

Genome Sequence of *Staphylococcus arlettae* Strain CVD059, Isolated from the Blood of a Cardiovascular Disease Patient

Vasudevan Dinakaran,^a Manoharan Shankar,^a Sathyanarayanan Jayashree,^a Andiappan Rathinavel,^b Paramasamy Gunasekaran,^a and Jeyaprakash Rajendhran^a

Department of Genetics, Center for Excellence in Genomic Sciences, School of Biological Sciences, Madurai Kamaraj University, Madurai, Tamilnadu, India,^a and Department of Cardiothoracic Surgery, Madurai Medical College and Government Rajaji Hospital, Madurai, India^b

We have isolated a *Staphylococcus arlettae* strain, strain CVD059, from the blood of a rheumatic mitral stenosis patient. Here, we report the genome sequence and potential virulence factors of this clinical isolate. The draft genome of *S. arlettae* CVD059 is 2,565,675 bp long with a G+C content of 33.5%.

Coagulase-negative staphylococci (CoNS) are among the most common organisms causing bacteremia worldwide (5). We have reported the prevalence of CoNS in the circulation systems of cardiovascular disease (CVD) patients (3). Genome sequencing and comparative genomics of different CoNS strains will form the basis for understanding their pathogenicity and possible clinical implications in CVD patients.

Staphylococcus arlettae, one of the CoNS species, was first isolated from the skin and nares of poultry and goats (9). Subsequently, *S. arlettae* strains with azo dye degradation potential and plant growth promoting property were isolated from textile and tannery industrial effluents, respectively (4, 8). We have isolated an *S. arlettae* strain from the blood of a 47-year-old male patient suffering from rheumatic mitral stenosis at the Government Rajaji Hospital, Madurai, India, using Bactec automated blood culture system (Becton, Dickinson, NJ). Here, we report the draft genome sequence and potential virulence factors of this strain.

Genomic DNA from *S. arlettae* strain CVD059 was isolated using DNeasy miniprep kit (Qiagen, Hilden, Germany), and genome sequencing was performed using Ion Torrent Personal Genome machine (Life Technologies, CA). A total of 2,997,560 reads with an average read length of 245 bp were obtained, which yielded 734,402,200 sequenced bases (~280-fold coverage of ~2.6-Mb genome). The *de novo* assembly of the sequence was done using MIRA (mimicking intelligent read assembly) version 3.4 (2), which yielded 233 contigs (N_{50} length of ~27 kb). The Staden package version 2.0 (10) was used to visualize *de novo* assembly, and contigs with significant overlaps were joined manually. Finally, 57 contigs were obtained; the longest and shortest contigs were 501,983 bp and 993 bp, respectively. The draft genome of *S. arlettae* CVD059 is 2,565,675 bp long with 33.5% G+C content. The genome sequence was annotated using the RAST (Rapid Annotations using Subsystems Technology) server (1) and NCBI Prokaryotic Genomes Automatic Annotation Pipeline (<http://www.ncbi.nlm.nih.gov/genomes/static/Pipeline.html>). A total of 2,439 protein-encoding genes and 79 RNA coding regions were predicted.

The genome of *S. arlettae* CVD059 contains virulence genes coding for fibronectin/fibrinogen binding protein, programmed cell death toxin *ycdD*, hemolysin III, autolysins (*atl*) and their precursors. The immunodominant surface antigen B (IsaB), which elicits an immune response during septicemia, and secretory antigen SsaA, which was reported to induce elevated anti-Ssa IgG levels in endocarditis patients (6), were identified. Genes respon-

sible for resistance to methicillin, chloramphenicol, teicoplanin, tetracycline, bleomycin, bicyclomycin, polymyxin, and fluoroquinolones were identified. In addition, genes/operons coding for colicin V synthesis, bile hydrolysis, copper resistance, cobalt-zinc-cadmium resistance, and mercury resistance are present in the genome. The genome of *S. arlettae* contains *Staphylococcus* accessory gene regulators, such as *agrA*, *agrB*, *agrR*, *agrV*, and *agrZ*, that are involved in regulation of the expression of virulence genes (7, 11). Further research on the virulence genes and regulation factors may reveal the possible roles of this CoNS strain in pathogenicity.

Nucleotide sequence accession numbers. This whole-genome shotgun project has been deposited at DDBJ/EMBL/GenBank under the accession number [ALWK00000000](https://www.ncbi.nlm.nih.gov/nuccore/ALWK00000000). The version described in this paper is the first version, ALWK01000000.

ACKNOWLEDGMENTS

We gratefully acknowledge the Department of Biotechnology, New Delhi, India, for providing financial support (project BT/PR13253/GBD/27/237/2009). Central facilities, Centre of Advanced Studies (CAS), Center for Excellence in Genomic Sciences (CEGS), University Grants Commission-Networking Resource Centre in Biological Sciences (UGC-NRCBS), and Interdisciplinary Life Science Programme for Advanced Research and Education (DBT-IPLS) at the School of Biological Sciences of Madurai Kamaraj University (MKU) are gratefully acknowledged.

REFERENCES

1. Aziz RK, et al. 2008. The RAST server: rapid annotations using subsystems technology. *BMC Genomics* 9:75. doi:10.1186/1471-2164-9-75.
2. Chevreaux B. 2005. MIRA: an automated genome and EST assembler. Ph.D. thesis. German Cancer Research Center, University of Heidelberg, Heidelberg, Germany.
3. Dinakaran V, John L, Rathinavel A, Gunasekaran P, Rajendhran J. 2012. Prevalence of bacteria in the circulation of cardiovascular disease patients, Madurai, India. *Heart Lung Circ*. 21:281–283.
4. Elisangela F, et al. 2009. Biodegradation of textile azo dyes by a facultative *Staphylococcus arlettae* strain VN-11 using a sequential microaerophilic/aerobic process. *Int. Biodeter. Biodegrad.* 63:280–288.
5. Huebner J, Goldmann DA. 1999. Coagulase-negative staphylococci: role as pathogens. *Annu. Rev. Med.* 50:223–236.

Received 13 September 2012 Accepted 17 September 2012

Address correspondence to Jeyaprakash Rajendhran, rajendhran@gmail.com.

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

doi:10.1128/JB.01732-12

6. Lang S, Livesley MA, Lambert PA, Littler WA, Elliott TS. 2000. Identification of a novel antigen from *Staphylococcus epidermidis*. FEMS Immunol. Med. Microbiol. 29:213–220.
7. Li M, et al. 2004. Genetic polymorphism of the accessory gene regulator (*agr*) locus in *Staphylococcus epidermidis* and its association with pathogenicity. Med. Microbiol. 53:545–549.
8. Sagar S, Dwivedi A, Yadav S, Tripathi M, Kaistha SD. 2012. Hexavalent chromium reduction and plant growth promotion by *Staphylococcus arlettae* strain Cr11. Chemosphere 86:847–852.
9. Schleifer KH, Kilpper-Bälz R, Devriese LA. 1984. *Staphylococcus arlettae* sp. nov., *S. equorum* sp. nov. and *S. kloosii* sp. nov.: three new coagulase-negative, novobiocin-resistant species from animals. Syst. Appl. Microbiol. 5:501–509.
10. Staden R, Beal KF, Bonfield JK. 2000. The Staden package, 1998. Methods Mol. Biol. 132:115–130.
11. Vandenesch F, Projan SJ, Kreiswirth B, Etienne J, Novick RP. 1993. Agr-related sequences in *Staphylococcus lugdunensis*. FEMS Microbiol. Lett. 111:115–122.