



## Draft Genome Sequences of 11 Clinical Isolates of Acinetobacter baumannii

## P. G. Higgins,<sup>a,b</sup> J. Z.-M. Chan,<sup>c</sup> H. Seifert,<sup>a,b</sup> M. J. Pallen,<sup>c</sup> A. D. Millard<sup>c</sup>

Institute for Medical Microbiology, Immunology and Hygiene, University of Cologne, Cologne, Germany<sup>a</sup>; German Center for Infection Research (DZIF), partner site Bonn-Cologne, Cologne, Germany<sup>b</sup>; Microbiology and Infection Unit, Warwick Medical School, University of Warwick, Warwick, United Kingdom<sup>c</sup>

The development of multidrug-resistant *Acinetobacter baumannii* is of serious concern in the hospital setting. Here, we report draft genome sequences of 11 *A. baumannii* isolates that were isolated from a single patient over a 65-day period, during which time the isolates exhibited increased antimicrobial resistance.

Received 24 February 2016 Accepted 29 February 2016 Published 14 April 2016

Citation Higgins PG, Chan JZ-M, Seifert H, Pallen MJ, Millard AD. 2016. Draft genome sequences of 11 clinical isolates of Acinetobacter baumannii. Genome Announc 4(2): e00269-16. doi:10.1128/genomeA.00269-16.

Copyright © 2016 Higgins et al. This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International license.

Address correspondence to A. D. Millard, a.d.milard@warwick.ac.uk.

A cinetobacter baumannii is recognized as a serious threat in the hospital environment, particularly in the intensive care unit (ICU) setting (1). The treatment of *A. baumannii* infections can be complicated, because the organism is often multidrug resistant (MDR), mediated through acquired resistance determinants, or a mutant (2). In this study, we present the draft genome sequences of 11 MDR *A. baumannii* isolates that were isolated from a single patient over a 65-day period. Over this time period, the patient was treated with multiple antibiotics, a practice that is associated with increased antimicrobial resistance (3). This was reported previously to be associated with mutations in the genes encoding the efflux pump regulator AdeR and the acquired oxacillinase OXA-164 (3).

Bacterial genomic DNA was prepared from cultures using the Qiagen DNeasy kit, according to the manufacturer's instructions, and sequenced using MiSeq (Illumina, USA). One nanogram of genomic DNA was prepared using the Nextera XT DNA sample preparation kit (Illumina) prior to sequencing on the MiSeq platform using the paired-end  $2 \times 250$ -bp (version 2) protocol. The resulting FASTQ files were quality trimmed and assembled de novo using the Velvet integrated in the Ridom SeqSphere+ version 3.0 software (Ridom GmbH, Münster, Germany). Here, reads were trimmed at their 5' and 3' ends until an average base quality of 30 was reached in a window of 20 bases, and the assembly was performed with Velvet version 1.1.04 using optimized k-mer size and coverage cutoff values based on the average length of contigs with >1,000 bp. All genomes were sequenced to a minimum depth of  $20 \times$ , with a median coverage of  $41 \times$  across all 11 isolates. The median genome size was 3.980 Mb and ranged from 3.878 Mb (isolate C) to 3.996 Mb (isolate K). The resulting number of contigs per genome ranged from 122 (isolate J) to 836 (isolate C). Contigs were annotated with Prokka 1.11 (4) using the Acinetobacter-specific database for annotation. The number of predicted tRNAs per genome ranged from 39 (isolate C) to 58 (isolate F). The range of predicted coding sequences (CDSs) per genome was from 3,543 CDSs (isolate C) to 3,797 CDSs (isolate I), with a median of 3,780 CDSs from all 11 genomes.

Multilocus sequence typing using the Pasteur scheme (http: //pubmlst.org/abaumannii/) shows that all isolates belong to sequence type 1 (ST1), which corresponds to the international clone lineage 1 (5). The genome sequences confirmed the previously observed resistance-associated mutations (3). Furthermore, *in silico* analysis using the CGE server (https://cge.cbs.dtu.dk/) revealed that these isolates possess multiple aminoglycoside-modifying enzymes: all possessed aac(3)-Ia and aph(3')-Ic, while nine amikacin-resistant isolates had aph(3')-via, which was missing in two amikacin-susceptible isolates.

The genome information of these 11 sequential *A. baumannii* isolates will aid our understanding of genetic changes that occur during a prolonged infection treated with multiple antimicrobial therapies.

**Nucleotide sequence accession numbers.** The draft genome sequences of isolates B, C, D, E, F, G, I, J, K, L, and M have been deposited in DDBJ/ENA/GenBank under the accession numbers FITR01000001, FCNC01000001, FCND01000001, FCNG01000001, FCNJ01000001, FCNF01000001, FCNK01000001, FCNH01000001, FCNI01000001, FCNE01000001, and FJMY01000001, respectively.

## **FUNDING INFORMATION**

The project was supported by start-up funds from Warwick Medical School to M.J.P. Illumina sequencing was performed at Warwick Medical School, University of Warwick.

## REFERENCES

- Peleg AY, Seifert H, Paterson DL. 2008. Acinetobacter baumannii: emergence of a successful pathogen 21:538–582. http://dx.doi.org/10.1128/ CMR.00058-07.
- Dijkshoorn L, Nemec A, Seifert H. 2007. An increasing threat in hospitals: multidrug-resistant *Acinetobacter baumannii*. Nat Rev Microbiol 5:939–951. http://dx.doi.org/10.1038/nrmicro1789.
- Higgins PG, Schneiders T, Hamprecht A, Seifert H. 2010. *In vivo* selection of a missense mutation in *adeR* and conversion of the novel *bla*<sub>OXA-164</sub> gene into *bla*<sub>OXA-58</sub> in carbapenem-resistant *Acinetobacter baumannii* isolates from a hospitalized patient. Antimicrob Agents Chemother 54:5021–5027. http://dx.doi.org/10.1128/AAC.00598-10.
- Seemann T. 2014. Prokka: rapid prokaryotic genome annotation. Bioinformatics 30:2068–2069. http://dx.doi.org/10.1093/bioinformatics/btu153.
- Diancourt L, Passet V, Nemec A, Dijkshoorn L, Brisse S. 2010. The population structure of *Acinetobacter baumannii*: expanding multiresistant clones from an ancestral susceptible genetic pool. PLoS One 5:e10034. http://dx.doi.org/10.1371/journal.pone.0010034.