

# Draft Genome Sequence of *Streptococcus agalactiae* PR06

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***Streptococcus agalactiae* (group B streptococcus [GBS]) is a Gram-positive bacterium that was first recognized as a causative agent of bovine mastitis. *S. agalactiae* has subsequently emerged as a significant cause of human diseases. Here, we report the draft genome sequence of *S. agalactiae* PR06, which was isolated from a septicemic patient in a local hospital in Malaysia.**

Received 28 April 2013 Accepted 3 May 2013 Published 13 June 2013

Citation MZ IS, Teh LK, Salleh MZ. 2013. Draft genome sequence of *Streptococcus agalactiae* PR06. *Genome Announc.* 1(3):e00351-13. doi:10.1128/genomeA.00351-13.

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*Streptococcus agalactiae* is well-adapted to asymptomatic colonization in the gastrointestinal and genitourinary tracts of healthy individuals (1). However, under certain circumstances, it may turn into a life-threatening pathogen causing septicemia, meningitis, and pneumonia in neonates (2). It is also a serious cause of mortality and morbidity in nonpregnant adults, particularly in elderly persons and those with underlying diseases (3). Here, we report the draft genome sequence of the *S. agalactiae* PR06 isolate from a local hospital in Malaysia.

*S. agalactiae* PR06 was grown in Todd-Hewitt broth under optimal growth conditions. Gram stain and biochemical tests were performed to determine the purity of the bacterium. The genomic DNA of *S. agalactiae* PR06 was isolated using the Wizard genomic DNA purification kit (Promega). The draft genome of local *S. agalactiae* PR06 was determined using the Genome Analyzer IIX platform (Illumina). This genome was sequenced to produce ultra-deep coverage of 1,000×, and the quality was checked using FastQC. The reads were trimmed using the CLC bio Genomic Workbench 5.1 and assembled by mapping against the genome of the closest reference strain, *S. agalactiae* A909. A total of 67 contigs were produced, and these contigs were ordered with respect to the best-aligned positions compared to the reference genome of *S. agalactiae* A909 using Optimal Syntenic Layout of Unfinished Assemblies (OSLay) (4). The genome was annotated using the Rapid Annotations using Subsystems Technology (RAST) server (5), and putative coding sequences (CDSs) and annotated genes were identified by comparing outputs from the Bacterial Annotation System (BASys) (6).

The draft genome of *S. agalactiae* contains a circular chromosome of 2,120,750 bp with a G+C content of 37% and 2,143 protein-coding sequences. The genome revealed >37 RNA genes and several virulence genes, including the *cyl* locus, which is responsible for pulmonary epithelial cell damage; the *cfb* gene, which codes for CAMP factor (7); the *scp* gene, which codes for C5a peptidase (8); and gene loci involved in capsule synthesis.

These observations and comparative genomic studies would be

useful in providing better knowledge of the genetic and molecular elements that might reveal drug-resistance profiles and vulnerabilities. The genome offers targets for the development of diagnostic tests, as well as new antimicrobial drugs and vaccines.

**Nucleotide sequence accession number.** The genome sequence of *S. agalactiae* PR06 has been deposited at DDBJ/EMBL/GenBank under the accession no. [AOSD000000000](https://www.ncbi.nlm.nih.gov/nuccore/AOSD000000000).

## ACKNOWLEDGMENT

This research was funded by the Pharmacogenomic Centre (PROMISE), Faculty of Pharmacy, Universiti Teknologi MARA.

## REFERENCES

1. Koenig JM, Keenan WJ. 2009. Group B streptococcus and early-onset sepsis in the era of maternal prophylaxis. *Pediatr. Clin. North Am.* 56:689–708.
2. Melin P. 2011. Neonatal group B streptococcal disease: from pathogenesis to preventive strategies. *Clin. Microbiol. Infect.* 17:1294–1303.
3. Schrag SJ, Zywicki S, Farley MM, Reingold AL, Harrison LH, Lefkowitz LB, Hadler JL, Danila R, Cieslak PR, Schuchat A. 2000. Group B streptococcal disease in the era of intrapartum antibiotic prophylaxis. *N. Engl. J. Med.* 342:15–20.
4. Richter DC, Schuster SC, Huson DH. 2007. OSLay: optimal syntenic layout of unfinished assemblies. *Bioinformatics* 23:1573–1579.
5. Aziz RK, Bartels D, Best AA, DeJongh M, Disz T, Edwards RA, Formsma K, Gerdes S, Glass EM, Kubal M, Meyer F, Olsen GJ, Olson R, Osterman AL, Overbeek RA, McNeil LK, Paarmann D, Paczian T, Parrello B, Pusch GD, Reich C, Stevens R, Vassieva O, Vonstein V, Wilke A, Zagnitko O. 2008. The RAST server: rapid annotations using subsystems technology. *BMC Genomics* 9:75.
6. Van Domselaar GH, Stothard P, Shrivastava S, Cruz JA, Guo A, Dong X, Lu P, Szafron D, Greiner R, Wishart DS. 2005. BASys: a web server for automated bacterial genome annotation. *Nucleic Acids Res.* 33:W455–W459.
7. Chuzeville S, Puymège A, Madec JY, Haenni M, Payot S. 2012. Characterization of a new CAMP factor carried by an integrative and conjugative element in *Streptococcus agalactiae* and spreading in streptococci. *PLoS One* 7:e48918.
8. Beckmann C, Waggoner JD, Harris TO, Tamura GS, Rubens CE. 2002. Identification of novel adhesins from group B streptococci by use of phage display reveals that C5a peptidase mediates fibronectin binding. *Infect. Immun.* 70:2869–2876.