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# Frequency of periodontal pathogens in equivalent peri-implant and periodontal clinical statuses

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ABSTRACT

*Objectives:* This study tested the hypotheses that there is: (1) higher bacterial frequency in peri-implantitis/periodontitis, followed by mucositis/gingivitis and peri-implant/periodontal health; (2) similar bacterial frequency between comparable peri-implant and periodontal clinical statuses.

*Design of study:* The presence of *Porphyromonas gingivalis*, *Tannerella forsythia*, *Campylobacter rectus*, *Prevotella intermedia*, *Treponema denticola* and *Aggregatibacter actinomycetemcomitans* was evaluated in peri-implant ($n = 53$) and periodontal ($n = 53$) health; mucositis ($n = 50$), gingivitis ($n = 50$), peri-implantitis ($n = 50$) and periodontitis ($n = 50$).

*Results:* The pattern of peri-implant bacterial frequency was not as expected (peri-implantitis > mucositis > health). Except for *P. intermedia* ($p > 0.05$), bacterial frequency was higher in peri-implantitis than health ($p < 0.05$). The frequency of *P.gingivalis* and red complex species were higher in peri-implantitis than mucositis ($p < 0.05$). In periodontal samples, *T. forsythia* and *T. denticola* showed the expected pattern of frequency (periodontitis > gingivitis > health). The frequencies of *C. rectus* and *T. forsythia* were higher in healthy teeth/gingivitis than healthy implants/mucositis, respectively ($p < 0.05$). The frequency of *P. gingivalis* and *A. actinomycetemcomitans* were similar between periodontitis and peri-implantitis ($p > 0.05$) while all other species occurrences were higher in periodontitis than peri-implantitis ($p < 0.05$).

*Conclusions:* Bacterial frequency increased from peri-implant/periodontal health to peri-implantitis/periodontitis but not from mucositis/gingivitis to peri-implantitis/periodontitis. There was a trend towards higher bacterial frequency in teeth than implants.



## 1. Introduction

Despite many crucial histological and structural differences between teeth and implants, their clinical similarities lead researchers to apply some general well accepted statements in periodontal field to implant dentistry. The inflammation restricted to soft tissues in early stages followed by bone loss and increased pocket depth could exemplify these similarities.

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In addition, peri-implant and periodontal diseases share some risk factors such as age, tobacco use and levels of oral hygiene.[1–4] The fact that risk factors for periodontal disease could also increase the risk of development of peri-implant disease confirms that both disorders share some etiopathogenic aspects. Moimaz et al.[5] reported smoking, a recognized risk factor for periodontitis, as the most important risk factor for the development of mucositis. For peri-implant disease similar findings were also observed by Karbach et al.[6] in a sample of 100 patients with single implants. Interestingly, periodontitis history per se may also be considered a risk factor for peri-implant disease.[4] Schou et al.,[7] in a systematic review, showed a significantly increased incidence of peri-implantitis and peri-implant bone loss in subjects with periodontitis associated tooth loss. Similarly, Safii et al.[8] demonstrated in a meta-analysis study that periodontitis subjects showed a higher risk of implant failure and greater marginal bone loss than periodontally healthy subjects. This relation was recently reviewd by Donos et al.[3]

Although periodontal diseases are multifactorial disorders, it is well established that subjects that harbour periodontal pathogens are more susceptible to gingivitis/periodontitis development.[9] The microenvironment (i.e. sulcus/pockets) around teeth favours selective bacterial colonization and, the successive interactions among bacterial species ultimately contribute to the aggregation of microorganisms forming periodontopathogenic communities.[10] The microorganisms considered to be periodontal pathogens may perpetuate the imbalance in the microbiota and the inflammatory response in periodontal tissues. Therefore, the presence of some key pathogenic species is well recognized to be related to the progression and severity of periodontal disease.[11–13] Although present in smaller number in healthy periodontal sites, target periodontal species tend to increase as a healthy periodontal condition shift to a diseased periodontal status. This tendency was demonstrated in a well-known paper in which the authors compared the microbiota of healthy, gingivitis and initial periodontitis sites[13] and confirmed by other investigations.[14–16]

It has been suggested that bacteria which cause periodontal breakdown could migrate and colonize peri-implant sites.[17] Quirynen et al.[18] analysed the subgingival microbiota present in so-called ''pristine pockets'', namely pockets created after insertion of transmucosal abutments in previously submerged dental implants. The authors demonstrated that periodontal pathogens were more frequently found when adjacent teeth also harboured them, showing that the development of subgingival plaque in implants is directly influenced by the supragingival environment. This plausible finding was corroborated by studies that observed that, even after the complete loss of teeth, some of these target species still remain in the oral cavity[19,16] and, bacteria may be also detected in apparently healed alveolar bone.[20] Therefore, not only teeth but also the oral soft tissues could act as important reservoirs of bacteria that can subsequent colonize the sulcus/pockets around dental implants. As observed in periodontal tissues, studies have suggested that the presence of periodontal pathogens could also lead to damage in the peri-implant tissues.[21–24] However, it is not completely clear if there is a progressive increase in pathogens frequencies when different peri-implant statuses are compared; i.e. healthy peri-implant sites vs. mucositis vs. peri-implantitis. The pathogens *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Prevotella intermedia*, *Treponema denticola*, and *Tanerella forsythia* were detected in Brazilians with healthy and diseased implants.[25] In addition, little evidence arose from studies which concomitantly compared the microbiota of peri-implant and periodontal sites from healthy to diseased statuses.[26,27]

Therefore, the first aim of this cross-sectional study is to verify if there is a tendency towards an increase in pathogen frequency from peri-implant health to established peri-implant diseases, as previously observed from healthy to diseased periodontal conditions. The second aim of the present study is to test if bacterial frequency is comparative between equivalent periodontal and peri-implant clinical statuses, i.e. healthy peri-implant vs. healthy periodontal sites, mucositis vs. gingivitis and, peri-implantitis vs. periodontitis.

## 2. Materials and methods

This research protocol was reviewed and approved by the Institutional Ethics Committees from University of Taubaté (2008/0098) and Guarulhos University (09/2005). After verbal and written explanations, individuals who agreed to participate signed an informed consent form. Participants received oral hygiene instructions and dental treatment according to their individual needs.

### 2.1. *Study population*

This convenience sample population was composed of subjects selected, from January 2006 to June 2010, according to six specific diagnoses: peri-implant ($n = 53$ subjects) or periodontal health ($n = 53$ subjects); peri-implant mucositis ($n = 50$ subjects) or gingivitis ($n = 50$ subjects); peri-implantitis ($n = 50$ subjects) or chronic periodontitis ($n = 50$ subjects).

Eligible subjects were screened from two Clinical Centres, Department of Dentistry of the University of Taubaté and Department of Periodontics of the University of Guarulhos, according to the following inclusion criteria: male or female; aged between 26 and 52 years; at least fifteen natural teeth; at least one single titanium implant (MKIII, Nobel Biocare) under function for at least one year (for the implant groups). In addition, some exclusion criteria were considered: smoking (current smokers and former smokers); alcohol abuse; diabetes mellitus; immunosuppressive systemic conditions; pregnancy and lactation; extensive fix or removable orthodontic or prosthetic appliances; local or systemic antibiotic therapy within 6 months prior to biofilm sampling; daily regular use of mouthwash two months prior to the study; any type of periodontal treatment in the past 12 months (for periodontal groups).

### 2.2. *Clinical examination*

Clinical parameters were measured by two trained and calibrated examiners at six sites per tooth or implant using