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Three Cells, One Surface: A 2026 Update on the Divergent Cellular Mechanisms Driving Next-Generation Dental Implant Coatings

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Abstract
Dental implant surface engineering has moved decisively, over 2025–2026, from roughness-based osseointegration strategies toward multifunctional nanoengineered coatings that simultaneously target three distinct cellular audiences: osteoblasts, macrophages, and oral bacteria. This narrative review compares the cellular and molecular mechanisms by which three representative classes of contemporary surface modification act — (a) superhydrophilic nanotopographic coatings (e.g., anatase-TiO2 and nanocrystalline hydroxyapatite films) that act principally through polar surface free energy, protein conditioning, and integrin-mediated osteoblast adhesion; (b) osteoimmunomodulatory microarc oxidation and related nanotopographies that steer macrophage M1-to-M2 polarization via the PI3K-Akt axis and iNOS/Arg-1 signaling; and (c) silver-nanoparticle antibacterial coatings that act through ionic Ag+ release, reactive oxygen species (ROS) generation, and direct bacterial membrane and DNA disruption, at the cost of a narrow therapeutic window against host osteoblasts. Rather than converging on a single pathway, these strategies engage fundamentally different receptors, signaling cascades, and cellular targets, which explains both their complementary clinical promise and the persistent difficulty of combining them without mechanistic trade-offs. Implications for peri-implantitis prevention and for future multifunctional coating design are discussed.

Key Word: *Dental implant surface, osseointegration, osteoblast, macrophage polarization, silver nanoparticles, osteoimmunomodulation, peri-implantitis*

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I. Introduction

The clinical success of endosseous titanium dental implants depends on osseointegration, the direct structural and functional connection between living bone and the implant surface[1]. For decades, surface engineering pursued this goal principally through roughness: sandblasted, large-grit, acid-etched (SLA) surfaces increased bone-to-implant contact relative to machined surfaces by providing mechanical interlocking and a larger area for cell attachment[1]. Over the past two years, however, the field's center of gravity has shifted. The most consequential advances no longer concern how rough a surface is, but which cellular populations it is designed to instruct, and through which specific molecular pathway.

Three cellular audiences now dominate the surface-engineering literature. The first is the *osteoblast*, whose adhesion, proliferation, and differentiation remain the classic endpoint of osseointegration research. The second is the *macrophage*, increasingly recognized as a gatekeeper of the peri-implant healing cascade through its M1 (pro-inflammatory) and M2 (pro-reparative) polarization states[2,3]. The third is the *oral bacterium*, the target of antibacterial nanocoatings designed to pre-empt peri-implantitis, the leading cause of late implant failure[4]. This review compares, mechanism by mechanism, how contemporary coatings speak to each of these three cell types, and argues that the differences between these mechanisms — not merely their individual efficacy — are what should guide the design of the next generation of multifunctional implant surfaces. A complementary sixteen-year chronological synthesis of the antimicrobial and osteoinductive nanocoating literature (2010–2026), tracing the field's progression from single-mechanism nanoparticle coatings toward the multifunctional, stimuli-responsive systems discussed here, is provided by Bastos et al.[5], whose historical framing usefully situates the mechanistic comparison developed in the present review.

II. Osteoblast-Directed Mechanisms: Surface Free Energy, Protein Conditioning, and Integrin Signaling

The most rigorously characterized 2026 addition to this literature is the superhydrophilic anatase-TiO2 coating deposited onto clinically relevant SLA titanium by reactive pulsed-DC magnetron sputtering[6]. Unlike conventional SLA, which remains hydrophobic (water contact angle ≈ 115°) because of air entrapment and adsorbed atmospheric hydrocarbons within its micro-pitted topography, the anatase-coated variant (SLA-anatase) achieved a near-zero contact angle (3.8°) without any post-deposition activation step[6]. Mechanistically, this transition is not attributable to roughness — Sa values for SLA and SLA-anatase differed only marginally — but to a substantial increase in the *polar* (electron-donor/base) fraction of surface free energy (SFE), which lowers the barrier to interfacial hydration and calcium-phosphate nucleation[6]. In simulated body fluid, only the superhydrophilic surface supported homogeneous hydroxyapatite formation, and in vitro osteoblasts (SAOS-2 and primary human osteoblasts alike) showed accelerated early adhesion, greater spreading with prominent filopodial extension, higher metabolic activity, and significantly elevated alkaline phosphatase activity and RUNX2/osterix gene expression relative to uncoated SLA[6]. The proposed causal chain is therefore: increased polar SFE → accelerated protein adsorption and hydration layer formation → integrin-mediated focal adhesion → RUNX2/osterix-driven osteogenic transcription — a pathway that is fundamentally physicochemical and receptor-mediated rather than immunological or antimicrobial.

An earlier but mechanistically convergent strategy achieves a similar polar-SFE effect through nanocrystalline hydroxyapatite (nano-HA) blasting, in which a roughly 20-nanometer-thick layer of pure HA nanocrystals is deposited without altering the underlying SLA microstructure[1]. Because HA is the principal inorganic constituent of bone, this coating is thought to act through a biomimetic recognition mechanism in addition to a purely energetic one: osteoblasts encounter a chemically bone-like substrate rather than an inert metal oxide, which is proposed to lower the threshold for osteoblastic lineage commitment. Whether the dominant driver in any given nanotopographic coating is the physical SFE effect, the chemical HA-recognition effect, or an inseparable combination of both remains an open mechanistic question that the 2026 anatase data help to partially disentangle by isolating a TiO2 (non-HA) system that reproduces much of the same biological outcome through SFE alone[6].

III. Macrophage-Directed Mechanisms: Osteoimmunomodulation via M1/M2 Polarization

A second, mechanistically distinct family of 2025–2026 coatings targets not the osteoblast directly but the macrophage, reflecting the broader recognition that titanium is an immunomodulatory biomaterial whose implantation triggers a transient inflammatory response that shapes downstream bone formation[1]. Macrophages recruited to the implant interface polarize along a spectrum between a classically activated, pro-inflammatory M1 phenotype (marked by inducible nitric oxide synthase, iNOS, and secretion of TNF-α and IL-1β) and an

alternatively activated, pro-reparative M2 phenotype (marked by CD206 and arginase-1, Arg-1, and associated with angiogenic and osteogenic cytokine release)[2]. A transient early M1 response supports debris clearance and recruitment of mesenchymal stem cells, but a prolonged M1 state is associated with fibrotic encapsulation and impaired bone formation, whereas a timely M1-to-M2 transition is associated with favorable osseointegration.

A 2026 study modified titanium with a "cortex-like" microarc oxidation (MAO) coating engineered to mimic the hierarchical micro/nano architecture of cortical bone, and found that this topography — independent of any drug loading or ionic doping — promoted macrophage adhesion, proliferation, and an elongated morphology, and shifted the RAW264.7 macrophage population away from the iNOS+ M1 phenotype and toward the Arg-1+ M2 phenotype[3]. Pathway analysis implicated the PI3K-Akt signaling cascade as the mechanistic link between topography sensing and polarization outcome, distinguishing this mechanism sharply from the SFE/integrin pathway described in Section II: here the relevant surface property is architectural mimicry of a specific tissue geometry, transduced through an intracellular kinase cascade in an immune cell, rather than a purely energetic effect on protein adsorption acting on a bone-forming cell.

This immunomodulatory route carries a strategic implication that the osteoblast-only literature does not fully capture: a coating can, in principle, accelerate osseointegration without directly touching an osteoblast at all, by instead shortening the pro-inflammatory window and hastening the arrival of the pro-reparative, cytokine-rich M2 microenvironment in which osteoblasts subsequently operate[2,3]. This positions macrophage-directed coatings as an upstream intervention relative to the osteoblast-directed coatings discussed above, rather than a competing alternative to them.

IV. Bacteria-Directed Mechanisms: Silver Nanoparticle Antibacterial Action and Its Cytotoxic Ceiling

The third and most extensively documented mechanism operates on an entirely different cellular target — the bacterium — and is dominated by silver nanoparticle (AgNP) coatings, motivated by the role of biofilm colonization in peri-implant mucositis and peri-implantitis[4]. The antibacterial mechanism is multimodal and, unlike the osteoblast and macrophage pathways above, is not receptor-mediated in the classical sense but proceeds through direct physicochemical damage: released Ag+ ions bind sulfur- and phosphorus-containing groups in bacterial membrane proteins and DNA, disrupting membrane permeability and inhibiting ATP synthesis and DNA replication, while electron transfer between silver and the underlying substrate generates bursts of reactive oxygen species that cause additional intracellular oxidative damage[7]. This mechanism is bactericidal against both Gram-positive and Gram-negative oral pathogens and, critically, does not appear to select for classical antibiotic-resistance mechanisms in the same way that pharmacological antibiotics do[7].

However, this same ROS/ion-release mechanism is not cell-type-selective at the physicochemical level: at sufficient local concentration, Ag+ and AgNP-induced ROS are equally capable of damaging osteoblast membranes, mitochondria, and DNA, producing a documented narrow therapeutic window in which antibacterial efficacy and host-cell cytotoxicity trade off directly against one another[4]. A systematic narrative review of 45 controlled studies found that the determining variable was not the presence of silver per se but its *release kinetics*: AgNPs embedded within nanotubular titanium structures or polymeric linker layers (e.g., polydopamine, chitosan) released silver ions more slowly and sustained antibacterial activity for over two weeks with substantially reduced cytotoxicity, whereas surfaces with rapid, unbuffered ion release achieved strong short-term antibacterial effect at the cost of measurable osteoblast toxicity[4]. This kinetic dependency has no direct analogue in the SFE-driven osteoblast mechanism or the topography-driven macrophage mechanism described above, both of which are steady-state surface properties rather than time-dependent release phenomena — a distinction with direct implications for how each mechanism should be evaluated preclinically and dosed clinically.

This same release-kinetics problem is the organizing thread of the broader sixteen-year narrative traced by Bastos et al.[5], who show that the field's response to the antibacterial—cytotoxicity trade-off evolved in successive waves: chitosan- and polydopamine-mediated chelation to slow silver-ion release (2013–2019), pH-responsive multilayers that confine antimicrobial release to the acidic microenvironment of an active infection rather than releasing continuously (first demonstrated in 2019 and matured by 2025), and, most recently, photothermally activated gold-nanostar coatings that place antibacterial triggering entirely under clinician control via near-infrared irradiation[5]. Read alongside the mechanistic account given here, this trajectory indicates that the field has not resolved the ionic/ROS cytotoxicity ceiling described above so much as engineered around it, by converting a constant-exposure mechanism into an intermittent or on-demand one.

V. Comparative Synthesis: Three Mechanisms, Three Target Cells, Three Time Signatures

Placed side by side, these three mechanistic families diverge along at least three axes. First, the *target cell* differs categorically: osteoblasts for SFE/nanotopography coatings, macrophages for immunomodulatory microarc/topographic coatings, and bacteria (with osteoblasts as an unintended bystander target) for AgNP

coatings. Second, the *proximate molecular event* differs: integrin engagement and RUNX2/osterix transcription in the osteoblast pathway; PI3K-Akt-mediated polarization switching in the macrophage pathway; and direct ionic/oxidative membrane and DNA damage in the antibacterial pathway. Third, the *temporal signature* differs: SFE effects are essentially instantaneous surface properties present from the moment of implantation; macrophage polarization unfolds over the first days to weeks of healing; and AgNP antibacterial action is release-rate-dependent and must be sustained, but not indefinitely, to avoid cumulative host-cell toxicity.

This divergence carries a direct design implication: because the three mechanisms act on different cells through different pathways at different times, combining them in a single multifunctional coating is not simply additive. A coating optimized to maximize polar SFE for osteoblast adhesion, for instance, does not thereby confer any macrophage-polarizing or antibacterial property, and a coating engineered for sustained low-dose Ag+ release must be designed so that its ionic microenvironment does not interfere with the superhydrophilic, protein-conditioning surface chemistry on which the osteoblast pathway depends[2]. The most promising 2025–2026 designs are therefore explicitly layered or spatially segregated — for example, a topographically engineered base layer for macrophage polarization combined with a controlled-release reservoir for antibacterial ions — rather than a single homogeneous chemical modification expected to satisfy all three cellular audiences simultaneously[2,3].

VI. Discussion and Limitations

Several limitations qualify these comparisons. Most of the mechanistic evidence reviewed here derives from in vitro cell-culture systems (SAOS-2, primary human osteoblasts, RAW264.7 macrophages) or short-duration animal models, and the translation of isolated single-cell-type mechanisms to the multicellular, dynamically remodeling peri-implant environment in humans remains incompletely validated[6]. The anatase-coating mechanism, while mechanistically well characterized in vitro, still awaits in vivo confirmation of early bone-to-implant contact and long-term coating stability under masticatory load[6]. Similarly, the clinical evidence base for AgNP-coated implants remains limited to short-duration studies (maximum documented antibacterial monitoring of approximately 15 days), and no AgNP dental implant surface has yet been validated in controlled human clinical trials[4]. The macrophage-polarization literature, while mechanistically the most novel of the three families discussed, is also the most recent and carries the least depth of independent replication.

A further limitation is definitional: this review has organized coatings by their dominant reported mechanism, but many real-world experimental surfaces plausibly act on more than one cell type simultaneously in ways their original studies were not designed to detect (for example, an anatase or MAO topography could conceivably also influence macrophage behavior, or an AgNP coating could alter osteoblast gene expression sublethally). Future mechanistic studies that assay osteoblast, macrophage, and bacterial responses concurrently on the same experimental surface would meaningfully strengthen the comparative framework proposed here.

VII. Conclusion

The most recent generation of dental implant surface coatings should not be understood as competing refinements of a single osseointegration mechanism, but as three mechanistically distinct interventions addressing three different cellular populations at the implant interface. Superhydrophilic nanotopographic coatings act on osteoblasts through polar surface free energy and integrin-mediated signaling; osteoimmunomodulatory microarc and nanotopographic coatings act on macrophages through PI3K-Akt-mediated M1-to-M2 polarization; and silver nanoparticle coatings act on bacteria through ionic and oxidative membrane and DNA damage, constrained by a cytotoxicity ceiling shared with host osteoblasts. Recognizing these mechanisms as parallel rather than convergent pathways is essential both for interpreting the 2025–2026 literature accurately and for designing the layered, multifunctional implant surfaces most likely to translate successfully into long-term clinical reductions in peri-implantitis and implant failure.

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