

Editors: N. Donos, S. Barter, D. Wismeijer

ITI Treatment Guide

Volume 12

<u>Peri-Implant Soft-Tissue</u> <u>Integration and Management</u> Authors: M. Roccuzzo A. Sculean

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<u>Peri-Implant Soft-Tissue</u> <u>Integration and Management</u>



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"... to serve the dental profession by providing a growing global network for life-long learning in implant dentistry through comprehensive quality education and innovative research to the benefit of the patient."



<u>Preface</u>

The field of implant dentistry has developed significantly in recent years. As a result, practitioners are faced with higher demand as well as expectations from their patients, not only in terms of successful implant treatment but also the long-term esthetics of the final result. At the same time, the growing number of patients with soft-tissue-related problems is an undeniable reality. This volume therefore provides readers with guidance and reference material for the treatment of patients with mucogingival deformities. Its aim is to reduce the risk of biological and esthetic complications around dental implants, and to ensure predictable and stable longterm treatment outcomes.



As with every ITI Treatment Guide, this volume illustrates clinical approaches to peri-implant soft-tissue integration and management, step by step, in a variety of clinical situations. We hope this volume provides clinicians support and orientation towards optimal long-term maintenance of peri-implant soft-tissue health and esthetics.

N. Donos

S. Barter

D. Wismeijer

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Editors and Authors

Editors:

Nikolaos Donos DDS, MS, FHEA, FDSRC, PhD Professor, Head and Chair, Periodontology and Implant Dentistry Head of Clinical Research Institute of Dentistry, Barts and The London School of Medicine and Dentistry Queen Mary University of London Turner Street London E1 2AD United Kingdom n.donos@qmul.ac.uk Stephen Barter BDS, MSurgDent, RCS Specialist in Oral Surgery

Honorary Senior Clinical Lecturer/Consultant Oral Surgeon Centre for Oral Clinical Research Institute of Dentistry, Barts and The London School of Medicine and Dentistry Turner Street London E1 2AD United Kingdom s.barter@gmul.ac.uk

Daniel Wismeijer Professor, DMD Oral Implantology and Prosthetic Dentistry Private Practice Zutphensestraatweg 26 6955 AH Ellecom Netherlands Danwismeijer@gmail.com

Authors:

Mario Roccuzzo DMD, Dr med dent Private practice (periodontology) Corso Tassoni 14 10143 Torino (TO) Italy mroccuzzo@icloud.com

Anton Sculean

Professor, Dr med dent, Dr h c, MSc Executive Director and Chairman Department of Periodontology University of Bern School of Dental Medicine Freiburgstrasse 7 3010 Bern Switzerland anton.sculean@zmk.unibe.ch



Contributors

Sofia Aroca Dr med dent. PhD Private practice 35, Rue Franklin 78100 Saint Germain en Laye France sofiaaroca@me.com Paolo Casentini DDS. Dr med dent Private practice Via Anco Marzio 2 20123 Milano (MI) Italy paolocasentini@fastwebnet.it Raffaele Cavalcanti DDS, PhD Adjunct Professor of Periodontology University of Catania, CLMOPD, Via S. Sofia 78, 95123 Catania (CT), Italy and Private practice Studio Odontoiatrico Associato Cavalcanti & Venezia (periodontology, implantology, oral surgery) Via Giuseppe Posca 15 70124 Bari (BA) Italy raffaelecavalcanti@gmail.com Nikolaos Donos DDS, MS, FHEA, FDSRC, PhD Professor, Head and Chair, Periodontology and Implant Dentistry Head of Clinical Research Institute of Dentistry, Barts and The London School of Medicine and Dentistry Queen Mary University of London **Turner Street** London E1 2AD United Kingdom n.donos@qmul.ac.uk

Daniel Etienne Dr chir dent. MSc Private practice 1, Avenue Bugeaud 75116 Paris France etienne@paro-implant.com Jason R Gillespie BS DDS MS Private prac:ce (Prosthodon:cs) 105 W El Prado Dr San Antonio, TX 78212-2024 United States of America jason@gillespie.dental Alfonso Gil DDS, MS, PhD **Resident Physician** Clinic of Reconstructive Dentistry Center of Dental Medicine University of Zurich Plattenstrasse 11 8032 Zurich Switzerland alfonso.gil@zzm.uzh.ch Christoph Hämmerle Professor, Dr med dent, Dr h c Chair Clinic of Reconstructive Dentistry Center of Dental Medicine University of Zurich Plattenstrasse 11 8032 Zurich Switzerland christoph.hammerle@zzm.uzh.ch Vincenzo Iorio-Siciliano DDS, MS, PhD Department of Periodontology University of Naples Federico II Via Sergio Pansini 5 80131 Napoli (NA) Italy enzois@libero.it



Ronald Jung Professor, Dr med dent, PhD Head, Oral Implantology Clinic of Reconstructive Dentistry Center of Dental Medicine University of Zurich Plattenstrasse 11 8032 Zurich Switzerland ronald.jung@zzm.uzh.ch Eduardo Lorenzana DDS, MSc Private practice (periodontology) 3519 Paesano's Parkway Suite 103 San Antonio, TX 78231-1266 United States of America drlorenzana@yahoo.com Neil MacBeth BDS, MFGDP, MGDS RCS, MFDS RCS, FFGDP (UK), MSc, FDS RCS (Rest Dent), CDLM, RAF Consultant in Restorative Dentistry -**Defence Primary Health Care** Clinical Senior Lecturer in Periodontology Institute of Dentistry, Queen Mary University of London Institute of Dentistry, Barts and The London School of Medicine and Dentistry Turner Street London E1 2AD United Kingdom n.d.macbeth@gmul.ac.uk Kurt Riewe DDS

Private practice, Stone Oak Dental 335 E Sonterra Blvd Suite 150 San Antonio, TX 78258-4295 United States of America kurt.riewe@gmail.com Shakeel Shahdad BDS, MMedSc, FDS RCSEd, FDS (Rest. Dent.) RCSEd, DDS, FDT FEd Consultant in Restorative Dentistry Barts Health NHS Trust The Royal London Dental Hospital and Honorary Clinical Professor in Oral Rehabilitation and Implantology Barts and The London School of Medicine and Dentistry Queen Mary University of London **Turner Street** London, E1 1DE United Kingdom shakeel.shahdad@nhs.net Daniel Thoma

Professor, Dr med dent Vice-Chairman Head, Reconstructive Dentistry Clinic of Reconstructive Dentistry Center of Dental Medicine Vice Chairman, Center for Dental Medicine University of Zurich Plattenstrasse 11 8032 Zurich Switzerland daniel.thoma@zzm.uzh.ch

Pietro Venezia DDS Adjunct Professor Department of Prosthodontics University of Catania (Italy) and Private practice Via G. Posca, 15 70124 Bari (BA) Italy pierovenezia@gmail.com



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1 Introduction

M. Roccuzzo

In the earlier days of implant dentistry, osseointegration was considered to be a sufficient condition for long-term successful implant rehabilitation. With time, however, it became evident that soft-tissue integration is of significant importance and that the formation of an early and long-standing effective mucosal barrier, capable of biologically protecting the peri-implant structures, is essential. This soft-tissue barrier is mainly the result of a wound-healing process that results in an effective interface between "living tissues" and a "foreign body" (Rompen and coworkers 2006).

Whether the presence of a minimum amount of keratinized mucosal (KM) is necessary for the long-term maintenance of peri-implant health has been controversial for many years. Several researchers have found that insufficient KM may be correlated with plaque accumulation, bleeding on probing, discomfort when brushing, mucosal recession, and peri-implant mucositis (Bouri and coworkers 2008; Boynueğri and coworkers 2013; Chung and coworkers 2006; Roccuzzo and coworkers 2016). Other researchers were unable to obtain similar findings (Frisch and coworkers 2015), with some even suggesting that KM may not be essential in the presence of scrupulous oral hygiene and rigorous compliance with a professional maintenance regimen (Lim and coworkers 2019). On the other hand, complete osseointegration and perfect soft-tissue integration are not necessarily correlated with successful esthetic rehabilitation of a missing tooth or teeth. Indeed, success criteria for esthetically sensitive areas must include measurements of the peri-implant mucosa, as well as the restoration and its relationship to the surrounding dentition (Belser and coworkers 2004).

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Apart from the prosthetic aspects, sufficient horizontal and vertical volume is also essential for long-term esthetic soft-tissue stability. Where soft-tissue deficiencies exist, appropriate augmentation procedures may be required for comprehensive rehabilitation. Recent advances in implant dentistry have provided clinicians with various treatment options to treat peri-implant soft-tissue defects. At the same time, though, soft-tissue grafting procedures are of moderate to high complexity and may be associated with a significant risk of complications. For this reason, various step-by-step procedures have been outlined and illustrated by individual case descriptions for the reader of this book.

The aim of this ITI Treatment Guide is to foster awareness of the increasing demands on clinicians to provide treatment for a growing population of patients with soft-tissue related problems. The authors hope that Volume 12 will be a valuable resource and reference work for the treatment of patients with mucogingival deformities to reduce the risk of biological and esthetic complications and to ensure predictable and stable long-term results.

2 <u>Importance of the Peri-Implant</u> Soft Tissues

A. Sculean

Dental implants are anchored in jawbone via direct contact between the bone and the implant, a phenomenon called osseointegration (Albrektsson and coworkers 1981). Emerging evidence indicates that the long-term success and survival of implants does not depend solely on osseointegration, but also on the soft tissues around the transmucosal aspect of the implant that separate the peri-implant bone from the oral cavity. This soft-tissue seal or collar is also called the peri-implant mucosa (Lindhe and coworkers 2008). The attachment of the soft tissue to the implant serves as a biological seal that ensures healthy conditions and prevents the development of peri-implant infections (peri-implant mucositis and peri-implantitis). Consequently, the peri-implant soft tissues play a crucial role for long-term implant survival (Lindhe and coworkers 2008).

The soft tissue around teeth develops during tooth eruption and seals the supporting tissues (the alveolar bone, periodontal ligament, and cementum) against the oral cavity (Bosshardt and Lang 2005). The peri-implant mucosa forms after traumatizing the oral soft and hard tissues to accommodate osseointegrated implants. The following presents a brief description of the most important anatomical features of the periodontal and peri-implant tissues.

Structure of periodontal tissues in health

The periodontium comprises the tissues supporting the teeth: the tooth-facing part of the *gingiva*, the *root cementum*, the *periodontal ligament*, and the part of the alveolar process that lines the tooth socket, termed *alveolar bone* (Schroeder and Listgarten 1997) (Figs 1 to 5).

As they develop, the teeth penetrate the epithelial lining of the oral cavity and then persist as transmucosal organs. Their root portion is anchored in the bone, while the crown resides in the oral cavity. The most important function of the gingiva is to protect the underlying soft and hard connective tissues from penetration by microorganisms from the oral cavity. The gingiva terminates coronally at the gingival margin; apically it ends at the mucogingival junction or becomes continuous with the mucosa of the hard palate. The gingival sulcus has an approximate depth of 0.5 mm; however, in a completely healthy situation, it may not be clinically detectable (Schroeder and Listgarten 1997)

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The interdental region contains a structure called the *gingival papilla*. The gingiva consists of two parts, the *free gingiva* and the *attached gingiva*. The free gingiva comprises the coronal portion of the gingiva and follows the contour of the cementoenamel junction, varying in width between 1 and 2 mm (Ainamo and Löe 1966). Its apical boundary is accentuated by a stippled line; a gingival groove may also be present. The attached gingiva stretches between the end of the free gingiva and the alveolar mucosa, or the mucosa of the floor of the mouth. Because the palatal mucosa extends to the free gingiva, there is no attached gingiva in the palate. The width of the attached gingiva may range from 1 to 10 mm (Ainamo and Löe 1966).

Junctional epithelium

The *junctional epithelium* is a non-keratinized epithelium that, due to its unique structural and functional adaptation, plays a critical role in maintaining periodontal health by providing a functional barrier to microbial challenges. Cell division occurs in the basal layer facing the lamina propria, while the innermost cells constitute the *epithelial attachment*. It consists of the basal lamina and hemidesmosomes that connect the epithelial cells with the tooth surface (Bosshardt and Lang 2005).

Connective tissue of the gingiva

The connective tissue of the gingiva consists mainly of fibroblasts exhibiting phenotypes that differ from those from the periodontal ligament (Bartold and coworkers 2000). They are arranged as groups of collagen fibers with a complex three-dimensional architecture that allows polymorphonuclear neutrophils (PMNs) and mononuclear cells to migrate through the connective tissue until they can pass the basement membrane bordering the junctional epithelium. Even in clinically healthy circumstances, an inflammatory cell infiltrate will be present and can be considered a common (normal) characteristic of the connective tissue adjacent to the junctional epithelium.



Fig 1 Photomicrograph. Tooth with a healthy periodontium. Supporting tissues of the tooth consisting of the root cementum, periodontal ligament, alveolar bone, and gingiva.



Fig 2 Photomicrograph. Supra-alveolar soft tissue consisting of the oral sulcular epithelium, junctional epithelium, and connective-tissue attachment (collagen fibers inserting into the root cementum). The junctional epithelium ends at the cementoenamel junction (CEJ) at the point of the insertion of the collagen fibers into the root cementum.



Fig 3 Higher magnification. Supra-alveolar soft tissue comprising the junctional epithelium and root cementum with inserting collagen fibers. Well-encapsulated minor inflammatory cell infiltrate (arrow) located adjacently to the junctional epithelium.

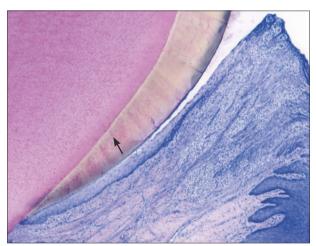


Fig 4 Higher magnification. Oral sulcular epithelium and junctional epithelium. The apical extension of the junctional epithelium ends at the cementoenamel junction. The well-encapsulated inflammatory cell infiltrate (arrow) is clearly distinguishable next to the junctional epithelium.

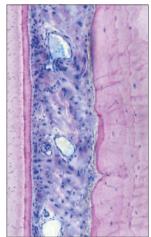


Fig 5 Higher magnification. Intact periodontal ligament connecting the root cementum with the alveolar bone. The collagen fibers invest in both root cementum and alveolar bone.

Periodontal ligament

The soft connective tissue interposed between the alveolar bone and the root cementum is called the *periodontal ligament*. Coronal to the alveolar crest, the periodontal ligament merges with the lamina propria of the gingiva, while it is continuous with the dental pulp periapically. The width of the periodontal ligament measures approximately 200 μ m, being thinnest in the middle third of the root. Its width decreases with age. The most important function of the periodontal ligament is to attach the tooth to the surrounding bone. Another important function is the damping of occlusal forces. Additionally, the periodontal ligament serves as an important reservoir for cells that are constantly needed for tissue homeostasis and play a crucial role in periodontal wound healing and regeneration (periodontal fibroblasts, cementoblasts, odontoclasts, osteoblasts and osteoclasts, epithelial cell rests of Malassez, monocytes and macrophages, and undifferentiated mesenchymal progenitor and stem cells).



Fig 6 Photomicrograph. Osseointegrated dental implant with direct bone-to-implant contact and supracrestal soft-tissue implant contact.

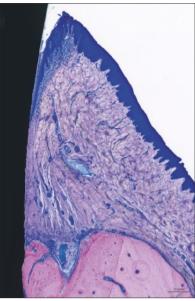


Fig 7 Higher magnification- Supracrestal peri-implant soft tissues consisting of oral and sulcular epithelium and connective tissue adhesion to the implant surface.



Fig 8 Higher magnification. Coronal portion of the supracrestal peri-implant soft tissues. The oral and sulcular epithelium are clearly visible. The collagen fibers located apically to the junctional epithelium run parallel to the implant surface. A more diffuse inflammatory infiltrate (arrow) is located immediately adjacent the junctional and sulcular epithelium.



Fig 9 Higher magnification. Supracrestal portion of the peri-implant soft tissues. The collagen fibers located apically to the junctional epithelium run parallel to the implant surface.

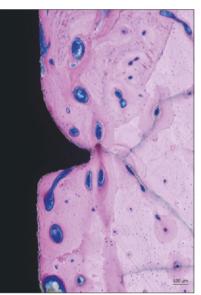


Fig 10 Higher magnification. Direct contact between the bone and the implant surface (osseointegrated implant).

The fibroblasts of the periodontal ligament synthesize, structure, and remodel the extracellular matrix, which consists of collagen fibers and an amorphous ground substance composed of non-collagenous proteins. Due to its structural configuration, the periodontal ligament provides a flexible attachment of the tooth to the surrounding bone via *Sharpey's fibers* into the mineralized tissues (Nanci and Bosshardt 2006).

Root cementum

Root cementum is a mineralized connective tissue coating the roots of teeth, usually extending from the cementoenamel junction to the root apex. Its primary function is to invest and attach the fibers of the periodontal ligament to the root surface (the acellular extrinsic fiber cementum, AEFC, and the cellular mixed stratified cementum, CMSC). However, root cementum also has other important functions, such as adjusting the tooth position to new physiologic requirements and repair of root defects (cellular intrinsic fiber cementum, CIFC) (Nanci and Bosshardt 2006).

Alveolar bone

The teeth are anchored in the *alveolar bone*, a part of the alveolar process that consists of an outer cortical plate, an inner cortical plate, and a central spongiosa. The alveolar process is continuous with the jawbone and can only develop in the presence of teeth. The inner cortical plate lines the alveolus and is also referred to as the alveolar bone.

In fully erupted and periodontally healthy teeth, the contour of the alveolar crest follows the contour of the cementoenamel junction in a coronoapical direction for approximately 2 mm (Saffar and coworkers 1997). The alveolar bone consists of compact bone characterized by the presence of osteons, the structural unit for cortical bone remodeling. The socket wall exhibits many perforations that connect the periodontal ligament with the endosteal or bone-marrow spaces, thus enabling blood and lymph vessels, and nerve fibers, to pass through these openings.

A characteristic component of the alveolar bone is the *bundle bone*, which is deposited in successive layers running parallel to the socket wall. Its typical appearance is determined by the Sharpey's fibers penetrating its layers. The alveolar bone responds to the functional demands placed on it by the processes of resorption and deposition, known as *bone remodeling*.

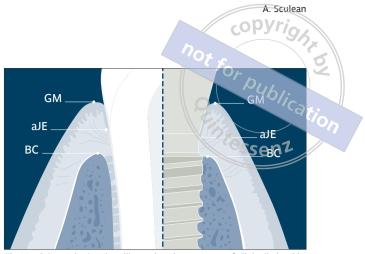


Fig 11 Schematic drawing. illustrating the structure of clinically healthy supra-alveolar soft tissues adjacent to a tooth or an implant (GM: gingival margin, aJE: apical extent of the junctional epithelium, BC: bone crest).

Structure of peri-implant tissues in health

During the process of wound healing following the placement of dental implants, the features of the peri-implant mucosa are established (Sculean and coworkers 2014) (Figs 6 to 10).

Berglundh and coworkers (1991) examined the anatomical and histological features of the peri-implant mucosa in dogs, formed in a two-stage procedure, and compared these with those of the gingiva around teeth. The peri-implant mucosa consisted of a keratinized oral epithelium located at the external surface, connected to a thin barrier epithelium facing the abutment (the equivalent to the junctional epithelium around teeth), the peri-implant junctional epithelium. It terminated 2 mm apical to the coronal soft-tissue margin and 1.0 to 1.5 mm coronal to the peri-implant bone crest. The mean supracrestal soft tissue (including the sulcus depth) measured 3.80 mm around implants and 3.17 mm around teeth. While there was no statistically significant difference in the height of the junctional epithelium and sulcus depth between implants and teeth, the height of the soft connective tissue was statistically significantly greater around implants than around teeth (Fig 11).

The peri-implant junctional epithelium and the soft connective tissue adjacent to the abutment appeared to be in direct contact with the implant/abutment surface (Berglundh and coworkers 1991). In summary, the findings of this study showed that the peri-implant mucosa displays comparable anatomical features to those of gingiva around teeth (Berglundh and coworkers 1991). Subsequent studies provided evidence that a similar mucosal attachment is formed on titanium with different implant systems (Buser and coworkers 1992; Abrahamson and coworkers 1996) and around implants placed using both non-submerged and submerged approaches (Abrahamson and coworkers 1999; Arvidson and coworkers 1996; Weber and coworkers 1996). However, the peri-implant junctional epithelium was significantly longer in implants placed using a submerged approach, where an abutment was connected in a second-stage surgical procedure, than in implants placed using a non-submerged approach (Weber and coworkers 1996).

The *biologic width* (of the supracrestal soft tissue) was revisited in a further dog experiment, following connection of the abutment to the implant with or without a reduced vertical dimension of the oral mucosa (Berglundh and coworkers 1996). It was found that while the peri-implant junctional epithelium was about 2 mm in depth, the supra-alveolar soft connective compartment had a depth of approximately 1.3 to 1.8 mm.

Interestingly, sites with reduced mucosal thickness consistently revealed marginal bone resorption, thus adjusting the width of the supracrestal soft tissue. Evaluating the biologic width around one- and two-piece titanium implants placed in a non-submerged or submerged approach in the mandibles of dogs, Hermann and coworkers (2001) suggested that the gingival margin is located coronally and the biologic width is more similar to teeth around one-piece non-submerged implants than either two-piece non-submerged or two-piece submerged implants. These findings were later confirmed in a comparably designed dog study with another implant system (Pontes and coworkers 2008).

Several studies evaluated the impact of surface topography (surface roughness measurements) on the peri-implant mucosa. Cochran and coworkers (1997) failed to show any differences in the dimensions of the sulcus depth, peri-implant junctional epithelium, and soft connective tissue in contact with implants with a titanium plasma-sprayed (TPS) surface or a sandblasted and acid-etched surface. Abrahamsson and coworkers (2001, 2002) observed similar epithelial and soft connective tissue components on rough (acid etched) and smooth (turned) titanium surfaces. The biologic width (supracrestal soft tissue) was greater on the rough surfaces, although with no statistically significant difference to that around smooth surfaces. Findings from two human histologic studies revealed less epithelial downgrowth and a longer soft connective tissue compartment in conjunction with oxidized or acid-etched titanium compared to a machined supface (Glauser and coworkers 2005; Ferreira Borges and Dragoo 2010). In a study in baboons, Watzak and coworkers (2006) showed that implant surface modifications had no significant effect on the biologic width after eighteen months of functional loading. Following a healing period of three months, nanoporous TiO₂ coatings of one-piece titanium implants showed similar length of peri-implant soft connective tissue and epithelium than the uncoated, smooth neck portion of the control titanium implants in dogs (Rossi and coworkers 2008). Schwarz and coworkers (2007) have suggested that soft-tissue integration was more influenced by hydrophilicity than by microtopography.

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A number of studies revealed that epithelial cells attach to different implant materials in a comparable manner to that in which junctional epithelial cells attach to the tooth surface via hemidesmosomes and a basal lamina (Sculean and coworkers 2014).

Analyzing the intact interface between soft connective tissue and titanium-coated epoxy resin implants, Listgarten confirmed the parallel orientation of collagen fibers to the titanium layer (Listgarten and coworkers 1992, 1996). Since implants lack a cementum layer into which the peri-implant collagen fibers can invest, the attachment of the soft connective tissue to the transmucosal portion of an implant is regarded as being weaker than the soft connective tissue attachment to the surface of a tooth root (Sculean and coworkers 2014). Therefore, improving the quality of the soft tissue-implant interface is of great relevance for maintaining healthy peri-implant tissues (Sculean and coworkers 2014).

The wound-healing sequence leading to the establishment of the soft tissue seal at implants was evaluated by Berglundh and coworkers (2007). Immediately after implant placement, a coagulum occupied the implant-mucosa interface. Numerous neutrophils infiltrated the blood clot, and at four days an initial mucosal seal was established. In the next few days, the number and distribution of leukocytes decreased, becoming confined to the coronal portion, with fibroblasts and collagen dominating the apical part of the implant-tissue interface.

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Between one and two weeks of healing, the peri-implant junctional epithelium was located approximately 0.5 mm apical to the mucosal margin. At two weeks, the peri-implant junctional epithelium began to proliferate in an apical direction. After two weeks, the peri-implant mucosa was rich in cells and blood vessels. At four weeks of healing, the peri-implant junctional epithelium migrated further apically and occupied 40% of the total soft-tissue/implant interface. This soft connective tissue was rich in collagen and fibroblasts and was well-organized.

The apical migration of the peri-implant junctional epithelium was completed between six and eight weeks, and the fibroblasts formed a dense layer over the titanium surface at that time. From six to twelve weeks, maturation of the soft connective tissue had occurred; the peri-implant junctional epithelium occupied about 60% of the entire implant/soft-tissue interface. Further away from the implant surface, the number of blood vessels was low; fibroblasts were located between thin collagen fibers, running mainly parallel to the implant surface.

These findings indicate that the soft-tissue attachment to transmucosal (non-submerged) implants made of commercially pure titanium with a polished surface in the neck portion requires at least six weeks (Berglundh and coworkers 2007). These findings from animal experiments were corroborated in human studies by Tomasi and coworkers (2013), indicating that a soft-tissue barrier adjacent to titanium implants may form completely within eight weeks. Further studies have provided evidence indicating that in dogs, the dimensions of the soft-tissue seal (the biologic width or supracrestal soft tissue) around implants are stable for at least twelve (Cochran and coworkers 1997; Assenza and coworkers 2003) or fifteen months, respectively (Hermann and coworkers 2000).

The role of keratinized mucosa in maintaining peri-implant tissue health

It is generally accepted that the assessment of peri-implant health is based on clinical and radiographic parameters bleeding on probing (BOP), probing depth (PD), and marginal peri-implant bone level (Salvi and coworkers 2012; Jepsen and coworkers 2015).

The influence of the presence or absence and the thickness of keratinized or attached mucosa (KAM) on peri-implant tissue health and stability is controversial (Bengazi and coworkers 1996; Schou and coworkers 1992; Strub and coworkers 1991; Wennström and coworkers 1994).

On one hand, a number of clinical studies have failed to show a correlation between the presence of an "adequate" band (2 mm or more) of KAM and implant stability, as assessed by peri-implant bone level or probing depths (Bengazi and coworkers 1996; Wennström and coworkers 1994; Chung and coworkers 2006; Bouri and coworkers 2008; Boynueğri and coworkers 2013). These results were also supported by findings from an animal study indicating that the presence of an "adequate" width of KAM does not significantly influence peri-implant tissue conditions (Strub and coworkers 1991).

However, other clinical studies have suggested that an inadequate (2 mm or less) width of KAM is related to a higher risk of peri-implant inflammation and loss of soft and hard tissue (Warrer and coworkers 1995; Block and coworkers 1996; Zarb and coworkers 1990). A number of other studies have reported statistically significant associations between a peri-implant KAM width of less than 2 mm and higher bleeding scores (Zigdon and coworkers 2008; Adibrad and coworkers 2009; Schrott and coworkers 2009; Lin and coworkers 2013), greater plaque accumulation (Chung and coworkers 2006: Bouri and coworkers 2008; Boynueğri and coworkers 2013; Adibrad and coworkers 2009; Schrott and coworkers 2009; Crespi and coworkers 2010), and more mucosal inflammation (Chung and coworkers 2006; Bouri and coworkers 2008; Boynueğri and coworkers 2013; Adibrad and coworkers 2009; Crespi and coworkers 2010), compared to sites with adequate KAM width (2 mm or more).

Conversely, results from a retrospective study reported low rates of peri-implant diseases in patients enrolled in a maintenance program irrespective of the width of the KAM (Frisch and coworkers 2015). The authors of this study suggest that maintaining an optimal level of plaque control seems to be more important for ensuring peri-implant tissue health than the presence of an adequate width of KAM. Schou and coworkers (1992) showed that peri-implant health can be ensured in the absence of keratinized mucosa, provided adequate oral hygiene is established.

These findings were later confirmed in systematic reviews that concluded that the lack of an adequate zone of keratinized attached tissue may not be mandatory for maintaining soft-tissue health around dental implants, as long as an optimal level of oral hygiene is ensured (Wennström and Derks 2012; Gobbato and coworkers 2013; Lin and coworkers 2013). However, preclinical and clinical data indicate that in the absence of stable keratinized attached mucosa, plaque control is more difficult, which in turn may lead to peri-implant soft-tissue inflammation and, eventually, bone loss (Warrer and coworkers 1995; Wennström and Derks 2012; Gobbato and coworkers 2013; Lin and coworkers 2013).

Roccuzzo and coworkers (2016) evaluated the clinical conditions around dental implants placed in the posterior mandible of healthy or moderately periodontally compromised patients as a function of the presence or absence of keratinized attached mucosa (KAM). The results showed that the absence of KAM was associated with higher plaque accumulation, greater soft-tissue recession (REC), and a higher number of sites that required additional surgical or antibiotic treatment, indicating that implants not surrounded by KAM are more prone to plaque accumulation and to developing soft-tissue recessions despite adequate oral hygiene and supportive periodontal therapy. These findings are in line with the results of three recent reviews, which concluded that the presence of an adequate width of KAM around dental implants is associated with better soft and hard tissue stability, less plaque accumulation, soft-tissue recession, and a lower incidence of peri-implant mucositis (Sculean and coworkers 2017; Chackartchi and coworkers 2019).

Taken together, the by far greater part of the available evidence indicates that the lack of an adequate width of KAM around dental implants is associated with more plaque accumulation, inflammation, soft-tissue recession, and attachment loss (Warrer and coworkers 1995; Wennström and Derks 2012; Gobbato and coworkers 2013; Lin and coworkers 2013; Sculean and coworkers 2017; Chackartchi and coworkers 2019; Iorio-Siciliano and coworkers 2019).

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A recent systematic review evaluated the effects of soft-tissue augmentation procedures on peri-implant health or disease in partially and fully edentulous patients (Thoma and coworkers 2018a), using soft-tissue grafting procedures to increase the width of the KAM or the thickness of the peri-implant mucosa. The findings indicated that soft-tissue grafting by means of autologous grafts may favor peri-implant health through a gain of KAM, improved bleeding scores, and less marginal bone loss.

In the esthetic zone, autologous connective-tissue grafts resulted in increased mucosal thickness around implants and were associated with statistically significantly less marginal bone loss over time. However, the data failed to reveal statistically significant changes in terms of bleeding on probing, probing depths, or plaque scores at grafted sites compared to sites without grafting. Nevertheless, the authors concluded that based on the available evidence, it is generally accepted that soft-tissue augmentation is beneficial to establishing and maintaining peri-implant health (Thoma and coworkers 2018a).

Regarding the thickness of the peri-implant mucosa, findings of preclinical and clinical studies suggest a threshold value of 2 mm for establishing a natural appearance of the peri-implant mucosa and minimal soft-tissue discoloration at implant-supported prosthetic reconstructions (Jung and coworkers 2007; Cosgarea and coworkers 2015; Ioannidis and coworkers 2017; Thoma and coworkers 2016). Moreover, an adequate mucosal thickness was associated with a decreased risk of mucosal recessions in immediate-placement protocols or in specific anatomic situations (e.g., minimal or no facial bony wall, orofacial implant malposition, various angles of the implant fixtures) (Buser and coworkers 2004; Evans and coworkers 2008; Sculean and coworkers 2017).



In summary, despite the fact that the available evidence is still inconclusive, there is reason to suggest that the presence of KAM favors peri-implant health through facilitating oral hygiene measures, with a consequent reduction in both inflammation (lower bleeding scores) and marginal bone loss. Furthermore, its presence or absence also plays a key role in ensuring esthetics.

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Photography

Professor Dieter D. Bosshardt - Bern, Switzerland

3.2 <u>Soft-Tissue Management Before</u> <u>Placement</u>

On the occasion of the 2017 World Workshop, Hämmerle and Tarnow (2018) reported that a significant amount of controlled prospective studies with medium-size patient samples indicated that thin soft tissue around implants leads to increased peri-implant marginal bone loss compared to thick soft tissue. Most of the data, however, were published by one group of researchers.

Linkevicius and coworkers (2009) placed 46 implants in 19 patients. The implants were divided into two groups related to soft-tissue thickness. At the one-year follow-up, the marginal bone loss at the implants in the thin-tissue group was on the order of 1.5 mm, compared to only 0.3 mm in the thick-tissue group.

In addition, the same investigators analyzed the effects of buccal soft-tissue thickness on marginal bone-level changes in 32 patients. They found a significant correlation between soft-tissue thickness and bone loss, with thin soft-tissue sites presenting more bone loss (0.3 mm versus 0.1 mm) at the one-year follow-up. That thin soft tissue leads to increased marginal bone loss was confirmed in another recent study (Linkevicius and coworkers 2015). In addition to the thin-tissue and thick-tissue groups, the investigators followed a third group of about 30 patients whose thin soft tissue was augmented by grafting at the time of implant. The resulting bone loss was not different from that in thick soft-tissue group. These findings seem to indicate that adequate soft-tissue thickness benefits the stability of the peri-implant bone levels.

In another study, Puisys and Linkevicius (2015) concluded that, since significantly less bone loss can occur in naturally thick soft tissue than in patients with a thin tissue phenotype, augmenting the tissue could be the way to reduce crestal bone loss.

Based on the observation that significantly less bone loss occurs around implants placed in thick tissue phenotypes compared to thin phenotypes, clinicians may be encouraged to augment thin soft tissue before or during implant placement in order to facilitate crestal bone stability. Figures 7a-i show an example of this treatment approach in the posterior mandible of a 63-year-old woman.

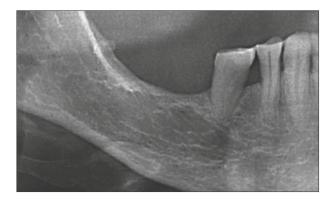


Fig 7a Panoramic radiograph of the edentulous sites 46 and 47. There is barely enough bone available for implant placement above the mandibular canal.



Fig 7b Edentulous area, buccal view. Very shallow vestibule and absence of keratinized mucosa.



Fig 7d Free gingival graft harvested from the palate sutured above a split-thickness flap in the area where the implants are planned.



Fig 7c Edentulous area, occlusal view. Very thin crest.



Fig 7e Graft sutured with 4-0 Vicryl, occlusal view



Fig 7f At three months, a full-thickness flap was raised lingually and buccally for placing the implants. Thicker keratinized tissue on both sides of the flap.



Fig 7g At the time of the final impression, occlusal view. Both implants are surrounded by a thick collar of keratinized tissue, that creates an effective barrier that protects the peri-implant structures.



Figs 7h-i Clinical and radiographic views of the screw-retained ceramic crowns at 6 years. Prosthetic procedures: Dr. Nicola Scotti – Torino, Italy



Fig 8a Preoperative view. Bone atrophy associated with the presence of very thin mucosa, with almost no keratinization.



Fig 8b Two free gingival grafts sutured in the area where implant placement and bone regeneration was planned.



Fig 8c Three months after soft-tissue augmentation. A thick band of keratinized tissue was present on the lingual and buccal aspects of the full-thickness flap.

From a clinical perspective, the presence of a wide band of keratinized tissue facilitates the transmucosal healing of dental implants, even in cases where bone regeneration is required, as it allows the creation of a thick soft-tissue cuff around the collar of the implant. Figures 8a-I show an example of this treatment approach in the posterior mandible of a 57-year-old woman, for whom horizontal bone regeneration was needed in conjunction with implant placement.

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Several studies have argued the use of various techniques for vertical ridge augmentation in cases of severe atrophy of the alveolar ridge, using either non-resorbable or resorbable membranes supported by a space-making device or a titanium mesh (Esposito and coworkers 2008; Fontana and coworkers 2011; Roccuzzo and coworkers 2017a).

All these studies also showed that the use of a barrier device is a technique-sensitive procedure and subject to surgical complications (Jepsen and coworkers 2019). One of the main reasons for GBR failures is related to exposure of the barrier membrane, leading to bacterial contamination of the surgical area and infection and thereby compromising the regeneration outcome (Sanz and coworkers 2019). Even though there have been no specific studies on this matter, it might be suggested that membrane exposure, especially during the first four weeks postoperatively, may be higher in patients with very thin mucosa, or without keratinization, or with scar tissue. In specific circumstances, it is therefore reasonable to consider optimizing the quantity and quality of the soft tissue before hard-tissue regenerative procedures are carried out.

Fig 8d Implants at sites 35 and 37 (S, RN, diameter 3.3 mm, length 10 mm, and S, RN, diameter 4.8 mm, length 10 mm; Institut Straumann AG) with a large dehiscence-type bone defect on the buccal aspect.



Fig 8e Guided bone regeneration with autologous bone in contact with the implant surface, followed by a layer of deproteinized bovine bone mineral (DBBM). Resorbable collagen membrane adapted around the implants to stabilize the graft.



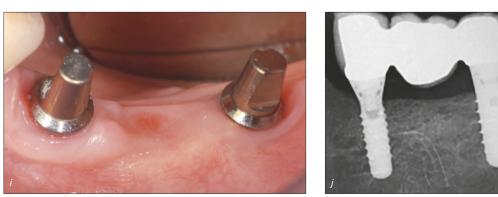
Fig 8f Sutures for transmucosal healing. Ideal soft-tissue seal around the collar of the implants thanks to the preliminary soft-tissue augmentation.



Fig 8g At the time of delivery of the final prosthesis, occlusal view.



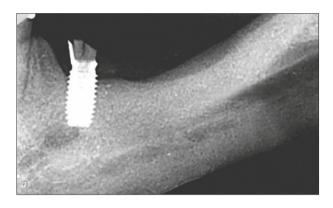
Fig 8h Three-unit ceramic bridge delivered and secured with temporary cement.



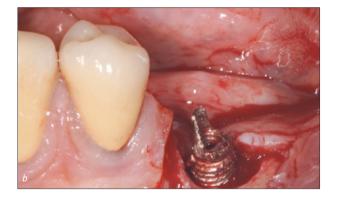
Figs 8i-j One-year clinical and radiographic follow-up. The prosthesis was removed to double-check the condition of the soft tissues and later then reinserted using definitive cement.

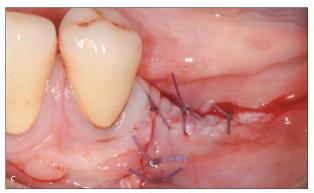


Figs 8k-I Ten-year follow-up. Minor pigmentation of the ceramic crown on the buccal side. Healthy peri-implant soft tissue with minimal probing depth.

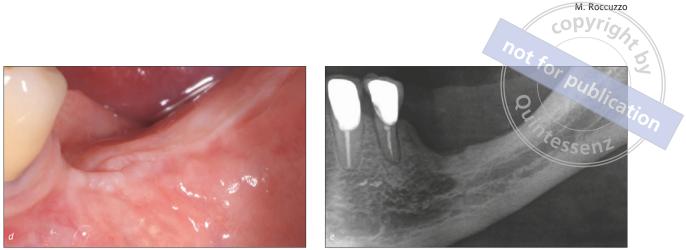


Figures 9a-p exemplify this approach in the mandible of a 63-year-old patient, a dentist and current cigarette smoker. He had previously received an implant at site 35, but it had recently fractured. After surgically removing the fractured implant, vertical bone augmentation was required, as the bone was not high enough to place an implant above the mandibular canal. The examination of the local soft tissue revealed minimal keratinized mucosa and the presence of scar tissue as a result of previous surgery. To reduce the risk of soft-tissue dehiscence and of exposure or infection of the area following GBR, the patient was advised that preliminary soft-tissue augmentation was required prior to any attempt at vertical bone regeneration.





Figs 9a-c Surgical removal of the fractured implant.



Figs 9d-e After three months, site 35 presented with minimal keratinized mucosa and scar tissue, considered not to be ideal in view of the planned vertical bone augmentation.



Fig 9f Free gingival graft sutured on the periosteum after elevating a split-thickness flap, with 4-0 Vicryl.



Fig 9g Four months after soft-tissue augmentation, lateral view.



Fig 9h Custom-made Ti-mesh filled with autologous bone combined with DBBM and secured with two screws to contain and protect the bone graft. The presence of thick mucosa reduced the need for a collagen membrane.

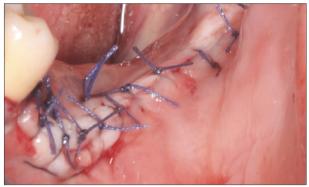


Fig 9i Flap closed without tension despite coronal advancement and adapted to completely cover the augmented area. The flap was stabilized with Vicryl 3-0 horizontal mattress sutures at the apical aspect and Vicryl 4-0 multiple single interrupted sutures at the far coronal aspect.

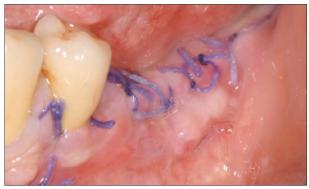
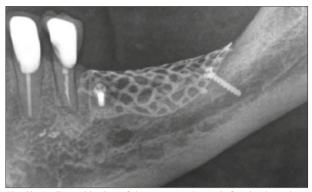


Fig 9j Two weeks after the surgery. The flap had healed well, and the sutures could be removed.



Figs 91 Radiographic view of the augmented area before implant placement surgery.







Fig 90 Three months after surgery. Implants surrounded by a thick cuff of healthy keratinized mucosa. The impressions could now be taken for the final restoration.



Fig 9k Clinical view six months after regeneration surgery. Optimal healing.



Fig 9m After removal of the Ti-mesh, two Straumann Tissue Level implants were placed at sites 35 and 36 (SP, RN, diameter 3.3 mm, length 8 mm, and SP, RN, diameter 4.1 mm, length 6 mm; Institut Straumann AG).



Fig 9p Six months after implant placement, the probe indicated a shallow sulcus with no signs of inflammation. Prosthetic procedures: Dr. Walter Gino – Torino, Italy

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Based on the conclusions of the 2017 World Workshop, namely that a significant amount of controlled prospective studies indicated that thin soft tissue around implants leads to increased marginal bone loss compared to thick soft tissue, clinicians may be encouraged to create ideal soft-tissue conditions before placing implants. Mucogingival surgery may be indicated particularly in patients with thin soft tissue and no keratinization. Each of the two steps of this approach is relatively easy to perform. However, the patient will have to accept the discomfort of two separate interventions not less than a month apart from each other. Even though recent publications provided guidelines for decision-making if the clinician considers autologous soft-tissue grafting to promote peri-implant health or preserve marginal bone levels at implant sites with insufficient soft-tissue dimensions (Thoma and coworkers 2018a; Giannobile and coworkers 2018), the ideal clinical solution should be individually determined and should represent the results of a proper patient-clinician discussion.



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