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Lisa Sullivan

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

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Editor's Notes

It is with great pleasure to present the Editorial Report for the first quarter of 2025. First, I'd like to thank all our contributors, reviewers, and editorial team for their hard work and support. We are immensely grateful to our dedicated reviewers who have generously contributed their time and expertise to the review process to maintain its high standards.

For this journal issue, we have published two articles. The first article by *Dr Oleg Pushkarev* (University of Queensland), is titled *'Clinical implications of maxillary incisor implants and an angled screw channel concept as a restorative solution.'* This is an important review to address the challenges of replacing a single anterior maxillary incisor with an implant-supported restoration, considering anatomical bone constraints. It also examines the use of angulated screw channel designs and the limited clinical data on their complications and performance.

The second article, by *Dr Miriam Lee* (University of Queensland), is a narrative review on the bidirectional association between periodontitis and rheumatoid arthritis. The author examines recent studies on the role of periodontal keystone bacteria in the pathogenesis of rheumatoid arthritis, potential immunological mechanisms linking the two conditions, as well as the effects of periodontal therapy on rheumatoid arthritis and the influence of rheumatoid arthritis treatment on periodontal health.

I find the reviews in this issue to be very informative and helpful in understanding interesting topics in periodontology and implant dentistry. I hope you enjoy reading them as well.

In closing, I'd like to thank Geistlich Pharma, Megagen, Straumann and ZimVie for their support for the journal. We look forward to your continued partnership in the years to come.

Regards,

A/Prof Ryan Lee
Editor-in-chief

President's Notes

It is my great honour to report to our society in the role of Federal President of the Australian Society of Periodontology (ASP) for the term spanning 2024 to 2026. I would like to express my sincere gratitude to the Perio Society for their trust and unwavering support. I stepped into this position marked by the exceptional leadership in the past that has defined ASP since its inception in 1963.

On behalf of the Society, I extend my heartfelt thanks to the immediate past president , A/P Ryan Lee, for his outstanding stewardship from 2022 to 2024. His tenure culminated in a memorable showcase at the 2024 ASP/AOS/APS Gold Coast Conference—an event that truly exemplified his dedication and vision. I am pleased to note that Ryan continues to contribute meaningfully to ASP through his ambassadorship with the European Federation of Periodontology (EFP) and his editorial role with the Australian Journal of Periodontology and Implant Dentistry (AJPID).

Cherishing on ASP's successful collaborations with the AOS and APS, we resolved to return to our foundational principles by reinstating our biannual federal ASP conference. I am delighted to announce that the inaugural event under this framework will take place in Perth, Australia, in September 2026 under the theme "Perio Launchpad; Boosting dentistry". Scientific and logistical planning is already underway, and we are committed to delivering a program worthy of ASP's esteemed reputation.

Additionally, I am thrilled to share that ASP's modernized website redesign is progressing well. This initiative reflects our commitment to innovation and accessibility, and I extend my deep appreciation to Dr. Luan Ngo and past leadership for their tireless efforts in driving this transformative project.

As we embark on this new chapter, We, together with our state branches are energized by the opportunity to collaborate with our members, partners, and global colleagues to advance periodontal science, education, and clinical excellence. Together, we will ensure ASP continues to lead with integrity and purpose.

Regards,

Mehdi Valizadeh

Dr Mehdi Valizadeh
ASP Federal President

President's Notes

On behalf of the AOS, I would like to extend my sincere gratitude to Dr. Angelos Sourial and the scientific committee for delivering an outstanding conference on the Gold Coast in September 2024. This event marked the first collaboration between AOS, ASP, and APS, and it was a tremendous success.

Looking ahead, the AOS and APS are now actively planning the 2026 conference, set to take place in Melbourne in May 2026. I would also like to acknowledge the incredible behind-the-scenes efforts of Bella Cherkasskaya, whose dedication was instrumental in making the 2024 conference a success.

As I step into my role as AOS Federal President, I am committed to serving our community and working tirelessly to make the upcoming conference our best one yet. I look forward to what lies ahead and to continuing to elevate our shared vision.

Finally, I would like to congratulate all the authors who have published in this issue of the journal—your contributions are invaluable. Wishing everyone an insightful and enjoyable read!

Regards,

Dr Jonathan Ng
AOS Federal President

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Upcoming Hands-On Courses and Events 2025

PROF. EM. DR. DANIEL BUSER (CH)

LECTURE

6 CPD HOURS

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A Day with Dani Buser

35 Years of GBR in Implant Dentistry: Surgical Key Factors to Achieve Successful Outcomes with Good Long-term Stability.

In this 1-Day Seminar, Prof. Buser will present all aspects of GBR, based on 35+ years of surgical experience. In addition, the three treatment options of post-extraction implant placement will be discussed including the concept of:

- Immediate implant placement with a flapless approach, computer-assisted implant surgery (CAIS), internal grafting and immediate restoration
- Early implant placement with GBR 4 to 8 weeks post extraction and spontaneous socket healing
- Socket grafting post extraction for alveolar ridge preservation (ARP) followed by a delayed implant placement after 4 to 6 months, most often flapless and computer-assisted.

Sydney - Saturday, 12 April 2025, Park Hyatt Sydney

DR MARKUS TROELTZSCH (DE)

Hands-On WORKSHOP

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Contemporary Management of Challenging Dentoalveolar Defects

This full-day workshop will present a variety of surgical options available for reconstructing hard and soft tissues in challenging cases. The role of customised bone regeneration (e.g., Yxoss CBR®) will be discussed, as will the importance of selecting correct biomaterials to help maximise long-term regenerative potential.

This full-day workshop will cover:

- CBR and case selection • Applying CBR – planning, flap design, and titanium mesh seating
- Soft tissue release • Primary tension-free wound closures suturing techniques

Sydney - Monday, 4 August 2025, Park Hyatt Sydney
Perth - Wednesday, 6 August 2025, The University Club

DR EUGENE SHEFTEL (VIC)

Hands-On WORKSHOP

3 CPD HOURS

$445

Mastering Ridge Preservation - Improving Patient Outcomes

This workshop, led by Dr. Eugene Sheftel, will explore the evidence for ridge preservation techniques and provide hands-on practice on simulation models. Participants will gain practical skills and knowledge to integrate these procedures into their clinical practice.

Key topics include:

- Ridge Preservation: Scientific evidence and surgical principles
- Managing Partially Edentulous Sites: Pre-extraction and post-extraction considerations
- Role of Biomaterials in Ridge Preservation: Properties and applications

Melbourne - Friday, 22 August 2025, Element Hotel

SAVE THE DATE

GEISTLICH MINI-SYMPOSIUM

LECTURE

We're excited to announce that the Geistlich Mini Symposium will be held at the Park Hyatt Sydney! Don't miss the opportunity to hear from industry experts and discover the latest advancements in bioregeneration. Registration opens soon—stay tuned!

Sydney - Friday, 24 October 2025, Park Hyatt Sydney

To register please visit dentevents.com.au or contact Belinda at education@geistlich.com.au

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The Australian Journal of Periodontology and Implant Dentistry

Clinical Implications of Maxillary Incisor Implants and an Angled Screw Channel Concept as a Restorative Solution

Oleg Pushkarev
The University of Queensland, School of Dentistry, DClinDent (Prosthodontics)

Challenges of the anterior maxilla

Implant rehabilitation of a missing anterior tooth has proven to be a valid and predictable treatment option. The successful aesthetic outcome of an implant-retained restoration depends on the soft tissue contour and a three-dimensional positioning of an implant[19]. The appearance of soft tissue, apart from its biotype, primarily relates to the height of a bony crest at adjacent teeth and the thickness of the buccal bone[4].

It is well known that post-extraction changes for anterior incisors and premolars in the maxilla result in a marked reduction of the buccal and marginal aspects of the alveolar bone, potentially eventuating in the formation of a triangular-shaped cross-section of the ridge[29]. A study by Kan et al. (2011) demonstrated the prevalence (76-86.5%) of Class I ridges in the anterior maxilla, suggesting the use of palatal bone for the implant placement[25]. Less favourable ridges constituted the minority, however, significantly complicating implant placement in the anterior maxilla. A CBCT-based study by Gluckman, et al. (2018) attempted to classify the radial tooth position and anterior bony wall dimensions[15]. In most (82-95%) cases, a thin <1mm facial bone prevailed. A retroclined incisor group, was found in 76.5% of cases. The authors stated that this type of incisal root angulation would best suit the palatal positioned osteotomy. This, however, may challenge the ideal three-dimensional position for the implant proposed previously in the literature[4,19].

"Ideal" position for an anterior maxillary implant

Buser et al. (2004) defined comfort zones for implant placement, facilitating aesthetic restorative outcomes[4]. One of the parameters is the mesiodistal dimension and the distance to the adjacent teeth, which was recommended to be at least 1.5mm for tissue-level implants. The orofacial dimension defines an implant shoulder position

Abstract:

Replacement of a single anterior maxillary incisor with an implant-supported restoration often poses a significant challenge due to anatomical constraints of the available bone. Placement of a dental implant palatal to the incisal edge was recommended to facilitate a cingulum screw-channel access, allowing screw retention for the implant crown. In majority of cases, this position requires delayed implant placement and augmentation. Clinical implications of the resultant emergence profile has been previously discussed in the literature. Changing the angle of a dental implant to coincide with the long axis of a maxillary incisor or engaging the remaining palatal alveolar bone and reducing the extent of bone augmentation can be used in some clinical scenarios. However, this implant position requires an angled screw channel design in the implant crown to facilitate a screw-retained restoration. Limited clinical data are available on complications and clinical performance of angulated screw channel restorations.

in the buccal comfort zone as at least 1mm palatal to the line connecting emergence profiles of adjacent teeth. If, however, the buccal implant shoulder is positioned more than 2mm palatal from this line, it could result in restoration with a ridge-lap design. Over-contoured in any dimension, implant restorations promote plaque accumulation, make oral hygiene procedures difficult, complicate periodontal probing and could lead to peri-mucositis and peri-implantitis[8].

A restorative (emergence) angle is often used to describe the subcritical contour of the restoration, measuring the angle formed between a line parallel to the implant axis and a line tangent to the highest point of the proximal contour of the restoration. A prospective single cohort study by Strauss et al. (2022) assessed bone loss in anterior maxillary and single mandibular implants, restored with zirconia crowns on titanium bases with various restorative angles. A greater restorative (emergence) angle, exceeding 40 degrees, has been associated with increased marginal bone loss within the first year[36]. Limited evidence suggests a narrow, concave emergence profile with an emergence angle below 30 degrees is more favourable for prevention of early bone loss and potentially reducing the risk of peri-implant disease[28].

If a dental implant is located off-centre in the mesiodistal dimension, this will impact the restorative contours of the crown to achieve interproximal contact[8]. Similarly, the discrepancy in labiolingual position (palatal placement) may affect hygiene measures for the patient. At this stage, no solid evidence shows the detrimental effects of over-contoured implant restorations in the buccolingual dimension. However, if such restoration is cemented, it could result in significant difficulties in removing the residual cement and promoting further plaque retention[35].

Implant design and its effect on the emergence profile

In the past decade, dental implants with a platform-switching concept have become more prevalent, allowing better tissue stability and flexibility in three-dimensional placement. By implementing an abutment narrower than the implant platform, the microgap at the connection interface is moved further away from the bone, allowing for more biological width[8].

This allows intentional change in the vertical position of the implant, modifying the resultant emergence profile. For instance, a shallow-placed implant will result in more significant buccal undercut. In contrast, a subcrestal implant placement will result in a less angled emergence profile, facilitating a gradual development of the crown contour and easier access to oral hygiene.

In the natural maxillary incisor, the cervical contour is convex and represented with enamel overlapping the root cementum. This constitutes an emergence angle between the long axis of the tooth and a tangent to the point of emergence at the gingival level. According to a recent study by Du et al., this angle is 15 degrees on average[10]. The buccolingual position and depth of a dental implant directly affect the convexity of the cervical contour. For instance, the more palatal an implant is, the greater the emergence angle, and vice versa[38].

Testori et al. (2018) argue that traditional guidelines on ideal implant positioning (at the cingulum) were developed on tissue-level and platform-matching implants, therefore, would lead to a subgingival contour of the crown significantly different to a natural tooth, resulting in a more pronounced buccal ridge-lap[38]. The authors discuss that positioning the centre of the implant under the incisal edge will result in the emergence angle more closely resembling the natural tooth. This would lead to the necessity of fabricating a cement-retained crown, and obtuse subgingival angles will facilitate benefits in easier cement removal.

The risk of undetected cement increases with the more apical location of a crown-to-abutment interface. A strong association (81%) between the incidence of peri-implantitis in cemented implant restorations was found by Wilson et al.[40]. Cement residue, being a rough surface, facilitates biofilm accumulation and hampers biofilm removal[28].

Therefore, anatomical constraints in the anterior maxilla and the natural shape of incisors often dictate the facial angulation of an implant following the palatal osteotomy. Traditionally, this has been solved by utilising angled abutments and cemented restorations or placing the implants more palatal to facilitate the cingulum direct screw channel access. More recently, a method of redirecting the screw access has been described in the literature by Beroeta et al., allowing a retrievable screw-retained implant restoration on a buccally tilted implant[2].

Angled screw channel restoration: Is it a potential solution or a new challenge?

The debate in the literature on angled abutments and non-axial stress on the surrounding bone resulted in numerous studies[24,39]. Even though increased stress was recorded, it did not result in a scientifically proven reduction of the longevity of restorations or increased bone loss. This coincided with mounting evidence of peri-implant inflammatory complications resulting from cement residue. Therefore, utilising an angled abutment with an angled screw channel became a promising solution to many restorative challenges and allowed the prevention of some biological complications.

The concept of a novel abutment design, allowing a change in the angulation of a screw channel up to 28 degrees, was proposed in 2004 by Esteban Xam-mar, a founder of Dynamic Abutment. However, the idea did not gain significant attention until 2014[2]. Angulated Screw Channel (ASC) can be applied to CAD/CAM monolithic and veneered Zirconia restorations and conventional porcelain fused to metal ones[33]. The authors used a CAD/CAM-modelled custom abutment from Heraeus Kulzer. However, long-term clinical evidence remains scarce.

A CBCT observational study revealed that 76% of maxillary incisors would require an angled abutment if replaced with an implant-retained prosthesis[11]. Authors reported that 71% of sites required 5-degree angular correction, 27% required 10-degree modification, and only 1% required 15-degree angulation. Lateral incisors presented a greater need for angled screw-channel abutments. However, one limitation of this study is that the depth of a final position of a screw will affect the required angle and the depth of implant placement to the CEJ of the tooth. The other limitation was that all virtually planned implants were positioned in the "ideal" position relative to the tooth morphology, ignoring the ridge anatomy. Immediate placement protocol and reduction of alveolar ridge post-extraction would dictate the palatal position of the fixture and, thus, result in more significant angle corrections.

Another recent CBCT study by Kan et al. (2023) of 200 patients discussed a possibility of immediate implant placement in the anterior maxilla with immediate provisionalization[26]. The inclusion criteria was the engagement of at least 35% of the implant with 1mm of surrounding bone. Authors found that the majority of cases (66-77%) will require an angle screw channel restoration, whereas a straight screw channel access would be possible in 10-24% of cases.

Clinical performance of Angled screw channel restorations

The first critical outlook on the performance of angled screw channel restorations assessed the mechanical complications of the Nobel Biocare ASC system[18]. Out of 99 restorations evaluated over three years, two returned with mechanical complications: screw loosening and porcelain fracture. The authors acknowledged that even though complication rates were comparable to conventional screw-retained restorations, the study suffered from a limitation of a relatively short follow-up time. An important notable feature of Nobel ASC is a custom-made abutment on a titanium base, where the angled screw clamps the abutment to the titanium insert.

An attempt to increase the area of cement retention between the abutment and the crown was described by Sakamoto et al. (2018)[34]. Satoshi Sakamoto's abutment consisted of a custom-milled titanium insert of the maximum possible height on the interproximal and a zirconia coping with a palatal screw channel. The authors noted better light transmission through the zirconia coping as an additional advantage. A core3D angle screwdriver was used for the screw access.

An application of Nobel ASC was reviewed in a prospective study involving 42 implants of the anterior maxilla[12]. A short follow-up time of 1 year did not reveal any prosthetic complications. The amounts of bone loss around fixtures were in-line with similar other reports, and the cumulative survival rate of implants was 98%. A similar study reviewed outcomes of full-contour zirconia restorations with angled screw channels (Nobel Biocare, ASC) in 1 year[32]. No prosthetic complications were encountered, given a small sample size and a short follow-up period.

A split-mouth study compared the marginal bone loss of CAD/CAM-designed screw-retained restorations with and without angle correction[1]. The results did not reveal statistical differences between groups over 39.65± 15.2 months of follow-up. A significant limitation of the study was the inclusion of splinted abutments, where angulated

screws were used in one abutment and non-angulated in the other abutment of the bridge.

Another study with a 2-year follow-up also reported a high success rate (94%) of ASC concept, including monolithic and veneered zirconia restorations[33]. The Kaplan-Meier survival analyses presented in this paper indicated that monolithic zirconia restorations exhibit a higher success rate, showing greater resistance to mechanical complications.

A recent systematic review and meta-analysis investigating the mechanical and biological complications of angled screw channel restorations revealed a comparable performance to straight screw channel (SC) restorations with a moderate level of evidence based on the wide range of assessed parameters, follow-up periods and a limited number of long-term studies[7]. ASC implant-supported prostheses demonstrated similar outcomes to SC prostheses regarding marginal bone loss (MBL) development and aesthetic performance, as evaluated by the Pink Esthetic Score (PES).

Mechanical complications

Mechanical complications are a significant concern in the long-term success and functionality of implant-supported restorations. These complications, which encompass issues such as screw loosening, fractures of the framework or prosthetic components, and wear of restorative materials, can compromise the stability, longevity, and clinical outcomes of the prosthesis. To date, no clinical studies reported fractures in monolithic zirconia ASC restorations. However, a laboratory study of two-piece zirconia abutments revealed a nearly two-fold difference in the maximum load before failure in straight abutments compared to ASC[13]. In this study, however, the minimal thickness of zirconia was 0.4mm at the abutment level in the angled screw channel group. The was no discussion of the minimal thickness in the control group. It should also be noted that loading exceeded the physiological range.

In a different study by Mulla et al. (2021) application of 200N loading in a fatigue testing scenario revealed fracturing of the friction-fitted two-piece zirconia abutments at approximately 2500 cycles[30]. The design of the crown for Nobel Biocare universal base in this study was notably different to the other titanium abutments, being thinner at the apical level. This affected the observed mode of failure, as crowns designed with thicker zirconia walls would instead fracture the titanium abutment at 400000-500000 cycles. Similarly, an importance of the thickness of the zirconia in the cingulum area, being a weak point in angled abutments, explained fractures of all ASC zirconia abutments and 4 out of 5 straight zirconia abutments in a study by Drew et al. (2020)[9].

One of the most frequent (6%) prosthetic complication of screw-retained restorations is screw loosening[23]. A laboratory study on reverse torque values for 10- and 20-degree angulations for abutment screws revealed significant statistical differences between straight and angulated screws, with the 20-degree group demonstrating the lowest reverse torque value. Similarly, another study confirmed the lowest reverse torque values for 28^0 angled Dynamic abutments[16]. A reduced reverse torque value of angled (25^0) screw channel abutments was reported after applying cyclic loading[30]. Similar conclusions were drawn from another study employing cycling loading at a lower magnitude (40N)[6]. The authors also noted the most significant wear on the screw in the ASC group with 25^0. Further reduction of reverse torque values has been reported in the presence of saliva, which, being a lubricant, may have a particular impact on screw loosening of screw-retained restorations[21,27].

A study by Cakmak et al. (2023) compared reverse torque loss between Straumann CrossFit and TorqueFit connections, in straight and angle screw scenarios[5]. The difference in reverse torque was more significant between the different connections, rather than between straight or angled screws. Similarly, in a study by Bhumpattarachai et al. (2023) the loss in preload was found to be related to a screw design, rather than an angle of screw engagement[3]. A tapered-head screw performed better than the flat-head ones.

More recent study by Hahn et al. (2024) found no statistically significant reduction in reverse torque values in 25-degree angled screw channel abutment compared to the straight one following the 5000 cycles of 155N loading[22]. Additionally, a micro-CT study was performed on 4 random samples, demonstrating a similar amount of counter-clockwise rotational micromovement of an abutment and a screw within the connection following the non-axial loading.

Overall, most authors agree that the stability of preload is inherent within the design for the implant-abutment connection, screw thread geometry and a screw head design. However, lower reversal torque values may not necessarily directly translate to higher incidence of screw loosening.

Conclusion

In summary, the anterior maxilla poses significant challenges in successful and aesthetic implant rehabilitation. Anatomical constraints often significantly affect implant position, resulting in possibly suboptimal prosthetic designs prone to plaque retention and subsequent biological complications. The best three-dimensional placement of the implant, allowing primary stability and lesser emergence angles, mimicking the natural tooth, will often result in a screw channel on the incisal edge or the buccal surface. Angled screw-retained restorations are preferable in such scenarios due to lesser risks of biological complications compared to a cement-retained restoration.

Short-term clinical evaluations of angled screw-retained restorations reveal a favourable prognosis on par with that of restorations featuring straight screw access. Technical complications of angled screw channels are rarely reported in the literature and mainly include screw loosening. There is, however, lack of evidence that angled screw restorations lead to more frequent screw loosening. The existing long-term clinical data on the performance of ASC is still lacking.

The current literature lacks practical recommendations for predictable treatment planning of an angled implant to replace a maxillary incisor. Thus, if a practitioner uses the traditional "ideal" parameters for implant planning but changes the angulation of the implant to match the maximum for the ASC, the resultant necessary angular correction may be more significant than ASC abutments could correct. These aspects require further investigation to improve the restorative predictability of single anterior implants and reduce the incidence of technical and biological complications.

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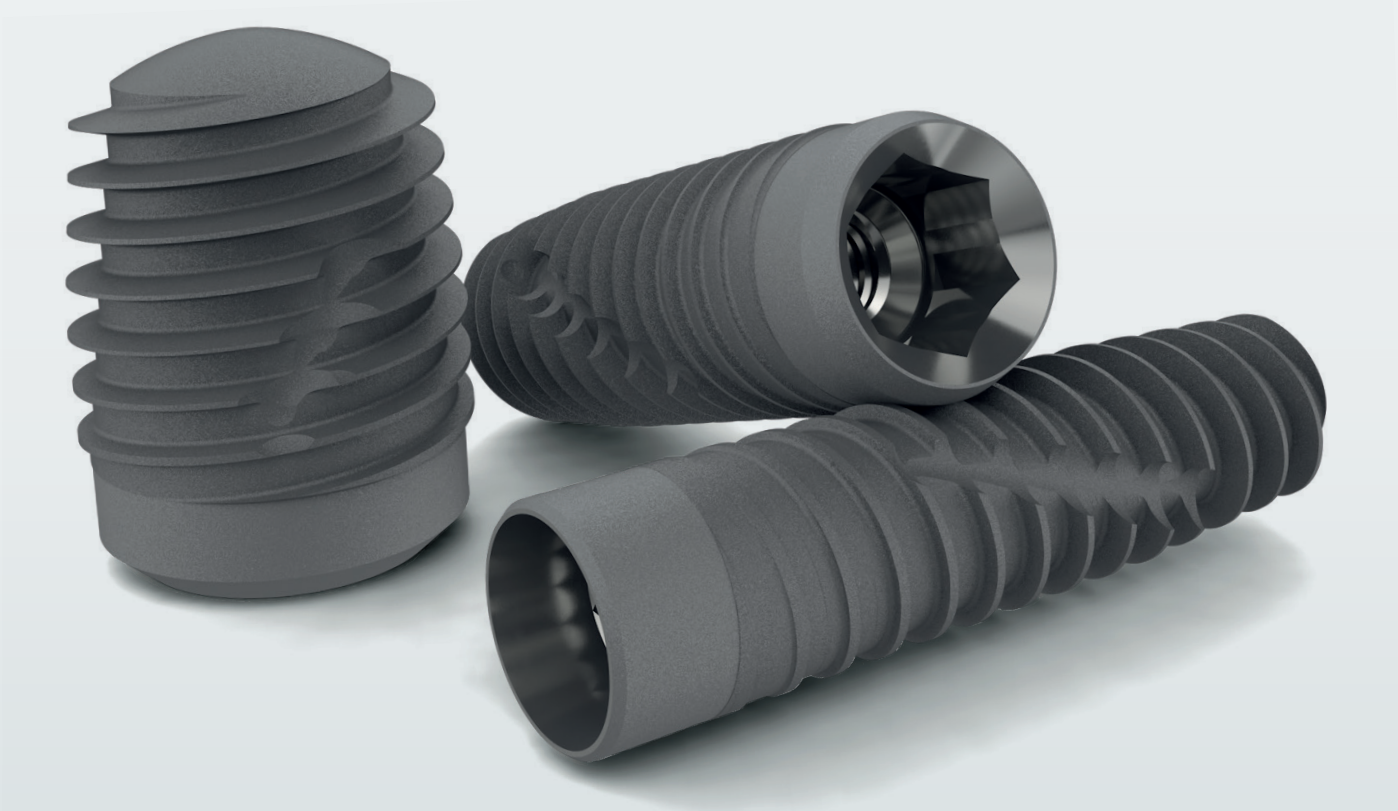

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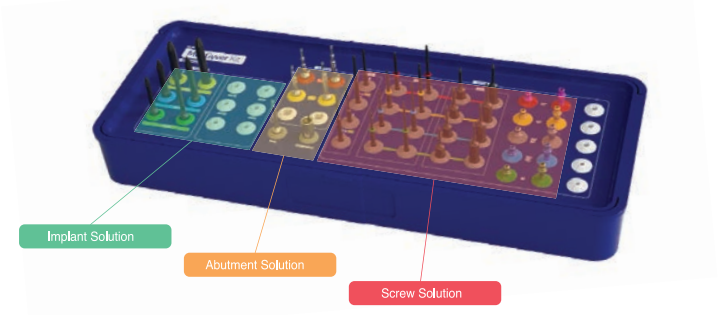

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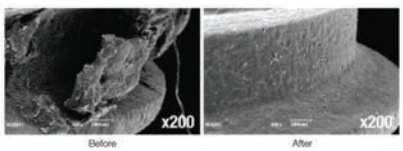

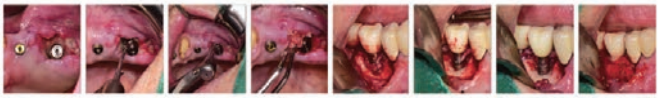

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The Bidirectional Relationship Between Periodontitis and Rheumatoid Arthritis – Where Do We Stand Now? A Narrative Review

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Abstract:

Periodontitis and rheumatoid arthritis are chronic inflammatory conditions that may share underlying pathophysiological mechanisms. This narrative review examines the most recent studies exploring the role of periodontal bacteria in the pathogenesis of rheumatoid arthritis, the immunological pathways potentially linking these two conditions, and the bidirectional impact of treatment—specifically, the effects of periodontal therapy on rheumatoid arthritis and the influence of rheumatoid arthritis treatment on periodontal health.

Introduction

Periodontitis and rheumatoid arthritis (RA) are both chronic inflammatory conditions affecting millions of people worldwide. While periodontitis primarily affects the periodontium, leading to the destruction of alveolar bone and connective tissue, RA is a systemic autoimmune condition targeting synovial joints. Despite affecting different body parts, both conditions share underlying immune system dysfunction.

In recent years, the potential link between these diseases has gained increasing attention, particularly in understanding the role of periodontal bacteria in RA pathogenesis, shared immunological pathways, the effect of periodontal treatment on RA and the effect of RA treatment on periodontal health. This narrative review will explore the latest studies on this connection, including bacterial involvement and proposed mechanisms such as molecular mimicry, post-translational protein modifications, and immune system activation that may underpin the bidirectional relationship between these diseases.

Rheumatoid Arthritis: A Brief Overview.

RA is a chronic, systemic autoimmune disease that primarily affects synovial joints, leading to progressive inflammation, cartilage destruction, and bone erosion over time. The severity of RA varies from person to person, with symptoms ranging from mild discomfort to debilitating pain, significantly impacting patients' quality of life. Joint symptoms include pain, swelling, stiffness, redness, and warmth, often occurring symmetrically – meaning both sides of the body are typically affected at the same time. If left untreated or unmanaged, RA can lead to joint deformities and reduced mobility. While RA predominantly affects the small joints of the hands, wrists, and feet, larger joints such as the knees, elbows, hips, and shoulders can also be involved. Systemic

symptoms include fatigue, fever, and weight loss, while extra-articular manifestations encompass ocular dryness and irritation, rheumatoid nodules, interstitial lung disease, and vasculitis (1, 2).

The 2010 American College of Rheumatology (ACR)/ European Alliance of Associations for Rheumatology (EULAR) classification criteria are widely used to facilitate early RA diagnosis and intervention with disease-modifying antirheumatic drugs (DMARDs). These criteria incorporate a scoring system based on joint involvement, serological markers such as rheumatoid factor (RF) and anti-citrullinated protein antibodies (ACPA), acute-phase reactants, and symptom duration. Importantly, RA diagnosis requires the exclusion of other types of arthritis to ensure diagnostic accuracy (3).

The global incidence and prevalence of RA vary due to genetic, environmental, and socioeconomic factors. In Western nations, the prevalence ranges from 0.5% to 1%, whereas it is lower in developing countries (4, 5). A recent meta-analysis of 67 studies estimated the global prevalence at 0.46% (95% CI of 0.39-0.54%) (6). A population-based cohort study in Western Australia reported an RA prevalence of 0.34% based on hospital records and 0.36% based on biological therapy usage, with an adjusted increase to 0.72% for 2010–2014 when corrected for DMARD utilisation (7). The impact of COVID-19 on RA incidence has also been investigated. A global federated health research network study reported an incidence of new-onset RA at 0.14% among COVID-19-diagnosed patients, compared to 0.147% in controls. However, reliance on electronic health record-derived data introduces the possibility of misclassification, particularly among asymptomatic COVID-19 cases (8).

A systematic analysis by the Global Burden of Disease (GBD) 2021 RA Collaborators examined RA epidemiology from 1990 to 2020, projecting trends to 2050. In 2020, an estimated 17.6 million individuals had RA, with a global prevalence rate of 208.8 cases per 100,000 population, marking a 14.1% increase since 1990. Despite this, the global mortality rate associated with RA decreased by 23.8% from 1990 to 2020, with a rate of 0.47 per 100,000 population. By 2050, the global RA prevalence is projected to rise to 31.7 million cases (9). These global trends align with Australian projections, where RA prevalence is expected to increase from 422,309 cases in 2015 to 579,915 by 2030, accompanied by direct healthcare costs exceeding $755 million (10).

While advancements in treatment have contributed to a decline in RA severity, the increasing prevalence remains a significant concern. Variability in incidence trends across studies may be attributed to differences in study methodologies. Additionally, disparities in access to diagnostic and therapeutic services, particularly in developing countries, pose challenges in generalising study findings and ensuring equitable RA management worldwide (4, 5).

The Link Between Periodontitis and Rheumatoid Arthritis

The association between RA and periodontitis has been extensively explored by researchers, with multiple studies indicating an increased prevalence of periodontitis among RA patients compared to healthy controls. However, findings have varied due to differences in study designs, populations, and diagnostic criteria. Several observational studies have demonstrated a higher prevalence of periodontitis in RA patients. A case-control study in Argentina found that individuals with RA had a significantly higher prevalence of severe periodontitis compared to controls (12% vs. 4%) (11). Similarly, a Korean case-control study reported a greater prevalence of periodontal diseases among RA patients, though no significant difference was observed between seronegative and seropositive RA groups (12).

Cross-sectional studies reinforce these findings. A study conducted in China reported periodontitis prevalence of 51.5% among RA patients, compared to 31.2% in healthy individuals, with disease severity correlating with age and RA duration (13). In Vietnam, RA patients exhibited a markedly higher prevalence of periodontitis (67% vs. 28%) compared to osteoarthritis patients, with severe periodontitis affecting 22.7% of RA patients compared to 8% in the osteoarthritis group (14). Similarly, Renvert and colleagues found that periodontitis was significantly more frequent in RA patients (61.1%) than in controls (33.7%, p=0.001) (15).

Conversely, studies also indicate that periodontitis may increase the risk of developing RA. A longitudinal study in Korea followed participants for a median of 16.7 years and found that individuals with periodontitis had a 20% increased risk of developing RA (HR = 1.2), with a higher risk observed in those with more than 15 missing teeth (HR = 1.5) (16). Another population-based cohort study in South Korea reported a slightly increased incidence of RA in the periodontitis group (16.8% vs. 15.4% in controls), with an adjusted hazard ratio of 1.09 ($p < 0.001$) (17). Similarly, a retrospective longitudinal study found an 18.7% prevalence of RA among individuals with periodontal disease, compared

to 11.2% in those with healthy periodontium ($p < 0.001$), yielding an odds ratio of 1.16 (18). Beyond prevalence, periodontitis has also been linked to increased RA disease activity. A Vietnamese study found that RA patients with periodontitis had higher disease activity scores (DAS 28), increased ACPA positivity, and elevated C-reactive protein (CRP) levels. The study concluded that periodontitis was associated with a 5.14-fold increased risk of RA (95% CI: 3.14–8.41) and a 2.7-fold increased likelihood of higher RA disease activity (95% CI: 1.14–6.42) (14).

However, not all studies have confirmed a strong association. An observational cross-sectional study utilising the US National Health and Nutrition Examination Survey (2009–2014) found that RA was associated with non-functional dentition but showed no significant link between RA and periodontitis when fully adjusted for confounding factors, including demographics, lifestyle, and comorbidities (19). Likewise, a Colombian cross-sectional study found no significant association between RA and periodontal clinical parameters. Interestingly, this study reported that RA patients without periodontitis had higher ACPA levels, suggesting that periodontal disease may not always be a determinant in RA severity. However, the study acknowledged that RA patients with reduced periodontium might have had sequelae of prior periodontitis rather than ongoing active disease (20)

Most studies suggest a bidirectional association between RA and periodontitis, with RA patients more likely to develop periodontal disease and individuals with periodontitis having a higher risk of developing RA. Nevertheless, variations in study design, diagnostic criteria, and confounder adjustments contribute to inconsistencies in findings. Standardising diagnostic protocols and controlling for factors such as smoking, socioeconomic status, and access to healthcare are crucial for refining our understanding of this relationship.

Periodontal Bacteria and Rheumatoid Arthritis

Periodontal disease is gaining recognition for its potential role in the onset and progression of RA. Evidence suggests that periodontal pathogens, particularly *Porphyromonas gingivalis* and *Aggregatibacter actinomycetemcomitans*, may be responsible for inducing systemic inflammation and autoimmunity, key processes in RA pathogenesis.

A recent meta-analysis identified a significant increase in RA risk among individuals exposed to *P. gingivalis*, with a pooled OR of 1.86. Notably, regional differences were observed, with higher ORs in European and North American populations compared to Asia. However, the study reported substantial heterogeneity in study designs, population characteristics, and methods for assessing *P. gingivalis* exposure and RA diagnosis, which may affect the generalisability of the findings (21).

A cross-sectional study in Türkiye investigated the microbial composition of subgingival plaque in RA patients and healthy controls. From this study, they found that RA patients with moderate to high DAS28 scores exhibited poorer oral health conditions, with a higher prevalence of *Treponema denticola*. The presence of cyclic citrullinated peptide (CCP) antibodies was associated with *T. denticola* and *Campylobacter rectus*, while CRP levels correlated with *Capnocytophaga gingivalis* and Epstein-Barr virus (22). Another Swedish study (15, 23-25) analysed anti-*P. gingivalis* antibodies in serum and saliva and their association with RA and periodontitis. The study found that salivary IgA antibodies against arginine-specific gingipain B (RgpB) of *P. gingivalis* were significantly higher in RA patients than healthy controls, even after adjusting for age, gender, smoking, and ACPA levels. However, anti-RgpB antibodies were not associated with periodontitis or serum IgG ACPA (26). Similarly, a Colombian study reported the presence of *Porphyromonas gulae* in 15.8% of RA patients compared to 9.5% of controls. While ACPA levels were higher in *P. gulae*-positive RA patients, the difference was not statistically significant. However, a strong correlation was observed between *P. gingivalis* and elevated antibodies against citrullinated proteins, reinforcing its potential role in RA pathogenesis (27). In this research group's other study, they identified a significant association between RA diagnosis and the presence of RF, ACPAs, and double-positive antibodies against *P. gingivalis* arginine-gingipains (RgpA) and peptidyl arginine deiminase produced by *P. gingivalis* (PPAD). The combination of anti-RgpA/anti-PPAD antibodies demonstrated high specificity (93.7%) and positive predictive value (82.5%) for diagnosing RA, suggesting its potential as a biomarker for RA associated with periodontal disease (28).

A longitudinal study assessing the link between serum antibodies to periodontal pathogens and RA risk found no significant differences in anti-*P. gingivalis*, *Fusobacterium nucleatum*, or *Prevotella intermedia* antibody levels between pre-RA patients and controls. However, among RA patients, anti-*P. intermedia* antibody levels were positively associated with several RA autoantibodies (e.g., anti-CCP2, anti-vimentin, anti-histone, anti-α-enolase, IgA RF,

IgG RF, and IgM RF) before RA diagnosis. These findings suggest that *P. intermedia* may play a role in preclinical RA progression, although the study did not assess periodontal disease status, limiting direct correlation (29). With *A. actinomycetemcomitans* and RA, an analysis using two prospective cohorts (30, 31) found no significant differences in anti-leukotoxin A serum levels between RA-at-risk patients and controls. However, in the British cohort, anti-leukotoxin A positivity was more frequent in ACPA-positive arthralgia patients. In the Swedish cohort, anti-leukotoxin A-positive patients had a higher risk of progressing to arthritis, though this association was not observed in the UK cohort. These population-specific variations suggest potential geographical differences in the microbial-immune interactions influencing RA development (32).

A Taiwanese study comparing subgingival microbial composition in RA patients and healthy controls found significant differences, particularly in *Aminipila butyrica* and *Peptococcus simiae*, which correlated with ACPA positivity in RA patients with periodontitis. However, the study focused on only two bacterial species, potentially overlooking broader microbial interactions (33). Similarly, a study in Indonesia examined the relationship between subgingival microbiota and IgA ACPA levels in gingival crevicular fluid (GCF). While *Porphyromonas* species were linked to local ACPA presence, overall subgingival microbiome composition did not significantly differ between ACPA-positive and ACPA-negative patients. This suggests that the link between periodontal disease and systemic autoimmunity may be more complex than direct microbial shifts (34). A Korean study found that RA patients with preclinical disease had an increased abundance of *Porphyromonadaceae* family bacteria in their subgingival microbiome compared to those with new-onset or chronic RA, despite similar periodontitis severity. In a collagen-induced arthritis mouse model, oral inoculation with *P. gingivalis* and *T. denticola* worsened arthritis symptoms, with differential effects on RF production. Transcriptomic analysis suggested that these bacteria influence B-cell and T-cell responses, potentially driving autoimmunity (35). Not all studies support a direct association between *P. gingivalis* and RA. A Malaysian study found no significant correlation between periodontal disease severity and RA disease activity. Furthermore, P. gingivalis bacterial load varied extensively without a significant association with ACPA levels ($p = 0.58$). However, the study lacked a non-RA control group and did not measure anti-*P. gingivalis* antibody levels, which may have provided additional insights (36).

The reviewed studies highlight the complex interplay between periodontal bacteria and RA, particularly the role of P. gingivalis, A. actinomycetemcomitans, and other oral pathogens in driving systemic inflammation and autoimmunity. While significant heterogeneity exists across studies, growing evidence suggests that periodontal bacteria may contribute to the initiation or exacerbation of RA. Future research should address methodological variability, standardise microbial and immunological assessments, and study broader microbiome-host interactions to refine our understanding of this association.

Mechanisms Underlying the Link between Periodontitis and Rheumatoid Arthritis

The complex relationship between RA and periodontitis has gained increasing attention, with research focusing on the roles of citrullination, inflammation, and the oral microbiome in linking these two conditions. RA patients consistently demonstrate elevated levels of autoantibodies against citrullinated proteins, and the presence of citrullinated proteins in periodontal tissues suggests a potential shared pathogenic mechanism. In particular, *P. gingivalis* has been implicated in the breakdown of immune tolerance through molecular mimicry, post-translational modifications, and immune system activation, contributing to systemic autoimmune responses (37).

Molecular Mimicry: A Pathogen-Induced Autoimmune Response

Molecular mimicry is a well-established immunological concept where microbial proteins closely resemble host proteins, triggering cross-reactive immune responses. This phenomenon is thought to play a role in RA pathogenesis, particularly in cases involving periodontal pathogens such as *P. gingivalis*.

1. Mechanism of Molecular Mimicry in RA and Periodontitis
   - Pathogens such as *P. gingivalis* express proteins that structurally resemble host antigens.
   - The immune system mistakenly targets and attacks host tissues, leading to chronic inflammation and autoimmune responses.
   - This cross-reactivity promotes autoantibody production, including ACPA, a hallmark of RA.

2. Evidence Supporting Molecular Mimicry in RA
   - *P. gingivalis* produces peptidyl arginine deiminase (PPAD), an enzyme capable of modifying host proteins through citrullination.
   - Citrullinated peptides from *P. gingivalis* trigger an immune response that cross-reacts with human citrullinated proteins, potentially driving ACPA formation (37).
   - Epidemiological data link *P. gingivalis* exposure to increased RA risk, particularly in genetically predisposed individuals (38)

A large cohort study from the Epidemiological Investigation of RA demonstrated that RA patients exhibited significantly higher antibody levels against *P. gingivalis* citrullinated peptides than controls. However, due to a lack of periodontal data, a direct correlation with periodontitis could not be established (38).

Post-Translational Protein Modifications and RA Pathogenesis

1. Citrullination - A Central Mechanism:
   Citrullination is a post-translational modification catalysed by PAD enzymes, converting arginine residues in proteins into citrulline. This process plays a key role in RA autoimmunity:

   - In RA patients, citrullinated proteins such as α-enolase, vimentin, and fibrinogen become immunogenic, driving ACPA production.
   - PAD2 and PAD4, the primary PAD enzymes implicated in RA, are also detected in inflamed periodontal tissues (39).
   - *P. gingivalis* produces PPAD, which citrullinates bacterial and host proteins, enhancing the breakdown of immune tolerance (40).

   A study analysing gingival tissues from RA patients found B cells reactive to citrullinated peptides, suggesting a role for periodontal infection in triggering systemic autoimmune responses (38).

2. Role of Carbamylation and Malondialdehyde-acetaldehyde (MAA) abduct formation:
   Beyond citrullination, carbamylation and MAA adduct formation have been implicated in RA pathogenesis:

   - Carbamylation of proteins (CarP) has been detected in inflamed gingival tissues and is associated with higher RA disease activity.
   - MAA-modified proteins also increase in periodontitis-affected tissues, potentially contributing to autoantibody formation in RA (41).

   A pilot case-control study demonstrated that serum CarP levels correlated with periodontitis severity in RA patients and that periodontal therapy reduced these autoantibody levels, highlighting a potential treatment target (42)

Immune System Activation and Chronic Inflammation

1. Th17 Cells and the IL-17/IL-23 Axis:
   The Th17/IL-17 immune axis plays a central role in both RA and periodontitis:
   - IL-17 is predominantly produced by Th17 cells and promotes chronic inflammation by stimulating cytokine production (IL-6, TNF-α) and neutrophil recruitment.
   - Th17 cells differentiate in response to IL-6, IL-1β, and TGF-β, with IL-23 sustaining their activation (43).
   - In RA, IL-17 promotes synovial inflammation and bone erosion, while in periodontitis, it exacerbates gingival inflammation and tissue destruction (44).

2. Neutrophil Extracellular Traps (NETs) and RA Progression:
   NETs are web-like structures released by activated neutrophils to capture pathogens. However, excessive NET formation is linked to autoimmune activation in RA:
   - NETs contain citrullinated histones, which can act as autoantigens for ACPA production.
   - Periodontitis has been associated with increased NET formation, and RA patients have been shown to exhibit higher circulating NET markers.
   - A retrospective case-control study found that periodontal treatment reduced NET markers, suggesting potential benefits for RA management (42).

The Two-Hit Model: Linking Periodontitis and RA

The "Two-Hit" Model, proposed by Golub and colleagues, provides a framework for how periodontal inflammation may act as a precursor for autoimmune disease:

1. First Hit: Periodontal bacteria trigger local inflammation and ACPA production.
2. Second Hit: Systemic factors (e.g., genetic predisposition, smoking) enhance inflammatory responses, leading to RA onset in susceptible individuals (40, 45).

This model aligns with mouse studies demonstrating that pre-existing periodontitis accelerates experimental arthritis progression (46, 47).

The interplay between periodontitis and RA appears to involve complex immunological mechanisms, including molecular mimicry, post-translational modifications, and immune activation. Although substantial progress has been made, further longitudinal studies and interventional trials are needed to fully elucidate the exact mechanisms.

The Effect of Periodontal Therapy on Rheumatoid Arthritis

Due to the shared inflammatory pathways between these two conditions, the potential impact of periodontal therapy on RA disease activity has been an area of growing interest. Researchers have investigated whether improving periodontal health can influence RA disease progression and treatment outcomes. While some studies have reported significant improvements in RA parameters following periodontal therapy, others have found no additional benefit beyond standard RA treatment.

A systematic review and meta-analysis by Mustufvi's group examined 21 studies, including randomised controlled trials (RCTs) and non-randomised studies, assessing the effect of periodontal therapy on RA disease activity. The analysis primarily focused on DAS28 score and key RA biomarkers such as ACPA and CRP. The review found that non-surgical periodontal therapy significantly improved DAS28 scores in the short term, with some studies showing reductions in ACPA levels, suggesting a potential systemic effect of periodontal therapy. However, variability in study designs and bias levels affected the generalisability of the results, with mixed findings on other RA biomarkers. These findings highlight the need for more standardised methodologies to assess the long-term effects of periodontal therapy on RA outcomes (48).

Several RCTs have indicated that periodontal therapy improves both periodontal and RA parameters. Thilagar and colleagues' study investigated periodontal therapy's effects on RA patients with periodontitis in a double-blind, randomised controlled trial. The study included 28 patients, with the treatment group receiving non-surgical periodontal therapy and the control group receiving no periodontal intervention. The treatment group showed significant improvements in periodontal parameters, including reductions in plaque index, bleeding on probing, probing pocket depth (PPD), and clinical attachment loss (CAL). Importantly, this group also exhibited a reduction in DAS28 scores, suggesting an improvement in RA disease activity. However, serum ACPA and RF levels increased from baseline to follow-up, indicating that while periodontal therapy improved clinical periodontal parameters and inflammatory markers, it did not necessarily reduce autoimmune reactivity. The study was limited by a small sample size and a short follow-up period of 8 to 12 weeks, which may not have been sufficient to observe the long-term effects of periodontal therapy on RA parameters (49).

Another notable study, the OPERA feasibility trial conducted by de Pablo's group, explored whether periodontal therapy could reduce disease activity in RA patients with periodontitis. The study found that periodontal therapy was associated with reductions in periodontal inflamed surface area (PISA), PPD, RA disease activity scores, and ultrasound-detected synovitis. However, the complete resolution of periodontal inflammation remained challenging, which could have influenced the results. Despite this, the study demonstrated that periodontal therapy could improve both periodontal health and RA symptoms, suggesting its potential as a non-drug adjunctive treatment for RA patients with periodontitis (50).

However, contradictory findings exist, with some studies reporting no additional benefit of periodontal therapy on RA disease activity. A randomised controlled trial conducted in Kampala, Uganda, included 58 RA patients with periodontitis to evaluate the impact of non-surgical periodontal therapy on RA disease activity. Patients were divided into an intervention group receiving periodontal therapy and a control group without periodontal treatment. The primary outcome measure was the change in DAS28 scores over two three-month follow-up periods. The study found that

DAS28 scores improved significantly in the intervention and control groups, but no significant difference was observed between them. These findings suggest that periodontal therapy did not offer supplementary benefits in enhancing RA parameters beyond the effects of conventional RA treatment (51).

The evidence on the effectiveness of periodontal therapy in improving RA outcomes remains varied, with promising and conflicting results. Several studies suggest that periodontal treatment can significantly improve RA disease activity, as measured by clinical parameters such as DAS28, CRP, and RF levels. However, others find no additional benefit beyond standard RA treatment, suggesting that the effect of periodontal therapy on RA disease activity may be influenced by various factors such as the duration of follow-up, RA treatment regimens, disease duration, and individual immune responses. Also, differences in study designs, patient populations, and periodontal therapy protocols contribute to the variability in findings.

Despite these inconsistencies, the potential for periodontal treatment as a non-pharmacological adjunct in RA management remains an important area for further exploration. Given the shared inflammatory pathways between periodontitis and RA, maintaining good periodontal health could have broader implications for systemic inflammation and overall disease management. Future research should focus on conducting large-scale, long-term, and well-controlled clinical trials to determine the role of periodontal therapy in RA management conclusively. Additionally, further investigation into the underlying biological mechanisms linking periodontitis and RA could help identify which subsets of RA patients may benefit most from periodontal interventions.

The Effect of Rheumatoid Arthritis Therapy on Periodontitis

The impact of RA therapies on periodontal health has gained increasing attention, as both conditions share common inflammatory pathways. Understanding how RA treatments, including anti-TNF therapy, B cell depletion therapy, and DMARDs, influence periodontal conditions can assist in optimising treatment strategies for patients with both RA and periodontitis. Recently, studies have explored the effects of these therapies on clinical periodontal parameters and inflammatory biomarkers, emphasising the complex interactions between RA treatment and oral health.

Anti-TNF therapy, which targets TNF-α, a key pro-inflammatory cytokine in RA and periodontitis, has been investigated for its effects on periodontal conditions. This study examined the effect of anti-TNF therapy on periodontal health in RA and ankylosing spondylitis patients over six months. The findings showed that anti-TNF therapy reduced PPD and CAL, suggesting an overall improvement in periodontal condition. However, it also increased the gingival index, indicating worsened gingival inflammation. The study's small sample size limited the generalisability of the findings, but the results suggest that while anti-TNF therapy may reduce systemic inflammation and improve periodontal parameters, it might also exacerbate localised gingival inflammation due to immune modulation affecting the host response to oral pathogens (52).

B cell depletion therapy, particularly with rituximab, is used in RA patients who do not respond to TNF inhibitors and has been studied for its effects on periodontal inflammation. Hatipoglu and colleagues examined the effects of B cell depletion therapy on periodontal health by measuring inflammatory biomarkers in GCF. This study included 70 patients, divided into a group receiving B cell depletion therapy, a group receiving DMARDs, and a control group without RA. The study found that clinical periodontal parameters such as PPD, CAL, bleeding on probing, gingival index, and plaque index were similar across all groups. However, IL-1β levels, a key inflammatory cytokine, were significantly lower in the B cell depletion therapy group compared to the DMARD and control groups, suggesting that this therapy may reduce local periodontal inflammation. Conversely, MMP-8 levels, which are associated with tissue degradation, were significantly lower in the DMARD group than in the B cell depletion and control groups, indicating that different RA therapies may have distinct effects on the biochemical markers of periodontal inflammation (53).

In addition to biological therapies, conventional anti-rheumatic drugs such as NSAIDs, corticosteroids, and methotrexate have been studied for their effects on cytokine release and microbial adherence. Stähli's group carried out an in vitro study investigating the influence of ibuprofen, prednisolone, and methotrexate on cytokine release from oral cells in response to microbial stimulation. Their findings showed that *F. nucleatum* strongly stimulated pro-inflammatory cytokine release by periodontal ligament fibroblasts and monocytes. While prednisolone increased IL-8 release in monocytes after microbial stimulation, methotrexate reduced it, indicating differential effects on

inflammatory responses in the periodontium. Additionally, anti-rheumatic drugs enhanced microbial adherence to epithelial cells, particularly *Candida albicans*, suggesting that prolonged medication use may change oral microbial dynamics. However, this study had limitations, as it relied on isolated cells rather than complex in vivo immune interactions, focused only on *F. nucleatum* rather than the diverse range of bacteria present in dental biofilm, and measured cytokine levels at a single time point, failing to capture the dynamic nature of periodontal inflammation (54).

The effects of RA therapies on periodontitis highlight a complex relationship between systemic inflammation management and local periodontal health. Anti-TNF therapy may improve the periodontal condition by reducing systemic inflammation but could also contribute to local gingival inflammation. B cell depletion therapy appears to reduce IL-1β levels in the periodontium, potentially benefiting periodontal health, while DMARDs may lower MMP-8 levels, suggesting protective effects against tissue degradation. Additionally, conventional anti-rheumatic drugs can influence cytokine release and microbial adherence, which may affect periodontal disease progression. Given these varying effects, a more integrated treatment approach is needed for patients with both RA and periodontitis.

The impact of RA therapies on periodontal health remains an evolving area of research, with studies demonstrating both beneficial and potentially adverse effects. While biologic and conventional RA treatments may help reduce systemic inflammation, their influence on local periodontal conditions remains inconsistent, with potential effects on gingival inflammation, cytokine levels, and microbial interactions. The conflicting findings emphasise the need for more extensive clinical studies with larger patient cohorts and extended follow-up periods to better understand these interactions. Future research should focus on optimising RA treatments to minimise negative effects on periodontal health while maintaining systemic disease control. Additionally, interdisciplinary collaboration between rheumatologists and periodontists is essential in developing comprehensive management strategies that address both systemic and oral health.

Conclusion

RA and periodontitis share common inflammatory and immune pathways, with evidence suggesting a bidirectional relationship between these conditions. Periodontal bacteria, particularly *P. gingivalis*, may contribute to autoimmune responses in RA through citrullination and molecular mimicry, while chronic inflammation in RA may exacerbate periodontal disease.

Studies indicate that periodontal therapy can reduce RA disease activity, though findings remain inconsistent, highlighting the need for further research. Conversely, RA therapies' effects on periodontal health are varied, with some treatments reducing inflammation and others potentially worsening gingival conditions. These complex interactions emphasise the importance of an integrated treatment approach involving collaboration between rheumatologists and periodontists.

Although more long-term studies are needed, maintaining good periodontal health may serve as a valuable adjunct to RA management, potentially reducing systemic inflammation and improving overall patient outcomes.

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Email: aspnsw@asp.asn.au

Meeting name: ASP NSW Sub Branch Dinner Meeting

Meeting date & time: Thursday 26 June 25

Meeting location: Swissotel Sydney - 68 Market Street, Sydney

Speakers: Dr. Anastasia Georgiou

Topics: Oral Medicine in Periodontal Practice: Collaborative Approaches for Comprehensive Care

Cost & other details: Members Free, Guests; $145

Meeting name: ASP NSW Sub Branch Dinner Meeting

Meeting date & time: Thursday 25 September 25

Meeting location: Swissotel Sydney - 68 Market Street, Sydney

Speakers: Dr. Luan Ngo

Topics: Salvage Operation – Extending the life of Failing Implant Restorations

Cost & other details: Members Free, Guests; $145

Meeting name: ASP NSW Sub Branch Dinner Meeting

Meeting date & time: Full Day Meeting: 17 October 25

Meeting location: Swissotel Sydney - 68 Market Street, Sydney

Speakers: Nikos Mattheos

Topics: Implant Dentistry as a Design Discipline: Potential and challenges of a new digital paradigm

Cost & other details: All $495

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Meeting name: ASPQ May Dinner Meeting - Periodontitis & Oral Cancer

Meeting date & time: Monday May 12th - 6:00pm

Meeting location: Location: Victoria Park, Herston

Speakers: A/Prof Omar Breik (OMFS)

Topics: Periodontitis and Oral Cancer

Cost & other details: Sponsors: Geistlich, Straumann; Neoss; Samson Medical Technologies

ASP QLD Branch Committee Details and Meetings (cont'd)

Meeting name: ASPQ September Dinner Meeting - Prof MP Cullinan & Prof Seymour Research Medallion Competition

Meeting date & time: Monday September 29th

Meeting location: Location: Victoria Park, Herston

Speakers: Judges to be confirmed

Topics: Research Medallion Competition

Cost & other details: Sponsors: Geistlich, Straumann; Neoss; Samson Medical Technologies

Meeting name: ASPQ Clinical Day

Meeting date & time: November TBC - speakers to be confirmed

Meeting location: TBC

Speakers: TBC

Topics: ASPQ Clinical Day

Cost & other details:

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Vice President: Dr Isaac He

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State Branch Councillor: Dr Geoff Harvey

Email: aspsa2@gmail.com

Meeting name: ASP SA Dinner Meeting

Meeting date & time: 7 May 2025 6pm

Meeting location: Rozelle, 46 Carrick Hill Drive, SA

Speakers: Dr Brittny Roberts and Mr Josh Galpin

Topics: PG Presentation. Diagnostic consideration for dental hygienists in stage IV periodontal care

Cost & other details: Members: Free. Guests: $125

Meeting name: ASP SA Dinner Meeting

Meeting date & time: 13 August 2025 6pm

Meeting location: Lenzerheide, 146 Belair Road, SA

Speakers: Prof Saso Ivanovski

Topics: TBC

Cost & other details: Members: Free. Guests: $125

Meeting name: ASP SA Dinner Meeting

Meeting date & time: 24 September 2025 6pm

Meeting location: The Gallery, 30 Waymouth Street SA

Speakers: Prof Andrew Tawse-Smith

Topics: Implant Surface Decontamination

Cost & other details: Members: Free. Guests: $125

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Branch Councillor: Dr Sarah Chin
Email: aspvic@gmail.com

Meeting name:
Meeting date & time: 19th Nov 2025
Meeting location: TBA
Speakers: Dr Leela Movva Specialist Periodontist
Topics: Lasers In Periodontal Therapy
Cost & other details: $130 for members , $220 for non-members

Meeting name:
Meeting date & time: 30th July 2025
Meeting location: TBA
Speakers: Professor Marie Cornelis Professor in Orthodontics Head of Orthodontics Melbourne University
Topics: TBA
Cost & other details: $130 for members , $220 for non-members

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Email: aspwa@asp.asn.au

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Meeting date & time: June 27/28 (TBC)
Meeting location:
Speakers:
Topics: Hands On Course
Cost & other details:

Meeting name:
Meeting date & time: 19-Aug-25
Meeting location:
Speakers:
Topics: AGM
Cost & other details:

Meeting name:
Meeting date & time: 13-Nov-25
Meeting location:
Speakers:
Topics: End of Year Dinner
Cost & other details:

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Meeting name: AOS (NSW) Half Day Meeting

Meeting date & time: Wednesday, 18th June 2025 3pm

Meeting location: View Sydney, 17 Blue Street North Sydney

Speakers: Prof. Dean Morton

Topics: Contemporary Treatment of Completely and Partially Edenrtulous Patients: Consensus, Workflows and Future Possibilities

Cost & other details: AOS Members – Free
Remote Member - Free
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Meeting name: AOS(NSW) Dinner Meeting

Meeting date & time: Monday, 4th August 2025 6pm

Meeting location: View Sydney, 17 Blue Street North Sydney

Speakers: Dr Markus Troeltzsch

Topics: Implants in the medically compromised patient.

Cost & other details: AOS Members – Free
Remote Member - Free
Non-Member - $200
Non-Member Online – $100 (plus eventbrite booking fee)
Bookings via Eventbrite - https://www.eventbrite.com.au/e/1085640974379?aff=oddtdtcreator
Dr Markus Troeltzsch is an invited speaker sponsored by Geistlich

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Federal Councillor: Dr Angelos Sourial
Admin/Secretariat: Ms Bella Cherkasskaya
Email: infovic@aos.org.au aosvic@gmail.com

Meeting name: Online broadcasting
Meeting date & time: April 2025
Meeting location:
Speakers: TBA
Topics: TBA
Cost & other details: Members - free, Non nembers - $50

Meeting name: Dinner meeting and online broadcasting
Meeting date & time: 1-Jun-25
Meeting location: Royal South Yarra Lawn Tennis Club 310 Williams Road North, Toorak 3142
Speakers: Dr Varun Gang Prosthodontist and Dr Sarah Byrne Periodontist
Topics: TBC
Cost & other details: Members- free, Students - $55, Online members (dinner) - $110, Non-members - $190

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President: Dr Tony Strangio
Secretary: Dr Andrew Ziepe
Treasurer: Dr Richard Williams
Federal Councillor: Dr Roy Sarmidi
Email: infowa@aos.org.au

Meeting Name: AOS WA Dinner Meeting
Meeting date & time: Wednesday, 30th of April 2025
Meeting location: The University Club, UWA
Speakers: A/Prof Lisa Heitz-Mayfield
Topics: TBA
Cost & other details: TBA

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Meeting details are also available online:

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