The impact of the type and configuration of abutments and their (repeated) removal on the attachment level and marginal bone

Key words: abutment disconnection, platform switching, soft tissue–implant interface, transmucosal components

Purpose: The quality and stability of the soft tissue–implant interface is most likely of paramount importance for the preservation of marginal bone and for the long-term prognosis of oral implants. The aim of the present review was to screen existing data in the literature concerning the influence of transmucosal components’ composition, type, design and disconnection on peri-implant tissues.

Results: The influence of the type of implant system (one-piece or soft tissue level implants versus two-piece or bone level implants) has been poorly studied in humans, and data from animal studies are controversial; both concepts demonstrated comparable long-term stability of peri-implant tissues. The chemical composition of abutments has been mostly studied in animals, with the conclusion that only a few materials (namely titanium, aluminium and zirconium oxides) allow the proper formation of a soft tissue interface. However, there is a severe lack of information about the clinical impact of this parameter, as well as of surface contamination of abutments. Mobility of transmucosal components has been shown to increase marginal bone loss in animals, while the influence of abutment disconnection is more controversial. Only one clinical study suggests that a ‘one abutment – one time’ technique preserves marginal bone. Studies investigating the influence of platform switching suggest that using an abutment narrower than the implant’s platform could have a positive effect on the fate of marginal bone. But those studies are extremely heterogenous and their results controversial.

Conclusion: There is still a severe lack of information about the clinical impact of implant type, of implant/abutment connection, and of abutment composition and stability on peri-implant bone remodelling.

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Introduction

Soft tissue interface

The soft tissue–implant/abutment interface has been evaluated in animals and has a dimension of 3 to 4 mm in the apico-coronal direction. The interface consists of two zones: one of epithelium that covers about 2 mm of the surface, while the rest is devoted to connective tissue adhesion.

Both of these tissues contribute to the establishment of the so-called ‘biological width’, which may prevent oral bacteria and their products from penetrating into the body.
Junctional epithelium

Due to its capacity to proliferate and to move on surfaces, the epithelium found at the border of the incision creeps over the bridge of the fibrin clot/granulation tissue that rapidly starts forming after implant/abutment installation. Upon reaching the surface of the implanted component, it moves in a corono-apical direction, giving rise to a junctional epithelium about 2 mm long\textsuperscript{9,10}.

Once the epithelial cells have reached the implant surface, their attachment occurs directly via a basal lamina (<200 nm) and the formation of hemidesmosomes\textsuperscript{11-17}. Hemidesmosomes can be formed after 2 to 3 days of healing\textsuperscript{18}.

It is generally recognised that the epithelium lining the peri-implant sulcus shares many structural, ultrastructural and functional characteristics with the corresponding gingival tissue. Studies conducted in humans\textsuperscript{19-21} indicate that the epithelium surrounding transmucosal oral implants possesses patterns of differentiation and function similar to gingival epithelium around teeth.

The presence of granulation tissue adhering to the surface of transmucosal implant components is considered the principal factor that stops the epithelium from moving further apically\textsuperscript{9}. The role of the connective tissue in preventing epithelium downgrowth has been clearly demonstrated in animal models\textsuperscript{22,23}. It seems that mature connective tissue interferes more effectively than granulation tissue with epithelial downgrowth\textsuperscript{23}. At initial phases of healing, the quality and stability of the fibrin clot adhesion to the surface of the transmucosal components most probably plays a role in the formation and positioning of the junctional epithelium\textsuperscript{24}.

Connective tissue adhesion

Apart from the orientation of the fibres, the major difference between the connective tissue at teeth and at implants is related to their connection to the natural or artificial root surface.

Around teeth, the dento-gingival collagen fibres are firmly inserted into the cementum and the bone, and oriented perpendicular or oblique to the tooth surface. This serves as a barrier to epithelial migration, and thus impedes bacterial invasion\textsuperscript{25,26}.

In contrast, implants lack cementum. The orientation of the ‘attachment’ fibres in the supracrestal soft tissue compartment is parallel to the implant surface and, more importantly, they are not inserted in the implant surface\textsuperscript{5-7,27,28}.

As a consequence, the connective tissue adhesion at implants is considered as having a poor mechanical resistance as compared to that around natural teeth\textsuperscript{29}. In other words, the mucosa at implants can hardly be qualified as ‘attached’.

Improving the quality and preserving the stability of the soft tissue–implant interface is thus most likely of paramount importance for the short- and long-term prognosis of oral implants. This can theoretically depend on numerous parameters, in particular the type of implant system (bone level versus soft tissue level), the chemical composition and surface texture of the abutment, the (repeated) removal or loosening of the abutment, the type of abutment (platform switch or not), and the mobility/stability of the peri-implant soft tissues.

Implant system

In ‘bone level’ implant systems, the transmucosal component (the abutment) dedicated to soft tissue integration is a separate part from the implant body. The interface between the transmucosal component and the implant is generally located in the vicinity of the buccal bone level.

In ‘soft tissue level’ implant systems, the transmucosal component facing the soft tissues is an integral part of the implant. The biological width will thus be facing this transmucosal neck, which is supposed to be as biocompatible as the implant, which cannot become loose or be removed, and which harbours no microgap.

Comparative studies were performed in dogs to determine the influence of implant design on soft tissue integration. Abrahamsson et al\textsuperscript{4} demonstrated that the dimensions of the junctional epithelium and of the connective tissue are similar on soft tissue level and on bone level implants. In addition, their position relative to the bone crest was also comparable, with the soft tissue integration located on the smooth implant’s neck on one-piece implants and at the abutment level on two-piece implants.
With the same experimental conditions, but after 6 months of undisturbed plaque accumulation, it was shown\(^3\) that the extent of the plaque-related inflammatory infiltrate was comparable around one- and two-piece implants.

Using experimental implants with either a one-piece (soft tissue level) or a two-piece (bone level) design, Hermann et al\(^{30,31}\) showed significantly more apical migration of the soft tissues and more marginal bone resorption with two-piece implants, suggesting a role for the subgingival position of the abutment/implant connection (the so-called ‘microgap’) on tissue remodelling. It must be noted that in this experiment, all two-piece implants were clinically and histologically surrounded by an intense inflammatory process. This is in contradiction with several other animal studies\(^1-6,10,32-36\) in which soft tissue integration occurred at the abutment level.

An inflammatory cell infiltrate has been demonstrated around bone level implants, but not at soft tissue level ones, in the close vicinity of the abutment–implant interface\(^{36}\). This infiltrate (abutment ICT) does not impair effective soft tissue integration.

It has not been clearly shown if the bacterial contamination of the internal components of some two-piece implant systems\(^{37}\) is responsible for the inflammatory cell infiltrate seen at the abutment–implant interface. So, while it has been shown that the seal provided by a locking taper connection at the implant–abutment interface effectively impairs bacterial leakage\(^{38}\), the abutment ICT has been shown both at implants systems with an external implant–abutment connection as well as at systems with an internal Morse taper connection, but not at one-piece implants\(^1,3,4\).

The size of the inflammatory infiltrate seems to be rather limited in size; on the order of 0.5 mm in diameter in dogs\(^1,3,39\) as well as in humans\(^40\).

In animal studies, the abutment ICT was found to be linked to increased bone loss compared to soft-tissue level implants for some authors\(^39\), but not for others\(^1-3\).

Several clinical studies have demonstrated long-standing stability of the soft tissue interface and comparable marginal bone remodelling at both soft tissue level and bone level implant systems\(^61-45\). However, no clinical study is available comparing the two concepts with the same intra-bony design and surface in terms of bone remodelling and attachment loss.

### Abutment composition

The reaction of cells and tissues to implanted foreign bodies depends on the material’s properties and its behaviour upon contact with body fluids. It must be noted that the chemical composition of the bulk material is sometimes significantly different from that of the surface at the interface with the living tissues. Some materials demonstrate surface oxidation (such as titanium that exhibits instantaneously a surface layer of titanium oxide), while the mode of preparation or of sterilisation of others results in chemical contamination of the surface.

### In vitro studies

**Ti, gold, Al\(_2\)O\(_3\) and dental ceramic**

Räisänen et al\(^{46}\) studied, in vitro, how epithelial cells attach to five different material surfaces (titanium, Ti6Al4V titanium alloy, dental gold alloy, dental porcelain and aluminium oxide). Epithelial cells adhered and spread more avidly on metallic surfaces (commercially pure [c.p.] titanium, Ti6Al4V titanium alloy and dental gold alloy) than on ceramic surfaces (dental porcelain and aluminium oxide). Well-organised focal contacts and pre-hemidesmosomes were found on metallic surfaces, but not on porcelain and aluminium oxide.

Previously, Jansen et al\(^{47}\) found focal contacts, hemidesmosome-like structures and extracellular matrix contacts between epithelial cells and titanium, gold, hydroxyapatite and carbon apatite.

Simion et al\(^{48}\) examined the human gingival fibroblast–implant material interface in vitro using a specific model that has not been validated elsewhere. Their results showed effective cell growth on acid-etched titanium and titanium alloy, on gold and porcelain, and a ‘tenacious’ cell adherence only on etched titanium.

When Ti6Al4V was compared to c.p. titanium\(^{49}\), gingival fibroblasts demonstrated a rounded cell shape and a reduced area of spreading on the alloy, presumably because of minor toxicity to vanadium or aluminium.

Titanium nitride also proved to be suitable for fibroblast adhesion and growth\(^{50}\).
Modified dental ceramics

Kokoti et al\textsuperscript{51} modified the chemical composition and surface morphology of dental ceramics and evaluated them, in vitro, for their ability to support fibroblast attachment and proliferation. Four modified ceramics were constructed from body or shoulder porcelain after treatment with CaO, or CaO and P\textsubscript{2}O\textsubscript{5}. All modified ceramics promoted cell proliferation compared to controls. Shoulder-modified ceramics proved to be the most effective.

HA surfaces

Kasten et al\textsuperscript{52} found higher epithelial cell adhesion on HA compared to c.p. titanium, but the extremely low number of samples limits the significance of their results. Human gingival fibroblast attachment to c.p. titanium proved to be significantly higher than to non-porous and porous hydroxyapatite\textsuperscript{53}.

Animal studies

Ti, gold, Al\textsubscript{2}O\textsubscript{3} and dental ceramic

Abrahamsson et al\textsuperscript{4} observed, in a dog model, that abutments made of c.p. titanium or highly sintered aluminium-based ceramic (Al\textsubscript{2}O\textsubscript{3}) allowed the formation of a mucosal attachment that included one epithelial and one connective tissue portion of about 2 mm and 1.5 mm, respectively.

In contrast, at gold or dental porcelain, no mucosal attachment formed at the abutment level, but the soft tissue margin receded and bone resorption occurred. The mucosal barrier was thus partially established at the fixture portion of the implant.

HA surfaces

Çomut et al\textsuperscript{54} observed in a dog model effective formation of a mucosal attachment on c.p. titanium and on HA-coated titanium. Other studies indicate a favourable soft tissue response to dense HA\textsuperscript{55-56}.

Zirconia

Kohal et al\textsuperscript{57} compared bone and soft tissue integration of rough titanium versus zirconia implants in a monkey model. They found effective formation of a mucosal attachment at both implant materials, the mean length of connective tissue being 1.5 mm on zirconia versus 2.4 mm on titanium, without evidence of perpendicular fibres. These differences did not reach the level of statistical significance.

Welander et al\textsuperscript{58} confirmed, in dogs, the quality of the soft tissue attachment to titanium abutments and showed that zirconium oxide abutments are as effective as titanium. In contrast, an apical shift of the biological width and bone resorption occurred again at Au/Pt alloy abutments.

Clinical studies

Degidi et al\textsuperscript{59} conducted a comparative immunohistochemical evaluation of peri-implant soft tissues of titanium and zirconium oxide healing caps in five patients. Statistically significant differences were observed, with an overall lower inflammatory level in tissues surrounding zirconium oxide healing caps than at titanium caps.

In a systematic review, Linkevicius and Apse\textsuperscript{60} pointed out a lack of information about the clinical performance of zinc oxide and gold alloy abutments compared to titanium abutments.

Surface contamination of abutments

The ultimate goal of procedures to clean abutments (re-use of healing abutments or cleaning of abutments modified in the dental laboratory) should be to remove the contaminants and restore the original composition of the surface oxide without changing the surface topography, either after the fabrication process, after handling in the dental laboratory, or when transgingival components are re-used.

Although specific protocols have been developed, it proves to be rather difficult to effectively clean a contaminated titanium surface, most probably because of the strong binding of proteins and amino acids\textsuperscript{51-53}.

Krozer et al\textsuperscript{64} showed that rinsing in water, saline solution or 5% H\textsubscript{2}O\textsubscript{2} did not remove the amino alcohol from the surface, while exposure to ozone resulted in complete removal of the adsorbed amino alcohol. The results show that the amino alcohol
used forms a stable and dense film at the implant surface in vitro. Presence of such a film most likely prevents reintegration from occurring at the implant–tissue interface in vivo, but this has not been clinically investigated.

Vezeau et al. evaluated the surface changes and effects on in vitro cell attachment and spreading on prepared c.p. titanium caused by multiple exposures to common sterilisation methods. In vitro analysis of cell attachment and spreading using gingival fibroblasts was performed. Results indicated that steam autoclave sterilisation contaminated and altered the titanium surface, resulting in decreased levels of cell attachment and spreading in vitro.

Keller et al. had also observed that sterilisation of c.p. titanium surfaces by steam autoclaving caused surface alteration and contamination, and a reduction of fibroblast cell attachment and spreading, in vitro.

Zöller and Zentner studied in vitro the influence of contamination of titanium by saliva or serum on initial attachment of fibroblasts. Pre-treatment with serum showed a consistent enhancing effect on cell adhesion. In contrast, pre-treatment with saliva significantly diminished cell adhesion.

These in vitro results suggest that exposure of transgingival components to saliva at placement might inhibit adhesion of gingival fibroblasts and thus indirectly induce epithelial downgrowth, a hypothesis not yet supported by clinical evidence.

Kawahara et al. investigated in vitro cell contact with titanium surfaces and adhesive strength of epithelial cells and fibroblasts under the influence of dental plaque extracts. Their results suggest that the difference in growth, contact and adhesive strength of the epithelial and fibroblastic cells to dental titanium surfaces may promote apical epithelialisation under exposure to dental plaque.

Sennerby and Lekholm placed titanium abutments in rats after intraoral contamination in humans for 1 min or 2 weeks and either rinsing in saline or ultrasonic treatment in amino alcohols. All pre-contaminated abutments induced an altered tissue response compared to pristine abutments, irrespective of the cleaning procedure.

In contrast, Ericsson et al. failed to show differences in soft tissue reaction between pristine titanium abutments with varying surface roughness and corresponding contaminated abutments.

### Influence of abutment disconnection or removal

#### Animal studies

Hermann et al. and King et al. demonstrated in dogs that the size of the microgap between implants and abutments has little influence on marginal bone remodelling, whereas micromovements of the abutments induce significant bone loss, independent of the microgap’s size. This strongly suggests that the mechanical disruption of the soft tissue interface is of importance. The presence of a transmucosal component at two-piece implant systems can lead to intentional or unintentional disconnections of this abutment.

Based on these results, it can be hypothesised that unintentional abutment loosening will lead to a disruption of the soft tissue integration and to increased bone remodelling, but it has not been clinically demonstrated so far.

It has also been shown that five intentional abutment disconnections and reconnections (after alcohol disinfection) with monthly intervals induce an apical repositioning of the soft tissues and marginal bone resorption. In contrast, a single shift of a healing abutment and replacement by a final abutment proved to induce no marginal bone remodelling.

Hermann and co-authors also performed five disconnections of healing abutments in dogs with 1-month intervals, but without alcohol disinfection. They observed no noticeable influence.

#### Clinical studies

Canullo et al. compared the fate of the peri-implant marginal bone from surgery to 3 years later at implants placed in fresh extraction sockets and having received either a provisional or a definitive titanium abutment. All implants immediately received a provisional dental crown, and all abutments were platform-switched. Several dis/reconnections of the abutments were performed in the first group only. The ‘one abutment – one time’ group exhibited significantly less marginal bone loss.
Influence of platform switching

Using a transmucosal abutment narrower than the implant's platform is considered to have a positive effect on peri-implant marginal bone preservation. Some finite element studies have shown that platform switching could shift the stress concentration area away from the cervical bone to the implant interface, but this has not been confirmed by others. Besides this mechanical hypothesis of a potential positive effect of platform switching, a biological hypothesis also exists: at platform-switched implants, the inflammatory cell infiltrate observed at bone level implants (see above) could be laterally displaced and thus moved away from the marginal bone.

In a recent systematic review and meta-analysis that included 10 studies, Atieh et al. showed that platform switching may preserve inter-implant bone height and soft tissue levels, the degree of marginal bone resorption being inversely proportional to the extent of the abutment-implant mismatch. An implant-abutment diameter difference ≥0.4 mm was associated with a more favourable marginal bone response (0.5 mm less marginal bone loss).

Nevertheless, extreme heterogeneity can be observed in the 10 included studies: some authors compared internal Morse taper connections to external hexagons, others compared identical implants but placed them at different levels relative to the bone crest, and the design of the threads and/or the transmucosal necks was often different between groups. Some studies used a 1-stage procedure, others 2-stage procedures, some used implants with a transmucosal neck, others not.

Furthermore, only limited information is available concerning the prosthetic procedures that were used, for instance the number of disconnections of the transmucosal components or the material used for the abutments. In the Canullo et al. studies, the transmucosal components or the material used, for instance the number of disconnections of the abutments were made, and radiographs seem to show that non-biocompatible materials were used at transmucosal levels.

Only two studies compared bone level implants placed at the same vertical level with adequate abutments. Cappiello et al. showed a 0.7 mm difference in marginal bone resorption between matching and switched abutments after 1 year of function. Vigolo and Givani also showed a significant difference in marginal bone loss after 1 year, but the difference was not significant anymore at 2, 3, 4 and 5 years of function.

It can be concluded that further clinical studies are necessary to elucidate the impact of platform switching on peri-implant tissue remodelling.

Conclusions

Despite the fact that the quality and stability of the soft tissue interface with implants or abutments is most likely of paramount importance for the short- and long-term prognosis of oral implants, there is still a severe lack of information about the clinical influence of implant type, of implant/abutment connection, of abutment composition and stability, and of soft tissue type and stability on peri-implant bone remodelling.

References


