

***omp25* from *B. canis* clustering two phylogenetic groups**

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Brucella canis is a contagious agent responsible for reproductive failure in dogs and is potentially zoonotic for humans. A high genetic similarity among *B. canis* strains is evidenced by phylogenetic analysis. In this context, some targets have been used for epidemiology analysis, as the *omp25* gene, which encoded an outer-membrane protein. Also, the Omp25 protein has been used as a vaccine target study. Thus, the objective of this study was achieving a phylogenetic analysis of *omp25* sequences from *B. canis* isolated in South of Brazil in comparison of worldwide sequences. Phylogenetic analysis was performed using 19 isolates from our laboratory culture collection and all available (until August, 2021) *omp25* sequences from GenBank (41 strains). Nucleotide alignments were performed with MAFFT software, the best model (F81+F) was identified by JModeltest v.2.1.4, and a maximum likelihood (ML) phylogenetic tree was constructed in PhyML software. *B. suis* strain U39397 was used as a root for the tree. Besides a high similarity among the *omp25* sequences evidenced by the alignments, the worldwide *omp25* sequences diverged in two largely branches at phylogenetic analysis. A single nucleotide polymorphism at position 326 of *omp25* sequence was the point that divided the sequences in two clades. The spontaneous T>C substitution provoke a Leucine to Proline change at the 109 codon position (L109P mutation). The mutated *B. canis* strains was named *B. canis*^{L109P}, while wild strains were named *B. canis*^{WT}. The clade of *B. canis*^{WT} was composed by 42 sequences from 12 different countries. All Brazilian sequences were grouped in *B. canis*^{WT} clade. Also, composed this clade in majority the sequences from Latin America countries, United States and Europe. In the other hand, the *B. canis*^{L109P} clade was composed mostly by sequences from Asian, European and African countries. Human strains were randomly distributed at both clades. Finally, remembering the Omp25 function, we suppose that this conserved mutation at *omp25* gene may be a genetic adaptation for some worldwide strains. This adaptation can change the virulence potential of *B. canis* by either help evasion of host immune system or invasion and maintenance on host cells. These results may contribute for future acellular vaccine development based on Omp25 protein and for epidemiology investigations of *B. canis*.

Keywords: canine brucellosis, phylogenetic analysis, outer-membrane protein.

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