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Bacterial colonisation of interior implant threads with and without sealing

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Premature loss of dental implants is due, apart from mechanical factors, to germ-related inflammation. Gaps and hollow spaces within the implant system, for example the gap between implant and abutment in the two-part implant system, may provide a bacterial reservoir causing or maintaining inflammation. The bacterial spectrum involved is similar to that found in periodontitis.
This in vitro *study aimed to scrutinise the capability of* Porphyromonas gingivalis *(DSM 20709), the bacterium blamed for inducing peri-implantitis, to pass the implant/abutment gap in titanium implant systems used for orthodontic anchorage and to remain vital in the interior. Additionally, the* in vitro *effectiveness of gutta percha for gap sealing was examined. Twelve titanium implants (Straumann®, diameter: 3.3 mm, length 5.5 mm) were provided with abutments at a defined torque (20 Ncm), six of which were sealed with gutta percha before screwing in the abutment. Subsequently the implants were placed in a nutrient solution (thioglycolate boullion with haemin-menadione solution) that contained* Porphyromonas gingivalis*. Microbiological specimens were sampled from the implant interiors after 24 and 72 hours and analysed using culture methods. There was evidence that penetration of the periodontal pathogen* Porphyromonas gingivalis *to the implant interior may occur as early as after 24 hours. Microbes were also detected in the interior of implants sealed with gutta percha. The abutment/implant interface* in vitro *provides a microbiological leakage for the prospective peri-implantitis-inducing bacterium* Porphyromonas gingivalis*. Survival of the bacterium is possible in the interior, so that development of a bacterial reservoir is assumed. This* in vitro *trial produced no evidence that sealing with gutta percha is an effective means to prevent secondary bacterial colonisation in the implant interior.*

Key words: bacterial colonisation, implant interior, bacterial reservoir, sealing

INTRODUCTION

Beside mechanical factors, specific microbes may be responsible for premature implant loss. Multi-part implant systems display a gap between implant and abutment which permits new bacterial colonisation on the one hand and represents a bacterial reservoir on the other [6] (Fig. 1, 2). The size of these gaps ranges from 1 to 50 μm, depending on the respective implant system and the screwing-in torque [1, 3]. Although the size of these gaps is relatively small as

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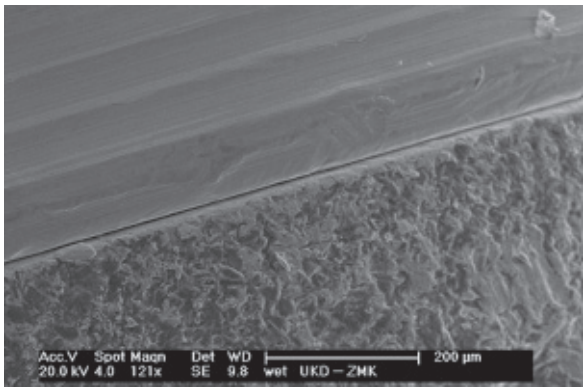

Figure 1. SEM image of implant/abutment interface at 121-fold magnification. The gap area between abutment and implant is clearly visible.

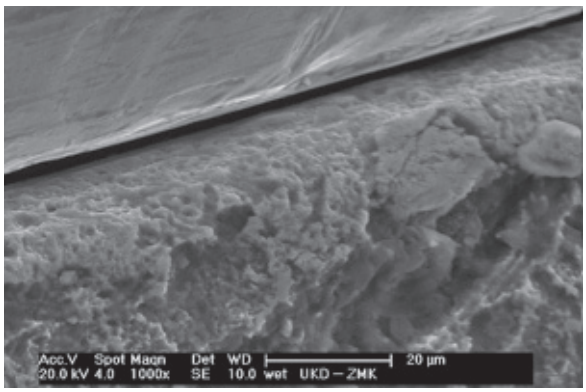

Figure 2. SEM image of implant/abutment interface at 1000-fold magnification. The gap width between implant and abutment amounts to about 2.5 μm.

compared to other dental restorations such as crowns, there are microleakages, which permit bacterial penetration. Bacterial colonisation of the interior of two-part implants induced by contamination during insertion or abutment installation may be prevented by relatively simple measures. Secondary penetration and vitality of bacteria within the implant interior lead to maintenance of a bacterial reservoir, which is more difficult to eliminate. Owing to vertical and transversal occlusal forces, gap width may additionally change and a pumping effect may develop, transporting even primarily immobile micro-organisms from the interior to the exterior and vice versa [7].

The study aimed to examine whether or not the peri-implantitis-associated bacterium *Porphyromonas gingivalis* (DSM 20709) penetrates to the interior through an existing microleakage of this two-part implant system (Straumann®, diameter: 3.3 mm, length 5.5 mm) even under *in vitro* conditions and whether gutta percha is primarily suited for sealing.

MATERIAL AND METHODS

Twelve titanium implants (Straumann®, diameter: 3.3 mm, length 5.5 mm) and 12 abutments were divided into two groups. In group I implant and abutment were screwed together at a defined torque (20 Ncm) under sterile conditions, while group II underwent additional sealing between implant and abutment using gutta percha. All implants were simultaneously placed in a nutrient solution (thioglycolate boullion with haemin-menadione solution) containing *Porphyromonas gingivalis* (DSM 20709). After 24 hours three implants of each group were picked, cleaned and opened. The samples from the implant interior were placed in a fresh thioglycolate boullion with haemin-menadione solution. Additionally, the specimens were pour-plated on a Schaedler agar with 5% sheep blood (Becton Dickinson Co.®). After 72 hours, the remaining implants of both groups were gathered for cultivation using the same procedure.

RESULTS

After about 4 days a distinct turbidity of the thioglycolate bouillon with haemin-menadione solution and marked growth of *Porphyromonas gingivalis* on the Schaedler agar was observed in all three implants of group I. Slight bacterial growth in the bouillon and agar was shown in the three implants of group II (with sealing). The group I implants picked up after 72 hours exhibited very strong growth of *Porphyromonas gingivalis* in boullion and agar. Similarly, the three implants of group II (with sealing) showed clear bacterial growth in boullion and agar.

In summary, the microleakage occurring in the gap area is sufficient for penetration of the peri-implantitis-associated bacterium *Porphyromonas gingivalis* under *in vitro* conditions, (i.e., devoid of the pumping effect resulting from occlusal implant loading). Sealing with gutta percha is not safe.

DISCUSSION

The impact of mechanical and microbiological factors on premature bone resorption and implant loss is still a subject of controversy. Mechanical stress resulting from occlusal overloading primarily affects the marginal bone. Of particular importance are the magnitude of the force, the frequency of force exposure, the direction of the force, implant and supraconstruction geometries and the presence of parafunctions [4, 8]. The bacterial spectrum blamed for the inflammatory response in peri-implantitis is similar to that of periodontitis. Neither radiographic

defect morphology nor probing depth nor microbiological analysis lends themselves to diagnostic clarification of pathogenesis. Nevertheless, there is a significant correlation between peri-implant loss of attachment and increasing bacterial counts and detection of *Porphyromonas gingivalis*, *Prevotella intermedia* and *Fusobacterium nucleatum* associated with a shift of the entire bacterial spectrum to gram-negative species [5]. The micro-gap between abutment and implant in two-part implants is not only a mechanical weak point but permits penetration of micro-organisms and the development of a bacterial reservoir. Preventive sealing of the implant/abutment interface is anyway not expected to increase implant survival substantially in view of the high rate of clinical implant success and the infrequent occurrence of a manifest peri-implantitis. Rather, sealing is better considered a supporting measure in the therapy of already manifest peri-implantitis [2].

CONCLUSION

The cavity between implant and abutment represents a bacterial reservoir. Sealing with gutta percha is ineffective in preventing penetration of the periodontal pathogen *Porphyromonas gingivalis*. Additional abutment deflection with increased gap formation is to be expected under load, even with secondary sealing. Thus microleakage further increases. Efforts should aim at improved primary sealing of the implant/abutment interface, for instance by cold welding.

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