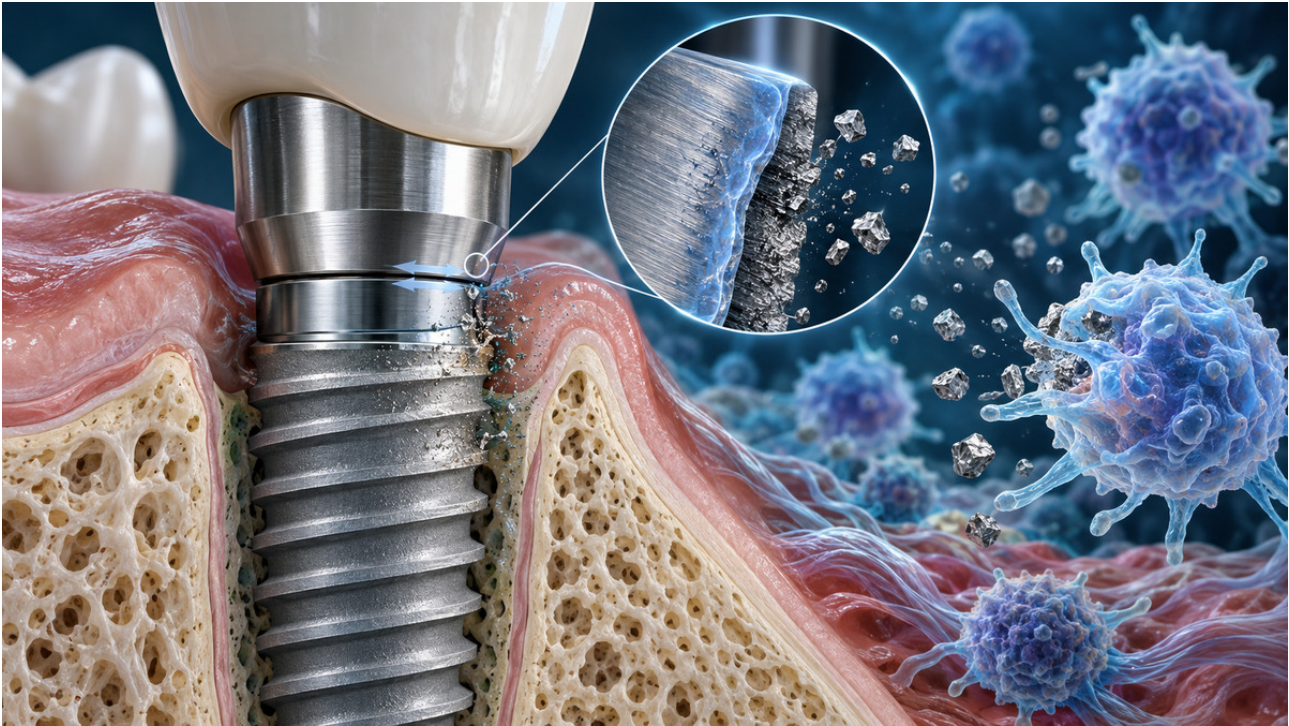


Titanium at the Peri-implant Interface: Corrosion, Wear and Particle Biology

Material degradation, immune recognition and the limits of causal interpretation in peri-implant disease



Core message: titanium is highly corrosion resistant, but it is not completely inert. Ions and particles can arise during placement, function, maintenance or treatment. Their presence can influence cells, yet detection alone does not prove that titanium caused peri-implantitis.

Educational material for dental students, postgraduate trainees and academic teachers

Passivation protects titanium

Titanium rapidly forms a thin oxide film when exposed to oxygen and water. This passive layer limits electrochemical dissolution and supports the high clinical biocompatibility of titanium implants. Damage to the film can be repaired by repassivation when the local chemical environment allows it.

Concept	Material process	Clinical relevance
Passive oxide film	A nanometre-scale titanium oxide layer separates the metal from the environment.	Reduces corrosion and interacts with proteins, cells and biofilm.
Repassivation	A damaged oxide film reforms after exposure of fresh metal.	Helps resist isolated mechanical injury.
Oxide disruption	Friction, abrasion, micromotion or instrumentation removes part of the film.	Temporarily exposes reactive metal and can release debris.
Chemical destabilization	Low pH, reactive species or selected chemical agents alter electrochemical conditions.	May slow repassivation or increase ion release in experimental settings.
Surface modification	Blasting, etching or coatings change roughness and chemistry.	Influences osseointegration and the pattern of surface damage.

Biocompatibility does not mean zero interaction

The implant surface adsorbs proteins and contacts bone, connective tissue, immune cells and microorganisms. Successful osseointegration is an active biological equilibrium, not proof that no metal products are ever released.

Material perspective: titanium performs well because passivation usually controls degradation. Clinical longevity depends on maintaining mechanical and biological conditions that support that protection.

Corrosion in the oral environment

Corrosion is electrochemical degradation of a material. The oral cavity provides electrolytes, changing pH, oxygen gradients, proteins and microbial metabolites. The implant, abutment and prosthesis can also create dissimilar-material and crevice conditions.

Process	Mechanism	Where it may occur
Uniform corrosion	Relatively even dissolution across an exposed surface.	Uncommon as a dominant pattern on well-passivated titanium.
Crevice corrosion	Restricted fluid exchange creates a locally aggressive chemical environment.	Implant-abutment interfaces, gaps and shielded regions.
Galvanic corrosion	Electrically connected materials with different potentials drive current.	Mixed alloys or restorative components under suitable conditions.
Pitting	Localized breakdown of the passive film produces small corrosion sites.	Defects, inclusions or chemically aggressive microenvironments.
Fretting corrosion	Small repetitive movements disrupt the passive film and accelerate chemical attack.	Connections exposed to micromotion.
Inflammation-associated corrosion	Low pH and reactive oxygen species can alter surface stability.	Inflamed peri-implant tissues and biofilm-rich pockets.

Cause and consequence can reinforce each other

Inflammation can make the environment more corrosive. Material products can then stimulate cells under experimental conditions. This feedback model is biologically plausible, but clinical studies cannot always determine which process began first.

Fluoride requires concentration and pH context

Laboratory studies show that acidic, fluoride-containing media can challenge titanium passivation. Routine fluoride use should not automatically be equated with implant corrosion. Product concentration, pH, contact time and surface condition determine relevance.

Avoid simple attribution: biofilm, inflammation, micromotion and material degradation can coexist. Finding one component does not establish the initiating cause.

Wear and tribocorrosion

Wear is mechanical material loss. Tribocorrosion describes the interaction between wear and corrosion, where friction damages the passive film and the chemical environment attacks the newly exposed surface. Repassivation then occurs, and repeated cycles can generate ions and particles.

Source	Mechanical event	Possible material product
Implant insertion	Thread-bone friction and high local resistance abrade surface peaks.	Titanium particles near thread crests and within the osteotomy.
Implant-abutment interface	Functional micromotion and fretting at contacting components.	Metal debris, ions and changes at the connection.
Loose or fractured components	Abnormal movement increases repeated contact and wear.	Greater local debris and mechanical damage.
Professional instrumentation	Curettes, ultrasonic tips or rotary devices contact the implant surface.	Scratches, displaced oxide and instrument-derived material.
Implantoplasty	Deliberate rotary smoothing removes exposed implant threads.	Substantial titanium debris unless capture and irrigation are effective.
Prosthetic adjustment	Grinding or polishing of components creates particulate matter.	Metal and abrasive debris outside or within the operative field.

Size and chemistry matter

Metal products range from dissolved ions and nanoscale particles to visible fragments. Size, shape, surface charge, oxide state and protein coating influence transport, cellular uptake and biological activity. Studies using one particle preparation cannot represent every clinical exposure.

Prevention starts with mechanics: correct component fit, controlled placement, stable connections and appropriate maintenance reduce avoidable surface damage.

How titanium is detected

Titanium has been identified in peri-implant mucosa, bone and cellular samples. Detection methods vary in spatial resolution and chemical specificity. Differences in sampling and analysis partly explain why studies report inconsistent particle concentrations.

Method	Information provided	Limitation
Histology with light microscopy	Tissue architecture and pigmented or refractile material.	Cannot identify titanium reliably without elemental analysis.
SEM with EDS	Particle morphology and elemental composition at selected points.	Small sampled area and limited sensitivity for dissolved ions.
ICP-MS or related spectroscopy	Highly sensitive total elemental concentration.	Usually loses spatial information and does not show particle form.
Micro-XRF or synchrotron methods	Elemental maps with improved spatial resolution.	Specialized access, preparation and interpretation.
Macrophage markers	CD68 or related staining identifies macrophage-rich responses.	Shows cell phenotype, not the source or causal effect of particles.
Clinical cytology or fluid sampling	Potential minimally invasive detection of metal products.	No validated diagnostic threshold for peri-implantitis causation.

Presence is not provenance

A detected titanium signal may originate from implant placement, functional wear, maintenance, implantoplasty or sample contamination. The analysis must consider when and how the specimen was obtained.

Presence is not causation

Titanium particles are also found around clinically healthy implants. A 2025 human study reported that microparticles were common in peri-implant soft tissues and were not associated with the occurrence of peri-implantitis.

Analytical discipline: report the method, sample location, particle size range and controls before interpreting biological significance.

Cellular responses to particles and ions

Macrophages can phagocytose titanium particles and respond according to particle characteristics and the surrounding microbial or inflammatory signals. Experimental studies describe inflammasome activation, cytokine release, osteoclast support and impaired osteoblast function under selected exposure conditions.

Cell or pathway	Potential response	Evidence boundary
Macrophages	Particle uptake, polarization changes and release of IL-1 beta, TNF-alpha or other mediators.	Responses depend strongly on dose, particle preparation and contamination.
Inflammasome pathways	Danger-signal recognition can amplify IL-1 family cytokine processing.	In vitro activation does not establish clinical disease initiation.
Osteoclast lineage	Pro-inflammatory signaling can favor differentiation and resorptive activity.	Biofilm and host inflammation provide overlapping stimuli.
Osteoblasts and stromal cells	High experimental particle loads may reduce viability or osteogenic function.	Clinical tissue exposure is difficult to quantify.
T lymphocytes	Adaptive immune activation or altered local profiles may occur.	This is not equivalent to a validated diagnosis of titanium allergy.
Neutrophils and biofilm	Reactive species and microbial products may modify corrosion and inflammation.	The direction of cause and effect remains difficult to separate.

Macrophages are responders, not a chairside test

CD68-positive cells containing particles demonstrate tissue interaction. They do not define why the implant failed, quantify the relevant dose or distinguish wear from corrosion without additional analysis.

Biological interpretation: particle responses are plausible modifiers of an established inflammatory environment. Their independent contribution in individual patients remains uncertain.

Titanium and peri-implantitis: an evidence hierarchy

Peri-implantitis is a plaque-associated inflammatory disease with progressive supporting-bone loss. Material degradation may interact with this process, but the evidence differs substantially between laboratory experiments and patient outcomes.

Evidence type	Finding	Strength for clinical causation
Cell culture	Particles or ions can provoke inflammatory signaling and reduce osteogenic activity.	Mechanistic plausibility only; dose and preparation may not match clinical exposure.
Animal models	Debris can alter local inflammation or bone response under controlled conditions.	Supports biological potential but not routine human incidence.
Human tissue studies	Titanium is often detected around implants and sometimes at higher levels in diseased sites.	Association can reflect disease-driven corrosion, treatment or sampling.
Cross-sectional clinical studies	Particle burden and peri-implant status can be compared at one time.	Cannot establish temporal direction.
Longitudinal studies	Could test whether early material products predict later disease.	Currently limited and methodologically difficult.
Intervention studies	Would test whether reducing debris changes clinical outcomes.	Sparse and confounded by simultaneous biofilm treatment.

Association can run in both directions

Inflamed pockets expose more implant surface to acidic biofilm and instrumentation, which can increase particle release. Therefore, higher titanium levels in peri-implantitis may be a consequence, a modifier or both.

Particle detection does not redefine diagnosis

Clinical diagnosis remains based on bleeding or suppuration, increased probing depth and progressive bone loss compared with baseline. Tissue titanium measurements do not replace these criteria and have no accepted chairside threshold.

Calibrated conclusion: titanium products can participate in local biology, but current evidence does not justify describing peri-implantitis as a metal-particle disease.

Hypersensitivity and “titanium intolerance”

True immune-mediated reactions to titanium are considered uncommon and remain difficult to define. Local inflammation, failed osseointegration, infection, cement, mechanical problems and reactions to other alloy components can produce overlapping symptoms.

Approach	Possible role	Major limitation
Clinical history	Identifies timing, prior metal reactions, repeated failures and systemic or dermatological symptoms.	Symptoms are non-specific and susceptible to attribution bias.
Patch testing	Can evaluate contact allergy to selected compounds.	Titanium salts have limited skin penetration and no universally validated reagent.
Lymphocyte transformation tests	Measure lymphocyte proliferation after laboratory exposure.	Variable methods, uncertain clinical thresholds and limited predictive validity.
MELISA-type testing	A modified in vitro lymphocyte response assay.	No consensus that it establishes implant causation or predicts outcome.
Histology and elemental analysis	Characterizes tissue inflammation and material presence.	Invasive and unable to prove systemic hypersensitivity alone.
Response after removal	Symptom improvement can support a clinical hypothesis.	Placebo effects, removal of infection and simultaneous treatment confound interpretation.

Routine pre-implant screening is not supported

Current reviews and the German S3 guideline do not support allergy testing as a general screening tool for titanium implant candidates. Testing may be considered only within specialist evaluation of a convincing clinical problem.

Evaluate the complete system

Abutments, prosthetic alloys, cements and instruments may contain other metals or materials with better-established allergenic potential. A narrow focus on titanium can miss the actual cause.

Terminology: use “suspected material-related reaction” until a structured differential assessment supports a more specific conclusion.

Clinical prevention and investigation

Stage	Risk-reduction action	Reason
Implant placement	Use the correct osteotomy, controlled torque and minimal insertion-removal cycles.	Limits avoidable thread damage and particle release.
Prosthetic connection	Verify component compatibility, fit, preload and occlusal design.	Reduces micromotion and fretting at interfaces.
Home care	Control biofilm with products appropriate for the implant and mucosa.	Reduces inflammation and an acidic microbial environment.
Professional maintenance	Use effective instrumentation that minimizes unnecessary surface damage.	Balances debridement with material preservation.
Peri-implantitis treatment	Capture debris during implantoplasty and use copious irrigation when the procedure is chosen.	Reduces retention of generated particles in the surgical field.
Unexplained failure	Assess infection, position, loading, connection, cement, medication and tissue pathology.	Prevents premature attribution to titanium intolerance.

A focused diagnostic pathway

- Document the timing and pattern of symptoms, previous implants and relevant skin or metal reactions.
- Evaluate plaque, probing findings, suppuration, progressive bone loss and prosthetic cleansability.
- Check implant position, connection stability, component compatibility and occlusal loading.
- Review systemic disease, medication and alternative mucosal or bone diagnoses.
- Seek oral medicine, dermatology, pathology or biomaterials expertise when the clinical pattern remains unexplained.

When tissue analysis is reasonable

Histology and elemental analysis may add value when tissue is already being removed for a clinical indication, when a neoplastic or unusual inflammatory lesion requires biopsy, or when a structured research protocol addresses material degradation.

Clinical priority: first identify treatable plaque, prosthetic and mechanical causes. Material analysis is an adjunct for selected unresolved cases.

Clinical synthesis and selected references

Clinical synthesis

- Titanium resists corrosion through a self-repairing passive oxide layer.
- Mechanical damage and aggressive chemistry can disrupt passivation and release ions or particles.
- Tribocorrosion couples friction with electrochemical degradation.
- Placement, connection micromotion, maintenance and implantoplasty are potential debris sources.
- Analytical methods measure different aspects of particle burden and require appropriate controls.
- Macrophage uptake demonstrates tissue interaction but does not prove disease causation.
- Experimental particles can stimulate inflammatory and osteolytic pathways under selected conditions.
- Titanium particles also occur around clinically healthy implants.
- Routine patch or lymphocyte testing does not reliably screen for titanium intolerance.
- Interpret peri-implantitis through biofilm, mechanics, material factors and host response together.

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Educational use and limitations

This material supports biomaterial-aware implant care and differential diagnosis. It does not establish material causation in an individual patient or replace periodontal, prosthetic, pathological, dermatological or biomaterials specialist assessment.

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