<table>
<thead>
<tr>
<th>Title</th>
<th>Porphyromonas gingivalis and related bacteria: from colonial pigmentation to the type IX secretion system and gliding motility</th>
</tr>
</thead>
<tbody>
<tr>
<td>Author(s)</td>
<td>Nakayama, Koji</td>
</tr>
<tr>
<td>Citation</td>
<td>Journal of Periodontal Research, 50(1), pp.1-8; 2015</td>
</tr>
<tr>
<td>Issue Date</td>
<td>2015-02</td>
</tr>
<tr>
<td>URL</td>
<td><a href="http://hdl.handle.net/10069/35084">http://hdl.handle.net/10069/35084</a></td>
</tr>
<tr>
<td>Rights</td>
<td>© 2014 The Authors Journal of Periodontal Research Published by John Wiley &amp; Sons Ltd; This is an open access article under the terms of the Creative Commons Attribution-NonCommercial-NoDerivs License, which permits use and distribution in any medium, provided the original work is properly cited, the use is non-commercial and no modifications or adaptations are made.</td>
</tr>
</tbody>
</table>
Porphyromonas gingivalis and related bacteria: from colonial pigmentation to the type IX secretion system and gliding motility


Porphyromonas gingivalis is a gram-negative, non-motile, anaerobic bacterium implicated as a major pathogen in periodontal disease. P. gingivalis grows as black-pigmented colonies on blood agar, and many bacteriologists have shown interest in this property. Studies of colonial pigmentation have revealed a number of important findings, including an association with the highly active extracellular and surface proteinases called gingipains that are found in P. gingivalis. The Por secretion system, a novel type IX secretion system (T9SS), has been implicated in gingipain secretion in studies using non-pigmented mutants. In addition, many potent virulence proteins, including the metallocarboxypeptidase CPG70, 35 kDa hemin-binding protein HBP35, peptidylarginine deiminase PAD and Lys-specific serine endopeptidase PepK, are secreted through the T9SS. These findings have not been limited to P. gingivalis but have been extended to other bacteria belonging to the phylum Bacteroidetes. Many Bacteroidetes species possess the T9SS, which is associated with gliding motility for some of these bacteria.

Porphyromonas gingivalis is a gram-negative anaerobic bacterium considered a major pathogen in chronic periodontitis (1). Recently, it has been proposed that even in low abundance P. gingivalis is a keystone pathogen with community-wide effects that are critical for the development of dysbiosis in periodontal biofilm (2). In addition, epidemiological and experimental studies have shown that the bacterium may be associated with systemic conditions, such as cardiovascular diseases (3), preterm low birth weight (4), rheumatoid arthritis (5) and non-alcoholic fatty liver disease (6,7).

P. gingivalis requires proteoheme for growth. In heme-deprived medium, P. gingivalis cells grow slowly and eventually stop growing after several passages in this medium. On blood agar, these bacterial cells form black-pigmented colonies (Fig 1). The black pigment is derived from the proteoheme in erythrocytes. The black pigment phenotype of P. gingivalis has been attributed to the accumulation of the μ-oxo bisheme complex of Fe(III) protoporphyrin IX, [Fe(III)PPIX]2O (8–10). This heme complex, also termed μ-oxo oligomers or dimeric heme, comprises two Fe(III) protoporphyrin IX moieties bridged by an oxygen atom (8). As the optimum pH for P. gingivalis growth is approximately 8 and this bacterium produces an alkaline terminal growth pH because of peptide and amino acid metabolism (11–14), the μ-oxo dimer [Fe(III)PPIX]2O is maintained at an alkaline pH. Interestingly,
pigmented Prevotella species, such as Prev. intermedia and Prev. nigrescens, generate monomeric Fe(III)PPIX.OH from Fe(III)PPIX.OH because the terminal growth pH of these bacteria on blood agar for 8 d is approximately 6 (14). In this review, we discuss novel findings, including the type IX secretion system (T9SS), obtained from genetic studies of colonial pigmentation.

Spontaneous pigment-less mutants

When P. gingivalis cells were grown under hemin excess in a chemostat at pH 7.5 for 2–3 wk (49–73 bacterial generations) and subsequently plated on to blood agar, colonies with atypical morphology were observed (15). One colony variant (W50/BE1) was beige in color, and another colony variant (W50/BR1) was brown. Both colonial variants exhibited decreased virulence (15), and W50/BE1 lacked gelatinase, collagenase and dipeptidyl aminopeptidase activities compared with the parent strain and exhibited reduced hydrophobicity, hemagglutination activity, fimbriation and extracellular matrix proteins, cyto- kines, complement proteins, antibodies and proteinase inhibitors (17–19). Collinson et al. (20) showed that BE1 exhibited decreased Rgp activity compared with the wild type, and no Rgp enzyme with large glycan additions, which are associated with the outer membrane, was observed. The gene(s) responsible for the phenotypes of W50/BE1 and W50/BR1 has not been elucidated. However, these early findings suggest that colonial pigmentation is associated with the activity and localization of proteases in P. gingivalis cells. Moreover, Rgp was purified from the P. gingivalis strain HG66, which secreted soluble Rgp and lacked pigmentation (21,22).

The isolation of pigment-less mutants using transposon mutagenesis

Several studies have applied transposon mutagenesis to isolate pigment-less P. gingivalis mutants (23–26). Simpson et al. (26) reported a non-pigmented mutant with an insertion sequence element (IS1126) at the promoter locus of kgp. In addition, Chen et al. (25) isolated non-pigmented mutants with transposon Tn4351 insertion in a putative glucosyl (rhamnosyl) transferase-encoding gene in several non-pigmented mutants, and Abaibou et al. (28) demonstrated that the vimA gene, located downstream of recA, is responsible for pigment. Using Tn4351 transposon mutagenesis, we isolated and characterized two non-pigmented mutants (porR and porT) (29,30).

Pigmentation-related genes

Pigmentation-related genes encode proteins with three types of functions: gingipain activity, gingipain transport and gingipain attachment (31). Rgp and Kgp proteinases are encoded by rgpA, rgpB and kgp, rgpA and kgp also encode hemagglutinins (adhesins) and the hemoglobin receptor at the 3′-terminal region of these genes. kgp single mutants and rgpA rgpB kgp triple mutants form less-pigmented and non-pigmented colonies, respectively, whereas rgpA rgpB double mutants form pigmented colonies (27,32). Smalley et al. (33) revealed that Rgp activity is crucial for converting oxy-hemoglobin into methemoglobin, a form more susceptible to Kgp-mediated degradation, resulting in the release of iron(III) protoporphyrin IX and the production of μ-oxo heme dimers.

The porR mutant exhibited a pleiotropic phenotype: Rgp and Kgp proteinases were mainly present in the culture supernatant, mutant cells had no hemagglutinating activity and Rgp-mediated processing of fimbrillin was delayed (29). The porR mutant had altered phenol extractable polysaccharides. The monoclonal antibody (mAb) 1B5, which reacts with the sugar portions of P. gingivalis cell surface polysaccharides and membrane-type Rgp proteinases (17), did not react with cell lysates from the porR mutant, indicating that porR is involved in the biosynthesis of cell surface polysaccharides that might function as anchors for Rgp, Kgp, hemagglutinins and the hemoglobin receptor protein. P. gingivalis has two different lipopolysaccharide (LPS) molecules, O-LPS and A-LPS. O-LPS...
possesses the conventional O-antigen, whereas A-LPS has a different O-antigen comprising an anionic polysaccharide repeat unit that reacts with mAb 1B5 (34,35). Recently, another mAb (TDC-5-2-1) that recognizes the O-antigen of O-LPS, which is present in almost all wild-type cells, was generated; however, the glycan epitope recognized by this mAb has not been identified (36,37). As the porR mutant reacts with mAb TDC-5-2-1, this mutant might possess O-LPS but lacks A-LPS. The porR gene encodes a putative transaminase (29). We recently proposed that the final product synthesized through the Wbp pathway, which involves WbpA (PGN_0613 [UgdA]), PGN_1243), WbpB (PGN_0168), WbpE (PGN_1236 [PorE]) and WbpD (PGN_0002), is a sugar substrate required for the biosynthesis of A-LPS (38). The P. gingivalis strain HG66, typically used for gingipain purification, exhibits no pigmentation on blood agar. This strain has a nonsense mutation in the wbpB gene, which is responsible for the pigmentless phenotype of the strain (38).

The porT mutant was also isolated as a non-pigmented mutant using Tn4351 transposon mutagenesis; however, the porT mutant was quite different from the porR mutant (30). The porR mutant exhibited gingipain activity in the culture supernatant, whereas the porT mutant demonstrated no gingipain activity either in the cell extract or in the culture supernatant. Subcellular fractionation and immunoblot analysis revealed that gingipain proteases accumulate in the periplasmic space, indicating that the PorT protein is involved in gingipain transport across the outer membrane. The subcellular localization of the PorT protein is controversial. We treated the membrane fraction of P. gingivalis cells with 1% Triton X-100 to separate the outer and inner membrane fractions, and the PorT protein was detected in the inner membrane fraction (Triton X-100-soluble fraction). However, using fractionation with Sarkosyl treatment, Nguyen et al. (39) reported that the PorT protein is located in the outer membrane.

Genome sequence of Porphyromonas gingivalis

In 2003, researchers at TIGR and the Forsyth Institute determined the whole genome sequence of P. gingivalis W83 (40). The P. gingivalis W83 genome comprises 2.3 megabase pairs and encodes a range of pathways and virulence determinants associated with the novel biology of this oral pathogen. This genome size is consistent with previous measurements using pulsed field gel electrophoresis (41). We determined the whole genome sequence of a different strain, ATCC 33277, typically used as a type strain in studies of the pathogenicity and physiology of P. gingivalis (42). Via genomic comparison with strain W83, we identified 461 ATCC 33277-specific and 415 W83-specific CDSs, and extensive genomic rearrangements were observed between the two strains, including 175 regions in which genomic rearrangements occurred. Interestingly, the genomes of P. gingivalis strains did not encode proteins involved in known secretion systems, such as the type II and III secretion systems, suggesting that P. gingivalis possesses a novel secretion system.

Discovery of a new protein secretion system

Genes homologous to porT of P. gingivalis have been identified in many members of the large and diverse Bacteroidetes phylum, whereas there are no porT homologs in bacteria belonging to other phyla. In addition, a porT homolog is not present in a bacterium belonging to the genus Bac teroides, B. thetaiotaomicron. Most bacterial protein secretion systems comprise multiple proteins that form a complex in the cell envelope. Thus, a set of proteins, including PorT, required for a protein secretion system must exist in bacteria with the protein secretion system, but not in bacteria lacking the system. Therefore, we used Venn diagram analysis to identify genes involved in these protein secretion systems. We identified 55 genes, including porT, that are present in P. gingivalis and Cytophaga hutchinsionii but absent in B. thetaiotaomicron and constructed deletion mutants of the genes (43). P. gingivalis strains with deletion mutations in 46 of these genes were generated to determine involvement of these genes in a secretion system for gingipains. Among the 46 mutants, 10 mutations in sov (PGN_0832), which was previously implicated in gingipain secretion (44), porK (PGN_1676), porL (PGN_1675), porM (PGN_1674), porP (PGN_1677), porQ (PGN_0645), porU (PGN_0022), porW (PGN_1877), porX (PGN_1019) and porY (PGN_2001) resulted in decreased Rgp or Kgp activity in cells and culture supernatants. We named this novel secretion system the Por secretion system (PorSS), now referred to as the T9SS (43,45,46) (Fig. 2). PorK, PorN and PorP were detected in the outer membrane fraction, whereas PorL and PorM were detected in the inner membrane fraction. Blue native gel analysis revealed that PorK was associated with PorN, and PorL was associated with PorM (43,47,48). PGN_0023 (pg27/lptO/porY) is located downstream of porU (PGN_0022) on the P. gingivalis chromosome. PGN_0023, a mutant deficient in gingipain secretion (48,49), has been implicated in the O-deacylation of LPS (50). PGN_0022 (porU)-encoding protein is a C-terminal signal peptidase for proteins that possess conserved C-terminal domains (CTDs) and utilize the T9SS for translocation across the outer membrane (49). Veith et al. (51) first identified the CTDs of P. gingivalis proteins. Seers et al. (52) predicted a role for CTDs in export and attachment to the cell surface, and Shoji et al. (53) verified this role. PG27/LptO/PorV has been associated with PorU and several CTD proteins (54).

Two-component system for the T9SS of Porphyromonas gingivalis

porX (PGN_1019) and porY (PGN_2001) encode the response regulatory protein and histidine sensor kinase, respectively, of a two-component signal transduction system and have roles in regulating the expression of the genes in the transport system (43) (Fig. 2). These genes are located separately on the P. gingivalis chromosome, although the cognate
Fig. 2. Model of the T9SS of Porphyromonas gingivalis. T9SS comprises more than 10 proteins, including PorK, PorL, PorM, PorN, PorP, PorQ, PorT, PorU, PorV, PorW and Sov. Some of these proteins were expressed using the two-component system PorXY. PorX and PorY are a response regulator and a sensor kinase, respectively. CTD proteins, such as Kgp (K) and Rgp (R), are translocated across the IM via Sec machinery and subsequently secreted across the OM through the T9SS. CTD, C-terminal domains; CP, cytoplasm; IM, inner membrane; OM, outer membrane; PP, periplasm; T9SS, type IX secretion system.

partners of a two-component signal transduction system are typically located in tandem. Microarray and reverse transcription polymerase chain reaction analyses revealed that 20 genes were downregulated by < 60% in the porX deletion mutant compared with the wild-type parent strain. Among these genes were porK, porL, porM, porN, porP, porT and sov, which are involved in the T9SS. These genes were also downregulated in the porY mutant, suggesting that decreased expression of gingipains in the porX and porY single mutants reflects the downregulation of porK, porL, porM, porN, porP, porT and sov.

Extracellular and surface proteins secreted through the T9SS in Porphyromonas gingivalis

The T9SS was identified in a study examining the secretion of gingipains. PGN_1728 (kgp), PGN_1733 (hagA), PGN_1970 (rgpA) and PGN_1466 (rgpB) encode gingipain group proteins, with proproteins that possess CTDs at the C-terminus, and the secretion of these proteins is dependent on the T9SS. However, the T9SS is not specific for gingipain secretion. Seers et al. (52) predicted that CTD-containing proteins other than gingipains, including PG1326 (PGN_1115), PG2100 (PGN_0152, tapA), PG2102 (no PGN, tapC), PG1427 (PGN_0900, prlT), PG1374 (PGN_0852), PG0495 (PGN_1476), PG0232 (PGN_0335, cpg70), PG0611 (PGN_0654), PG0654 (PGN_0693), PG1798 (PGN_1767), PG0553 (PGN_1416, pepK), PG2216 (PGN_2080), PG0350 (PGN_1611), PG1795 (PGN_1770), PG0616 (PG N_0659, hbp35), PG1424 (PGN_0898, pad), PG0614 (PGN_0657), PG1030 (PGN_1321) and PG0290 (PGN_1674), are secreted through this secretion system. In addition, Veith et al. (55) reported that as well as the CTD proteins described above, the outer membrane vesicle contains the following CTD proteins: PG0026 (PGN_0022, porU), PG0182 (PGN_0291), PG0183 (no PGN), PG0411 (PGN_1556), PG0626 (no PGN), PG1548 (no PGN), PG1604 (PGN_0509), PG1969 (PGN_1770) and PG2172 (PGN_0123). We compared the proteomes of P. gingivalis strains kgp rgpA rgpB (T9SS-sufficient strain) and kgp rgpA rgpB porK (T9SS-deficient strain) using two-dimensional gel electrophoresis and peptide mass fingerprinting to identify other proteins secreted through the T9SS and identified the following 10 proteins: PGN_0152 (PG2100, tapA), PGN_0291 (PG0182), PGN_0335 (PG0232, cpg70), PGN_0654 (PG0611), PGN_0659 (PG0616, hbp35), PGN_0795 (PG0769), PGN_0898 (PG1424, pad), PGN_1416 (PG0553, pepK), PGN_1476 (PG0495) and PGN_1767 (PG1798) (56).

tapA (PGN_0152, PG2100) was associated with tprA (PGN_0876, PG1385). TprA is a tetratricopeptide repeat (TPR) protein that was upregulated in wild-type P. gingivalis (W83) cells placed in a mouse subcutaneous chamber, and the tprA mutant was clearly less virulent in the mouse subcutaneous abscess model (57). When the tprA mutant was placed in a mouse subcutaneous chamber, nine genes, including PG2102 (tapA), PG2101 (tapB) and PG2100 (tapC), were downregulated in the tprA mutant compared with the wild-type bacteria (58). These mutant genes were also downregulated in the culture medium. Yeast two-hybrid system analysis and in vitro protein binding assays with immunoprecipitation and surface plasmon resonance
N- and C-terminally truncated peptides (59). Purified CPG70 is a peptidase (CPG70) that cleaves C-terminally truncated forms of the 40 kDa protein, and the initiation codons for truncated forms of the 40 kDa protein are N-terminal (63). The 29 kDa protein on the cell surface (63). The 29 kDa protein, and the initiation codons for these proteins are located in the middle of the coding sequence of hbp35. HBP35 exhibits thioredoxin activity and is essential for hemin-depleted conditions. The CTD of HBP35 has been extensively characterized (53). The 22 C-terminal amino acid residues of the CTD of HBP35 are required for cell surface translocation and glycosylation. The CTD region functions as a recognition signal for the T9SS, and the glycosylation of CTD proteins occurs after removal of the CTD region, as CTD-containing peptides were not detected in samples of glycosylated HBP35 protein through peptide map fingerprinting analysis, and antibodies against CTD region peptides did not react with glycosylated HBP35 protein (53).

pad (PGN_0898, PG1424) encodes a prokaryotic peptidylarginine deiminase (PAD). McGraw et al. (64) purified and characterized the biochemical and enzymatic properties of the PAD enzyme from P. gingivalis and proposed that PAD, acting in concert with arginine-specific proteinases from P. gingivalis, promotes the growth of the pathogen in the periodontal pocket by enhancing the survivability of this bacterium and mediating the circumvention of host humoral defenses (64). Subsequently, research interests were focused on the relationship between P. gingivalis PAD and rheumatoid arthritis (5,65,66). Experimental evidence of a relationship between PAD and rheumatoid arthritis has recently been proposed. Using the chamber model, Maresz et al. (67) showed that infection with viable wild-type P. gingivalis exacerbated collagen-induced arthritis in a mouse model, manifested through earlier onset, accelerated progression and enhanced disease severity, including significantly increased bone and cartilage destruction. Additional studies showed that infection with wild-type P. gingivalis significantly increased levels of autoantibodies to collagen type II and citrullinated epitopes, as a PAD null mutant did not elicit similar host responses. Consistently, Guly et al. (68) reported that the development of experimental periodontitis was significantly reduced in PAD-deficient P. gingivalis, and the extent of collagen-induced arthritis was significantly reduced in animals exposed to previous induction of periodontal disease through oral inoculation with a PAD-deficient strain vs. the wild type.

PepK protein, encoded by pepK (PGN_1416, PG0553), is secreted via the T9SS and anchored on to the cell surface through binding to A-LPS (56,69). Enzymatic analysis using outer membrane fractions of wild-type, pepK and gingipain-deficient mutant strains suggests that PepK has Lys-specific serine endopeptidase activity, and the activation of this protein requires processing through Rgp (69).

**T9SSs in other bacteria**

The comparative analysis of 37 Bacteroidetes bacteria genomes revealed T9SS genes in bacteria belonging to the phylum Bacteroidetes (45). Mutant analysis has revealed functional T9SSs in three other bacterial species (C. hutchinsonii, Flavobacterium johnsonii, Tannerella forsythia) in the phylum Bacteroidetes.

In F. johnsoniae, a gliding bacterium that digests insoluble chitin, a chiA-encoded chitinase (Fjoh_4555) is secreted via the T9SS (43,70). The F. johnsoniae genome encodes proteins with CTDs similar to the P. gingivalis CTD. However, the C-terminal region of ChiA is not similar to that of P. gingivalis CTD, although it is necessary for T9SS-mediated secretion (70).

Wang et al. (71) constructed an orthologous porU mutant in C. hutchinsonii, a widely distributed gram-negative cellulolytic bacterium, and this mutant showed defects in cellulose degradation and protein secretion. In addition, C. hutchinsonii CHU_0344, a dominant extracellular protein that possesses a C-terminal CTD, is secreted through the T9SS (71).

T. forsythia is one of the three bacteria implicated in the ‘Red Complex’ with P. gingivalis and Treponema denticola, which are important for chronic periodontitis (72). We constructed porK, porT and sov orthologous genes from T. forsythia mutants and observed that these single mutants lack the surface layer (S-layer) and express less-glycosylated versions of the S-layer glycoproteins TfsA and TfsB (73). Compared with the proteins secreted from the porK and wild-type strains, the secretion of several proteins containing CTD-like sequences is porK gene-dependent. Tomek et al. (74) obtained similar results using porK and porU orthologous mutants, showing that the TfsA and TfsB glycoproteins in these mutants, which are N-terminally processed for Sec-mediated translocation across the cytoplasmic membrane, are
O-glycosylated, revealing that T9SS-mediated translocation across the outer membrane is not associated with O-glycan attachment. In wild-type bacteria, TfsA and TfsB are likely further glycosylated with rough-type LPS on the cell surface (74).

**T9SS and gliding motility**

While the periodontal pathogens *P. gingivalis* and *T. forsythia* are non-motile, the phylum Bacteroidetes includes many gliding bacteria, such as *F. johnsoniae* and *C. hutchinsonii* (75). *F. johnsoniae* cells attach to and move along surfaces at speeds of up to 5 μm/s in a process known as gliding motility (76). Electron microscopic analyses have failed to identify motility machines such as flagella and type IV pili on cells of *F. johnsoniae*, and analysis of the genome failed to identify genes encoding critical components of flagella and type IV pili, suggesting that *F. johnsoniae* gliding motility is achieved by another mechanism (77).

Bacteroidetes gliding motility is closely associated with the T9SS (43,45,46). *F. johnsoniae* genes *gldK*, *gldL*, *gldM*, *gldN*, *sprA*, *sprE* and *sprT*, which are essential for gliding motility, are homologous to *P. gingivalis* T9SS-related genes. Our unpublished data obtained from mutant studies suggest that *Prev. intermedia* has a functional T9SS. The proteins secreted through T9SSs include many virulence factors, such as gingipains in *P. gingivalis*. More than 10 proteins comprising T9SSs have been identified, but the precise interactions of these proteins remain unknown. Thus, the elucidation of T9SSs is only just commencing.

**Coda**

Many oral bacteria associated with periodontal diseases belong to the phylum Bacteroidetes. These bacteria are members of the genera *Porphyromonas*, *Prevotella*, *Tannerella* and *Capnocytophaga* and possess T9SS-related genes. Our unpublished data obtained from mutant studies suggest that *Prev. intermedia* has a functional T9SS. The proteins secreted through T9SSs include many virulence factors, such as gingipains in *P. gingivalis*. More than 10 proteins comprising T9SSs have been identified, but the precise interactions of these proteins remain unknown. Thus, the elucidation of T9SSs is only just commencing.

**Acknowledgements**

Koji Nakayama would like to thank the members of the Division of Microbiology and Oral Infection, Department of Molecular Microbiology and Immunology, Nagasaki University Graduate School of Biomedical Sciences, for their assistance.

**References**


---

**Fig. 3**. Model of *Flavobacterium* gliding motility. A translocating cell on glass. SprB exhibits two different states: SprB moving toward the front of the cell, and SprB moving toward the rear of the cell. In a translocating cell, SprB moving toward the rear of the cell results in adhesion to the surface, generating left-handed rotation and right-directed translocation of the cell. SprB moves toward the front of the cell twice as fast with respect to the glass surface than SprB in a non-translocating cell. Reproduced from Nakane et al. (78).
Colonial pigmentation, secretion and motility


30. Sato K, Sakai E, Veith PD et al. Identification of a new membrane-associated protein that influences transport/matura-


35. Rangarajan M, Aduse-Opoku J, Paramo-


37. Shoji M, Yukitake H, Sato K et al. Iden-
tification of an O-antigen chain length regula-
tor, WzP, in Porphyromonas gingi-

38. Shoji M, Sato K, Yukitake H, Naito M, Nakayama K. Involvement of the Wbp pathway in the biosynthesis of Porphyro-


40. Nelson KE, Fleischmann RD, DeBoy RT et al. Complete genome sequence of the oral pathogenic bacterium Porphyro-


42. Naito M, Hirakawa H, Yamashita A et al. Determination of the genome sequence of Porphyromonas gingivalis strain ATCC 33277 and genomic comparison with strain W83 revealed extensive genome rearrange-

43. Sato K, Naito M, Yukitake H et al. A pro-


45. McBride MJ, Zhu Y. Gliding motility and Por secretion system genes are widespread among members of the phylum bacterio-

46. Chagnon C, Zorgani MA, Astruc T, Desvaux M. Proteinaceous determinants of surface colonization in bacteria: bacte-


