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IN THIS ISSUE

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Contents

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Editor's Notes

Welcome

The last 6 months has been truly exciting as we were slowly getting back to the new 'Norm'. There were multiple face-to-face conferences, and it was a surreal experience to meet and see friends and colleagues once again in person. The recent joint conference in Sydney between the ASP and AOS was a great success as it brought us together to share new scientific knowledge and clinical practices in periodontology and implant dentistry. I'd like to congratulate and thank Dr Eugene Foo, Dr Rajiv Verma and the organising committee members for putting together such a wonderful meeting despite all the COVID related difficulties. I hope to see more of those joint meetings between two societies in the future.

EuroPerio 10 in Copenhagen 2022 was another major highlighted event. Even after the unprecedent one year of postponement, the congress was a huge success, attracting more than 7000 participants from all around the world. Australian presence was well noted; more than 150 Australians attended the congress, and for the first time the ASP was invited to the Opening Flag ceremony as an international associate society of the European Federation of Periodontology.

This time around, the journal has received multiple manuscript submissions on a wide range of topics in periodontology and implant dentistry. I am grateful for your interests and continuous support for the journal.

The first article by *Dr Stella Lee* is a review on aetiology and management of halitosis. Halitosis is a common dental problem, causing many different negative psychosocial impacts on one's quality of life. This review explores the literature to give us an overview of the

current understanding of halitosis and its management strategies, particularly in the general dental setting.

In recent years, there has been a lot of attention on quality of peri-implant soft tissue mucosa. What is the role of keratinised mucosa? Is it necessary to have it for a long-term success? If so, how much of the keratinised tissue is sufficient? The second article by *Hamza Masood* examines and summarises the relevance of peri-implant keratinised mucosa and its significance in maintaining peri-implant health.

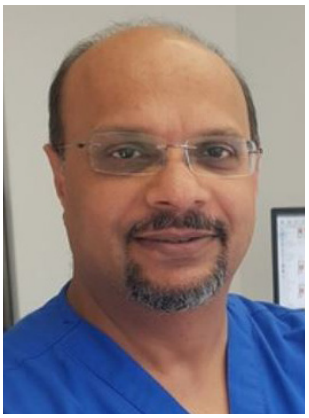
Intraoral scanning and digital dentistry have surged in popularity over the past few years. However, there has not been clear evidence to support the use of digital impression techniques over conventional impressions. The last article by *Dr Casey Walsh* provides a review on the role and importance of using scan bodies in the CAD/CAM implant restoration workflow and the effects of scan body design on data acquisition and processing accuracy.

I hope you enjoy the issue.

Regards,

A/Prof Ryan Lee
Editor-in-chief

President's Notes



Welcome to this edition of AJPID Journal and my final letter as President. It has been an unprecedented journey with more time spent in lock downs than in face-to-face interaction with our colleagues. It was a welcome change to have our biennial conference in Sydney as a face-to-face meeting rather than a hybrid or a zoom meeting.

A big thank you to our organizing committee members, Dr Eugene Foo (President AOS), his team and Kayla for the excellent synergy in organizing a successful convention in Sydney. It was the first Joint Conference of ASP & AOS and the feedback has been very positive. It was wonderful to meet all our colleagues after a long Covid induced hiatus and to interact with the industry.

I would like to thank all the members of the Federal Council for their support during my term as President, a special thanks to Dr Robert Fell for his role of Secretary/Treasurer and to our secretariat Kayla for all her support and contribution in making this Society run efficiently.

The Ray Williams Awards Committee awarded Prof Saso Ivanoski for the triennial period 2018-2020 and Prof Ivan Darby for the period 2015-2017 this esteemed award for eminence in research in Australia.

I would like to welcome A/Prof Ryan Lee to the role as President of our Society. It has been an honour to serve the society and I wish Ryan, his team in Queensland and the Council all the very best and success for the coming period.

A/Prof Ryan Lee and his editorial team are doing excellent work in providing us with scientific articles of such a high calibre in our journal.

Hope you all enjoy this edition of AJPID.

Best Wishes

Dr Rajiv Verma
ASP Federal President

President's Notes

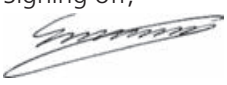
How time flies! The joint 2022 AOS & ASP Conference in Sydney during 17th to 20th of August has come and gone with excellent feedback from attendees regarding the high level of presentations and speakers who delivered them. Our speakers had a focus on research based data and there is always more delivered by those involved in the research than can be gleaned from the paper alone. AOS continues to encourage submissions from clinicians whose desire to "get to the bottom of things" could benefit their colleagues. As there was a variety of clinical and academic research presented, one can never know where those first steps could lead them!

Both societies acknowledged the efforts of the previous journal editor – Professor Ivan Darby – at the AOS & ASP Conference Gala Dinner, with our new editor A/Prof Ryan Lee handing over a keepsake to him. The Gala dinner was also an opportunity to acknowledge the incredible contribution of two members of the AOS Committee - A/Prof George Pal and Dr Bruce Munroe – who have provided decades of commitment to AOS with A/Prof George Pal being one of the members involved in its inception and Dr Bruce Munroe being our very long-term Treasurer. Thanks should also go to our Secretary Dr Cecilia So and our Organizing Committee counterparts in Dr Rajiv Verma and his ASP representatives. Both groups helped to steer what would become a successful face-to-face conference. Special thanks should go to our Scientific Committee comprising A/Prof Dale Howes, Dr Richard Vickers and Dr Kwan Yat Zee who did a fantastic effort to secure our highly regarded speakers and manage all the "detours" along the way. I would also like to thank the industry involved for their support and last but not least our Secretariat Kayla, without whom our society could not function effectively.

Dr Bruce Munroe, Dr Eugene Foo, A/Prof George Pal & Dr Cecilia So

The conference also marks the end of my tenure as the AOS Federal President with the baton handed over to Victoria where Dr Angelos Sourial will take over as the new Federal President. Global challenges continue to impact the environment that clinicians work in as well as the conference scene. I look forward to the content that is to come in future journals as well as the next conference delivered by our Victorian counterparts.

Signing off,



Dr Eugene Foo
AOS Federal President

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The Australian Journal of Periodontology
and Implant Dentistry

Aetiology and Management of Halitosis

Stella Lee
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Abstract:

Background: Halitosis, also known as oral malodour or bad breath, is a common condition experienced by most individuals at some point in their life. Although mostly transient and temporary in nature, it can be persistent with underlying intraoral or extraoral causes and can have a negative psychosocial impact on quality of life. Hence, it is important for dental practitioners to understand possible aetiological factors and management directions for patients who present with concerns of halitosis.

Aim: To review aetiology and management of halitosis by evaluating the current literature with relevance to general dental practice.

Method: MEDLINE (Ovid) and Google Scholar searches were performed for articles published in the English language. Key words containing 'halitosis', 'oral malodour', 'breath odour', 'bad breath', 'halitosis aetiology', 'halitosis treatment' and 'halitosis management' were used to identify relevant articles, as well as manual searching of references from previous publications.

Results: Halitosis is a complex, multifactorial phenomenon. Transient halitosis is often related to momentary behaviours such as certain food intake, smoking and alcohol. Pathological halitosis has predominantly intraoral aetiology, ranging from periodontal disease, dry mouth, tongue coating to various infections in the oral cavity. However, there is also a wide array of extraoral or systemic causes of halitosis. Psychogenic or delusional halitosis is possible when a clear measure of halitosis cannot be established. Management of halitosis is based on correct identification of specific aetiological factors and appropriate, individualised cause-related

Introduction

Halitosis is the presence of any disagreeable or unpleasant breath odour of expired air, independent of its origin. The term stems from the Latin word *halitus* for breath or exhalation and the Greek suffix *osis*, meaning abnormal. Other phrases such as oral malodour, fetor oris, fetor oralis, fetor ex ore and bad breath are also used to describe the same condition.

It is anticipated that over 70% of Australians have experienced halitosis at some point in their life. (1) Prevalence of halitosis is estimated to range between 30-60%. (2-5) The exact prevalence is difficult to calculate due to deficient epidemiological data, lack of standardised assessments and subjective nature of reporting. (6, 7)

Dental practitioners commonly encounter patients presenting with concerns of bad breath, either self-perceived or mentioned to them by others. (8, 9) Current literature shows that up to 90% of halitosis cases originate from the oral cavity. (10) Absence of intraoral causes suggests possibility of more serious, underlying systemic conditions that may require urgent referral to a medical colleague. (11, 12)

There is a growing number of studies demonstrating the negative psychosocial impact of halitosis, with associated embarrassment and decreased self-confidence compromising social interactions of the affected individuals. (9, 13-15) Hence, it is imperative that dental practitioners understand the possible causes halitosis and treat these patients appropriately.

This paper aims to review the current literature on aetiology and management of halitosis, particularly in the general dental setting.

Aetiology of halitosis

Genuine halitosis

Transient halitosis

Transient halitosis is self-limiting and rarely concerning. Malodour occurs through putrefactive processes in the oral

cavity, especially the tongue, resulting in volatile compounds. (16)

Mild oral malodour is commonly noted after waking up in the morning. This physiological halitosis, or "morning breath", is thought to be related to reduced salivary flow following a circadian rhythm, lack of oral movement and self-cleansing overnight and varying effectiveness of oral hygiene measures before sleep. (17-19)

Certain volatile food items such as garlic, onion and spices are accompanied by characteristic odours, which resolve upon passing through the body. (17, 20) 'Low carbohydrate, high protein' ketogenic diet may be linked to distinct malodour due to excessive ketone production. (21). Smoking and alcohol may contribute to uniquely identifiable breath for a few hours. Alcohol also has a dehydrating effect. (17)

Dry mouth is an indirect cause of oral malodour and can be related to mouth breathing, snoring, dehydration and side effects of many medications. (22-24)

Pathologic halitosis

Intraoral causes

While halitosis has dynamic, multifactorial aetiology, around 90% of the cases originate from the oral cavity. (10)

Periodontal disease, tongue coating and poor oral hygiene practices are the most common causes of intraoral halitosis according to a recent systematic review. (25) Other intraoral causes include xerostomia, salivary gland hypofunction, caries, pericoronitis, dental abscess, mucosal ulcers, oral candidiasis and oral cancer. (20, 22, 26, 27)

Oral malodour is mainly due to microbial degradation of sulfur-containing and non-sulfur containing amino acids derived from proteins in exfoliated epithelium, plaque, saliva, blood and tongue coatings. (28, 29) Putrefaction of sulfur-containing amino acids, such as cysteine and methionine, creates volatile sulfur compounds (VSC), which are key ingredients of bad breath intraorally. Numerous studies have indicated periodontal disease and tongue coatings as major sources of VSC. (30-32).

The exact mechanism of periodontal disease leading to halitosis is complex and still unclear. (32) Gram-negative proteolytic anaerobic bacteria involved in periodontal pathogenesis allow activation of enzymes involved in the putrefaction process. Most predominant compounds produced are hydrogen sulfide (H_2S) and methyl mercaptan (CH_3SH), making up 90% of VSC. A deep periodontal pocket is a prime environment for production of VSC due to presence of a large variety of subgingival anaerobic bacterial species and substrate availability. (16, 22, 33, 34)

Examination of periodontal pathogens through polymerase chain reactions of saliva from halitosis patients has shown that presence of *Tannerella forsythia, Provotella intermedia* and *Porphyromonas gingivalis* influences the production of VSC. In particular, *Tannerella forsythia* is strongly associated with higher concentrations of VSC in the oral cavity; *Porphyromonas gingivalis* is related to higher levels of methyl mercaptan production. (35)

A study by Morita and Wang (2001) measured volatile VSC levels in seventy systemically healthy, periodontal patients with varying levels of radiographic bone loss. The results indicated that sulcular sulfide levels significantly increased with the severity of periodontal disease. Untreated patients showed higher sulcular sulfide levels compared to stable patients enrolled in maintenance. A follow-up study demonstrated that oral malodour is significantly associated with the volume of tongue coating and percentage of periodontal sites with bleeding on probing.

The tongue is a large muscle in the oral cavity and displays an extensive surface area, enhanced by papillae and fissures,

Abstract: *(continued)*

therapy. It may be as simple as limiting transient causes of halitosis and implementing effective oral hygiene measures, or may require more complex, multidisciplinary involvement.

Conclusion: While extensive research has been conducted on halitosis, the strength of the studies on diagnosing and managing the condition is limited due variations in study design, oral health and medical status of the population, method of measuring halitosis, interventions and observation periods. Further research may be directed towards measure of halitosis with greater sensitivity and specificity, as well as practicality in the clinical setting. Additional studies are required on long-term interventional outcomes of halitosis management.

Keywords: Halitosis, oral malodour, mouth odour, bad breath, fetor oris, halitosis aetiology, halitosis management

that can harbour large amounts of diverse microorganisms. Tongue coating in the posterior dorsum has been strongly implied as a primary source of intraoral halitosis, in both periodontally affected and healthy individuals. Greater counts of *Porphyromonas gingivalis* have also been found in this location. (22, 34, 36)

Miyazaki et al. (1995) suggested that intraoral halitosis is mainly caused by tongue coating in younger individuals and by a combination of both periodontal disease and tongue coating in an older cohort.

Reduced resting salivary flow and higher viscosity saliva have been associated with diminished cleansing, increased deposition of food debris and dental plaque resulting in oral malodour. (25, 37)

Although many bacterial pathogens have been identified to produce noxious-smelling compounds populating the oral cavity, no definitive link has been established between halitosis and bacterial infections, suggesting very complex interactions of microbes and the oral environment. (25, 38)

Extraoral causes

Only 10% of halitosis has extraoral aetiology. Many cases are a manifestation of an underlying systemic medical condition and cannot be treated with dental interventions alone. (27) (22)

Unlike volatile sulfur compounds dominating intraoral halitosis, extraoral halitosis is mostly influenced by non-sulfuric volatile organic compounds. These compounds circulate in the bloodstream, get transported to the lungs, diffuse across the pulmonary alveolar membrane and get excreted into exhaled air. (39)

Possible causes of extraoral halitosis include but are not limited to:

(12, 21, 22, 25, 40)

- Acute cold or flu
- ENT conditions: post-nasal drip, nasal rhinitis, throat infections, tonsilitis, tonsil stones, sinusitis
- Respiratory disease: respiratory tract infections such as pneumonia and bronchitis, bronchiectasis, chronic lung infection with "acidic" or "cheesy" odour
- Liver disease: liver cirrhosis, liver failure
- Kidney disease: kidney insufficiency, kidney failure with ammonia-like odour, also known as "uremic fetor"
- Gastrointestinal disease: gastro-oesophageal reflux disease, oesophagitis, *Helicobacter pylori* infection, irritable bowel syndrome, Crohn's disease, ulcerative colitis
- Uncontrolled diabetes, ketoacidosis with distinct "sweet" odour
- Trimethylaminuria
- Menstruation
- Undiagnosed infection or malignancy
- Certain medications such as dimethyl sulfoxide, cysteamine, disulfiram, penicillamine

Psychogenic halitosis

Psychogenic or delusional halitosis occurs in a small number of individuals, who have heightened fear of having a bad breath that is offensive to others, in the absence of such odour. It can affect up to 1% of the adult population. (40, 41)

In a study of over two thousand patients presenting to a halitosis clinic, up to 16% of the participants had no obvious dental or medical causes related to their complaint of bad breath. Halitosis was not identifiable by the examiners. (27). Such belief of oral malodour in its absence is called pseudo-halitosis. If there is persistent fear of ongoing oral malodour following treatment of genuine halitosis or pseudo-halitosis, it is termed halitophobia. (33, 42) A German study also showed a particularly high prevalence of combined pseudo-halitosis and halitophobia of 27%. (43)

Management of halitosis

Measuring halitosis

Organoleptic measurement remains the gold standard of assessing halitosis. (36, 39, 44) It involves directly smelling exhaled air from the mouth and nose at a distance and comparing the two. This method has issues of being subjective, having to rely on skills of the examiner. Conditions for measurement are strict, including avoiding systemic antibiotics for three weeks, refraining from intake of garlic, onion and spicy foods for 48 hours and not using scented cosmetics for 24 hours prior to the planned assessment. (21, 40) The process can be socially uncomfortable for both parties involved. Moreover, such assessment may be inappropriate during the Covid-19 pandemic with implications of potential respiratory viral transmission.

Table 1: Organoleptic scoring scale (42)

Score	Category	Description
0	Absence of odour	Odour cannot be detected
1	Questionable odour	Odour is detectable, although the examiner could not recognise it as malodour
2	Slight malodour	Odour is deemed to exceed the threshold of malodour recognition
3	Moderate malodour	Malodour is definitely detected
4	Strong malodour	Strong malodour is detected, but can be tolerated by examiner
5	Severe malodour	Overwhelming malodour is detected and cannot be tolerated by examiner (examiner instinctively averts the nose)

Gas chromatography offers a more objective measurement of halitosis. Using an electrochemical meter such as Halimeter and Breathron®, volatile sulfur compound levels are measured in expired air samples. Despite superior objectivity and sensitivity, it does not differentiate between different sulfuric compounds and completely ignores the presence of non-sulfuric volatile compounds, which may be related to extraoral halitosis. Moreover, these machines are expensive and not practical to implement in a general dental practice. (39)

Sulfide monitors are less costly and simple to use, however, have similar issues of lacking sensitivity for certain sulfuric compounds and failing to detect non-sulfuric volatile organic compounds.

BANA test detects presence of an enzyme found in proteolytic gram-negative anaerobic bacteria that breaks down the benzoyl-DL-arginine-α-naphthylamide (BANA), a synthetic substrate of trypsin, which produces a blue compound. Although useful in detecting periodontal disease activity, the relationship between the BANA test and sulfide monitor measurements are inconsistent. (45, 46)

Cause-related management in dental practice

Successful management of halitosis in general dental practice relies upon detailed medical and dental history, comprehensive examination, and accurate identification of intraoral aetiological factors to facilitate effective cause-related therapy. (41)

A thorough history of halitosis, its onset, duration, severity, impact on everyday life including personal and social implications should be questioned. Examination includes clinical and radiographic assessments and any further special investigations such as diet analysis. Clinical evaluations of the patient's oral hygiene, tongue coating, periodontal status, caries and presence of other plaque-retentive factors such as overhanging or defective restorations are documented. (36)

Treatment needs for halitosis can be categorised into 5 groups.

Table 2: Treatment needs for breath malodour *Adapted from Miyazaki et al. (1999)*

Category	Description	Recommended management
TN-1	Transient halitosis	Explanation of halitosis and oral hygiene instructions (support and reinforcement of the patient's own self-care regime to further improve of their oral hygiene)
TN-2	Pathological halitosis with intraoral aetiology	Oral prophylaxis, professional cleaning and treatment of oral diseases, especially periodontal diseases
TN-3	Pathologic halitosis with extraoral aetiology	Referral to a physician or medical specialist
TN-4	Pseudo-halitosis	Explanation of examination data, further professional instruction, education and reassurance
TN-5	Halitophobia	Referral to a clinical psychologist, psychiatrist or other psychological specialist

Transient halitosis can improve by reducing the causes such as consumption of volatile, malodorous food items, alcohol and smoking. Physiological oral malodour is most effectively resolved by breakfast intake and tongue brushing. (47) Dry mouth can be alleviated by staying hydrated, chewing sugar-free gum to stimulate saliva production and using saliva lubricants or substitutes as appropriate. However, possible systemic reasons for hyposalivation need to be considered, such as Sjogren's syndrome, salivary gland hypofunction and medication-related xerostomia.

Treatment approaches for pathological halitosis of intraoral origin may include mechanical reduction of intraoral nutrients, substrates and microorganisms; chemical reduction of the intraoral microbial load; masking strategy and conversion of volatile sulfur compounds. (32)

Mechanical reduction of intraoral microbial load

Oral hygiene instructions should be detailed, effective and customised for individual patients to achieve optimal mechanical plaque control. Correct toothbrushing and interdental cleaning techniques are reviewed, including use of appropriate cleaning tools such as floss and interdental brushes. (32)

If periodontal disease is confirmed upon screening examination, complete periodontal analysis should be conducted to establish a periodontal diagnosis, followed by active non-surgical treatment involving scaling and root debridement. Plaque-retentive factors need to be modified, such as restoring carious lesions, polishing overhanging restorations and replacing defective restorations. Other intraoral pathology is identified and managed, such as root canal therapy or extraction of teeth with active endodontic lesions.

A Nigerian study has reported over 90% successful outcomes following routine dental treatment, regardless of the type of halitosis. (48) In a Taiwanese study, statistically significant improvements in organoleptic scores were found after conservative treatment involving tongue scraping, non-surgical periodontal treatment and oral hygiene instructions. (31)

Cleaning the dorsum of the tongue helps to disrupt the tongue biofilm and reduce the available nutrients for microbes, thereby decreasing the production of volatile compounds. Combined toothbrushing and tongue cleaning have been shown to be effective at lowering VSC compared to toothbrushing alone. (47, 49) A soft manual toothbrush may be used to sweep between the papillae. (50) If the tongue coating is thick, a tongue scraper is preferred for more effective reduction of VSC. (51) However, the evidence comparing different modes of tongue cleaning is weak. (3) In fact, evidence for long-term benefit of any halitosis intervention is weak, although tongue cleaning appears to be the best practice at this stage. (46)

Chemical reduction of the intraoral microbial load

Use of triclosan, zinc and sodium bicarbonate in toothpaste has shown improvement in reducing VSC levels and promoting freshness of breath as measured by a Halimeter and organoleptic scoring. (52-54) Triclosan is a broad-spectrum antibacterial agent that has been found to be effect against most oral bacteria and has good compatibility with other compounds used for home oral care. However, the anti-VSC effect of triclosan seems to be strongly dependent on the solubilising agents, as it is not maintained when oils, oily substances and uncharged detergents are used as solubilisers. A clinical study by Young et al. (2002) demonstrated rinsing with triclosan solubilised in sodium lauryl sulphate, propylene glycol and water to give a marked and long-lasting anti-VSC effect. Metal ions such as zinc, sodium and copper, owing to their positive charge, can bind to negatively loaded sulfur radicals and oxidise the thiol groups to retard bacterial growth and reduce the expression of VSC. Zinc has low toxicity, is non-cumulative, gives no visible discolouration and therefore is one of the most commonly used ingredients in anti-halitosis products. Schmidt and Tarbet (1978) reported that a rinse containing zinc chloride was remarkably more effective than a saline rinse or no treatment in reducing the levels of both VSC (80% reduction) and organoleptic scores (40% reduction) over 3 hours. Waler compared different concentrations of zinc in a chewing gum and found that retention of chewing gum containing 2mg of zinc acetate in the mouth for 5 minutes resulted in an immediate reduction in the VSC levels by up to 45% (Waler 1997b). Sodium bicarbonate in dentifrices has been shown to have a significant odour-reducing effect for periods up to three hours. (55)

Incorporation of stannous fluoride in dentifrices has also shown positive outcomes in terms of reducing organoleptic scores and VSC levels over a 8-hour period. (56) A superior short-term and overnight benefit of a stannous-containing dentifrice compared with a control dentifrice on morning bad breath has been highlighted in a meta-analysis by Feng et al. (2010)

Positive short-term benefits have been reported with chlorhexidine mouthrinse, a broad-spectrum antimicrobial agent. It is well known for inhibiting microbial action and reducing accumulation of plaque, most well-studied in managing gingivitis. Numerous studies have shown significantly reduced VSC production with chlorhexidine use. (31, 57) 0.2% chlorhexidine mouthrinse has demonstrated reduced microbial levels and organoleptic scoring even when lacking mechanical plaque control measures. (58) However, various side effects documented such as teeth staining, mucosal desquamation, increased calculus deposition and altered taste perception mean its use is limited. (59)

A randomised, double-blinded clinical trial compared the efficacy of four different mouthrinses in oral malodour measurements after 4 weeks. Cetylpyridinium chloride-containing formulation was shown to be the most effective in improving organoleptic scores, reducing oral malodour

from baseline values after 4 weeks of daily use. Essential oil and chlorine dioxide mouthrinses showed improved malodour within 4 hours after a single use, however this effect was negligible after 4 weeks when compared to baseline. (60) Another clinical study compared two groups of halitosis subjects, one that employed toothbrushing alone with fluoride toothpaste, and the other with adjunctive use of a 0.075% cetylpyridinium chloride mouthrinse on top of toothbrushing. Both groups had significantly improved organoleptic scores, however, the level of volatile sulfur compounds measured via gas chromatography only improved in the latter group at 3 weeks. (61)

A randomised controlled trial compared the effect of toothbrushing and mouthwash versus toothbrushing and tongue cleaning on halitosis over five weeks. No significant reduction in oral malodour was identified after one week of toothbrushing alone in both groups. However, significant reductions in VSC levels were shown from week two to week four after adding mouthwash and tongue cleaning; the tongue cleaning group showed superior results. The most significant reduction in VSC levels occurred when all three oral hygiene measures were implemented. (62)

A Cochrane review conducted in 2019 assessed the effects of various interventions used to control intraoral halitosis specifically. Overall, quality of evidence was low to very low, including the studies mentioned above, due to risk of bias, small sample size and inconsistent study designs. (63)

Masking strategy

Odour masking agents are commonly used by the public to cover symptoms of halitosis. Products come in many forms such as toothpaste flavouring, mint tablets, flavoured strips, chewing gum and mouth spray. They are generally affordable, easily accessible and widely utilised due to immediate effect of covering malodour; although underlying aetiological factors are not being managed, most of these items produce short-lived effect up to three hours. (46, 64)

Multidisciplinary management

Referral to dental specialists

If intraoral causes of halitosis cannot be adequately managed in a general dental setting, referral to dental specialists may be warranted.

Periodontic specialist referral is suitable for periodontitis that is unstable following conservative periodontal treatment. (65) Necrotising periodontal diseases can be difficult to manage, often accompanied by rapid tissue destruction, pain and breath malodour. (66) With global growth of Covid-19 cases, rising prevalence of acute periodontal conditions such as necrotising gingivitis has been projected, thought to be due to increased co-infections with pathogenic oral bacterial species. (67, 68)

Referral to an oral medicine specialist may be appropriate for patients with immune compromise, severe symptoms of dry mouth, mucosal conditions and/or persistent oral candidiasis, all of which can be contributory towards halitosis. In addition to symptomatic relief, underlying systemic conditions and effects of medications can be further examined. (24, 69-71)

Referral to medical practitioners

When dental strategies are exhausted and ineffective in managing halitosis, referral to medical practitioners is advised. (20) A general medical practitioner can conduct a detailed, methodical assessment to rule out possible underlying systemic reasons behind halitosis. Based on suspected aetiology, further diagnostic testing or referral to other medical specialties can be arranged.

Psychogenic halitosis is challenging to manage, as simple reassurance following exclusionary dental and medical examinations may not be sufficient to improve the condition. Detailed explanations and ongoing counselling may be required. Symptoms of anxiety and depression are often associated with pseudo-halitosis and halitophobia. (72). In such cases, referral to a psychiatrist or clinical psychologist is warranted. (17, 73)

Collaborative care model

A current Australian research project through the eviDENT Foundation involved interviewing the general public, general dental practitioners, general medical practitioners and community pharmacists regarding their experience in managing halitosis and confirmed the lack of collaboration among the three professions. (1) Each profession plays a unique role in diagnosing halitosis, reinforcing basic management strategies and facilitating referral to each other when appropriate for more specialised investigations and care. (74, 75)

Conclusion

Majority of halitosis stems from the oral cavity. Therefore, dental practitioners play a truly relevant and valuable role in helping patients presenting with concerns of halitosis. Positive

outcomes not only establish optimal oral health and effective oral hygiene habits but may also boost psychosocial profile of affected patients and increase their self-esteem. Effective treatment relies on detailed medical and dental history and comprehensive clinical examinations to identify specific causative factors for individually tailored management.

Conservative dental management involves establishing meticulous home plaque control regime and tongue cleaning, eliminating plaque-retentive factors, stabilising periodontal disease, managing caries and intraoral infections.

Various products are available on the market, claiming their ability to eliminate oral malodour. However, there is insufficient evidence in the literature for current halitosis interventions. More well-designed, longitudinal studies with sufficient sample size are required to examine long-term efficacy of the available interventions, along with practical, standardised and objective measurement of halitosis.

Further work is required to increase awareness of halitosis in the general public. Through various modes of education on the prevalence, aetiology and management of oral malodour, the extent of social stigma and negativity associated with halitosis can hopefully be reduced, so that individuals suffering from the condition firsthand or otherwise can more openly discuss their experience and seek help. Collaborative multidisciplinary care in managing halitosis should be further researched and promoted.

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Is Keratinised Mucosa Surrounding an Implant Necessary?

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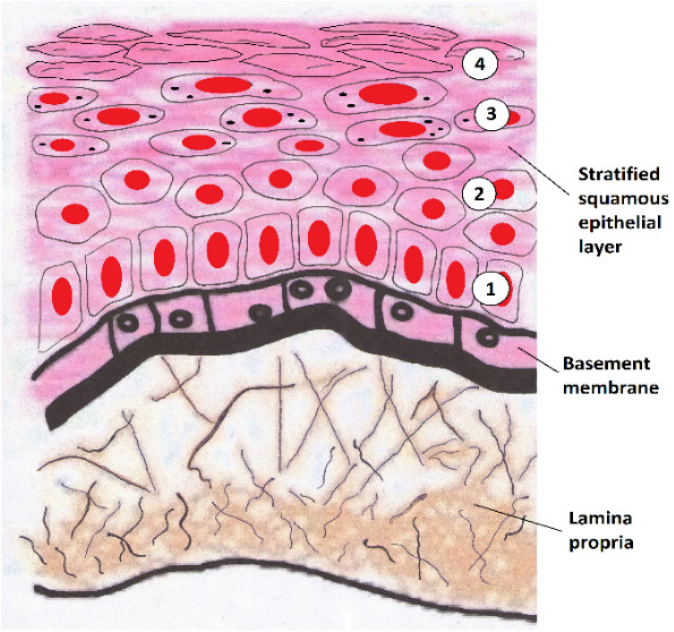
Introduction

Schematic illustration of the layers found in keratinized oral mucosa that include a deeper lamina propria and basement membrane in-between and superficial layers of stratified squamous epithelium that include from deepest to most superficial: 1: Stratum basale 2: Stratum spinosum 3: Stratum granulosum 4: Stratum corneum



https://en.wikipedia.org/wiki/Oral_mucosa

The function of epithelia is to protect the underlying tissues from various environmental influences. When analysing oral epithelium, three types of epithelia depending on morphology and specific differentiation patterns can be found which include; *keratinized stratified squamous epithelium* (masticatory mucosa found in hard palate, dorsum of tongue and attached gingiva), *non-keratinized stratified squamous epithelium* (soft palate, buccal, labial and alveolar mucosa) and *specialized mucosa* (dorsal surface of the tongue). (1) In the keratinized type, the stratified squamous epithelia

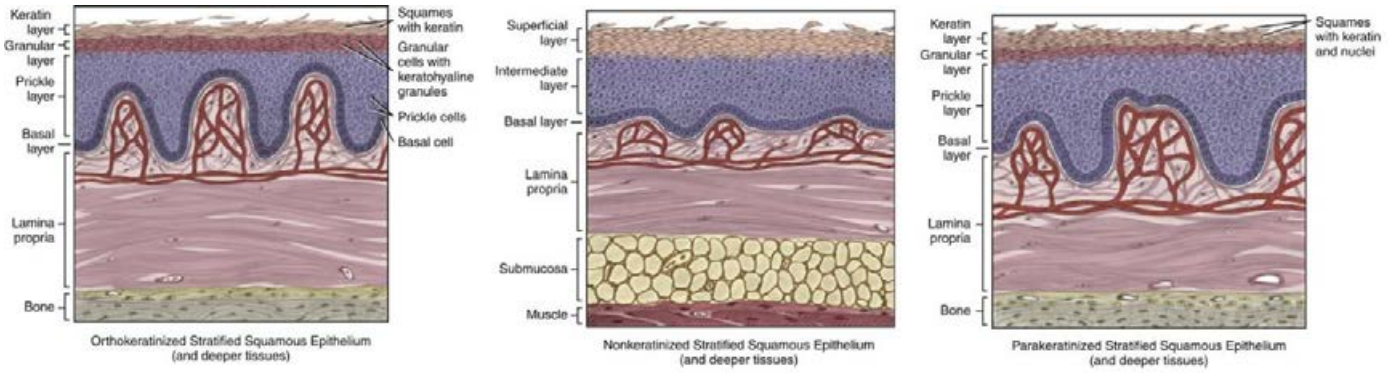
Abstract:

Aim: The aim of this paper is to ascertain the relevance of keratinised mucosa around dental implants and its significance in maintaining peri-implant health.

Method: An electronic search of Medline (Ovid), PubMed and Google Scholar were used. Both basic and advanced electronic searches were conducted using the search terms; dental implant, keratinised mucosa, keratinised mucosa width attached mucosa, peri-implantitis, peri-implant mucositis, plaque accumulation, attachment loss, bone loss and complications. Various titles were screened, and full text obtained where relevant. Some articles were identified from references in other articles after which full texts were obtained. Broadly, the search was restricted to studies conducted in the English language, published in the past 10 years (with certain exceptions to acquire historical references) and conducted on humans only.

Result: 47 articles and a textbook were identified and reviewed for this paper. The studies were highly heterogenic in design but mostly observed clinical parameters which impact peri-implant soft/hard tissue conditions such as gingival inflammation, plaque accumulation, bleeding on probing, periodontal pocket depth, mucosal recession, and bone level changes. After detailed analysis, 20 studies supported the assertion that keratinized mucosa is significant in maintaining peri-implant health whereas 9 studies rejected it. The impact of other factors like supportive implant maintenance, brushing discomfort, implant surface properties and soft tissue augmentation, on the relationship between keratinized mucosa and peri-implant clinical parameters were also analysed. From the data available in this review, it was concluded that insufficient keratinized mucosa appears to be associated with poor clinical parameter values which indicates

Histological illustration of three functional types of Oral Epithelium



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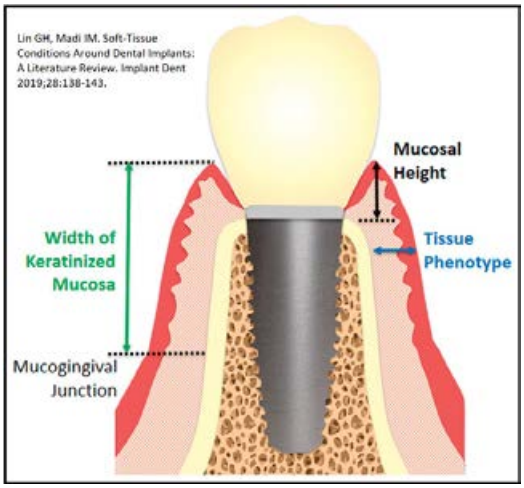
undergo terminal differentiation resulting in the formation of a mechanically tougher surface composed of proteinaceous cornified cells cross linked to keratin filaments, which provides a flexible and insoluble structure to protects the underlying epithelial cells. (2) This makes keratinized epithelium more stable and resistant against physical damage and infectious infiltration, therefor making its presence crucial for the long-term stability of underlying structures.

Keratinized mucosa includes the free and attached gingiva and its width is measured from the gingival margin up to the mucogingival junction. (3)



Based on clinical data it has been believed for several years that the presence of sufficient keratinized mucosa is critical for maintaining long-term gingival health. (4, 5) A scanning electron microscope study compared the microbiota in the "plaque-free" zone between healthy and chronic periodontitis patients and showed that an inadequate zone

Abstract: *(continued)*

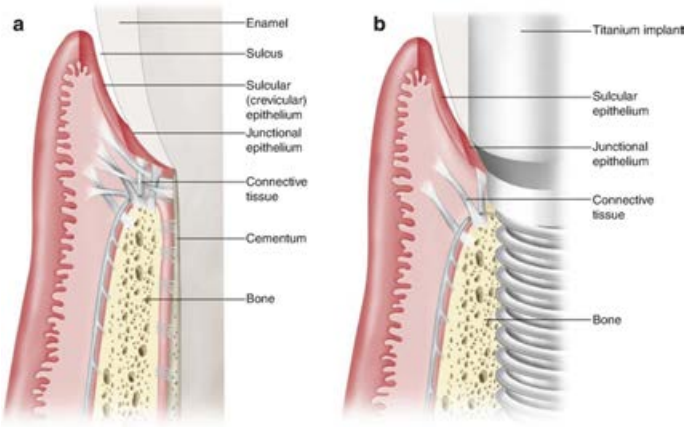
a higher risk of peri-implant complications. There is evidence available to suggests that regular implant maintenance can help to improve peri-implant health, however a lack of keratinized mucosa is still a risk factor irrespective of supportive implant therapy.

Keywords: dental implants, keratinised mucosa, peri-implantitis, peri-implant mucositis, clinical parameters, mucosal tissue, maintenance, implant surface, soft tissue augmentation, brushing discomfort.

of gingiva would increase subgingival plaque formation due to improper pocket closure, therefor concluding that the main function of the attached gingival tissue complex is to prevent access of plaque to the surrounding tissues. (6) A 6-week observational study following 32 dental students, determined that at least 2mm of keratinized mucosa of which 1mm was to be attached is necessary to maintain gingival health. 80% of tooth surfaces with >2mm keratinized gingiva were healthy whereas all surfaces with <2mm of keratinized gingiva showed signs of inflammation despite being plaque free. (7) However, several papers have disagreed with this concept and have shown that gingival health is maintainable even in the absence of adequate keratinized tissue or attached gingiva. (8-10)

The significance of keratinized mucosa is more important around restorations then natural teeth. It has been

demonstrated that subgingival restorations are prone to much higher rates of gingival inflammation, recession, and attachment loss (11) and subgingival restorations around narrow zone of keratinized mucosa and subsequent inadequate plaque control show significantly higher chances of gingival inflammation. (12) As dental implants are inherently subgingival restorations; it can be postulated that lack of keratinized mucosa around implants can negatively impact the maintenance of peri-implant tissue which may lead to increased peri-implant complications. There are also significant structural differences when it comes to periodontal and peri-implant tissues. Around teeth, the fibres present in the connective tissue layer subjacent to the junctional epithelium are perpendicular to the root surfaces and insert in the cementum whereas peri-implant connective tissue fibres are parallel or oblique and do not insert into the implant surface instead the attached mucosa adheres to the implant surface by means of an hemidesmosomes thus making the quality of attachment low.(13, 14) Plus, due to the absence of periodontal ligaments, the blood supply around implants is less then natural teeth.(15) These factors can make implants more susceptible to peri-implant complications which in turn increases the relevance of the protective function of keratinized mucosa as it firmly bounds to the underlying bone and constitutes a functional barrier between the oral environment and underlying dental implants.



https://pocketdentistry.com/introduction-to-understanding-the-basics-of-teeth-vs-dental-implants-similarities-and-differences/

The need of keratinized mucosa around implants is a controversial topic as different studies have proposed different conclusion. The aim of this paper is to review current literature and find evidence for the significance of keratinized mucosa in maintenance of long-term peri-implant health.

Discussion

Based on long-term studies, it appears that dental implant therapy has overall high survival rates, irrespective of the presence or absence of keratinized mucosa (KM). (16-19) However, weather there is a significant role of keratinized mucosa in preventing peri-implant complications in humans needs to be determined. Both, studies which support the relationship between adequate KM and peri-implant health and those against it, will be evaluated. The impact of other variables, like oral hygiene maintenance, brushing discomfort, implant surfaces and soft tissue augmentation, will also be examined in relation to peri-implant keratinized mucosa. Peri-implant soft-tissue inflammation, recession, plaque accumulation, probing depth, bleeding on probing, attachment level and peri-implant bone level are some of the clinical parameters commonly used to monitoring soft and hard tissue status of dental implants and indicate the presence or absence of peri-implant disease. (20)

Evidence supporting the relationship between adequate keratinized mucosa and peri-implant health

Baqain et al (21) conducted a prospective observational study to evaluate potential factors which may lead to early implant failure. Failure was defined as peri-implant radiolucency, infection, and/or implant mobility. As it was an early failure study, implants were evaluated from the time of placement till either the second stage or at the time of restoration and prosthetic treatment was not factored into the findings. From a total of 399 implants, only 4% were deemed early failures. An interesting finding was that implants placed in sites with narrow keratinized gingiva (<2mm) were nearly 5 times more likely of early implant failure when compared to implants placed in sites with adequate keratinized gingiva (>2mm).

Adibrad et al (22) carried out a cross sectional study to determine the relationship between the width of keratinized mucosa around implants supporting overdentures and its effect on the health of peri-implant supporting tissues in patients attending a regular maintenance program. A relatively small sample of 27 patients with 66 restored implants was

used, with no baseline keratinized mucosa readings and lack of standardized radiographs to accurately determine alveolar bone loss. It should be noted that due to the retrospective design of this study, the data is only explorative in nature and several prognostic factors like, surgical protocol, loading protocol, implant design and presence or absence of grafting were adjusted for in the results. Nonetheless, the findings showed that implants without adequate width of keratinized mucosa (<2mm) showed significantly higher plaque accumulation, gingival inflammation, bleeding on probing, periodontal attachment loss and mucosal recession. Even though probing pocket depth and radiographic bone loss were also higher in narrow keratinized mucosa group, they did not reach statistical significance. These findings were later confirmed by *Boynuegri et al* (23) in a prospective study which also looked at implants supported overdentures and found significantly higher plaque accumulation and mucosal inflammation at the 12-month follow-up around implants with absent keratinized mucosa. Peri-implant crevicular fluid analysis showed a significantly higher levels of pro-inflammatory TNF-a cytokine in implants without keratinized mucosa which may indicate a greater risk of peri-implantitis. (24) Short follow-up period and a small sample size (15 patients with 4 implants each) impede the study in providing a long-term comprehensive outcome.

Chung et al (25) agreed with the above findings in another short-term retrospective cross-sectional study following 69 patients and 339 endosseous implants with variations in implant surfaces. The objective of the study was to investigate the effect of keratinized mucosa in the maintenance of smooth and rough surface implants. Like the previous study (22), gingival inflammation and plaque accumulation were significantly higher in the group with inadequate keratinized mucosa (<2mm) specially in implants placed in the posterior region and no significant association was found with radiographic bone levels as bone loss was present irrespective of the presence or absence of keratinized mucosa. Furthermore, when comparing the impact of keratinized mucosa with different implant surfaces, smooth surface implants with inadequate keratinized mucosa (SKL) showed significantly higher plaque accumulation and gingival inflammation when compared to smooth surface implants with adequate keratinized mucosa (SKM), rough surface implants with inadequate keratinized mucosa (RKL) and rough surface implants with adequate keratinized mucosa (RKM). However keratinized mucosa was deemed less significant when comparing RKL and RKM groups even through there was a slightly higher amount of plaque accumulation and gingival inflammation noticed in RKL. Even though variables such as smoking and oral hygiene maintenance were not controlled in the, the results concluded that presence keratinized mucosa is not critical in reducing peri-implant bone loss, however its presence showed a significant advantage in maintaining soft tissue health irrespective of implant surface treatment.

Ladwein et al (26) in a long-term retrospective study, agreed with the above findings. 211 patients with 967 tissue level implants were divided into two groups, NKM (0mm of keratinized mucosa) and KM (>0mm of keratinized mucosa) with a follow up period between 4 to 15 years. This was different from other studies as adequate (>2mm) or inadequate (<2mm) keratinized mucosa were not being compared, rather the comparison was between complete absence or presence of any of keratinized mucosa. NKM showed significantly higher plaque accumulation, bleeding on probing and gingival inflammation, thus suggesting that even a presence of a small amount of keratinized mucosa may be sufficient for peri-implant soft tissue maintenance. However, like the previous studies (22, 25), no significant correlation was present regarding vertical bone loss and keratinized mucosa. Missing baseline radiographic data, the use of panoramic radiographs instead of standardized peri-apical radiographs and not excluding the initial bone loss due to bone remoulding could have increased the inaccuracy of the findings. Variables like surgical protocol, oral hygiene maintenance, type of prosthesis, history of periodontitis and smoking were not adjusted for which may have impacted the results.

Crespi et al (27) conducted a long-term prospective study which looked at 164 implants placed in freshly extracted sites with immediate loading and the effect of keratinized mucosa width on the peri-implant health over a period of 4 years. The results again supported a positive relationship between keratinized mucosa and peri-implant health by showing a statistically significant higher gingival index, plaque index and bleeding index in implants surrounded by a narrow-keratinized mucosa (<2mm) however like the previous studies (22, 25, 26), marginal bone loss between the two groups did not reach statistical significance. The majority of the gingival recession was seen in the first 6 month in both groups however it was significantly more around narrow keratinized mucosa, which was also confirmed by *Zigdon et al* (28) who showed that over a 3-year period, a wider and/or thicker keratinized mucosal band (>1mm) was associated

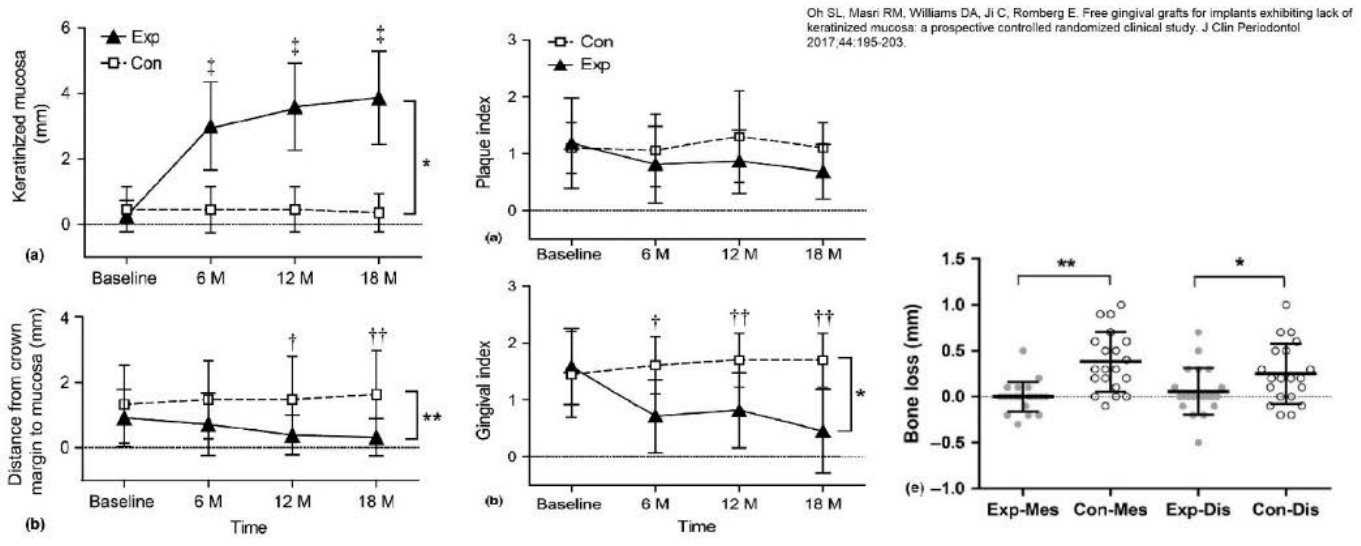
with significantly less mucosal recession (mean 0.27mm v/s 0.9mm) and attachment loss when compared to narrower/ thinner mucosal band (<1mm)

Kim et al (29) in contrast to the previous papers (22, 23, 25-27) showed in a retrospective study that implants with in-sufficient keratinized mucosa (<2mm) experienced significantly higher alveolar bone loss together with gingival recession whereas plaque accumulation was found not to be associated with keratinized mucosa and even though gingival inflammation was higher in the deficient keratinized mucosa group, it did not reach statistical significance. Also, in agreement with a previous study (25), no correlation was observed between implant surface and keratinized mucosa nor did implant surface have any significant impact of crestal bone loss. Limitations of the study included a relatively short follow-up period (average 13months), not including smoking or supportive peri-implant therapy as variables and as measurement were taken at the time of final prosthesis installation (baseline), there was lack of data regarding the soft tissue condition both pre-surgery and immediately after it. Similarly, *Bouri et al* (30) in another cross sectional study following 200 implants, showed that implants with <2mm of keratinized mucosa had a significantly higher chance of bleeding on probing (more than 3 times), gingival inflammation, plaque accumulation and alveolar bone loss as compared to implant with wide keratinized mucosa (>2mm). In both the above studies, baseline bone levels were taken at the time of implant placement and bone loss related to initial remodelling were not factored in the result which could have distorted the findings.

In one of the few prospective randomized control trials investigating this topic, *Oh SL et al* (31) followed 41 single unit implants in 28 patients over a period of 18 months. All implants had <2mm of keratinized mucosa and were randomly divided into those receiving free gingival graft to improve keratinized mucosal width (experimental group) together with prophylactic treatment and control group which received prophylactic treatment only. The baseline values (KM width, PI, GI, BoP, PPD and mucosal margins level) were similar in both groups at baseline however at follow-up, the results showed a significant gain of keratinized mucosa in the experimental group together with significantly reduced mucosal recession, gingival inflammation, bleeding on probing and crestal bone loss when compared to the control group throughout the follow-up period. Plaque accumulation was higher in the control group but it did not reach statistical significance.

Even with the small sample size and a relatively short follow up period, the study showed that free gingival graft is a predictable solution to attain adequate peri-implant keratinized mucosa and is in support of the previous findings (29, 30) that lack of keratinized mucosa can cause a higher degree of peri-implant bone loss and thus may lead to peri-implant mucositis and/or peri-implantitis.

A recent meta-analysis based on four prospective studies, *Thoma et al* (32) agreed with the above finding and showed favourable results towards soft tissue grafting to improve width of the keratinized mucosa and subsequently improved peri-implant health. The outcomes of the review were limited due to the lack of negative control groups and only up to 12 months follow up period for the included studies.



Canullo et al (33) also reported that implants with a height of keratinized mucosa equal to or greater then 2mm are 0.36 times less likely to develop peri-implantitis (>3mm bone loss, >4mm PPD together with BoP).

Grischke et al (34) in a cross-sectional study on 231 implants with regular maintenance care, reported that implants with <2mm keratinized mucosa width are 3.3 times more likely to develop peri-implant mucositis and there was a significant association between the reduced width of keratinized mucosa (<2mm) and the severity of the peri-implant mucositis both with and without adjusting for variable such as pocket depth, loading time, plaque index, implant position and sex of patient (1.7 and 2.5 difference of mean respectively). This shows that a reduced keratinized mucosa can not only increase the risk of mucositis but can also increase the severity of the disease as well. One factor which may have reduced the accuracy of the study is the fact that a gingival index system created to assess inflammation around teeth was used here to assess inflammation around implants, which could have led to misdiagnosis as in thin phenotype cases, the implant abutment could have shined through the soft tissue and mislead the examiner. Other limitations include the lack of temporality in the study design and limited to patients following regular maintenance.

A systemic review by *Gobbato et al* (35) concluded that implants with <2mm of keratinized mucosa showed significantly higher gingival inflammation, plaque accumulation and bleeding on probing when compared to implants with >2mm of keratinized mucosa. Thus, showing a higher risk of peri-implant mucositis in implants with narrow keratinized mucosa. These findings were confirmed by other systemic reviews analysing the significance of keratinized mucosa. (36, 37)

Evidence not supporting the relationship between adequate keratinized mucosa and peri-implant health

In a cross sectional study, *Esper et al* (38) followed 202 implants in 109 patients with cleft lip and/or palate who underwent significant bone grafting to improve thickness of the alveolar ridge. The results showed no significant difference in gingival inflammation or plaque accumulation between implants with adequate keratinized mucosa (>2mm) and implants without adequate keratinized mucosa (<2mm). No radiographic analysis of bone levels was included in this study. The probing depth was found to be higher around adequate keratinized mucosa, which backs *Zigdon et al* (28) results and the possible reasoning can be that due to increased mucosal recession around narrow keratinized mucosa, the pockets become shallower therefor showing reduced probing depths. It should be noted that the soft tissue conditions were analysed only 1-year after restoring the implants which is not sufficiently long enough to evaluate the impact of a reduced keratinized mucosa on peri-implant health.

In a 5-year retrospective study, *Lim et al* (39) not only investigated the influence of keratinized mucosa on peri-implant health but also tried to determine the threshold value of keratinized mucosal width. Based on the results, negligible association could be drawn between the buccal keratinized mucosa width and clinical parameters of peri-implant mucositis (BoP, Plaque index and Pocket depth) or peri-implantitis (Marginal bone-loss). Moreover, no visible pattern emerged between the recorded parameters and keratinized mucosa width, thus no threshold value could be determined which indicates that the use of thresholds like >2mm or <2mm of width may be meaningless. These results were a confirmation of another long term (>12 years) study by *Frisch et al* (40) in which 60 patients were analysed who enrolled in regular supportive postimplant therapy (SIT) and had at least one implant with <1mm of keratinized mucosal width. Mucogingival surgical procedures at implants (MGSI) with either free gingival graft (FGG) and connective tissue graft (CTG) were offered to all patients approximately 2 years after implant placement to help increase the keratinized mucosal width and patients who accepted were placed in the Intervention group and those who rejected it were the control group. At a 10 year follow up period both FGG and CTG had significant success in improving long term keratinized mucosa width with FGG showing slightly better results (3.3mm v/s 2.87mm), whereas no width gain was seen in the control group. Overall success and survival rates in both groups were high and no significant difference was observed between the two groups in relation to peri-implant mucositis (+ve BoP) and peri-implantitis (+ve BoP, PPD >5mm and Bone loss >3.5mm). Both studies had a retrospective design, small sample sizes and only included patients following strict postimplant maintenance program with overall good oral hygiene scores therefor these results cannot be applied to patients not compliant to regular maintenance.

Dalago et al (41), similar to previous studies but with a larger sample size, was unable to find any correlation between a lack or absence of peri-implant keratinized mucosa and incidence of peri-implantitis (PPD >5mm, BoP and BL >2mm). One major flaw in the study design was that digital peri-apical radiographs obtained at the time of data collection were compared to panoramic radiographs from baseline to calculate bone level changes, which could lead to obvious miscalculations in determining bone level changes and subsequently diagnosing peri-implantitis.

Todisco et al (42) in a recent 5-year prospective cohort study, 128 implants were divided into those having no keratinized mucosa either in the vestibular or lingual region (KMH Def=0) and those having keratinized mucosa at both vestibular and lingual region (KMH Def=1). No significant association between the two groups was identified during the follow-up period with respect to marginal bone loss and bleeding on probing. In fact, when keratinized mucosal height was analysed as a dichotomous variable, KMH Def=1 group showed significantly greater marginal bone loss however a statistically non-significant trend of reduced bleeding of probing was also observed when compared to KMH Def=0. These results contradict the apparent protective role of keratinized mucosa around implants. Like previous studies (38-40), a limited sample size was analysed and only included patients who followed strict maintenance regimes.

A meta-analysis by *Wennstrom et al* (43) reviewed 17 publications on humans. When analysing plaque accumulation between adequate (KM >2mm) and inadequate keratinized (KM <2mm) mucosal width, 4 studies showed significantly more plaque accumulation in the KM <2mm group and 6 studies showed no significant difference between the groups. Similarly, 5 studies showed significantly higher bleeding on probing values in KM <2mm group whereas 5 studies showed no significant difference. 8 out of the 10 studies reporting periodontal pocket depth (PPD) showed no significant difference between the groups with only *Zigdon et al* (28) showing significantly greater PPD in patients with KM <1mm. Soft tissue recession evaluated by 3 studies and only *Crespi et al* (27) showed a significant relationship between lack of keratinized mucosa and gingival recession. No human studies included in the review showed any relation between changes in bone level or implant loss and width of keratinized mucosa. Overall, this review concluded that there is limited evidence supporting the need of peri-implant keratinized mucosa in maintaining long term per-implant health.

Impact of regular maintenance on the relationship between adequate keratinized mucosa and peri-implant health

When evaluating the data from the studies not supporting the idea of a protective role of keratinized mucosa around implants, it can be deduced that in most of these studies (38-40, 42), regular supportive implant therapy protocol is followed by the participating patients and one can wonder if this continuous maintenance throughout the follow up period had an impact in improving clinical soft tissue parameters around implants with inadequate width of keratinized mucosa. Even the previously discussed systemic review (43) concluded that studies which had adequate maintenance showed no significant relationship between reduced peri-implant health and lack of keratinized mucosa, whereas a significant relationship was found in studies which lacked proper maintenance protocol.

Roccuzzo et al (44) in a prospective comparative study followed 98 posterior mandible implants in patients having excellent oral hygiene and a regular tailored made maintenance program throughout the 10-year follow-up. In this study, keratinized mucosa was dichotomized into either present (KT) or absent (AM) at the time of implant surgery. During the duration of follow up, AM required significantly more antibiotic or surgical intervention for biological complications as compared to KT (51.4% v/s 12.7%). 42.9% of patients in AM showed pain or discomfort during oral hygiene procedures (tooth brushing) whereas no such complain was present in the KT group, a finding supported by *Souza et al* (45) and *Perussolo et al* (46) who showed significantly higher peri-implant discomfort during tooth brushing in patients with <2mm of KT. Plaque accumulation, soft tissue recession, bleeding on probing (BoP) and mean bone loss values (mBL) were all higher in AM when compared to KT however BoP and mBL did not reach statistical significant values. Free gingival grafts were done for some patients in the AM group to help achieve adequate keratinized mucosal width and improve oral hygiene maintenance, which created a significant treatment bias in the study design. These findings are even more relevant as good long-term compliance was shown in both groups (<20% FMBS and FMPS), which indicates that even with good long-term supportive therapy, keratinized mucosa is valuable in maintaining peri-implant health and reducing the incidence of biological complications. These results should

only be interpreted for implants placed in the posterior mandibular region only.

In contract, *Monje et al* (47) conducted a cross sectional study on 37 patients who had received 66 implants with screw retained fixed prosthesis for minimum 3 years and did not adhere to regular peri-implant supportive programs. The results again showed that implants with insufficient of keratinized mucosa (KM <2mm) showed significantly higher values of periodontal pocket depths, bleeding on probing, plaque accumulation and marginal bone loss together with a significantly higher rate of peri-implantitis (BoP+PPD>6mm+MB level>3mm apical). However, no significant difference was seen between the two groups with regards to peri-implant mucositis. The KM<2mm group also showed significantly higher discomfort in tooth brushing and shallower vestibular depth with no attached mucosa, which can lead to difficulty in oral hygiene maintenance and may negatively impact peri-implant health. Patients with a keratinized mucosal band of 2.5mm or more showed maximum tooth brushing comfort. Due to the cross-sectional study design lacking any temporal element, a cause-effect relationship between peri-implant parameter and keratinized mucosa cannot be determined. In addition, prognostic variables like history of periodontitis, gingival recession, surgical protocol and implant design were not factored in the results. Nonetheless, it was concluded that erratic compliers have a higher risk of peri-implant disease in the absence of adequate keratinized mucosa whereas its presence can play a significant role in protecting the peri-implant tissue.

Romanos et al (48) in a retrospective study not only evaluated the relationship between keratinized mucosa and peri-implant soft tissue stability, it also further subdivided the groups into patient compliant to implant maintenance therapy (IMT) and those who are not. Bone level changes were not assessed and implants with any crestal bone loss were excluded, which helped eliminate the variable of hard tissue change association with soft tissue stability. As per previous studies, plaque accumulation, bleeding on probing, and mucosal recession (3 times more) were significantly higher in the narrow-keratinized mucosa group. Non-compliant patients with narrow keratinized mucosa had significantly higher plaque accumulation when compared to non-compliant patients with wide keratinized mucosa whereas no significant difference was seen within the compliant group, which shows that good compliance to IMT can reduce plaque accumulation irrespective of keratinized mucosa. However, inflammation was only related to reduced keratinized mucosa width, irrespective of IMT. Therefor it was concluded that even though regular IMT assists in reducing plaque accumulation, the presence of sufficient keratinized mucosa is highly recommended in reducing peri-implant inflammation. Absence of baseline soft tissue records was a major limitation of this study as a temporal relationship of implant therapy and soft tissue health in these groups could not be determined.

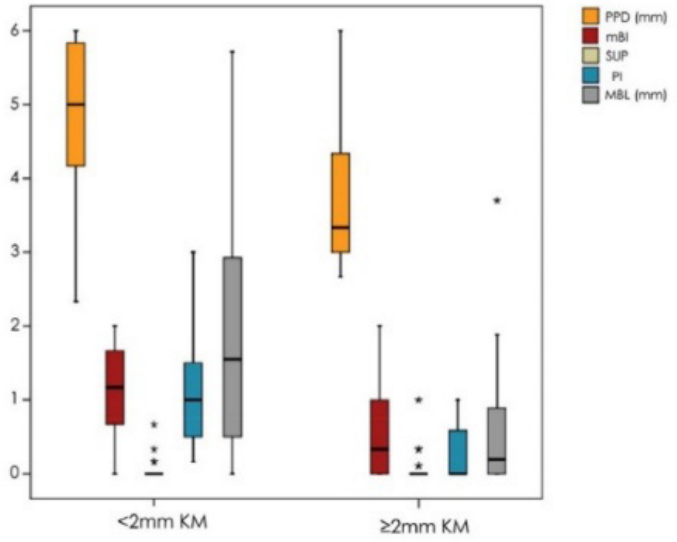


FIGURE 1 Clinical and radiographic parameters according to the width of the band of keratinized mucosa (<2mm/≥2mm). Asterisks

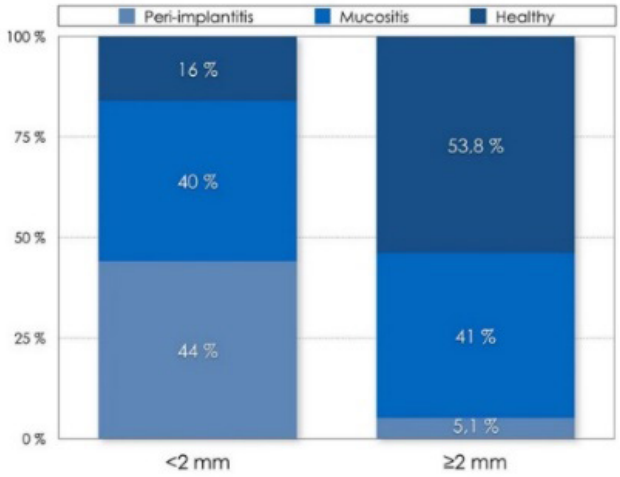


FIGURE 2 Implant diagnosis according to the width of keratinized mucosa

Monje A, Blasi G. Significance of keratinized mucosa/gingiva on peri-implant and adjacent periodontal conditions in erratic maintenance compliers. J Periodontol 2019;90:445-453.

Conclusion

The majority of the identified articles support the relationship between keratinized mucosa and peri-implant health. Despite one study showing early implant failure risk of implants lacking keratinized tissue (21), there is little evidence supporting the role of keratinized mucosa on implant survival. In reporting the peri-implant clinical parameters, several studies showed significantly higher plaque accumulation (22, 23, 25-27, 30, 35, 44-48), gingival inflammation (22, 23, 25-27, 30, 31, 34, 35), bleeding on probing (22, 26-28, 30, 31, 34, 35, 45-48), mucosal recession (22, 27-29, 31, 44, 48), pocket depths (47), radiographic bone loss (29-31, 44, 46, 47) and attachment loss (22, 28) in the presence of insufficient peri-implant keratinized mucosa, which are clear signs of peri-implant disease. Lack of keratinized mucosa also seem to increase brushing discomfort (44-47) which may lead to increased plaque accumulation and subsequently peri-implant complications. These parameters can be even more significant in the aesthetic zone as gingival inflammation or recession can lead to compromised aesthetic outcomes.

Even though multiple studies failed to correlate the presence of adequate keratinized mucosa with improved peri-implant clinical parameters (38-43), it should be noted that most of these studies followed regular implant maintenance regimes which could have played a key role in the outcomes. It was shown that in erratic maintenance compliers, presence of keratinized mucosa helped in significantly reducing the risk of peri-implantitis (47) and regular maintenance compliers are still at risk of peri-implant complications without adequate keratinized mucosa. (22, 34, 44, 48)

Implant surface configuration had no significant impact on peri-implant health with respect to keratinized mucosa width. (25, 29) Whereas soft tissue augmentation with both free gingival graft or connective tissue graft are successful in increasing the width of keratinized mucosa and subsequently improving soft tissue health. (31, 32, 44)

After reviewing the current literature, it can be concluded that an adequate zone of keratinized tissue around an implant is crucial for the long-term maintenance of both soft and hard peri-implant tissue, irrespective of regular implant maintenance therapy. Soft tissue augmentation can be used to improve the keratinized tissue width however in the absence of adequate tissue, good oral hygiene maintenance and continued supportive peri-implant therapy can aid in reducing peri-implant complications.

It should be noted that most of the literature addressing this topic have a high degree of heterogeneity in design and consist mostly of short-term cross sectional or retrospective studies with limited access to baseline data and lack of temporal relationship between exposure and outcome. This heterogeneity is also evident in determining the keratinized width threshold as some studies take 2mm as the threshold, some take 1mm and some only discuss presence or absence keratinized tissue. Important variable like; type of prosthesis, contour of restoration, cemented or screw retained, history of periodontitis, smoking, implant design, implant position, bone augmentation and oral hygiene maintenance need to be factored in the results to acquire a more accurate conclusion. Due to these study design shortfalls, the role of keratinized mucosa in peri-implant health is still controversial and therefor more prospective multi-cantered long-term randomized control trials with sufficiently large sample size are required to gain a more definitive answer to this question.

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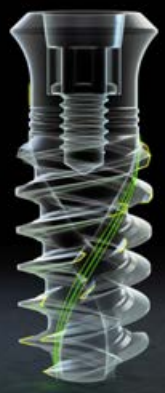

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The Effect of Scan Body Design on the Accuracy of Digital Dental Implant Restoration Workflows

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Introduction

The first commercially available intraoral scanning and CAM systems were released in 1985. CEREC 1 allowed for chairside construction of inlays for immediate restoration by using limited two-dimensional (2D) images. (1) Since 1985, developments in computer technology and workflow refinements have assisted the evolution of these systems from use in single unit restorations, into systems with applications in model creation, prosthodontics, orthodontics, and implant dentistry. (2)

A recent review of the literature regarding intraoral scanners in dentistry reported many advantages of using this technology, including less patient discomfort, improved time efficiency, simplified clinical procedures, no plaster casts, and better communication with dental technicians and patients. (3)

The use of digital scanners for the restoration of dental implants requires the use of componentry referred to as scan bodies. The scan body allows for the indirect visualisation and digitisation of the intraosseous position of the implant by the scanner, without which this process would not be possible. The accuracy of digital implant impressions, with both intraoral and laboratory scanners, has been well researched and documented. (4, 5) However, studies of scan bodies themselves are comparatively underreported and emerging in the academic literature.

Advancements in digital technology have driven significant interest and investment in the global intraoral scanner (IOS) market from the dental community. This market was valued at USD 428.9m (AUD 594.13m) in 2020 and is predicted to rise to USD 890.90m (AUD 1.234b) by 2028. (6) A survey completed in 2021 by the American Dental Association reported that 53% of respondents currently used an intra-oral scanner in their practice, with 34% of non-users considering buying a system. (7)

With the surge in access among practitioners, improved patient-centred outcomes and efficiencies compared to conventional impressions (8), it is only reasonable to assume the use of digital impression techniques for implant

Abstract:

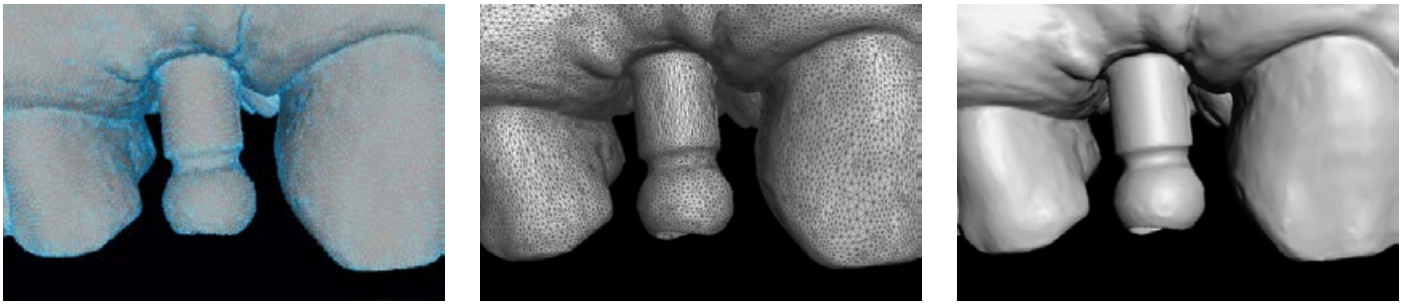
Intraoral scanning and digital dentistry have surged in popularity over the past few years, bringing with it a host of advantages to clinical practice. One of the areas which has more recently introduced digital techniques is restoration of dental implants. Despite this, there has not been clear evidence to support the use of digital impression techniques over conventional impressions.

CAD/CAM techniques comprise three basic elements, digital data acquisition, data processing, and computer aided manufacturing. Scan bodies were developed to assist in the capture of the position of the dental implant within the dental arch by a digital scanner, akin to an impression coping in a conventional impression.

This review of the current literature reveals there is limited understanding in the literature on how the design of scan bodies may affect the accuracy of both data acquisition and data processing, and how this impacts the outcome of the prosthesis.

Keywords: Dental; Implant; scan body; design; accuracy

Figure 1. a) Point cloud data of an implant scan body. b) Polygon mesh reconstructed. c) Rendered surface visualisation (2)



restorations will increase in the future. This literature review aims to review the role and importance of using scan bodies in the CAD/CAM implant restoration workflow and the impact that scan body design may have on data acquisition and processing accuracy.

Background

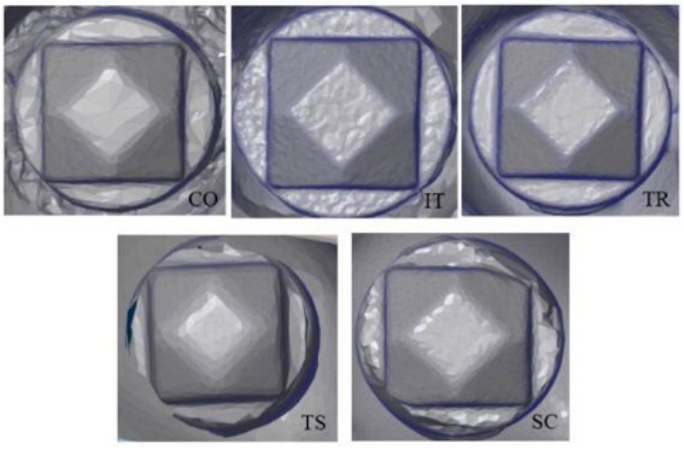
The restoration of a dental implant requires the capture of the implant's precise location and connection indexation within the context of the dental arch. Conventionally, impression copings and implant analogues were used to transfer this information to a stone model. Scannable impression copings, later termed 'scan bodies' by the Straumann Group, enable the transfer of the required information to a digital model via a computer-aided design and computer-aided manufacturing (CAD/CAM) workflow. (2)

The CAD/CAM workflow is dependent on three main components: 1) digital data acquisition, 2) data processing and design, and 3) computer-aided manufacturing by subtractive (milling/grinding) or additive (3D printing) methods. (9) Digital data acquisition is performed using digital scanners, which can be classified as mechanical/contact (touch-probe) or non-contact/optical (Laser/white light). Non-contact optical scanners dominate the dental market in both intra-oral and laboratory settings. (10)

All scanners, regardless of type, operate with the same basic premise: they collect data to digitally recreate a 3-dimensional image of the surface of the object being scanned. The raw data collected via the scanner is used to create a 'point cloud', with each point representing a coordinate on the x, y, and z-axis of 3-dimensional space. The density of the point cloud relates to the number of points in a measured area and is an outcome of the scanner resolution and the rendering software. Typically, a higher density point cloud will provide a more accurate surface reconstruction. (2, 11, 12)

However, a series of points will not form a visually complete surface for the purpose of clinical use. Rendering the point cloud creates a series of triangles by linking three adjacent data points to create a polygon mesh; a process called computerised polygonisation. The surface of the scanned image is represented by a series of flat triangle surfaces between discrete data points. Scans with a low point cloud density will have larger interpoint areas where there is no data, which may result in dimensional inaccuracies or surface characteristics that are unrepresentative of the scanned object. (11)

Figure 2. Surface topography of the same scan body from different intraoral scanners, demonstrating differences in point cloud resolution (9)



Digital Data Acquisition techniques for implant restoration

Digital data acquisition of the implant position can be completed with both *direct* and *indirect* techniques using scan bodies and intra-oral or laboratory scanners. (13)

Indirect:
Conventional impression → stone model → Digitised by the laboratory with scan body and benchtop scanner→ data processing

Direct:
Intraoral digitisation with scan body and intraoral scanner → Laboratory for data processing

Both methods have the potential to introduce errors into the data acquisition process, which may impact the accuracy of fit of the final prosthesis. The literature is yet to demonstrate the definitive superiority of either technique for implant impressions. Two comprehensive systematic reviews and meta-analyses by Flügge et al. (2018) and Papaspyridakos et al. (2020) comparing digital and conventional implant impressions provide concise summaries of the current literature.

Flügge et al. (2018) concluded that with the limited high-quality evidence and insufficient in vivo data available in the literature, no clinical guidelines could be derived recommending the superiority of either technique. From the data available, two preliminary conclusions were presented. 1) Angled implants showed significantly higher inaccuracy than parallel implants, regardless of the impression technique employed. 2) Scan protocol impacts the accuracy and precision of digital impressions. (14)

Papaspyridakos et al. (2020) surmised that, based on mainly in vitro studies, digital scans appear to have comparable 3D accuracy to conventional impressions. The authors also agreed that based on the current evidence and lack of in vivo studies and clinical studies, a recommendation for the routine use of intraoral digital scans for partial or complete arch implant impressions cannot yet be made. Scan body shape and design and clinically relevant factors such as implant angulation, implant depth, operator experience and intraoral scanning strategy may affect 3D accuracy. (4)

Extrapolating in vitro results for IOS accuracy to clinical applications of intraoral scanners should be done with caution. Intraoral conditions can have a negative influence on scan accuracy. These factors have been reported to include moisture, salivary flow, space restriction and ambient room lighting conditions. (15, 16) Extraoral scanning techniques are immune from these variables. However, although conventional elastomer materials have demonstrated excellent dimensional stability and precision, the indirect technique introduces variables such as temperature, the time between impression and pouring, wettability of the gypsum, and dimensional stability of the gypsum product. (17)

Digital data processing for implant model creation

Once the digital scan has been acquired, rendered, and recreated in the polygon mesh, it can be exported in a working file format called a 'standard triangle language' file or 'STL'. Some IOS systems are referred to as "closed" and export in a proprietary file format, which can restrict how or where the file is used.

The scan body manufacturer provides a reference surface representation of the scan body design as part of a 'digital library' within the dental CAD software. This reference information is aligned with the digital scan using a surface matching algorithm, which automatically aligns the digital implant analogue in the correct 3d position. The best-fit algorithm is most used for surface matching. It aims to reduce the global distances between the point cloud of the digital scan and the point cloud of the reference scan body from the library, reducing the root-mean-square error of the two data sets. (2, 18)

The process of surface alignment may be aided using three-point or manual shape matching algorithms, which reduces the quantity of point-cloud data requiring sampling. Once the scan body has been correctly aligned within the software, the original data representing the scan body is removed from the point cloud using the Boolean subtraction algorithm, leaving the 3D position of the implant analogue merged into the original scan data. (2)

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Scan body design

The design of scan bodies, including the shape and material, is highly variable among manufacturers and systems. Despite variations in design, they typically share three main design elements: (19)

1) Scan region: This is responsible for locating the 3D position of the implant and the indexing of the connection (if engaged) and may even contain identifiable information on implant manufacturer, connection type, and size.
2) Base: forms the interface between the scan body and the implant or abutment, may or may not engage the connection for indexing.
3) Transition zone: connects the base to the scan region

Several factors of the scan body may influence the outcome of the digital workflow, including shape and primary design factors, materials used for construction of the scan regions and base, wear and distortion from use, manufacturer machining tolerances, the resultant fit of components to implant connections or laboratory analogues, and scan body library provided for surface matching. (2, 13, 18, 20, 21)

Influence of scan body design on scan accuracy

Comparatively, little is known about scan bodies and their impact on the digital workflow. Extrapolating more broadly from the literature on digital scanning demonstrates that primary structures (overall shape) that are opaque, with smooth flat surfaces and rounded corners, are more accurately scanned than those with shiny, translucent, or rough surfaces with sharp edges or undercut areas. (2, 22)

Mizumoto et al. (2020) and Motel et al. (2019) both compared various scan body designs and scan strategies with a single IOS system (Trios 3, 3Shape). Mizumoto et al. concluded that both scan body and scan technique affected the accuracy of the digital scans and that scan bodies of shorter, simple shapes with fewer undercuts improved scan time and accuracy. (13) Motel et al. concurred with this summation, finding that within the scope of the study, scan bodies with flatter, simpler structures were associated with significantly smaller deviations. (23) However, Revilla-Leon et al. (2021) compared three scan body designs using a different IOS system (iTero Element, Cadent). Results showed that both scan bodies transferred accurate linear positions, with differences only in the XY angular deviation, favouring a more complex scan body design. (24) This contradiction may indicate that different scanner technology may have improved accuracy with different scan body designs.

Huang et al. (2020) demonstrated that adding an extension arm to a scan body improves precision for full arch implant scans by effectively reducing the scanning distance between the scan bodies. No differences in trueness or precision were noted between the scan body designs without the extension, and all groups were less accurate than a conventional splinted impression and laboratory digitisation. (25)

Scan bodies are typically constructed of a single material such as PEEK (Polyetheretherketone) or titanium alloy, or a dual material with a PEEK scan region and a metal base made from either aluminium or titanium alloy. (2, 18, 26) Arcuri et al. (2020) compared scan body material on scan accuracy, concluding PEEK showed the highest accuracy, followed by titanium and PEEK with a titanium base, respectively. (26) PEEK has demonstrated to be susceptible to dimensional and material change over multiple sterilisation cycles (27) and to suffer negative wear patterns when used repeatedly in a single material scan body design, impacting accuracy. (20) It is therefore prudent to follow the manufactures recommended number of cycles when using PEEK scan bodies.

Some studies have reported vertical distortion of scan bodies made entirely of PEEK, with speculation this was due to a lack of manufacturer recommendations leading to over-torque of the scan body and compression of the material. (28) However, when comparing differing torques levels, Tan et al. (2021) found no difference between single material groups and those with a metal base. All but one scan body tested experienced vertical distortion with increased torque levels. (18) Kim et al. (2020) also examined scan body distortion on screw tightening at "hand tight", which is often recommended by manufacturers, 5Nm and 10Nm. They demonstrated that the average "hand tight" torque level was 15.7Nm (± 1.3Nm) and resulted in over 100µm of vertical displacement in PEEK scan bodies. It was therefore recommended that manufacturers recommend 5Nm for PEEK scan bodies. (29) Interestingly, in this study, the genuine PEEK scan body matching the tested implant (Straumann CARES, Straumann) did not demonstrate the same deformation as the other PEEK scan bodies at higher torque levels, possibly representing differences in manufacture materials and tolerances. (29)

Two studies have examined the accuracy of repositioning scan bodies directly onto either implants or laboratory analogues. Pan et al. (2020) examined the reproducibility of digital scans when scan bodies were removed and replaced in the same positions or removed and randomly replaced in any position in the model. A significant increase in distance distortion was demonstrated when the scan bodies were replaced both in the original positions and randomly replaced. Random repositioning also introduced significant differences in angular trueness and an increased distance error. From the data, it was concluded that the differences were likely due to machining tolerances of the scan body base connection to the implant or analogue, which has been shown to vary significantly between manufacturer and component, and the manufacturing tolerances of the scan body itself. However, they acknowledged using third-party components in the study, which may have a reduced fit compared to original components. Accordingly, the outcome should be viewed with caution before extrapolating to other scan bodies or scanning systems. (30)

Stimmelmayr et al. (2012) did examine the reproducibility of genuine manufacturer scan body fit onto both implants and laboratory analogues and found a significant difference between the groups in favour of the laboratory analogue group. The differences exceeded that of the manufacturing tolerance reported (15µm), however, were still within limits reported for impression copings, abutment replicas, and abutments in previous studies. (31) An update to this study would be beneficial in assessing whether improvements in manufacturing over the past decade have led to an improvement in component fit and differences between original and third-party scan bodies.

Influence of scan body design on data processing and model creation

Few studies exist that examine the effects data processing and model creation have on scan bodies. However, they are beginning to emerge in the literature.

Choi et al. (2020) demonstrated that the reduction in scan body exposure due to increased gingival height or depth of implant placement negatively impacts the virtual implant positioning. This finding was due to inaccuracy in the surface matching of the scan body reference library to the digital scan. However, the authors acknowledged that the results reflect an outcome of one scan body design and one desktop scanner, and comparisons with other manufacturer products were recommended. (32)

Donmez et al. (2022) and Mangano et al. (2020) both attempted to compare the accuracy of mesh data of a particular scan body from different intraoral scanners to surface match with the corresponding scan body library file. Donmez et al. concluded that all scanners produced similar congruence of files, whereas Mangano et al. concluded there was a statistically significant difference between scanners. (33, 34)

Both studies utilised a single scan body to compare the scanners, with the scan body chosen in each study differing significantly in design and geometry. This may indicate that specific scan body designs may provide a more favourable congruence between mesh files of specific scanners to the corresponding scan body library file. Another noted issue with the studies from Choi, Donmez and Mangano are that they all used engineering grade geometric software for the surface matching of the scan body libraries to the scan meshes which does not mimic the workflow in digital restorations manufacturing.

Pan et al. (2022) attempted to improve the above studies by utilising dental-specific CAD/CAM software for the surface matching process before comparing the results for two different scan body designs from the same manufacturer after digitisation by a laboratory scanner. They noted differences between the dome and cuboidal shaped scan bodies, with improved surface matching accuracy of the dome-shaped group. They concluded that the geometry of a scan body might influence the transfer accuracy in the digital workflow. (21)

Conclusion

Scan body design and its effect on scan accuracy and data processing are not well understood in the literature. There are some indications that both scan body design and the type of scanner used may influence both the data acquisition and data processing component of the CAD/CAM workflow.

Gaps exist in the literature regarding whether a single scan body design is suitable for accurate data capture for modern scanning systems or whether scan body design should be tailored to the scanning system used. It is also unknown whether the scan body design and digital scanning system impact the surface matching of the scan mesh to the digital scan body library file. In Vivo data is also lacking, along with an understanding of how these variables may impact the fit of the final restoration and clinical outcomes.

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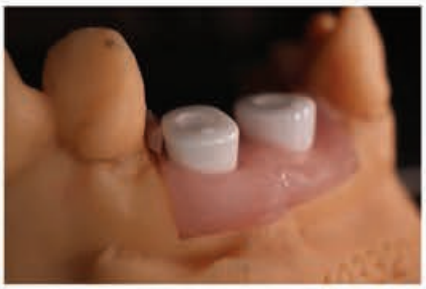

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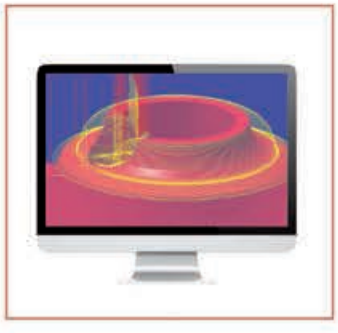

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State Branch Councillor: Dr Robert Fell

Secretariat: Mrs Helen Mooney

Email: helen.mooney4@gmail.com

Meeting name: Full Day Meeting & AGM

Meeting date & time: Friday, 25 November 2022 at 8:30am

Meeting location: Sofitel Wentworth Hotel, Phillip Street, Sydney

Speakers: Dr Giulio Rasperini

Topics: Interproximal attachment gain: The challenge of periodontal regeneration

Cost & other details: Members: $100 Guests $440 Register online helen.mooney4@gmail.com

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Meeting name: The GJ Seymour and MP Cullinan Research Medallion Competition and AGM

Meeting date & time: Monday 17th October 2022, 6:30pm-9pm

Meeting location: The Inchcolm Ovolo Hotel, 73 Wickham Terrace Spring Hill QLD 4000

Speakers: Periodontics post-graduate and PhD students presenting their original research

Topics: Original postgraduate and post-doc research relating to the field of periodontics. The winner will be awarded $500 cash, many thanks to sponsorship from Geistlich Pharma Australia.

Cost & other details: Members: Free, Guests: $150, Send your RSVP to aspqld@gmail.com

Meeting name: ASPQ Annual Clinic Day

Meeting date & time: Friday 18th November, 9-5pm

Meeting location: The Inchcolm Ovolo Hotel, 73 Wickham Terrace Spring Hill QLD 4000

Speakers: Professor Axel Spahr, Dr Jaya Seneviratne, Dr Stephen Robinson

Topics: Professor Axel Spahr will be presenting updates in periodontal regenerative techniques and ITI implant guidelines, as well as management of soft tissue aroundn dental implants. Dr Jaya Seneviratne will be talking about microbiology in periodontology. Dr Stephen Robinson will be talking about prosthodontic design and how it can affect implant health and longevity, and principles around prosthesis design to preserve implant health.

Cost & other details: Members: Free, Guests $250, Send your RSVP to aspqld@gmail.com

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Meeting name: ASP SA fourth dinner meeting (including AGM)

Meeting date & time: Wednesday, 19 October 2022, 6pm for 6:30pm start

Meeting location: The Lion Hotel, 161 Melbourne Street, North Adelaide

Speakers: Dr James Badlani, Oral and Maxillofacial Surgeon

Topics: Oral rehabilitation of head and neck cancer patients

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Meeting name: ASP (VIC) November 2022 Dinner-Lecture meeting

Meeting date & time: Wednesday, 16 November 2022 6pm registration for 6.30pm start

Meeting location: Woodward Conference Centre, University of Melbourne

Speakers: Dr Giselle D'Mello

Topics: Periodontal Interactions in Paediatric Dentistry

Cost & other details: Members: Free Guests: $180

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Meeting name: End of Year Dinner Lecture

Meeting date & time: Thursday, 17 November 2022, 7pm

Meeting location: Matilda Bay Restaurant

Speakers: A/Prof Tino Mercado

Topics: Enamel Matrix Derivative (Emdogain), a 25 Year Journey on the Biology, Development and Clinical Indications.

Cost & other details: TBA

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Meeting name: AOS QLD Branch Young Lecturer's Competition Dinner Meeting
Meeting date & time: Wednesday 19th of October 2022
Meeting location: Inchcolm by Ovolo
Speakers: Young Researchers and Lecturers
Topics: Research in Dental Implantology
Cost & other details: Free for Members, $140 for Non-Members

Meeting name: AOS (QLD) Dinner Lecture Meeting
Meeting date & time: Wednesday 22nd of February 2023
Meeting location: TBC
Speakers: Prof Saso Ivanovski
Topics: Implant Surfaces and Clinical Implications
Cost & other details: Free for Members, $140 for Non-Members

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President: Dr Angelos Sourial

Secretary: Dr Gaurika Sud

Treasurer: Dr Betty Lisa Matthews

Federal Councillor: Dr Gabriel Rodriguez Ortiz

Admin/Secretariat: Ms Bella Cherkasskaya

Email: infovic@aos.org.au

Meeting name: Dinner meeting and online broadcasting

Meeting date & time: 04 October 2022 at 7.00 PM

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

Speakers: Main Presenter: Dr Vahid Parson (Prosthodontist) Moderator Dr Chee Chang (Prosthodontist). Panel discussion and case presentation with Prosthodontist, Periodontist, General Dentist and OMFS.

Topics: Decision making process in implant dentistry. Choice of the treatment configuration from implant positioning to restoration.
Cases: 1 implant; Multiple implants (3-4 unit bridges on multiple implants); Full arch

Cost & other details: Members- free, Students - $55, Online members - $110, Non-members - $190

Meeting name: Dinner meeting and online broadcasting

Meeting date & time: 07 February 2023 at 6.00 PM

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

Speakers: Dr David Attia (GP Sydney) and Mr Russell Young (Laboratory technician)

Topics: Maximising aesthetics. Clinical and technical aspects.

Cost & other details: Members- free, Students - $55, Online members - $110, Non-members - $190

Meeting name: Dinner meeting and online broadcasting

Meeting date & time: 28 March 2023 at 7.00 pm

Meeting location: Osteo Medical 767 Springvale Rd, Mulgrave VIC 3170

Speakers: Dr Fadi Yassmin (NSW) ; Osteon

Topics: Full arch digital workflow. Who is leading the digital workflow? Scientific and practical. Full arch topic. Lab topic: Full arch scanning

Cost & other details: Members- free, Students - $55, Online members - $110, Non-members - $190

Meeting name:

Meeting date & time: May-June 2023

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

Speakers: TBA

Topics: Joint meeting with Periodontic and Prosthdontic societies.

Cost & other details: Members- free, Students - $55, Online members - $110, Non-members - $190

Due to the impact of COVID-19, please check your state branch website for the most up to date event information.

AOS Victoria Committee Details and Meetings (cont'd)

Meeting name:

Meeting date & time: TBA

Meeting location: Online 2x45 mins lectures

Speakers: Jessy Green – How to talk to the patient about implants.

Topics: Dr Gabriel Rodrigues Ortiz – How to integrate the implants to your dental practice.

Cost & other details: Members- free, Students - free, Online members - free, Non-members - $50

AOS WA Committee Details and Meetings

President: Dr Tony Strangio

Secretary: Dr Frances Denney

Treasurer: Dr Richard Williams

Federal Councillor: Dr Roy Sarmidi

Email: infowa@aos.org.au

Meeting Name: AOS WA Dinner Meeting

Meeting date & time: Friday 18 November 2022 6.30pm

Meeting location: The University Club of WA

Speakers: Prof Alex Quaranta

Topics: TBA

Cost & other details: www.aos.org.au

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.

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