Original Article

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Dentistry Section

Evaluation of Gapseal® as a Sealing Material at the Implant-abutment Interface in Type II Diabetes Patients: A Split-mouth Randomised Clinical Trial

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ABSTRACT

Introduction: Peri-implantitis is a prevalent complication in implant dentistry, particularly among patients with Type II Diabetes Mellitus (T2DM). It is characterised by impaired wound healing and a high susceptibility to infectious processes, as interfacial microleakage between the implant and abutment creates a channel for bacterial ingress. Porphyromonas gingivalis is a key pathogen whose colonisation triggers peri-implant inflammatory cascades that ultimately lead to osteolytic sequelae. Given the heightened risk of peri-implantitis in T2DM patients, the rationale for the present study is that evaluating GapSeal® as a sealing material at the Implant Abutment Interface (IAI) may provide a preventive strategy to reduce bacterial colonisation and improve implant outcomes.

Aim: To evaluate the efficacy of GapSeal® as a sealing material at the IAI in patients with T2DM.

Materials and Methods: This single-blinded, split-mouth randomised clinical trial was conducted at the Department of Prosthodontics, Crown and Bridge, Sri Ramachandra Dental College and Hospital, Chennai, Tamil Nadu, India, over a period of six months, from January 2025 to June 2025. The study included 12 partially edentulous patients with T2DM requiring implant-supported prostheses. A total of 24 implants were placed following a split-mouth design, with GapSeal® applied at the IAI in the test group, while the control group received no sealing material. Submucosal plaque samples were collected from the IAI at baseline and at 2, 4, and 12 weeks following abutment connection. The samples were analysed for Porphyromonas gingivalis using quantitative real-time Polymerase Chain Reaction (qPCR) to assess microbial colonisation. Statistical analysis was performed using an Independent t-test, and for within-group comparison over a period of time, repeated measures Analysis of Variance (ANOVA) followed by a Bonferroni post-hoc test was used. The p-value <0.05 was considered statistically significant.

Results: The GapSeal® group demonstrated significantly lower P. gingivalis colonisation compared to the control group at all post-baseline time points ($p<0.001$), with the most pronounced reduction observed at 12 weeks. P. gingivalis counts were significantly lower in the GapSeal® group at two weeks (2.18±0.35 vs. 4.30±0.85; $p<0.001$), four weeks (2.39±0.39 vs. 4.68±0.77; $p<0.001$) and 12 weeks (2.59±0.36 vs. 5.57±0.86; $p<0.001$).

Conclusion: GapSeal® significantly reduces P. gingivalis colonisation at the IAI in patients with T2DM, indicating its potential to minimise microleakage and reduce bacterial colonisation associated with peri-implant inflammatory risk. These findings support the use of GapSeal® as an adjunctive prophylactic measure in implant therapy in patients for systematically compromised patients to improve oral healthcare outcomes.

Keywords: Dental implants, Non insulin-dependent diabetes mellitus, Peri-implantitis

INTRODUCTION

Dental implants have emerged as the gold standard of rehabilitation of edentulous areas with predictable long-term outcomes due to the development of biomaterial science, implant design, and surgical guidelines [1]. Although the survival rates are high, more than 90% in a decade, the real success of implants hinges on the lack of peri-implant bone loss and inflammatory complications [2,3]. Peri-implantitis is one of them, and it is of significant concern, especially in populations that are systemically compromised, like those with T2DM.

The IAI is a critical zone of vulnerability, and micro-gaps of 1-49 µm allow bacterial infiltration and biofilm formation [4]. These microleakage pathways facilitate colonisation by anaerobic pathogens, notably *Porphyromonas gingivalis*, triggering inflammatory cascades that culminate in crestal bone resorption and implant failure [5-7]. The challenge is exacerbated in diabetic patients, where chronic hyperglycaemia impairs wound healing, alters immune responses, and promotes dysbiosis through Advanced Glycation End-products (AGEs) and elevated pro-inflammatory cytokines (IL-1β, TNF-α, IL-6) [3].

Considering the global increase in the prevalence of T2DM and its adverse effect on the health of peri-implant tissues, there is an urgent need to come up with measures to reduce microbial ingress at the Implant-Abutment Interface (IAI). GapSeal® is a highly viscous silicone matrix with 5% thymol, which has been developed to seal the IAI by filling microgaps and inhibiting the growth of microorganisms. It does not shrink and forms gaps as hardening sealants do. Unlike hardening sealants, it maintains long-term viscosity, hydrophobicity, and mechanical strength, which ensure high retention. The active ingredient, thymol, is a bactericidal, virucidal and fungicidal agent, about 30 times more effective than phenol, and is biocompatible and non allergenic. The manufacturer recommends reapplication every five years with regular visits to the recall centre to ensure effectiveness. GapSeal® is particularly appropriate in patients with a high risk of peri-implantitis as it provides a long-lasting microbial barrier that minimises the risk of infection and enhances the survival of implants. Its single-use ampoules are sterile, which makes their use hygienic, and the material can be easily removed with alcohol, which makes it a convenient and effective solution to the maintenance of implants [8].

Despite the proven in-vitro and clinical effectiveness of GapSeal® in the reduction of microleakage, its effectiveness in diabetic populations is not established. Hence, this study aimed to assess the effectiveness of GapSeal® as a sealing agent at the IAI in patients with T2DM, with a specific focus on the quantification of the *P. gingivalis* colonisation by the use of Quantitative PCR (qPCR) or real-time PCR. The objectives were to quantify the presence of *P.gingivalis* at the IAI in patients with T2DM using qPCR, to compare the microbial colonisation between implants treated with GapSeal® and those without the sealing agent (control group), and to evaluate the changes in *P. gingivalis* levels at baseline, 2nd week, 4th week, and 12th week following abutment connection.

The null hypothesis (H_0) stated that there would be no statistically significant difference in the colonisation of *P. gingivalis* at the IAI between implants treated with GapSeal® and those without the sealing agent.

The alternative hypothesis (H_1) proposed that the use of GapSeal® would result in a statistically significant reduction in *P. gingivalis* colonisation at the IAI compared with the control group.

MATERIALS AND METHODS

This study was designed as a single-blinded, split-mouth randomised clinical trial conducted at the Department of Prosthodontics, Crown & Bridge, Sri Ramachandra Dental College and Hospital, Chennai, Tamil Nadu, India, over a period of six months, from January 2025 to June 2025. Ethical clearance was obtained from the Institutional Ethics Committee (IEC Approval No: CSP/23/AUG/134/726) and written informed consent was obtained from all participants before enrollment. The trial was registered retrospectively with the Clinical Trials Registry of India CTRI/2026/03/105955).

Sample size calculation: G*Power software (v3.1.9.2) was used to calculate the sample size. The estimated sample size was nine participants, which would be enough to identify a large effect size (1.84). (Based on effect size derived from a pilot study conducted on three participants (not included in the final analysis) with 95% power at a 5% significance level. To increase the statistical strength and reduce the possibility of attrition, 12 participants were eventually recruited. Each participant received two implants, with one implant placed per mandibular quadrant, resulting in a total of 24 implant sites.

Inclusion criteria: Twelve individuals between 40 and 65 years of age with bilaterally missing mandibular posterior dentition, who agreed to an implant-supported prosthesis and had controlled type II diabetes with HbA1c levels less than seven, were recruited.

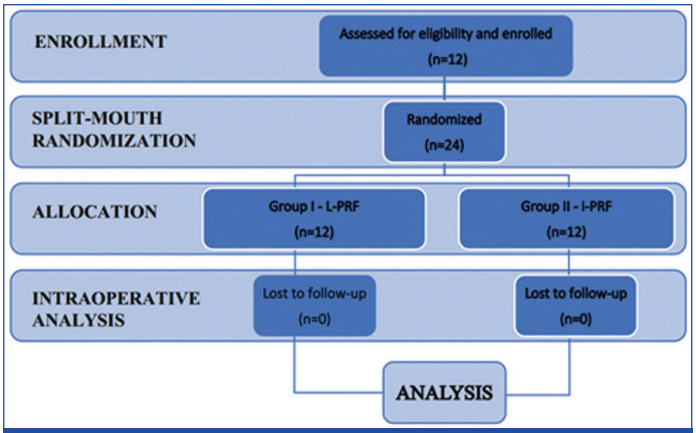
Exclusion criteria: Any systemic disease that may compromise the surgical outcomes, history of smoking, bisphosphonate use, psychological disorders, or pregnancy or lactation during the recruitment process were excluded.

Randomisation was done using a computer-generated sequence to assign the experimental (GapSeal®) and control (no sealant) conditions to either the right or left mandibular arch. Sequentially Numbered, Opaque, Sealed Envelopes (SNOSE) were used to maintain allocation concealment, which were only opened when the abutment was placed. The subjects were not aware of the intervention side, but operator blinding was not possible because of the nature of the procedures.

The present study was conducted according to the Consolidated Standards of Reporting Trials (CONSORT) guideline [Table/Fig-1].

Study Procedure

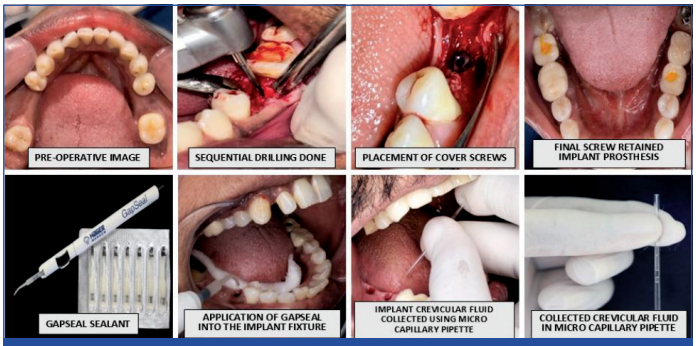
All the surgical procedures were performed by one clinician in accordance with a standardised protocol. The local anaesthesia was administered with 2% lignocaine with 1:80,000 adrenaline, and its sufficiency was checked before the incision. A mid-crest incision was done at the intended implant site and extended to the mesial and distal teeth through crevicular incisions. Mucoperiosteal flaps were raised buccally and lingually to expose the alveolar bone, which made it possible to measure the width of the ridges and bone volume.



[Table/Fig-1]: CONSORT flowchart.

The dimensions of the implants were determined based on clinical and radiographic assessment. A surgical template was used to mark each site and a pilot drill was performed to start the osteotomy preparation, which was then followed by sequential drilling as per the protocol of the manufacturer. Root-form implants 4.3/4.7 x 11.5/13 mm (DIO Implant Co., Ltd., Busan, South Korea) were inserted and fixed using cover screws. Flaps were repositioned and sutured, and patients were recalled after one week for suture removal. Three months of healing were noted before the rehabilitation of the prosthetics.

After haemostatic resolution, the cover screws were replaced with gingival formers that were maintained over three weeks. Abutments were then joined together. On the experimental side, GapSeal®- a silicone-based matrix containing 5% thymol- was applied at the implant-abutment interface before abutment placement. The abutment was fixed to the control side without the sealant. GapSeal® was applied using a single-use ampoule (0.06 mL) that was sterile and inserted into a reusable applicator [8]. The cannula was then carefully bent to access the implant site after the cap was removed. The sealant was applied to the IAI by rotating the applicator wheel. In cases where a closure gap was present and subsequently screwed in, the extrusion of excess material confirmed adequate sealing [Table/Fig-2].



[Table/Fig-2]: Surgical workflow.

Before the collection of Peri-Implant Crevicular Fluid (PICF), the implant sites were dried using cotton rolls and air-dried to avoid contamination. White, colour-coded microcapillary pipettes that were calibrated to 1-5 µL volumes were used to sample extra-crevicularly at the gingival margin to prevent tissue contact. Calibrated pipettes were used to be consistent, and samples that showed the presence of visible blood or plaque contamination were eliminated. A single calibrated examiner collected all the samples and had gone through training and pilot testing to obtain an intra-examiner reliability coefficient of 0.91.

After the collection of the specimen, the samples were immediately transferred into a sterile Eppendorf tube that had been prefilled with 98 µL of buffered alkaline phosphate saline. The samples were transported in tightly controlled conditions and kept at -800 C until further analysis. qPCR was subsequently employed to detect and quantify *Porphyromonas gingivalis*, serving as a biomarker for microbial leakage at the IAI. The second, fourth and twelfth weeks after abutment connection were scheduled as follow-up appointments. During these visits, PICF samples were collected at both sites to determine the presence of *P.gingivalis* through qPCR. The species-specific primers targeting the 16S rRNA gene of *P. gingivalis* were used. The primer sequences were as follows: Forward primer: 5′-TGCAACTTGCCTTACAGAGGG-3′; Reverse primer: 5′-ACTCGTATCGCCCGTTATTC-3′ [9]. The amplification was performed using SYBR Green chemistry following standard qPCR protocols and expressed as Cycle threshold (Ct) values.

Although dye penetration remains the most commonly employed technique for assessing microleakage, practical difficulties in directly accessing the implant-abutment microgap necessitated the use of PICF sampling at the gingival margin. While PICF does not directly access the microgap, it represents the most clinically feasible and reproducible method for evaluating bacterial leakage, and its microbial profile reflects the bacterial migration from internal reservoirs and correlates with peri-implant disease progression.

STATISTICAL ANALYSIS

All the data were compiled in Microsoft Excel and then analysed statistically with IBM Statistical Pacakge for Social Sciences (SPSS) Statistics version 31.0. Normality was assessed using the Shapiro-Wilk test. The demographic and clinical parameters were summarised as descriptive statistics. To compare the microbial counts of the groups at each time point, an Independent t-test was used and for within-group comparison over a period of time, repeated measures ANOVA followed by a Bonferroni post-hoc test was used. The significance was set as p<0.05.

RESULTS

The mean age of the 12 patients was 54.17±6.28 years, with 7 (58.3%) being male and 5 (41.7%) being female. The colonisation of *Porphyromonas gingivalis* was assessed at baseline and at 2nd, 4th and 12th weeks following abutment connection. No statistically significant difference was found between the GapSeal® and control cohorts at baseline. But starting in the 2nd week, the GapSeal® cohort showed significantly lower levels of colonisation compared to the control group. This reduction continued to be statistically significant at every follow-up time, with the highest difference being at the 12th week [Table/Fig-3].

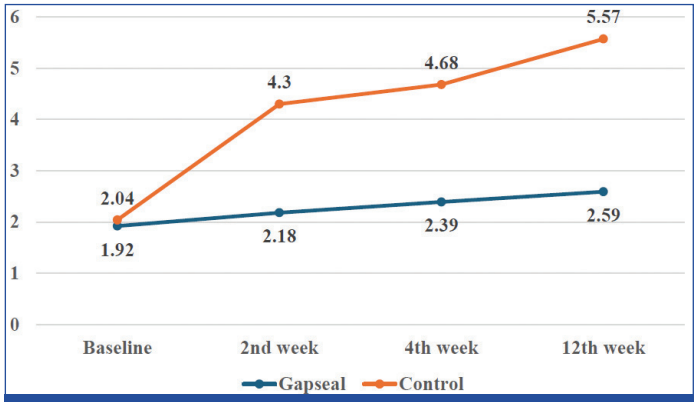
P.gingivalis	Gapseal (Mean±SD)	Control (Mean±SD)	Mean difference	Test statistic[a]	p-value
Baseline	1.92±0.38	2.04±0.36	-0.12	-0.775	0.447
2nd week	2.18±0.35	4.30±0.85	-2.12	-7.971	<0.001*
4th week	2.39±0.39	4.68±0.77	-2.28	-9.177	<0.001*
12th week	2.59±0.36	5.57±0.86	-2.97	-11.057	<0.001*
Test statistica	61.444	119.372			
p-valueb	<0.001*	<0.001*			

[Table/Fig-3]: Assessment of *P.gingivalis* Colonisation (Ct) between groups at various time intervals and within groups.
[a]Independent t-test, [b]Repeated measures ANOVA, *p<0.05 considered significant; SD: Standard deviation, *P.gingivalis*: *Porphyromonas gingivalis*

Intra-group comparison of *P. gingivalis* colonisation demonstrates that both GapSeal® and control groups showed a statistically significant increase over time, but the extent of colonisation differed. Within the GapSeal® group, the rise from baseline to subsequent weeks reflects a gradual accumulation of bacterial load. In contrast, the control group exhibited a much sharper increase, with baseline to later week comparisons revealing substantially higher mean differences. Even short-interval comparisons highlighted pronounced elevations, suggesting that colonisation expanded more aggressively in the absence of intervention [Table/Fig-4,5].

Group	Timeline comparison	Mean difference	Std. error	Sig.	95% Confidence interval for difference	
					Lower Bound	Upper Bound
Gapseal	Basline- 2nd week	-0.250*	0.036	<0.001	-0.365	-0.135
	Baseline- 4th week	-0.467*	0.068	<0.001	-0.684	-0.249
	Baseline- 12th week	-0.667*	0.063	<0.001	-0.869	-0.464
	2nd week- 4th week	-0.217*	0.041	0.001	-0.347	-.087
	2nd week- 12th week	-0.417*	0.047	<0.001	-0.569	-0.265
	4th week- 12th week	-0.200*	0.048	0.009	-0.353	-0.047
Control	Basline- 2nd week	-2.258*	0.269	<0.001	-3.122	-1.395
	Baseline- 4th week	-2.633*	0.248	<0.001	-3.430	-1.836
	Baseline- 12th week	-3.525*	0.276	<0.001	-4.410	-2.640
	2nd week- 4th week	-0.375*	0.075	0.002	-0.616	-0.134
	2nd week- 12th week	-1.267*	.091	<.001	-1.558	-0.976
	4th week – 12th week	-.892*	.045	<.001	-1.037	-0.747

[Table/Fig-4]: Pair-wise comparison of *P.gingivalis* Colonisation (Ct) between timelines in each group.
Bonferroni post-hoc test, *p<0.05 considered significant



[Table/Fig-5]: *P. gingivalis* colonisation over time between groups.

DISCUSSION

Peri-implantitis is a multifactorial condition, with microleakage at the IAI being one of the primary contributors to its onset. This condition is particularly common in patients with T2DM, who exhibit an elevated risk for peri-implant inflammation. Bacterial biofilm formation at the IAI, especially in the presence of pathogens like *P. gingivalis* and S. aureus, is a key factor in the pathogenesis of peri-implantitis and subsequent bone loss. Although significant efforts have been made to minimise microgaps at the IAI, oral cavity contamination is inevitable due to design gaps inherent in the implant system [5,10]. Out of the list of measures that can be taken to reduce this issue, the use of GapSeal®, an antibacterial sealing compound, has shown promising initial outcomes in reducing microleakage. However, there is limited clinical evidence regarding its efficacy in patients with T2DM.

GapSeal®, a silicone-based sealing material, has been shown to be effective in solving the problem of microgaps and microleakage at

the IAI, reducing both mechanical complications, including screw loosening, and biological complications, including peri-implant infections. The effectiveness of the material can be partly explained by the fact that it reduces the friction coefficient between the parts of the implants, which prevents the occurrence of micromotion and limits the loss of torque. Besides, the high viscosity and bactericidal effect of GapSeal®, which is due to the addition of thymol, reduce bacterial growth at the IAI, thereby reducing the risk of infection. Biocompatibility also guarantees low levels of adverse reactions with the surrounding tissues. Previous research has demonstrated that GapSeal® reduces the size of microgaps and microleakage in implant systems, and thus, it is a safe choice to improve the long-term stability and performance of dental implants [11-13].

The chosen timeline was aimed at assessing the short-term and long-term outcomes of the GapSeal® application on the IAI. Baseline sample collection will provide initial data on the presence of pathogens before the intervention, thus giving a clear picture of the state of the peri-implant environment. The 2nd and 4th -week follow-up visits are important to determine the initial impact of the intervention, whereas the twelfth-week visit provides information about the stability and permanence of the results over time [13]. The mandibular posterior region was chosen as this typically demonstrates relatively favourable bone density and cortical bone thickness compared with other implant sites, which contributes to improved primary implant stability and predictable osseointegration.

Porphyromonas gingivalis, one of the main pathogens in the pathogenesis of peri-implantitis, was selected in this study due to its classification as part of the so-called red complex of bacteria, which are closely related to periodontal and peri-implant diseases [14]. *P.gingivalis* is also known to colonise the implant surface within 30 minutes of implant placement and contributes greatly to the development of inflammatory responses that cause implant failure. By assessing the presence and quantity of *P. gingivalis* using qPCR, the study aims to determine whether the application of GapSeal® can effectively reduce bacterial colonisation at the IAI and potentially prevent peri-implantitis [15-17].

Fürst MM et al., study noted that colonisation of titanium implants by *P. gingivalis* began in thirty minutes after surgical placement. The bacterial load at the implant sites increased significantly within a period of twelve weeks. It is important to note that the load of the implant sites at the twelfth week of the implantation of the *P. gingivalis* was higher than the load at the beginning of the postoperative period. These results indicate that the colonisation of titanium implants by *P. gingivalis* is time-dependent and can be chronic and eventually lead to peri-implant pathology unless it is wisely managed [15].

The findings of the present study demonstrated a statistically significant reduction in *P.gingivalis* colonisation at the implant-abutment interface in the GapSeal® group compared with the control group at subsequent follow-up intervals; therefore, the null hypothesis was rejected. The present study indicates that the use of GapSeal® significantly reduced colonisation by *P.gingivalis* compared to the control group, and the effect continued after the second week and peaked at the twelfth week, thus highlighting its potential in the future to be used in the sustained modulation of microbes.

The mechanism of GapSeal® that could be involved in reducing colonisation of the bacteria may be antimicrobial agents that directly prevent bacterial growth or viability by disrupting cellular membranes, preventing protein synthesis, or disrupting key metabolic pathways [18]. Additionally, Gapseal® may disrupt biofilms formed by *P. gingivalis* on host surfaces, making it easier for the immune system to target and eliminate the bacteria [13]. It can also boost the host's immune response by stimulating macrophages and neutrophils and increasing the generation of antimicrobial peptides. The other possible mechanism is the inhibition of bacterial adhesion, in which case GapSeal® may inhibit receptors that are used by *P. gingivalis* to bind to host tissues, thereby inhibiting colonisation [19]. In addition, GapSeal® may have anti-inflammatory properties, which will decrease the inflammation in the area and provide a less favourable environment to bacteria. Furthermore, it can help restore a natural balance of the oral microbiome by promoting the growth of commensal microorganisms that can outcompete opportunistic pathogens like *P. gingivalis*. These mechanisms will tend to work in synergy to reduce bacterial colonisation [11-13].

This is in line with Zarbakhsh A et al., and Mostofy Naser SH et al., findings, who found a significant reduction in microgap size in the GapSeal® group [10,11]. Moreover, all control specimens showed leakage, whereas none of the GapSeal® specimens showed leakage. This demonstrates that GapSeal® effectively seals the IAI, preventing microleakage. According to Smojver I et al., GapSeal® reduced micro-leakage compared to other sealer agents (Flow.Sil, Oxysafe Gel), particularly with respect to Candida albicans [13]. In contrast to 80-90 per cent of control samples that leaked when exposed to Staphylococcus aureus and C. albicans, GapSeal® was able to close the IAI and prevent microbial contamination. Conversely, Mohammadi F et al., found that Atridox had better efficacy in reducing bacterial micro-leakage than other sealing agents, such as GapSeal® [20]. Although this method reduces microbiota at the IAI, a complete hermetic barrier against microbial invasion is currently unattainable.

Therefore, the results suggest that GapSeal® is an effective barrier to the IAI, which reduces microleakage and bacterial infiltration. GapSeal® has the potential to increase the success rate of dental implants and decrease the risk of associated biological and mechanical complications by reducing the risk of infection at the implant-abutment junction.

Limitation(s)

The limitations of the present study are that they did not measure the microgap dimensions at the IAI, which means no correlation between microgap size and microleakage could be established. In addition, the research was limited to a single microbial species, which limited the extrapolation of the findings to other bacterial or fungal pathogens that may also influence the success of implants. The small sample size increases the risk of inflated statistical significance and limits the generalisability of the findings. There was also no long-term follow-up to assess the durability of the sealing material over time and its performance under continuous mechanical loading. Furthermore, the evaluation was limited to GapSeal® only, eliminating the possibility of making a comparative evaluation with other sealing agents, which would have provided a more detailed evaluation of its effectiveness in preventing microleakage. Also, the duration and severity of diabetes, variability in oral hygiene practices, prior periodontal or peri-implant history, medication use, and lifestyle factors, including diet may act as confounders influencing implant success and the sealing efficacy.

CONCLUSION(S)

GapSeal® attenuated the rate of *P. gingivalis* colonisation compared with controls, indicating a partial protective effect against microleakage. However, colonisation continued to increase significantly within the treatment group, underscoring that the material reduces but does not completely prevent microbial ingress. These findings highlight its potential as an adjunctive measure to mitigate peri-implant inflammatory risk, particularly in susceptible populations such as patients with T2DM. Nevertheless, additional studies are required to validate these results and to evaluate the long-term stability and therapeutic efficacy of GapSeal® under conditions of sustained mechanical loading. Moreover, comparative studies involving alternative sealing agents are necessary to establish their relative effectiveness and broaden the clinical understanding of their therapeutic potential.

Authors' contribution: KP: Conceived and designed the study, performed the statistical analyses, developed the study protocol, and drafted the initial version of the manuscript; S: Contributed to the management of the study analyses and supported the interpretation of the results; NP: Assisted in managing the study analyses and conducted the literature search. All authors critically reviewed the manuscript, contributed to revisions, and approved the final version for submission.

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