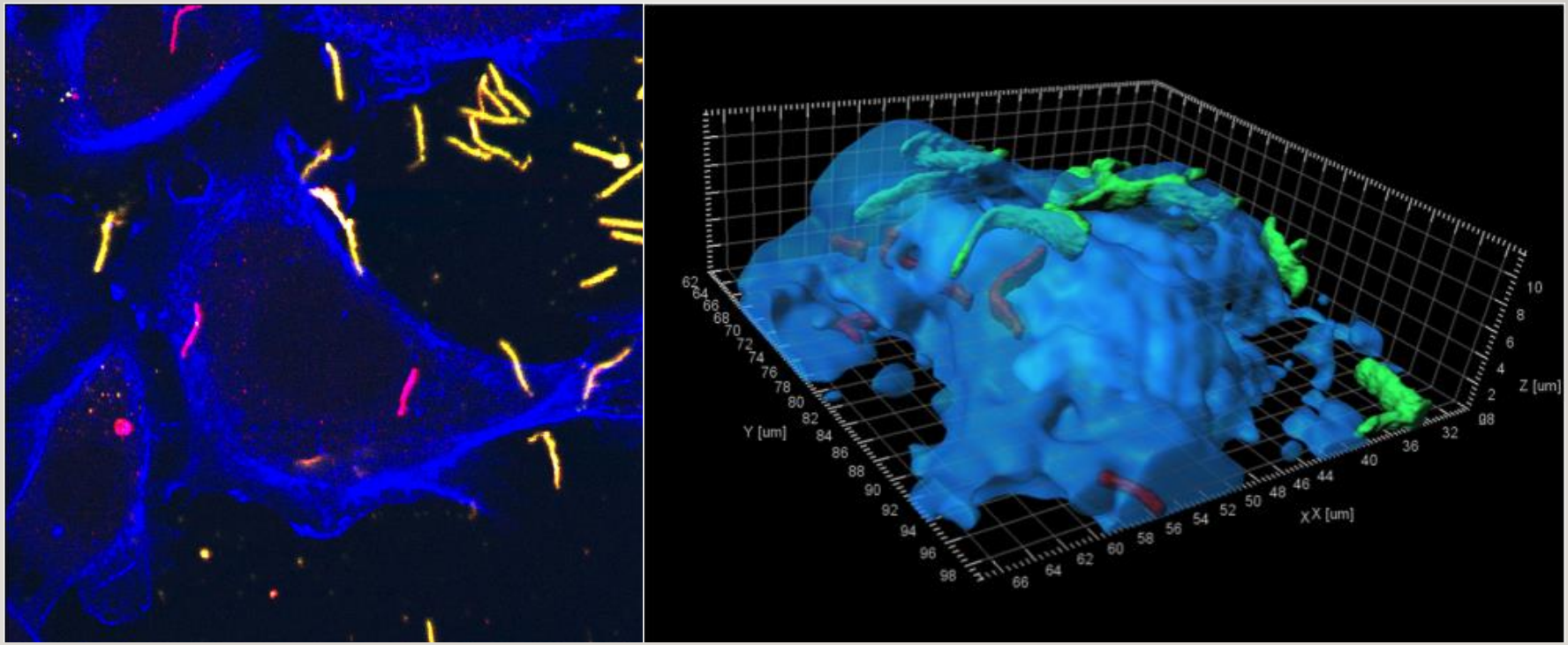
微生物学講座



微生物学講座の研究対象

- 歯周病原性細菌の遺伝子解析
  - 病原遺伝子の解析
  - Systemic diseaseとの関わり
- 歯周炎・根尖性歯周組織炎の細菌叢解析

歯周病原性細菌の遺伝子解析

AMERICAN SOCIETY FOR MICROBIOLOGY | Applied and Environmental Microbiology

Involvement of the Type IX Secretion System in *Capnocytophaga ochracea* Gliding Motility and Biofilm Formation

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Capnocytophaga ochracea **is a Gram-negative, rod-shaped bacterium that demonstrates gliding agar surfaces. *C. ochracea* possesses the ability to form biofilms; however, factors involved in bi rium are unclear. A type IX secretion system (T9SS) in *Flavobacterium johnsoniae* was shown to proteins (e.g., several adhesins) to the cell surface. Genes orthologous to those encoding T9SS pr been identified in the genome of *C. ochracea*; therefore, the T9SS may be involved in biofilm for constructed three ortholog-deficient *C. ochracea* mutants lacking *sprB* (which encodes a gliding *sprT* (which encode T9SS proteins in *F. johnsoniae*). Gliding motility was lost in each mutant, s the proteins encoded by *sprB*, *gldK*, and *sprT* are necessary for gliding motility, and SprB is trans T9SS. For the Δ*gldK*, Δ*sprT*, and Δ*sprB* strains, the amounts of crystal violet-associated biofilm, were 49%, 34%, and 65%, respectively, at 48 h. Confocal laser scanning and scanning electron m films formed by wild-type *C. ochracea* were denser and bacterial cells were closer together than i strains. Together, these results indicate that proteins exported by the T9SS are key elements of t formation of *C. ochracea*.**

PLOS ONE

RESEARCH ARTICLE

Involvement of *luxS* in Biofilm Formation by *Capnocytophaga ochracea*

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Abstract

Systemic diseaseとの関わり

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Differential ability of periodontopathic bacteria to modulate invasion of human gingival epithelial cells by *Porphyromonas gingivalis*

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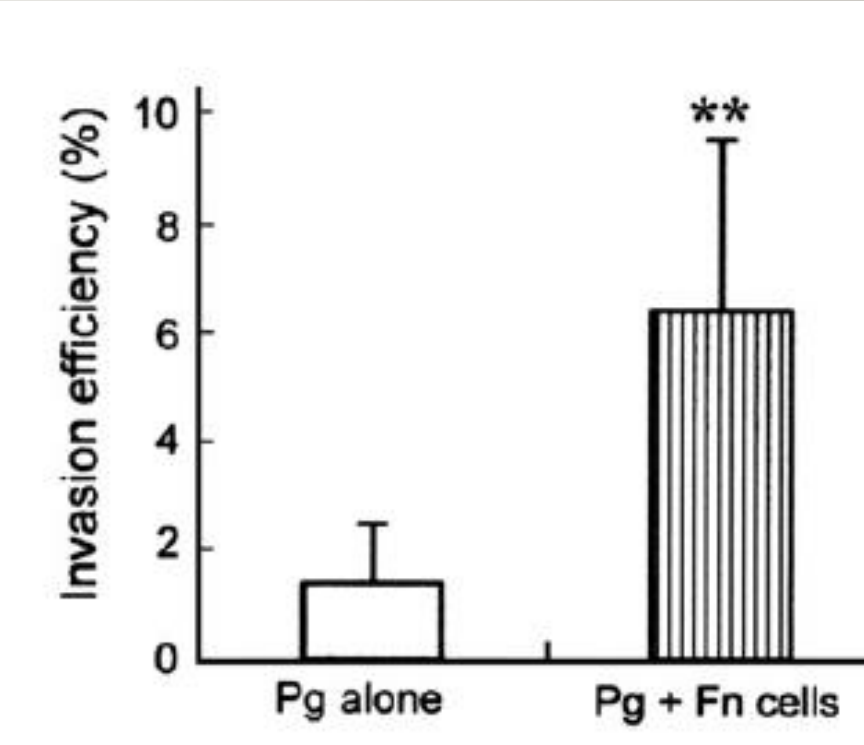
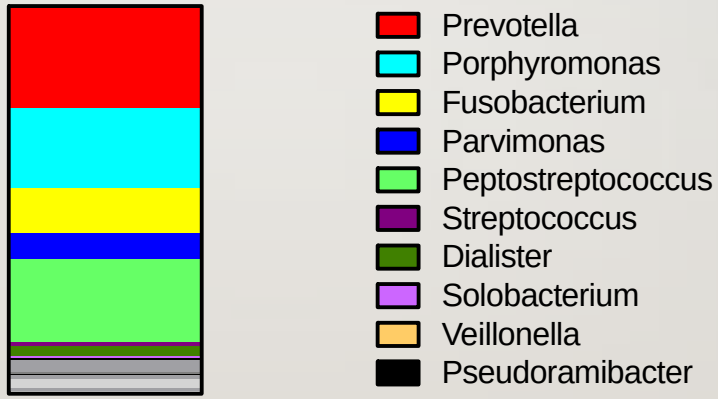
ABSTRACT

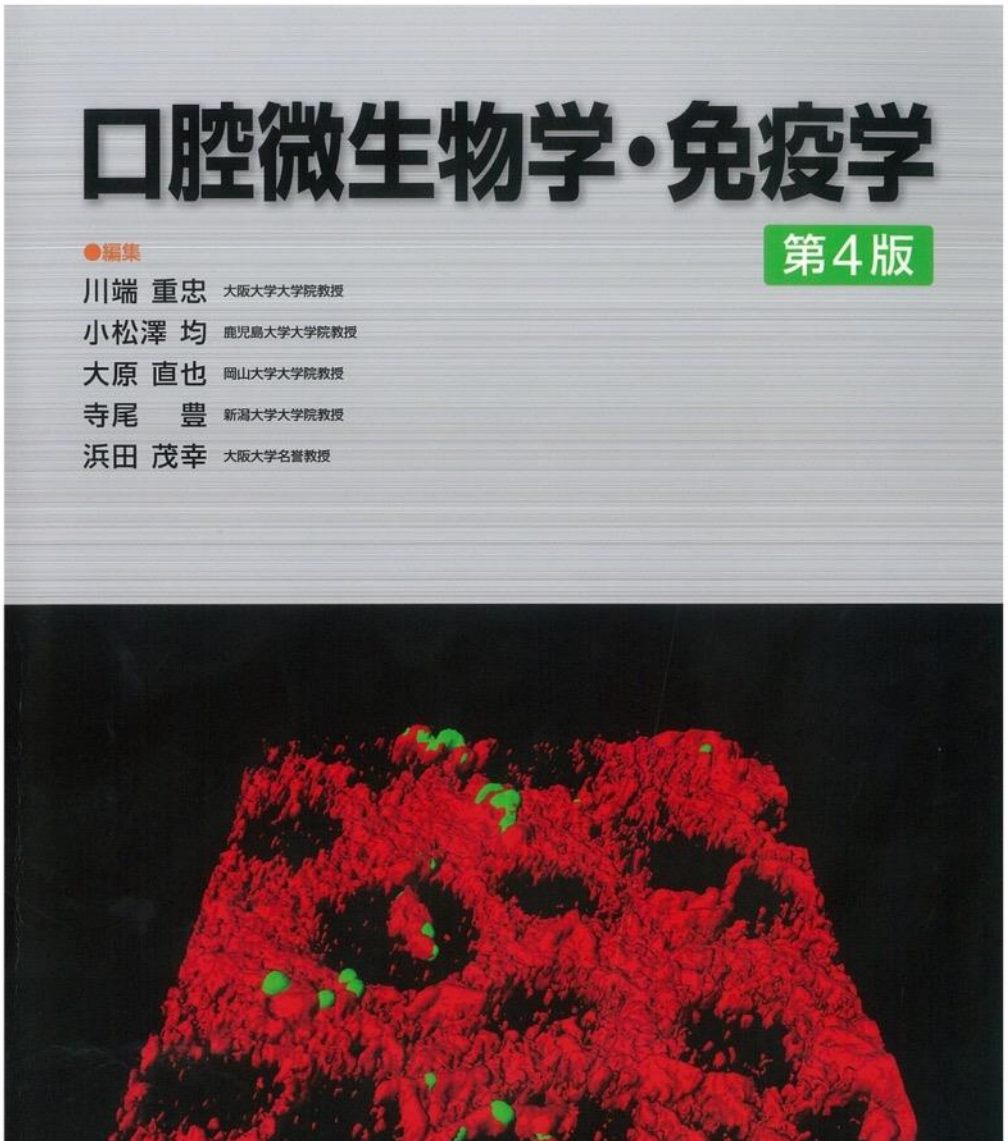
Periodontitis is a polymicrobial infection caused by selected gram-negative bacteria including *Porphyromonas gingivalis*. Host cell invasion by *P. gingivalis* has been proposed as a possible mechanism of pathogenesis in periodontitis. The aim of the present study was to assess the influence of periodontopathogens on *P. gingivalis* invasion of gingival epithelial cells in polymicrobial infection. *P. gingivalis* was tested for its ability to invade a human gingival epithelial cell line Ca9-22 in co-infection with periodontopathogens, using an antibiotic protection assay. Among the pathogens tested, only *Fusobacterium nucleatum* demonstrated the ability to significantly promote *P. gingivalis* invasion ($P < 0.01$). This increased invasion was confirmed by confocal scanning laser microscopy utilizing a dual labeling technique. In contrast, co-infection with *Aggregatibacter actinomycetemcomitans* or *Tannerella forsythia* attenuated *P. gingivalis* invasion. The fusobacterial enhancement of host cell invasion was not observed in co-incubation with other periodontopathogens tested. These results suggested that complex synergistic or antagonistic physiologic mechanisms are intimately involved in host cell invasion by *P. gingivalis* in polymicrobial infection.



歯周炎・根尖性歯周組織炎の細菌叢解析



教科書を変える。



られている．Msp はエンベロープの構成タンパク質であり，フィブロネクチンへの結合能，細胞傷害作用などが報告されている．Dentilisin はプロテアーゼ活性を有し，種々のマトリックスやサイトカインなどに対して分解活性を有する．また，本菌は dentipain とよばれるシステインプロテアーゼも産生している．

本菌は慢性歯周炎患者の歯周ポケットから特に顕著に検出されるが，*T. denticola* に対する抗体価の上昇は認められない．これは本菌が産生する免疫抑制因子による可能性が考えられている．

2．*T. vincentii*

T. vincentii の菌体は幅 0.2～0.3 μm，長さ 5～16 μm で，

臨床分野の研修

- 歯周病学会
  - 認定医

  - 昨年度卒業　Dr. 藤瀬和隆

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