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Titanium degradation and peri-implant tissue health: A review

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Abstract:
Titanium and its alloys are widely used in dental implants because of their strength, corrosion resistance and predictable osseointegration. However, corrosion, mechanical wear and tribocorrosion can release titanium particles and ions into peri-implant tissues, potentially contributing to inflammation and marginal bone loss. Therefore, it is of interest to examine the titanium degradation mechanisms, particle distribution and biological pathways involving macrophage activation, cytokine release, osteolysis and inflammasome signalling. Diagnostic challenges include the absence of validated biomarkers and limited evidence establishing a direct causal relationship between titanium degradation and peri-implantitis. Thus, improved diagnostic methods, surface modifications, antimicrobial coatings and alternative implant materials may help reduce degradation-associated tissue damage and improve long-term peri-implant health.

Keywords: Dental implants (DI), titanium particles, peri-implantitis, tribocorrosion, osteolysis, titanium hypersensitivity, peri-implant tissue health

Background:
Endosseous dental implants have become a predictable and widely used option for the rehabilitation of partially and fully edentulous patients, with reported survival rates exceeding ninety percent over the first decade of function. Despite this success, biological complications affecting the tissues surrounding implants remain common and peri-implantitis, an inflammatory condition associated with progressive loss of supporting bone, is now recognised as a leading cause of late implant failure **[1]**. Identifying the causative factors of peri-implant inflammation and osteolysis is therefore central to prevention and treatment, yet the detailed pathogenesis of the disease remains incompletely understood **[2]**. Titanium-based implants inevitably release titanium particles into the adjacent tissue and the concentration of these particles rises markedly at peri-implantitis sites, positioning material degradation as a potential contributing risk factor alongside the established role of bacterial biofilm **[3]**. The concept of the implant as a permanently stable, inert biomaterial has been progressively revised. Titanium owes its biocompatibility to a thin, self-repairing titanium dioxide passivation layer; when this layer is disrupted, the underlying metal becomes vulnerable to further degradation. The combined effect of mechanical wear and corrosion, a process termed tribocorrosion, leads to the liberation of particles and ions, which in turn have been associated with inflammation, immune activation and biological complications, although a strict unidirectional chain of causation has not been demonstrated **[4]**. Because material degradation and the host response appear to interact through several feedback loops, understanding titanium behaviour in the oral cavity is essential to interpreting peri-implant disease. This review brings together current evidence on how titanium degrades, where the resulting particles accumulate, how they may injure peri-implant tissues, why they are difficult to diagnose and how future implant design may mitigate their effects **[5]**. Therefore, it is of interest to evaluate the mechanisms of titanium degradation, the biological effects of titanium particles and ions on peri-implant tissues.

Titanium as an implant biomaterial and mechanisms of degradation:
Several interrelated processes drive titanium degradation in the oral environment. Corrosion is an electrochemical phenomenon that is accelerated by acidic pH, high electrolyte concentrations, fluoride exposure and the metabolic by-products of adherent bacteria **[6]**. When implants or prosthetic components made of dissimilar metals are present in the same mouth, saliva acts as an electrolyte and permits galvanic coupling; the resulting micro-currents can accelerate breakdown of the protective oxide layer and electrogalvanic interactions have been described as an under-recognised contributor to implant corrosion and mucosal irritation **[7].** Titanium ions and particles already present in saliva may themselves aggravate corrosion, establishing a self-reinforcing cycle in which released products further compromise the surface **[8].** Mechanical factors act in concert with corrosion. Frictional loading during implant insertion generates measurable quantities of loose particles even before the implant is placed in function and the size of the released particles influences both the extent of ion release and their cytotoxic behaviour, with smaller particles presenting a greater surface-area-to-volume ratio and correspondingly higher biological reactivity **[9]**. Micro movements at the implant-abutment interface, masticatory loading and instrumentation during maintenance and decontamination each add to the cumulative particle burden. The interplay of wear and corrosion, exacerbated by environmental factors and biofilm, is what ultimately defines tribocorrosion as the principal route of titanium surface breakdown **[10]**.

Titanium particle release and distribution in peri-implant tissues:
Once liberated, titanium particles are consistently recovered from the tissues surrounding dental implants. Histological and analytical studies have identified titanium wear particles within peri-implantitis biopsies, where they are accompanied by overexpression of receptor activator of nuclear factor kappa-B ligand and other inflammatory markers, positioning particle accumulation as an emerging risk factor for peri-implant bone loss even though a direct causal relationship cannot be confirmed from cross-sectional data alone **[6]**. Particles vary widely in shape and size, spanning a range from roughly one

hundred nanometers to several tens of micrometres and are located around the implant as well as inside epithelial cells, connective tissue, macrophages and bone **[11]**. More recent tissue-level analyses confirm that titanium micro-particles are a common finding in the soft tissues adjacent to implants, reinforcing that their presence is not restricted to failing implants but occurs as a general consequence of implantation, while their quantity tends to be greater at sites of disease **[7]**. The immunopathological profile of peri-implantitis granulation tissue containing titanium particles differs from that of periodontitis, suggesting that the particles may modify the local inflammatory pattern rather than acting merely as passive debris **[12]**. Taken together, these observations establish that titanium is redistributed from the implant surface into a range of peri-implant compartments, providing the anatomical basis for a sustained host response.

Biological mechanisms linking titanium particles to peri-implant tissue damage:

The release of titanium particles and ions can influence peri-implant tissues through foreign body reactions, direct cellular effects, epigenetic changes and modulation of the oral microbiome and it has been proposed that marginal bone loss reflects a disturbance of the equilibrium between the host immune system and the implant **[13]**. Central to this response is the macrophage. *In vitro* exposure of human macrophages to titanium and zirconium oxide micro particles under sterile inflammatory conditions elicits a concentration-dependent pro-inflammatory response, indicating that the particles alone, in the absence of bacteria, are sufficient to activate innate immune cells **[14].** Activated macrophages secrete interleukin-1 beta, interleukin-6 and tumour necrosis factor alpha and adopt a pro-inflammatory M1 phenotype that favours osteoclast recruitment. The downstream consequence of this activation is osteolysis. Experimental work has shown that titanium particles stimulate osteoclastogenesis and bone resorption through nuclear factor kappa-B signalling and autophagy-related pathways and that interrupting these pathways can attenuate particle-induced inflammation and bone loss. Beyond direct cytokine release, the inflammasome represents an additional node; a correlation between inflammasome activity and the peri-implant microbiota suggests that particle-driven innate immune sensing and microbial dysbiosis are intertwined in the peri-implant lesion **[15]**. Epigenetic regulation adds a further layer of complexity: the presence of titanium particles has been associated with altered DNA and RNA methylation in peri-implant tissues, which can influence macrophage and lymphocyte polarisation and thereby shape the intensity and persistence of the inflammatory response **[16]**. Collectively, these mechanisms describe a self-amplifying loop in which particles provoke inflammation, inflammation aggravates corrosion and further particle release and the resulting osteoclastic activity drives the bone loss that characterizes peri-implantitis.

Titanium hypersensitivity and systemic implications:

In a subset of patients, the response to titanium degradation products appears to extend beyond a generic foreign body reaction toward hypersensitivity. Reviews of implant failures attributed to titanium allergy report cases in which symptoms resolved after removal of the titanium fixtures and their replacement with ceramic implants, although true titanium allergy is difficult to separate from other causes of failure and no single confirmatory test exists **[17]**. The immune basis is debated, with evidence favouring innate, particle-driven macrophage hyperreactivity, particularly toward nanoparticles, over classical adaptive allergic sensitisation **[18]**. The concern is not confined to the peri-implant site. Titanium and other metallic ions released from implants can be detected in the systemic circulation and studies assessing blood metal-ion levels in implant-bearing patients have reported measurable concentrations attributable to the implants **[19]**. Although the clinical significance of low-level systemic titanium remains uncertain, the possibility that degradation products migrate to distant organs underlines the importance of characterising and, where possible, minimising particle release.

Diagnostic challenges:

Diagnosing the contribution of titanium degradation to peri-implant disease is inherently difficult. Because titanium particles are present around both healthy and diseased implants, their mere detection is not diagnostic and the challenge lies in distinguishing a clinically relevant particle-driven process from bacterially driven peri-implantitis. Salivary and fluid-based biomarkers have been explored as non-invasive indicators: analyses of salivary titanium, nickel, vanadium and arsenic in implant patients have shown detectable metal concentrations, though some studies found no statistically significant difference between healthy and diseased implants, highlighting the limited discriminatory power of a single measurement **[20].** Tests intended to identify titanium hypersensitivity face similar limitations. Systematic evaluation of epicutaneous testing and lymphocyte-based assays such as the lymphocyte transformation test and the memory lymphocyte immunostimulation assay has revealed inconsistent reliability and validity, leading to the recommendation that inflammatory clinical signs, rather than allergy tests alone, should guide decision-making. To improve early detection, computational approaches have been proposed, including artificial neural network models that aim to predict the onset of peri-implantitis from clinical parameters, offering a potential adjunct to conventional radiographic and probing assessment **[21].** Nonetheless, the field still lacks a validated, particle-specific diagnostic marker and interpretation continues to rely on the integration of clinical, radiographic and, where indicated, histological findings.

Future perspectives:

Efforts to preserve peri-implant health increasingly focus on preventing degradation and its biological consequences at the material level. Physical and chemical modification of the titanium surface, together with the application of metal-element and antimicrobial coatings, has demonstrated the capacity to

reduce bacterial colonisation and biofilm formation *in vivo*, addressing one of the drivers of the corrosion-inflammation cycle **[22]**. More sophisticated delivery systems, such as layer-by-layer coatings capable of releasing therapeutic agents in a controlled manner, are being developed to combat implant-related infection while maintaining favourable surface properties **[23]**. In parallel, ceramic materials, notably zirconia, are being investigated as alternatives that are non-corrosive and appear less prone to eliciting the immune responses associated with titanium degradation, particularly for patients with suspected hypersensitivity **[24]**. Realising the promise of these approaches will require long-term clinical evidence and standardised methods for quantifying particle release, so that material innovation can be matched to individual patient risk.

Conclusion:

Titanium degradation through corrosion, wear and tribocorrosion releases particles and ions that may contribute to peri-implant inflammation, immune activation and marginal bone loss. Although current evidence supports a biological association, a direct causal relationship remains unproven and diagnosis is limited by the absence of validated biomarkers. Surface modifications, antimicrobial coatings and ceramic alternatives offer promising strategies to preserve peri-implant tissue health, pending robust long-term clinical validation.

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