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WHEN BIOFILMS BITE BACK: MICROBIOLOGICALLY INFLUENCED CORROSION AND ITS ROLE IN PERI-IMPLANTITIS – A PERIODONTICS PERSPECTIVE

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ABSTRACT Dental implants, predominantly composed of titanium and its alloys, have revolutionized oral rehabilitation by restoring function and esthetics with high biocompatibility and mechanical strength. However, within the dynamic oral environment, implants are exposed to biochemical, microbiological, and mechanical stresses that can compromise their longevity. Recent evidence implicates microbiologically influenced corrosion (MIC)-a microbe-mediated electrochemical degradation of metals-as a critical but under recognized factor in peri-implantitis, the leading cause of late implant failure. This review synthesizes current understanding of MIC from a periodontics perspective, integrating insights how bacterial biofilms alter surface electrochemistry through acidogenic, sulfate-reducing, and redox-active metabolic pathways, destabilizing the protective TiO_2 film and promoting ion release, pitting, and nanoparticle formation. The synergy between microbial activity, mechanical wear, and host inflammation-termed bio-tribocorrosion-emerges as a central mechanism linking material degradation with peri-implant tissue destruction. Preventive strategies, including advanced surface modifications, antimicrobial coatings, and biofilm control, are discussed with this review.

KEYWORDS : Peri-Implantitis, Titanium implants, microbiologically influenced corrosion, tribo-corrosion

INTRODUCTION

Dental implants have transformed oral rehabilitation, offering reliable restoration of function and esthetics. Titanium and its alloys are widely used because of their exceptional biocompatibility, mechanical strength, and ability to form a stable passive titanium-oxide (TiO_2) layer that resists corrosion [1].Yet, despite their apparent inertness, implants function in a complex oral ecosystem where biochemical, microbiological, and mechanical stresses coexist. Within this environment, **microbiologically influenced corrosion (MIC)**-the deterioration of metals mediated or accelerated by microbial activity has emerged as a hidden contributor to **peri-implantitis**, the major cause of post-osseointegration implant failure [2,3].

Recent insights from materials science and microbiology reveal that corrosion of titanium in vivo is not merely electrochemical but **bio-electro-mechanical**, driven by interactions between biofilm metabolites, mechanical wear, and host inflammatory responses [1,4]. The result is **bio-tribocorrosion**, a synergistic degradation process that links material instability with biological inflammation [1].This review consolidates current evidence on MIC in dental implants from a **periodontics perspective**, emphasizing the microbial, electrochemical, and host factors underlying peri-implant disease.

Background On Metals In Dentistry

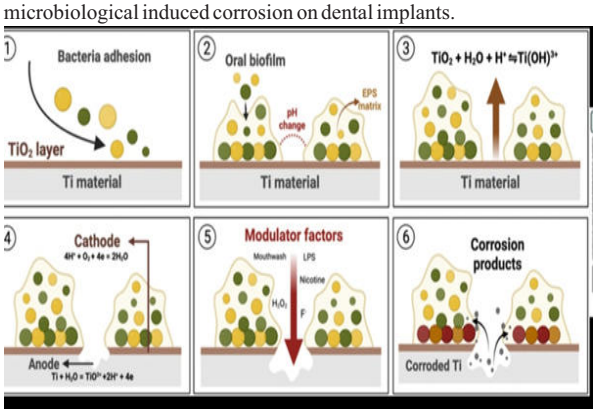
Metals have long underpinned restorative and prosthodontic practice. Among them, **titanium** dominates implantology for its high strength-to-weight ratio and ability to spontaneously form a protective TiO_2 film [5,6]. The alloy Ti-6Al-4V further enhances fatigue resistance. Stainless steel, cobalt–chromium, and nickel–chromium alloys are also used in orthodontics and prosthodontics but display higher corrosion susceptibility [7].

Despite its passivity, titanium is **not immune** to degradation. Surface oxide breakdown can occur through **acidic or oxidative microbial metabolites**, **fluoride exposure**, or **mechanical micromovement**. Dissolved titanium ions and particles have been identified in peri-implant crevicular fluid, saliva, and even systemic circulation [8,9]. These findings challenge the long-held notion of titanium's inertness and highlight the importance of understanding corrosion within the oral microenvironment.

Overview Of Corrosion In Oral Environments

Corrosion is fundamentally an **electrochemical reaction** where metal atoms oxidize to ions. The oral cavity provides ideal conditions for such reactions: an electrolyte (saliva), fluctuating pH, microbial biofilms, and cyclic mechanical loads [5,7]. Different corrosion forms—uniform, pitting, galvanic, and **fretting corrosion**—can develop on implant surfaces. Fretting corrosion, caused by micromotions at the implant–abutment junction, exposes underlying metal and accelerates oxide removal [10].

In diseased peri-implant sites, bacterial metabolism generates **acidic and reductive microenvironments** that destabilize the TiO_2 layer [2,11]. This facilitates localized ion release and nanoparticle formation, both implicated in peri-implant inflammatory cascades [8,12]. Elevated titanium concentrations in diseased tissue samples strongly correlate with peri-implantitis severity [9].Fig 1 illustrate how microbiological induced corrosion on dental implants.



Relevance Of Microbiologically Influenced Corrosion (MIC)

MIC describes corrosion processes initiated or accelerated by microorganisms. In dentistry, this occurs when adherent biofilms alter the **electrochemical potential** and chemistry of the implant surface [13]. Bacteria secrete acids, hydrogen sulfide, ammonia, and redox-active metabolites that promote metal dissolution [14].

Unlike purely chemical corrosion, MIC involves **biological catalysis**: microbes facilitate electron transfer between metal and environment. Oral biofilms contain diverse species-facultative anaerobes, acidogenic streptococci, and sulfate-reducing bacteria - capable of driving MIC reactions [15,16]. These microbes generate **differential aeration zones** within the biofilm, forming anodic and cathodic sites that trigger localized corrosion [13,17].

Clinically, MIC contributes to **ion release, oxide thinning, and nanoparticle generation**, which, in turn, aggravate **host inflammation and bone resorption**. Thus, MIC represents both a **microbial** and **biomaterial** pathology [2,3,8].

Mechanisms of Microbiologically Influenced Corrosion (MIC)

The pathogenesis of MIC involves interconnected **electrochemical**, **biochemical**, and **microbial** mechanisms:

1. Biofilm-induced potential heterogeneity: Biofilms create oxygen and ion gradients, forming localized anodes and cathodes on the metal surface [13,14].

2. Acidogenic metabolism: Species such as *Streptococcus mutans* and *Lactobacillus spp.* produce lactic acid, lowering pH and dissolving the passive film [11,15].

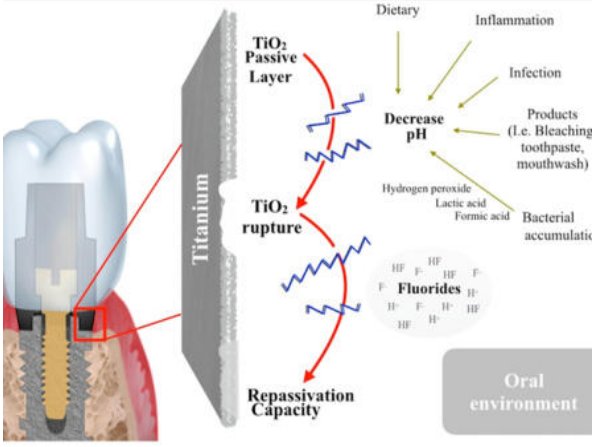
3. Sulfate reduction: *Desulfovibrio* spp. generate hydrogen sulfide, reacting with titanium to form non-protective sulfides and deep pits [16].

4. Redox-active metabolites: *Pseudomonas aeruginosa* secretes pyocyanin and phenazines that shuttle electrons between microbial cells and metal, accelerating corrosion [17].

5. Exopolymeric substances (EPS): EPS retains electrolytes, enhances conductivity, and sustains corrosive reactions [18].

6. Synergy with mechanical wear: Micromovements abrade the oxide layer, exposing fresh titanium for microbial colonization and continued MIC [1,10].

These mechanisms culminate in **localized pitting**, **surface roughening**, and **metallic ion release**, fueling peri-implant inflammation and structural degradation [8,12].Fig 2 depicts, mechanism of MIC on dental implant corrosion.



Biofilm Formation Sequence

Biofilm development on titanium follows classic stages: **pellicle formation**, **initial adhesion**, **coaggregation**, and **maturation** [15,19]. Salivary proteins first form a conditioning layer enabling attachment of early colonizers (*Streptococcus oralis*, *Actinomyces naeslundii*). These early communities alter surface charge and hydrophobicity, attracting bridging species such as *Fusobacterium nucleatum* and late anaerobes including *Prevotella intermedia* and *Porphyromonas gingivalis* [15,20].

Within mature biofilms, metabolic cooperation maintains acidic and hypoxic microzones that destabilize TiO_2. EPS acts as both **diffusion barrier** and **ionic reservoir**, fostering MIC [18]. *Pseudomonas fluorescens*, for instance, produces extracellular polymers that mediate electron transfer and corrosion of metals [18]. Thus, biofilm maturation directly transforms the titanium surface into an **electrochemically active interface** vulnerable to MIC.

Electrochemical Disruption Of The TiO_2 Passive Layer

Titanium's corrosion resistance derives from its TiO_2 film; however, this layer is dynamic and easily perturbed. Acidogenic biofilms can locally drop pH below the oxide's stability range (pH 3.5–4.5), dissolving or thinning the film [5,11]. Moreover, microbial redox processes generate anodic–cathodic heterogeneity across the surface, initiating pitting [13,16].

Experimental work on orthodontic wires colonized by bacteria demonstrated increased corrosion currents and potential shifts due to microbial electron mediation [21].

In peri-implantitis, this biochemical disruption manifests clinically as elevated titanium levels in crevicular fluid [9] and histologically as **metallic particle deposits surrounded by inflammatory infiltrate** [22].

Tribocorrosion Synergy

The oral cavity imposes repeated mechanical loads that induce **fretting wear** and **tribological stress** on implant components. These forces remove protective oxide layers, exposing fresh titanium to saliva and biofilms [1,10]. Heintze et al. demonstrated that wear and surface roughness enhance bacterial adhesion, initiating a feedback cycle between **mechanical wear** and **microbial corrosion** [10].

The synergistic interplay-**wear-induced corrosion** and **corrosion-enhanced wear**-is termed **bio-tribocorrosion** [1]. Clinically, this results in titanium particle release, abutment screw loosening, and microleakage at the implant–abutment interface, all of which intensify bacterial colonization [8,22].

Microbial Communities in Microbiologically Influenced Corrosion

Metagenomic sequencing reveals that peri-implantitis biofilms harbor distinct microbial signatures compared to healthy implants [2,15,20]. Liang et al. identified enrichment of *Prevotella*, *Fusobacterium*, and *Treponema* species associated with pathways in sulfur and nitrogen metabolism [15]. Alves et al. demonstrated that host–microbiome crosstalk modulates inflammation, where microbial virulence factors up-regulate host cytokines, compounding corrosion-related damage [3].

Microorganisms implicated in MIC include *Pseudomonas aeruginosa*, *Desulfovibrio*, *Streptococcus mutans*, *Actinomyces*, *Lactobacillus*, and *Veillonella* spp. [14-17,20]. Their metabolic diversity establishes **heterogeneous redox conditions** that sustain corrosion reactions at the biofilm–metal interface [13].

Species	Metabolic Activity	Role in MIC
Streptococcus mutans	Lactic acid fermentation	Acid dissolution of TiO_2[11,15]
Pseudomonas aeruginosa	Phenazine secretion	Electron shuttle; anodic dissolution[17]
Desulfovibrio spp.	Sulfate reduction to H_2S	Sulfide-induced pitting[16]
Fusobacterium nucleatum	Coaggregation bridge	Biofilm maturation, pH reduction[15,20]
Prevotella intermedia	Proteolytic metabolism	EPS production, acidification[15,20]
Actinomyces naeslundii	Early colonizer	Surface conditioning[19,20]
Treponema denticola	Proteolysis, motility	Deep invasion, MIC synergy[15,20]

Clinical Implications

Corrosion and biofilm activity compromise implant integrity and host tissues simultaneously. Titanium ions and nanoparticles act as **pro-inflammatory mediators**, stimulating macrophage activation and osteoclastogenesis [3,8,12]. Surface roughening and altered wettability following corrosion favor recolonization by pathogenic species, perpetuating a **vicious cycle** of degradation [2,13].

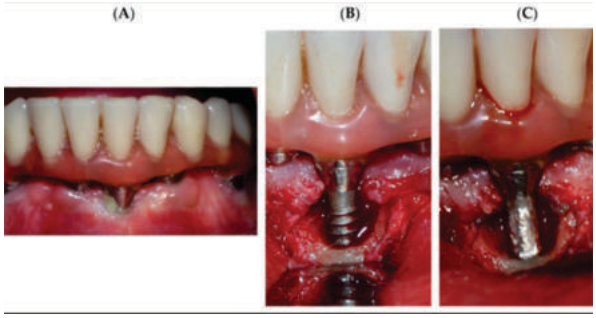
Histopathologic analyses of failed implants reveal **metallic debris**

embedded within peri-implant granulation tissue, signifying chronic exposure to corrosion products [22]. MIC thus bridges material failure with biological inflammation, explaining why some peri-implant lesions resist conventional mechanical debridement.

Peri-Implantitis Link

The association between MIC and peri-implantitis is substantiated by both clinical and molecular evidence. Safioti et al. observed higher titanium concentrations in peri-implantitis tissues than in healthy controls [9]. Asa'ad et al. demonstrated that titanium ions up-regulate inflammatory mediators such as TNF-α and IL-1β, intensifying bone resorption [8].

According to the **Biofilm-Induced Inflammation and Bone Dysregulation (BIND) hypothesis**, corrosion products serve as DAMPs (damage-associated molecular patterns), activating macrophages and triggering osteoclastic pathways [23]. Thus, MIC not only contributes to biofilm dysbiosis but also to **immune dysregulation**, unifying mechanical, microbial, and host factors in peri-implantitis pathogenesis. Fig 3, depicts clinical picture of plaque induced corrosion of implant.



Allergic And Toxic Reactions

Although titanium is deemed biocompatible, released ions can form complexes with proteins, eliciting **type IV hypersensitivity** in predisposed patients [9,24]. Hubenova et al. demonstrated that microbial corrosion accelerates ion release to levels sufficient to trigger immune sensitization and even implant rejection [14]. Cytotoxic effects include **oxidative DNA damage**, mitochondrial dysfunction, and apoptosis in peri-implant tissues [1,8]. Clinically, patients may report mucosal erythema, burning, or unexplained inflammation unresponsive to therapy, warranting hypersensitivity testing [20].

Structural Failures

At the structural level, MIC leads to **pitting, crevice formation, and surface cracking** that weaken implants [7,22]. Fretting corrosion at the implant–abutment interface amplifies stress concentration, predisposing to **mechanical fatigue and fracture** [10]. Histopathology of failed implants shows corrosion pits colonized by bone-associated microbes, suggesting a self-propagating cycle of **corrosion–infection–fracture** [22]. Such deterioration undermines load distribution, ultimately compromising long-term prognosis [5,7,10].

Prevention And Mitigation

Preventive strategies target both **material properties** and **microbial activity**. Surface engineering approaches—such as **anodization, plasma-spraying, and alloying with niobium or zirconium**-enhance corrosion resistance [6,24]. **Bioactive coatings** incorporating hydroxyapatite, chitosan, silver, or zinc ions exhibit antimicrobial and anticorrosive potential [18,24].

Clinically, routine peri-implant maintenance with **mechanical debridement, pH-neutral irrigants, and fluoride-free formulations** reduces risk. The **Indian Society of Periodontology** recommends meticulous prosthetic design to minimize microgaps and occlusal overloading [20]. Emerging **probiotic therapies** and **nanostructured coatings** show promise in modulating biofilm ecology toward health [18,24].

Biofilm Control Strategies

Controlling biofilms remains central to mitigating MIC. Conventional antiseptics (chlorhexidine, hydrogen peroxide) reduce microbial load but may not penetrate mature biofilms [20]. Research into **probiotic strains (e.g., *Lactobacillus reuteri*)** suggests they can outcompete pathogens and restore ecological balance [18]. Advanced coatings releasing antimicrobial peptides or nitric oxide offer **sustained antibiofilm activity** while preserving osseointegration [24]. Nevertheless, over-reliance on broad antimicrobials risks resistance; hence, **multimodal prevention** integrating mechanical, chemical, and probiotic approaches is advocated.

Research Gaps And Future Directions

Despite significant progress in understanding microbiologically influenced corrosion (MIC) and its role in peri-implantitis, several critical gaps remain. There is limited in vivo quantification of MIC rates and titanium ion release, and the threshold between physiological and toxic concentrations in peri-implant tissues is not well established [8,9,23]. Existing experimental models often fail to integrate mechanical wear, microbial colonization, and host tissue interactions, limiting their clinical relevance. Data on patient-specific susceptibility, including genetic factors and immune hypersensitivity, are also insufficient [8,9,23]. Future research should leverage metagenomics, electrochemical impedance spectroscopy, and in situ imaging to unravel the dynamic interactions between implants, biofilms, and host tissues. Additionally, the development of corrosion-resistant β-type titanium alloys and biomimetic surface coatings holds promise for enhancing implant longevity and reducing the incidence of peri-implant disease [6,24].

CONCLUSION

Microbiologically influenced corrosion represents a paradigm shift in understanding peri-implant diseases. Titanium degradation is no longer viewed as a passive chemical process but as an **active bio-electro-mechanical phenomenon** involving microbial metabolism, electrochemical instability, and host inflammation. Recognizing MIC's role helps clinicians appreciate peri-implantitis as not merely infectious but **bio-corrosive**, demanding interdisciplinary management. Prevention through **surface optimization, biofilm control, and patient-specific risk assessment** remains the cornerstone for sustaining implant health.

REFERENCES

1. Gaur S, Agnihotri R, Albin S. Bio-Tribocorrosion of Titanium Dental Implants and Its Toxicological Implications: A Scoping Review. *Dent J (Basel)*. 2023;11(5):133.
2. Takahashi N, Igarashi K, Nishimura F. Microbiologically influenced corrosion in dentistry: A review. *J Prosthodont Res*. 2022;66(3):325–33.
3. Alves LR, Bianchini MA, Siqueira AB, et al. Host-microbiome crosstalk in peri-implantitis: The missing link in implant corrosion. *Front Oral Health*. 2023;4:1212762.
4. Sridhar S, Narayanan R. Tribocorrosion in titanium dental implants: A systematic insight. *Mater Sci Eng C Mater Biol Appl*. 2020;109:110585.
5. Elias CN, Lima JHC, Valiev R, Meyers MA. Biomedical applications of titanium and its alloys. *JOM*. 2008;60(3):46–9.
6. Geetha M, Singh AK, Asokamani R, Gogia AK. Ti based biomaterials, the ultimate choice for orthopaedic implants – A review. *Prog Mater Sci*. 2009;54(3):397–425.
7. Schiff N, Grosgogeat B, Lissac M, Dalard F. Influence of fluoride content and pH on corrosion resistance of titanium and its alloys. *Biomaterials*. 2002;23(19):3979–88.
8. Asa'ad F, Pagni G, Pilipchuk SP, et al. Titanium particle release and host response in peri-implantitis. *Clin Oral Investig*. 2023;27(5):2135–46.
9. Safioti LM, Kotsakis GA, Pozhitkov AE, Chung WO, Daubert DM. Increased levels of dissolved and particulate titanium in peri-implantitis granulation tissue and healthy peri-implant mucosa. *J Periodontol*. 2017;88(4):436–42.
10. Heintze SD, Cavalleri A, Forjanic M, Zellweger G, Rousson V. Wear of ceramic and antagonist—A systematic evaluation of influencing factors in vitro. *Dent Mater*. 2008;24(4):433–49.
11. Souza JGS, Bertolini M, Costa RC, et al. Biofilm interactions with titanium: A critical review of the role of microbial-induced corrosion in implant failure. *Biofouling*. 2020;36(2):185–98.
12. Alrabeah GO, Knowles JC, Petridis H. The effect of titanium ions on macrophage cytokine expression and osteogenic potential in vitro. *J Dent*. 2017;66:91–101.
13. Videla HA, Herrera LK. Microbiologically influenced corrosion: Looking to the future. *Int Microbiol*. 2009;12(3):169–80.
14. Hubenova Y, Rashkov R, Hubenov V, et al. Bioelectrochemical corrosion of titanium by sulfate-reducing bacteria. *Electrochim Acta*. 2021;388:138565.
15. Liang S, Hosomi K, Kobayashi T, et al. Microbial communities associated with peri-implantitis and peri-implant mucositis: A systematic review. *Int J Implant Dent*. 2022;8(1):14.
16. Li Y, Xu J, Zhang C, et al. The influence of sulfate-reducing bacteria on the corrosion of titanium in anaerobic environments. *Corros Sci*. 2019;157:10–22.
17. Hernandez ME, Kappler A, Newman DK. Phenazines and other redox-active metabolites as electron shuttles in microbial redox processes. *Appl Environ Microbiol*. 2004;70(2):921–8.
18. Flemming HC, Wingender J, Szewzyk U, et al. Biofilms: An emergent form of bacterial life. *Nat Rev Microbiol*. 2016;14(9):563–75.
19. Kolenbrander PE, Palmer RJ Jr, Periasamy S, Jakubovics NS. Oral multispecies biofilm development and the key role of cell-cell distance. *Nat Rev Microbiol*. 2010;8(7):471–80.
20. Renvert S, Quirynen M. Risk indicators for peri-implantitis. A narrative review. *Clin Oral Implants Res*. 2015;26(Suppl 11):15–44.
21. Arana-Chavez VE, Pacheco LM, Teixeira FB, et al. Microbial corrosion of orthodontic wires: Electrochemical and microbiological analysis. *Angle Orthod*. 2018;88(1):44–50.
22. Noronha Oliveira M, Schunemann WVH, Mathew MT, et al. Can degradation products released from dental implants affect peri-implant tissues? *J Periodontal Res*. 2018;53(1):1–11.
23. Mathew MT, Abbey S, Hallab NJ, et al. Tribocorrosion and osteolysis: The missing link in biological corrosion. *Acta Biomater*. 2012;8(3):1086–93.
24. Balamurugan A, Balossier G, Rajeswari S. Surface modification and corrosion behavior of titanium-based implants. *J Mater Sci Mater Med*. 2008;19(8):2799–807.