



# The Australian Journal of Periodontology and Implant Dentistry Limited

The Official Journal of the Australian Society of Periodontology and the Australasian Osseointegration Society

## IN THIS ISSUE

- Understanding the Peri-Implant Mucosa: Key Insights and Implications for Predictable Implant Therapy
- Strategies to Improve Peri-Implant Soft Tissue Integration: Micro to Nanoengineered Surface Modifications
- Socket Shield – is it just the Latest Trend or a Revolutionary Technique for Maintaining the Buccal Bone?
- AOS-ASP-APS Combined Conference 2024 Poster Abstracts
- ASP & AOS State Branch News

*Australasian  
Osseointegration  
Society limited*

leading regeneration

Geistlich

# Geistlich Bio-Oss Pen<sup>®</sup>



Available in:  
Small granules 0.5cc (0.25g) / 1.0cc (0.5g)  
Large granules 1.5cc (0.5g)

A treatment made  
simple for **you**  
and **your patient.**



  
170  
YEARS  
Geistlich

## Contents



■ Authors Guidelines	2
■ Editor's Notes	3
■ President's Notes	4
■ Understanding the Peri-Implant Mucosa: Key Insights and Implications for Predictable Implant Therapy	7
■ Strategies to Improve Peri-Implant Soft Tissue Integration: Micro to Nanoengineered Surface Modifications	23
■ Socket Shield – is it just the Latest Trend or a Revolutionary Technique for Maintaining the Buccal Bone?	33
■ AOS-ASP-APS Combined Conference 2024 Poster Abstracts	44
■ ASP State Branch News	56
■ AOS State Branch News	59

**Editor**

A/Prof Ryan Lee

MCD (Perio), PhD  
 School of Dentistry  
 The University of Queensland  
 288 Herston Road  
 Cnr Bramston Tce & Herston Rd  
 QLD 4006 Australia  
 Email: editor@ajpid.org.au

**Editorial Board**

Dr Fritz Heitz,  
 Western Australia

Prof. Saso Ivanovski,  
 Queensland

A/Prof. George Pal,  
 New South Wales

Dr Simon Watson, Victoria

**How to reach us:**

**www.ajpid.org.au**

**Paper submission & letters to the editor**

editor@ajpid.org.au

**Journal annual subscription (for non-members)**

admin@ajpid.org.au

**ASP Membership enquiries**

contact@asp.asn.au

www.asp.asn.au

**AOS Membership enquiries**

info@aos.org.au

www.aos.org.au

**Journal enquiries**

admin@ajpid.org.au

**Journal Compilation**

Lisa Sullivan

## Submissions welcome!

**Australian Journal of Periodontology and Implant Dentistry** aims to promote the field of *Periodontics and Implant Dentistry* through clinical papers, original research, review articles, case reports and other correspondence that the Editor thinks appropriate to print. Submissions of articles covering these areas are welcomed. See website for submission guidelines <http://www.ajpid.org.au/>

If the reported research involves either human or animal experimentation then the authors must indicate that the ethical guidelines of the Australian National Health and Medical Research Council or the Declaration of Helsinki have been applied. If the research involves human subjects then the authors must state that informed consent was obtained.

Material is received on the understanding that it may be subject to Editorial revision and that, on acceptance, becomes the property of the Australian Journal of Periodontology and Implant Dentistry. All expressions of opinion or statements of supposed facts are those of the authors and are not to be regarded as the views of the ASP/AOS or Editorial Board of Australian Journal of Periodontology and Implant Dentistry.

## Digital Subscription

All members of the Australian Society of Periodontology and the Australasian Osseointegration Society will receive two publications per year as part of their membership.

The annual subscription for 2024 will be \$68.00 plus GST.

Order queries should be sent to the AJPID Administrator Kayla Ashkar:  
[admin@ajpid.org.au](mailto:admin@ajpid.org.au)



It is with great pleasure to present the Editorial Report for the last quarter of 2024. We are grateful to our dedicated reviewers who have generously contributed their time and expertise to the review process. Your insightful feedback and constructive criticism have been instrumental in shaping the content of our journal and maintaining its high standards.

For this journal issue, we have published three articles and conference abstracts. The first article by *Dr Rafael Mata Santos* (ITI Scholar, University of Queensland), is titled '*Understanding the Peri-Implant Mucosa: Key Insights and Implications for Predictable Implant Therapy*.' This narrative review explores the anatomy and clinical significance of peri-implant soft tissue phenotype components. It discusses the role of peri-implant soft tissue mucosa in implant therapy success and complications.

The second article, by *Dr Sae Mi Bok* (University of Queensland), is another comprehensive narrative review that explores innovative strategies to enhance peri-implant soft tissue integration. The review discusses micro- and nano-scale modifications of implant surfaces, highlighting cutting-edge advancements designed to enhance biological response and promote the long-term success of implants.

The last article by *Dr Alana Smith* (University of Melbourne) is a review paper on '*Socket shield technique: its efficacy and predictability*.' The review examines the evolution of the socket-shield protocol, tracing its original concept and assessing the technique's efficacy. By analysing existing clinical evidence, the review provides interesting insights into the clinical applicability of this innovative approach in implant dentistry.

Additionally, I am proud to present the accepted poster abstracts from the AOS-ASP-APS Conference Poster Competition. It was divided into two streams: pre-clinical and clinical research, offering a platform for emerging researchers to present their work to a distinguished panel of experts.

In closing, I'd like to thank all the contributors, reviewers, and editorial team for their continued support and dedication. We look forward to your continued partnership in the years to come.

Regards,

A/Prof Ryan Lee  
Editor-in-chief





## President's Notes



As my term as Federal President of Australian Society of Periodontology comes to an end, I reflect on what has been an amazing journey, both in the privilege of serving our society and in the rewarding experience of organising this year's conference. It has been an honour to work alongside such a dedicated group of people, and I am truly grateful for the opportunity to lead ASP.

One of the highlights of my presidency was the combined **AOS, ASP, and APS Conference** held in Gold Coast. I believe the conference was a tremendous success, featuring world-class international and local speakers who provided not only practical and up-to-date scientific content but also enriching social events and networking opportunities. I would like to extend my deepest thanks to the organisation and scientific committees for their dedication and hard work in making this conference such a success.

Beyond the academic and professional excellence, we had the pleasure of enjoying an unforgettable **Gala Dinner at Sea World, Gold Coast**. The evening was a memorable one, highlighted by a thrilling dolphin show and exciting roller coaster rides, offering everyone the chance to relax, connect, and enjoy the unique atmosphere. The night was filled with laughter, camaraderie, and a shared sense of celebration, making it a perfect conclusion to a fantastic conference.

At the AGM, I had the pleasure of officially passing the presidency to **Dr. Mehdi Valizadeh**. I am confident that under Medhi's guidance, ASP will continue to thrive and grow.

Thank you.

Regards,

A handwritten signature in black ink, appearing to read 'Ryan Lee', written in a cursive style.

A/Prof Ryan Lee  
ASP President

## President's Notes



Following an incredibly successful conference in Sydney in 2022 in collaboration with the Australian Society of Periodontology, the decision was made to continue the partnership and plan the 2024 conference in the Gold Coast. This time however, we invited the Australian Prosthodontic Society to partner with us in an effort to pool resources and increase our exposure to a wider pool of members and make the opportunity more attractive to our industry partners. The joint conference was a great success, with over 400 delegates registered and several high profile speakers invited to present as part of a jam-packed scientific program.

The AOS continues to be one of the leading independent and highly regarded implant societies across Australia. Our flagship event, the biennial conference at a federal level is coupled with local branch meetings at a local level including online streaming of several meetings to provide members with as much value as possible.

On a federal level, there were two ongoing projects that had been commenced and were handed to us from the previous committee, being an update of an outdated constitution and a rollout of a new website. Both projects involved a significant amount of work. The constitution is nearing its final draft and will be passed on to the individual states for feedback in the coming weeks. The new website development was particularly complex due to the specific features that were required by each state and the website developer engaged to carry out the project was not capable of devoting the necessary amount of time and effort to see it through. Therefore a decision was made to part ways with him and engage the services of another company who have made excellent progress in the last few months and will be performing testing very shortly in preparation for going live in 2025.

As I end my term as federal president, I would like to acknowledge a few people who have assisted me in my duties over the last couple of years. Namely, Ms. Bella Cherkasskaya, Dr. Betty-Lisa Matthews, Dr. Simon Watson and Dr. George Pal. I would also like to wish my successor Dr. Jonothan Ng from the Queensland branch every success with federal responsibilities over the next term, after both South Australia and Western Australian branches declined their respective rotations.

Dr Angelos Sourial

AOS Federal President

# Precision, innovation and reliability



**stoma** hy-light®  
Hygienic lightness



For more information please contact your local  
Geistlich Product Specialist or call 1800 776 326.





# Understanding the Peri-Implant Mucosa: Key Insights and Implications for Predictable Implant Therapy

Rafael P. da Mata Santos, DDS, MSc

School of Dentistry, The University of Queensland, Queensland, Australia

## 1. Introduction

Implant dentistry offers a reliable long-term solution for replacing missing teeth, with numerous studies documenting stable results and achieving success and survival rates of approximately 95% over a 10-year follow-up (1-4). This high survival rate is based on the biocompatibility of titanium, which facilitates osseointegration with the host bone and ensures stable anchorage for functional dental reconstruction (5). Despite technological advances enhancing osseointegration and survival rates, biological, prosthetic, and aesthetic complications remain common. Consequently, implant survival is no longer the sole measure of success, but the absence of complications and patient satisfaction have become primary outcomes (6-8).

Peri-implant soft tissue integration is crucial for success (9, 10). The achievement of a successful outcome is based on the understanding of the biology and anatomy of peri-implant hard and soft tissues, alongside restorative and technical considerations. Composite outcome measures, including patient-reported outcomes, peri-implant tissue health, and the functional and aesthetic results of implant-supported reconstructions are essential for evaluating it (11, 12).

Aesthetics in implant dentistry increasingly focus on both "white aesthetics" and "pink aesthetics" (13, 14). Besides the aesthetic aspect of the prosthetic reconstruction, it includes the presence or absence of papillae, the emergence profile of the implant crown, the position of the mucosal margin, and the colour of the soft tissue. Among the factors influencing soft tissue aesthetics and stability, the peri-implant phenotype is one of the most critical. Minimum soft tissue dimensions are necessary to ensure mucosal margin and marginal bone stability, cleansing feasibility, aesthetics, and implant health (9, 15-17). Thus, tissue augmentation procedures are often necessary. The positive impact of these procedures on clinical, aesthetic, radiographic, and biological outcomes has been extensively documented in the literature (10, 12, 15). Addressing the critical role of peri-implant soft tissues is essential for effective treatment planning. This review will explore the anatomy and clinical significance of peri-implant soft tissue phenotype components. It aims to

## Abstract:

**Background:** The three soft tissue components of the peri-implant phenotype, namely keratinised mucosa width (KMW), supracrestal tissue height (STH), and mucosal thickness (MT) have been linked to various aspects of aesthetics, tissue stability, and peri-implant health. These components play a crucial role in successful implant therapy. Understanding of its biology is a keystone for achieving predictable and stable treatment outcomes.

**Objective:** To provide an overview of peri-implant mucosa biology and its clinical relevance, highlighting its impact on implant therapy success and complications.

**Conclusions:** Understanding the clinical significance of peri-implant mucosa dimensions, as well as its biological and morphological features, is essential for making pertinent clinical decisions and achieving predictable outcomes. A thorough assessment of these factors before or during implant placement is advised for preventing future complications.

**Keywords:** Dental Implants; Phenotype; Dental aesthetics; Peri-implant soft tissue.

evaluate their impact on implant therapy success and the relationship with implant-related complications, providing a comprehensive perspective of how keratinised mucosa width, mucosal thickness, and supracrestal tissue height influence peri-implant tissues stability, health, and aesthetics.

## 2. Anatomy of Peri-Implant Mucosa and Susceptibility to Inflammation

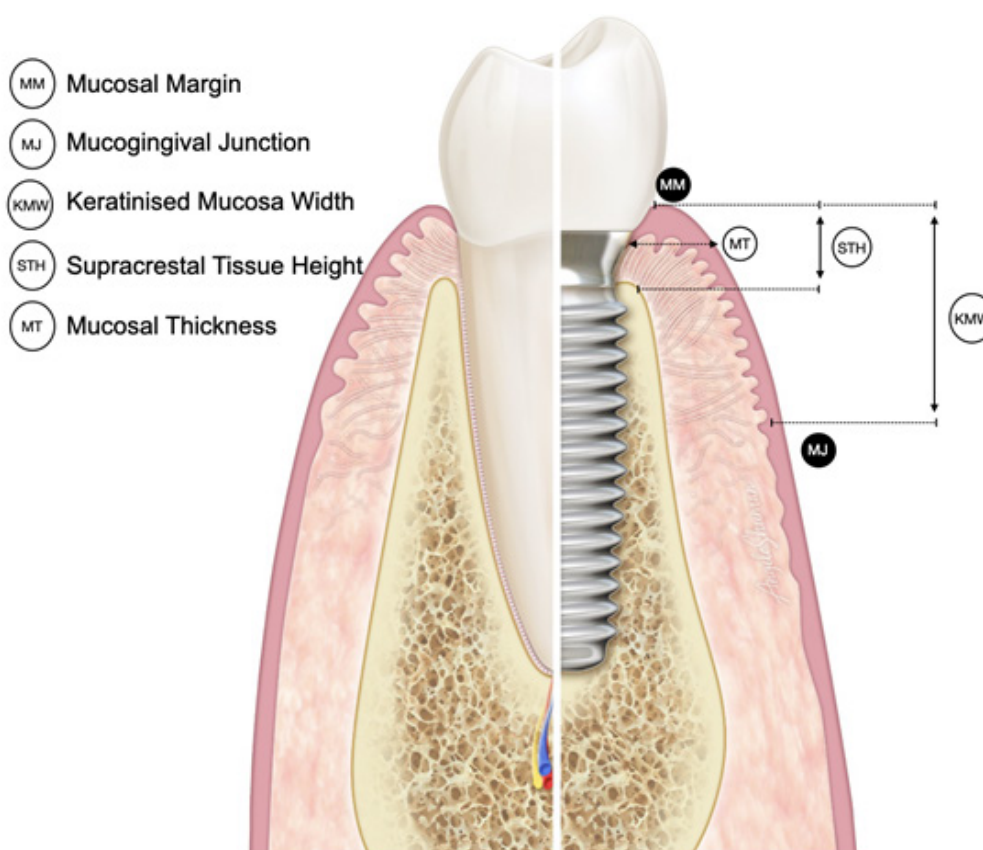
### 2.1 Anatomy of Peri-Implant Mucosa

Peri-implant tissues can be divided into hard and soft tissue compartments (18). The hard tissue compartment forms a direct contact relationship with the implant surface to secure implant stability. It carries the function of supporting the implant. The soft tissue, termed peri-implant mucosa, is established during a well-orchestrated wound healing process that occurs subsequent to the closure of the mucoperiosteal flap following implant installation (one-stage procedures) or following abutment connection in second-

stage surgery (19). It carries the function of protecting the underlying bone, serving as a biological seal that prevents the development of inflammatory peri-implant diseases, thus ensuring healthy conditions and stable osseointegration (20).

A classical study conducted by Berglundh et al. (21) in dogs investigated the anatomical and histological characteristics of the peri-implant mucosa, comparing them to those of gingival tissue. The authors found that the peri-implant mucosa consisted of a keratinised oral epithelium on the external surface, which continued with a thin, non-keratinised sulcular epithelium facing the abutment and ending in a junctional epithelium, similar to that found around natural teeth. The peri-implant junctional epithelium terminated 2 mm apical to the coronal mucosal margin and 1.0–1.5 mm coronal to the peri-implant bone crest. The mean supracrestal soft tissue measured 3.80 mm around implants and 3.17 mm around natural teeth. Notably, the height of the soft connective tissue was significantly greater around implants than around teeth.

**Fig. 1. Periodontal and peri-implant anatomy, and soft tissue components of the peri-implant phenotype.**



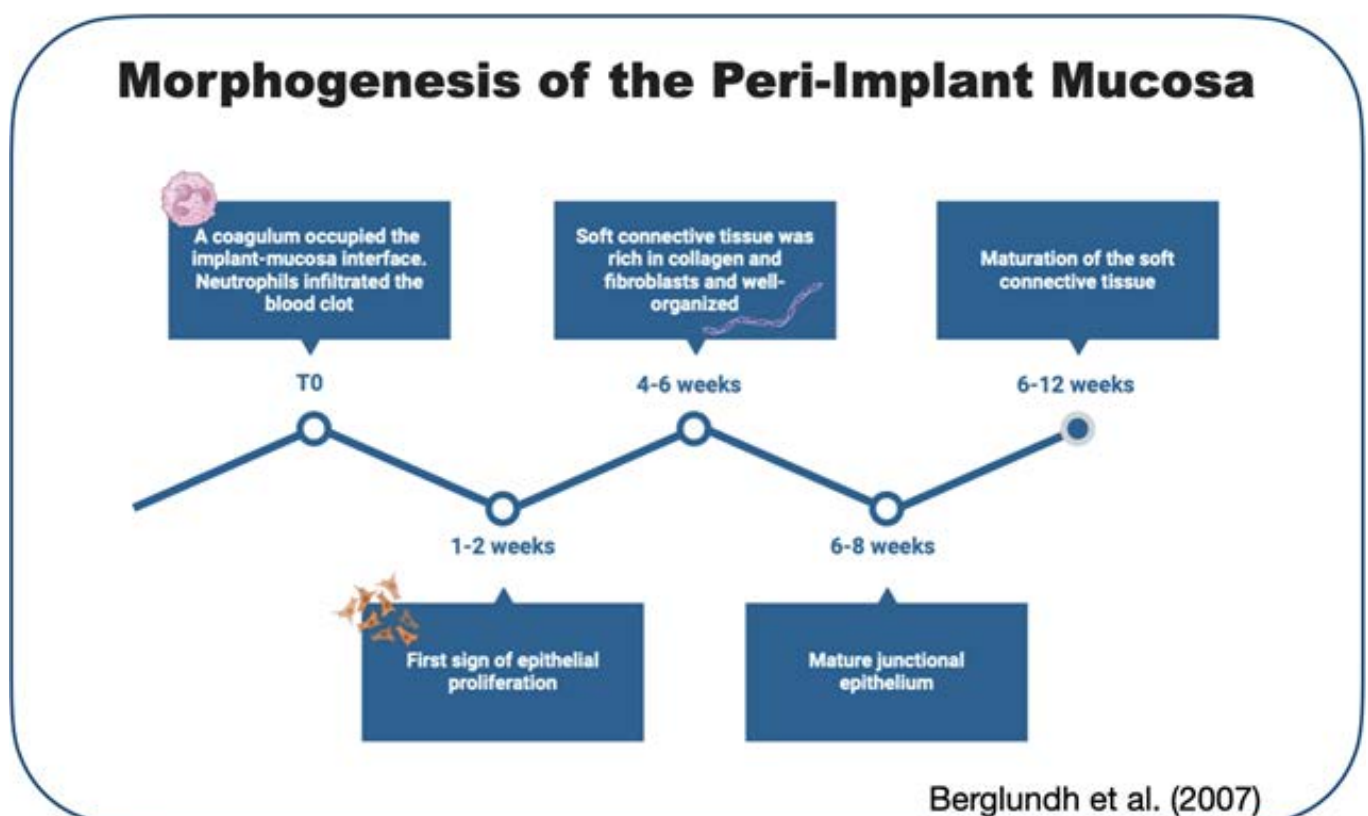
In summary, this study revealed that the peri-implant mucosa exhibits anatomical features comparable to those of the gingiva surrounding natural teeth. Importantly, the study identified that peri-implant connective tissue fibres run parallel to the implant surface and do not attach to it, unlike dento-gingival fibres, which are oriented perpendicularly and attach directly to the root cementum. This finding was later confirmed in a pre-clinical experiment (22), and in humans based on explanted implants (23,24). Additionally, the connective tissue adhesion zone appears to contain limited vascular structures, the vascular supply at implant sites is reduced due to the absence of the periodontal ligament, with nourishment derived solely from the supra-periosteal blood vessels (25) (Fig. 1).

Moreover, Berglundh et al. (19) described the morphogenesis of the peri-implant mucosa on non-submerged implants in a pre-clinical *in vivo* experiment. Healing periods varied from 2 hours to 12 weeks, and the morphogenesis was analysed in histological sections and through histomorphometry. During the initial phase of healing, a substantial number of neutrophils infiltrated and degraded the coagulum at

the implant-mucosa interface. The first signs of epithelial proliferation appeared after 1-2 weeks of healing, and at 4-6 weeks, collagen fibres were organised. Subsequently, a mature barrier epithelium was observed after 6-8 weeks. From 6 to 12 weeks, maturation of the soft connective tissue occurred (Fig. 2).

Later studies demonstrated that similar mucosal attachments formed around both non-submerged and submerged implants (26-28). The peri-implant junctional epithelium was significantly longer in initially submerged implants with second-stage transmucosal abutments than in non-submerged implants (28), showing dimensions more similar to natural teeth around one-piece non-submerged implants compared to two-piece either submerged or non-submerged implants (29). Furthermore, epithelial cells attach to various implant materials via hemidesmosomes and a basal lamina (20). Nevertheless, abutments made of gold alloy or dental porcelain resulted in poor mucosal healing, leading to a connective tissue attachment in a more apical location and greater marginal bone loss (30). It is noteworthy that most data regarding the structural features of the peri-implant

**Fig. 2. Morphogenesis of the peri-implant mucosa timeline. Adapted from Berglundh *et al.* (19).**



mucosa are based on animal studies using dog models where implants were placed in the edentulous ridge (18).

The height of the peri-implant biological width was investigated in a further experiment in dogs, comparing tissue establishment after abutment connection with or without a reduced ( $< 2$  mm) vertical tissue thickness (31). The peri-implant junctional epithelium was approximately 2 mm in length and supra-alveolar soft connective tissue measured about 1.3-1.8 mm in height. Additionally, a paradigm shift was introduced, as sites with reduced mucosa thickness consistently showed wound healing including bone resorption, suggesting that a minimum height of the peri-implant mucosa is necessary, and that bone resorption occurs to create space to accommodate the epithelial and connective tissue components of the transmucosal attachment. Moreover, surface topography does not appear to impact supracrestal soft tissue, which is considered a physiologically formed and stable dimension (29, 32). However, the horizontal mismatch of platform-switching implants leads to the medialisation of the biologic width and seems to reduce marginal bone resorption (34). Clinical studies have reported favourable soft and hard tissue responses to this newer design (35-37).

In summary, the attachment of soft tissue to implants is effectively established only after several weeks of healing (19). The literature consistently shows that the peri-implant STH is about 3-4 mm. The soft peri-implant mucosa acts as a physiological barrier between the oral mucosa and the peri-implant bone, protecting the surrounding bone and providing anatomical support to withstand functional loads while facilitating the host tissue's immunological response (38).

## 2.2 Susceptibility to Inflammation

Peri-implant diseases are described as inflammatory entities triggered by biofilm pathogenic factors and are prevalent, affecting up to 47% of patients (39, 40). Peri-implant mucositis is an inflammatory reaction in the soft tissues surrounding a dental implant and is considered a precursor to peri-implantitis (41, 42). Peri-implantitis is characterised by inflammation in the peri-implant mucosa and progressive loss of supporting bone, it progresses in an accelerating and nonlinear manner (43, 44).

The anatomical and compositional differences between peri-implant mucosa and the periodontium, such as parallel-oriented fibres (21), diminished vascular supply (25), and a thinner, more permeable junctional epithelium with a

connective tissue containing fewer fibroblasts (45), make dental implants more susceptible to inflammation and subsequent bone loss due to microbial challenge (20, 46, 47). Additionally, the host response of peri-implant mucosa is less pronounced than that of the gingiva (48).

It has been demonstrated that an adequate quantity and quality of mucosa surrounding the peri-implant bone is essential for maintaining peri-implant health (6, 39, 44, 49).

## 3. Significance of Soft Tissue Dimension Around Dental Implants

Avila-Ortiz et al. (50) defined the peri-implant phenotype as the "morphologic and dimensional features characterising the clinical presentation of the tissues that surround and support osseointegrated implants." This includes peri-implant keratinised mucosa width, mucosa thickness, supracrestal tissue height, and peri-implant bone thickness. The peri-implant soft tissue phenotype specifically comprises KMW, MT, and STH (51). Understanding and respecting the peri-implant phenotype critical dimensions is crucial for achieving predictable, long-term success with implant therapy (6, 52, 53).

### 3.1 Keratinised Mucosa

The soft tissues around dental implants are classified as keratinised or non-keratinised alveolar mucosa (54). The keratinised mucosa (KM) is the portion of the masticatory mucosa extending from the mucosal margin to the mucogingival junction. It consists of dense connective tissue rich in collagen fibres, firmly connected to the lamina propria and covered by keratinised epithelium. It may or may not be attached to the periosteum, particularly if the transition between keratinised and non-keratinised mucosa is located coronal to the bone. The lining mucosa, on the other hand, is covered with non-keratinised epithelium and has a lamina propria rich in elastic fibres, making it a mobile tissue that adapts to muscle tensions (55). Movable mucosa facilitates the introduction of microorganisms into the crevice, resulting in bacterial plaque (56). The significance of keratinised mucosa width has been a subject of much debate (49, 57).

Studies investigating patient-reported outcome measures (PROMs) have shown a direct relationship between narrow or "inadequate" ( $< 2$  mm) KMW and brushing discomfort, especially in the posterior region (58) and lower teeth (59, 60). There appears to be consensus regarding the brushing comfort conferred by adequate KMW (61). Additionally, sites with narrow KMW are more prone to plaque accumulation,





bleeding on probing, peri-implant soft tissue inflammation, and buccal soft tissue dehiscence (BSTD) compared to sites with  $\geq 2$  mm KMW (58-64). Further, a negative correlation between the presence of adequate KMW and marginal bone loss (MBL) has been reported (58, 62, 63). In line, a recent consensus report with a meta-analysis stated that a greater MBL can be expected around implants with  $< 2$  mm KMW (67). Moreover, these sites are associated with increased expression of pro-inflammatory mediators (64). Inadequate KMW also represents a risk indicator for peri-implant mucositis severity (6, 65, 66), and was associated with an increased prevalence of peri-implantitis (49, 57). In contrast, although lack of adequate KMW may be linked to a higher prevalence of peri-implant mucositis, recent clinical evidence suggests that it is not necessarily associated to a higher prevalence of peri-implantitis (62). Additionally, adequate KMW plays a positive role in the resolution of peri-implant mucositis (39, 65). And has been linked to a significant impact on long-term success of reconstructive treatment of peri-implantitis (67). These findings were corroborated by a recent systematic review and meta-analysis (68). Congruently, several authors advocate that possessing at least 2 mm of KMW has a protective effect on peri-implant tissues and health (6, 49, 57-59). Furthermore, in the presence of brushing discomfort when KMW is  $< 2$  mm, it has been advised that the augmentation of KMW may be considered to preserve peri-implant health (69). However, while the presence of KMW of at least 2 mm seems to reduce the incidence of peri-implant mucosal inflammation, a width beyond 2 mm does not appear to provide additional benefits compared to an adequate KMW. (62, 65) (Fig. 3).

Moreover, the presence of keratinised mucosa is linked to better aesthetic outcomes, as the natural appearance of soft

tissues around a dental implant is primarily determined by the position, colour, and texture of the peri-implant mucosa (14, 70, 71). A survey by Bonino et al. (54) demonstrated that patient-reported satisfaction following implant therapy is significantly influenced by the presence or absence of KM.

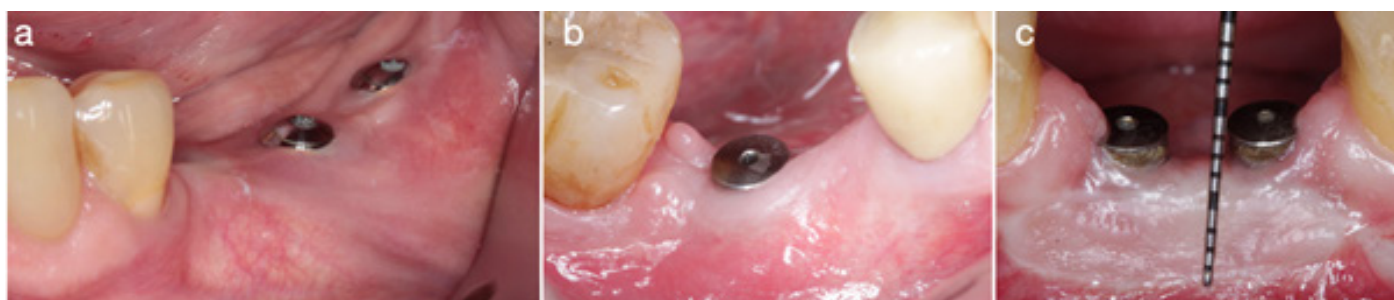
In conclusion, an adequate width of keratinised mucosa plays an important role in supporting peri-implant health by enabling effective self-performed oral hygiene. This can lead to a reduction in plaque accumulation, tissue inflammation, BSTD, and bone loss. Furthermore, the presence of KM helps maintain the natural architecture and colour of the soft tissue, resembling the natural dentition.

### 3.2 Mucosal Thickness

Peri-implant mucosal thickness refers to the horizontal dimension of the peri-implant soft tissue, which can be either keratinised or non-keratinised. This thickness can vary in different locations relative to the mucosal margin around a given implant. It plays a crucial role in aesthetics and tissue stability. Based on pre-clinical and clinical studies that established a threshold mucosal thickness of 2 mm to assess the natural appearance of the peri-implant mucosa and tissue stability, a categorisation of  $< 2$  mm (thin) and  $\geq 2$  mm (thick) mucosal thickness was proposed (50, 51).

The role of mucosal thickness on implant-related outcomes has been extensively investigated (6, 15, 72). Thin MT compromises aesthetics by affecting the peri-implant mucosa's ability to mask abutment shades. This results in noticeable colour differences with neighbouring gingival tissues, limiting abutment material choices. A minimum MT of 2 mm is required to reduce discolouration from restorative abutment materials such as gold and zirconia. Additionally, a pre-clinical study found that a 3 mm MT could mask all restorative materials (72-78).

**Fig. 3. Keratinised mucosa width around non-restored dental implants. (a) Inadequate ( $< 2$  mm) KMW, (b) Adequate ( $\geq 2$  mm) KMW, (c) More than 2 mm of KMW.**





A recent systematic review analysing the influence of peri-implant soft tissue thickness on aesthetic outcomes reported that changes in the pink aesthetic score (Furhauser et al., 2005) (14) during follow-up, papilla index (Jemt, 1999) (79), and patient-reported outcome measures were significantly more favourable for thick MT (16). Accordingly, patients with a thick phenotype were associated with more papilla fill, consistent with the findings of a previous retrospective study (80).

The literature suggests that proper MT is crucial for tissue stability. Thin MT is a risk factor for BSTD, particularly in immediate placement protocols, where a 1 mm apical migration of the mucosal margin can be expected in the first year, without soft tissue thickening procedures (81–84). A recent systematic review and meta-analysis (85) demonstrated that BSTD is consistently related to thin MT and buccally placed implants, with the odds of BSTD being 2.85 times greater in patients with thin MT compared to those with thick MT. Based on these results and previous reports, thick MT is suggested to be a protective factor against BSTD. Soft tissue dehiscence can expose prosthetic components or the implant surface, compromising aesthetics. This condition can impair soft tissue health, as the exposure of the implant's rough surface creates a favourable environment for bacterial colonisation, which can be a critical factor in the initiation of peri-implantitis (15, 85, 86).

In addition, a recent network meta-analysis highlighted the benefit of thick MT and phenotype modification in augmenting peri-implant MT for marginal bone level stability (51), which was later supported in a systematic review with meta-analysis by Stefanini et al. (2023) (15). Consequently, surgical procedures to augment soft tissue thickness are recommended for both biological and aesthetic purposes. However, mucosa thickening, regardless of the graft used, was not associated with KMW gain in bilaminar techniques (51). Furthermore, while mucosa thickening is linked to greater marginal bone stability over time, no improvement in other clinical parameters such as probing depth, bleeding indices, and plaque indices was identified (72, 87). Notably, most available studies evaluated MT through mucosal probing transparency, thus not assessing the peri-implant phenotype as a whole, which also includes STH, KMW and bone thickness.

The relationship between a thin MT and BSTD could be based on the experiment conducted to investigate the pathogenesis of gingival recessions around teeth by Baker & Seymour in 1976 (88), which noted that thin tissues may

not withstand localized inflammatory processes, leading to the breakdown of connective tissue and allowing epithelial proliferation into areas of connective tissue destruction.

In summary, mucosal thickness is a crucial factor influencing most aesthetic parameters. Thin MT can result in increased bone remodelling, reduced papilla fill, BSTD, and an unnatural mucosal appearance. Furthermore, adequate MT can enhance the emergence profile, facilitating better self-performed hygiene (72). Current evidence recommends a minimum of 2 mm MT to prevent complications and support long-term tissue stability. In cases where the mucosa is thin, soft tissue augmentation procedures are advised.

### 3.3 Supracrestal Tissue Height

Peri-implant soft tissue height refers to the vertical measurement of the soft tissue surrounding a dental implant, extending from the mucosal margin to the crestal bone. This includes the sulcular epithelium, the junctional epithelium, and the supracrestal connective tissue (50, 89). It is greater in interproximal areas and typically 1–1.5 mm higher than the adjacent gingiva (18).

The importance of STH was highlighted in animal studies as early as 1996 (30). Clinically, it has been demonstrated that a minimum vertical soft tissue height of at least 3 mm is required to maintain crestal bone stability. Implants placed in thin tissue sites (mean 1.95 mm) were reported to show greater marginal bone loss compared to those placed in thicker tissue sites (mean 3.3 mm) (90). Similarly, a series of studies by the same research group demonstrated that thin STH is associated with greater MBL and increasing STH through soft tissue augmentation effectively reduced peri-implant bone loss (52, 90–92). The association between thin STH and higher MBL is particularly evident for implants placed at the bone level (93). Additionally, STH is strongly associated with papilla volume (80, 94–97).

Recent systematic reviews have confirmed the crucial role that soft tissue height plays in maintaining crestal bone stability. Additionally, they highlight that increasing soft tissue height can provide this stability (15, 51, 93). However, evidence also indicates that a short prosthetic abutment represents a strong predisposing factor for early MBL, regardless of STH (98, 99).

Moreover, caution should be exercised during planning as excessive STH may be disadvantageous for peri-implant health. A mucosal tunnel (distance between the bottom of the sulcus and the mucosal margin) of more than 3 mm can affect the resolution of peri-implant mucositis and hinder



proper hygiene (100). Excessive STH around implants in patients with a history of periodontitis adversely affects peri-implant tissue health, with the risk of peri-implantitis increasing 1.5 times for each 1 mm increase in STH (101). To avoid complications, implant placement should follow the concept of “as shallow as possible, as deep as necessary” (50, 89, 102).

The contour of a prosthesis, particularly the emergence profile, significantly influences crestal bone maintenance. The relationship between the height of peri-implant tissue and the prosthesis contour is crucial for the health of the implant supracrestal complex (52, 103, 104). Careful manipulation of the peri-implant soft tissue emergence profile is of utmost importance for optimal results.

Establishing adequate soft tissue height to create sufficient space for the supra-crestal soft tissue is essential. Current evidence supports this rationale regardless of implant design (bone or tissue level) or restorative interface (such as platform switching). To prevent aesthetic complications, ensuring optimal 3D implant positioning and a proper emergence profile are crucial (50, 89).

### 3.4 Papilla Height

The height of the papilla is a key aspect for aesthetics and, consequently, fundamental for the success of implant rehabilitation. It is strongly related to implant positioning as well as the establishment and dimensions of the peri-implant soft tissue (14, 16, 80).

For single-tooth implant-supported restorations, the level of the connective tissue attachment on the adjacent tooth and the position of the contact point between the crowns are key factors in determining whether a complete papilla fill will be achieved (94, 10, 106). Choquet et al. (94) found that when the distance from the contact point to the bone crest was  $\leq 5$  mm, the papilla was fully or almost fully present. A clear shift was observed at a distance of 5 to 6 mm, where the papilla was missing 50% of the time. Additionally, the height of the papilla at single-implant restorations seems to have a biologic limit of about 4 mm. Papilla fill does not appear to be related to whether the prosthetic crown is positioned immediately after surgery or after soft tissue healing (96).

The position of the papilla between adjacent implants is determined by the topography of the bone crest and the STH. Tarnow et al. (107) using transmucosal probing to assess papillae STH, found that the mean height of the papillae was 3.4 mm. Later, Gastaldo et al. (95) evaluated

the presence or absence of the papilla between two adjacent implants, and reported that complete papilla fill occurred only at sites where the distance from the bone crest to the contact point between the crown restorations was less than 4 mm. Furthermore, it does not seem to be influenced by different systems (108). Moreover, inter-implant distance directly affects the inter-implant bone crest. Although there is a different behaviour between platform matching and platform switching implants, a distance of at least 3 mm is recommended to preserve the surrounding bone and ensure optimal results (95, 109, 107).

Papilla reconstruction is reported as an unpredictable procedure (95, 111), as papillae are dependent on the bone crest position. It was reported that when a complete papilla fill is observed at baseline, the risk of papilla loss is less than 25% (80). Moreover, a “tissue rebound” with papilla increase was observed during follow-up both between tooth-implant and between implants, although sometimes not significant (80, 82, 112, 113).

## 4. Considerations to Prevent Soft Tissue Complications

Dental implants can experience a range of complications, including technical, prosthetic, biological, and patient-reported related issues (89). Soft tissue complications are not rare in dental implant rehabilitations (47, 51). Frequent issues include the asymmetric appearance of peri-implant mucosa, incomplete papilla fill, unnatural soft tissue colour, and aesthetic voids such as black spaces and volume deficiency (89). The discrepancies between the ideal gingival aspects and the peri-implant mucosa affects the patients' perception of the overall treatment. These deficiencies can arise due to various factors, and can lead to increased marginal bone loss, soft-tissue inflammation, and compromised aesthetics. Correlations between peri-implant soft and hard tissues, implant prosthetic abutments, and implant restorations are complex due to variable patient and implant factors. Thus, thorough planning is essential, as surgical and prosthetic treatments are more predictable when performed in the earlier stages of implant therapy, making the later management of complications a clinical challenge (47, 114).

### 4.1 Implant Positioning

The alveolar bone surrounding an osseointegrated implant provides essential support for soft tissues, which is crucial

for achieving aesthetic outcomes. Implant malpositioning is significantly associated with peri-implant tissue breakdown, including marginal bone resorption, absence of papilla fill, BSTD, and peri-implant diseases (44, 107, 115). Each orientation dimension of implant placement impacts the peri-implant hard and soft tissues in a particular way.

A facial bone wall with adequate height and thickness is vital for long-term stability of harmonious mucosal margins around implants (102). Buccally positioned implants are linked to BSTD (114, 116, 117). Indeed, a buccal bone wall thickness of at least 1.5 mm is known to be necessary to prevent bone resorption and marginal changes (118). A study with single immediate implant reconstructions in the aesthetic zone found that for every 1 mm the implant was placed more buccally from the centre of the alveolus, the buccal crest was positioned 0.22 mm more apically (117). Moreover, in a case control study, implants placed buccally (out of the alveolar envelope) were 34 times more likely to have BSTD than the control group, and KMW > 2 mm was considered a protective factor (119). Interestingly, BSTD of up to 1mm are usually not noticed by patients, unless there is metal surface exposure (120, 121). Implants placed too far facially can cause soft tissue recession and prosthetic complications, while implants placed too far palatally can result in unesthetic and hard-to-maintain ridge-lap restorations.

Additionally, the mesiodistal dimension is crucial for preserving the width of the interproximal bone and the interdental papillae (107, 122). Improper placement of implants too close to each other or to an adjacent tooth may lead to compromised vascular supply, resulting in the loss of the interproximal crest, and subsequent reduction in papillary height. Furthermore, such misplacement can result in restorative complications (102, 123).

The supracrestal tissue dimension should be considered in apico-coronal positioning to provide adequate space for the soft tissue height establishment and a smooth, progressive transition of the restoration emergence profile. The peri-implant STH is about 3-4 mm in height (18). Implants placed too deep may present with more vertical bone resorption and subsequent soft-tissue loss, besides leading to a greater mucosal tunnel. While implants placed too shallow can result in bone resorption for the establishment of the supracrestal soft tissues, impair the ideal emergence profile and lead to restorative complications (124, 125).

In line, implant malpositioning may influence the onset and progression of peri-implantitis (126, 127). Therefore,

prosthetic-driven planning that respects recommended dimensions promotes favourable outcomes, with augmentation procedures performed as needed (1, 9, 102).

## 4.2 Soft Tissue Dimensions

Critical soft tissue dimensions are essential for successful implant therapy as discussed throughout this review. Significantly influencing the biologic and aesthetic outcomes. Recent evidence recommends soft tissue augmentation when necessary, enhancing tissue stability, peri-implant health, and compensating for volume deficiencies. While the timing of augmentation does not significantly affect keratinised mucosal width or soft tissue thickening, augmenting soft tissue height at implant placement time may prevent marginal bone loss. Moreover, addressing soft tissue conditions before reconstruction helps prevent complications and ensures peri-implant tissues stability (15, 47, 51).

The 7<sup>th</sup> ITI Consensus stated that single implant sites augmented with connective tissue grafts maintain stable soft tissue margins, increased thickness, and keratinised mucosa width for up to five years. These sites also show stable or improved aesthetic outcomes, including pink aesthetic scores and patient-reported aesthetics for the same period (128).

## 4.3 Prosthetic Reconstruction

The transmucosal portion of the prosthetic abutment, commonly referred to as the emergence profile, arises from the restorative platform and extends through the mucosa, progressing toward the cervical contour of the implant restoration, providing support for the peri-implant soft tissues (129). Gomez-Meda et al. (130) categorised the extension of the emergence profile into three zones: the aesthetic zone, the bounded zone, and the crestal zone.

The aesthetic zone, approximately 1 mm in height, is located immediately apical to the mucosal margin. This zone should be convex to establish the morphology of the clinical portion of the implant crown, determining the level of the mucosal margin on the buccal side and the crown shape in interproximal areas.

The bounded zone, 1-2 mm in height, is situated apical to the aesthetic zone and is influenced by the thickness of the surrounding tissues, implant position, and implant neck design. It should preferably be concave and contains the junctional epithelium.

The crestal zone, the most apical one, is 1-1.5 mm in height. In this zone, the abutment should be concave or

**Fig. 4. Manipulation of the emergence profile during provisional phase (a). Emergence profile with the definitive abutments from an occlusal view (b), and from a buccal view (c), showing adequate marginal contour and inter-implant papilla. Note that there is presence of thick MT, which is favourable for the treatment outcomes longevity. Source: Dr. Rafael Lazarin.**



straight to avoid pressuring the adjacent bone, with the supracrestal connective tissue present in this area.

An improper emergence profile (greater than a 30-degree angle) and over-contoured restoration are linked to increased plaque accumulation and early marginal bone loss (104). When designing the emergence profile, a convex contour can help displace buccal soft tissues, while a concave contour maintains their position. This allows for manipulation of the tissue to achieve optimal aesthetics. Therefore, manipulating the emergence profile architecture through provisional restoration or customized abutment is recommended for optimal results, especially in the aesthetic zone (131, 132) (Fig. 4).

Moreover, iatrogenic factors, such as poor marginal fit and excessive submucosal cement deposits, also contribute to biological complications (126). Therefore, the correct selection of restorative material and restoration modality (cemented or screw-retained) is of paramount importance for avoiding complications.

#### 4.4 Supportive Therapy

Soft tissue stability is directly influenced by its health, as peri-implant diseases lead to tissue inflammation and destruction. Adhering to recommended recall intervals has a well-documented positive impact on peri-implant health and long-term maintenance (41, 57, 126). The 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions identified non-compliance with maintenance protocols as one of the major risk factors for peri-implant diseases, alongside with history of periodontitis and poor plaque control (61). Adequate KMW has been described as a positive factor in facilitating self-performed hygiene (58, 59, 60). Additionally, it has been

associated with better peri-implant health in patients with erratic compliance to supportive care (57).

## 5. Conclusion

Understanding the biology and clinical significance of the peri-implant mucosa is crucial for successful implant therapy. The components of the peri-implant soft tissue phenotype play a pivotal role in maintaining tissue stability, influencing both health and aesthetics. Thus, establishing adequate dimensions of peri-implant anatomical structures is crucial for successful clinical outcomes with minimal complications. Achieving optimal clinical, biological, and patient-reported outcomes requires a comprehensive approach, from implant selection to tissue grafting and prosthetic design. This integrated strategy is essential for predictable, durable, and aesthetically pleasing results in implant therapy.

## References

1. Buser D, Janner SF, Wittneben JG, Bragger U, Ramseier CA, Salvi GE. 10-year survival and success rates of 511 titanium implants with a sandblasted and acid-etched surface: a retrospective study in 303 partially edentulous patients. *Clin Implant Dent Relat Res*. 2012;14(6):839-51.
2. Buser D, Sennerby L, De Bruyn H. Modern implant dentistry based on osseointegration: 50 years of progress, current trends and open questions. *Periodontol 2000*. 2017;73(1):7-21.
3. Gotfredsen K. A 10-year prospective study of single tooth implants placed in the anterior maxilla. *Clin Implant Dent Relat Res*. 2012;14(1):80-7.



4. Jung RE, Zembic A, Pjetursson BE, Zwahlen M, Thoma DS. Systematic review of the survival rate and the incidence of biological, technical, and aesthetic complications of single crowns on implants reported in longitudinal studies with a mean follow-up of 5 years. *Clin Oral Implants Res*. 2012;23 Suppl 6:2-21.
5. Guo T, Gulati K, Arora H, Han P, Fournier B, Ivanovski S. Race to invade: Understanding soft tissue integration at the transmucosal region of titanium dental implants. *Dent Mater*. 2021;37(5):816-31.
6. Gharpure AS, Latimer JM, Aljofi FE, Kahng JH, Daubert DM. Role of thin gingival phenotype and inadequate keratinized mucosa width (<2 mm) as risk indicators for peri-implantitis and peri-implant mucositis. *J Periodontol*. 2021;92(12):1687-96.
7. De Bruyn H, Raes S, Matthys C, Cosyn J. The current use of patient-centered/reported outcomes in implant dentistry: a systematic review. *Clin Oral Implants Res*. 2015;26 Suppl 11:45-56.
8. Monje A, Nart J. Management and sequelae of dental implant removal. *Periodontol 2000*. 2022;88(1):182-200.
9. Buser D, Chappuis V, Belser UC, Chen S. Implant placement post extraction in esthetic single tooth sites: when immediate, when early, when late?. *Periodontol 2000*. 2017;73(1):84-102.
10. Thoma DS, Zeltner M, Hilbe M, Hämmerle CH, Hüsler J, Jung RE. Randomized controlled clinical study evaluating effectiveness and safety of a volume-stable collagen matrix compared to autogenous connective tissue grafts for soft tissue augmentation at implant sites. *J Clin Periodontol*. 2016;43(10):874-885.
11. Pjetursson BE, Asgeirsson AG, Zwahlen M, Sailer I. Improvements in implant dentistry over the last decade: comparison of survival and complication rates in older and newer publications. *Int J Oral Maxillofac Implants*. 2014;29 Suppl:308-324.
12. Thoma DS, Muhlemann S, Jung RE. Critical soft-tissue dimensions with dental implants and treatment concepts. *Periodontol 2000*. 2014;66(1):106-18.
13. Belser UC, Grütter L, Vailati F, Bornstein MM, Weber HP, Buser D. Outcome evaluation of early placed maxillary anterior single-tooth implants using objective esthetic criteria: a cross-sectional, retrospective study in 45 patients with a 2- to 4-year follow-up using pink and white esthetic scores. *J Periodontol*. 2009;80(1):140-151.
14. Fürhauser R, Florescu D, Benesch T, Haas R, Mailath G, Watzek G. Evaluation of soft tissue around single-tooth implant crowns: the pink esthetic score. *Clin Oral Implants Res*. 2005;16(6):639-644.
15. Stefanini M, Barootchi S, Sangiorgi M, et al. Do soft tissue augmentation techniques provide stable and favorable peri-implant conditions in the medium and long term? A systematic review. *Clin Oral Implants Res*. 2023;34 Suppl 26:28-42.
16. Bienz SP, Pirc M, Papageorgiou SN, Jung RE, Thoma DS. The influence of thin as compared to thick peri-implant soft tissues on aesthetic outcomes: A systematic review and meta-analysis. *Clin Oral Implants Res*. 2022;33 Suppl 23(Suppl 23):56-71.
17. Tettamanti S, Millen C, Gavric J, et al. Esthetic Evaluation of Implant Crowns and Peri-Implant Soft Tissue in the Anterior Maxilla: Comparison and Reproducibility of Three Different Indices. *Clin Implant Dent Relat Res*. 2016;18(3):517-526.
18. Araujo MG, Lindhe J. Peri-implant health. *J Clin Periodontol*. 2018;45 Suppl 20:S230-S6.
19. Berglundh T, Abrahamsson I, Welander M, Lang NP, Lindhe J. Morphogenesis of the peri-implant mucosa: an experimental study in dogs. *Clin Oral Implants Res*. 2007;18(1):1-8.
20. Sculean A, Gruber R, Bosshardt DD. Soft tissue wound healing around teeth and dental implants. *J Clin Periodontol*. 2014;41 Suppl 15:S6-22.
21. Berglundh T, Lindhe J, Ericsson I, Marinello CP, Liljenberg B, Thomsen P. The soft tissue barrier at implants and teeth. *Clin Oral Implants Res*. 1991;2(2):81-90.
22. Listgarten MA, Buser D, Steinemann SG, Donath K, Lang NP, Weber HP. Light and transmission electron microscopy of the intact interfaces between non-submerged titanium-coated epoxy resin implants and bone or gingiva [published correction appears in *J Dent Res* 1992 May;71(5):1267]. *J Dent Res*. 1992;71(2):364-371.
23. Degidi M, Piattelli A, Scarano A, Shibli JA, Iezzi G. Peri-implant collagen fibers around human cone Morse connection implants under polarized light: a report of three cases. *Int J Periodontics Restorative Dent*. 2012;32(3):323-328.





24. Schierano G, Ramieri G, Cortese M, Aimetti M, Preti G. Organization of the connective tissue barrier around long-term loaded implant abutments in man. *Clin Oral Implants Res.* 2002;13(5):460-464. doi:10.1034/j.1600-0501.2002.130503.x
25. Berglundh T, Lindhe J, Jonsson K, Ericsson I. The topography of the vascular systems in the periodontal and peri-implant tissues in the dog. *J Clin Periodontol.* 1994;21(3):189-193.
26. Abrahamsson I, Berglundh T, Wennström J, Lindhe J. The peri-implant hard and soft tissues at different implant systems. A comparative study in the dog. *Clin Oral Implants Res.* 1996;7(3):212-219.
27. Abrahamsson I, Zitzmann NU, Berglundh T, Linder E, Wennerberg A, Lindhe J. The mucosal attachment to titanium implants with different surface characteristics: an experimental study in dogs. *J Clin Periodontol.* 2002;29(5):448-455.
28. Weber HP, Buser D, Donath K, et al. Comparison of healed tissues adjacent to submerged and non-submerged unloaded titanium dental implants. A histometric study in beagle dogs. *Clin Oral Implants Res.* 1996;7(1):11-19.
29. Hermann JS, Buser D, Schenk RK, Schoolfield JD, Cochran DL. Biologic Width around one- and two-piece titanium implants. *Clin Oral Implants Res.* 2001;12(6):559-571.
30. Abrahamsson I, Berglundh T, Glantz PO, Lindhe J. The mucosal attachment at different abutments. An experimental study in dogs. *J Clin Periodontol.* 1998;25(9):721-727.
31. Berglundh T, Lindhe J. Dimension of the periimplant mucosa. Biological width revisited. *J Clin Periodontol.* 1996;23(10):971-973.
32. Cochran DL, Hermann JS, Schenk RK, Higginbottom FL, Buser D. Biologic width around titanium implants. A histometric analysis of the implanto-gingival junction around unloaded and loaded nonsubmerged implants in the canine mandible. *J Periodontol.* 1997;68(2):186-198.
33. Cochran DL, Bosshardt DD, Grize L, et al. Bone response to loaded implants with non-matching implant-abutment diameters in the canine mandible. *J Periodontol.* 2009;80(4):609-617.
34. Jung RE, Jones AA, Higginbottom FL, et al. The influence of non-matching implant and abutment diameters on radiographic crestal bone levels in dogs. *J Periodontol.* 2008;79(2):260-270.
35. Canullo L, Fedele GR, Iannello G, Jepsen S. Platform switching and marginal bone-level alterations: the results of a randomized-controlled trial. *Clin Oral Implants Res.* 2010;21(1):115-121.
36. Hämmerle CHF, Jung RE, Sanz M, et al. Submerged and transmucosal healing yield the same clinical outcomes with two-piece implants in the anterior maxilla and mandible: interim 1-year results of a randomized, controlled clinical trial. *Clin Oral Implants Res.* 2012;23(2):211-219.
37. Hürzeler M, Fickl S, Zuh O, Wachtel HC. Peri-implant bone level around implants with platform-switched abutments: preliminary data from a prospective study [published correction appears in *J Oral Maxillofac Surg.* 2008 Oct;66(10):2195-6]. *J Oral Maxillofac Surg.* 2007;65(7 Suppl 1):33-39.
38. Romanos G. *Saving Dental Implants.* 1st ed. Wiley-Blackwell; 2024.
39. Blasi G, Chierico F, Amerio E, et al. Influence of keratinized mucosa width on the resolution of peri-implant mucositis: A prospective cohort study. *Clin Implant Dent Relat Res.* 2024;26(3):581-591.
40. Lee CT, Huang YW, Zhu L, Weltman R. Prevalences of peri-implantitis and peri-implant mucositis: systematic review and meta-analysis. *J Dent.* 2017;62:1-12.
41. Costa FO, Takenaka-Martinez S, Cota LO, Ferreira SD, Silva GL, Costa JE. Peri-implant disease in subjects with and without preventive maintenance: a 5-year follow-up. *J Clin Periodontol.* 2012;39(2):173-181.
42. Renvert S, Polyzos I. Risk indicators for peri-implant mucositis: a systematic literature review. *J Clin Periodontol.* 2015;42 Suppl 16:S172-S186.
43. Derks J, Schaller D, Håkansson J, Wennström JL, Tomasi C, Berglundh T. Peri-implantitis - onset and pattern of progression. *J Clin Periodontol.* 2016;43(4):383-388.
44. Schwarz F, Derks J, Monje A, Wang HL. Peri-implantitis. *J Periodontol.* 2018;89(Suppl 1):S267-S290.
45. Moon IS, Berglundh T, Abrahamsson I, Linder E, Lindhe J. The barrier between the keratinized mucosa and the dental implant. An experimental study in the dog. *J Clin Periodontol.* 1999;26(10):658-663.

46. Salvi GE, Aglietta M, Eick S, Sculean A, Lang NP, Ramseier CA. Reversibility of experimental peri-implant mucositis compared with experimental gingivitis in humans. *Clin Oral Implants Res.* 2012;23(2):182-190.
47. Thoma DS, Gil A, Hämmerle CHF, Jung RE. Management and prevention of soft tissue complications in implant dentistry. *Periodontol 2000.* 2022;88(1):116-129.
48. Zitzmann NU, Berglundh T, Marinello CP, Lindhe J. Experimental peri-implant mucositis in man. *J Clin Periodontol.* 2001;28(6):517-523.
49. Sanz M, Schwarz F, Herrera D, McClain P, Figuero E, Molina A, et al. Importance of keratinized mucosa around dental implants: Consensus report of group 1 of the DGI/SEPA/Osteology Workshop. *Clin Oral Implants Res.* 2022;33 Suppl 23:47-55.
50. Avila-Ortiz G, Gonzalez-Martin O, Couso-Queiruga E, Wang HL. The peri-implant phenotype. *J Periodontol.* 2020;91(3):283-8.
51. Tavelli L, Barootchi S, Avila-Ortiz G, Urban IA, Giannobile WV, Wang HL. Peri-implant soft tissue phenotype modification and its impact on peri-implant health: A systematic review and network meta-analysis. *J Periodontol.* 2021;92(1):21-44.
52. Puisys A, Vindasiute-Narbutė E, Razukevicius D, Akhondi S, Gallucci GO, Pedrinaci I. Influence of mucosal tissue height on implant crestal bone: A 10-year follow-up of a controlled clinical trial. *J Dent.* Published online June 30, 2024.
53. Schwarz F, Ramanauskaite A. It is all about peri-implant tissue health. *Periodontol 2000.* 2022;88(1):9-12.
54. Bonino F, Steffensen B, Natto Z, Hur Y, Holtzman LP, Weber HP. Prospective study of the impact of peri-implant soft tissue properties on patient-reported and clinically assessed outcomes. *J Periodontol.* 2018;89(9):1025-32.
55. Nanci, A. (2018). Ten Cate's oral histology: development, structure, and function (Ninth edition). Elsevier.
56. Lang NP, Loe H. The relationship between the width of keratinized gingiva and gingival health. *J Periodontol.* 1972;43(10):623-7.
57. Monje A, Blasi G. Significance of keratinized mucosa/gingiva on peri-implant and adjacent periodontal conditions in erratic maintenance compliers. *J Periodontol.* 2019;90(5):445-53.
58. Perussolo J, Souza AB, Matarazzo F, Oliveira RP, Araujo MG. Influence of the keratinized mucosa on the stability of peri-implant tissues and brushing discomfort: A 4-year follow-up study. *Clin Oral Implants Res.* 2018;29(12):1177-85.
59. Rocuzzo M, Grasso G, Dalmaso P. Keratinized mucosa around implants in partially edentulous posterior mandible: 10-year results of a prospective comparative study. *Clin Oral Implants Res.* 2016;27(4):491-6.
60. Souza AB, Tormena M, Matarazzo F, Araujo MG. The influence of peri-implant keratinized mucosa on brushing discomfort and peri-implant tissue health. *Clin Oral Implants Res.* 2016;27(6):650-5.
61. Berglundh T, Armitage G, Araujo MG, Avila-Ortiz G, Blanco J, Camargo PM, et al. Peri-implant diseases and conditions: Consensus report of workgroup 4 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Periodontol.* 2018;89 Suppl 1:S313-S8.
62. Mancini L, Strauss FJ, Lim HC, et al. Impact of keratinized mucosa on implant-health related parameters: A 10-year prospective re-analysis study. *Clin Implant Dent Relat Res.* 2024;26(3):554-563.
63. Suzuki A, Nakano T, Inoue M, Isigaki S. Multivariate analysis of the effect of keratinized mucosa on peri-implant tissues with platform switching: A retrospective study. *Clin Implant Dent Relat Res.* 2024;26(3):592-603.
64. Boynuegri D, Nemli SK, Kasko YA. Significance of keratinized mucosa around dental implants: a prospective comparative study. *Clin Oral Implants Res.* 2013;24(8):928-33.
65. Schwarz F, Becker J, Civalé S, Sahin D, Iglhaut T, Iglhaut G. Influence of the width of keratinized tissue on the development and resolution of experimental peri-implant mucositis lesions in humans. *Clin Oral Implants Res.* 2018;29(6):576-82.
66. Grischke J, Karch A, Wenzlaff A, Foitzik MM, Stiesch M, Eberhard J. Keratinized mucosa width is associated with severity of peri-implant mucositis. A cross-sectional study. *Clin Oral Implants Res.* 2019;30(5):457-65.
67. Isler SC, Soysal F, Tunc S, Kaymaz Ö, Unsal B, Rocuzzo A. Impact of peri-implant keratinized mucosa width on the long-term reconstructive outcomes of peri-implantitis: A retrospective analysis with a follow-up up to 10 years. *Clin Implant Dent Relat Res.* Published online June 24, 2024.



68. Ramanauskaite A, Schwarz F, Sader R. Influence of width of keratinized tissue on the prevalence of peri-implant diseases: A systematic review and meta-analysis. *Clin Oral Implants Res.* 2022;33 Suppl 23:8-31.
69. Herrera D, Berglundh T, Schwarz F, et al. Prevention and treatment of peri-implant diseases-The EFP S3 level clinical practice guideline. *J Clin Periodontol.* 2023;50 Suppl 26:4-76.
70. Stefanini M, Felice P, Mazzotti C, Mounssif I, Marzadori M, Zucchelli G. Esthetic evaluation and patient-centered outcomes in single-tooth implant rehabilitation in the esthetic area. *Periodontol 2000.* 2018;77(1):150-164.
71. Zucchelli G, Sharma P, Mounssif I. Esthetics in periodontics and implantology. *Periodontol 2000.* 2018;77(1):7-18.
72. Jung RE, Becker K, Bienz SP, et al. Effect of peri-implant mucosal thickness on esthetic outcomes and the efficacy of soft tissue augmentation procedures: Consensus report of group 2 of the SEPA/DGI/OF workshop. *Clin Oral Implants Res.* 2022;33 Suppl 23(Suppl 23):100-108.
73. Bressan E, Paniz G, Lops D, Corazza B, Romeo E, Favero G. Influence of abutment material on the gingival color of implant-supported all-ceramic restorations: a prospective multicenter study. *Clin Oral Implants Res.* 2011;22(6):631-637.
74. Jung RE, Holderegger C, Sailer I, Khraisat A, Suter A, Hämmerle CH. The effect of all-ceramic and porcelain-fused-to-metal restorations on marginal peri-implant soft tissue color: a randomized controlled clinical trial. *Int J Periodontics Restorative Dent.* 2008;28(4):357-365.
75. Jung RE, Sailer I, Hämmerle CH, Attin T, Schmidlin P. In vitro color changes of soft tissues caused by restorative materials. *Int J Periodontics Restorative Dent.* 2007;27(3):251-257.
76. Park SE, Da Silva JD, Weber HP, Ishikawa-Nagai S. Optical phenomenon of peri-implant soft tissue. Part I. Spectrophotometric assessment of natural tooth gingiva and peri-implant mucosa. *Clin Oral Implants Res.* 2007;18(5):569-574.
77. Kim A, Campbell SD, Viana MA, Knoernschild KL. Abutment Material Effect on Peri-implant Soft Tissue Color and Perceived Esthetics. *J Prosthodont.* 2016;25(8):634-640.
78. Lops D, Stellini E, Sbricoli L, Cea N, Romeo E, Bressan E. Influence of abutment material on peri-implant soft tissues in anterior areas with thin gingival biotype: a multicentric prospective study. *Clin Oral Implants Res.* 2017;28(10):1263-1268.
79. Jemt T. Restoring the gingival contour by means of provisional resin crowns after single-implant treatment. *Int J Periodontics Restorative Dent.* 1999;19(1):20-29.
80. Garabetyan J, Malet J, Kerner S, Detzen L, Carra MC, Bouchard P. The relationship between dental implant papilla and dental implant mucosa around single-tooth implant in the esthetic area: A retrospective study. *Clin Oral Implants Res.* 2019;30(12):1229-1237.
81. Bittner N, Schulze-Späte U, Silva C, et al. Changes of the alveolar ridge dimension and gingival recession associated with implant position and tissue phenotype with immediate implant placement: A randomised controlled clinical trial. *Int J Oral Implantol (Berl).* 2019;12(4):469-480.
82. Kan JY, Rungcharassaeng K, Lozada JL, Zimmerman G. Facial gingival tissue stability following immediate placement and provisionalization of maxillary anterior single implants: a 2- to 8-year follow-up. *Int J Oral Maxillofac Implants.* 2011;26(1):179-187.
83. Sculean A, Chappuis V, Cosgarea R. Coverage of mucosal recessions at dental implants. *Periodontol 2000.* 2017;73(1):134-140.
84. Seyssens L, De Lat L, Cosyn J. Immediate implant placement with or without connective tissue graft: A systematic review and meta-analysis. *J Clin Periodontol.* 2021;48(2):284-301.
85. Sanz-Martín I, Regidor E, Cosyn J, Wiedemeier DB, Thoma DS. Buccal soft tissue dehiscence defects at dental implants-associated factors and frequency of occurrence: A systematic review and meta-analysis. *Clin Oral Implants Res.* 2022;33 Suppl 23:109-124.
86. Zucchelli G, Tavelli L, McGuire MK, et al. Autogenous soft tissue grafting for periodontal and peri-implant plastic surgical reconstruction. *J Periodontol.* 2020;91(1):9-16.
87. Thoma DS, Naenni N, Figuero E, et al. Effects of soft tissue augmentation procedures on peri-implant health or disease: A systematic review and meta-analysis. *Clin Oral Implants Res.* 2018;29 Suppl 15:32-49.

88. Baker DL, Seymour GJ. The possible pathogenesis of gingival recession. A histological study of induced recession in the rat. *J Clin Periodontol.* 1976;3(4):208-219.
89. Wang II, Barootchi S, Tavelli L, Wang HL. The peri-implant phenotype and implant esthetic complications. Contemporary overview. *J Esthet Restor Dent.* 2021;33(1):212-223.
90. Linkevicius T, Apse P, Grybauskas S, Puisys A. The influence of soft tissue thickness on crestal bone changes around implants: a 1-year prospective controlled clinical trial. *Int J Oral Maxillofac Implants.* 2009;24(4):712-719.
91. Linkevicius T, Linkevicius R, Alkimavicius J, Linkeviciene L, Andrijauskas P, Puisys A. Influence of titanium base, lithium disilicate restoration and vertical soft tissue thickness on bone stability around triangular-shaped implants: A prospective clinical trial. *Clin Oral Implants Res.* 2018;29(7):716-724.
92. Puisys A, Linkevicius T. The influence of mucosal tissue thickening on crestal bone stability around bone-level implants. A prospective controlled clinical trial. *Clin Oral Implants Res.* 2015;26(2):123-129.
93. Díaz-Sánchez M, Soto-Peñaloza D, Peñarrocha-Oltra D, Peñarrocha-Diogo M. Influence of supracrestal tissue attachment thickness on radiographic bone level around dental implants: A systematic review and meta-analysis. *J Periodontol Res.* 2019;54(6):573-588.
94. Choquet V, Hermans M, Adriaenssens P, Daelemans P, Tarnow DP, Malevez C. Clinical and radiographic evaluation of the papilla level adjacent to single-tooth dental implants. A retrospective study in the maxillary anterior region. *J Periodontol.* 2001;72(10):1364-1371.
95. Gastaldo JF, Cury PR, Sendyk WR. Effect of the vertical and horizontal distances between adjacent implants and between a tooth and an implant on the incidence of interproximal papilla. *J Periodontol.* 2004;75(9):1242-1246.
96. Roccuzzo M, Roccuzzo A, Ramanuskaite A. Papilla height in relation to the distance between bone crest and interproximal contact point at single-tooth implants: A systematic review. *Clin Oral Implants Res.* 2018;29 Suppl 15:50-61.
97. Valles C, Vilarrasa J, Barallat L, Pascual A, Nart J. Efficacy of soft tissue augmentation procedures on tissue thickening around dental implants: A systematic review and meta-analysis. *Clin Oral Implants Res.* 2022;33 Suppl 23:72-99.
98. Galindo-Moreno P, León-Cano A, Monje A, Ortega-Oller I, O'Valle F, Catena A. Abutment height influences the effect of platform switching on peri-implant marginal bone loss. *Clin Oral Implants Res.* 2016;27(2):167-173.
99. Spinato S, Stacchi C, Lombardi T, Bernardello F, Messina M, Zaffe D. Biological width establishment around dental implants is influenced by abutment height irrespective of vertical mucosal thickness: A cluster randomized controlled trial. *Clin Oral Implants Res.* 2019;30(7):649-659.
100. Chan D, Pelekos G, Ho D, Cortellini P, Tonetti MS. The depth of the implant mucosal tunnel modifies the development and resolution of experimental peri-implant mucositis: A case-control study. *J Clin Periodontol.* 2019;46(2):248-255.
101. Zhang Z, Shi D, Meng H, Han J, Zhang L, Li W. Influence of vertical soft tissue thickness on occurrence of peri-implantitis in patients with periodontitis: a prospective cohort study. *Clin Implant Dent Relat Res.* 2020;22(3):292-300.
102. Buser D, Martin W, Belser UC. Optimizing esthetics for implant restorations in the anterior maxilla: anatomic and surgical considerations. *Int J Oral Maxillofac Implants.* 2004;19 Suppl:43-61.
103. Puisys A, Janda M, Auzbikaviciute V, Gallucci GO, Mattheos N. Contour angle and peri-implant tissue height: Two interrelated features of the implant supracrestal complex. *Clin Exp Dent Res.* 2023;9(3):418-424.
104. Souza AB, Alshihri A, Kämmerer PW, Araújo MG, Gallucci GO. Histological and micro-CT analysis of peri-implant soft and hard tissue healing on implants with different healing abutments configurations. *Clin Oral Implants Res.* 2018;29(10):1007-1015.
105. Kan JY, Rungcharassaeng K, Umez K, Kois JC. Dimensions of peri-implant mucosa: an evaluation of maxillary anterior single implants in humans. *J Periodontol.* 2003;74(4):557-562.
106. Schropp L, Wenzel A, Kostopoulos L, Karring T. Bone healing and soft tissue contour changes following single-tooth extraction: a clinical and radiographic 12-month prospective study. *Int J Periodontics Restorative Dent.* 2003;23(4):313-323.





107. Tarnow D, Elian N, Fletcher P, et al. Vertical distance from the crest of bone to the height of the interproximal papilla between adjacent implants. *J Periodontol*. 2003;74(12):1785-1788.
108. Lee DW, Park KH, Moon IS. Dimension of interproximal soft tissue between adjacent implants in two distinctive implant systems. *J Periodontol*. 2006;77(6):1080-1084.
109. Rodríguez-Ciurana X, Vela-Nebot X, Segalà-Torres M, et al. The effect of interimplant distance on the height of the interimplant bone crest when using platform-switched implants. *Int J Periodontics Restorative Dent*. 2009;29(2):141-151.
110. Tarnow DP, Cho SC, Wallace SS. The effect of inter-implant distance on the height of inter-implant bone crest. *J Periodontol*. 2000;71(4):546-549.
111. Degidi M, Novaes AB Jr, Nardi D, Piattelli A. Outcome analysis of immediately placed, immediately restored implants in the esthetic area: the clinical relevance of different interimplant distances. *J Periodontol*. 2008;79(6):1056-1061.
112. Cosyn J, Eghbali A, Hermans A, Vervaeke S, De Bruyn H, Cleymaet R. A 5-year prospective study on single immediate implants in the aesthetic zone. *J Clin Periodontol*. 2016;43(8):702-709.
113. Koster M, Dias DR, Zimiani GS, Santos R, de Oliveira RP, Araujo MG, et al. Four single implant-supported crowns replacing the maxillary incisors: A retrospective report of 10 cases with 2-9 years of follow-up. *Clin Oral Implants Res*. 2023.
114. Hämmerle CHF, Tarnow D. The etiology of hard- and soft-tissue deficiencies at dental implants: A narrative review. *J Periodontol*. 2018;89 Suppl 1:S291-S303.
115. Canullo L, Tallarico M, Radovanovic S, Delibasic B, Covani U, Rakic M. Distinguishing predictive profiles for patient-based risk assessment and diagnostics of plaque induced, surgically and prosthetically triggered peri-implantitis. *Clin Oral Implants Res*. 2016;27(10):1243-1250.
116. Chen ST, Darby IB, Reynolds EC. A prospective clinical study of non-submerged immediate implants: clinical outcomes and esthetic results. *Clin Oral Implants Res*. 2007;18(5):552-562.
117. Tomasi C, Sanz M, Cecchinato D, et al. Bone dimensional variations at implants placed in fresh extraction sockets: a multilevel multivariate analysis. *Clin Oral Implants Res*. 2010;21(1):30-36.
118. Monje A, Roccuzzo A, Buser D, Wang HL. Influence of buccal bone wall thickness on the peri-implant hard and soft tissue dimensional changes: A systematic review. *Clin Oral Implants Res*. 2023;34(3):157-176.
119. Sanz-Martín I, Regidor E, Navarro J, Sanz-Sánchez I, Sanz M, Ortiz-Vigón A. Factors associated with the presence of peri-implant buccal soft tissue dehiscences: A case-control study. *J Periodontol*. 2020;91(8):1003-1010.
120. Cosyn J, De Rouck T. Aesthetic outcome of single-tooth implant restorations following early implant placement and guided bone regeneration: crown and soft tissue dimensions compared with contralateral teeth. *Clin Oral Implants Res*. 2009;20(10):1063-1069.
121. De Rouck T, Collys K, Cosyn J. Immediate single-tooth implants in the anterior maxilla: a 1-year case cohort study on hard and soft tissue response. *J Clin Periodontol*. 2008;35(7):649-657.
122. Ramanauskaite A, Roccuzzo A, Schwarz F. A systematic review on the influence of the horizontal distance between two adjacent implants inserted in the anterior maxilla on the inter-implant mucosa fill. *Clin Oral Implants Res*. 2018;29 Suppl 15:62-70.
123. Testori T, Weinstein T, Scutellà F, Wang HL, Zucchelli G. Implant placement in the esthetic area: criteria for positioning single and multiple implants. *Periodontol 2000*. 2018;77(1):176-196.
124. Albrektsson T, Chrcanovic B, Östman PO, Sennerby L. Initial and long-term crestal bone responses to modern dental implants. *Periodontol 2000*. 2017;73(1):41-50.
125. Piattelli A, Vrespa G, Petrone G, Iezzi G, Annibali S, Scarano A. Role of the microgap between implant and abutment: a retrospective histologic evaluation in monkeys. *J Periodontol*. 2003;74(3):346-352.
126. Kumar PS, Dabdoub SM, Hegde R, Ranganathan N, Mariotti A. Site-level risk predictors of peri-implantitis: A retrospective analysis. *J Clin Periodontol*. 2018;45(5):597-604.
127. Roccuzzo A, Imber JC, Salvi GE, Roccuzzo M. Peri-implantitis as the consequence of errors in implant therapy. *Periodontol 2000*. 2023;92(1):350-61.



128. Jensen SS, Aghaloo T, Jung RE, et al. Group 1 ITI Consensus Report: The role of bone dimensions and soft tissue augmentation procedures on the stability of clinical, radiographic, and patient-reported outcomes of implant treatment. *Clin Oral Implants Res.* 2023;34 Suppl 26:43-49.
129. Chu SJ, Kan JY, Lee EA, et al. Restorative Emergence Profile for Single-Tooth Implants in Healthy Periodontal Patients: Clinical Guidelines and Decision-Making Strategies. *Int J Periodontics Restorative Dent.* 2019;40(1):19-29.
130. Gomez-Meda R, Esquivel J, Blatz MB. The esthetic biological contour concept for implant restoration emergence profile design. *J Esthet Restor Dent.* 2021;33(1):173-184.
131. Siegenthaler M, Strauss FJ, Gamper F, Hämmerle CHF, Jung RE, Thoma DS. Anterior implant restorations with a convex emergence profile increase the frequency of recession: 12-month results of a randomized controlled clinical trial. *J Clin Periodontol.* 2022;49(11):1145-1157.
132. Steigmann M, Monje A, Chan HL, Wang HL. Emergence profile design based on implant position in the esthetic zone. *Int J Periodontics Restorative Dent.* 2014;34(4):559-563.



## Strategies to Improve Peri-Implant Soft Tissue Integration: Micro to Nanoengineered Surface Modifications

Sae Mi Bok, BDS

School of Dentistry, The University of Queensland, Queensland, Australia

### Introduction

Dental implants have revolutionized modern dentistry since the introduction of the first Brånemark implant in 1965, becoming a preferred treatment modality for replacing missing dentition to restore function, aesthetics and quality of life. The biological process of osseointegration has been extensively studied to enhance the implant survival. However, despite its critical role in the long-term success of implants, soft tissue integration has been relatively overlooked. With the rise in implant-related complications, such as peri-implant diseases, and increasing patient expectations for aesthetic outcomes, there is growing emphasis on improvement of peri-implant soft tissue integrity.

This narrative review briefly explores the current understanding of the anatomical features of peri-implant soft tissues and discuss various strategies to improve peri-implant soft tissue integration.

### Structure of peri-implant soft tissue

Peri-implant tissues share several anatomical and histological similarities with periodontium, but there are notable differences. The peri-implant mucosal apparatus comprises keratinised oral epithelium, sulcus epithelium and transmucosal connective tissue, which are comparable to those found in the natural periodontium. However, major differences exist in the connective tissue arrangement and its associated structures (e.g. vascular network and nerve innervation), attributing to somewhat different inflammatory responses in disease.

The outer surface of peri-implant mucosa is covered by keratinised oral epithelium, which is joined to non-keratinised barrier epithelium in a marginal border. The barrier epithelium facing towards the implant abutment surface creates a tight seal against bacteria and/or external stimuli and continues to line with connective tissue below. The barrier epithelium and connective tissue are termed transmucosal attachment, measuring from the buccal aspect of mucosal margin to the crestal bone (1).

### Abstract:

The long-term success of dental implants depends on the effective integration of both hard and soft tissues. The biological process of osseointegration has been extensively studied, however peri-implant soft tissue integration has only recently gained attention. Peri-implant tissues share several structural similarities with the natural periodontium but fundamentally differ as they lack cementum, periodontal ligaments, and alveolar bone proper. The collagen fiber structure in peri-implant tissue is organised parallel to the long axis of the implant, forming a cuff-like dense connective tissue around it with reduced vascularity and cellularity. These characteristics may lead to rapid disease progression when peri-implant disease occurs. To address inherent limitations of peri-implant tissues, implant surface improvements at the micro- to nano-scale have been extensively investigated. Nano-engineered titanium implants offer several advantages over conventional surfaces, including improved rate of osseointegration and quality of peri-implant soft tissue integration. Furthermore, surface nano-modifications could enhance both antibacterial and immunomodulatory effects, thereby promoting better healing and long-term maintenance. However, the current literature on nano-surface implants remains limited, and their clinical applications have not been fully explored.

The barrier epithelium, resembling the junctional epithelium of natural teeth, is approximately 2mm in length and 100µm in thickness. It tapers a few cell layers towards the apical end and attaches to connective tissue via basal lamina and hemidesmosomes (2). The mucopolysaccharides in the basal lamina in contact with the implant, help to protect the epithelial layer from trauma or mechanical stress on the basal lamina, promoting a stable integration with surrounding tissues (2). This layer also contains various immune cells, such as immunoglobulins, neutrophils, lymphocytes and plasma cells within wide intercellular space with a fewer desmosome. This feature allows significantly faster cellular turnover (4-6 days) compared to oral epithelium (6-12days) (3).

Connective tissue ranges between 1.0 and 1.5mm in height, with collagen fibres originating at crestal bone level and projecting cuff-like orientation from the periosteum to the gingival margin (4). In contrast, collagen fibres in natural teeth are oriented perpendicular to the long axis of the tooth and in various directions, creating a fan-like arrangement between gingiva and bone, as well as among adjacent teeth. These multi-directional orientations of collagen bundles provide an adequate soft tissue support and integrity around natural teeth, facilitating resistance to physiological and pathological challenges. On the other hand, the orientation of collagen fibres around peri-implant tissue is less dynamic and more focused on adaptation rather than insertion, which may be associated with increase the risk of disease initiation and progression (5).

The histologic and morphometrical examination of connective tissue around the implant in dogs identified two distinct connective tissue layers: (i) Zone A, 40µm wide, central in direct contact with implant fixture, (ii) Zone B, 160 µm wide, lateral to zone A, not in contact with the implant. Zone A contained high volume of fibroblasts (28.12% by volume) intermingled with collagen fibres without blood vessels, while zone B had fewer fibroblasts (11.59% by volume) and more collagen and blood. The study concluded dense fibroblast layer was essential to create a tight seal between the implant and mucosa (6).

The extracellular matrix protein of keratinised gingival tissue provides the structural stability against proteolytic degradation and the bacterial penetration. Immunohistochemical analysis comparing peri-implant tissue and natural periodontium in the presence of inflammation revealed a similar distribution of collagen types (I, III, IV, and VII) and fibronectin in both groups. However, peri-implant gingival tissue presented significantly higher collagen type V. Increased collagen type V

is typically observed during the early stage of wound healing, playing a key role in soft tissue remodelling and organisation and stability of newly formed tissue (7). Nonetheless, it is important to note that the study had a limited sample size, and the structural differences were not consistently observed across different studies.

## Vascularity of peri-implant tissue

Berglundh et al. (1994) compared the vascularity of peri-implant tissue with that of natural periodontium in animals (8). In natural teeth, the supracrestal connective tissue attachment is richly vascularized through the PDL vascular plexus and suprapariosteal blood vessels adjacent to the alveolar process. These vascular structures supply the connective tissue in multiple areas: laterally to the root cementum, coronally to the crestal bone, and apically to the epithelium. The suprapariosteal blood vessels vary in diameter (ranging from >7µm to 10-40µm) and are densely packed near the marginal gingiva, forming a crevicular plexus. In contrast, the vascularization of the periodontal ligament close to the cementum is generally less pronounced, with a thinner network of vessels. The subepithelial oral plexus, a terminal extension of the suprapariosteal vessels, is found beneath the oral epithelium.

Conversely, in peri-implant tissues, the vascular supply to transmucosal attachment is limited to the terminal branch of suprapariosteal blood vessels outside of the alveolar ridge, due to the absence of periodontal ligament. While a crevicular plexus is present, the vascular patterns in the transmucosal connective tissue vary with the proximity of implant contact. Notably, almost no capillary vessels were observed in the central portion (300-500µm wide from the implant), while the greater blood vessels were observed laterally, extending toward the suprapariosteal arterioles.

## Role of peri-implant tissue and limitations

The peri-implant soft tissue plays a crucial role in ensuring the stability of dental implants. It acts as a protective barrier against external factors such as masticatory forces and oral bacteria (9). This tissue is vital for transporting essential nutrients and oxygen from blood vessels to the associated structures surrounding the implant, which is critical for the implant's survival and successful integration with adjacent bone and tissues (10). Peri-implant tissue also contributes



to the structural integrity necessary for maintaining the aesthetics of implant prosthesis, allowing for a natural appearance and a seamless transition between the implant and surrounding tissues. Thus, the peri-implant soft tissue provides essential protection, support, and aesthetic value to dental implant.

Nevertheless, when inflammation is present, the peri-implant soft tissue is generally more susceptible to biological complications than the periodontium surrounding natural teeth (5). This increased vulnerability is associated with several factors, such as limited blood supply, reduced cellularity, and less diverse fibre orientation. These characteristics can compromise the health and stability of the tissue, increasing the risk of complications around dental implants.

## Failure of peri-implant tissue integrity

Peri-implant tissues may exhibit compromised integrity, displaying unique features compared to periodontitis. Peri-implantitis lesions show distinct cellular and histopathologic characteristics. In experimental animal models induced by ligature, inflammatory infiltrates were found to be larger, with a higher number of osteoclasts, and these lesions extended closer to the crestal bone than those found in periodontitis (11). Clinical studies have confirmed these findings, revealing that peri-implantitis lesions are twice as extensive as those seen in patients with severe chronic periodontitis. These lesions show significantly higher numbers and densities of CD138 (plasma cell markers), CD68 (macrophages markers), and MPO-positive cells (neutrophils markers) compared to periodontitis lesions (12). Furthermore, peri-implantitis lesions often extend beyond the junctional epithelium and are surrounded by a denser network of vascular structures in the connective tissue adjacent to the infiltrated areas, whereas periodontitis lesions are surrounded by non-infiltrated connective tissue (12).

The integrity of peri-implant tissue is critical for the long-term success and durability of dental implants. The peri-implant mucosa seal, essential for maintaining implant health, is constantly threatened by microorganisms that form biofilms on the implant surface (13).

Despite the increase in complications related to peri-implantitis, there is currently no universally accepted management strategy (14). Existing treatment approaches, often adapted from those for periodontal disease, may be less effective due to the unique characteristics of peri-implant tissues. Therefore, enhancing the quality and integration

of peri-implant soft tissues – potentially through different implant surface designs that mimic the natural connective tissue around teeth – could be an effective strategy to minimise the risk of biological complications.

## Surface modifications influencing soft tissue integrity

### Acid-etched surface

The Sandblasted, Large-grit, Acid-etched (SLA) surface was primarily developed in the mid-1990s and has proven highly effective in enhancing osseointegration. This modification involves first sandblasting the implant with 200µm grit, followed by acid etching with HCl and H<sub>2</sub>SO<sub>4</sub> to create 20µm cavities, resulting in a multi-level rough surface (15). While primarily aimed at improving osseointegration, several pre-clinical studies revealed interesting effects of SLA surfaces on the quality and quantity of peri-implant soft tissues.

Glauser et al. (2005) performed a microscopic and histometric analysis comparing barrier epithelium and connective tissue in one-piece mini-implants with different surfaces in humans (16). Implants with oxidized, acid-etched, or machined surfaces underwent transmucosal healing for 8 weeks. The study found that smooth implant surfaces had a barrier epithelium length of 2.9 mm, whereas those with oxidized or acid-etched surfaces had shorter epithelium, ranging from 1.4 to 1.6 mm. This suggested that oxidized and acid-etched surfaces were associated with reduced apical migration of the epithelium and a more extensive connective tissue seal compared to smooth machined implants. The authors speculated that the reduced height of junctional epithelium at rough surfaces might facilitate better connective tissue adhesion during healing process, thereby limiting epithelial migration.

In contrast, an animal study by Abrahamsson et al. (2002) found no significant quantitative or qualitative differences in soft tissue healing between acid-etched and turned surface abutments in beagle dogs. The study concluded that the roughness of titanium did not notably affect the dimension or orientation of soft tissue around the implants (17). Another pre-clinical animal study, comparing three different implant designs – commercially pure titanium, titanium plasma-sprayed, and sand blasted acid-etched surfaces – after 1.5 years of function showed no significant differences in the conditions of the peri-implant soft tissues. The design and surface modifications did not influence plaque accumulation or the development of peri-implant mucositis (18).

## Hydrophilicity

Hydrophilicity is a surface characteristic that promotes rapid protein adsorption, which accelerates osseointegration and significantly impacts the integration of soft tissue around dental implants (19). In animal study, it was found that collagen fibres formed perpendicular to the hydrophilic implant surface (SLActive) with a high density of blood vessels within the newly formed loose connective tissue. In contrast, the hydrophobic surface (SLA), exhibited collagen fibres aligned parallel to the long axis of implant with a relatively lower level of angiogenesis compared to the SLActive surface (20). These preliminary findings suggest that a hydrophilicity of implant surface may promote perpendicular connective tissue attachment at transmucosal level. Further exploration of surface hydrophilicity's effect on transmucosal healing involving 30 patients (21). Three types of abutment surfaces were compared - hydrophobic machined, chemically modified hydrophilic acid-etched titanium, and chemically modified hydrophilic acid-etched titanium-zirconium alloy. Histological analysis at 8 weeks of healing showed that the hydrophilic acid-etched surface achieved superior epithelial and connective tissue contact compared to the hydrophobic surfaces. This highlights the effectiveness of hydrophilic surfaces in enhancing tissue integration and improving overall soft tissue healing around implants.

## Laser-modified microgroove abutments

Laser-micro-grooved surface modification, produced by laser ablation, has been shown to promote soft tissue integration by promoting perpendicular connective tissue attachment at transmucosal level in multiple preclinical and clinical studies (22-25). *In vitro* and animal studies support these findings, indicating that the micro-grooved surface facilitates the proliferation of osteoblasts and fibroblasts towards the grooves on the cervical portion of abutments (22, 23). This surface modification is believed to create a direct and stable connection between the implant abutment and the connective tissue, inhibiting apical epithelial migration and preventing bone resorption. As a result, bone remodelling tends to occur in a coronal direction, which reduces epithelial attachment and improves connective tissue integration. However, these findings should be interpreted with caution as they have not yet been fully validated.

The Laser-Lok abutment is a commercially available example of laser-micro-grooved surface modification. A recent longitudinal randomized clinical trial showed a significant improvement in clinical parameters, including reductions in plaque and bleeding scores, peri-implant sulcus depth and

the mean crestal bone loss (24). The test group with laser-microgroove abutments showed a 0.15mm bone gain over three years of function. These results were consistent with previous study by Guarnieri et al. (2021), which also reported decreased plaque and bleeding scores, along with a reduction in inflammatory infiltrate due to the soft tissue seal formed at the laser-microgroove sites (25). In contrast, the control group experienced greater crestal bone loss, likely due to lack of functional fibre attachment to the machined transmucosal portion. It may have allowed bacteria from the sulcus to infiltrate micro gaps, leading to increased plaque-induced inflammation. However, despite these promising results, the evidence is limited by small sample sizes and case reports. To fully understand the benefits and potential limitations of laser-micro-grooved abutments, further research with larger sample sizes and additional prospective studies is warranted.

## Abutment materials

Abrahamsson et al. (1998) investigated the impact of various abutment materials on the quality of peri-implant mucosa using a canine model. The materials compared included pure titanium, highly sintered aluminium-based ceramic (aluminium oxide), gold alloy, and dental porcelain fused to gold (26). The study found that both pure titanium and aluminium-based ceramic led to superior healing and normal peri-implant formation. In contrast, gold alloy and dental porcelain resulted in compromised tissue healing, leading to the apical migration of barrier epithelium, recession of soft tissue, and bone resorption. These outcomes were consistent with those of Welanders et al. (2008), who also observed inferior healing with gold alloy, characterised by higher levels of inflammatory cells and fewer collagen fibres and fibroblasts in the connective tissue (27).

In subsequent research, the preclinical study by Abrahamsson et al. (2007) demonstrated that the dimensions of implant soft tissue were not significantly influenced by either pure titanium or gold (28). Similarly, no clinical differences were found in peri-implant crestal bone levels or healing outcomes between these materials (29).

Recent *in vivo* study in rats have further explained the advantages of abutment materials. Titanium and zirconia exhibited superior cell adhesion properties as compared to platinum-gold. These materials expressed laminin-5 immunoreactivity not only at the apical portion of the epithelium but also at the cervical region. Additionally, a greater number of gingival epithelial-like cells (Sa3) and fibroblastic cells (NIH3T3) were observed on titanium and zirconia surfaces, indicating their favourable interactions with soft tissue (30).





## Repeated abutment removal

The impact of repeated abutment removal on peri-implant tissues has been explored in both experimental and clinical studies. In a study by Abrahamsson (1997), an experimental dog model was used to assess the effect of removing and reattaching abutments on marginal peri-implant tissues. During a plaque control phase, abutments at test sites were removed, cleaned, and reattached five times, while the contralateral abutments were left undisturbed for six months as control sites. Histological analysis revealed that repeated abutment manipulation compromised mucosal barrier, as indicated by apical migration of connective tissue and marginal bone resorption. This disruption suggested changes in the biological width and the formation of a new biological interface (31).

More recently, a multicentre randomized controlled trial investigated the impact of repeated abutment removal on hard and soft tissue changes over a three-year period following implant loading (32). This study involved 80 patients who required either single crowns or fixed partial prostheses supported by up to three implants. The transmucosal healing abutments were removed at least three times for various procedures including impression taking, checking the zirconium core on titanium abutments for single crowns or fitting the metal structure for multi-implant prostheses, and final prosthesis delivery. The findings indicated an average bone loss of 0.43 mm associated with repeated abutment changes. However, the clinical significance of this bone loss remains debatable.

## Nanoengineered implant surface

### Development of nanoengineered implant surface

An ideal surface modification for implants should enhance epithelial and fibroblast cell functionality, improving their adhesion to the implant surface while regulating the inflammatory response to accelerate healing and minimize bacterial adhesion and colonisation. Despite advancements in increasing surface microroughness and various surface modifications to improve tissue integration, implant failures continue to occur (33). To address these challenges, there is a growing emphasis on nanoscale modifications of implant surfaces.

Nanotechnology involves the design, synthesis, characterization, and application of materials and devices with at least one dimension is at the nanometre scale or one billionth of a meter (34). Nano-engineered titanium surface can be created using both subtractive and additive methods. Subtractive methods include micro-machining,

chemical etching, polishing, grinding, particle blasting, and electrochemical anodization. Additive methods encompass techniques such as titanium plasma spraying, ion deposition and the application of hydroxyapatite and calcium phosphate coating (35, 36). Additionally, the fabrication of nano-engineering implant can be modified through mechanical, chemical and electrochemical process (37). Among these, electrochemical anodization has been extensively studied for its cost-effectiveness and versatility in creating nanostructures on metal surfaces like titanium. This process involves immersing a metal electrode or implant (anode) in an electrolyte solution containing water and fluoride ions, along with a counter metal electrode (cathode). An optimal voltage and current are applied to promote the self-organisation of various metal-oxide nanostructures, such as nanotubes and nanopores, on the surface of the implant (38, 39). Nanotubes, resembling test tubes with an open top and a closed bottom, may experience strain and failure due to the inter-tube gaps, whereas nanopores, characterised by fused tubes, offer a pore-like appearance (40). Nano-engineered surfaces can be structured either in aligned or random fashion, depending on the number of electrochemical anodization process employed. Removal of the anodic film can create nano-templates, leading to dual micro-nano structures that enhance mechanical stability (38, 41). Further surface treatments, such as alkali-heat processing, can produce a range of nano-topographies, including nanospikes and nano-grass, which can be customised to influence epithelial cells and fibroblasts activity (42). Generally, smaller nanopores (50nm) exhibit greater resistance against bending and fracture compared to larger nanopores (70nm) and traditional nanotubes (38). This increased resistance is attributed to the higher density and reduced depth of smaller nanopores on the implant threads (41).

### Effects of nanoengineered implant surface

Nano-engineered implants have positive impacts on cellular activity by promoting blood coagulation, protein adhesion, and improving osteoblast attachment and alignment during the early stages of the healing process (43-45). These findings are consistent with the results of other studies by Park et al. (2009) and Oh et al. (2009) indicated that the maximum activity occurred at a diameter of 15nm (46, 47). Smaller nanotubes (30nm) stimulate higher adhesion and growth of mesenchymal stem cells compared to larger nanotubes (70-100nm). The bioactivity of these nano-surfaces can be further enhanced by loading various nanoparticles, ions, and coatings, such as BMP-2, PDGF-BB, strontium,

tantalum, lanthanum, and zinc, which collectively support early bone formation (45-50). However, further research is required as some additional ions and coatings may lead to immunotoxicity in high doses (40).

Recent *in vitro* studies have observed that human gingival fibroblasts proliferate early and aligned parallel to dual-micro-nano titanium oxide nanopores as soon as day 1 of cell culture. While macrophages were randomly distributed across the nanopore surface, osteoblasts and fibroblasts exhibit parallel alignment in the direction of the nanopores (51). These nanopores increased the gene expression of type I and III collagen, as well as integrin- $\beta$ 6 which are essential in wound healing and improving cell interactions (52). Activated fibroblasts enhance the production of TGF- $\beta$ 1 and type IV collagen in the peri-implant epithelium, stimulating laminin- $\beta$ 3 expression, and strengthens epithelial sealing activity (53). Epithelial attachment to anodized titanium implants is significantly higher at 90.16% compared to non-anodized implants, which show only 3.62% attachment after 8 weeks. Soft tissue frequently infiltrates the nanopores of titanium implants, suggesting potential soft tissue integration at the transmucosal region (54). In addition, hydrothermal treatment of nanotubes has been shown to upregulate the expression of key molecules involved in soft tissue integration. This included integrin  $\alpha$ -5 and  $\beta$ -4 in gingival epithelial cells, as well as adhesion kinase and murine fibroblast-like NIH/3T3 cells (55, 56).

Nano-modified implant surfaces also demonstrate promising therapeutic effects in drug delivery. Pre-clinical studies show that drugs such as bisphosphonate, aspirin, and vitamin C were more effectively released on larger nanotubes (100nm) (57-59). The use of polymeric micelles enhances the simultaneous delivery of multiple drugs. These micelles can encapsulate hydrophobic drugs on the top and hydrophilic drugs on the bottom, preventing them from mixing (58). Once the first drug is released, the inverted micelles containing the hydrophilic drugs at the bottom can be gradually released, allowing for a controlled and sustained release of the drug and effectively addressing common challenges in drug delivery, such as poor biodistribution and burst release effects.

Titania nanopores exhibit immunomodulatory functions, including reduced adhesion of macrophages, monocytes, and neutrophils, as well as decreased production of inflammatory cytokines. The voltage applied during anodization process significantly influences macrophage activity (60). For instance, anodisation at 5V improves

osteogenesis by reducing inflammation and CD 68+ macrophage distribution, promoting activity of pro-healing M2 macrophages, whereas anodization at 20V is associated with pro-inflammatory M1 macrophage. These findings are consistent across both *in vitro* and *in vivo* pre-clinical studies.

Advancements in nanostructured titanium implants enhance soft tissue healing and reduce bacterial infections. Improved healing may be linked to a reduction in reactive oxygen species produced by macrophages, thereby minimising post-surgical inflammation (61). For example, titanium oxide nanotubes with a diameter of 70nm had significantly lower nitride oxide activity than unmodified titanium, leading to reduced macrophage infiltration and better healing outcomes one week after surgery (61). Electrochemically nano-engineered titanium implants have demonstrated antimicrobial properties, effectively reducing bacterial adhesion, metabolic activity, and salivary biofilm formation (43, 62). Nanotube surfaces exhibit lower bacterial adherence than traditional micro-rough SLA surfaces and smooth surfaces (63). It could be attributed to decreased expression of bacterial adhesins and increased fibronectin protein attachment (64, 65). Thermally treated nanoscale implants with 80nm diameter nanotubes (66), as well as those with smaller nanotubes around 15nm in diameter (67), have shown enhanced antibacterial effects compared to implants with diameters of 50nm and 100nm (66, 67). This suggests that both the size and treatment of the nanotubes play a crucial role in optimizing the antibacterial properties of titanium implants.

## Conclusion

Nano-engineered implants have been shown a great potential in addressing the challenges associated with conventional implants. By incorporating nanoscale surface modifications, these implants can enhance soft tissue integration and reduce peri-implant infections by modulating the immune response and enabling targeted delivery of bioactive molecules and multi-drugs directly to the treatment site. However, their clinical application remains limited due to the novelty of nano-engineering techniques and a lack of comprehensive clinical trials.

Soft tissue integration is crucial for maintaining implant function and integrity, highlighting the need for further *in vivo* research to fully explore the potential of nano-engineered surface implants.



## Reference

1. Araujo M, Lindhe J. Peri-implant health. *J Clin Periodontol*. 2018;45(20):s230-s6.
2. Misch CE. *Implantología Contemporánea*. Madrid: Mosby; 1995.
3. Demetriou NA, Ramfjord SP. Premitotic labeling and inflammation in the gingiva of Rhesus monkeys. *J Periodontol*. 1972;43(10):606-13.
4. Berglundh T, Lindhe J, Ericsson I, Marinello CP, Liljenberg B, Thomsen P. The soft tissue barrier at implants and teeth. *Clin Oral Implants Res*. 1991;2(2):81-90.
5. Ivanovski S, Lee R. Comparison of peri-implant and periodontal marginal soft tissues in health and disease. *Periodontol 2000*. 2018;76(1):116-30.
6. Moon IS, Berglundh T, Abrahamsson I, Linder E, Lindhe J. The barrier between the keratinized mucosa and the dental implant. An experimental study in the dog. *J Clin Periodontol*. 1999;26(10):658-63.
7. Romanos GE, Schröter-Kermani C, Weingart D, Strub JR. Health human periodontal versus peri-implant gingival tissues: an immunohistochemical differentiation of the extracellular matrix. *Int J Oral Maxillofac Implants*. 1995;10(6):750-8.
8. Berglundh T, Lindhe J, Jonsson K, Ericsson I. The topography of the vascular systems in the periodontal and peri-implant tissues in the dog. *J Clin Periodontol*. 1994;21(3):189-93.
9. Salvi GE, Bosshardt DD, Lang NP, Abrahamsson I, Berglundh T, Lindhe J, et al. Temporal sequence of hard and soft tissue healing around titanium dental implants. *Periodontol 2000*. 2015;68(1):135-52.
10. Atsuta I, Ayukawa Y, Kondo R. Soft tissue sealing around dental implants based on histological interpretation. *J Prosthodont Res*. 2016;60(1):3-11.
11. Carcuac O, Abrahamsson I, Albouy JP, Linder E, Larsson L, Berglundh T. Experimental periodontitis and peri-implantitis in dogs. *Clin Oral Implants Res*. 2013;24(4):363-71.
12. Carcuac O, Berglundh T. Composition of human peri-implantitis and periodontitis lesions. *J Dent Res*. 2014;93(11):1083-8.
13. Seymour GJ, Gemmell E, Lenz LJ, Henry P, Bower R, Yamazaki K. Immunohistologic analysis of the inflammatory infiltrates associated with osseointegrated implants. *Int J Oral Maxillofac Implants*. 1989;4(3):191-8.
14. A. I, A. I, G. I, I. C, H. U. PERIIMPLANTITIS: MANAGEMENT AND PROGNOSTIC. *Romanian J Med Dent Ed*. 2022;11(2):6-10.
15. Y Z, S L, Y L, P D, Y L. Macro to Micro: Surface Modification of Titanium Dental Implants. In: Gulati, K. (eds) *Surface Modification of Titanium Dental Implants*.: Springer, Cham; 2023.
16. Glauser R, Schüpbach P, Gottlow J, Hämmerle CH. Periimplant soft tissue barrier at experimental one-piece mini-implants with different surface topography in humans: A light-microscopic overview and histometric analysis. *Clin Implant Dent Relat Res*. 2005;7 Suppl 1:S44-51.
17. Abrahamsson I, Zitzmann NU, Berglundh T, Linder E, Wennerberg A, Lindhe J. The mucosal attachment to titanium implants with different surface characteristics: an experimental study in dogs. *J Clin Periodontol*. 2002;29(5):448-55.
18. Watzak G, Zechner W, Tangl S, Vasak C, Donath K, Watzek G. Soft tissue around three different implant types after 1.5 years of functional loading without oral hygiene: a preliminary study in baboons. *Clin Oral Implants Res*. 2006;17(2):229-36.
19. El Chaar E, Zhang L, Zhou Y, Sandgren R, Fricain JC, Dard M, et al. Osseointegration of Superhydrophilic Implants Placed in Defect Grafted Bones. *Int J Oral Maxillofac Implants*. 2019;34(2):443-50.
20. Schwarz F, Herten M, Sager M, Wieland M, Dard M, Becker J. Histological and immunohistochemical analysis of initial and early subepithelial connective tissue attachment at chemically modified and conventional SLA titanium implants. A pilot study in dogs. *Clin Oral Investig*. 2007;11(3):245-55.
21. Schwarz F, Mihatovic I, Becker J, Bormann KH, Keeve PL, Friedmann A. Histological evaluation of different abutments in the posterior maxilla and mandible: an experimental study in humans. *J Clin Periodontol*. 2013;40(8):807-15.
22. Kim SY, Oh N, Lee MH, Kim SE, Leesungbok R, Lee SW. Surface microgrooves and acid etching on titanium substrata alter various cell behaviors of cultured

- human gingival fibroblasts. *Clin Oral Implants Res.* 2009;20(3):262-72.
23. Cho SA, Jung SK. A removal torque of the laser-treated titanium implants in rabbit tibia. *Biomaterials.* 2003;24(26):4859-63.
  24. Ahamed AS, Prakash PSG, Crena J, Victor DJ, Subramanian S, Appukuttan D. The influence of laser-microgrooved implant and abutment surfaces on mean crestal bone levels and peri-implant soft tissue healing: a 3-year longitudinal randomized controlled clinical trial. *Int J Implant Dent.* 2021;7(1):102.
  25. Guarnieri R, Miccoli G, Reda R, Mazzoni A, Di Nardo D, Testarelli L. Laser microgrooved vs. machined healing abutment disconnection/reconnection: a comparative clinical, radiographical and biochemical study with split-mouth design. *Int J Implant Dent.* 2021;7(1):19.
  26. Abrahamsson I, Berglundh T, Glantz PO, Lindhe J. The mucosal attachment at different abutments. An experimental study in dogs. *J Clin Periodontol.* 1998;25(9):721-7.
  27. Welander M, Abrahamsson I, Berglundh T. The mucosal barrier at implant abutments of different materials. *Clin Oral Implants Res.* 2008;19(7):635-41.
  28. Welander M, Abrahamsson I, Linder E, Liljenberg B, Berglundh T. Soft tissue healing at titanium implants coated with type I collagen. An experimental study in dogs. *J Clin Periodontol.* 2007;34(5):452-8.
  29. Vigolo P, Givani A, Majzoub Z, Cordioli G. A 4-year prospective study to assess peri-implant hard and soft tissues adjacent to titanium versus gold-alloy abutments in cemented single implant crowns. *J Prosthodont.* 2006;15(4):250-6.
  30. Furuhashi A, Ayukawa Y, Atsuta I, Rakhmatia YD, Koyano K. Soft Tissue Interface with Various Kinds of Implant Abutment Materials. *J Clin Med.* 2021;10(11).
  31. Abrahamsson I, Berglundh T, Lindhe J. The mucosal barrier following abutment dis/reconnection. An experimental study in dogs. *J Clin Periodontol.* 1997;24(8):568-72.
  32. Bressan E, Grusovin MG, D'Avenia F, Neumann K, Sbricoli L, Luongo G, et al. The influence of repeated abutment changes on peri-implant tissue stability: 3-year post-loading results from a multicentre randomised controlled trial. *Eur J Oral Implantol.* 2017;10(4):373-90.
  33. Mendonça G, Mendonça DB, Aragão FJ, Cooper LF. Advancing dental implant surface technology- from micron- to nanotopography. *Biomaterials.* 2008;29(28):3822-35.
  34. Silva GA. Introduction to nanotechnology and its applications to medicine. *Surg Neurol.* 2004;61(3):216-20.
  35. Chopra D, Gulati K, Ivanovski S. Understanding and optimizing the antibacterial functions of anodized nano-engineered titanium implants. *Acta Biomater.* 2021;127:80-101.
  36. Rupp F, Liang L, Geis-Gerstorf J, Scheideler L, Hüttig F. Surface characteristics of dental implants: A review. *Dent Mater.* 2018;34(1):40-57.
  37. del Olmo R, Czerwiński M, Santos-Coquillat A, Dubey V, Dhoble SJ, Michalska-Domańska M. Nano-scale Surface Modification of Dental Implants: Fabrication. In: Gulati K, editor. *Surface Modification of Titanium Dental Implants. Cham: Springer International Publishing; 2023. p. 83-116.*
  38. Gulati K, Li T, Ivanovski S. Consume or Conserve: Microroughness of Titanium Implants toward Fabrication of Dual Micro-Nanotopography. *ACS Biomater Sci Eng.* 2018;4(9):3125-31.
  39. Shin YC, Pang KM, Han DW, Lee KH, Ha YC, Park JW, et al. Enhanced osteogenic differentiation of human mesenchymal stem cells on Ti surfaces with electrochemical nanopattern formation. *Mater Sci Eng C Mater Biol Appl.* 2019;99:1174-81.
  40. Zhang Y, Gulati K, Li Z, Di P, Liu Y. Dental Implant Nano-Engineering: Advances, Limitations and Future Directions. *Nanomaterials* (Basel). 2021;11(10).
  41. Li T, Gulati K, Wang N, Zhang Z, Ivanovski S. Understanding and augmenting the stability of therapeutic nanotubes on anodized titanium implants. *Mater Sci Eng C Mater Biol Appl.* 2018;88:182-95.
  42. Kato E, Sakurai K, Yamada M. Periodontal-like gingival connective tissue attachment on titanium surface with nano-ordered spikes and pores created by alkali-heat treatment. *Dent Mater.* 2015;31(5):e116-30.
  43. Guo T, Gulati K, Arora H, Han P, Fournier B, Ivanovski S. Race to invade: Understanding soft tissue integration at the transmucosal region of titanium dental implants. *Dent Mater.* 2021;37(5):816-31.



44. Gulati K, Maher S, Findlay DM, Losic D. Titania nanotubes for orchestrating osteogenesis at the bone-implant interface. *Nanomedicine* (Lond). 2016;11(14):1847-64.
45. Balasundaram G, Yao C, Webster TJ. TiO<sub>2</sub> nanotubes functionalized with regions of bone morphogenetic protein-2 increases osteoblast adhesion. *J Biomed Mater Res*. 2008;84:447-53.
46. Zhang W, Jin Y, Qian S, Li J, Chang Q, Ye D, et al. Vacuum extraction enhances rhPDGF-BB immobilization on nanotubes to improve implant osseointegration in ovariectomized rats. *Nanomedicine*. 2014;10(8):1809-18.
47. Zhao L, Wang H, Huo K, Zhang X, Wang W, Zhang Y, et al. The osteogenic activity of strontium loaded titania nanotube arrays on titanium substrates. *Biomaterials*. 2013;34(1):19-29.
48. Frandsen CJ, Brammer KS, Noh K, Johnston G, Jin S. Tantalum coating on TiO<sub>2</sub> nanotubes induces superior rate of matrix mineralization and osteofunctionality in human osteoblasts. *Mater Sci Eng C Mater Biol Appl*. 2014;37:332-41.
49. Zhang X, Zhang X, Wang B, Lan J, Yang H, Wang Z, et al. Synergistic effects of lanthanum and strontium to enhance the osteogenic activity of TiO<sub>2</sub> nanotube biological interface. *Ceramics International*. 2020;46(9):13969-79.
50. Huo K, Zhang X, Wang H, Zhao L, Liu X, Chu PK. Osteogenic activity and antibacterial effects on titanium surfaces modified with Zn-incorporated nanotube arrays. *Biomaterials*. 2013;34(13):3467-78.
51. Gulati K, Moon HJ, Li T, Sudheesh Kumar PT, Ivanovski S. Titania nanopores with dual micro-/nano-topography for selective cellular bioactivity. *Mater Sci Eng C Mater Biol Appl*. 2018;91:624-30.
52. Gulati K, Moon HJ, Kumar PTS, Han P, Ivanovski S. Anodized anisotropic titanium surfaces for enhanced guidance of gingival fibroblasts. *Mater Sci Eng C Mater Biol Appl*. 2020;112:110860.
53. Tan J, Zhao C, Zhou J, Duan K, Wang J, Lu X, et al. Co-culturing epidermal keratinocytes and dermal fibroblasts on nano-structured titanium surfaces. *Mater Sci Eng C Mater Biol Appl*. 2017;78:288-95.
54. Chen GJ, Wang Z, Bai H, Li JM, Cai H. A preliminary study on investigating the attachment of soft tissue onto micro-arc oxidized titanium alloy implants. *Biomed Mater*. 2009;4(1):015017.
55. Miura S, Takebe J. Biological behavior of fibroblast-like cells cultured on anodized-hydrothermally treated titanium with a nanotopographic surface structure. *J Prosthodont Res*. 2012;56(3):178-86.
56. Miyata K, Takebe J. Anodized-hydrothermally treated titanium with a nanotopographic surface structure regulates integrin- $\alpha 6\beta 4$  and laminin-5 gene expression in adherent murine gingival epithelial cells. *J Prosthodont Res*. 2013;57(2):99-108.
57. Kwon DH, Lee SJ, Wikesjö UME, Johansson PH, Johansson CB, Sul YT. Bone tissue response following local drug delivery of bisphosphonate through titanium oxide nanotube implants in a rabbit model. *J Clin Periodontol*. 2017;44(9):941-9.
58. Wang Q, Huang JY, Li HQ, Zhao AZ, Wang Y, Zhang KQ, et al. Recent advances on smart TiO<sub>2</sub> nanotube platforms for sustainable drug delivery applications. *Int J Nanomedicine*. 2017;12:151-65.
59. Yang W, Deng C, Liu P, Hu Y, Luo Z, Cai K. Sustained release of aspirin and vitamin C from titanium nanotubes: An experimental and stimulation study. *Mater Sci Eng C Mater Biol Appl*. 2016;64:139-47.
60. Ma QL, Zhao LZ, Liu RR, Jin BQ, Song W, Wang Y, et al. Improved implant osseointegration of a nanostructured titanium surface via mediation of macrophage polarization. *Biomaterials*. 2014;35(37):9853-67.
61. Smith GC, Chamberlain L, Faxius L, Johnston GW, Jin S, Bjursten LM. Soft tissue response to titanium dioxide nanotube modified implants. *Acta Biomater*. 2011;7(8):3209-15.
62. Jayasree A, Ivanovski S, Gulati K. ON or OFF: Triggered therapies from anodized nano-engineered titanium implants. *J Control Release*. 2021;333:521-35.
63. Miao X, Wang D, Xu L, Wang J, Zeng D, Lin S, et al. The response of human osteoblasts, epithelial cells, fibroblasts, macrophages and oral bacteria to nanostructured titanium surfaces: a systematic study. *Int J Nanomedicine*. 2017;12:1415-30.



64. Rizzello L, Cingolani R, Pompa PP. Nanotechnology tools for antibacterial materials. *Nanomedicine* (Lond). 2013;8(5):807-21.
65. Puckett SD, Taylor E, Raimondo T, Webster TJ. The relationship between the nanostructure of titanium surfaces and bacterial attachment. *Biomaterials*. 2010;31(4):706-13.
66. Ercan B, Taylor E, Alpaslan E, Webster TJ. Diameter of titanium nanotubes influences anti-bacterial efficacy. *Nanotechnology*. 2011;22(29):295102.
67. Narendrakumar K, Kulkarni M, Addison O, Mazare A, Junkar I, Schmuki P, et al. Adherence of oral streptococci to nanostructured titanium surfaces. *Dent Mater*. 2015;31(12):1460-8.



## Socket Shield – is it just the Latest Trend or a Revolutionary Technique for Maintaining the Buccal Bone?

Alana Smith, BDS, DCLinDent

School of Dentistry, The University of Melbourne, Victoria, Australia

### Introduction

Extraction of a tooth triggers a cascade of physiological processes that results in the loss of the bundle bone, being a tooth-dependent structure, which modifies the alveolar ridge dimensions (1-3). Rehabilitation of the edentulous space with an implant-supported prosthesis that achieves biological, functional and aesthetic success may be compromised by insufficient hard and soft tissue. Current concepts of guided bone regeneration (GBR) to minimise the buccal wall loss have not been successful in preventing the resorption of the bundle bone and are associated with a certain degree of ridge collapse (4). The socket-shield is a type of partial extraction therapy first introduced by Hürzeler in 2010, which maintains the buccal part of a tooth root within the socket, with simultaneous immediate implant placement palatally, which preserves the periodontal attachment and blood supply to the facial bundle bone. Consequently, bone remodelling following extraction is not triggered and ridge architecture is preserved (5). The literature consists mainly of small cohort case studies of limited follow up periods, typically performed by a single clinician, and a few larger retrospective studies. These studies are largely heterogenous in the socket-shield protocol and outcomes measured making it difficult to draw high quality evidence based conclusions. The purpose of this discussion is to introduce the concept of the socket-shield and establish what is currently reported about its efficacy in maintaining the buccal bone, the safety of the technique with regard to biological and implant-related complications long term and what questions are yet to be answered, in helping clinicians decide where they stand on introducing the procedure into their practice.

### Buccal bone changes following extraction

The bundle bone constitutes the inner lining of the socket which forms from the ectomesenchyme alongside root formation during development. It provides anchorage for the Sharpey's fibres of the periodontal ligament, making

### Abstract:

**Aim:** Extraction of a tooth results in the resorption of the tooth-dependent bundle bone and changes in the alveolar ridge dimension which can compromise biologic and aesthetic outcomes with implant-supported prostheses. The introduction of the socket-shield technique, which maintains the buccal fragment of the tooth root to preserve the periodontium and the buccal bone, has been combined with immediate implant placement to deliver promising outcomes. The purpose of this discussion is to gain more insight into the socket-shield protocol evolution, the efficacy of the technique in maintaining the buccal wall, the safety of the technique with regard to biologic and implant-related complications long term and what questions are yet to be answered before the procedure can be recommended to clinicians.

**Methods:** The search resulted in a selection of publications on the socket-shield technique since the first publication by Hürzeler in 2010 that described principles behind the concept and treatment protocols, assessed clinical outcomes including implant survival, buccal bone preservation, aesthetic outcomes and complications.

**Results:** Analysis of available dental literature showed various updates to the initial socket-shield protocol to address the incidence of complications. Studies were mostly case reports with a selection of larger retrospective studies, and a small number of randomised controlled trials. The socket-shield technique was shown to successfully preserve the buccal wall and achieve excellent aesthetic results, however due to the lack of long-term data on the potential complications which may result in compromised biologic and aesthetic outcomes, the procedure cannot yet be considered predictable.

it an essential part of the attachment apparatus (6). It is a tooth-dependent structure that receives blood supply largely from the periodontal ligament. When a tooth is lost and blood supply disrupted, physiological changes occur that lead to osteoclastic resorption of the bundle bone (7). In an animal model, bundle bone was absent at four weeks following extraction of a posterior tooth. Where the crest of the buccal bone was thinner and consisted solely of bundle bone, a substantial vertical and horizontal reduction in ridge dimension was observed (2). This pattern was consistent in the anterior maxillary sites of humans where thin facial wall phenotypes less than 1mm showed a 62% vertical bone loss. Comparatively, only 9% vertical loss was observed in thick wall phenotypes (3). Although full maintenance of the facial bone wall has been observed following immediate implant placement in animal extraction sockets with a buccal wall thickness of 2mm (8), sites in the anterior maxilla of humans have shown to be less than 1mm thickness in 90% of cases, and less than 0.5mm in approximately 50% of cases (9-11). This suggests the majority of patients are at high risk of post-extraction resorption of the thin buccal bone wall in the aesthetic zone. Other factors affecting the amount of buccal bone resorption includes thin gingival phenotypes, positioning of implants (12), and multiple adjacent extractions. Immediate implant placement and GBR do not prevent the resorption of bundle bone. The dimensional reductions in bone volume and formation of dehiscence defects in the facial bone may adversely affect the subsequent placement of dental implants, as well as aesthetic and clinical outcomes (13).

## Proof of concept

The concept behind the socket-shield technique is that when a tooth is indicated for extraction and replacement with an implant, that would otherwise be associated with collapse of the buccal bone, the buccal portion of the root is instead retained. This avoids the destruction of Sharpey's fibres inserting into the bundle bone, preserving the periodontal apparatus, including the blood supply, and therefore prevents the facial alveolar ridge from resorption (5). Retaining the roots of unrestorable teeth to avoid tissue alterations after tooth extraction is not a completely new concept. The technique was adopted from *Dent Traumatol* which recommended the decoronation of ankylosed teeth (14) to eventually undergo a replacement-resorptive process, or enabling better bone volume conditions for later

## Abstract: (continued)

Further well-designed longitudinal studies supporting the safety and biological compatibility of the socket-shield are required before the procedure can be recommended.

removal and implant placement (15). Retained roots have also been used in prosthodontics to improve the retention and stability of overdentures (16). Salama described the root submergence technique which involves the burial of a tooth root in the pontic site, which in turn allows for complete preservation of the alveolar bone frame and assists in creation of an aesthetic result, particularly in adjacent multiple tooth replacement cases (17).

A German group led by Hurzeler published the first proof-of-principle experiment in 2010 to histologically assess and clinically demonstrate the effect of the socket-shield technique in combination with immediate implant placement. In a beagle dog the buccal root fragment was retained approximately 1mm coronal to the buccal bone plate, a titanium implant with a healing abutment was placed lingual to the fragment with or without contacting it. Enamel matrix derivative was also applied to the buccal root-fragment to encourage cementum formation. At four months analysis confirmed that all implants placed were osseointegrated with an absence of any histologic inflammatory reaction or resorptive processes of the tooth fragment. Buccally to the socket-shield the periodontal ligament was maintained preserving the facial bone plate and lingually newly formed cementum was detected. In areas where the implant was placed into the fragment, newly formed cementum was identified directly on the surface of the implant, while the gaps between the socket shield and the implant were filled with new bone formation (5). These findings were confirmed in a similar canine model by Baumer where new bone was visible between the gap with no interference of implant osseointegration, even when the socket-shield was split vertically into two fragments to resemble a vertical root fracture (18).

Human histological evidence was reported upon in a case of an unplanned socket-shield, whereby a fragment of root was retained next to an implant that presented clinical and radiographic signs of peri-implantitis. The patient elected for implant removal enabling histological analysis. Bone filled



each thread space intimately interfacing the implant and dentine. It was verified to be mature, vital bone, organised in concentric lamellae, containing osteocytes with lacunae. Failure of the socket-shield was a result of the extension of the root fragment beyond the bone crest that lead to its exposure and contribution to peri-implantitis (19). These findings were able to demonstrate that socket-shield does not obstruct the passage of peri-vascular pluripotent cells and trabecular bone-lining mesenchymal cells from reaching the implant surface.

## Case Selection

Successful outcomes with the socket-shield technique begin with comprehensive assessment and appropriate case selection as outlined in Table 1 (20-22).

## Protocol

Hurzeler's original protocol has seen a variety of updates since it was first published in 2010, due to the incidence of complications (5). Preparation of the socket-shield had initially instructed the implant osteotomy to be prepared through the tooth creating a thin buccal root fragment, and the implant was often placed directly up against the socket shield. However, the root position does not always coincide

with the ideal implant position, particularly in retroclined teeth, and there would be a high risk of perforating the facial bone. The thin socket-shield was also at risk of fracture and loosening during the vibration caused by the preparation and implant placement. The socket-shield was initially prepared to 1mm above the crestal bone with the intension to preserve the supra-crestal fibres and papillae. However, the vertical height provided limited prosthetic space and resulted in internal exposure of the socket seal. Enamel matrix was also applied to promote cementum formation on the lingual surface of the socket shield and no grafting used between the gap between socket-shield and implant surface. There is currently no consensus on the benefits of additional use of grafting materials with the socket shield technique.

Gluckman and colleagues reviewed socket-shield outcomes in the previous 10 years and their 2020 publication provides an updated step-by-step protocol to mitigate the above limitations (22). The steps include (1) decoronation and removal of any endodontic posts, (2) canal preparation and measurement of root length, (3) mesio-distal sectioning of the root, (4) removal of palatal/lingual portion of the root, (5) coronal preparation of buccal portion of the root, (6) osteotomy and implant placement, (7) management of the gap with or without grafting, and (8) management of the gingival seal to protect the implant, socket shield, graft and

**Table 1.**

### Indications

- Maxillary anterior teeth in the aesthetic zone, can also be performed in the posterior region
- Unrestorable tooth crown or tooth indicated for extraction
- Tooth root with or without apical pathology – excluding the contraindicated/relative contraindicated apical pathology considerations below
- Immediate implant placement, can also be performed as delayed implant placement with the Glocker protocol modification (23)
- High risk of buccal bone wall collapse – thin buccal bone phenotype assessed using cone beam computed tomography (CBCT), thin gingival phenotype, multiple adjacent extractions

### Contraindications

- Tooth root with apical pathology if the apical site is inaccessible to instrumentation to adequately remove it
- Infection or pathology that involves the buccal root fragment to be retained e.g. horizontal or vertical root fractures through the buccal portion of the root
- Extensive facial bone dehiscence
- Mobility or widening of the periodontal ligament space that would compromise stability of the buccal fragment
- Lack of clinician skill/experience/training in surgical procedures including immediate implant placement

### Relative contraindications

- Tooth root with apical pathology if the extent of the defect is such that primary stability cannot be obtained with immediate implant placement, instead a delayed implant placement approach is indicated
- Deep residual periodontal bony defects
- Apico-facial bone fenestration

blood clot. Readers are recommended to refer to the original publication for specific protocol details.

## Variations

Glocker described a delayed socket-shield technique (23). Once the socket-shield has been prepared, reducing it to the level of the bone crest and thinned to a horizontal dimension of less than 1mm, a collagen cone is placed in the socket, left to fill with the maximum amount of bone and implant placement delayed by two to six months. The purpose was to achieve an implant position where all boundaries are formed by bone to maximise bone-implant contact principles of osseointegration (24), as the dentine-implant interface long-term behaviour had not been studied sufficiently and the possible formation of a fibrous sheath around the implant (25) was to be avoided. The delayed socket-shield technique provides an opportunity for resolution of any extensive apical pathology in scenarios where that primary stability of an immediate implant would not have been achievable.

In pontic site development, where entire root submergence is contraindicated due to the presence of periapical pathology, the socket-shield principles can also be used to preserve the bucco-palatal dimension and maintain a positive contour to create aesthetic harmony between the restoration and the alveolar ridge. The 'pontic-shield' described by Gluckman (20) involves preparation of the buccal root fragment, careful removal of the palatal component of the root and placement of adjunctive augmentation materials (particulate xenogeneic bone) within the extraction sockets. The 14 sites in the case series reportedly closed the sockets using various techniques including buccal flap advancement, connective tissue grafting, cytoplasmic membrane, socket seal and management without closure. The sites were then left to heal for a minimum of 90 days, followed by pontic site development with moderate pressure from an interim fixed partial denture before final restorations were placed. Three sites were complicated by exposure of the pontic-shield as a result of omitting soft tissue closure which led to the authors' recommendation that surgical soft tissue closure is a necessary step within the treatment protocol.

## Outcomes

The majority of publications are case reports and of the clinical studies, most are retrospective with outcomes assessed no longer than 12 months later. Therefore, little is known about

the long-term biocompatibility of the socket-shield. There are a small selection of larger trials published in 2018 including Gluckman and co-workers who reported their findings on 128 socket-shield cases with up to four years follow up, of which almost half the patients were reviewed at three years (26). The largest study to date has been Siormpas' retrospective review of 250 immediate implants with socket-shield with a mean review of over four years. However, 10 of those patients did have a follow up at 10 years, 15 patients at 9 years, and 10 patients at 8 years (27). Since the socket-shield technique was first introduced in 2010 (5), clinical studies have been largely heterogeneous in regards to the treatment protocol, as refinements are constantly introduced (20, 22, 23), making comparison between publications challenging. It should also be recognised that due to the high technique sensitivity of the socket-shield protocol it is usually performed by a single, highly-skilled and surgically experienced clinician in a single centre which introduces a certain level of performance bias (28). Clinicians should keep the limitations of these studies in mind when extrapolating the following data on treatment outcomes to routine practice.

## Implant survival

Socket-shield studies mostly evaluated the implant survival rate, according to criteria reported by Buser (29), reporting rates comparable to that of the conventional implant survival of 95-100% at 10 years (30, 31) and immediate implant survival of approximately 95% (32). Case reports of immediate implant placement using the socket shield technique with significantly smaller cohorts and shorter observation periods generally report an osseointegration rate of 100% (33-35). Lower survival rates were reported in the larger retrospective studies with Gluckman reporting 5 of their 128 cases failing to maintain osseointegration 1-4 years following restoration (survival rate 96.1%), however it was not possible to determine whether failure was a direct result of the additional socket-shield procedure. The socket-shield was retained in three of the cases, implants were replaced in two of these and the third retained as a pontic shield (26). Siormpas reported 5 implant failures out of 250 for a 10 year cumulative implant survival of 97.3% (implant-level) and 96.5% (patient-level). Two implants failed to integrate within three months of placement. The other three were removed due to presence of recurrent untreatable peri-implantitis that had become symptomatic with extensive bone loss, attributable in two cases due to exposure, mobility





and infection of the socket-shield (27). Recent systematic review of the literature up to 2021 reported implant survival rate with socket-shield to be 98.6% at a mean follow up of 18 months (21). Assuming the socket-shield is performed correctly, there should be no interference with implant osseointegration that is different from immediate implant placement. Late complications appear to be the main cause of implant failure with socket-shield.

## Preservation of buccal bone

The methods used to measure the effectiveness of buccal tissue maintenance with the socket-shield technique were heterogenous. Preservation of the alveolar ridge was analysed using either 3-dimensional surface scans (33), pre- and post-operative CBCT scans (36), or a non-specific comment on whether a 'good' outcome was achieved (5, 20, 23, 26, 34). The 3-dimensional surface scans of plaster casts taken by Baumer prior to extraction and five years post-implant placement with socket-shield showed a low degree of contour change with a mean horizontal tissue loss on the facial side of  $-0.21 \pm 0.18$  mm (33). CBCT cross sections revealed stability of the buccal bone volume in three out of the four cases with available pre- and post-operative CBCT images in a retrospective case series of 46 implants up to five years (36). In a randomised controlled trial of 42 patients, six month follow up CBCT demonstrated the socket-shield group yielded significantly less vertical and horizontal buccal bone resorption of  $0.35 \pm 0.62$  mm and  $0.29 \pm 0.34$  mm compared to conventional immediate implant therapy with xenograft placed in the buccal gap with  $1.71 \pm 1.02$  mm and  $1.45 \pm 0.72$  mm, respectively (37). Generally the socket-shield technique appears to be effective at maintaining the buccal bone dimensions in extraction sockets in the medium-term.

## Aesthetic outcomes

Bramanti used the Pink Esthetic Score (PES) (38) to demonstrate a significantly superior aesthetic outcome using the socket-shield technique  $12.15 \pm 0.94$  compared to conventional immediate implant placement  $10.3 \pm 2.53$  ( $p = .00008$ ) at three years in one of the few randomized controlled trials (35). PES evaluation also showed positive results in socket-shield cases assessed retrospectively by Baumer, with a mean score of 12 (range 11-14) at five years (33). Additional assessment of mucosal recession reported an

average of  $-0.33 \pm 0.23$  mm at the implant restoration, which was comparable to that of  $-0.38 \pm 0.27$  mm at neighbouring teeth. Similarly, Gluckman reported no recession sufficient to expose the implant-abutment interface or any blue-grey hue as a sign of implant showing through the translucent gingival tissue in any cases (26).

## Complications

The consensus across the current literature is the prerequisite that the clinician performing the socket-shield procedure possess a high level of skill and experience. The protocol is highly technique sensitive, requires the correct use of specific armamentarium (39) and is most successful when a clinician has the ability to anticipate complications and manage these appropriately. This includes understanding when to abort the socket-shield procedure, such as identification of mobility or fracture of the root fragment, and when to proceed with a conventional implant technique. A recent systematic review reported mean complication rates after a mean of 18 months to be reasonably low (3.81%) and implant survival rates high (98.6%) (21). Perhaps a more realistic complication rate is closer to the 19.5% reported by Gluckman in a larger cohort for a medium-term assessment period (26). The risks a clinician should weigh when considering performing socket-shield procedures is that the true incidence of these complications is not definitively known, these complications may arise later than reported examination time frames, the potential for failure could be catastrophic and these can be challenging to successfully manage by the clinician, requiring with further complex treatment or have a compromised final result.

## Internal and external exposure

The most frequent complication reported is internal exposure of the root fragment whereby the coronal portion of the socket-shield was found to perforate the restoration-facing soft tissue at the time of provisional restoration removal. Gluckman reported internal shield exposure in 9.4% of cases (26), thought to be caused by excessive height of the socket-shield with sharp edges in a limited prosthetic space based on the initial protocol recommendation of the vertical preparation of the socket shield to 1mm above crestal bone (5). External exposures occurred in 3.1% of sites, with the root fragment perforating the gingival tissues. Treatment of external exposures involves raising a micro-flap to reduce the

height of the socket-shield to bone level and smoothing of the sharp edges with a high speed diamond bur. For larger external exposures, an additional connective tissue graft can be included to assist with closure (39). Neither of these complications were considered difficult to manage or caused an adverse aesthetic outcome, however 2mm of tissue recession was reported at follow-up in two adjacent socket-shields that had received vertical reduction to manage internal exposure. Gluckman has indicated that a change in the protocol to reduce the socket-shield height to bone level and include a chamfer preparation in the most coronal portion provides 2-3mm of prosthetic space between the margin of the subgingival crown and the shield, providing an adequate space for soft tissue (40). Although Gluckman has reported these protocol changes have 'led to an almost complete elimination of complications related to exposure of the shield', sufficient long term observation on the updated protocol has not yet been published.

## Infection and peri-implantitis

Infection of the socket-shield with suppuration and fistula formation can result in extensive resorption of hard and soft tissues resulting in a large defect compromising peri-implant health and aesthetics. A case report of implants unintentionally placed in proximity to undetected retained root fragments (3-5 mm) developed adverse effects due to severe coronal bone loss 6-48 months post implant placement. Three out of seven implants were removed due to infection of the retained roots (41). Scanning electron microscope analysis revealed extensive bacterial infiltration on the surface of the implant and calculus formation consistent with peri-implantitis (42). These late-failures occur up to 10 years post loading indicating long-term follow up of socket-shield cases is necessary in quantifying the frequency of this problem. Socket-shields cannot be interpreted exactly the same as the above case reports of unintentional root retention as these teeth have often had previous endodontic therapy, possibly involved with some degree of ankylosis and difficulty of extraction, that through attempted removal may have resulted in mobilisation of the fragment (41).

Mobility of the fragment is often the cause of infection of the socket-shield. Mobility may be undetected at the time of preparation due to previous tooth mobility, diseased periodontium, or traumatic occlusion. Iatrogenic mobilisation of the socket shield can occur during buccal root fragment preparation. Early protocols where the osteotomy

preparation was through the tooth root, blunting the drills rapidly and creating massive vibration and chattering can cause loosening of the shield. Similarly, inadequate vertical reduction of the socket shield and insufficient prosthetic space can result in exposure and unfavourable mobilising forces applied by the prosthesis (40). Additionally inadequate removal of apical infection, including root canal filling material and granulation tissue, and over-preparation of the fragment resulting in fracture, can result in infection (39). These factors are all generally avoidable by comprehensive case assessment, following a meticulous, up-to-date surgical technique and a high level of clinician experience.

The reported incidence of socket-shield infection in Gluckman's retrospective evaluation was 3 out of 128 cases (2.3%) and was attributed to mobility of the fragment. In all cases the root fragment was removed and two implants lost (26). Infection of the socket-shield occurred in 5 out of 250 sites (2%) reviewed by Siormpas. In two of the cases, the infection involved the occurrence of peri-implantitis that caused the loss of the mobile socket-shields and implants up to five years after placement. Two other infected sites did not involve the implants and only the socket shields were removed, as is mandatory for mobile fragments (27). The residual peri-implant soft tissue dehiscence (PSTD) can be extremely challenging to repair and success of treatment is limited even when the implant is placed in the appropriate position. With the additional introduction of bacterial contamination of the implant rough surface, implant survival may also be compromised. Further data is required to assess the long-term implications of socket-shield infection and outcomes of their management.

The fifth infected socket-shield case in the retrospective study by Siormpas et al. (2018) occurred 9.4 years after placement. The root fragment was non-mobile and associated with peri-implant mucositis in a heavy smoker with a history of periodontal disease. What is unknown is whether the infection of the socket-shield caused the peri-implant mucositis, which may have eventually progressed to peri-implantitis (43), or whether inflammation of the peri-implant tissues resulted in the infection of the socket-shield. The interesting questions raised in this scenario is 'what is the influence of the socket shield on implants that develop peri-implantitis? Once crestal bone loss occurs, does the socket-shield create a pathway for bacteria to spread apically and amplify the rate of bone destruction around the implant?' This complication has not been addressed in the literature possibly because peri-implantitis has not yet developed in the



limited assessment period of most clinical trials. The reality is that peri-implantitis has a prevalence of 45% of patients or 25% of implants after 9 years of loading with majority of cases exhibiting bone loss within 3 years of function (30) and may be a major limitation of the socket-shield technique due to the potential risk of developing a significant defect in the long term. Perhaps in the future patients who present with peri-implantitis risk factors such as smoking, history of periodontitis (30, 44) and associated medications (45) will be considered as relative contraindications for the socket-shield technique.

## Migration

Little information exists on the potential for continued eruption of the buccal root fragment. Only one case of socket-shield migration in the literature evaluated was confirmed via CBCT scan and resulted in internal exposure. No vertical reduction was performed and the site was monitored (26). It is possible that exposure may be the only complication of migration for which aforementioned management by adjustment of the vertical height and creation of the chamfer is relatively straightforward. However, it is unclear whether the socket-shield could keep migrating following reduction and require repeated reduction in the future. What is also unknown is the impact that craniofacial growth might have on the position of the socket-shield relative to the osseointegrated implant which is described to have functional ankylosis (46). A continuous rate of growth can lead to infra-occlusion of the implant when placed in both adolescent (47) and even mature adult patients (48). Whether the socket-shield remains in position with the implant long term, or if migration occurs with the rest of the dentition as part of continued bone apposition and deposition and leads to unfavourable positioning of the root fragment is yet to be determined.

## Comparison to implant therapy with guided bone regeneration

Current approaches to rebuilding the buccal bone volume lost following extraction involves augmentation procedures with immediate, early or delayed implant placement. The majority of studies use a combination of bone grafts and barrier membranes to promote GBR in peri-implant defects {Becker, 1990}{Dahlin, 1995}{Buser, 2009}{Chen, 2005} {Chappuis, 2018}. Compared to the socket-shield technique, a wealth

of literature supports these reproducible and predictable GBR approaches to recreating the alveolar ridge, however these procedures are not without their own limitations.

Overall, 4-6 year follow up of implants placed with concomitant guided bone regeneration did not perform differently from implants placed into native bone with respect to implant survival, marginal bone height and peri-implant soft tissue parameters (49). This is supported in the retrospective analysis by Bazrafshan and Darby whereby cumulative survival rate of 98% was calculated for implants both with and without augmentation (50). These survival rates are similar to socket-shield according to reports thus far.

The use of a xenograft such as deproteinized bovine bone mineral (DBBM) does not prevent the loss of bundle bone and a 1mm vertical resorption is still expected when augmented around immediate implants {Chen, 2007}. Augmentation tends to be more successful in minimising the horizontal resorption (4). This was reflected in a prospective case series by Buser on 20 patients with a single tooth tissue level implant placement with simultaneous contour augmentation using locally harvested autogenous bone chips and DBBM and a porcine non-crosslinked collagen membrane. At the six year review, all implants were osseointegrated and a mean facial bone thickness of 1.9mm identified by CBCT scans (51). Conversely, socket-shield has shown to maintain both the horizontal and vertical dimensions of the buccal bone (37). Success of GBR around implants is also influenced by factors such as bone quality type, maxillary or mandibular site, and timing of placement which determines defect morphologies (52). The relevance of these factors in socket-shield sites is probably limited as they are unlikely to interfere with maintenance of the buccal bone which is preserved by the periodontal attachment to the root fragment.

Good aesthetic outcomes were also achieved by Buser with simultaneous augmentation achieving a mean PES of 8.25 (range 5-10) (51). A similar protocol of early implant placement using bone level implants with simultaneous contour augmentation reported a 95% success rate and an acceptable median PES of 8 at 10 year follow up (53). However, these PES are notably lower than the 'excellent' scores of ~12 reported for the socket-shield technique although these are described in smaller clinical trials (33, 35). Recession of the midfacial mucosa, even when combined with bone grafts or substitutes is a common complication with immediate implant placement. Mucosal recession of 1mm or greater was observed in around 20% (range 8 –

40%) of sites (13). Conversely, early placement with soft tissue healing combined with GBR using DBBM is associated with a relatively low incidence at 5% of sites demonstrating 0.5-1.0mm recession (54). Although less recession is seen with the socket-shield technique than with early implant placement, the minimal dimensional change may lie within the visual threshold of detecting a difference in mucosal levels and therefore have minimal effect on satisfaction with aesthetic outcomes.

Healing of any procedure is not without complication. Wound dehiscence and membrane exposure after GBR can lead to postoperative infection, inadequate healing and loss of graft material. One implant that had been placed with simultaneous augmentation in Chappuis' 10 year follow up demonstrated no facial bone wall and 5.92mm of vertical bone loss in review CBCT as a result of post-operative infection in a patient with a history of smoking and bisphosphonate use (53). Complications such as membrane exposure have been reduced with the use of resorbable membranes (Dahlin, 1995). Data on the long-term complications associated with socket-shield are scarce, however may be catastrophic and difficult to repair, even involving loss of the implant.

The expense of additional grafting materials, invasive surgical procedures, increased morbidity and associated soft tissue alterations (scarring, loss of papillae, recession on adjacent teeth) serves as a disadvantage for GBR approaches. However, the abundance of longitudinal data supporting stable clinical outcomes of an acceptable standard, low rates of complication that are more easily managed when they arise, and clinician comfort with these conventional approaches common in routine practice present as the main advantages. Socket-shield does not involve any additional material cost using the patient's own natural tooth to maintain the buccal bone and requires a single surgery with the benefits of immediate implant placement. The main disadvantages are the high sensitivity of the technique and the lack of long-term data particularly on reliability, predictability and complications including their complexity of management (55).

## Conclusion

The socket-shield is an effective technique for preserving the tooth-dependent bundle bone when tooth extraction with implant replacement is indicated. When comparing outcomes on preservation of buccal hard and soft tissue dimensional and aesthetics, the early literature on the socket-shield

technique shows superior results and similar implant survival rates in the short- to medium-term compared to conventional implant therapy with or without augmentation procedures. It becomes a philosophical question of how much risk sits comfortably with a clinician with their particular level of skill and experience to take on. Socket-shield has the potential for excellent results for most patients but longer term may present significant complications in a small handful, of which the full extent is unknown due to the lack in high-quality, long-term evidence. If the main duty of the clinician is to practice evidence-based treatment, then it becomes their responsibility to properly inform the patient that a said treatment may still be under evaluation. Future multi-centre clinical trials are required that compare conventional approaches with socket-shield of a standardized protocol. Outcomes measured should include implant survival and success, buccal ridge changes using volumetric scans and CBCT, PES, and peri-implant tissue health. A longer examination period would allow for better assessment of the long-term stability of socket-shield outcomes and the true incidence and implications of complications, including the impact of peri-implantitis and migration of the buccal root fragment. Once high quality evidence is presented that the socket-shield technique is a safe and predictable treatment the next step is for clinicians to seek education on appropriate case selection, hands-on training of a refined treatment protocol and use of correct instruments to provide patients with the best treatment outcomes.

## References

1. Schropp L, Wenzel A, Kostopoulos L, Karring T. Bone healing and soft tissue contour changes following single-tooth extraction: a clinical and radiographic 12-month prospective study. *Int J Periodontics Restorative Dent*. 2003;23(4):313-23.
2. Araujo MG, Lindhe J. Dimensional ridge alterations following tooth extraction. An experimental study in the dog. *J Clin Periodontol*. 2005;32(2):212-8.
3. Chappuis V, Engel O, Reyes M, Shahim K, Nolte LP, Buser D. Ridge alterations post-extraction in the esthetic zone: a 3D analysis with CBCT. *J Dent Res*. 2013;92(12 Suppl):195S-201S.
4. Chen ST, Darby IB, Reynolds EC. A prospective clinical study of non-submerged immediate implants: clinical outcomes and esthetic results. *Clin Oral Implants Res*. 2007;18(5):552-62.



5. Hurzeler MB, Zuhr O, Schupbach P, Rebele SF, Emmanouilidis N, Fickl S. The socket-shield technique: a proof-of-principle report. *J Clin Periodontol*. 2010;37(9):855-62.
6. Nanci A. Ten cate's oral histology-pageburst on vitalsource: development, structure, and function: Elsevier Health Sciences; 2007.
7. Cardaropoli G, Araújo M, Lindhe J. Dynamics of bone tissue formation in tooth extraction sites. *J Clin Periodontol*. 2003;30(9):809-18.
8. Qahash M, Susin C, Polimeni G, Hall J, Wikesjö UM. Bone healing dynamics at buccal peri-implant sites. *Clin Oral Implants Res*. 2008;19(2):166-72.
9. Braut V, Bornstein M, Belser U, Buser D. Thickness of the Anterior Maxillary Facial Bone Wall—A Retrospective Radiographic Study Using Cone Beam Computed Tomography. *Int J Perio & Restor Dent*. 2011;31:125-31.
10. Huynh-Ba G, Pjetursson BE, Sanz M, Cecchinato D, Ferrus J, Lindhe J, et al. Analysis of the socket bone wall dimensions in the upper maxilla in relation to immediate implant placement. *Clin Oral Implants Res*. 2010;21(1):37-42.
11. Januário AL, Duarte WR, Barriviera M, Mesti JC, Araújo MG, Lindhe J. Dimension of the facial bone wall in the anterior maxilla: a cone-beam computed tomography study. *Clin Oral Implants Res*. 2011;22(10):1168-71.
12. Evans CD, Chen ST. Esthetic outcomes of immediate implant placements. *Clin Oral Implants Res*. 2008;19(1):73-80.
13. Chen ST, Buser D. Clinical and esthetic outcomes of implants placed in postextraction sites. *Int J Oral & Maxillofac Impl*. 2009;24 Suppl:186-217.
14. Malmgren B, Cvek M, Lundberg M, Frykholm A. Surgical treatment of ankylosed and infrapositioned reimplanted incisors in adolescents. *Scand J Dent Res*. 1984;92(5):391-9.
15. Andersson L, Emami-Kristiansen Z, Höglström J. Single-tooth implant treatment in the anterior region of the maxilla for treatment of tooth loss after trauma: a retrospective clinical and interview study. *Dent Traumatol*. 2003;19(3):126-31.
16. Allen PF, McKenna G, Creugers N. Prosthodontic care for elderly patients. *Dental Update*. 2011;38(7):460-70.
17. Salama M, Ishikawa T, Salama H, Funato A, Garber D. Advantages of the root submergence technique for pontic site development in esthetic implant therapy. *Int J Periodontics Restorative Dent*. 2007;27(6):521-7.
18. Bäumer D, Zuhr O, Rebele S, Schneider D, Schupbach P, Hurzeler M. The Socket-Shield Technique: First Histological, Clinical, and Volumetric Observations after Separation of the Buccal Tooth Segment – A Pilot Study. *Clin Impl Dent & Related Res*. 2015;17(1):71-82.
19. Schwimer C, Pette GA, Gluckman H, Salama M, Du Toit J. Human Histologic Evidence of New Bone Formation and Osseointegration Between Root Dentin (Unplanned Socket-Shield) and Dental Implant: Case Report. *Int J Oral Maxillofac Implants*. 2018;33(1):e19-e23.
20. Gluckman H, Du Toit J, Salama M. The Pontic-Shield: Partial Extraction Therapy for Ridge Preservation and Pontic Site Development. *Int J Periodontics Restorative Dent*. 2016;36(3):417-23.
21. Oliva S, Capogreco M, Murmura G, Lupi E, Mariachiara DC, D'Amario M. The socket shield technique and its complications, implant survival rate, and clinical outcomes: a systematic review. *J Periodontal Implant Sci*. 2023;53(2):99-109.
22. Gluckman H, Du Toit J, Salama M, Nagy K, Dard M. A decade of the socket-shield technique: a step-by-step partial extraction therapy protocol. *Int J Esthet Dent*. 2020;15(2):212-25.
23. Glocker M, Attin T, Schmidlin PR. Ridge Preservation with Modified "Socket-Shield" Technique: A Methodological Case Series. *Dent J*. 2014;2(1):11-21.
24. Brånemark PI. Osseointegration and its experimental background. *J Prosthet Dent*. 1983;50(3):399-410.
25. Parlar A, Bosshardt DD, Unsal B, Cetiner D, Haytaç C, Lang NP. New formation of periodontal tissues around titanium implants in a novel dentin chamber model. *Clin Oral Implants Res*. 2005;16(3):259-67.
26. Gluckman H, Salama M, Du Toit J. A retrospective evaluation of 128 socket-shield cases in the esthetic zone and posterior sites: Partial extraction therapy with up to 4 years follow-up. *Clin Impl Dent & Related Res*. 2018;20(2):122-9.
27. Siormpas KD, Mitsias ME, Kotsakis GA, Tawil I, Pikos MA, Mangano FG. The Root Membrane Technique: A Retrospective Clinical Study With Up to 10 Years of Follow-Up. *Implant Dent*. 2018;27(5):564-74.



28. Pannucci CJ, Wilkins EG. Identifying and avoiding bias in research. *Plast Reconstr Surg*. 2010;126(2):619-25.
29. Buser D, Weber HP, Lang NP. Tissue integration of non-submerged implants. 1-year results of a prospective study with 100 ITI hollow-cylinder and hollow-screw implants. *Clin Oral Implants Res*. 1990;1(1):33-40.
30. Derks J, Schaller D, Håkansson J, Wennström JL, Tomasi C, Berglundh T. Effectiveness of Implant Therapy Analyzed in a Swedish Population: Prevalence of Peri-implantitis. *J Dent Res*. 2016;95(1):43-9.
31. Jung RE, Zembic A, Pjetursson BE, Zwahlen M, Thoma DS. Systematic review of the survival rate and the incidence of biological, technical, and aesthetic complications of single crowns on implants reported in longitudinal studies with a mean follow-up of 5 years. *Clin Oral Implants Res*. 2012;23 Suppl 6:2-21.
32. Cosyn J, De Lat L, Seyssens L, Doornewaard R, Deschepper E, Vervaeke S. The effectiveness of immediate implant placement for single tooth replacement compared to delayed implant placement: A systematic review and meta-analysis. *J Clin Periodontol*. 2019;46(S21):224-41.
33. Bäumer D, Zühr O, Rebele S, Hürzeler M. Socket Shield Technique for immediate implant placement - clinical, radiographic and volumetric data after 5 years. *Clin Oral Implants Res*. 2017;28(11):1450-8.
34. Mitsias ME, Siormpas KD, Kotsakis GA, Ganz SD, Mangano C, Iezzi G. The Root Membrane Technique: Human Histologic Evidence after Five Years of Function. *Biomed Res Int*. 2017;2017:7269467.
35. Bramanti E, Norcia A, Cicciù M, Maticena G, Cervino G, Troiano G, et al. Postextraction Dental Implant in the Aesthetic Zone, Socket Shield Technique Versus Conventional Protocol. *J Craniofac Surg*. 2018;29(4):1037-41.
36. Siormpas KD, Mitsias ME, Kotsiotou-Siormpa E, Garber D, Kotsakis GA. Immediate implant placement in the esthetic zone utilizing the "root-membrane" technique: clinical results up to 5 years postloading. *Int J Oral Maxillofac Implants*. 2014;29(6):1397-405.
37. Atef M, El Barbary A, Dahrous MSE, Zahran AF. Comparison of the soft and hard peri-implant tissue dimensional changes around single immediate implants in the esthetic zone with socket shield technique versus using xenograft: A randomized controlled clinical trial. *Clin Implant Dent Relat Res*. 2021;23(3):456-65.
38. Fürhauser R, Florescu D, Benesch T, Haas R, Mailath G, Watzek G. Evaluation of soft tissue around single-tooth implant crowns: the pink esthetic score. *Clin Oral Implants Res*. 2005;16(6):639-44.
39. Gluckman H, Salama M, Du Toit J. Partial Extraction Therapies (PET) Part 2: Procedures and Technical Aspects. *Int J Periodontics Restorative Dent*. 2017;37(3):377-85.
40. Gluckman H, Nagy K, Du Toit J. Prosthetic management of implants placed with the socket-shield technique. *J Prosthet Dent*. 2019;121(4):581-5.
41. Langer L, Langer B, Salem D. Unintentional root fragment retention in proximity to dental implants: a series of six human case reports. *Int J Periodontics Restorative Dent*. 2015;35(3):305-13.
42. Nevins ML, Langer L, Schupbach P. Late Dental Implant Failures Associated with Retained Root Fragments: Case Reports with Histologic and SEM Analysis. *Int J Periodontics Restorative Dent*. 2018;38(1):9-15.
43. Heitz-Mayfield LJA, Salvi GE. Peri-implant mucositis. *J Periodontol*. 2018;89(S1):S257-S66.
44. Roos-Jansåker A-M, Renvert H, Lindahl C, Renvert S. Nine- to fourteen-year follow-up of implant treatment. Part III: factors associated with peri-implant lesions. *J Clin Periodontol*. 2006;33(4):296-301.
45. Chappuis V, Avila-Ortiz G, Araújo MG, Monje A. Medication-related dental implant failure: Systematic review and meta-analysis. *Clinical Oral Implants Research*. 2018;29(S16):55-68.
46. Schroeder A, van der Zypen E, Stich H, Sutter F. The reactions of bone, connective tissue, and epithelium to endosteal implants with titanium-sprayed surfaces. *J Maxillofac Surg*. 1981;9(1):15-25.
47. Thilander B, Odman J, Jemt T. Single implants in the upper incisor region and their relationship to the adjacent teeth. An 8-year follow-up study. *Clin Oral Implants Res*. 1999;10(5):346-55.
48. Bernard JP, Schatz JP, Christou P, Belser U, Kiliaridis S. Long-term vertical changes of the anterior maxillary teeth adjacent to single implants in young and mature adults. A retrospective study. *J Clin Periodontol*. 2004;31(11):1024-8.
49. BeniĆ GI, Jung RE, Siegenthaler DW, Hämmerle CH. Clinical and radiographic comparison of implants in



- regenerated or native bone: 5-year results. *Clin Oral Implants Res.* 2009;20(5):507-13.
50. Bazrafshan N, Darby I. Retrospective success and survival rates of dental implants placed with simultaneous bone augmentation in partially edentulous patients. *Clin Oral Impl Res.* 2014;25(7):768-73.
  51. Buser D, Chappuis V, Kuchler U, Bornstein MM, Wittneben JG, Buser R, et al. Long-term stability of early implant placement with contour augmentation. *J Dent Res.* 2013;92(12 Suppl):176s-82s.
  52. Zitzmann NU, Schärer P, Marinello CP. Factors influencing the success of GBR. *J Clin Periodontol.* 1999;26(10):673-82.
  53. Chappuis V, Rahman L, Buser R, Janner SFM, Belser UC, Buser D. Effectiveness of Contour Augmentation with Guided Bone Regeneration: 10-Year Results. *J Dent Res.* 2018;97(3):266-74.
  54. Buser D, Halbritter S, Hart C, Bornstein MM, Grütter L, Chappuis V, et al. Early implant placement with simultaneous guided bone regeneration following single-tooth extraction in the esthetic zone: 12-month results of a prospective study with 20 consecutive patients. *J Periodontol.* 2009;80(1):152-62.
  55. Gluckman H, Toit JD, Salama M, editors. The socket-shield technique to support the buccofacial tissues at immediate implant placement 2015.



## AOS-ASP-APS Combined Conference 2024 Poster Abstracts

### Decontamination of titanium surface using novel nanoparticle activated by NIR laser.

Jackie Yiu

*School of Dentistry, The University of Queensland, Queensland, Australia*

#### Objectives

Decontamination of dental titanium implant surface is clinically challenging and often unpredictable. Photodynamic therapy may provide unique advantages over conventional decontamination methods, due to their ability to reach bacteria sheltered in irregular implant surfaces. A novel layered double hydroxide based nanoparticle with indocyanine green could offer unique advantages as a photosensitiser. The aim of this study was to evaluate the decontamination efficiency of adjunctive photodynamic therapy using NIR-activated LDH-ICG nanoparticles and compare it to mechanical and chemical decontamination methods on biofilm contaminated SLA titanium surface.

#### Material and methods

Titanium discs with SLA surfaces were contaminated with saliva-derived biofilm. Contaminated discs were first treated mechanically using a chitosan fiber brush. This is followed by adjunctive decontamination with chemical (EDTA), or photodynamic therapy using LDH-ICG nanoparticles of different durations. Decontamination outcome in terms of residual biofilm were evaluated by CV assay, XTT assay, SEM and live/dead staining. The influence on inflammatory mediators expression by macrophage were measured using qPCR.

#### Results

Adjunctive chemical and photodynamic therapy using LDH-ICG nanoparticles further reduced residual biofilm when compared to mechanical decontamination alone. No treatment modality, however, resulted in the complete elimination of biofilm. The application of LDH-ICG photodynamic therapy on contaminated titanium surface may result in a more favorable cytokine expression by macrophages.

#### Conclusion

Within the limitation of this in vitro study, mechanical cleaning with adjunctive photodynamic therapy using novel LDH-ICG nanoparticles is as effective as adjunctive chemical decontamination. The use of adjunctive decontamination measures is more effective in removal of biofilm than mechanical cleaning alone. However, no method of decontamination resulted in complete elimination of biofilm. Further investigations in vitro and in vivo studies are needed to evaluate the efficacy of LDH-ICG nanoparticles in decontamination and cytocompatibility following its usage.



## Long-term cumulative survival rate of dental implants in organ transplant recipients

Young Eun, Jang

*Department of Periodontology, Seoul Asan Medical Centre, Ulsan University, Republic of Korea*

### Objectives

The use of dental implants in organ transplant recipients is a contentious issue due to their modified wound healing processes and increased susceptibility to serious infections. The purpose of this retrospective study is to evaluate the long-term survival rate and related clinical parameters of dental implants after organ transplantation.

### Material and methods

This study was conducted on 356 dental implants installed in 113 patients at Asan Medical Center from 2001 to 2021. Parameters such as gender, age, transplanted organ, implant position (anterior, premolar and molar), jawbone (maxilla and mandible), GBR, types of immunosuppressants, and the etiology of implant failure were recorded. A life table analysis and Kaplan-Meier analysis was used to calculate the cumulative survival rate (CSR). Comparisons of 10-year CSR among gender, implant position (anterior, premolar and molar) and jawbone (maxilla and mandible) were performed using the log-rank test.

### Results

Among 356 dental implants installed in 113 patients, 30 implants in 8 patients were lost and the CSR up to 5-years and 10-years were 92.7% and 89.6%, respectively. Statistically significant differences were observed in the 10-year CSR among gender ( $P = 0.001$ ) and jawbone ( $P = 0.0003$ ) whereas no statistically significant differences were observed among implant position ( $P = 0.352$ ). The 10-year CSR of the male was 83.6%, that of the female was 100.0%, that of the maxilla was 81.0%, and that of the mandible was 97.2%. 23 fixtures were removed due to osseointegration failure, 4 due to peri-implantitis, 2 due to the fracture of abutment, and 1 due to the fracture of fixture.

### Conclusion

Organ transplant recipients exhibit a higher incidence of dental implant failure rates compared to otherwise healthy patients. It is noteworthy that this study reported longer follow-up periods and included a larger number of patients and implants compared to previous studies assessing dental implant failure in organ transplant recipients.

## **Influence of vertical level of implant abutment margin on residual cement occurrence**

Hyun Ju, Kim

*Seoul National University Dental Hospital, Seoul National University, Republic of Korea*

### **Objectives**

The aim of this study was to evaluate the influence of vertical level of implant abutment margin on residual cement occurrence in cement-retained implant restorations with customized abutments.

### **Materials and Methods**

A total of 109 single-unit cement-retained implant restorations with a screw-access channel were included. The crowns were intraorally cemented on the abutments, and excess cement was removed. After unscrewing, the abutment–crown complex and peri-implant tissue were photographed. The presence of residual cement was recorded by dividing the abutment–crown complex and peri-implant tissue into four quadrants: mesial, distal, buccal, and lingual. The prevalence of residual cement was compared according to the vertical level of the abutment margin at the corresponding quadrant. A multilevel model was used for statistical analysis ( $\alpha = .05$ ).

### **Results**

Cement remnants were observed on 72.5% of the total implants. When the restoration quadrants were compared, cement remnants were present on 51.4%, 39.5%, 20.2%, and 17.4% of the mesial, distal, buccal, and lingual surfaces, respectively ( $p < .01$ ). Regarding the abutment margin level, cement remnants were found in 60.2% and 61.4% of the 0.5 mm subgingival and  $\geq 1$  mm subgingival margin groups, respectively, which were significantly more than those in the supragingival (23.7%) and equigingival (26.6%) margin groups ( $p < .01$ ). After adjustment for confounding factors, the adjusted odds ratio (with 95% confidence interval) for residual cement in the subgingival margin groups was 3.664 (1.710, 7.852) when compared to the supragingival and equigingival margin groups.

### **Conclusions**

Subgingival abutment margin had a 3.7-fold greater risk of residual cement occurrence than supragingival or equigingival margin.





## Automated Identification of Dental Implants Using Artificial Intelligence

Rafael Santos

*School of Dentistry, The University of Queensland, Queensland, Australia*

### Objectives

To develop and evaluate the accuracy of a computer-assisted system based on artificial intelligence for detecting and identifying dental implant brands using digital periapical radiographs.

### Materials and Methods

A total of 1,800 digital periapical radiographs of dental implants from three distinct manufacturers ( $f1 = 600$ ,  $f2 = 600$ , and  $f3 = 600$ ) were split into training dataset ( $n = 1,440$  [80%]) and testing dataset ( $n = 360$  [20%]) groups. The images were evaluated by software developed by means of convolutional neural networks (CNN), with the aim of identifying the manufacturer of the dental implants contained in them. Accuracy, sensitivity, specificity, positive and negative predictive values, and the receiver operating characteristic (ROC) curve were calculated for the detection and diagnostic performance of the CNN algorithm.

### Results

At the final epoch (25), system accuracy values of 99.78% were obtained for group training data, 99.36% for group testing data, and 85.29% for validation data. The latter value corresponded to the actual accuracy of carrying out the system learning process.

### Conclusion

This study demonstrated the effectiveness of CNN for identifying dental implant manufacturers in periapical radiographs, which was proven to be a precise method of great clinical significance.

## Extracellular Vesicles as Dual Messengers: Deciphering Microbial and Host Interaction for Periodontitis

Chun Liu

*School of Dentistry, The University of Queensland, Queensland, Australia*

*Centre for Orofacial Regeneration, Rehabilitation and Reconstruction (COR3), School of Dentistry, The University of Queensland, Queensland, Australia*

### Objectives

Extracellular vesicles (EVs) are nanoscale lipid-bilayer particles derived from most cells of different species, including host and bacterial-derived EVs (BEVs) [1]. Oral bacterial-derived BEVs contain a variety of microbial molecules, including enzymes, toxins, and microbial-associated molecular patterns (MAMP) [1], that can be transported to recipient host cells locally and systematically to cause periodontitis or other systemic diseases [1, 2]. In terms of host response, host-derived EVs with encapsulated pro-inflammatory cytokines may contribute to the modulation of immune and inflammatory processes in oral disease pathogenesis [3]. Limited studies explored both dental plaque derived BEVs and saliva host EVs cytokine profiles. This study aims to a) understand the BEV component by comparing 16S sequencing profiles from 3D-mimicking saliva biofilm and b) assess the potential of immunoaffinity-enriched host EVs from saliva as diagnostic markers for periodontitis.

### Materials and Methods

For BEV profiling, oral biofilms were cultured on 3D polycaprolactone (PCL) scaffolds and 2D plates. BEVs were isolated using size exclusion chromatography (SEC) and characterized by multiple techniques, followed by genomic DNA qPCR and 16S sequencing. Simultaneously, host-derived EVs were enriched from 12 non-periodontitis and 20 periodontitis patients' saliva using SEC and bead-based immunoaffinity capture. After saliva-EVs characterization, inflammatory cytokines (IL-6, IL-8 and IL-10) in host EVs were compared between non-periodontitis (n=12) and periodontitis (n=20) groups.

### Results

16s sequencing results suggest that BEVs exhibit strong enrichment ability and sensitivity with genera *Capnocytophaga*, *Porphyromonas* and *Veillonella*, phylum *Firmicutes* and *Bacteroidota*, and species *Alloprevotella\_tanneriae*, *Capnocytophaga\_sputigena*, *Veillonella\_atypica* and *Prevotella\_melaninogenica*. Moreover, immunoaffinity-enriched salivary EVs from periodontitis patients displayed elevated pro-inflammatory cytokines (IL-6, IL-8) and reduced anti-inflammatory IL-10 compared to non-periodontitis individuals.

### Conclusion

Investigating BEVs from oral biofilm and cytokine signatures in salivary host EVs could enhance our understanding of periodontitis, leading to more accurate diagnosis and targeted therapeutic interventions.



## The BISHOP (Biomarkers In Saliva of Health or Periodontitis) Study

Stella Lee

*School of Dentistry, The University of Melbourne, Melbourne, Australia*

### Objectives

Periodontitis is a complex, chronic immune-mediated inflammatory condition associated with dysbiotic plaque biofilms, leading to soft and hard tissue destruction around teeth (Papapanou et al., 2018). *Porphyromonas gingivalis* is a keystone pathogen with extensive virulence factors implicated in periodontitis (Hajishengallis et al., 2012). Non-surgical cause-related periodontal therapy is the primary treatment modality and often improves clinical outcomes (Badersten et al., 1984). As periodontitis is host-mediated, its progression and treatment outcomes can be reflected in saliva. Numerous salivary microbial and immunological biomarkers have been investigated for periodontitis identification and monitoring.

### Materials and Methods

This study recruited 35 stage III/IV periodontitis patients and 16 healthy controls from university clinics to examine clinical, microbial, and immunological differences pre- and post-periodontal treatment. Unstimulated whole saliva samples were analysed for *P. gingivalis* counts via DNA extraction using a KqP test. A Bioplex assay quantified 25 inflammatory cytokines and chemokines in saliva. Comparisons were made between periodontitis and control subjects, and between baseline and post-treatment saliva samples from periodontitis cases.

### Results

Periodontal treatment led to significant reductions in mean probing depths and improvements in clinical attachment levels, plaque index, and modified gingival index. *P. gingivalis* was more prevalent in periodontitis cases compared to controls, with an AUC of 0.77. Post-treatment, mean *P. gingivalis* counts decreased three-fold. There were statistically significant changes in the concentrations of inflammatory cytokines and chemokines post-treatment, reflecting a shift towards levels observed in health. Significant differences were noted in the levels of IL-1, IL-2R, IFN-, IL-4, and IL-5.

### Conclusion

This study underscores the potential of saliva as a source of biomarkers for periodontitis detection and monitoring. *P. gingivalis* counts in saliva are a useful surrogate measure of disease. Future research should validate these biomarkers in larger cohorts to enhance personalised periodontal care.

## Effect of maxillary sinus morphological characteristics on de novo bone formation

Denise Hsueh

*School of Dentistry, The University of Queensland, Queensland, Australia*

### Objectives

A key objective of maxillary sinus augmentation (MSA) is to maximise the amount of new bone formation. However, the factors that influence this process are still poorly understood. This prospective clinical study aimed to investigate the degree of association between maxillary sinus morphological characteristics, particularly, the resident bone surface area in contact with the bone graft (CA), and graft volume (GV) on the fraction of new bone formation following MSA using the lateral window approach.

### Materials and Methods

Collagen-stabilised deproteinised bovine bone mineral (DBBM-C) was used as the sole grafting material. During implant placement, the biopsies were performed after a mean healing period of  $7.4 \pm 1.8$  months for histomorphometric analysis. Area percentage of new bone (%NB), residual graft (%RG) and non-mineralised components (%STM), and proportion of graft particle perimeter in direct contact with bone (%OI) were measured after dividing the grafted area into equal thirds (coronal, middle and apical). The region of interest (ROI) was the apical third of the graft. CA and GV were measured on CBCT scans taken 5 months after MSA and correlated with histomorphometric results. Simple linear regression, independent-samples t-test, Mann-Whitney U test and ANOVA were used to assess the impact of morphological characteristics and GV on %NB.

### Results

19 patients with a mean age of  $57.4 \pm 9.5$  were included in the results. No significant association was found between CA and GV, and %NB in the ROI. However, a statistically significant difference in %NB was observed in the coronal third of the graft ( $26.0 \pm 12.3\%$ ) compared to the apical third of the graft ( $13.0 \pm 7.9\%$ ).

### Conclusion

In conclusion, there is insufficient evidence to support the hypothesis that new bone formation following MSA is correlated to sinus morphological characteristics or graft volume.



## Effect of alveolar ridge preservation on hard and soft tissue dimensional changes

Daniel Mckenzie

*School of Dentistry, The University of Queensland, Queensland, Australia*

### Objectives

To investigate the volumetric and linear dimensional changes for implant supported restorations in the anterior maxilla (13-23) after alveolar ridge preservation (ARP) compared to implants placed into unassisted socket healing (USH) sites with an early placement protocol.

### Materials and methods

35 participants referred for single implant replacement were recruited. Following minimally traumatic extraction, patients were randomly allocated to two treatment groups: (a) ARP using demineralised bovine bone mineral with 10% collagen (DBBM-C) covered by a collagen matrix or membrane (CM), (b) unassisted socket healing. Implant placement was performed after 12-16 weeks in the ARP group and 6-8 weeks in the USH group. Patients were followed up after implant restoration and 12 months post-loading. Intra-oral scans were used to compare the change in alveolar ridge volume and mid-facial dimensional changes from pre-extraction up to 12 months post-restoration.

### Results

No significant difference was observed at any timepoint for the volumetric alveolar ridge changes or mid-facial linear dimensional changes. Increased atrophy was observed in both treatment groups when a buccal bone defect was present following extraction, with no significant difference between the groups. A reduced need for augmentation simultaneous to implant placement was observed in the ARP group compared to USH (50% vs 89%). The mid-facial mucosal margin changes showed a statistically significant difference from pre-extraction to implant restoration which remained significant 12 months post restoration in favour of the ARP group ( $-0.43 \pm 0.43\text{mm}$  vs  $-1.31 \pm 0.61\text{mm}$ ,  $p = 0.012$ ).

### Conclusion

Single tooth implant placement in the anterior maxilla following ARP or USH showed comparable alveolar ridge dimensional changes as assessed using volumetric and linear measurements. An increased incidence of mid-facial mucosal margin change  $\geq 1\text{mm}$  was observed following USH.



Combined Conference

Gold Coast

**AOS · ASP · APS**

19 – 21 September 2024

[www.aosaspaps.com](http://www.aosaspaps.com)







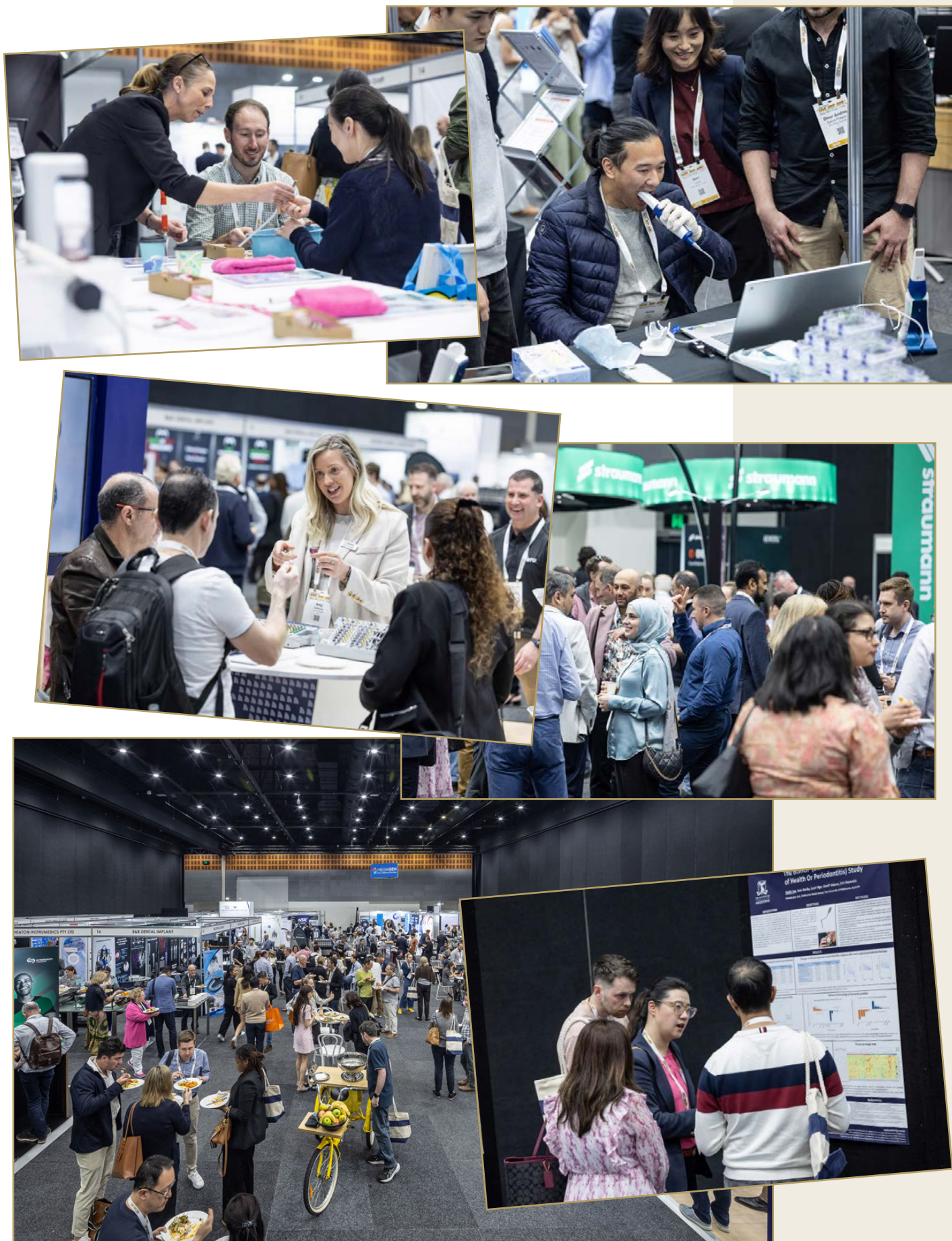




It would be a problem with the acts allegedly...  
com...  
"extr..."







### ASP NSW Branch Committee Details and Meetings

**President:** Dr Khai Nguyen

**Secretary/Treasurer:** Dr Wesley Wong

**State Branch Councillor:** Dr Rob Fell

**Secretariat:** Brooke Mcfarlane

**Email:** aspnsw@asp.asn.au

**Meeting name:** ASP (NSW) Full Day Meeting

**Meeting date & time:** Friday 15th November 2024

**Meeting location:** Swissotel Sydney

**Speakers:** Frank Strauss

**Topics:** Implants and Peri Implant Disease - Should I be changing my practice?

**Cost & other details:** Members \$100  
Guests; TBA

### ASP QLD Branch Committee Details and Meetings

**President:** Dr Marina Kamel

**Secretary:** Dr Miriam Lee

**Treasurer:** Dr Gabrielle Bou-Samra

**Federal Councillor:** A/Prof Ryan Lee

**Email:** aspqld@gmail.com

**Meeting name:** ASP (QLD) Full Clinic Day

**Meeting date & time:** Friday 8th Nov 2024 - 8:30am

**Meeting location:** The Inchcolm by Ovolo

**Speakers:** Professor Nikolaos Donos

**Topics:** Guided Bone regeneration and Treatment modalities for peri-implantitis

**Cost & other details:** Fees: Free for members and \$350 for non-members





## ASP SA Branch Committee Details and Meetings

**President:** Dr Geoff Harvey

**Secretary:**

**Treasurer:**

**State Branch Councillor:**

**Email:** aspsa2@gmail.com

**Meeting name:** ASP (SA) Dinner Meeting #4 and AGM

**Meeting date & time:** Wednesday 16 October 2024. 6pm for 6:30pm start

**Meeting location:** The Gallery, 30 Waymouth Street, Adelaide SA

**Speakers:** Dr Michael Stokes, Cardiologist

**Topics:** Cardiovascular Disease and Periodontitis

**Cost & other details:** No additional charge for paid members/sponsors. \$125 for single guest ticket

## ASP VIC Branch Committee Details and Meetings

**President:** Dr Larissa Ong

**Vice President:** Dr Alice Huynh

**Secretary/Treasurer:** Dr. Yevgeny (Eugene) Sheftel

**Branch Councillor:** Dr Sarah Chin

**Email:** aspvic@gmail.com

**Meeting name:** ASP (VIC) Dinner Meeting

**Meeting date & time:** Date: 20th November 2024 6.00pm registration for a 6.30pm start

**Meeting location:** Woodward Conference Centre - 10th Floor,

Melbourne Law, the University of Melbourne, 185 Pelham Street, Carlton VIC 3053

**Speakers:** A/Prof Neil McGregor

**Topics:** Precision medicine-based approach to diagnosis and treatment of periodontitis.

**Cost & other details:** RSVP: by 13th November 2024 with dietary requirements Cost: \$180 (includes 3-course dinner) via EFT to BSB: 083026 Acc: 609430668. Free for ASP (Vic) members. CPD hours: 1.0

### ASP WA Branch Committee Details and Meetings

**President:** Dr Nish Bhargava

**Secretary:** Ms Jennine Bywaters

**Treasurer:** Dr Samy Francis

**Federal Councillor:**

**Email:** aspwapert@gmail.com

**Meeting name:** ASP(WA) Dinner Meeting


**Meeting date & time:** Friday, 15 November 2024, 6pm

**Meeting location:** Mandoon Estate, Caversham

**Speakers:** Dr Ehsan Mellati

**Topics:** Hopeless prognosis - is there such a thing?

**Cost & other details:** Members: \$100



**EuroPerio11**  
Registration and abstract submission are open!

Visit [efp.org](http://efp.org) for more information

**EFP** | **EuroPerio11**  
An EFP initiative | MAY 14-17 | 2025  
VIENNA

The most anticipated event in periodontology and implant dentistry, *EuroPerio11*, is now open for registration. Secure your spot at the world's leading congress in our field. **EuroPerio11 will take place in Vienna, from 14 to 17 May 2025.**

Be part of the programme too! **Abstract submission** is also now open.  
Check out the **EuroPerio11 programme** today.



## AOS NSW Committee Details and Meetings

**President:** Dr Eugene Foo

**Secretary:** Dr Cecilia So

**Treasurer:** Dr Bruce Munroe

**Federal Councillor:** A/Prof George Pal

**Admin/Secretariat:** Heather Archer

**Email:** infonsw@aos.org.au

**Meeting name:** AOS (NSW) Half Day Meeting

**Meeting date & time:** Friday, 25th October 2024, 3:00pm -9:00pm

**Meeting location:** Dr Adam Hamilton

**Speakers:** The View, 17 Blue Street, Sydney.

**Topics:** Making the Right Choice: Explore the intricacies of Material Selection in Restorative . New Classification and Concepts: Understand the latest Implant Placement and Loading Protocols

**Cost & other details:** Members: Free Guest \$450, online \$100 Register Online- [https://www.eventbrite.com.au/e/aos-nsw-half-day-meeting-amg-october-registration-920534542597?aff=odeimcmalchimp&mc\\_cid=c6d1f85ecb&mc\\_eid=852a166990](https://www.eventbrite.com.au/e/aos-nsw-half-day-meeting-amg-october-registration-920534542597?aff=odeimcmalchimp&mc_cid=c6d1f85ecb&mc_eid=852a166990)

## AOS QLD Committee Details and Meetings

**President:** Dr Peter LC Chen

**Secretary:** Dr Daniel Hu

**Treasurer:** Dr Jonathan Ng

**Federal Councillor:** Dr Jonathan Ng

**Email:** aosqld@gmail.com

**Meeting name:** AOS (QLD) Research Competition

**Meeting date & time:** Wednesday 9th of October 2024

**Meeting location:** Tattersalls Club Brisbane

**Speakers:** Researchers with Osseointegration or Implant related Research

**Topics:**

**Cost & other details:** Free for Members, \$150 for Non-Members Register via email aosqld@gmail.com

### AOS SA Committee Details and Meetings

**President:** Dr Ramon Baba

**Secretary:** Mr Hab Awwad

**Treasurer:**

**Federal Councillor:** Dr Ramon Baba

**Admin/Secretariat:** Ms Francine Poole

**Main Email Address:** infoaos.sa@gmail.com

**Meeting name:** AOS (SA)

**Meeting date & time:** Tuesday, 8 October 2024

**Meeting location:** Lion Hotel North Adelaide - Tower room

**Speakers:** Dr Stephen Soukoulis

**Topics:** TBC

**Cost & other details:** "full member - no cost, non member \$110 + GST meeting registration and details: <https://www.eventbrite.com.au/e/aos-sa-dinner-lecture-dr-steve-soukoulis-plus-2024-agm-registration-998159390607>"

### AOS Victoria Committee Details and Meetings

**President:** Dr Angelos Sourial

**Secretary:** Dr Paul Fagiarone

**Treasurer:** Dr Betty Lisa Matthews

**Federal Councillor:** Dr Gabriel Rodriguez-Ortiz

**Committee Members:** Dr Brandon Krapf, Dr Larissa Ong, Dr Philip Ho, Dr Fady Tossoun, Dr David Laskey, Mr Michael Qiu

**Admin/Secretariat:** Ms Bella Cherkasskaya

**Email:** infovic@aos.org.au aosvic@gmail.com

**Meeting name:** Dinner meeting and online broadcasting

**Meeting date & time:** November 2024

**Meeting location:** Royal South Yarra Lawn Tennis Club 310 Williams Road North, Toorak 3142

**Speakers:** Dr Varun Gang Prosthodontist and Dr Sarah Byrne Periodontist

**Topics:** TBC

**Cost & other details:** Members- free, Students - \$55, Online members (dinner) - \$110, Non-members - \$190

**Meeting name:** Dinner meeting and online broadcasting

**Meeting date & time:** February-March 2025

**Meeting location:** Royal South Yarra Lawn Tennis Club 310 Williams Road North, Toorak 3142

**Speakers:** Panel Discussion and case presentation

**Topics:** Do we place an implant or.....? Alternative options for teeth replacement.

**Cost & other details:** Members- free, Students - \$55, Online members (dinner) - \$110, Non-members - \$190



## AOS WA Committee Details and Meetings

**President:** Dr Tony Strangio

**Secretary:** Dr Andrew Ziepe

**Treasurer:** Dr Richard Williams

**Federal Councillor:** Dr Roy Sarmidi

**Email :** infowa@aos.org.au

**Meeting Name:** AOS WA Dinner Meeting

**Meeting date & time:** Thursday, 17th of October 2024 6.30pm for 7.00pm lecture time

**Meeting location:** The University Club of Western Australia

**Speakers:** Drs Mithran Goonewardene and Brent Allan

**Topics:** Aspects of Implant Replacement

**Cost & other details:** TBA

## Find out online...

Meeting details are also available online:

**Australian Society of Periodontology**  
<https://www.asp.asn.au/>

Or check with your state branch Secretary/Secretariat for further details.

**Australasian Osseointegration Society**  
<https://www.aos.org.au/>

Or check with your state branch Secretary/Secretariat for further details.







AOS

# The Australian Journal of Periodontology and Implant Dentistry Limited

The Official Journal of the Australian Society of Periodontology  
and the Australasian Osseointegration Society

AOS

*Australasian  
Osseointegration  
Society limited*

