



Peri-implant mucosal tissue attachment: Narrative review

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ABSTRACT

The peri-implant mucosa (PIM) is formed during the wound healing process that follows implant and abutment placement. The PIM is known to play crucial roles in function; acting as a biologic barrier for bacterial infiltration and maintaining the mucosal health of the implant, and for implant esthetics. This narrative review presents:

- Differences between periodontal soft tissue attachment and peri-implant mucosal tissue attachment
- Development of the osseointegration and its effect on PIM
- Different Implant – abutment interface configurations including one- VS two-piece implants, bone-level VS tissue-level implants, and PIM in different implant collars
- Engineering in surface characteristics

1. Introduction

The peri-implant mucosa (PIM) is formed during a well-orchestrated wound healing process that follows implant and/or abutment placement. In other words, the PIM is oral mucosa that has adapted to the presence of the dental implant and/or transmucosal component. Beyond its pink esthetic role, the PIM arguably provides an even greater functional role—serving as a biologic barrier against bacterial infiltration and maintaining the mucosal health of the implant.

1.1. Functional role of peri-implant mucosa

Implants are a predictable long-term solution for replacing teeth, as studies indicate a 10-year survival of dental implants of 96 % [1]. However, the incidence of peri-implantitis has been steadily increasing, with an estimated weighted mean prevalence of 22 % with a range of 1–47 % [2]. Peri-implantitis is caused by a bacterial biofilm induced host inflammatory reaction yielding loss of supporting bone around an implant in function. Studies report that the microbiota linked to peri-implantitis is more diverse than that of healthy peri-implant tissues [3–5]. The microbiota present in peri-implantitis is more diverse and a large proportion is composed of Gram-negative bacteria species. Peri-implantitis was shown to be associated with certain pathogens (e.g., Gram-negative anaerobic periopathogens and opportunistic

microorganisms).

While the supracrestal attachment of natural teeth and dental implants have a similar function, there are specific differences in structure and composition. In teeth, the periodontal soft tissue attachment is a biologic barrier to bacterial invasion. Yet simultaneously the semi-permeable nature of components such as the junctional epithelium provide unique capabilities for immune surveillance. Unlike the dentogingival complex of dentition, the seal at the implant abutment has a weaker attachment with the surrounding soft tissue. Supracrestal tissue attachment height around teeth consists of a layer of junctional epithelium that is 0.97 mm on average and a connective tissue attachment averaging 1.07 mm [6]. For implants, this supracrestal tissue attachment forms only after the transmucosal abutment is placed. In the peri-implant tissues, these values are around 1.88 mm and 1.05 mm, respectively [Fig. 1] [7]. Implants have thinner and longer junctional epithelium with a hemidesmosome attachment and a thin basal layer. Implants also have a higher fiber content and collagen fibers arranged parallel to the implant surface with less fibroblasts than teeth, which only have perpendicular fibers [8]. According to the American Academy of Periodontology consensus report of the 2017 World Workshop [9], “healthy peri-implant mucosa averages 3 to 4 mm in height” compared to 2 to 3 mm we would expect with healthy periodontium around teeth. Thus, implants have a higher risk of bacterial penetration [5]. Additionally, a deeper physiologic probing depth with higher mean bleeding

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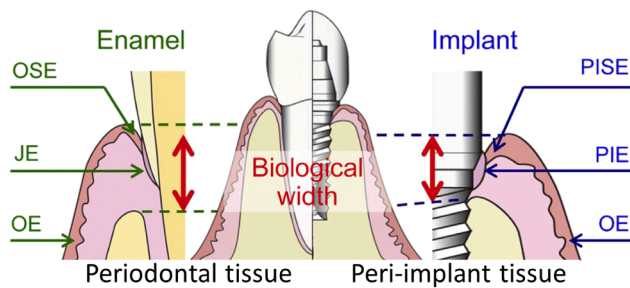


Fig. 1. Supracrestal tissue attachment of peri-implant and periodontal tissue. Diagram highlights the significant landmarks of the epithelial component of soft tissue attachment to periodontal tissue (left) and the counterpart in the peri-implant soft tissue attachment (right). (JE: junctional epithelium, OSE: oral sulcular epithelium, OE: oral epithelium, PIE: peri-implant epithelium, PISE: peri-implant sulcular epithelium).

on probing should be expected with implants compared to teeth, even in health. Implants also do not have periodontal ligament, so when disease or injury occurs, the bone is directly affected.

At the cellular level, the PIM features fewer human gingival fibroblasts (HGFs), less vascularity and a parallel orientation of collagen fibers, which make implants more susceptible to peri-implantitis initiation and progression. Additionally, during the healing of soft tissue around an implant, there is epithelial downgrowth due to the faster speed at which epithelial cells grow compared to connective tissue cells. This results in long junctional epithelial attachment, which is weaker than connective tissue attachment and provides less stability for the implant. This, along with the absence of a stable supracrestal connective tissue attachment, leads to much more accelerated progression of peri-implant disease around tissues. Therefore, there is an urgent need to improve the interface between the peri-implant mucosal tissue and the abutment material for a more effective biological seal. The soft tissue component of implants has less cellularity overall, with fewer HGFs and inflammatory cells. However, the soft tissue at the abutment interface has a higher degree of cellularity, suggesting cell turnover to preserve a functional seal [10]. The connective tissue zone of peri-implant tissues does not have inserting fibers into the implant surface. Additionally, the peri-implant tissues have less vascularity between the alveolar crest and the junctional epithelium in comparison to the connective tissue zone of the periodontium around teeth [9].

1.2. Esthetic role of peri-implant mucosa

A proper structure and quality of PIM may be necessary to provide esthetic standards. Although most of the currently available implants have shown 10-year survival rate of 96 % [1], the distinction between implant survival and success is important in determining clinical outcomes of different materials, techniques, and procedures [11]. Implant success as defined by Albrektsson et al. in 1986 is an implant with "no mobility, no peri-implant radiolucency, bone loss less than 0.2 mm per year after the first year of loading, and no persisting pain, discomfort, or infection" [12]. However, during last two decades, the focus of a successful implant shifted toward also achieving an esthetic which is in harmony with surrounding teeth [13,14]. The phenomena such as absence of interproximal papilla and/or mid facial recession play major role in defining implant esthetics [15]. One index for defining implant success is the pink esthetic score (PES). To achieve success, implant-supported restorations should replicate natural teeth. The decisive consideration of this seems to be the quality of the peri-implant mucosal tissue. PES measures presence or absence of papilla, soft tissue contour, gingival level, alveolar process resorption, coloring, and texture. The height of the peri-implant mucosal tissue alters the crown length, and the tissue color and texture are crucial to achieve a natural appearance of implant-supported restorations [11]. Thus, implant

success includes not only adequate function but also satisfactory esthetic outcomes. In contrast, implant survival implies that the implant is present in the mouth with no consideration of function or esthetics.

2. Historical narrative: implant and surface design focus on reliable osseointegration

Original dental implant research and design efforts focused on achieving successful and consistent osseointegration. The concept of osseointegration was discovered serendipitously by Per-Ingvar Brånemark in 1952, while studying blood flow in rabbit femurs through the placement of titanium chambers into bone [16]. With time, Brånemark found that the titanium became securely affixed to the bone and was not removable. The bone had bonded to the titanium surface. In the case of a fracture, it was always between bone and bone and never between bone and implant. In time, this idea was extended to the field of dentistry [17]. The term "osseointegration" was further nuanced and defined by Brånemark as, "a direct structural and functional connection between ordered, living bone, and the surface of a load-carrying implant."

The first report about the soft tissue reaction with titanium implants was documented a few years later and in 1974, ITI implant system which was made with rough titanium with a plasma sprayed (TPS) surface, was introduced [18]. Early reports showed favorable long-term survival of the ITI system. However, the TPS implants were found to have greater marginal bone loss [19] as well as higher failure rates, notably in periodontal patients and smokers [20]. The concept of soft tissue reactions with TPS implants was introduced in later years with the tissue-level implants discussed in the next paragraph. The one-piece ITI implant first introduced in 1974 was a prototype for the development of two-piece implants, which provided more prosthetic options but maintained the key notion of a tissue-level implant, which usually have a collar with a smooth titanium surface [18]. A two-piece threaded titanium root-form implant was introduced in 1978 by Brånemark [21]. A system of pure titanium screws was established and assessed, called "fixtures". The original Brånemark implant had a cylindrical form, and tapered designs later emerged [22].

In 1986, the one-piece, or tissue-level, implants were introduced by Straumann [12]. These implants, were designed with a TPS surface on the endosseous portion to enhance bone anchorage and a machined neck for the transmucosal portion to prevent peri-implant lesions. These implants were one of the first implant systems that considered the peri-implant mucosal tissue in the implant design with a smooth transgingival collar. These implants were intended to be used in a one-stage surgical protocol which would allow for transgingival healing without requiring a second-stage surgery to uncover the implant shoulder. Furthermore, the original Straumann tissue-level implant system was created to be biomimetic. By design, this implant allowed for the microgap present at the implant-abutment interface to be located at the gingival margin and away from the bone. Additionally, the smooth, transmucosal collar would promote adequate hygiene and plaque control, decreasing the risk of peri-implant disease. Specific design features that may influence the soft tissue interface will be reviewed in the following section.

In 1993, Tarnow discussed periodontal disease in dental implant patients. He presented the concept of peri-implant mucosal tissue stability and its role in implant integration and survival and introduced the hybrid design [23]. Since then, implant surfaces have changed to address the issue of peri-implant mucosal tissue for implant health and stability. A study by Lindhe found that there were more severe clinical and radiographic signs of tissue destruction around implants compared to teeth [24]. More significantly, the lesion around implants extended into the bone marrow. Because the inflammatory infiltration was not confined to the soft tissue, this could indicate that failed implants could lead to osteomyelitis. In the late 1990s, Straumann introduced the sandblasted, large grit (particle size between 250 and 500 μm), acid-etched (SLA™, Institut Straumann AG, Basel Switzerland) surface

on the two-piece implants, with acid-etching to enclose a macro-roughness ($Sa = 1.5 \mu m$) on the titanium surface [Table 1]. Microroughness affects soft tissue attachment as the cells present in the peri-implant mucosal tissue interface appear to attach and proliferate on rougher surfaces more favorably due to the increased surface area present [7,25].

3. Implant-abutment and surface design address the soft tissue interface

In order to promote biocompatibility at the implant/soft tissue interface, efforts continue to improve implant and abutment designs, as well as surface characteristics.

One-piece and two-piece implant[Fig. 2a,b]: There are two primary types of implant systems used in dentistry, each with distinct indications, but neither provides the ideal environment microscopically or macroscopically. One-piece implants have the endosseous portion and abutment as a single unit. Conversely, two-piece implants consist of an endosseous component placed into the bone, along with an independent supragingival portion, or prosthetic abutment. The absence of a micro-gap has long been thought of as an advantage of one-piece implants [26]. The presence of a microgap has been shown to contribute to peri-implant mucosal inflammation at the implant-abutment interface which often progressed to alveolar bone loss around the implant. Thus, it is important that the abutment has a precise fit to the implant in order to inhibit plaque accumulation that could potentially lead to inflammation and concomitant soft tissue loss around implants [27].

Bone-level vs. tissue-level implant [Figs. 3a-c]: Typically, the bone-level implant has a moderately rough surface implant body placed at epicrestal or the crestal level [28]. This design requires a transmucosal abutment (either healing or prosthetic abutment) independent of the implant so that the implant-abutment interface is located at the bone level (Straumann®, Institut Straumann AG, Switzerland) [Fig. 3a,b]. However, if bone-level implants are placed above the bone crest, or exposed due to bone loss, there is a risk that the rough surface of the implant will impinge on the soft tissues. In general, rough textured surfaces are more prone to plaque accumulation therefore causing inflammation that can lead to peri-implant mucositis and/or peri-implantitis [29]. To address the issue with plaque control on the rough textured surface, a tissue-level implant with smooth transmucosal collar placed at the level of the soft tissue, was introduced (Straumann®, Institut Straumann AG, Switzerland) [Fig. 3c]. It is imperative that this part is coronal to the level of bone so the polished portion will be in contact with the soft tissue [28]. Since there is one piece, there is also less concern for bacterial colonization around the microgap at the level of the bone [30].

Table 1
Surface roughness of different implants (Sa).

Sa category	Sa Value (μm)	Implant surface	Implant system(s)
Smooth surface	0.0–0.4	"Machined" implants	Experimental
Minimally rough surface	0.5–1.0	Turned (machined) surface implants	Brånemark System
Moderately rough surface	1.0–2.0	Blasted and/or acid-etched surface, oxidized surface, laser-microtextured surface	Straumann SLA ad SLActive, Nobel Biocare TiUnite, BioHorizons
Rough surface	> 2.0	TPS implants, HA-coated implants	Laser-Lok Straumman TPS, Zimmer Calcitrek Integral, Omnilock

3.1. Implant-Abutment interface

Recent studies have suggested that the soft tissue at the implant-abutment interface may affect plaque control, inflammation, and the progression of peri-implant disease. Modern implants have implant macrostructure designed to address the issue of peri-implant mucosal tissue stability to aid in implant integration and survival [31–33].

Platform switching design: Another concept of implants is platform-switching, in which the abutment size is a smaller diameter than that of the implant [33]. The rationale for this is to create an additional horizontal dimension for peri-implant mucosal tissue as well as the biologic width and shift the micro-gap and its associated inflammatory cell infiltrate medially [30]. Therefore, there should be less bone loss around the neck of the implant and satisfactory dispersion of load. Platform-switching implants permit adequate attachment of marginal epithelium to the surface of the implant and favorable soft tissue profile. Implant design and its position within the transmucosal portion may influence the development of the peri-implant mucosa and marginal bone remodeling during healing [34].

Implant-abutment connections: Short-term and mid-term follow-up studies have shown that the internal connections may exhibit less marginal bone loss [31]. For example, a conical connection design, in which the conical shape of the abutment fits inside the implant with a matching taper of equivalent angle, exhibits less marginal bone loss. This behavior may be attributed to a reduced gap, associated with less "leakage" of bacterial colonization [32].

3.2. Implant collar

Various modifications to the crestal implant collar zone have been suggested to optimize the implant-soft tissue interface. The hybrid design implant [Fig. 4] is composed of a smooth, machined surface in the coronal portion and a plasma-sprayed surface in the apical portion of the implant in one body. In theory, this will prevent plaque accumulation at the alveolar crest if the implant becomes exposed to the oral environment while maintaining maximal percentage of bone integration [23]. In Buser's review [35], he states that the simplest method of decreasing the risk of peri-implantitis is by utilizing hybrid design implants, as suggested by Tarnow in the 1990s. Most commercially available implants are non-hybrid implants. However, selecting an implant with a smooth, machined coronal surface portion for the patient with a history of periodontitis/peri-implantitis, may help reduce the occurrence of the peri-implantitis.

Implant design has explored the use of variations in macro-architecture. For example, microthreads have been added to the implant collar zone and abutment surfaces. The Laser-Lok™ (Bio-horizon®, Birmingham, AL, USA) surface treatment has two different thread pitches of cell-sized microthreads around the neck, engineered by laser ablation technology. The specific microthreads aim to promote the alignment and attachment of osteoblasts and fibroblasts to the implant surface ($Sa = 1.0\text{--}2.0 \mu m$). The Laser-Lok™ texture on the implant collar has been shown to allow connective tissue fibers to physically attach to the implant collar that is laser-machined onto the dental implant surface itself. This attachment results in a biologic seal surrounding the implant to protect and maintain crestal bone stability [36].

Another example of implant collar modification is the use of anodization and creation of differential surface roughness, to improve surface chemistry and topography with respect to cell/attachment biocompatibility. Nobel Biocare has advanced mucosal compatible smooth non-porous, nanostructured, and anodized surfaces. These implants are designed with surface topography which may promote soft tissue attachment to create a mucosal seal for optimized tissue integration (Xeal™ [$Sa = 0.2 \mu m$] and TiUltra™ [$Sa = 0.5 \mu m$], Nobel Biocare®, Brea, CA, USA). These implants feature a multizone implant surface with a gold colored minimally rough collar that appears after anodization. There is a progressive change in topography as the surface becomes

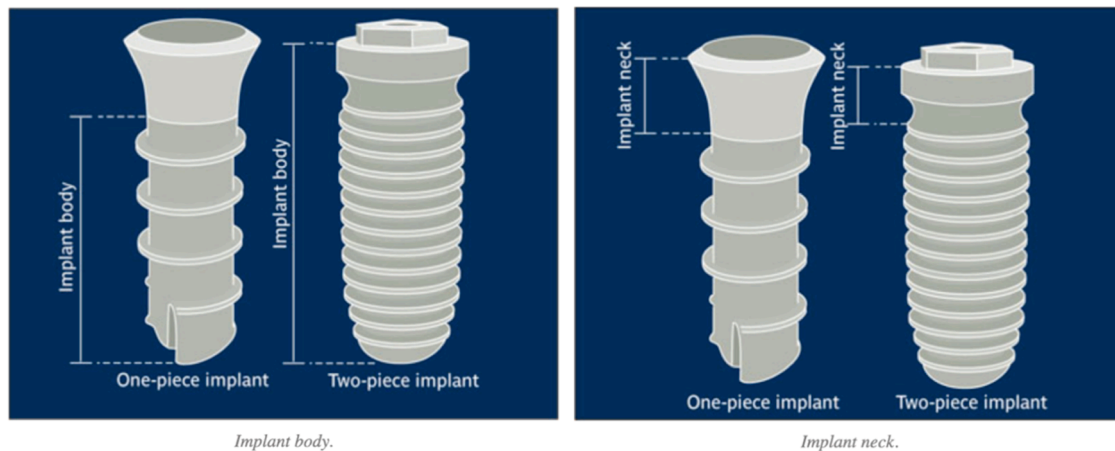


Fig. 2. a & b; One-piece vs. two-piece implants. Diagram highlights the differences in the structure of the implant body (Fig. 2a) in which the implant body and the implant neck are a single unit in one-piece implants vs. two independent parts in the two-piece implant where the prosthetic abutment is separate and attaches to the implant neck (Fig. 2b).



Fig. 3. a-c: Bone-level (a & b) vs tissue-level (c) implants. (a & b) the implant abutment interface (IAI) of bone level implant is positioned at the bone crest, and (c) IAI of tissue-level implant is placed at the level of the soft tissue so the polished portion contacts the soft tissue and not the bone.



Fig. 4. Hybrid design titanium implant with smooth, machined surface of the coronal portion and a plasma-sprayed surface of the apical portion of the implant in one body.

moderately rough and porous approaching the implant apex [37].

3.3. Engineering in surface characteristics

Traditionally, surface modification has been done by surface

topography, hydrophilicity, and chemical composition [38]. Along with the evolution of the implant and abutment designs of implant and abutment materials, there have been numerous surface characteristics studied. The surface properties of implants influence the adhesion and differentiation of cells. Efforts have been made to determine the ideal material or surface characteristics to improve mucosal integration thereby preventing peri-implant disease. Titanium is the current gold standard for its established functionality and biocompatibility with the cells of the peri-implant mucosal tissue [39].

Other materials that may meet the criteria for use in abutment fabrication may include polymer type materials (polymethylmethacrylate [PMMA] and polyetheretherketone [PEEK]) and zirconia. Although zirconia implants and abutments have been used clinically, the superiority and inferiority of the material compared to titanium is controversial [40]. However, studies have shown that the cellular response may not depend on the material, but rather, is more dependant on the surface characteristics [30].

There have been modifications on the morphology/roughness level to increase the mucosal attachment and adding nanotopography targeting to reduce bacterial accumulation and biofilm formation [41]. Early studies determined that epithelial cells preferred machined and polished surfaces as opposed to sand-blasted surfaces [42]. The data also indicated that fibroblasts adhere more readily on roughened versus smooth titanium surfaces than did epithelial cells [43]. Indeed, in our pilot study comparing commercially available zirconia surfaces, the surface roughness played an important role for the growth of human gingival fibroblast/epithelial cell in co-culture system (Fig. 5).

In contrast to the emphasis on surface modification to improve the process of osseointegration, the focus in the transmucosal area has been limited to maintaining healthy soft tissue attachments and the absence

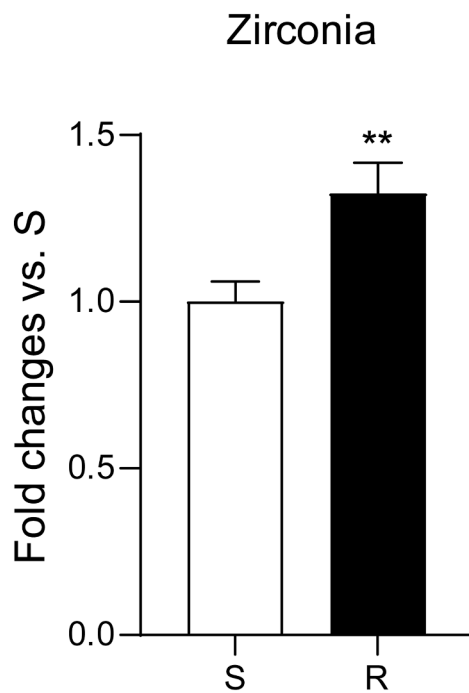


Fig. 5. Amount of cells present on smooth zirconia vs. rough zirconia. Smooth zirconia used as a standard of 1.0. Statistically significant difference found with rough zirconia surface ($P < .005$). (S: smooth zirconia, Ra value = $8.75 \text{ SD} \pm 0.52$. R: rough zirconia, Ra value = $2.56 \text{ SD} \pm 0.49$).

of contamination [29]. Identifying the optimal surface roughness for specific cell adhesion may prove to be foundational in efforts to guide PIM tissue formation. By engineering transmucosal abutments to encourage fibroblast attachment over epithelial cell attachment, the phenomenon of epithelial downgrowth may be prevented. In addition, in regard to bacterial adhesion and prevention of peri-implantitis, it has been recommended that the abutment and/or implant collar be smooth or polished as the forementioned concept of the hybrid implant was re-introduced [44]. More recently, bioactive coating of the surface such as antimicrobials to reduce bacterial formation or controlled drug release to enhance cellular viability has been tested [45]. Application of these technologies within the range of smooth surfaces may reduce bacterial adhesion and serve as a platform for peri-implant mucosal attachment. However, commercial availability is still limited.

4. Conclusion

Peri-implant mucosal tissue is essential for long-term maintenance of dental implants, serving as a biological shield against invasion of bacteria while influencing both functional and esthetic outcomes. The historical evolution from the initial focus on osseointegration to acknowledging mucosal integration underscores its growing importance. Advancements in implant design, implant-abutment interface, and engineered surface characteristics aim to enhance mucosal attachment thus minimizing bacterial adhesion and optimizing tissue integration. The future of implant dentistry relies on improving peri-implant mucosal interfaces by enhancing the material and its surface through engineering for peri-implant mucosal stability. There are varying levels of evidence; some are well-documented historically, while for others, there is little scientific evidence. More research is needed to better understand and promote long-term peri-implant mucosal health.

Declaration of competing interest

The authors declare the following financial interests/personal

relationships which may be considered as potential competing interests:

Authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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References

- [1] Howe M-S, Keys W, Richards D. Long-term (10-year) dental implant survival: a systematic review and sensitivity meta-analysis. *J Dent* 2019;84:9–21. <https://doi.org/10.1016/j.jdent.2019.03.008>.
- [2] Derks J, Tomasi C. Peri-implant health and disease. A systematic review of current epidemiology. *J Clin Periodontol* 2015;42(S16). <https://doi.org/10.1111/jcpe.12334>.
- [3] Yeh H-C, Lu J-J, Chang S-C, Ge M-C. Identification of microbiota in peri-implantitis pockets by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry. *Sci Rep* 2019;9(1):774. <https://doi.org/10.1038/s41598-018-37450-5>.
- [4] Charalampakis G, Belibasakis GN. Microbiome of peri-implant infections: lessons from conventional, molecular and metagenomic analyses. *Virulence* 2015;6(3): 183–7. <https://doi.org/10.4161/21505594.2014.980661>.
- [5] Belibasakis GN, Charalampakis G, Bostanci N, Stadlinger B. Peri-implant infections of oral biofilm etiology. In: Donelli G, editor. *Biofilm-based Healthcare-associated Infections*. Donelli G, editor. *Advances in experimental medicine and biology*, vol. 830. Cham: Springer International Publishing; 2015. p. 69–84. https://doi.org/10.1007/978-3-319-11038-7_4.
- [6] Gargiulo AW, Wentz FM, Orban B. Dimensions and relations of the dentogingival junction in humans. *J Periodontol* 1961;32:261–7. <https://doi.org/10.1902/jop.1961.32.3.261>.
- [7] Atsuta I, et al. Soft tissue sealing around dental implants based on histological interpretation. *J Prosthodont Res* 2016;60(1):3–11. <https://doi.org/10.1016/j.jpor.2015.07.001>.
- [8] Berglundh T, Lindhe J. Dimension of the periimplant mucosa: biological width revisited. *J Clin Periodontol* 1996;23(10):971–3. <https://doi.org/10.1111/j.1600-051X.1996.tb00520.x>.
- [9] Berglundh T, 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(S1). <https://doi.org/10.1002/JPER.17-0739>.
- [10] Miciak JJ, et al. Fast skeletal muscle troponin activator in the dy2J muscular dystrophy model: tirasemtiv in the dy2J Mouse. *Muscle Nerve* 2013;48(2):279–85. <https://doi.org/10.1002/mus.23848>.
- [11] 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. <https://doi.org/10.1111/j.1600-0501.2005.01193.x>.
- [12] Albrektsson T. The long-term efficacy of currently used dental implants: a review and proposed criteria of success. 1986.
- [13] Tarnow D, et al. vertical distance from the crest of bone to the height of the interproximal papilla between adjacent implants. *J Periodontol* 2003;74(12): 1785–8. <https://doi.org/10.1902/jop.2003.74.12.1785>.
- [14] Seyssens L, Eghbali A, Cosyn J. A 10-year prospective study on single immediate implants. *J Clin Periodontol* 2020;47(10):1248–58. <https://doi.org/10.1111/jcpe.13352>.
- [15] R.A. Levine et al., “10 keys for successful esthetic-zone single immediate implants,” vol. 38, no. 4, 2017.
- [16] Brånemark P-I, Breine U, Adell R, Hansson BO, Lindström J, Ohlsson Å. Intra-osseous anchorage of dental prostheses: I. *Experimental studies*. *Scand J Plast Reconstr Surg* 1969;3(2):81–100. <https://doi.org/10.3109/02844316909036699>.
- [17] 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;9:15–25. [https://doi.org/10.1016/S0301-0503\(81\)80007-0](https://doi.org/10.1016/S0301-0503(81)80007-0).
- [18] 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. <https://doi.org/10.1111/prd.12185>.
- [19] Becker W, et al. A prospective multicenter clinical trial comparing one- and two-stage titanium screw-shaped fixtures with one-stage plasma-sprayed solid-screw fixtures. *Clin Implant Dent Relat Res* 2000;2(3):159–65. <https://doi.org/10.1111/j.1708-8208.2000.tb00007.x>.
- [20] De Boever AL, Quirynen M, Coucke W, Theuniers G, De Boever JA. Clinical and radiographic study of implant treatment outcome in periodontally susceptible and non-susceptible patients: a prospective long-term study. *Clin Oral Implants Res* 2009;20(12):1341–50. <https://doi.org/10.1111/j.1600-0501.2009.01750.x>.

- [21] Brånemark P-I, Zarb G, Albrektsson T. Tissue-integrated prostheses: osseointegration in clinical dentistry. Chicago: Quintessence Publishing; 1985.
- [22] Adell R, Lekholm U, Rockler B, Brånemark P-I. A 15-year study of osseointegrated implants in the treatment of the edentulous jaw. *Int J Oral Surg* 1981;10(6): 387–416. [https://doi.org/10.1016/S0300-9785\(81\)80077-4](https://doi.org/10.1016/S0300-9785(81)80077-4).
- [23] Tarnow D. Dental implants in periodontal care. *Curr Opin Periodontol* 1993; 157–62.
- [24] Lindhe J, Berglundh T, Ericsson I, Liljenberg B, Marinello C. Experimental breakdown of peri-implant and periodontal tissues. A study in the beagle dog. *Clin Oral Implants Res* 1992;3(1):9–16. <https://doi.org/10.1034/j.1600-0501.1992.030102.x>.
- [25] M. Nossowitz, M. Teale, S. Mathes, A. Venturato, and A. Gasser, “Evaluation of anodized surfaces designed for improved soft tissue integration”.
- [26] Scarano A, et al. A 16-year study of the microgap between 272 human titanium implants and their abutments. *J Oral Implantol* 2005;31(6):269–75. <https://doi.org/10.1563/753.1>.
- [27] Moon I-S, 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. <https://doi.org/10.1034/j.1600-051X.1999.261005.x>.
- [28] Jones AA, Cochran DL. Consequences of implant design. *Dent Clin North Am* 2006; 50(3):339–60. <https://doi.org/10.1016/j.cden.2006.03.008>.
- [29] Kim J-J, Lee J-H, Kim J-C, Lee J-B, Yeo I-SL. Biological responses to the transitional area of dental implants: material- and structure-dependent responses of peri-implant tissue to abutments. *Materials* 2019;13(1):72. <https://doi.org/10.3390/ma13010072>.
- [30] Saito H, Aichelmann-Reidy MB, Oates TW. Advances in implant therapy in North America: improved outcomes and application in the compromised dentition. *Periodontol* 2000 2020;82(1):225–37. <https://doi.org/10.1111/prd.12319>.
- [31] Caricasulo R, Malchiodi L, Ghensi P, Fantozzi G, Cucchi A. The influence of implant-abutment connection to peri-implant bone loss: a systematic review and meta-analysis. *Clin Implant Dent Relat Res* 2018;20(4):653–64. <https://doi.org/10.1111/cid.12620>.
- [32] Camps-Font O, Rubianes-Porta L, Valmaseda-Castellón E, Jung RE, Gay-Escoda C, Figueiredo R. Comparison of external, internal flat-to-flat, and conical implant abutment connections for implant-supported prostheses: a systematic review and network meta-analysis of randomized clinical trials. *J Prosthet Dent* 2023;130(3): 327–40. <https://doi.org/10.1016/j.prosdent.2021.09.029>.
- [33] Lazzara RJ, Porter SS. Platform switching: a new concept in implant dentistry for controlling postrestorative crestal bone levels. *Int J Periodont Restorat Dent* 2006; 26(1):9–17.
- [34] Palombo D, Rahmati M, Vignoletti F, Sanz-Esporrin J, Haugen HJ, Sanz M. Hard and soft tissue healing around implants with a modified implant neck configuration: an experimental in vivo preclinical investigation. *Clin Oral Implants Res* 2021;32(9):1127–41. <https://doi.org/10.1111/clr.13812>.
- [35] Buser D. Hybrid Design Implants: is this the future in implant dentistry? *Acad News* 2022;33(1):6–7.
- [36] Shapoff CA, Lahey B, Wasserlauf PA, Kim DM. Radiographic analysis of crestal bone levels around Laser-Lok collar dental implants. *Int J Periodontics Restorative Dent* 2010;30(2):129–37.
- [37] Milleret V, Lienemann PS, Gasser A, Bauer S, Ehrbar M, Wennerberg A. Rational design and in vitro characterization of novel dental implant and abutment surfaces for balancing clinical and biological needs. *Clin Implant Dent Relat Res* 2019;21 (S1):15–24. <https://doi.org/10.1111/cid.12736>.
- [38] Kim HS, Kumbar SG, Nukavarapu SP. Biomaterial-directed cell behavior for tissue engineering. *Curr Opin Biomed Eng* 2021;17:100260. <https://doi.org/10.1016/j.cobme.2020.100260>.
- [39] Abrahamsson I, Berglundh T, Glantz P-O, Lindhe J. The mucosal attachment at different abutments: an experimental study in dogs. *J Clin Periodontol* 1998;25(9): 721–7. <https://doi.org/10.1111/j.1600-051X.1998.tb02513.x>.
- [40] Linkevicius T, Vaitelis J. The effect of zirconia or titanium as abutment material on soft peri-implant tissues: a systematic review and meta-analysis. *Clin Oral Implants Res* 2015;26(S11):139–47. <https://doi.org/10.1111/clr.12631>.
- [41] Gröner-Schreiber B, Herzog M, Hedderich J, Dück A, Hannig M, Griepentrog M. Focal adhesion contact formation by fibroblasts cultured on surface-modified dental implants: an in vitro study. *Clin Oral Implants Res* 2006;17(6):736–45. <https://doi.org/10.1111/j.1600-0501.2006.01277.x>.
- [42] Hormia M, Könönen M, Kivilahti J, Virtanen I. Immunolocalization of proteins specific for *adhaerens* junctions in human gingival epithelial cells grown on differently processed titanium surfaces. *J Periodontol Res* 1991;26(6):491–7. <https://doi.org/10.1111/j.1600-0765.1991.tb01800.x>.
- [43] Hormia M, Könönen M. Immunolocalization of fibronectin and vitronectin receptors in human gingival fibroblasts spreading on titanium surfaces. *J Periodontol Res* 1994;29(2):146–52. <https://doi.org/10.1111/j.1600-0765.1994.tb01103.x>.
- [44] Nascimento CD, Pita MS, Fernandes FHNC, Pedrazzi V, De Albuquerque Junior RF, Ribeiro RF. Bacterial adhesion on the titanium and zirconia abutment surfaces. *Clin Oral Implants Res* 2014;25(3):337–43. <https://doi.org/10.1111/clr.12093>.
- [45] Xing R, et al. Antibacterial effect of doxycycline-coated dental abutment surfaces. *Biomed Mater* 2015;10(5):055003. <https://doi.org/10.1088/1748-6041/10/5/055003>.