Aliiroseovarius crassostreae, formerly known as Roseovarius crassostreae, is the causative agent of Roseovarius oyster disease (ROD), which results in high mortality rates in hatchery-raised juvenile eastern oysters in the northeast United States (1-3). This alphaproteobacterium is a strict aerobe and a member of the Roseobacter clade (4). Strain CV919-312 was isolated by Boettcher et al. (4) from tissues of an infected oyster from the Damariscotta River in Maine. Here, we announce the genome sequence of A. crassostreae CV919-312Sm, a spontaneous streptomycin-resistant mutant, to help elucidate the mechanisms of virulence used by this bacterium to cause ROD.

A single isolated colony of A. crassostreae CV919-312Sm was grown overnight in yeast-peptone broth supplemented with 3% artificial sea salts (YP30) (5). Genomic DNA was isolated using the Promega Wizard genomic DNA purification kit, and DNA was resuspended in 2 mM Tris-HCl buffer (Bio Basic). Sequencing was performed at the Rhode Island Genomics and Sequencing Center using the Illumina MiSeq benchtop instrument.

Sequence trimming and de novo assembly were performed using the CLC Genomics Workbench (version 8.0.1). Contigs with an average coverage of >100X were assembled with the CLC Genomics Workbench and SPAdes genomic assembler (version 3.1.1). The resulting contigs were joined using the CLC Microbial Genome Finishing module. The draft genome consists of 26 contigs, with a total sequence length of 3,706,831 bp and G+C content of 58.4%, plus one complete plasmid of 18,548 bp with a G+C content of 58.3%.

Gene annotation was performed using Rapid Annotations using Subsystems Technology (RAST) (6-8) and Integrated Microbial Genomes/Expert Review ( IMG/ER) (9). Annotation revealed multiple clusters of genes involved in flp (fimbrial low-molecular-weight) pilus assembly, which may play an important role in attachment to the inner shell surface of oysters. Oyster hemocytes treated with extracellular products from A. crassostreae experienced high mortality, indicating that extracellular proteins play a role in pathogenicity (10). One putative channel-forming hemolysin/cytolysin gene was identified. Additionally, BLASTX results reveal that the genome also contains 11 open reading frames with conserved regions associated with RTX toxins and related Ca\(^{2+}\) binding proteins, one with strong similarity to serralysin peptidase (11). An acyltransferase is located directly adjacent to two of these open reading frames (ORFs). These genes may be hemolysin/leukotoxin-type toxins affecting oyster hemocytes (12). Some of the annotated RTX toxins might also be involved in surface attachment (13). Additionally, a putative type IVA secretion system (T4ASS) was located. This T4ASS gene cluster is missing VirB1 and VirB7 in annotations from both RAST and IMG/ER. However, two ORFs within the cluster contain soluble lytic murein transglycosylase domains, which are the same class of proteins as VirB1 (14). Identification of VirB7 is often challenging since VirB7 has not been described in all T4SSs (14, 15). A partial T4BSS was also located and annotated; T4BSSs are often involved in pathogenesis (16).

**Nucleotide sequence accession numbers.** This whole-genome shotgun project has been deposited in DDBJ/ENA/GenBank under the accession no. LKBA00000000. The version described in this paper is version LKBA01000000.

**ACKNOWLEDGMENTS**

We thank Katherine Boettcher for providing us with A. crassostreae strain CV919-312. This research is based in part upon work conducted using the Rhode Island Genomics and Sequencing Center, which is supported in part by the National Science Foundation under EPSCoR grants 0554548 and EPS-1004057. This work was supported by an award from the Rhode Island Science and Technology Advisory Council to David R. Nelson and David C. Rowley.

**FUNDING INFORMATION**

This work, including the efforts of David C. Rowley and David R. Nelson, was funded by Rhode Island Science and Technology Advisory Council.
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