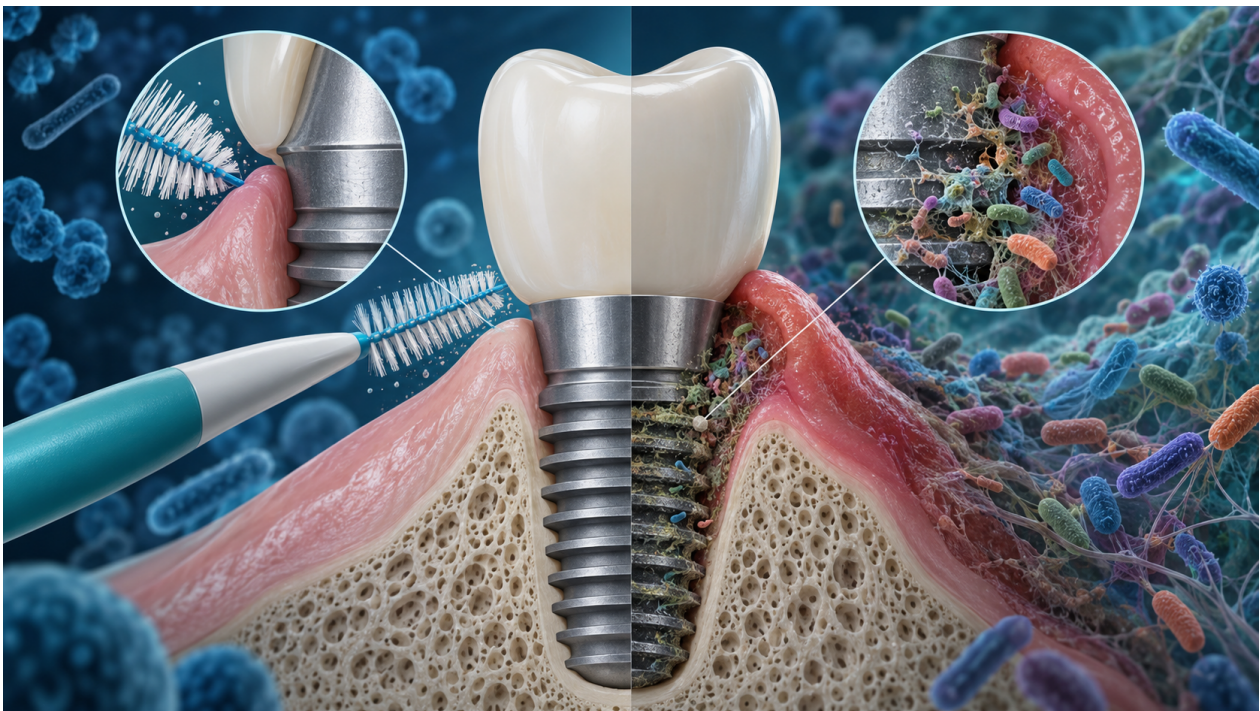


The Peri-implant Biofilm Ecosystem: Dysbiosis, Cleansability, and Local Disease Drivers

An evidence-based guide to microbial ecology, implant niches and modifiable local determinants



Conceptual scientific illustration. Microbial forms are illustrative and do not represent a diagnostic microbiological test.

Core message: biofilm provides the primary microbial exposure, while host response and local ecological niches determine whether inflammation resolves or develops into persistent peri-implant disease.

Educational material for dental students, postgraduate trainees and academic teachers

Purpose and learning outcomes

This material explains peri-implant disease through an ecological model. Biofilm, the host response, implant surface characteristics and prosthetic access interact continuously. The result depends on whether the patient and clinical team can disrupt the biofilm and maintain a cleanable environment.

After completing the material, the reader should be able to:

- Explain why biofilm is the primary etiological exposure in peri-implant mucositis and peri-implantitis.
- Describe dysbiosis as a change in microbial community structure and function rather than infection by one pathogen.
- Identify patient, implant, tissue and prosthetic features that create plaque-retentive niches.
- Distinguish established local determinants from associations with uncertain clinical thresholds.
- Translate cleansability into prevention, maintenance and treatment planning.

An ecological framework

A stable peri-implant microbiota coexists with the host under conditions of health. Persistent plaque accumulation triggers inflammation. The resulting exudate, deeper pocket and reduced oxygen tension alter the environment, favouring organisms adapted to the diseased niche. Dysbiosis and inflammation can then reinforce one another.

Clinical emphasis

Microbial complexity does not change the primary clinical priorities. The clinician should identify plaque-retentive anatomy, improve access for daily cleaning, disrupt biofilm professionally and monitor the tissue response with reproducible measures.

Dysbiosis is an ecological diagnosis. The presence or absence of one named organism neither proves nor excludes peri-implantitis.

Biofilm as the initiating exposure

Experimental plaque accumulation produces peri-implant mucositis, and removal of the microbial deposit can restore health in many sites. Peri-implantitis is therefore classified as a plaque-associated pathological condition, although plaque exposure alone does not explain every difference between patients or implants.

Stage	Ecological change	Clinical consequence
Early colonisation	Microorganisms attach to the acquired pellicle on titanium and prosthetic materials.	A structured biofilm begins to form on a non-shedding surface.
Mature biofilm	Co-aggregation and extracellular matrix create a protected microbial community.	Mechanical removal becomes more important than rinsing alone.
Inflammatory shift	Host-derived nutrients and reduced oxygen alter the local environment.	Inflammation and dysbiosis begin to reinforce each other.
Deepened niche	Pocket formation and exposed rough implant surfaces provide sheltered retention sites.	Daily disruption and closed professional debridement become less predictable.

The implant surface as a niche

Titanium implants have macrogeometry, microtopography and thread patterns that differ from natural teeth. Once rough surfaces become exposed to the submucosal environment, they retain complex biofilm and limit instrument contact. Defect anatomy and restoration contour can create additional sheltered areas.

Host response modifies the outcome

Similar plaque levels do not produce identical disease in every patient. Previous periodontitis identifies a susceptible phenotype, while diabetes control, smoking and other exposures may modify inflammatory regulation. Local ecology and host susceptibility should therefore be interpreted together.

Biofilm is necessary but not sufficient to predict severity. Disease expression reflects exposure, susceptibility, anatomy and time.

Dysbiosis without the single-pathogen fallacy

Culture-independent studies show that peri-implant health, mucositis and peri-implantitis have overlapping but distinguishable microbial communities. Diseased sites often show greater diversity, altered community networks and enrichment of anaerobic or opportunistic taxa. Findings vary across sampling methods, populations and analytical platforms.

Interpretation	Appropriate conclusion	Common error
A taxon is detected	The organism forms part of the sampled community at the time of collection.	Assuming that detection proves it caused disease.
A taxon is absent	The assay did not detect it above the method-specific threshold.	Assuming that absence excludes peri-implantitis.
Community composition differs	The ecological state may have shifted in association with inflammation.	Treating every compositional difference as a clinical action threshold.
Microbial load falls after treatment	Biofilm disruption changed the sampled community or quantity.	Assuming that microbiological change guarantees durable clinical resolution.

Bidirectional reinforcement

Biofilm initiates mucosal inflammation, but inflammation also changes the habitat. Gingival crevicular fluid, tissue breakdown products and a deeper anaerobic pocket provide new substrates and physical protection. The community adapts to the inflammatory environment, and the altered community can sustain further host activation.

Limits of routine microbial testing

Commercial assays sample only part of the community and use method-specific detection thresholds. Microbial profiles overlap across health and disease, and results rarely change the primary clinical action: evaluate inflammation, bone levels, cleansability and adherence. Testing may support selected research or complex cases, but it does not replace clinical diagnosis.

Clinical decisions should not depend on a microbial shopping list. Current evidence supports an ecological, polymicrobial interpretation.

Cleansability as a biological design requirement

Prosthetic design determines whether the patient can reach the peri-implant margin and whether the clinician can examine and instrument the site. A restoration can therefore convert a biologically manageable interface into a persistent plaque-retentive niche.

Design feature	Potential effect	Preventive response
Excess convexity	Limits brush and interdental access close to the mucosal margin.	Modify contour when feasible and select site-specific cleaning devices.
Deep or over-contoured margin	Restricts visual control and professional instrumentation.	Plan retrievability and assess access before final delivery.
Narrow embrasure	Prevents insertion of an appropriately sized interdental aid.	Create an emergence profile compatible with daily hygiene.
Cement-retained restoration	Residual cement can act as a local irritant and plaque-retentive factor.	Use controlled cementation and exclude residual material when inflammation is localised.
Non-retrievable prosthesis	Limits diagnostic access and correction of local contributors.	Include retrievability in treatment planning where possible.

Assessment before delivery

Cleansability should be tested before definitive prosthesis delivery. The patient should demonstrate access with the selected brush, interdental device or floss system. The clinical team should also confirm that the restoration permits probing and professional instrumentation.

Assessment after delivery

Re-evaluate the interface at maintenance visits. Tissue changes, reduced dexterity or new prosthetic components can alter access over time. Persistent plaque at the same surface suggests a design or technique problem rather than a need for generic oral hygiene advice.

A cleanable prosthesis is part of disease prevention. Access for self-care and professional examination should be treated as clinical outcomes.

Implant position, tissues and foreign material

Local determinant	Evidence-informed interpretation	Clinical response
Buccolingual position	Position affects tissue dimensions, restoration contour and hygiene access.	Use restorative-driven planning within the available bone envelope.
Vertical depth	Excess depth can require a taller transmucosal contour and complicate probing or debridement.	Balance aesthetic and surgical goals with long-term access.
Keratinized mucosa	No universal width threshold defines long-term health, but limited tissue may increase discomfort during cleaning.	Treat symptoms and access limitations rather than a number alone.
Residual cement	Cement can mimic or amplify plaque-associated inflammation and may escape radiographic detection.	Inspect clinically and consider prosthesis removal when necessary.
Defect anatomy	Intrabony and circumferential defects shelter exposed rough surfaces.	Recognise the limits of closed debridement and plan access appropriately.

Keratinized mucosa

Evidence does not establish one minimum width that prevents peri-implantitis in every patient. Adequate keratinized mucosa may improve comfort during brushing and facilitate plaque removal. Decisions about soft-tissue augmentation should therefore consider symptoms, recession, hygiene access and the planned restoration rather than a numerical threshold alone.

Residual cement and other foreign material

Local irritants can produce persistent inflammation or amplify the response to plaque. Radiographs do not reveal every cement deposit because material radiopacity, projection and location vary. A focal lesion adjacent to a cement-retained restoration should prompt careful clinical inspection and consideration of prosthetic retrievability.

Local risk assessment begins before surgery. Implant position, restoration design and tissue access should be planned as one system.

From mucositis control to treatment escalation

Peri-implant mucositis provides an important prevention checkpoint. Effective plaque control can resolve inflammation, although complete resolution may require more than three weeks and does not occur in every site after one intervention. Persistent bleeding should trigger reassessment of technique, access and adherence.

Phase	Primary objective	Practical elements
Self-performed care	Disrupt biofilm daily at every accessible surface.	Site-specific instruction, appropriate brush size and verification of patient technique.
Professional plaque removal	Remove biofilm and deposits that the patient cannot reach.	Implant-compatible instrumentation and correction of obvious local irritants.
Re-evaluation	Determine whether inflammation has resolved and access has improved.	Repeat bleeding, probing and plaque measurements using the same sites.
Peri-implantitis escalation	Gain access to residual diseased surfaces and defects.	Consider surgical access when deep pockets and exposed rough surfaces limit closed therapy.
Supportive care	Maintain a low biofilm burden and detect recurrence early.	Risk-based recall, reproducible site maps and periodic radiographic comparison.

Why decontamination is difficult

Implant threads, roughness and defect walls create sheltered regions that instruments cannot contact completely. No single mechanical, chemical or energy-based method predictably sterilises the implant surface. Treatment aims to reduce the bioburden, remove deposits, improve access and create conditions in which the host can resolve inflammation.

Behaviour and access come first

Non-surgical treatment starts with effective self-care and professional mechanical biofilm disruption. Adjunctive products cannot compensate for an inaccessible restoration or ineffective daily plaque control. Prosthetic obstacles should be corrected when the expected benefit justifies modification.

Treatment success depends on maintainability. A surface that cannot be reached after therapy remains vulnerable to recolonisation.

A four-level local risk assessment

Level	Questions to ask	Modifiable targets
Patient	Can the patient see, reach and disrupt plaque consistently? Are dexterity, motivation or discomfort limiting?	Technique, device selection, coaching and recall frequency.
Implant and site	Does position, depth, tissue architecture or defect anatomy create a protected niche?	Early planning, soft-tissue management and treatment access.
Prosthesis	Are contour, margins, embrasures and retrievability compatible with maintenance?	Contour adjustment, removal of residual cement and prosthetic modification.
Clinical system	Can the team probe, instrument and monitor the same sites reproducibly?	Site maps, consistent indices, appropriate instruments and risk-based recalls.

Maintenance designed around detectability

A maintenance visit should allow the clinician to detect change, not merely polish accessible surfaces. When the prosthesis blocks examination or instrumentation, removal or modification may be necessary. Use consistent site maps and increase recall frequency when inflammation persists or the patient has a history of peri-implantitis.

Common interpretation errors

- Attributing peri-implantitis to one named organism or treating its absence as exclusionary.
- Assuming that antimicrobial rinses can replace mechanical biofilm disruption.
- Giving generic oral hygiene advice without testing access around the actual prosthesis.
- Using keratinized mucosa width as an automatic treatment threshold.
- Ignoring cement, prosthetic contour or implant position when inflammation remains localised.
- Expecting one decontamination method to sterilise a complex exposed implant surface.

Key learning points

- Biofilm initiates peri-implant inflammation, while host susceptibility and local ecology modify the outcome.
- Dysbiosis describes a community shift and cannot be reduced to one pathogen.
- Implant position and prosthetic contour determine access for self-care and professional monitoring.
- Mucositis control provides the main opportunity to prevent destructive disease.
- Maintenance should combine biofilm control, correction of local niches and reproducible surveillance.

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Educational use and limitations

This material is intended for education and does not replace individual clinical assessment or specialist consultation. Microbiological testing, imaging and prosthetic modification should be selected only when the result can change diagnosis or management.

This work was co-funded by the European Union under grant agreement No. 2024-1-PL01-KA220-HED-000251208. Views and opinions expressed are those of the author(s) only and do not necessarily reflect those of the European Union or the Foundation for the Development of the Education System (FRSE). Neither the European Union nor FRSE can be held responsible for them.