

Dyslipidaemia and the Bone-Implant Interface: Oxidized Lipids, Redox Biology and Clinical Interpretation

Lipid measurements, oxidized LDL pathways, bone remodelling and evidence-calibrated decisions in implant care



Core message: dyslipidaemia can alter inflammatory and skeletal biology, but most direct evidence for impaired osseointegration comes from experimental research. A lipid result contributes to the medical context and does not independently diagnose implant risk or determine eligibility.

Educational material for dental students, postgraduate trainees and academic teachers

The lipid profile in clinical context

A standard lipid profile estimates circulating cholesterol and triglyceride transport. Its main validated use is cardiovascular risk assessment and treatment. Dental clinicians may encounter the results during medical history review, but interpretation should remain aligned with the patient's cardiovascular risk category and medical plan.

Measure	Biological meaning	Clinical boundary in implant care
Total cholesterol	Cholesterol carried across several lipoprotein classes.	A summary value that can conceal different atherogenic patterns.
LDL-C	Cholesterol carried in low-density lipoproteins and a central target of cardiovascular prevention.	The treatment target varies with global cardiovascular risk; no implant-specific target exists.
HDL-C	Cholesterol within particles involved in reverse cholesterol transport and other functions.	A high value does not neutralize elevated LDL-C or other risk.
Triglycerides	Lipids transported mainly in triglyceride-rich particles; elevation often accompanies insulin resistance.	Interpret with metabolic context, fasting status when relevant and secondary causes.
Non-HDL-C and apoB	Alternative estimates of atherogenic particle burden in selected clinical settings.	Useful for cardiovascular care but not validated as implant eligibility measures.

Reference ranges and treatment targets are different concepts

LDL-C goals depend on established cardiovascular disease, diabetes, kidney disease, calculated risk and other modifiers. The 2025 ESC/EAS focused update retained risk-based LDL-C goals. Dental teams should record results and medical treatment without assigning a new target for implant placement.

No dental cutoff: no cholesterol, LDL-C, HDL-C or triglyceride value has been validated as a universal threshold for implant surgery.

Dyslipidaemia and skeletal biology

Bone responds to endocrine, vascular, immune and nutritional signals. Dyslipidaemia can coexist with obesity, diabetes, metabolic liver disease, smoking and reduced physical activity. These conditions influence bone through overlapping pathways and make it difficult to isolate a lipid effect in human implant studies.

Biological domain	Potential effect of lipid dysregulation	Evidence boundary
Oxidative balance	Lipid oxidation can generate reactive products and amplify cellular stress.	The concentration, exposure time and tissue environment determine the response.
Inflammatory signalling	Modified lipoproteins may activate endothelial, immune and stromal pathways.	Systemic inflammation has many sources and cannot be attributed to LDL alone.
Vascular function	Atherogenic particles can impair endothelial biology and tissue perfusion.	A plasma result does not directly measure perfusion at an implant site.
Osteoblast-lineage cells	Experimental exposure may reduce differentiation, matrix production or mineralization.	Dose and model conditions may not reflect clinical exposure.
Osteoclast pathways	Oxidized lipids can modify RANKL signalling, differentiation and resorption.	Published effects differ by cell type, oxidation level and experimental system.

Association does not identify the operative pathway

A patient with high triglycerides may also have dysglycaemia, visceral adiposity and metabolic liver disease. Each condition may contribute to inflammation or altered healing. A clinical decision should therefore use the whole medical and local profile rather than assign causation to one laboratory value.

Mechanistic framework: lipid dysregulation may modify the healing environment, while local stability, vascularized tissue, plaque control and loading remain essential determinants.

Oxidized LDL and LOX-1 signalling

Oxidized LDL is a heterogeneous group of LDL particles modified by oxidative processes. It differs from the LDL-C concentration reported in a routine lipid profile. Cells can recognize modified particles through scavenger receptors, including lectin-like oxidized LDL receptor-1, known as LOX-1.

Step	Biological event	Interpretive caution
Particle modification	Lipids and apolipoprotein components undergo oxidative change.	There is no single uniform oxLDL molecule or universally standardized assay.
Receptor recognition	LOX-1 and other scavenger receptors bind modified lipoproteins.	LOX-1 expression varies by cell type and inflammatory environment.
Intracellular signalling	MAPK, NF-kappaB, oxidative and cytokine pathways may change.	Pathway activation in vitro does not quantify clinical implant risk.
Matrix and bone effects	Osteogenic differentiation, RANKL-related signalling and matrix turnover may be altered.	Direction and magnitude differ across experimental systems.
Clinical measurement	Research assays can quantify selected oxLDL epitopes or particles.	Routine oxLDL testing is not established for implant planning.

LOX-1 is part of a receptor network

CD36, LDL receptors, Toll-like receptors and other pathways also participate in lipid sensing. A single receptor model is useful for explaining biology but cannot capture the full response of bone, immune and vascular cells.

Oxidative stress is not measured by one marker

Oxidative stress describes an imbalance between oxidant production and protective systems. OxLDL can reflect part of this process, but it does not provide a complete or site-specific measure of redox status.

Clinical boundary: oxLDL and LOX-1 currently belong mainly to mechanistic and translational research, not routine dental risk stratification.

Osteoblasts, progenitor cells and marrow adiposity

Osteoblast-lineage cells form osteoid, regulate mineralization and communicate with osteoclast precursors. Experimental lipid excess and oxidized lipids can impair osteogenic differentiation through oxidative, inflammatory and mitochondrial pathways.

Cellular process	Potential disturbance	Possible tissue consequence
Progenitor commitment	Metabolic conditions may shift mesenchymal progenitor activity away from osteogenesis and toward adipogenic programmes.	Reduced regenerative capacity and greater marrow adiposity in some contexts.
Osteoblast differentiation	Oxidative products may suppress osteogenic transcription and phosphate-related mineralization pathways.	Less mature bone-forming activity.
Matrix production	Collagen synthesis and matrix organization may become less efficient.	Delayed formation of a stable bone-implant interface.
Cell survival	High experimental oxLDL exposure can reduce viability and increase cellular stress.	Loss of active bone-forming cells in susceptible models.
Coupling signals	Osteoblast-lineage cells can change RANKL and osteoprotegerin expression.	Altered communication with osteoclast precursors.

Marrow appearance on CBCT

Trabecular pattern and marrow density can prompt broader clinical thinking, but CBCT cannot diagnose marrow adiposity, dyslipidaemia or oxidative stress. Imaging findings require radiological interpretation and correlation with history. They should not trigger metabolic treatment without medical evaluation.

Human translation remains incomplete

Cell culture studies expose cells to defined lipid preparations at selected concentrations. Human tissues experience changing mixtures of native and modified particles, medications, hormones and mechanical loading. This gap limits direct conversion of laboratory findings into a clinical cutoff.

Translational principle: experimental effects explain plausibility. They do not provide an individual probability of osseointegration.

Osteoclasts and context-dependent remodelling

Osteoclasts remove damaged bone and support coupled remodelling. Lipid-related inflammation can promote osteoclastogenic signals, yet direct oxLDL exposure has also inhibited osteoclast differentiation or resorption in some experimental models. These findings are not necessarily contradictory because the result depends on the cells, dose, oxidation state and surrounding signals.

Experimental context	Reported direction	Why results can differ
Osteoblast-osteoclast co-culture	oxLDL may increase osteoblast RANKL and support osteoclast formation.	Indirect signalling differs from direct exposure of precursors.
Direct precursor exposure	Some studies report inhibition of RANKL-induced differentiation.	High oxidative burden may suppress fusion or cell function.
Inflammatory co-stimulation	Toll-like receptor and cytokine signals may increase LOX-1 and favour osteoclastogenesis.	The combined environment changes receptor expression and threshold responses.
Atherogenic diet in vivo	Net bone loss or impaired interface strength can occur.	The model includes hormones, adiposity, inflammation and mechanical effects beyond oxLDL alone.

Net bone balance matters more than one cell pathway

A reduction in osteoclast activity is not automatically beneficial. Effective osseointegration requires removal of damaged bone followed by coupled formation. Both excessive resorption and suppressed remodelling can impair interface maturation.

Marginal bone loss has multiple determinants

Biofilm-associated inflammation, implant position, prosthetic design, tissue dimensions and loading must be assessed before assigning bone loss to dyslipidaemia.

Avoid a simple activation model: oxidized lipids can disturb osteoclast biology in different directions. The clinically relevant issue is loss of coordinated remodelling.

Evidence for dental implant outcomes

Experimental models consistently support biological concern, but direct human evidence remains limited. A high-fat diet increased implant loss and reduced bone-interface formation and strength in a mouse model. Reviews have therefore proposed dyslipidaemia as a possible modifying factor. Human studies remain heterogeneous and vulnerable to confounding.

Evidence layer	What it contributes	Main limitation
Cell studies	Identify oxidative, inflammatory and bone-cell pathways.	Artificial exposure conditions and uncertain clinical dose equivalence.
Animal studies	Examine interface formation and mechanical strength in a whole organism.	Dietary models combine several metabolic changes and do not reproduce human care.
Observational human studies	Explore lipid values, peri-implant parameters and failures in clinical populations.	Small samples, variable definitions and incomplete adjustment for diabetes, obesity, smoking and plaque.
Systematic reviews	Organize the available studies and identify a possible association.	Conclusions remain limited by the quality and comparability of included evidence.
Intervention studies	Could test whether lipid improvement changes implant outcomes.	Adequate prospective trials with implant endpoints are still lacking.

What the evidence does not establish

- A validated lipid threshold above which implant placement should be cancelled.
- A proven benefit of ordering lipid tests for every implant candidate.
- A demonstrated improvement in osseointegration after changing lipid therapy specifically for dental purposes.
- A routine role for oxLDL, LOX-1 or redox panels in chairside diagnosis.

Evidence-calibrated conclusion: dyslipidaemia is a plausible systemic modifier, but it should not be presented as a proven independent cause of implant failure.

Clinical assessment and medical coordination

A known lipid disorder should form part of the medical history. Additional testing is most useful when it answers a medical question or when the patient already requires cardiovascular or metabolic assessment. Dental teams should avoid creating an unvalidated universal screening panel.

Clinical situation	Reasonable action	Avoid
Known dyslipidaemia	Record the recent profile, treatment, adherence and cardiovascular risk context when relevant.	Relabelling control from one value without medical context.
Obesity, diabetes, hypertension or metabolic liver disease	Confirm whether medical follow-up and recent metabolic assessment exist.	Using a TG/HDL ratio as a stand-alone diagnosis.
Unexpected severe abnormality in available results	Advise timely medical review and coordinate elective invasive care according to overall stability.	Diagnosing or adjusting lipid treatment in the dental clinic.
Repeated implant or graft complications	Reassess local, mechanical, behavioural and systemic factors in parallel.	Assuming that dyslipidaemia explains failure after local causes are overlooked.
No known metabolic risk	Follow routine medical history and local implant assessment.	Ordering oxLDL or advanced lipid tests solely for implant eligibility.

TG/HDL ratio

The triglyceride-to-HDL-C ratio can correlate with insulin resistance in some populations, but cutoffs vary with sex, ethnicity, fasting conditions and study methods. It should not replace validated diabetes testing or cardiovascular risk assessment and has no established implant-specific threshold.

Local readiness remains essential

Periodontal stability, plaque control, implant position, tissue phenotype, graft needs and prosthetic cleansability continue to guide treatment. Systemic optimization complements these measures rather than replacing them.

Focused coordination: ask the treating clinician about current cardiovascular risk management and systemic stability when the answer will affect timing or procedure complexity.

Lipid-lowering therapy and supportive care

Statins reduce atherosclerotic cardiovascular risk and remain first-line LDL-lowering therapy in appropriate patients. Their effects on bone cells and implant integration are scientifically interesting, but human implant evidence does not justify prescribing, stopping or changing a statin for dental purposes.

Issue	Clinical interpretation	Safe action
Existing statin therapy	May indicate active cardiovascular risk management.	Record the medicine and do not discontinue it without medical direction.
Bone-related pleiotropic effects	Animal and local-delivery studies report osteogenic signals in some settings.	Do not promise improved osseointegration from systemic statin use.
Local statin delivery	Studied experimentally and in selected periodontal or regenerative protocols.	Not a routine standard for implant osseointegration.
Muscle symptoms or intolerance	Can affect function and adherence but requires medical assessment.	Refer concerns to the prescriber rather than advising interruption.
Nutraceuticals and supplements	Products vary in efficacy, safety and interaction potential.	Do not recommend them as substitutes for evidence-based lipid management.

Supportive peri-implant care

Recall intensity should reflect combined risk, including history of periodontitis, plaque, smoking, diabetes control, prosthetic access and prior peri-implant disease. Dyslipidaemia alone does not dictate a universal maintenance interval.

Longitudinal communication

Document medical changes and local peri-implant measures over time. Explain that better lipid control benefits cardiovascular health and may improve the broader biological environment, while direct implant benefit has not been quantified.

Medication principle: cardiovascular benefit drives lipid-lowering therapy. Dental hypotheses must not override the medical indication or treatment plan.

Clinical synthesis and selected references

Clinical synthesis

- Interpret LDL-C and other lipid measurements within cardiovascular risk management, not through an implant-specific cutoff.
- Distinguish native LDL-C concentration from oxidized LDL research assays.
- Treat oxLDL and LOX-1 as mechanistic concepts rather than routine dental biomarkers.
- Recognize that oxidized lipids can alter osteoblast and osteoclast pathways in context-dependent ways.
- Use animal evidence to explain plausibility while acknowledging limited human translation.
- Assess local, mechanical and behavioural causes whenever osseointegration or marginal bone is compromised.
- Do not prescribe, stop or modify lipid-lowering therapy for a dental hypothesis.
- Coordinate care when systemic instability or severe untreated metabolic disease may affect elective invasive treatment.

Selected references

1. Mach F, Baigent C, Catapano AL, et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias. *European Heart Journal*. 2020;41:111-188.
2. European Society of Cardiology and European Atherosclerosis Society. 2025 focused update of the 2019 ESC/EAS Guidelines for the management of dyslipidaemias. 2025.
3. Sun H, et al. Effects of hyperlipidemia on osseointegration of dental implants and its strategies. 2023. PMID: 37103284.
4. Gurgel BCV, et al. Are dyslipidemias a risk factor for peri-implant complications? A systematic review. 2025. PMID: 40375843.
5. Keuroghlian A, et al. The effects of hyperlipidemia on implant osseointegration in the mouse femur. *Journal of Oral Implantology*. 2015. PMID: 24295508.
6. Maziere C, et al. Oxidized LDL inhibits phosphate signalling and impairs osteoblast differentiation. 2010. PMID: 20667472.
7. Maziere C, et al. Oxidized LDL enhances RANKL expression and modifies osteoblast-osteoclast crosstalk. 2013. PMID: 23756197.
8. Dawodu D, et al. oxLDL inhibits differentiation and functional activity of osteoclasts. *Scientific Reports*. 2018;8. PMID: 30087473.
9. Ohgi K, et al. Toll-like receptor 2 activation primes and upregulates LOX-1 in bone marrow cells. *Lipids in Health and Disease*. 2018;17:132.
10. Sendyk DI, et al. The influence of statins on osseointegration: a systematic review of animal studies. 2016. PMID: 27611923.
11. Rallis K, et al. Statins and dental implant osseointegration: mechanisms and clinical evidence. 2025. PMID: 41873920.

Educational use and limitations

This material supports clinical reasoning and medical communication. It does not replace cardiovascular risk assessment, laboratory interpretation, lipid treatment or individualized implant planning.

Funding statement

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.