% sheep blood essentially as described by Maki et al. (6). The genome sequence of S. epidermidis AU12-03 was determined on a 454 GS FLX system (Roche) (7). The sequence data consist of 125,924,967 bp of DNA sequence at 51× coverage. A total of 50 contigs (>500 bp) were *de novo* assembled using the GS De Novo assembler (version 2.3; Roche). The contig N_{50} was 81,766 bp, and the largest contig assembled was 210,685 bp. The contigs were then ordered and oriented into nine scaffolds using paired-end information. The average length of the scaffolds was 271,213 bp. Automatic genome annotation was performed on the RAST server (1).

The draft genome of S. epidermidis AU12-03 consists of a circular 2,440,923-bp chromosome with a G+C content of 31.98%. The genome contains 58 tRNA genes coding for all amino acids and 2,248 predicted protein-coding genes, consistent with other staphylococci (3, 5). We identified numerous putative virulence factors. S. epidermidis AU12-03 carried some predicted proteincoding genes involved in adhesion (cell wall-associated fibronectin-binding protein, elastin-binding protein, the bifunctional autolysin Atl, and the virulence-associated cell wall-anchored protein LPXTG motif binding to squamous nasal epithelial cells) and regulators involved in quorum sensing and biofilm formation (the staphylococcal accessory regulator SarA, the RNA polymerase sigma factor SigB, the bifunctional autolysin Atl, the antiadhesin Pls, the teicoplanin resistance-associated HTH-type transcriptional regulator TcaR, and the transcriptional regulator of biofilm formation AraC/XylS family). A range of genes related to bacteriocins and invasion were found. As expected, we also identified putative antibiotic resistance determinants (resistance to fluoroquinolone and beta-lactam antibiotics).

In short, the genomic information suggests that *S. epidermidis* AU12-03 may form biofilms on intravascular catheter surfaces that are resistant to a variety of antibiotics. As *S. epidermidis* species are the most prevalent cause of intravascular catheter-related bacteremia, characterization of virulence factors across a wide range of isolates may be important in elucidating the molecular principles of catheter-related infections and may provide insight into the genetic factors supporting pathogenesis.

Nucleotide sequence accession number. The genome sequence of *Staphylococcus epidermidis* AU12-03 has been deposited in NCBI GenBank under accession no. AMCS00000000.

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REFERENCES

- 1. Aziz RK, et al. 2008. The RAST server: rapid annotations using subsystems technology. BMC Genomics 9:75.
- 2. Fey PD, Olson ME. 2010. Current concepts in biofilm formation of *Staphylococcus epidermidis*. Future Microbiol. 5:917–933.
- 3. Gill SR, et al. 2005. Insights on evolution of virulence and resistance from the complete genome analysis of an early methicillin-resistant *Staphylococcus aureus* strain and a biofilm-producing methicillin-resistant *Staphylococcus epidermidis* strain. J. Bacteriol. **187**:2426–2438.
- Huebner J, Goldmann DA. 1999. Coagulase-negative staphylococci: role as pathogens. Annu. Rev. Med. 50:223–236.
- 5. Jiang S, et al. 2012. Whole-genome sequence of *Staphylococcus hominis*, an opportunistic pathogen. J. Bacteriol. **194**:4761–4762.
- Maki DG, Weise CE, Sarafin HW. 1977. A semiquantitative culture method for identifying intravenous catheter-related infections. N. Engl. J. Med. 296:1305–1309.
- 7. Margulies M, et al. 2005. Genome sequencing in microfabricated highdensity picolitre reactors. Nature 437:376–380.
- 8. Mermel LA, et al. 2009. Clinical practice guidelines for the diagnosis and management of intravascular catheter-related infection: 2009 update by the Infectious Diseases Society of America. Clin. Infect. Dis. **49**:1–45.

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