

Bacterial Colonization of the Dental Implant Fixture–Abutment Interface: An In Vitro Study

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Background: The geometry of the fixture–abutment interface (FAI) might influence the risk of bacterial invasion of the internal part of the implant. The aim of this study was to use an in vitro model to assess the potential risk for invasion of oral microorganisms into the FAI microgap of dental implants with different characteristics of the connection between the fixture and abutment.

Methods: Thirty implants were divided into three groups (n = 10 per group) based on their microgap dynamics. Groups 1 and 2 were comprised of fixtures with internal Morse-taper connections that connected to standard abutments and the same abutments with a 0.5-mm groove modification, respectively. Group 3 was comprised of implants with a tri-channel internal connection. Fixtures and abutments were assembled and allowed to incubate in a bacterial solution of *Aggregatibacter actinomycetemcomitans* (previously *Actinobacillus actinomycetemcomitans*) and *Porphyromonas gingivalis*. Two standard abutments were either exposed to bacterial culture or left sterile to serve as positive and negative controls. After disconnection of fixtures and abutments, microbial samples were taken from the threaded portion of the abutment, plated, and allowed to culture under appropriate conditions.

Results: Three of the 10 samples in group 1 developed one colony forming unit (CFU) for *A. actinomycetemcomitans*, whereas zero of 10 samples developed CFUs for *P. gingivalis*. Ten of 10 and nine of 10 samples from groups 2 and 3, respectively, developed multiple CFUs for *A. actinomycetemcomitans* and *P. gingivalis*.

Conclusion: This study indicated that differences in implant designs may affect the potential risk for invasion of oral microorganisms into the FAI microgap. *J Periodontol* 2009;80:1991–1997.

KEY WORDS

Bacteria; dental leakage; endosseous dental implantation; microbiology; osseointegration; titanium.

The quantity and quality of the bone surrounding a dental implant influences implant osseointegration and affects the shape and contour of the overlying soft tissues and, consequently, the esthetic outcome. Only with careful considerations of the biologic principles of peri-implant soft and hard tissues, as well as the appropriate selection of implant type and position, can a functional and esthetic treatment result be achieved.^{1,2} Early bacterial colonization around implants by microorganisms associated with periodontitis was reported,^{3–5} and this colonization of implant surfaces and peri-implant tissues can occur within minutes after implant placement.⁶

When a prosthetic abutment is connected to a fixture, a microgap is created between the components. Microorganisms may grow into this fixture–abutment interface (FAI) microgap^{7–9} and set up a bacterial reservoir, resulting in an area of inflamed soft tissue facing the fixture–abutment junction.¹⁰ A study by Callan et al.⁹ used DNA probe analysis to examine the bacterial colonization into the FAI in patients. The authors reported moderate to high levels of eight different putative periodontal pathogens, including *Aggregatibacter actinomycetemcomitans* (previously *Actinobacillus actinomycetemcomitans*) and *Porphyromonas gingivalis*, colonizing the FAI. These findings support the results of other researchers^{4,5} indicating a translocation

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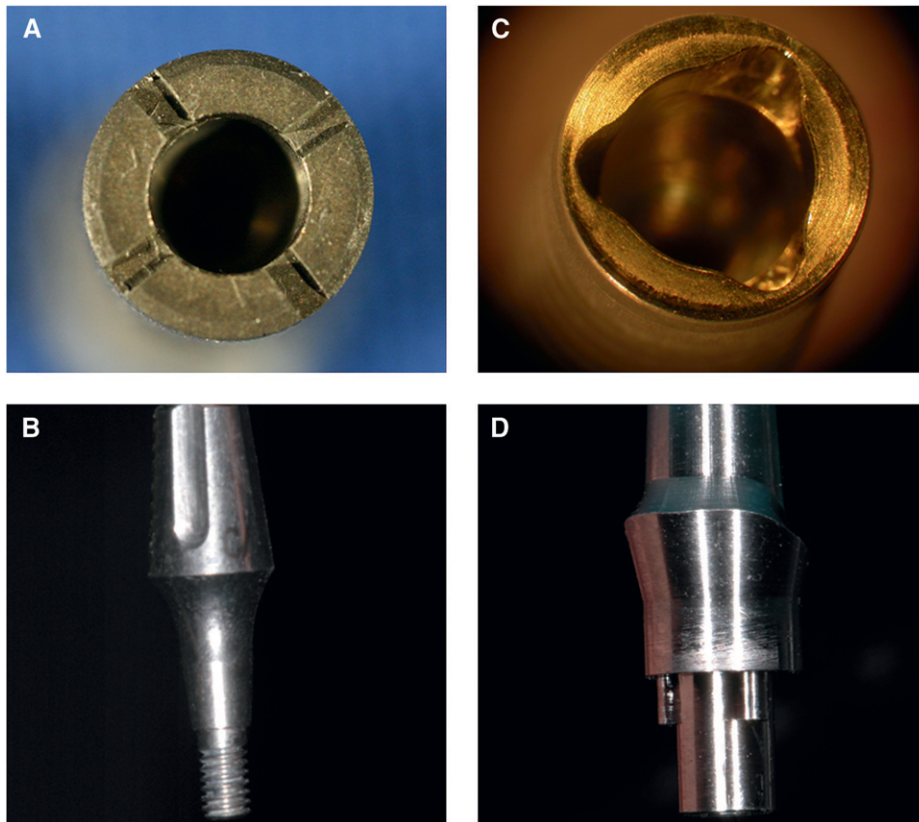


Figure 1.

A) Implant of group 1. **B)** Abutment of group 1. **C)** Implant of group 3. **D)** Abutment of group 3.

of microorganisms from the remaining dentition to implants.

Thus, the presence of an FAI microgap in close relation to bone may have a role in the development of peri-implant inflammation and bone loss.^{11–16} Furthermore, when using one-piece implants that do not have an FAI microgap, minimal early bone resorption was found.¹² This result is consistent with the favorable 8-year outcomes of one-piece implants in patients reported by Buser et al.¹⁷ and suggests the potential impact of the FAI microgap on successful implant therapy.

The design of the FAI may have an impact on the amount of microbial penetration into the internal part of dental implants.^{8,16,18} For instance, in an in vitro study, Quirynen et al.⁸ demonstrated the microbial penetration of the FAI microgap of fixtures with an external hex design. However, there was no comparison among implants with different FAI designs in the study. Jansen et al.¹⁸ reported microbial leakage of 13 different implant–abutment combinations using *Escherichia coli* as indicator bacteria. Among the different implant–abutment combinations, an implant with an internal connection and a silicon washer demonstrated the fewest cases of leakage. In the report by

Callan et al.,⁹ implants from different manufacturers were used without the authors specifying the characteristics of the FAI geometry. Therefore, despite the fact that they reported moderate to high levels of colonization of the FAI microgap by periodontal pathogens, it was not possible to evaluate the impact of the design of the FAI on the microbial penetration. Thus, there is limited information regarding differences in the microbial penetration of the FAI microgap of implants with different internal connection designs.

The aim of this study was to use an in vitro model to assess the potential risk for invasion of oral microorganisms into the FAI microgap in dental implants with different internal connection designs.

MATERIALS AND METHODS

Implant Experiment Groups

For this study, three groups of implants were compared based on their FAI microgap geometry.

Ten implants were tested in each experimental group: group 1 = fixtures with an internal Morse-taper connection[‡] were connected to standard straight abutments[§] with a height of 6 mm (Fig. 1); the abutments were connected to the fixtures with a torque of 25 Ncm according to the manufacturer's protocol; group 2 = identical fixtures and abutments as described in group 1 were used with the exception that prior to fixture–abutment connection, a vertical groove of ~0.5 mm depth was prepared with a fissure bur^{||} on one side of the abutment (Fig. 2). The fixtures and abutments were connected using a torque of 25 Ncm according to the manufacturer's protocol. The introduction of a 0.5-mm groove to the abutment was to ensure microbial penetration to the internal part of the implant, while allowing for the exact same torque for connecting the abutment as the implants in group 1; group 3 = fixtures with a tri-channel internal connection[¶] were connected to 3-mm high abutments[#]

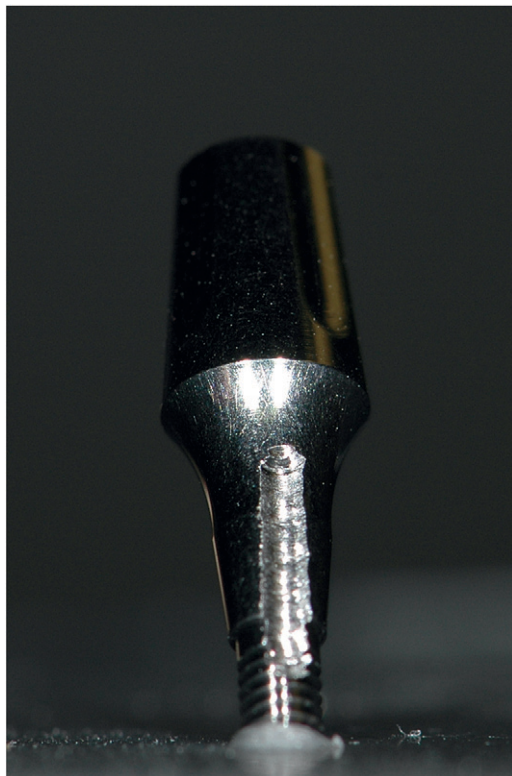
‡ Ankylos Fixtures (B14), 4.5 × 14 mm, Dentsply Friadent, Mannheim, Germany.

§ Standard C/Abutment b/3.0/6.0 straight, Dentsply Friadent.

|| US#557MX, XtremeCut, Brasseler, Savannah, GA.

¶ Nobel Biocare Replace Select, 4.3 × 13 mm, Nobel Biocare, Gothenburg, Sweden.

Esthetic Abutment Nobel Replace RP 3 mm, Nobel Biocare.

**Figure 2.**

Standard straight abutment of group 2 with 0.5-mm vertical groove.

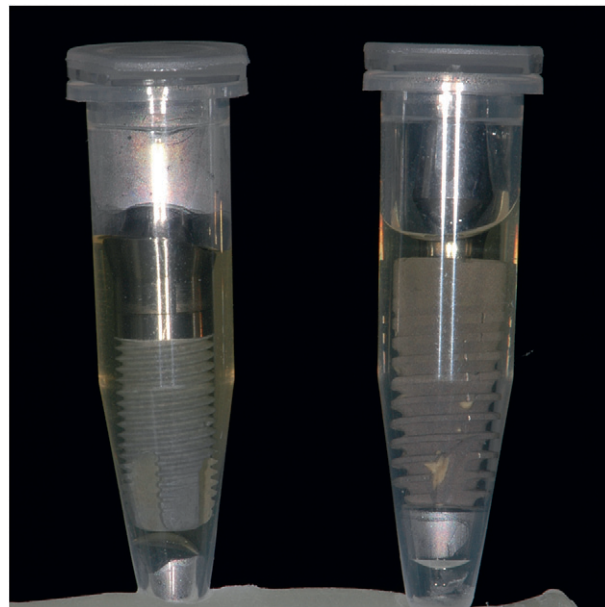
(Fig. 1). The components were connected with a torque of 35 Ncm according to manufacturer's recommendation.

To evaluate the microbial detection techniques, two standard straight abutments with a height of 6 mm were used as negative and positive controls. The negative-control abutment was not connected to a fixture and was not subjected to bacterial culture. The positive-control abutment was not connected to a fixture but was subjected to the same bacterial cultures as groups 1 through 3.

All fixtures and abutments were connected in a sterile environment and placed in a plastic container with a bacterial solution covering the FAI interface and containing microorganisms as described (Fig. 3).

Bacterial Culture Conditions

A. actinomycetemcomitans VT1169 (State University of New York [SUNY] 465 nalidixic acid resistant rifampicin resistant) was grown in liquid tryptic soy broth supplemented with yeast extract and cultured at 37°C in 10% CO₂ to the mid-logarithmic phase. *P. gingivalis* W83 was grown in liquid tryptic soy broth supplemented with hemin, vitamin K, yeast extract, and L-cysteine hydrochloride at 37°C under anaerobic

**Figure 3.**

Implant and abutment of group 1 (right) and group 3 (left) in a plastic container with the bacterial solution.

conditions to the mid-logarithmic phase. Implants were placed in an aliquot of a 1:10 dilution of a 1:1 stock solution of *A. actinomycetemcomitans* VT1169 and *P. gingivalis* W83 and incubated at 37°C under anaerobic conditions for 5 days.

Microbial Sampling and Detection

After disconnection of fixtures and abutments under sterile conditions, microbial samples were taken from the threaded portion of the abutment using sterile cotton swabs. Samples were plated onto tryptic soy-broth agar plates supplemented with yeast extract for the detection of *A. actinomycetemcomitans* and onto tryptic soy-broth agar plates supplemented with hemin, vitamin K, yeast extract, and L-cysteine hydrochloride for detection of *P. gingivalis* colony forming units (CFUs). Plates were incubated at 37°C in 10% CO₂ for 7 and 2 days, respectively. Individual CFUs were counted and recorded.

Statistical Analyses

Median values and interquartile ranges were calculated for the number of CFUs for *A. actinomycetemcomitans* and *P. gingivalis*. In addition, the total number of implants per group exhibiting bacterial colonization of the FAI microgap was calculated. The Kruskal-Wallis test with Dunn comparisons was applied to evaluate differences among the three groups regarding the number of CFUs for *A. actinomycetemcomitans* and *P. gingivalis*. The χ^2 test was used to evaluate differences in the number of implants

exhibiting bacterial colonization of the FAI microgap among the different groups. A *P* value <0.05 was considered significant.

RESULTS

To validate the colonization and detection techniques, abutments similar to those in group 1 were left unassembled and either exposed to bacterial culture or left sterile. Zero CFUs of *A. actinomycetemcomitans* or *P. gingivalis* were detected from sampling of abutments that were left sterile (negative control), whereas 188 CFUs of *A. actinomycetemcomitans* and 113 CFUs of *P. gingivalis* were detected in samples from abutments exposed to bacterial culture (positive control). These data indicate that the conditions for colonization and sample collection were appropriate for the experimental design.

To semiquantitate the ability of *A. actinomycetemcomitans* or *P. gingivalis* to colonize the FAI microgap, CFUs from cultured samplings were quantified (Table 1). Group 1 exhibited significantly lower numbers of CFUs for *A. actinomycetemcomitans* (median: 0; interquartile range: 0 to 1) compared to group 2 (median: 81; interquartile range: 44.5 to 96.5) (difference: -36.25; *P* <0.05) and group 3 (median: 24.5; interquartile range: 11 to 56.5) (difference: -22; *P* <0.05). There was a significant difference in the number of CFUs for *P. gingivalis* between group 1 (median: 0; interquartile range: 0 to 0) and group 2 (median: 55; interquartile range: 35.5 to 96) (difference: -35.8; *P* <0.05). However, the difference in the number of CFUs for *P. gingivalis* between group 1 and group 3 (median: 12, interquartile range: 6 to 29.5) did not reach a statistically significant level (difference: -19.05; *P* >0.05).

The number of implants that had an FAI microgap contaminated with *A. actinomycetemcomitans* and *P. gingivalis* according to the different implant groups is presented in Table 2. Three of ten implants of group 1 had FAI microgaps colonized by *A. actinomycetemcomitans*, whereas none of the implants of this group had FAI microgaps colonized by *P. gingivalis*. In contrast, 10 of 10 implants in group 2 and nine of 10 implants in group 3 had FAI microgaps colonized by both *A. actinomycetemcomitans* and *P. gingivalis*. There was a statistically significant difference for the number of implants that had FAI microgaps colonized by *A. actinomycetemcomitans* between groups 1 and 2 ($\chi^2 = 10.76$; *P* <0.05) and between groups 1 and 3 ($\chi^2 = 7.5$; *P* <0.05). Similarly, there was a statistically significant difference between groups 1 and 2 ($\chi^2 = 20$; *P* <0.05) and between groups 1 and 3 ($\chi^2 = 16.36$; *P* <0.05) regarding the number of implants that had FAI microgaps colonized by *P. gingivalis*.

Table 1.

Median Number of CFUs (interquartile range) for *A. actinomycetemcomitans* and *P. gingivalis* by Implant Group

Group	<i>A. actinomycetemcomitans</i> (CFU)	<i>P. gingivalis</i> (CFU)
1 (n = 10)	0 (0 to 1)*†	0 (0 to 0)‡
2 (n = 10)	81 (44.5 to 96.5)	55 (35.5 to 96)
3 (n = 10)	24.5 (11 to 56.5)	12 (6 to 29.5)

* *P* <0.05; group 1 versus group 2 for *A. actinomycetemcomitans* (Kruskal-Wallis test with Dunn comparisons).

† *P* <0.05; group 1 versus group 3 for *A. actinomycetemcomitans* (Kruskal-Wallis test with Dunn comparisons).

‡ *P* <0.05; group 1 versus group 2 for *P. gingivalis* (Kruskal-Wallis test with Dunn comparisons).

Table 2.

Number of Implants With an FAI Microgap Contaminated With *A. actinomycetemcomitans* and *P. gingivalis* by Implant Group

Group	Number of Implants Contaminated With <i>A. actinomycetemcomitans</i>	Number of Implants Contaminated With <i>P. gingivalis</i>
1 (n = 10)	3*†	0‡§
2 (n = 10)	10	10
3 (n = 10)	9	9

* *P* <0.05; group 1 versus group 2 for *A. actinomycetemcomitans* (χ^2 test).

† *P* <0.05; group 1 versus group 3 for *A. actinomycetemcomitans* (χ^2 test).

‡ *P* <0.05; group 1 versus group 2 for *P. gingivalis* (χ^2 test).

§ *P* <0.05; group 1 versus group 3 for *P. gingivalis* (χ^2 test).

DISCUSSION

The present study shows that the tested dental implants with a Morse-taper internal connection had negligible bacterial penetration down to the threaded part of the FAI under in vitro conditions. Three of 10 implants with this connection (group 1) had one CFU of *A. actinomycetemcomitans*. In addition, none of those implants developed CFUs for *P. gingivalis*. These results seem to be relevant with the geometry of the internal connection because nine of 10 implants with a tri-channel internal connection (group 3) developed multiple CFUs for both *A. actinomycetemcomitans* and *P. gingivalis*. However, there was no statistically significant difference between implants of groups 1 and 3 regarding the number of CFUs of *P. gingivalis*.

Microbial penetration along the internal part of dental implants was reported in some in vitro

studies^{8,16,18} using implants with different geometries of the FAI. For instance, Quirynen et al.⁸ demonstrated that bacterial invasion of the FAI microgap was detected when fixtures and abutments were assembled and installed in a liquid blood medium inoculated with oral microorganisms. Similarly, Jansen et al.¹⁸ reported microbial leakage of 13 different implant–abutment combinations using *E. coli* as the indicator bacteria. In addition, an in vivo study by Quirynen and van Steenberghe⁷ reported the presence of microorganisms in the inner threads of external hex implants. All screw threads in this study harbored significant quantities of microorganisms. Most recently, Callan et al.⁹ described moderate to high levels of eight different periodontopathogenic microorganisms, including *A. actinomycetemcomitans* and *P. gingivalis*, colonizing the FAI using DNA-probe analysis. Interestingly, the study did not detect the colonization of the screw-threads of the abutments. This is in contrast to what was found in the present study, where the threads of the abutments of groups 2 and 3 were colonized with bacteria. This difference may lie in the sample-collection technique. Callan et al.⁹ used paper points for sample collection, whereas in the present study, sterile cotton swabs were used for the microbial sampling. In addition, our group used CFUs, whereas Callan et al.⁹ used DNA-probe analysis.

In the present study, we tested for microbial colonization of the FAI microgap by *A. actinomycetemcomitans* and *P. gingivalis* because both microorganisms have an established role as putative periodontal pathogens.¹⁹ In this context, the bacterial flora associated with peri-implantitis resembles that of chronic periodontitis^{20,21} with significant levels of bacteria such as *Fusobacterium* spp., *Treponema* spp., *Tannerella forsythia* (previously *T. forsythensis*), *Prevotella intermedia*, *A. actinomycetemcomitans*, and *P. gingivalis*. An FAI that is colonized early by putative periodontal pathogens such as *A. actinomycetemcomitans* and *P. gingivalis* may act as a reservoir of bacteria. This contributes to the establishment and maintenance of microflora that resembles that of chronic periodontitis. In fact Quirynen et al.,⁵ using a checkerboard DNA–DNA hybridization and real-time polymerase chain reaction, revealed that a complex microbiota with several pathogenic species was established in peri-implant pockets within 2 weeks after abutment connection. However, the mere presence of putative periodontal pathogens does not indicate a direct etiologic relationship that may lead to a destructive process but may simply indicate a potential pathogenic environment.²²

Few studies^{23–25} focused on the decontamination of the inner-implant cavity of two-stage implants. In a recent study, Paolantonio et al.²⁵ reported that the application of a 1% chlorhexidine gel in the inter-

nal part of the fixture before abutment placement and screw tightening could be an effective method to reduce bacterial colonization over a 6-month period. The authors reported their findings for dental implants with an external hex design that was previously shown to exhibit microbial leakage at the FAI microgap.^{7,8} In addition, Groenendijk et al.²⁴ reported that, the internal implant decontamination with 0.2% chlorhexidine solution led to a reduced gingival index and crevicular fluid flow compared to saline treated controls. Although, the clinical impact of bacterial leakage on the implant survival rate seems to be very limited, as shown by longitudinal and cross-sectional studies,²⁶ the exclusion of bacteria from peri-implant regenerative procedures is considered of paramount importance to obtain clinical success.²⁷

Loading forces on implants may also contribute to the bacterial colonization of the FAI microgap. One disadvantage of the present in vitro study is that loading conditions were not applied. For instance, in an in vitro experiment using loading forces, Steinebrunner et al.¹⁶ evaluated bacterial leakage along the FAI microgap and discovered statistically significant differences between five implant systems with respect to the number of chewing cycles and bacterial colonization. Thus, it is important to confirm or contrast the results of the present study using loading conditions.

The importance of the position, size, and geometry of the implant on marginal bone levels was a subject of various studies^{13,14,28,29} demonstrating that several factors are important regarding peri-implant marginal bone loss. The bacterial colonization of the FAI microgap was reported to be one of these factors. The potential colonization of oral microorganisms of the FAI microgap is presumably impacted by multifactor conditions like the precision fit between the implant components, torque forces when the components are connected, and loading forces when the implants are in function. Indeed, Zipprich et al.³⁰ evaluated the dynamic behavior of dental implants with different designs of the fixture–abutment connection with respect to microbial colonization. The authors reported the micromovement of the fixture–abutment complex of implants loaded at an angle of 30° when a force of up to 200 N was applied. Interestingly, the same implant system used in our experiment was one of four systems reported to exhibit no micromovement when loaded at 100 N and one of two systems showing no measurable microgap when loaded at 200 N.³⁰ The authors speculated that certain implant designs would minimize the pumping effect between the fixture and the abutment, thus preventing bacterial colonization of the FAI interface.

CONCLUSIONS

The present study indicated that differences in implant design may affect the potential risk for colonization of oral microorganisms into the FAI microgap. Also, this study indicated a negligible bacterial penetration down to the threaded part of the FAI of dental implants with a Morse-taper connection, although the effects of functional loading still need to be assessed.

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