

Bone–Implant Interfaces for Photopolymer-Derived Devices: Surface Chemistry, Porosity, Mechanics and Biological Evidence

A multiscale framework for interfacial stability, tissue response and osseointegration claims

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Abstract

A bone-implant interface is formed by the final material surface, pore architecture, degradation behaviour, mechanics, fixation, surgical environment and host response. Photopolymer-derived devices span retained organic networks, particle-filled composites and debound or transformed ceramics; these states cannot share one biological inference. Surface chemistry can change during washing, post-cure, sterilisation, hydrolysis or sintering, while architecture controls fluid transport, cell access, mechanical stiffness and stress concentration. Early cytocompatibility therefore does not establish local tissue tolerance, bone formation or stable osseointegration. This review defines a non-compensatory qualification framework for photopolymer-derived bone interfaces. It freezes the material-transformation route and final device, measures chemistry and multiscale porosity, separates constituent properties from structural performance, and links micromotion and load transfer to staged biological evidence. In vitro assays, degradation studies, implantation histology, micro-computed tomography, mechanical fixation and clinically relevant comparators are assigned distinct evidentiary roles. Bone-implant contact is interpreted together with new-bone volume, tissue quality, inflammation, fibrous encapsulation and interfacial strength. The framework supports traceable engineering decisions without disclosing proprietary formulations or asserting that any generic printed material is suitable for implantation. Keywords: bone-implant interface; photopolymer-derived device; osseointegration; porosity; surface chemistry; micromotion; implantation; scaffold

1. Significance, scope and ownership

ISO 10993-6:2026 specifies current requirements for preclinical assessment of local effects after implantation and expressly separates local tissue response from mechanical or functional performance [1]. ISO 10993-1:2025 situates biological evidence in device risk management [2]. ISO 10993-14:2001 remains the published standard for identifying and quantifying ceramic degradation products [3].

1.1 Corpus boundary

ER-258 owns bone-interface surface state, pore architecture, interfacial mechanics, local implantation evidence and osseointegration claims for photopolymer-derived devices. ER-175 retains washing; ER-181 retains post-cure; ER-206 retains general micro-computed-tomography metrology; ER-221 to ER-225 retain general adhesion and surface-treatment science; ER-231 to ER-240 retain bulk structural reliability; ER-251 owns biological-evaluation planning; ER-252 owns cytotoxicity artefacts; ER-253 and ER-254 own chemical and toxicological assessment; ER-255 to ER-257 own skin, mucosal and blood contact; ER-259 owns antimicrobial-claim durability; and ER-260 owns patient-specific traceability.

2. Define the material family after processing

Classify the device as a retained organic photopolymer, hydrogel, polymer-ceramic composite, green body, debound ceramic, sintered ceramic or chemically transformed composite. Record which constituents remain at implantation and which are removed or converted. The phrase photopolymer-derived describes manufacturing history, not final chemistry. A sintered ceramic cannot inherit extractables assumptions from its sacrificial binder, while a retained network cannot inherit ceramic degradation behaviour from filler alone.

3. Freeze the final device and interface

Record resin or slurry lot, printer and wavelength, layer schedule, orientation, supports, wash, dry and cure, debinding or transformation profile, sintering, machining, surface treatment, sterilisation, packaging and ageing. Define the actual bone-contacting regions and any non-contacting backing or fixation components. Representative coupons must reproduce chemistry, roughness, pore accessibility and thermal history. Dense witness discs cannot represent a porous lattice when trapped residue, sintering gradients or local defects are material to the interface.

4. Characterise surface chemistry

Use surface-sensitive methods to identify elemental and functional composition, contamination, mineral phase, organic residue and treatment stability. Couple dry-state chemistry with hydrated behaviour, including ion release, dissolution, protein conditioning and pH change. Fagotto-Clavijo, Lodoso-Torrecilla, Diez-Escudero and Ginebra printed PEGDA/alpha-tricalcium-phosphate scaffolds that transformed in situ to calcium-deficient hydroxyapatite, increasing surface area and mechanical response [4]. This primary study shows that the implanted state can be chemically different from the as-printed state and must be verified after transformation.

5. Measure topography and multiscale porosity

Report external roughness, strut microtexture, pore-size distribution, interconnect diameter, closed porosity, tortuosity, directional anisotropy and accessible surface area. Compare design files with the manufactured structure using a validated metrology chain. Apparent porosity may be written as $\phi = 1 - \rho_{\text{app}} / \rho_{\text{solid}}$ where ϕ is apparent porosity, ρ_{app} is scaffold apparent density, and ρ_{solid} is the density of the solid phase used in the calculation. The result is method-dependent and does not establish pore interconnectivity or biological accessibility. Gatto, Cerqueni, Furlani, Riberti, Tognoli, Denti, Leonardi, Giuliani, Mattioli-Belmonte and Mengucci varied vat-photopolymerised trabecular geometry and related morphometry to compressive behaviour and MG-63 viability [6]. Their findings support joint interpretation of architecture, mechanics and cell response rather than a single nominal pore size.

6. Verify phase and transformation state

For ceramic and composite routes, quantify crystalline and amorphous phases, residual carbon or binder, grain size, shrinkage and spatial gradients. Thermal schedules can alter mineralogy and surface charge even when geometry is maintained. Ressler, Ohlsbom, Gobbo, Hannula, Keck, Swaminathan, Pakarinen, Mohammadi, Hyttinen, Massera, Schwentenwein, Frankberg, Levänen, Gebraad and Miettinen showed that porosity, sintering temperature, mineralogical phase and trace elements altered mechanical and osteogenic behaviour in ceramic vat-photopolymerised calcium-phosphate scaffolds [5]. The study demonstrates coupled processing effects, not a universal optimum.

7. Separate material and structural mechanics

Measure constituent modulus and strength where useful, then test the manufactured architecture under compression, tension, shear or torsion relevant to use. Report loading direction, platen friction, strain measurement, hydration, rate, cycles and failure mode. An apparent compressive modulus over a specified linear interval can be expressed as $E_{app} = \Delta \sigma / \Delta \epsilon$ where E_{app} is apparent structural modulus, $\Delta \sigma$ is the stress increment, and $\Delta \epsilon$ is the corresponding strain increment over the declared fitting interval. It is an architecture-dependent property, not the intrinsic modulus of the solid phase.

8. Control fixation and interfacial micromotion

Define press fit, screws, adhesive, cement, interference, defect geometry and load path. Measure initial stability and micromotion under representative multiaxial loading. Excess motion can favour fibrous tissue even when the material is intrinsically osteoconductive. Separate fixation success from tissue integration. A mechanically stable interface can lack new bone, while apparent bone contact can coexist with inadequate fatigue or pull-out resistance.

9. Characterise degradation and release

For retained polymers, evaluate chemical degradation and leachables in the final cured state. For ceramics, identify dissolution products, particulate release and phase-dependent transformation. For composites, treat both pathways and their interaction. Measure mass, molecular or phase change, ion release, pH, morphology and mechanical retention over relevant time. Accelerated conditions require a demonstrated relation to physiological mechanisms; an aggressive medium can create products not formed clinically.

10. Design in vitro biological evidence by mechanism

Use appropriate primary cells or justified models for adhesion, viability, morphology, osteogenic differentiation and inflammatory signalling. Distinguish direct contact, extract exposure and conditioned-medium designs. Confirm assay compatibility with coloured, porous or adsorptive specimens. The transformed-composite study reported cytocompatibility [4], while the ceramic-scaffold study linked phase and surface differences to attachment and osteogenic outcomes [5]. These examples support mechanistic screening but do not replace implantation evidence.

11. Evaluate immune and inflammatory response

Characterise macrophage and foreign-body response where relevant, including temporal phenotype, giant cells, necrosis and fibrous capsule. Avoid treating one cytokine ratio as a complete immune conclusion. Inflammation can reflect chemistry, wear particles, pore geometry, instability or infection. Study design should preserve enough information to discriminate these causes.

12. Select implantation models by question

Use subcutaneous or intramuscular implantation for local tissue tolerance when justified, and orthotopic bone models for bone formation, load transfer and integration. Match species, anatomical site, defect criticality, fixation, load and observation times to the intended claim.

ISO 10993-6:2026 covers local implantation effects but does not determine functional mechanical performance [1]. A single model should not be stretched beyond the question it can answer.

13. Use comparators and reference controls

Include a reference material with established tissue response and, when appropriate, a clinically relevant device or scaffold comparator. Comparators should match geometry and fixation sufficiently to isolate the factor under study. Empty-defect controls measure spontaneous healing but do not replace a material comparator. Historical controls are acceptable only when methods, site, scoring and laboratory conditions remain comparable.

14. Quantify bone formation in three dimensions

Use calibrated micro-computed tomography to measure new-bone volume, distribution, mineral density and pore occupancy, while controlling beam hardening and segmentation bias. Register longitudinal scans when repeated imaging is used. Two-dimensional histology remains necessary for cell and tissue identity. Three-dimensional mineral volume alone cannot distinguish viable lamellar bone from residual radiopaque scaffold or mineralised debris. Bai, Gui, Zhang, He, Peng, Zhou, Yang, Chen and Lai combined DLP scaffold architecture with in vitro and preclinical in vivo observations in a bio-root model [7]; the study illustrates staged evidence but does not validate other materials or indications.

15. Score histology transparently

Predefine regions of interest, section orientation, stains, pathologist blinding and scoring rules. Report inflammation, necrosis, fibrosis, vascularisation, new bone, marrow, resorption and material fragmentation. Bone-implant contact can be represented as $BIC = 100 L_{bone} / L_{eligible}$ where BIC is percentage bone-implant contact, L_{bone} is interface length directly contacting bone, and $L_{eligible}$ is the total evaluable interface length. Exclusions, section selection and observer uncertainty must be reported.

16. Couple biological and mechanical outcomes

Measure push-out, pull-out, torque or interfacial shear when the clinical claim includes fixation. Preserve the failure location: cohesive bone failure, interfacial separation and scaffold fracture have different meanings. A high bone volume with weak fixation may indicate poor interface quality or architecture. Conversely, high mechanical retention from geometric interlock should not be labelled osseointegration without tissue evidence.

17. Address sterilisation and ageing

Verify chemistry, phase, porosity, dimensions, mechanical properties and biological response after the final sterilisation and worst-case ageing states. Residual sterilant, oxidation, chain scission, additional crosslinking or ceramic-surface change can alter the interface. Package integrity and particulate generation during transport are part of the delivered state. Reprocessing and reuse require separate evidence if they are within intended use.

18. Apply non-compensatory gates

Gate	Required evidence	Blocking failure
Material identity	Final polymer, composite or ceramic state	Evidence is assigned to the wrong material
Surface	Chemistry, phase, contamination and hydration	Interface mechanism is undefined
Architecture	Manufactured open porosity and defects	Transport and mechanics are unbounded
Mechanics	Structural response and initial fixation	Biological result lacks load context
Degradation	Products, kinetics and mechanical retention	Long-term state is unknown
Local response	Controlled implantation histology	Tissue tolerance is unresolved
Bone evidence	Spatial bone formation and contact	Osseointegration claim is unsupported
Fixation	Interfacial mechanical outcome and failure mode	Functional integration is unproven

Success at one gate cannot compensate for failure at another. Unresolved gates require added characterisation, redesign or a narrower claim.

19. Integrate evidence by time and scale

Build a matrix linking process state, surface chemistry, architecture, mechanics, degradation, inflammation, bone formation and fixation at each time point. Preserve adverse local regions instead of averaging the whole implant. Interpret early cell data as mechanism, implantation as local response and mechanical testing as function. Concordance strengthens a claim; discordance should generate a discriminating next experiment.

20. Control changes

Trigger reassessment after changes in resin or powder, supplier, particle distribution, exposure dose, wash, cure, transformation, sintering, machining, surface treatment, sterilisation, geometry, pore network, fixation, packaging or shelf life. A geometrically minor change can alter local stress or pore accessibility. Equivalence requires evidence on sensitive attributes, not only nominal composition. Maintain traceability from digital design through the implanted lot.

21. Minimum reporting set

Report intended interface and claim; final material family; complete manufacturing and transformation route; surface chemistry and phase; topography and accessible porosity; dimensional fidelity; hydrated mechanics; degradation and release; sterilisation and ageing; in vitro methods; animal model and ethics; controls; imaging and histology; bone-contact and fixation outcomes; statistics; adverse regions; deviations; uncertainty and change triggers.

22. Knowledge boundary

The standards define risk-based implantation and ceramic-degradation frameworks [1-3]. The experimental studies show that transformation, porosity, phase, geometry and surface state can materially affect mechanics and osteogenic observations [4-7]. None establishes safety, efficacy or osseointegration for an unspecified photopolymer-derived device.

23. Limitations

References [1-3] were verified against current official ISO records; references [4-7] were verified at detailed primary-record depth. Controlled standards and jurisdictional requirements govern implementation. This review does not prescribe clinical indications, animal numbers, universal pore sizes or product-specific acceptance criteria.

24. Conclusions

A defensible bone-interface claim requires the final transformed material, manufactured architecture, mechanical environment and staged tissue response to agree. Surface chemistry and porosity must be measured rather than inferred from design; cytocompatibility is an early screen rather than osseointegration; and bone contact must be interpreted with inflammation, three-dimensional bone formation and interfacial mechanics. Non-compensatory gates prevent attractive results at one scale from hiding failure at another.

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