

Genome Sequence of *Propionibacterium acnes* Type II Strain ATCC 11828

Balázs Horváth,^a Judit Hunyadkúrti,^{a,b} Andrea Vörös,^a Csaba Fekete,^b Edit Urbán,^c Lajos Kemény,^d and István Nagy^a

Institute for Plant Genomics, Human Biotechnology and Bioenergy, Bay Zoltán Foundation for Applied Research, Szeged, Hungary^a; Department of General and Environmental Microbiology, University of Pécs, Pécs, Hungary^b; Institute of Clinical Microbiology, University of Szeged, Szeged, Hungary^c; and Department of Dermatology and Allergology, University of Szeged, Szeged, Hungary^d

***Propionibacterium acnes* is an anaerobic Gram-positive bacterium that forms part of the normal human cutaneous microbiota and is occasionally associated with inflammatory diseases (I. Kurokawa et al., *Exp. Dermatol.* 18:821–832, 2009). Here we present the complete genome sequence for the commercially available *P. acnes* type II reference strain ATCC 11828 (I. Nagy et al., *Microbes Infect.* 8:2195–2205, 2006) recovered from a subcutaneous abscess.**

Propionibacterium acnes is considered a skin commensal which, under certain conditions, acts as an opportunistic pathogen and is thus associated with several diseases such as acne vulgaris (9). Even though the significance of the involvement of *P. acnes* in inflammatory diseases is still controversial, the first complete genome sequence of *P. acnes* uncovered the pathogenic potential of this bacterium (1). This is further supported by recent studies showing that distinct *P. acnes* strains trigger the secretion of antimicrobial peptides and proinflammatory mediators from various cell types *in vitro* (4, 7, 8, 13, 14). By comparison of *recA* and *tly* sequences, *P. acnes* isolates may be subdivided into phylotypes IA, IB, II, and III (12). Further subdivision came from the multilocus sequence typing (MLST) approach, resulting in the identification of 60 sequence types (STs) (11). Importantly, there is a clear association between some STs and certain diseases, such as the association of type IA clone ST6 with acne vulgaris; in contrast, other STs, such as ST10 isolates, seem to be nonpathogenic (10, 11). In order to identify factors responsible for the diversity, it is mandatory to compare genomes within and across phylogenetic clusters. Yet, only three complete *P. acnes* genomes are currently available: one type IA ST25 genome (2) and two type IB ST10 genomes (1, 6).

Genome sequencing of a type II strain, ATCC 11828, was performed by the SOLiD sequencing technology. We have generated 14,040,094 reads, which yielded >280-fold coverage. Assembly was performed using the Genomics Workbench 4.7 and the Omixon Gapped SOLiD Alignment 1.2 plug-in (3) provided by CLC Bio and Omixon, respectively. Gap closing was accomplished using PCR followed by Sanger sequencing. Automatic annotation of the genome was performed by the NCBI Prokaryotic Genomes Automatic Annotation Pipeline (PGAAP) (<http://www.ncbi.nlm.nih.gov/genomes/static/Pipeline.html>), which utilizes GeneMark, Glimmer, and tRNAscan-SE searches. *P. acnes* strain ATCC 11828 has a single circular chromosome of 2,488,752 bp with a GC content of 60%. There are 2,260 putative coding sequences, 45 tRNAs, and 6 rRNA loci.

Previous phylogenetic analysis revealed that strain ATCC 11828 belongs to the *P. acnes* type II division (13). Based on the recently published MLST scheme for *P. acnes* (11), we have now determined that this strain belongs to the ST44 lineage. When cocultured with keratinocytes, *P. acnes* strain ATCC 11828 did not induce the expression of human β -defensin-2, nor did it have any effect on the viability of keratinocytes (14). Furthermore, type II

strains do not produce immunoreactive proteins, such as dermatan-sulfate binding adhesions (5, 11), which may be important in the context of acne pathogenesis. These data suggest that type II isolates are part of the normal cutaneous flora.

Nucleotide sequence accession number. The complete nucleotide sequence of *P. acnes* strain ATCC 11828 has been deposited in GenBank under accession number CP003084.

ACKNOWLEDGMENTS

This work was supported by the Hungarian National Office for Research and Technology Teller program OMF0-00441/2007, by the French-Hungarian Associated European Laboratory (LEA) SkinChroma OMF0-00272/2009, and by TÁMOP-4.2.1.B-10/2/KONV-2010-0002.

We thank Marianna Nagymihály and Judit Cseklye for their valuable work in sequencing.

REFERENCES

1. Bruggemann H, et al. 2004. The complete genome sequence of *Propionibacterium acnes*, a commensal of human skin. *Science* 305:671–673.
2. Brzuszkiewicz E, et al. 2011. Comparative genomics and transcriptomics of *Propionibacterium acnes*. *PLoS One* 6:e21581.
3. Csuros M, Juhos S, Berces A. 2010. Fast mapping and precise alignment of AB SOLiD color reads to reference DNA, p 176–188. In Moulton V, Singh M (ed), *Proceedings of the 10th International Conference on Algorithms in Bioinformatics*. Springer-Verlag, Berlin, Germany.
4. Graham GM, Farrar MD, Cruse-Sawyer JE, Holland KT, Ingham E. 2004. Proinflammatory cytokine production by human keratinocytes stimulated with *Propionibacterium acnes* and *P. acnes* GroEL. *Br. J. Dermatol.* 150:421–428.
5. Holland C, et al. 2010. Proteomic identification of secreted proteins of *Propionibacterium acnes*. *BMC Microbiol.* 10:230.
6. Hunyadkúrti J, et al. 2011. Complete genome sequence of *Propionibacterium acnes* type IB strain 6609. *J. Bacteriol.* 193:4561–4562.
7. Jugeau S, et al. 2005. Induction of toll-like receptors by *Propionibacterium acnes*. *Br. J. Dermatol.* 153:1105–1113.
8. Kim J, et al. 2002. Activation of toll-like receptor 2 in acne triggers inflammatory cytokine responses. *J. Immunol.* 169:1535–1541.
9. Kurokawa I, et al. 2009. New developments in our understanding of acne pathogenesis and treatment. *Exp. Dermatol.* 18:821–832.

Received 18 October 2011 Accepted 21 October 2011

Address correspondence to István Nagy, nagy@baygen.hu.

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

doi:10.1128/JB.06388-11

10. Lomholt HB, Kilian M. 2010. Population genetic analysis of *Propionibacterium acnes* identifies a subpopulation and epidemic clones associated with acne. *PLoS One* 5:e12277.
11. McDowell A, et al. 2011. A novel multilocus sequence typing scheme for the opportunistic pathogen *Propionibacterium acnes* and characterization of type I cell surface-associated antigens. *Microbiology* 157:1990–2003.
12. McDowell A, et al. 2005. *Propionibacterium acnes* types I and II represent phylogenetically distinct groups. *J. Clin. Microbiol.* 43:326–334.
13. Nagy I, et al. 2006. *Propionibacterium acnes* and lipopolysaccharide induce the expression of antimicrobial peptides and proinflammatory cytokines/chemokines in human sebocytes. *Microbes Infect.* 8:2195–2205.
14. Nagy I, et al. 2005. Distinct strains of *Propionibacterium acnes* induce selective human beta-defensin-2 and interleukin-8 expression in human keratinocytes through toll-like receptors. *J. Invest. Dermatol.* 124: 931–938.