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Distribution of 8 periodontal microorganisms in family members of Chinese patients with aggressive periodontitis

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ABSTRACT

Objective: To date, no information on the distribution of periodontal microorganisms among family members of Chinese patients with aggressive peridontitis (AgP) is available. The aim of the present study was to investigate the probability of transmission of eight periodontal microorganisms between patients with aggressive periodontitis and their family members.

Design: Saliva and pooled subgingival plaque samples were collected from 103 participants from 41 nuclear families (including 41 AgP probands, 19 mothers, 22 fathers, 21 siblings). Eight periodontal microorganisms, including *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Tannerella forsythia*, *Treponema denticola*, *Campylobacter rectus*, *Prevotella intermedia*, *Prevotella nigrescens* and *Fusobacterium nucleatum* were detected in these samples by the polymerase chain reaction (PCR). In addition, the distribution of *fimA* genotypes was assessed in *P. gingivalis*-positive individuals by PCR.

Results: *P. gingivalis*, *T. forsythia*, *T. denticola*, *C. rectus* and *F. nucleatum* were the most frequently detected species both in AgP probands and in their relatives. Kappa statistical analysis revealed that the detection of *A. actinomycetemcomitans* (Kappa = 0.503) and *F. nucleatum* (Kappa = 0.565) in probands was highly consistent with that in their relatives. Most probands shared the identical *fimA* genotype of *P. gingivalis* with their relatives.

Conclusions: Our results suggested that the intrafamilial transmission of periodontal microorganisms may occur between Chinese patients with aggressive periodontitis and their relatives.

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Abbreviations: AgP, aggressive periodontitis; CP, chronic periodontitis; PD, probing depth; BI, bleeding index; AL, attachment loss.

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1. Introduction

Aggressive periodontitis (AgP) is a group of infrequent types of periodontal diseases with rapid attachment loss and bone destruction initiated at a young age. Many studies[1,2] have indicated that the prevalence of AgP is disproportionately high among certain families. In many families, the percentage of affected siblings may reach 40–50%,[3,4] or even higher.[5] The notable familial aggregation of AgP cases indicates that genetic factors might be important in susceptibility to AgP[6–8]. In addition, familial aggregation of periodontal disease may also reflect exposure to common environmental factors. Nasidze's observations suggest that similar lifestyles and diet lead to more similar oral microbiomes.[9] Certain infectious agents may cluster in families. The intrafamilial transmission of periodontal microorganisms may in part explain the familial aggregation of AgP and may have important prophylactic and treatment implications.

It has been shown that periodontal microorganisms are not restricted to subgingival areas, also being found in the saliva, supragingival plaque and various mucous membranes in patients with periodontitis.[10–12] Saliva and direct mucosal contact might be the main transmission routes of periodontal microorganisms. In a study[13] on the transmission of *Porphyromonas gingivalis* within families, it was noted that a *P. gingivalis*-colonized mother became a risk for colonization of their child and the risk of colonization was highest for a child when both parents were colonized with *P. gingivalis*. Umeda observed that *Tannerella forsythia*, *Prevotella intermedia* and *Prevotella nigrescens* were detected more frequently in children whose parents were positive for these microorganisms than in children whose parents were negative.[14] Similar results were obtained from other studies on children and their mothers.[15,16] Taken these observations together, the intrafamilial transmission of periodontal microorganisms may be suggested. However, detection of the same bacterial species in family members does not prove transmission. For further evidence, typing of the bacterial isolates is necessary. Methods of bacteria typing include molecular methods and phenotypic methods. *Aggregatibacter actinomycetemcomitans* and *P. gingivalis* are detected frequently in patients with periodontitis, which makes these species prime candidates for studying person to person transmission.

To the best of our knowledge, no information on the distribution of periodontal pathogens among family members of Chinese patients with AgP is available. The aim of the present study was to investigate the probability of transmitting eight periodontal pathogens between patients with AgP and their family members.

2. Material and methods

2.1. Study population and clinical examination

From 2000 to 2011, a total of 103 participants from 41 nuclear families (including 41 AgP probands, 19 mothers, 22 fathers, 21 siblings) were recruited from the Department of Periodontology, Peking University School and Hospital of Stomatology. All the participants were members of the Chinese Han race. AgP patients who encouraged their relatives to participate in the study are termed probands. All the AgP patients were of the generalized type (GAgP). According to the 1999 international classification of periodontal diseases,[17] all participants were diagnosed as healthy, or having gingivitis, chronic periodontitis (CP), or AgP, based on full-mouth periodontal chartings (including assessments of probing depth (PD), attachment loss, bleeding index (BI)[18] at six sites per tooth) and full-mouth periapical radiographs. All the clinical parameters were recorded by three skilled, calibrated practitioners (HM, LX and LZ) as described by Shi.[19] Calibration was performed on 10 patients with severe periodontitis. The consistency of the replicated measurements of PD and AL for each examiner and paired measurements between two examiners were recorded. The clinical criteria used to define GAgP were as follows: (1) patients were under 35 years old; (2) they had rapid attachment loss and bone destruction; (3) at least eight teeth, three of them not being first molars and incisors, had PD $>$ 5 mm, AL $>$ 3 mm; (4) clinical diagnosis was confirmed by evidence of interproximal bone loss on full-mouth periapical radiographs. Participants were excluded if they (1) had a chronic medical disease or condition such as diabetes, cardiovascular disease, chronic kidney disease, hereditary disease and so on; (2) were pregnant or lactating; (3) had received periodontal treatment within the previous 6 months or antibiotic medication during the previous 3 months. Further classification of CP was based on the extent and severity of the clinically evident periodontal destruction. All the CP patients were divided into mild, moderate and severe types as described by Armitage.[20] According to the medical history, one relative was edentulous because he had a history of severe periodontitis. The study protocol was reviewed and approved by the Ethics Committee of the Peking University Health Science Center. The probands and all family members who agreed to attend the study provided written informed consent.

2.2. Sample collection and processing

Whole saliva and subgingival plaque samples were obtained 1 week after completing the full-mouth periodontal examination. Whole saliva samples were collected before subgingival plaque samples. Approximately 0.5 ml of unstimulated whole saliva from each individual was collected in a sterile Eppendorf tube. Subgingival plaque samples were obtained from the mesiobuccal sites of four first molars per participant (excluding sites with caries, interproximal restoration and crown, or if missing, premolars or second molars instead), and the four samples were pooled into a single sample tube. After isolating the sampled area with cotton rolls and gentle air drying, supragingival plaque was removed carefully with curettes, subgingival samples were obtained by placing a sterile Gracey curette at the apical extent of the pocket or gingival crevice and drawing it coronally with slight pressure. The sampled sites of periodontitis patients were characterized by PD $\geq$ 4 mm and AL $\geq$ 2 mm (if the mesiobuccal sites of the first molars had no pocket, other first or second molar sites were used).