

Capsular-defective *Porphyromonas gingivalis* mutant strains induce less alveolar bone resorption than W50 wild-type strain due to a decreased Th1/Th17 immune response and less osteoclast activity

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Background: Encapsulation of *Porphyromonas gingivalis* has been demonstrated as responsible of several host immunological changes, which have been associated with the pathogenesis of periodontitis. Using a murine model of periodontitis and two isogenic non-capsulated mutants of *P. gingivalis*, this study aimed to analyze whether *P. gingivalis* encapsulation induces more severe alveolar bone resorption, and whether this bone loss is associated with a T-helper (Th)1 and Th17-pattern of immune response. **Methods:** Experimental periodontal infections were generated by oral inoculation with the encapsulated W50 wild-type strain or isogenic non-encapsulated ?PG0116-PG0120 (GPA) and ?PG0109-PG0118 (GPC) mutants of *P. gingivalis*. Periodontal infections induced with the encapsulated HG184 or non-encapsulated ATCC 33277 strains of *P. gingivalis* were used as controls. Alveolar bone resorption was analyzed using microcomputed tomography and scanning electron microscopy. The expression levels of Th1, Th2, Th17, or T regulatory-associated cytokines and RANKL, as well as the periodontal bacterial load, were

quantified by quantitative polymerase chain reaction. The detection of Th1 and Th17 lymphocytes was analyzed by flow cytometry. Results: In the periodontal lesions, both capsular-defective knockout mutant strains of *P. gingivalis* induced less alveolar bone resorption than the encapsulated W50 wild-type strain. This decreased bone loss was associated with a diminished RANKL expression, decreased Th1- and Th17-type of cytokine expression, reduced Th1 and Th17 lymphocyte detection, and low osteoclast finding. Conclusion: These data demonstrate that encapsulation of *P. gingivalis* plays a key role in the alveolar bone resorption induced during periodontitis, and this bone loss is associated with a Th1- and Th17-pattern of immune response triggered in the periodontal lesions.