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GENOME ANNOUNCEMENT

Genome Sequence of *Rickettsia australis*, the Agent of Queensland Tick Typhus

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***Rickettsia australis* strain Phillips^T was isolated in Queensland, Australia, in 1950. It is the tick-borne agent of Queensland tick typhus, a disease endemic in Australia. The 1.29-Mb genome sequence of this bacterium is highly similar to that of *Rickettsia akari* but contains two plasmids.**

The genus *Rickettsia* is composed of small Gram-negative, obligate intracellular alphaproteobacteria (1) that underwent progressive genomic reduction (2). However, paradoxically, recent genomic studies have suggested that genome reduction was associated with increased virulence in rickettsiae (3). *Rickettsia australis* was first identified in residents of Northern Queensland, Australia, in 1950 (4). This bacterium is a spotted fever group rickettsia and causes Queensland tick typhus (QTT). QTT is endemic in the eastern part of Australia, where it is transmitted to humans through the bites of *Ixodes holocyclus* or *Ixodes tasmani* ticks (6). It is considered to be mostly mild and is characterized by fever, headache, and myalgia followed by the development of a maculopapular or vesicular rash, an inoculation eschar (65% of cases), and lymphadenopathy (71%) (5).

The genome sequencing of *R. australis* strain Phillips^T was performed by 454 shotgun and 454 paired-end sequencing. Briefly, the shotgun sequencing was performed using a GS-FLX Titanium sequencer (Roche, Meylan, France) with assembly into 1 complete circularized chromosome, 1 complete circularized plasmid, and 1 putative incomplete plasmid. The chromosome has a predicted size of 1,297,390 bp and a G+C content of 31.7%, which is similar to that of other rickettsial genomes. The plasmid (pRau01) has a size of 26,608 bp with a G+C content of 33.7%. The second plasmid is made of 13 contigs, for a total size of 30,176 bp, and has a G+C content of 21.7%. The chromosome contains 1,110 genes (1,426 open reading frames [ORFs]), including 820 complete genes (74%), 139 split genes (12%), and 151 gene fragments (14%). Among these 1,110 genes, 855 genes (77%) encode proteins with putative functions and 255 (23%) encode hypothetical proteins and proteins of unknown function. The *R. australis* genome contains 3 noncontiguous rRNAs (5S, 16S, and 23S rRNA), 33 tRNAs, and 3 other RNAs. The pRau01 plasmid carries 16 genes (25 ORFs), whereas the second plasmid carries 20 genes (24 ORFs).

R. australis exhibits an almost perfect genomic synteny with *Rickettsia akari*, its closest phylogenetic neighbor, with the exception of two inversions of 78,519 bp and 36,776 bp. *R. australis* lacks only 46 genes that are present in *R. prowazekii*, the agent of epidemic typhus, the most severe human rickettsiosis (7). These genes encode proteins of unknown function (23 genes), transposases (3 genes), ankyrin repeat-containing proteins (2 genes), transferases (6 genes), cell surface antigens (1 gene), synthetases (3 genes), and 1 gene in each of the following categories: BioY family protein, ABC-type transporter related to toluene tolerance protein, dCTP deaminase, putative transcriptional regulator, Sco2 protein precursor, DNA invertase Pin-like protein, VirD4 protein, and beta-glucosidase. None of these genes appears to be related to rickettsial virulence. In contrast, many genes involved in the biosynthesis and regulation of biosynthesis of amino acids and nucleotides present in *R. australis* are absent from *R. prowazekii*. Further investigations will be conducted to study the genomic basis of rickettsial virulence and the potential role of plasmids.

Nucleotide sequence accession number. The whole-genome shotgun project has been deposited in the GenBank database under accession number AKVZ00000000.

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REFERENCES

1. **Blanc G, et al.** 2007. Lateral gene transfer between obligate intracellular bacteria: evidence from the *Rickettsia massiliae* genome. Genome Res. **17**: 1657–1664.
2. **Blanc G, et al.** 2007. Reductive genome evolution from the mother of *Rickettsia.* PLoS Genet. **3**:e14. doi:10.1371/journal.pgen.0030014.
3. **Fournier PE, et al.** 2009. Analysis of the *Rickettsia africae* genome reveals that virulence acquisition in *Rickettsia* species may be explained by genome reduction. BMC Genomics **10**:166.
4. **Graves S, Stenos J.** 1999. Rickettsioses in Australia, p 244–246. *In* Raoult D, Brouqui P (ed), Rickettsiae and rickettsial diseases at the turn of the third millenium. Elsevier, Paris, France.
5. **McBride WJ, Hanson JP, Miller R, Wenck D.** 2007. Severe spotted fever group rickettsiosis, Australia. Emerg. Infect. Dis. **13**:1742–1744.
6. **Sexton DJ, Dwyer B, Kemp R, Graves S.** 1991. Spotted fever group rickettsial infections in Australia. Rev. Infect. Dis. **13**:876–886.
7. **Walker DH.** 1988. Pathology and pathogenesis of the vasculotropic rickettsioses, p 115–138. *In* Walker DH (ed), Biology of rickettsial disease. CRC Press Inc., Boca Raton, FL.

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