

# Complete Genome Sequence of *Pseudomonas aeruginosa* Mucoid Strain FRD1, Isolated from a Cystic Fibrosis Patient

Laura A. Silo-Suh,<sup>a</sup> Sang-Jin Suh,<sup>b</sup> Dennis E. Ohman,<sup>c,d</sup> Daniel J. Wozniak,<sup>e</sup> Julia W. Pridgeon<sup>f</sup>

Department of Basic Medical Sciences, Mercer University School of Medicine, Macon, Georgia, USA<sup>a</sup>; Department of Biological Sciences, Auburn University, Auburn, Alabama, USA<sup>b</sup>; Department of Microbiology and Immunology and Virginia Commonwealth University Medical Center, Richmond, Virginia, USA<sup>c</sup>; Hunter Holmes McGuire VA Medical Center, Richmond, Virginia, USA<sup>d</sup>; Center for Microbial Interface Biology, The Ohio State University, Columbus, Ohio, USA<sup>e</sup>; Aquatic Animal Health Research Unit, USDA-ARS, Auburn, Alabama, USA<sup>f</sup>

We announce here the complete genome sequence of the *Pseudomonas aeruginosa* mucoid strain FRD1, isolated from the sputum of a cystic fibrosis patient. The complete genome of *P. aeruginosa* FRD1 is 6,712,339 bp. This genome will allow comparative genomics to be used to identify genes associated with virulence, especially those involved in chronic pulmonary infections.

Received 6 February 2015 Accepted 10 February 2015 Published 19 March 2015

**Citation** Silo-Suh LA, Suh S-J, Ohman DE, Wozniak DJ, Pridgeon JW. 2015. Complete genome sequence of *Pseudomonas aeruginosa* mucoid strain FRD1, isolated from a cystic fibrosis patient. *Genome Announc* 3(2):e00153-15. doi:10.1128/genomea.00153-15.

**Copyright** © 2015 Silo-Suh et al. This is an open-access article distributed under the terms of the [Creative Commons Attribution 3.0 Unported license](https://creativecommons.org/licenses/by/3.0/).

Address correspondence to Laura A. Silo-Suh, silo-suh\_l@mercer.edu.

The Gram-negative bacterium *Pseudomonas aeruginosa* is the major etiological agent responsible for pulmonary infections in cystic fibrosis (CF) patients leading to morbidity and mortality. CF patients are frequently colonized by environment-borne *P. aeruginosa*, which initially causes intermittent infections (1). Chronic infections are associated with significant genetic changes in the bacterium that appear to facilitate the persistence of the pathogen in this niche (2, 3). One such change is the acquisition of the mucoid phenotype caused by mutations in the *mucA* gene leading to overproduction of the exopolysaccharide alginate (4, 5). Alginate is a major component of the *P. aeruginosa* biofilm matrix responsible for increased tolerance to antibiotics (6). A mucoid strain of *P. aeruginosa*, designated FRD1, was isolated from a sputum sample from a chronically infected CF patient in 1979 by Barbara Iglewski (Portland, OR) and first reported in 1981 as a strain that can be readily manipulated genetically (7). This isolate is arguably the best characterized CF *P. aeruginosa* isolate and has provided the basis for much of the information assembled on alginate biosynthesis and regulation in *P. aeruginosa* (8–11). The complete genome sequence of *P. aeruginosa* FRD1 was determined in this study in order to augment and facilitate a further investigation of the microevolution process of *P. aeruginosa* adaptation in the CF lung.

Genomic DNA was isolated from an overnight broth culture of *P. aeruginosa* FRD1 using the Wizard genomic DNA purification kit (Promega) and sequenced using the Pacific Biosciences RS II platform. HGAP3 was used to *de novo* assemble a total of 117,765 sequence reads, with an average length of 4,559 bp. The whole genome of *P. aeruginosa* FRD1 is 6,712,339 bp, with a G+C content of 66.1% and 6,439 predicted open reading frames. The contigs of the *de novo*-assembled genome of *P. aeruginosa* FRD1 share 99% identity with those of the genome of *P. aeruginosa* strain PA38182 (GenBank accession no. HG530068), a major epidemic strain throughout Europe (12). The annotation of the genome by Manatee detected the following breakdown within subsystems: 91 open reading frames in amino acid biosynthesis, 82 in fatty acid

and phospholipid metabolism, 58 in central intermediary metabolism, 320 in energy metabolism, 1,030 in transport and binding proteins, 117 in DNA metabolism, 90 in transcription, and 273 in cell envelope.

**Nucleotide sequence accession number.** The complete genome sequence of *P. aeruginosa* FRD1 was deposited in GenBank under the accession no. [CP010555](https://ncbi.nlm.nih.gov/nucl/CP010555). The version described in this paper is the first version.

## ACKNOWLEDGMENTS

We thank Leslie Carroll for isolation of the genomic DNA and the University of Maryland Genomics Resource Center for sequencing and annotation of the FRD1 genome.

This study was partially supported by funds from the Mercer University School of Medicine provided to L. A. Silo-Suh, The National Science Foundation's Experimental Program to Stimulate Competitive Research (EPSCoR) (NSF ESP 11-58862) to Auburn University, National Institutes of Health research grant AI19146 (to D.E.O.), Veterans Administration Medical research grant I01BX000477 (to D.E.O.), and the National Institutes of Health grants AI097511 and NR013898 to D.J.W.

## REFERENCES

- Burns JL, Gibson RL, McNamara S, Yim D, Emerson J, Rosenfeld M, Hiatt P, McCoy K, Castile R, Smith AL, Ramsey BW. 2001. Longitudinal assessment of *Pseudomonas aeruginosa* in young children with cystic fibrosis. *J Infect Dis* 183:444–452. [http://dx.doi.org/10.1086/318075](https://doi.org/10.1086/318075).
- Nguyen D, Singh PK. 2006. Evolving stealth: genetic adaptation of *Pseudomonas aeruginosa* during cystic fibrosis infections. *Proc Natl Acad Sci U S A* 103:8305–8306. [http://dx.doi.org/10.1073/pnas.0602526103](https://doi.org/10.1073/pnas.0602526103).
- Hoboth C, Hoffmann R, Eichner A, Henke C, Schmoltdt S, Imhof A, Heesemann J, Hogardt M. 2009. Dynamics of adaptive microevolution of hypermutable *Pseudomonas aeruginosa* during chronic pulmonary infection in patients with cystic fibrosis. *J Infect Dis* 200:118–130. [http://dx.doi.org/10.1086/599360](https://doi.org/10.1086/599360).
- Doggett RG. 1969. Incidence of mucoid *Pseudomonas aeruginosa* from clinical sources. *Appl Microbiol* 18:936–937.
- Martin DW, Schurr MJ, Mudd MH, Govan JR, Holloway BW, Deretic V. 1993. Mechanism of conversion to mucoidy in *Pseudomonas aeruginosa* infecting cystic fibrosis patients. *Proc Natl Acad Sci U S A* 90:8377–8381. [http://dx.doi.org/10.1073/pnas.90.18.8377](https://doi.org/10.1073/pnas.90.18.8377).

6. Hentzer M, Teitzel GM, Balzer GJ, Heydorn A, Molin S, Givskov M, Parsek MR. 2001. Alginate overproduction affects *Pseudomonas aeruginosa* biofilm structure and function. *J Bacteriol* 183:5395–5401. <http://dx.doi.org/10.1128/JB.183.18.5395-5401.2001>.
7. Ohman DE, Chakrabarty AM. 1981. Genetic mapping of chromosomal determinants for the production of the exopolysaccharide alginate in a *Pseudomonas aeruginosa* cystic fibrosis isolate. *Infect Immun* 33:142–148.
8. Chitnis CE, Ohman DE. 1990. Cloning of *Pseudomonas aeruginosa* *algG*, which controls alginate structure. *J Bacteriol* 172:2894–2900.
9. Franklin MJ, Ohman DE. 1993. Identification of *algF* in the alginate biosynthetic gene cluster of *Pseudomonas aeruginosa* which is required for alginate acetylation. *J Bacteriol* 175:5057–5065.
10. Wozniak DJ, Ohman DE. 1994. Transcriptional analysis of the *Pseudomonas aeruginosa* genes *algR*, *algB*, and *algD* reveals a hierarchy of alginate gene expression which is modulated by *algT*. *J Bacteriol* 176:6007–6014.
11. Jain S, Ohman DE. 1998. Deletion of *algK* in mucoid *Pseudomonas aeruginosa* blocks alginate polymer formation and results in uronic acid secretion. *J Bacteriol* 180:634–641.
12. Witney AA, Gould KA, Pope CF, Bolt F, Stoker NG, Cubbon MD, Bradley CR, Fraise A, Breathnach AS, Butcher PD, Planche TD, Hinds J. 2014. Genome sequencing and characterization of an extensively drug-resistant sequence type 111 serotype O12 hospital outbreak strain of *Pseudomonas aeruginosa*. *Clin Microbiol Infect* 20:O609–O618. <http://dx.doi.org/10.1111/1469-0691.12528>.