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Therapeutic

Aesthetic Breast Surgery

Supplement Article

From Bench to Bedside: Clinical Implications of a 4-Micron Surface-Modulated Immune Response in the Era of Breast Tissue Preservation

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Abstract

Breast implant surface topography plays a critical role in modulating host immune responses and long-term clinical outcomes. Although traditional classifications dichotomize implant surfaces as smooth or textured, emerging evidence demonstrates that specific architectural parameters differentially regulate macrophage polarization, T cell recruitment, and fibrotic capsule formation. This narrative review synthesizes preclinical, translational, and clinical evidence evaluating immune modulation associated with a specific 4 μ m surface architecture and discusses its implications for capsular contracture, chronic inflammation, biointegration, and the corresponding consequences for conservative surgical techniques. Across animal models, capsule histology, proteomic analyses, and gene expression studies, this specific 4 μ m surface consistently demonstrates reduced proinflammatory macrophage infiltration, enhanced FoxP3 + regulatory T cell recruitment, and decreased profibrotic signaling with thinner yet elastic structured capsules compared with both conventional smooth and high roughness surfaces, favoring immune regulation rather than chronic inflammation. Capsule behavior is governed not by thickness alone but by structural organization, elasticity, and immune profile. Clinically, these findings translate into low rates of capsular contracture without increased risks of implant instability, when appropriately managed. Collectively, these converging data support a broader conceptual framework in which foreign body response is a biologically modifiable process. Modulating this response through surface topography enables the development of an optimized implant-tissue interface that promotes physiologic healing, minimizes adverse outcomes, and supports natural breast biomechanics. Within this framework, coupling this specific architecture with breast tissue-preserving techniques, this biologic profile provides a foundation for stable, low-inflammatory implant integration without reliance on complex maneuvers, additional structural support or excessive fibrotic adhesion.

Level of Evidence: 5 (Therapeutic)

A growing body of research examining host immune responses to breast implant surfaces seeks to close critical gaps in understanding how mechanical properties, particulate shedding, bacterial adhesion,

and other device-related factors influence clinical outcomes. Increasing attention has focused on the interface between implant surface topography and the local immune environment, particularly as it

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relates to capsular contracture, breast implant-associated anaplastic large cell lymphoma (BIA-ALCL), and the variety of Systematic Symptoms Associated with Breast Implants (SSBI).¹

Recent studies have demonstrated an association between increased implant surface roughness and surface area and increased levels of bacterial adhesion and biofilm formation.^{2,3} Bacterial attachment and biofilm formation have been implicated in both BIA-ALCL⁴ and capsular contracture, potentially through chronic antigenic stimulation and sustained inflammatory signaling.⁵ In parallel, surface particulate shedding has been postulated as an additional pro-inflammatory stimulus, contributing to immune activation within the periprosthetic microenvironment and possibly playing a role in lymphoproliferative transformation.⁶

With limited consensus on the underlying etiology of these entities, there is an even greater need to understand the breast implant surface and its interaction with tissue *in vivo*, from *de novo* formation of the periprosthetic scar tissue to long-term embodiment. Traditional implant surfaces have historically been classified in broad terms as either smooth ($\sim 0\ \mu\text{m}$) or textured (macrotextured, $>50\ \mu\text{m}$). The current discourse continues to rely on this coarse distinction, categorizing surfaces from an engineering perspective, primarily by the presence or absence of surface modifications, without examining its relationship to a biologically complex continuum.⁷ Emerging evidence indicates that host immune responses are subject to more nuanced modulation.⁸ A specific surface architecture characterized by an average roughness of approximately $4\ \mu\text{m}$ has demonstrated translational promise, appearing capable of fine-tuning the fibrotic and inflammatory reactions elicited by the implant, distinct from both conventional smooth and textured designs.⁹

To further clarify these relationships, we conducted a targeted narrative review of preclinical animal models, human capsular analyses, and applied immunologic studies evaluating this specific surface architecture and its host response. The objective was to synthesize emerging links between defined topographic parameters, immune pathway activation, and downstream clinical implications. This review was conducted in accordance with the principles of the Declaration of Helsinki, and included studies published between January 2017 and May 2026. Emphasis was placed on combining rigorous studies reporting macroscopic findings such as capsule structure, immune cell population profiling, proteomic and gene expression. The 2021 Nature Biomedical Engineering study by Doloff et al⁹ served as a keystone investigation for mechanistic interpretation and contextual integration of these findings.

Surface Roughness and Architecture

Precisely defined microtopographical features in silicone substrates were pioneered by Prof. Bayat et al,¹⁰ who demonstrated that implant surface architecture exerts measurable and biologically significant effects on cellular behavior by modulating fibroblast adhesion, alignment, and cytoskeletal organization, thereby driving phenotypic and functional cellular responses. Furthermore, the ability of specific surface geometries to induce fibroblast remodeling and influence the foreign body response provides a clear mechanistic basis for surface-driven modulation of capsular outcomes. Collectively, this work establishes the scientific foundation underpinning the rational design and engineering of implant surfaces, with direct implications for enhancing biocompatibility and optimizing clinical performance.

Current classification standards for breast implant surfaces vary worldwide and are primarily regulatory and technical rather than biologically driven. Nonetheless, surface topographic complexity plays a decisive role in protein adsorption kinetics, macrophage polarization, fibroblast activation, and collagen organization. These early molecular and cellular events ultimately determine the quality and magnitude of the foreign body response (FBR).⁸ This underscores the need to

expand surface characterization parameters beyond conventional categorical distinctions in order to more accurately reflect biological behavior at the tissue–implant interface.

The SmoothSilk implant surface (Motiva Implants, Establishment Labs, Alajuela, Costa Rica) is produced via a proprietary mold-inversion manufacturing process that avoids residual particulate contamination and mitigates the shedding of silicone particles from the implant shell,¹¹⁻¹³ addressing a known trigger for exhaustive phagocytosis and inflammation.

Surface roughness can be quantified using profilometry, a technique designed to evaluate the topography of biomaterials.¹² In non-contact (optical) profilometry, 3-dimensional surface scans are generated to quantify microtopographic attributes. Roughness is calculated as the deviation of surface vectors relative to an ideal reference plane. Larger deviations denote rougher surfaces, whereas minimal deviations indicate smoother profiles.^{12,14} The aforementioned implant surface has been consistently characterized as having an average roughness of approximately $4\ \mu\text{m}$, among other features of its architecture.⁹ Importantly, despite its hierarchical microtopographic features, Atlan et al reported its effective surface area to be comparable to that of traditional smooth, distinguishing it from textured devices that significantly increase surface area.¹¹

Conventional smooth surfaces, when examined using differential interference contrast microscopy and scanning electron microscopy, were characterized by Barr as exhibiting directional ridges approximately $5\ \mu\text{m}$ in width, indicating that they are not as smooth as historically assumed (Figure 1).¹⁰ Consequently, a more appropriate description for these surfaces, which possess surface roughness values close to $\sim 0\ \mu\text{m}$, is to refer to them as nonintentionally modified rather than smooth.

Beyond average roughness, skewness provides an additional statistical descriptor of surface architecture. Skewness reflects the degree of symmetry of surface height distribution relative to the mean plane. A positively skewed surface demonstrates a predominance of peaks over valleys, whereas a negatively skewed surface demonstrates a predominance of valleys. A skewness value of zero indicates a symmetric distribution.⁹

Using optical profilometry (Figure 2A), Doloff et al⁹ calculated average roughness and skewness values across 5 different breast implant surfaces (with average roughness of ~ 0 , 4, 15, 50, and $83\ \mu\text{m}$). The $4\ \mu\text{m}$ surface demonstrated the highest positive skewness (more peaks than valleys) among all tested surfaces, with significantly greater positive skewness compared to the 15, 50, and $83\ \mu\text{m}$ groups. In contrast, the greatest negative skewness (more valleys than peaks) was observed with the $83\ \mu\text{m}$ surface, reflecting a valley-dominant architecture. The skewness values of the 50, 28, 15, and $\sim 0\ \mu\text{m}$ surfaces were closer to 0 (Figure 2B).

Additionally, the $4\ \mu\text{m}$ surface exhibited the highest number of contact points per unit surface area among the tested implant surfaces. This increased peak density may influence cellular anchorage and immune cell interaction at the interface.

Immune Response to Biomaterial Implantation

The FBR to biomaterial implantation is regulated by innate and adaptive immune cells, including neutrophils, macrophages, and lymphocytes. Immediately following implantation, serum proteins adsorb to the biomaterial surface, initiating neutrophil recruitment within minutes to hours.^{15,16} Neutrophils drive the early proinflammatory phase promoting monocyte recruitment and differentiation into pro-inflammatory (M1-like) macrophages. These macrophages secrete cytokines such

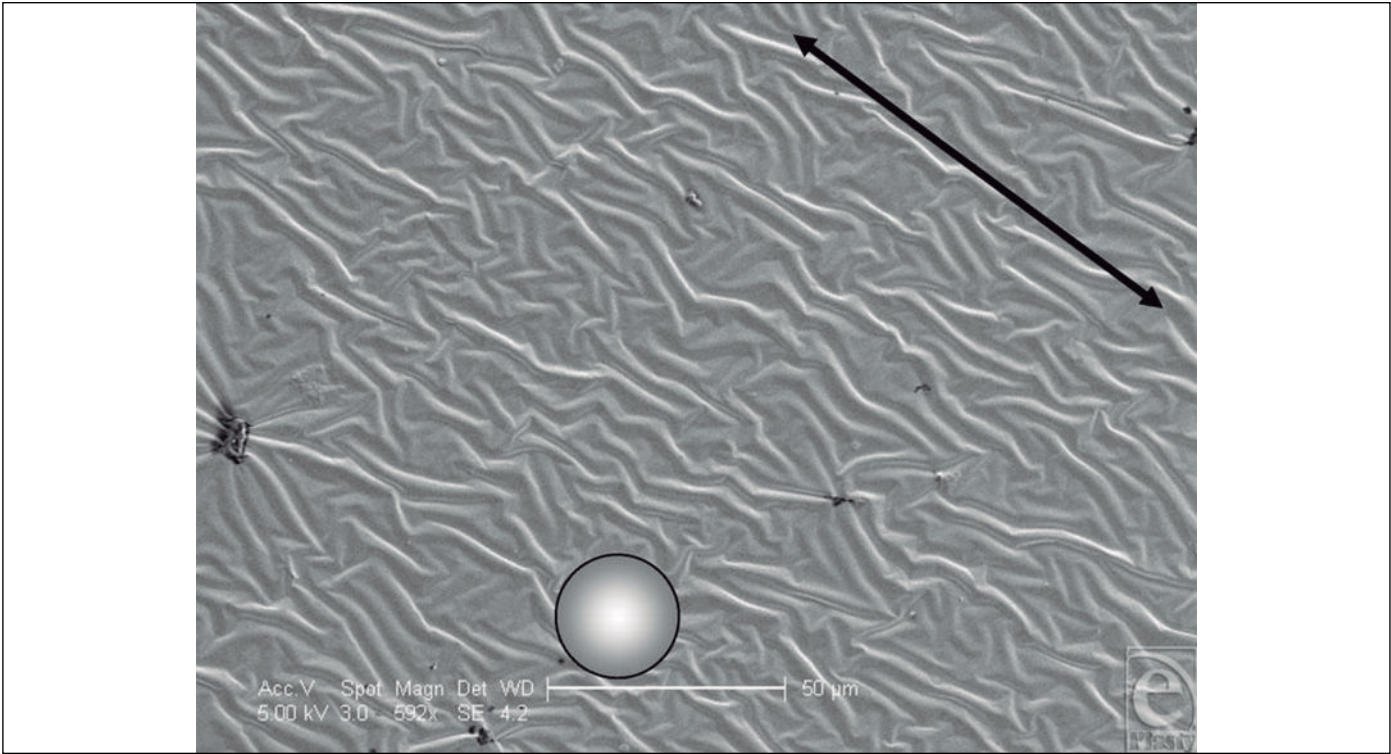


Figure 1. Scanning electron micrograph of a smooth Allergan breast implant surface, shown at 592x magnification with a 50 µm scale bar. The image depicts representative fibroblast dimensions and highlights the predominant orientation of surface ridges using an arrow. These ridges demonstrate a consistent directional topography, likely arising during the implant drying and manufacturing process. The scale of the ridged features is shown relative to an average fibroblast, illustrating potential interactions between surface architecture and cellular contact. Reprinted from Barr et al.

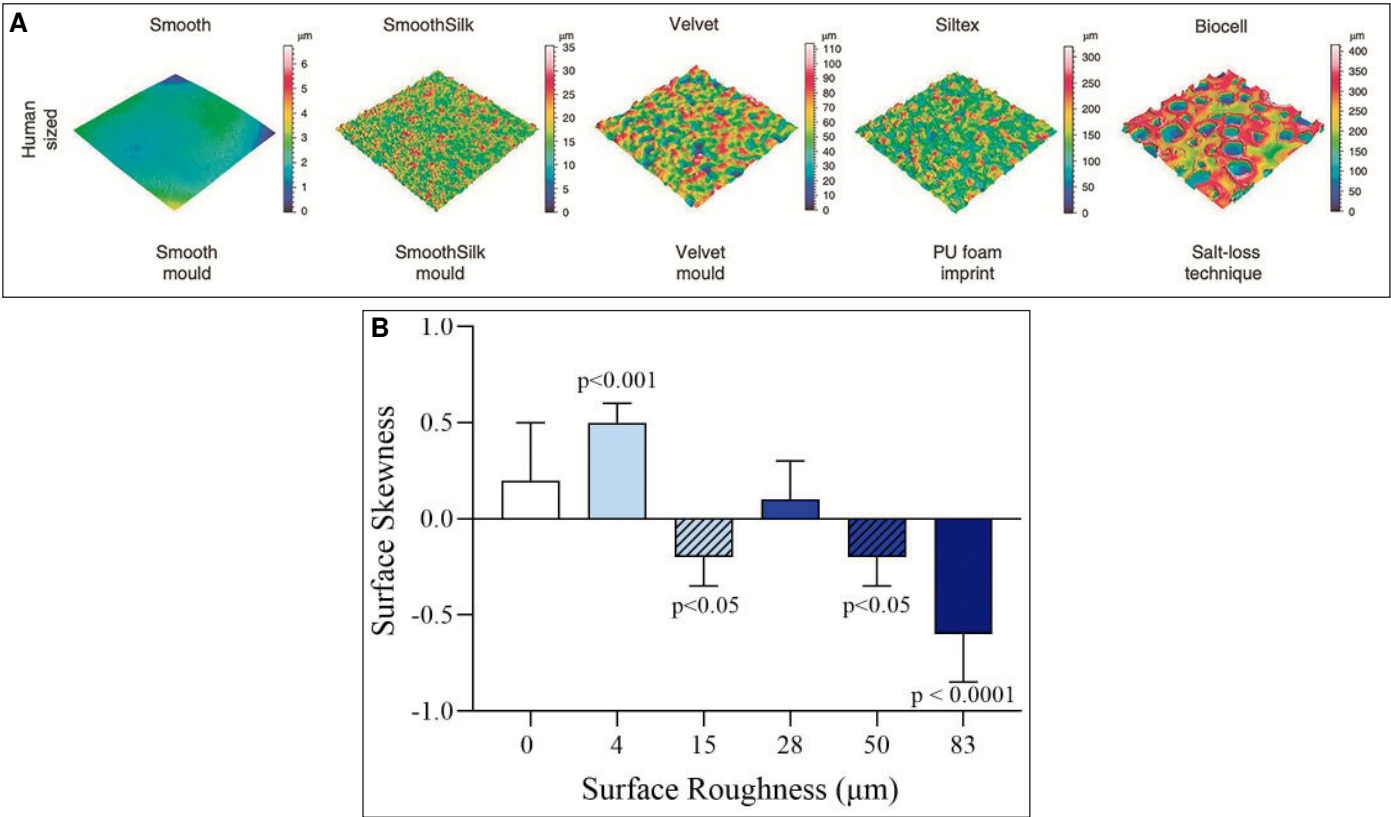


Figure 2. Surface topography and skewness analysis of breast implant surfaces. (A) Representative 3-dimensional surface topography images generated by optical profilometry for breast implant surfaces with average roughness values of approximately 0, 4, 15, 50, and 83 µm. Height scales represent relative surface elevations. (B) Quantification of surface skewness as a function of average surface roughness. Data are presented as mean ± SEM. SEM, standard error of the mean. Adapted from Doloff et al, 2021, with permission.

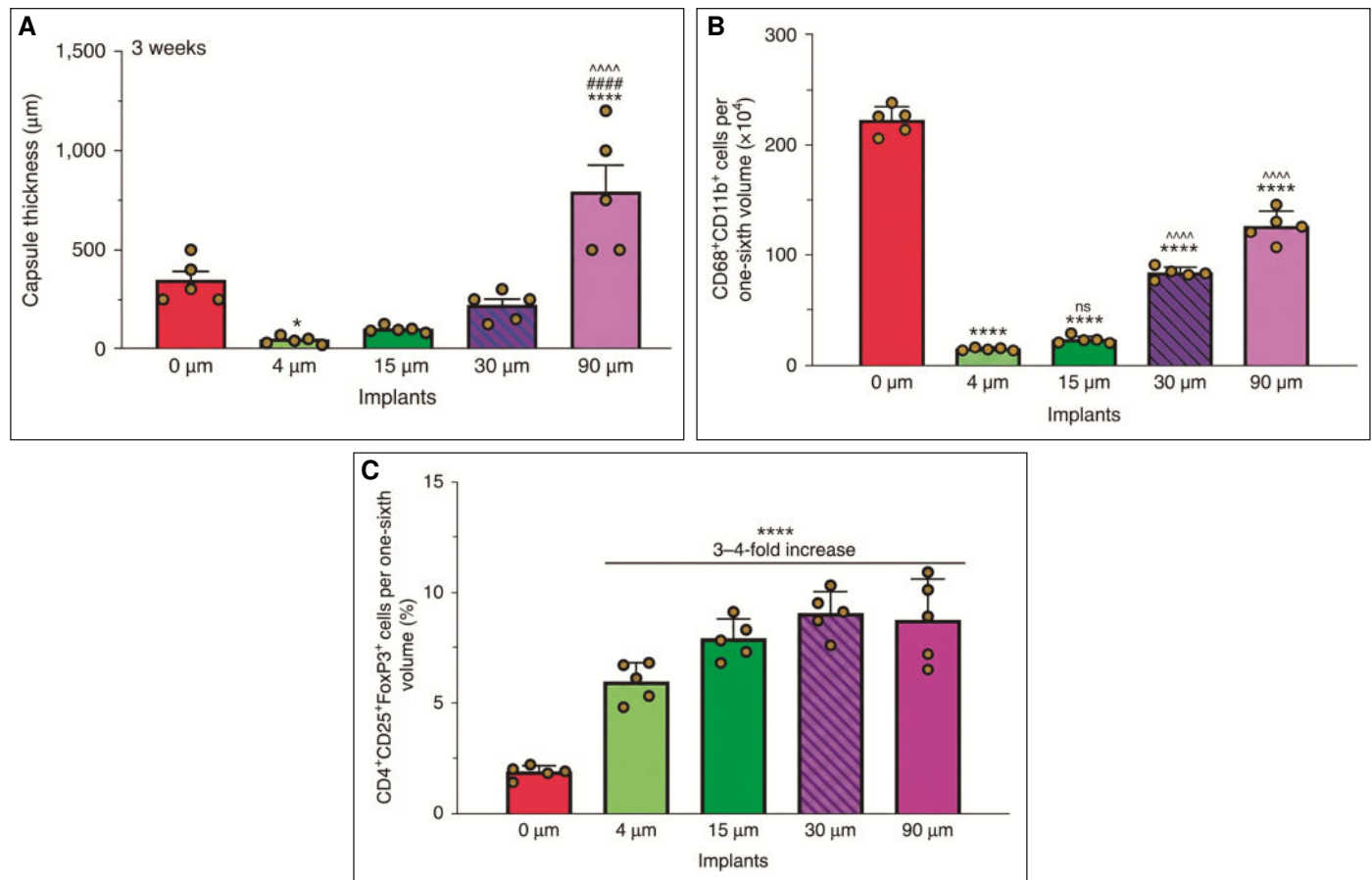


Figure 3. Short-term macrophage cell response in mice. Capsule thickness (A), proinflammatory macrophage recruitment (B), and anti-inflammatory Treg recruitment (C) across surface topographies at 3 weeks. One-way ANOVA with Bonferroni correction; * $P < .05$, ** $P < .01$, **** $P < .0001$ vs 0 μm ; #### $P < .0001$ vs 4 μm ; ^^^ $P < .0001$ vs 15 μm . Data represent mean $\pm$ SEM, $n = 5/\text{group}$. SEM, standard error of the mean; Treg, regulatory T cells. Adapted from Doloff et al, 2021, with permission.

as IL-1 β , IL-6, and TNF α , further amplifying immune cell infiltration and activating T helper 1 (Th1) responses.⁶ Continued macrophage accumulation and fusion into foreign body giant cells (FBGC) sustains local inflammation and contributes to fibrotic capsule formation, which serves to isolate the implant from surrounding tissue. Over time, macrophage polarization shifts toward an anti-inflammatory, profibrotic (M2-like) phenotype, marking progression from acute inflammation to chronic fibrosis and capsule maturation.¹⁷

Adaptive immune modulation is central to regulating this response. The 2025 Nobel Prize in Physiology or Medicine recognized discoveries in peripheral immune tolerance, particularly the identification and characterization of regulatory T cells (Tregs), which play a pivotal role in suppressing excessive immune activation and modulate chronic inflammation.¹⁸ At the implant site, Tregs, together with T helper 2 (Th2) cells and anti-inflammatory cytokines, help rebalance the immune microenvironment from a proinflammatory toward an immunoregulatory state. This transition promotes implant tolerance, controlled fibrotic responses, and improved long-term biointegration.¹¹ Rather than driving fibrosis, Tregs, as specialized subset of CD4⁺ T lymphocytes, attenuate macrophage activation, modulate fibroblast signaling, and limit exaggerated FBR.

Fibrotic Encapsulation

Capsule formation represents the natural endpoint of prolonged host–implant interaction and reflects the host's attempt to balance immune tolerance with structural isolation of the breast implant.

Capsules are clinically characterized by thickness, degree of contraction, pliability, the presence of calcifications, vascularity, and, in some cases, multilayered or “double capsule” morphology. While capsule formation is universal, qualitative properties determine clinical outcome. Capsules are also a biological imprint that results from the remodeling of periprosthetic tissues, one that is not inert. Its study provides a microsite of evidence.

Preclinical Evidence

Doloff and colleagues conducted a comprehensive multi-species analysis at the Massachusetts Institute of Technology (MIT, Boston, MA, USA),⁹ integrating murine mini-models with full-scale commercial implants in New Zealand White rabbits. Given the methodological rigor and translational relevance of this work, its findings warrant detailed consideration.

Cell Response

To characterize the early immune response, macrophage infiltration and T-cell recruitment were evaluated in wild-type C57BL/6 mice using fluorescence-activated cell sorting (FACS) 3 weeks following implantation. Peri-implant tissues were collected, enzymatically dissociated, and stained with antibodies targeting key immune markers, including CD11b, CD68, CD4, and CD25.

Capsule thickness was lowest in the 4- μm surface group compared with all other surface topographies, with statistically significant

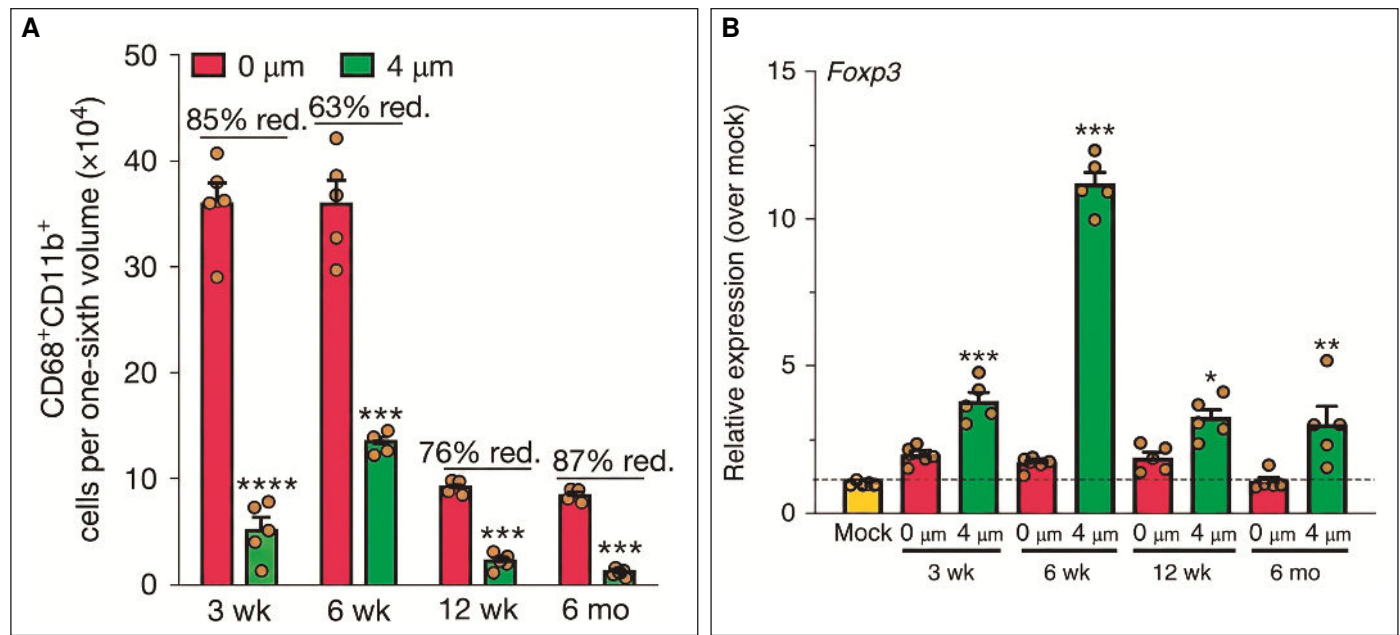


Figure 4. Long-term immune response to the ~0 and 4 μ m surfaces in mice. Flow cytometry analysis of macrophage response (A) and qPCR analysis of anti-inflammatory Treg markers (B) through 6 months. One-way ANOVA with Bonferroni correction; * $P < .05$, ** $P < .01$, *** $P < .001$, **** $P < .0001$ vs 0 μ m. Data represent mean $\pm$ SEM, $n = 5$ /group. ANOVA, analysis of variance; qPCR, quantitative polymerase chain reaction; SEM, standard error of the mean; Treg, regulatory T cells. Adapted from Doloff et al., 2021, with permission.

reductions relative to both the nonmodified smooth (~0 μ m) and highly textured (90 μ m) implants (Figure 3A). Consistent with this observation, FACS analysis demonstrated significantly fewer macrophages adherent to the 4 μ m surface compared with the ~0 μ m, 30 μ m, and 90 μ m surfaces (Figure 3B).

Notably, all implant surfaces—including the 4 μ m surface—exhibited a statistically significant compositional (percentage-based) increase in regulatory T cell (Treg) recruitment compared with the nonmodified ~0 μ m surface (Figure 3C). Further, the 4 μ m surface was shown to actively and directly recruit Tregs, rather than the other larger topography surfaces, for which Tregs were likely increasing as a bodily response and an attempt at maintaining peripheral tolerance due to the rise of proinflammatory lymphocytes, as confirmed by multiplexed NanoString gene expression as well as single-cell RNA sequencing data.⁹ Taken together, these findings highlight a marked immunological divergence between the ~0 and 4 μ m surfaces despite only a minimal absolute difference in surface roughness.

Long-Term Evaluation and Role of Adaptive Immune Regulation

Based on these short-term findings, a targeted long-term analysis comparing the ~0 μ m and 4 μ m surfaces was conducted in wild-type C57BL/6 mice for up to 6 months postimplantation. This comparison was both methodologically intentional and biologically informative, aiming to further investigate the pronounced divergence observed between implants classified as smooth surfaces despite their minimal difference in surface roughness. The analysis revealed persistent and statistically significant differences in immune response and capsule thickness across all evaluated time points (Figure 4A, B).

To directly assess whether the observed protective effects were mediated by adaptive immune regulation rather than nonspecific suppression of inflammation, immune responses to the nonmodified ~0 and 4 μ m surfaces were evaluated in T-cell-deficient mice models 3 weeks following implantation. These animals harbor a genetic

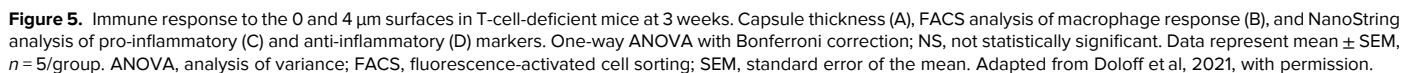
mutation that impairs T-cell maturation and differentiation, while preserving functional macrophages and FBGC, resulting in baseline FBR dominated by macrophage-mediated inflammation.

In contrast to wild-type animals, the protective effects observed with the 4 μ m surface—including reduced capsule thickness and favorable immune modulation—were completely absent in T-cell-deficient mice (Figure 5A, B). Specifically, the significantly reduced capsule thickness and increased Treg recruitment seen in wild-type mice implanted with the 4 μ m surface (Figure 3A–C) were no longer observed, demonstrating that Tregs are not merely correlated with reduced fibrosis, but are essential mediators of the protective response. The use of this model represents a key methodological strength, as it enables clear discrimination between innate inflammatory responses and true Treg-dependent immune modulation.¹⁹

This conclusion was further supported by comprehensive inflammatory gene expression analyses conducted 6 weeks postimplantation. These assays demonstrated reduced macrophage-associated markers and increased expression of anti-inflammatory mediators, including IL-4, along with elevated Treg signatures on the 4 μ m surface in wild-type mice. In contrast, this anti-inflammatory and regulatory profile was absent in T-cell-deficient animals (Figure 5C, D).

Importantly, the influence of Tregs should not be interpreted as simply inversely proportional to FBR intensity. Both micro- and macro-textured surfaces elicited robust Treg recruitment while also exhibiting increased capsule thickness. These findings suggest that multiple drivers contribute to fibrotic outcomes and support the concept of an optimal immunological balance—or “sweet spot”—in which regulatory mechanisms effectively counteract macrophage-driven inflammation. In this context, greater surface roughness may require increased Treg recruitment to modulate a stronger initial response, whereas the 4 μ m surface appears to promote a more rapid transition toward a regulated, wound-healing phenotype.

Further, it should be noted that implant surfaces with a similar average roughness (eg, 4 microns) are not always equivalent, as surface topography is defined by multiple parameters beyond roughness, including skewness (peaks vs valleys), kurtosis



Shell surface (μm)	Tension lines/rippling		
	3 months	6 months	1 year
0*	3/5	4/5	4/5
4	0/5	1/5	1/5
15	1/5	2/5	ND
28	4/5	5/5	ND
50	1/5	3/5	ND
83	3/5	4/5	5/5

(sharpness vs lack thereof, of peak size distribution or dispersity), and peak density, all of which influence cellular behavior. Recent literature²⁰ may not acknowledge that ostensibly similar 4-micron surfaces can differ substantially in these microstructural characteristics, meaning that similar manufacturing methods do not necessarily reproduce the same surface architecture as well as host response outcomes. Furthermore, artificial processing of implant materials (eg, cutting, inversion, or adhesion) can alter surface features and

Similarly, Bérniz et al.²² assessed implant surface disks in Wistar female rats 5 weeks postimplantation. Histopathologic analyses demonstrated that the same surface was associated with lower cellularity, reduced macrophage infiltration, and fewer myofibroblasts—features consistent with decreased susceptibility to fibrotic capsule formation.

To extend these findings in a clinically relevant large-animal model, commercially available, human-scale implants bearing the tested surface topographies were implanted in the subcutaneous region of New Zealand White rabbits. Capsule thickness, along with local cell and cytokine expressions associated with immune, inflammatory, and fibroblast-mediated fibrotic responses, was evaluated 3 weeks

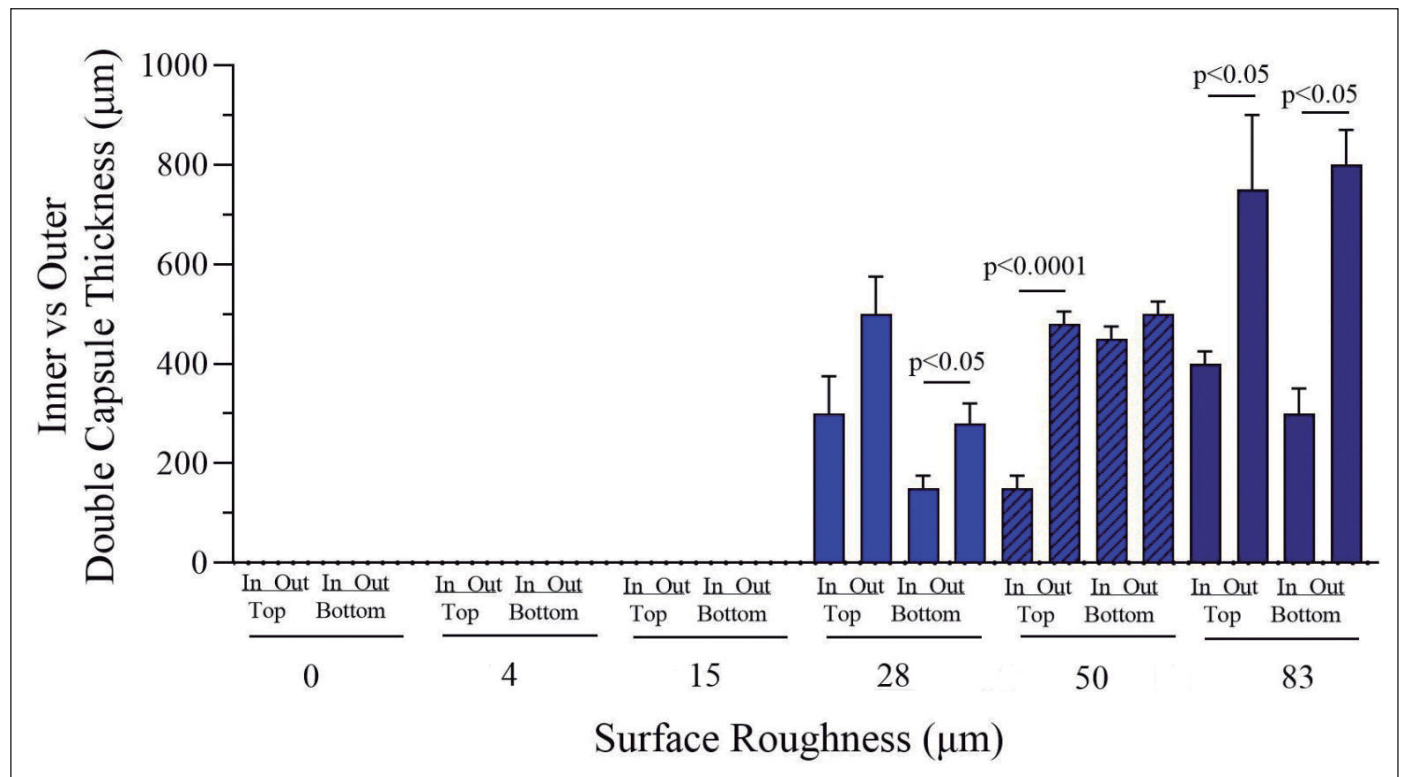


Figure 7. Surface-dependent double capsule formation at 3 weeks postimplantation in rabbits. Inner and outer capsule thickness was quantified on top and bottom implant surfaces. Double capsules were detected only for 28, 50, and 83 μm textured implants. Adapted from Doloff et al, 2021, with permission.

Capsular Tension in the Skin

The clinical appearance of implant rippling has been linked to the mechanical interaction between capsular fibrous tissue and the overlying skin, a process that is influenced by implant surface topography. Accordingly, tension lines and rippling (TL/R) were evaluated in the skin and implant capsules of New Zealand rabbits. As shown in Table 1, the 4 μm surface consistently exhibited the lowest incidence of TL/R in both the skin and implant capsule across all evaluated time points up to 1 year. This finding is particularly noteworthy given that reduced capsular thickness has traditionally been associated with an increased risk of rippling in breast implants. However, these results, together with the immunologic profile promoted by the 4 μm surface, suggest the formation of a more compliant and elastic extracellular matrix. Such a matrix may better dissipate mechanical stress, thereby reducing fibrosis-associated complications, including TL/R.

Clinical Implications

Inherently, downregulating the FBR at multiple stages results in a less fibrotic capsule. Evidence from the human sample arm of the Doloff et al,⁹ consisting of 21 explanted capsules from women with 3 different surfaces aligns with other published studies.

The senior author of this manuscript performed histological evaluation of periprosthetic capsular tissue obtained from 34 consecutive cases involving 4 μm implant replacement or explantation. Despite heterogeneity in the cohort and implantation times ranging from 1 to 16 years, the capsules exhibited strikingly consistent macroscopic features: pliable, uniform, translucent, and minimally vascularized. Histological analysis of 64 capsules reported an average thickness of 0.85 mm (range: 0.19–3.48 mm), characterized by low fibrotic content

with minimal cellular activity or evidence of ongoing immune response. Notably, the oldest capsule in the sample—depicted in Figure 8 and the video, available online at <https://doi.org/10.1093/asj/sjag115>, exemplifies these characteristics and highlights the durability of this tissue phenotype over time.

A noteworthy series of investigations from the Medical University of Innsbruck led by Wolfram has provided an important translational bridge between preclinical mechanistic findings and bedside validation.^{23–26} In these studies, temporary breast tissue expanders with 4 μm vs 60 μm surface roughness were randomized and implanted within the same patient, allowing intraindividual comparison of tissue responses to differing implant topographies. This design represents a major methodological strength, as it minimizes confounding variables related to host biology, surgical technique, and wound-healing variability. Analysis of peri-implant tissue using proteomic profiling and flow cytometry demonstrated that rougher surfaces trigger higher levels of pro-inflammatory cytokines and cellular stress-response proteins. Conversely, reduced surface roughness (4 μm) demonstrated a molecular profile more consistent with regulated wound healing, characterized by increased regulatory T-cell activity and reduced markers of inflammatory signaling.

In addition to the molecular and histologic findings, patient-reported outcomes from this cohort provide clinically relevant insight into how surface-mediated immune modulation may translate into patient experience. Patients consistently reported lower levels of breast pain, reduced discomfort, and decreased nipple sensation loss on the side implanted with the 4 μm surface.²³ These findings were accompanied by significantly higher ratings of overall comfort and aesthetic satisfaction. Such observations may reflect the underlying biologic response to the implant surface. A less pronounced inflammatory and fibrotic response during the early tissue-expansion period may therefore contribute to improved tissue compliance,

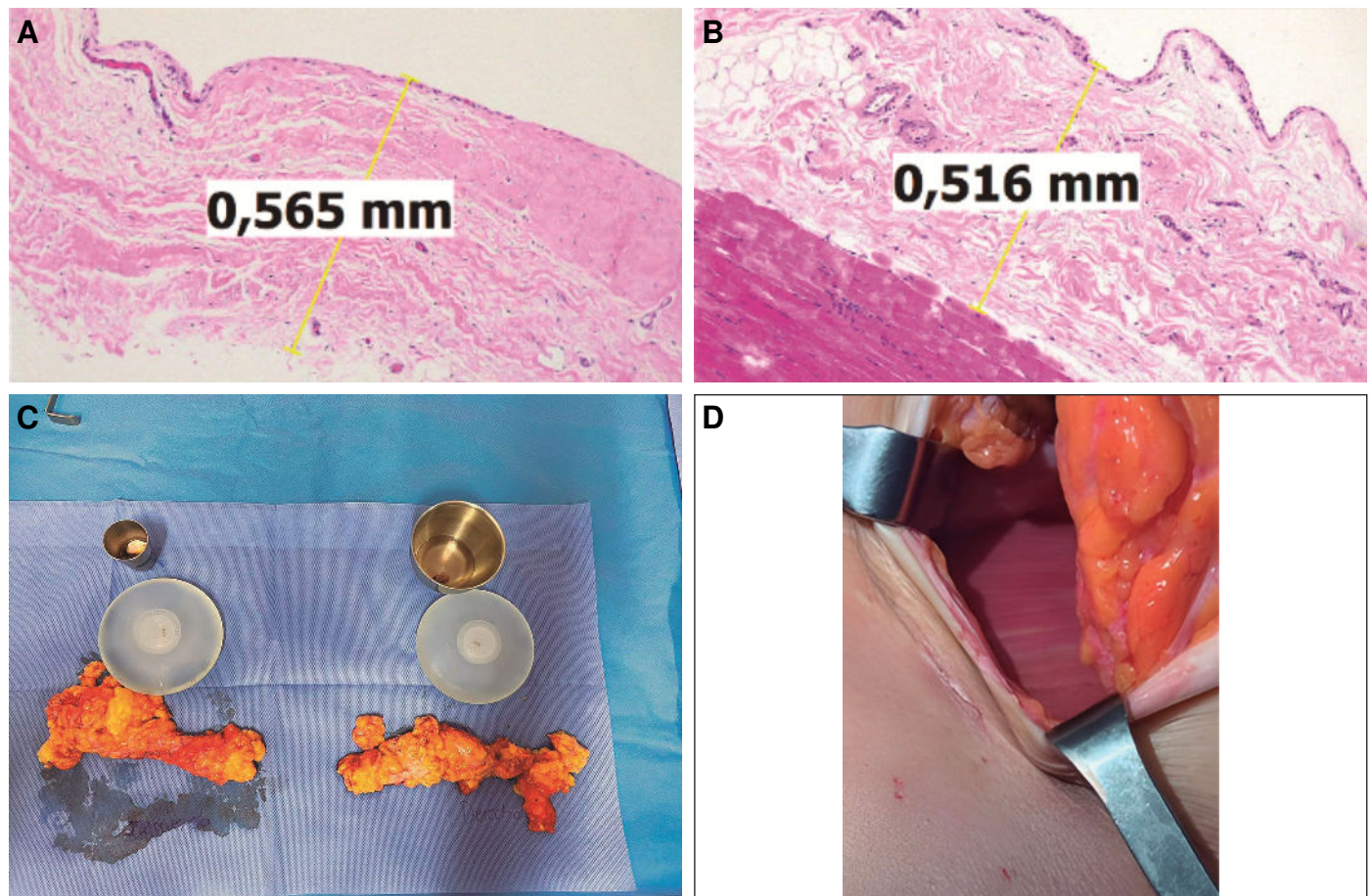


Figure 8. Sixteen-year capsule: histological measurements of (A) right side and (B) left side, alongside the macroscopic appearance on the operating table (C, D). This case is particularly noteworthy as it originates from the very first year of commercial availability of the 4 μ m implants. Images courtesy of Dr Manuel Chacon-Quiros, used with permission.

reduced nociceptive stimulation, and greater overall patient comfort. These translational findings support the concept that microtopographic modulation of the FBR may have measurable effects not only on capsule biology but also on patient-perceived outcomes during breast surgery.

Absence of Association to Lymphoproliferative Diseases

The absence of evidence is no evidence of absence. Nonetheless, to date, no cases of primary BIA-ALCL have been reported in association with implants possessing this 4 μ m surface architecture since their market introduction in 2010.²⁷ While continued surveillance remains essential, this observation aligns with current epidemiologic data linking the majority of BIA-ALCL cases to macrotextured devices.²⁸

Lower Incidence of Fibrotic Capsules and Cumulative Risk Capsular Contracture

Clinical outcome data further suggest that implant surface architecture may influence long-term capsular behavior. Recent data from the FDA Core Study evaluating implants with the 4 μ m surface roughness report a Kaplan–Meier estimated capsular contracture rate of 0.5% at 5 years,²⁹ which is comparatively low relative to rates historically reported in earlier core studies evaluating other implant surface types (Table 2).^{30–32} The majority of implants in this study were placed

in some variation of the dual-plane. Direct comparisons between FDA core studies should be interpreted cautiously, given differences in patient populations, surgical techniques, follow-up duration, and study methodology. Nevertheless, these findings are consistent with the biologic framework suggested by preclinical and translational studies, in which modulation of the FBR may influence capsule thickness and contracture risk. Importantly, the low capsular contracture rate observed in the 4 μ m FDA core study is further contextualized by international clinical experience.³³

Surgical Approach With Breast Tissue Preservation

Breast tissue preservation (BTP) is an advanced surgical concept that represents a paradigm shift in aesthetic breast surgery, encompassing primary augmentation, augmentation-mastopexy, and hybrid approaches. It is grounded in a framework that prioritizes preservation of the patient's native anatomy and physiology. This preservation-oriented philosophy integrates minimally invasive technologies with biologically informed surgical planning to limit tissue injury and protect the supporting structures of the breast, favorably modulate the host immune response, and optimize long-term functional and aesthetic outcomes.

Central to the success of a BTP strategy is the biologic interaction between the implant surface and the host immune response. Preservation of glandular, muscular, and neurovascular cellular integrity—while avoiding unnecessary tissue disruption—aims to prevent injury-induced chronic inflammation that may impair wound healing

Table 2. Summary of Capsular Contracture Rates Across FDA Core Studies

	Motiva 5 years ^a	Sientra 5 years ^a	Mentor 6 years ^a	Allergan 6 years ^a
KM risk of capsular contracture	0.5%	8.8%	9.8%	14.8%
N patients (primary augmentation)	451	1116	552	455
Compliance rate	88.5%	80%*	64%	81%

*From the 3-year report.

and compromise long-term implant integration. Within this context, the quality of the periprosthetic capsule becomes a critical determinant of device biointegration and tissue compliance. An optimal implant surface should promote formation of a well-organized capsule that supports mechanical stability while minimizing chronic inflammation and fibrosis. As demonstrated by the evidence synthesized in this review, the 4 μm surface currently represents the most favorable available option to achieve these objectives, aligning immune modulation with durable safety, stability, and implant biointegration.

By redefining the underlying surgical philosophy, BTP introduces an additional decision-making layer that emphasizes tissue biology, physiologic preservation, and long-term outcomes, rather than replacing established breast surgery algorithms. Within this expanded framework, surgical strategies can be more precisely tailored to the patient's native anatomy and individual biologic capacity for tissue preservation. Nevertheless, BTP may not be appropriate for all clinical scenarios. Patients presenting with native anatomical dimensions requiring large implant widths, marked soft-tissue deficiency, or a history of multiple prior procedures that have significantly altered tissue planes and compromised support structures may be better served by conventional or reconstructive approaches beyond the scope of a preservation-based strategy.

Contemporary tissue-preserving augmentation approaches that emphasize minimal tissue disruption and maintenance of native anatomical planes that constitute the natural support structures of the breast have demonstrated encouraging outcomes with intralamellar prepectoral placement of 4 μm implants. Prospective clinical studies have demonstrated zero capsular contracture and malposition rates at 3 years and 18 months follow-up periods, with 100 and 330 patients, respectively.^{34,35} These observations suggest that the 4 μm surface associated with an improved biocompatibility and tissue compliance help optimize outcomes in different prepectoral planes achieving an optimal equilibrium of low inflammatory breast stability, softer feel and reduced capsular contracture.

Surgical Approach With Conventional Techniques

Prior to the development of BTP techniques, efforts to achieve long-term stability in breast augmentation evolved in parallel with the use of conventional smooth, textured, and polyurethane-covered implants.³⁶ The rationale for textured and polyurethane surfaces was to promote implant-tissue adhesion through a heightened inflammatory response, leading to the formation of thicker fibrous capsules believed to confer improved long-term stability. However, this high inflammatory surgical approach to obtain breast stability was subsequently associated with new complications, including waterfall deformity, the formation of double capsules, late seromas, and, ultimately, proliferative conditions such as BIA-ALCL.³⁶

In contrast, stability with smooth implants has traditionally relied on surgical technique rather than implant surface characteristics. Strategies have included the use of internal surgically created capsular or fascial slings, multiplanar pocket creation, and the incorporation of adjunctive materials, such as synthetic scaffolds or acellular dermal matrices (ADM), to reinforce the soft-tissue envelope.³⁷

Across both conventional smooth and textured devices, surgical protocols historically have emphasized meticulous tissue dissection, creation of precise “glove-tight” pockets, and strict adherence to contamination-minimizing strategies. Over the past 2 decades, however, clinical concerns such as capsular contracture, implant visibility, and/or palpability contributed to the widespread adoption of the dual-plane technique irrespective of implant surface characteristics, in an effort to improve implant coverage and mitigate surface-related complications.³⁸

Beyond the incidence of capsular contracture, qualitative capsule properties—such as structure, organization, and elasticity—play a pivotal role in surgical planning and long-term implant behavior. Surgeons must consider anticipated capsular development, tissue elasticity, and implant–tissue integration when selecting the appropriate surgical strategies.

When breast tissue preservation techniques are not feasible, surgical protocols for the 4 μm surface generally align with those used for conventional smooth implants, albeit with enhanced attention to precise tissue management. Implants associated with reduced fibrotic capsule formation require detailed pocket dissection, accurate implant-to-pocket dimensional matching and respect of the lateral boundaries of the breast. In selected cases, based on the choice of large implant sizes in patients with lax tissue envelopes, additional soft-tissue support to mitigate risks of malposition may be required, as it is commonly done for conventional smooth implants.³⁹⁻⁴¹ Importantly, implant surfaces that induce fundamentally different capsular phenotypes should not be approached with identical techniques, and principles derived from experience with macrotextured or polyurethane-covered devices are not directly applicable to low-roughness surfaces.⁴²

In prepectoral planes, where muscular coverage is absent, the biologic interface between the surface and surrounding soft tissues assumes particular importance. Capsular structure and elasticity may directly influence implant stability, device wrinkling and rippling, and force transmission to overlying tissues. In this setting, the 4 μm surface appears particularly advantageous. A more elastic capsule may allow more harmonious mechanical interaction between the implant and the surrounding tissues, potentially reducing distortion while allowing expression of the implant's intrinsic biomechanical properties of the device to be expressed more naturally. These observations are consistent with the findings from New Zealand rabbit models (Table 1) demonstrating favorable capsule characteristics that reduce underlying traction rippling.

Further support for favorable outcomes in the prepectoral plane is provided by a 2-center international clinical evaluation reported by Randquist et al.⁴⁰ This series included 1053 breast augmentations with 4 μm implants, of which 588 were performed in the prepectoral plane, and demonstrated overall low complication rates across the cohort, including a capsular contracture rate of 0.7%, 0% rippling, and minimal implant malposition. Although outcomes were reported for the combined population and not stratified by implant plane, the substantial representation of prepectoral cases supports the relevance of meticulous pocket control, inframammary fold preservation, and maintenance of native soft tissue support in achieving stable clinical and aesthetic outcomes.⁴⁰

Concerns that reduced surface adhesion could predispose to implant displacement in prepectoral or subfascial planes have not been substantiated clinically. Munhoz et al in a large series reported no statistical significant difference in malposition rates between 4 μm

and macrot textured implants ($P = .695$).⁴³ Malposition rates were 2.7% in primary and 5.5% in secondary augmentation, reinforcing that implant stability depends more on precise surgical execution than on surface-induced adhesion alone.⁴³

Despite the advantages of the prepectoral pocket, limitations remain, particularly in thin patients with poor soft-tissue coverage. In such cases, the combination of the 4 μm surface with autologous fat grafting has shown consistent benefits.⁴⁴⁻⁴⁶ Hybrid techniques,⁴⁴ the use of smaller implant volumes,⁴⁵ and standardized zone-based fat grafting,⁴⁶ have further optimized upper pole fullness and reduced the intermammary distance in high-risk patients with no reported rippling and improved implant edge camouflage, even in patients with pinch test <2 cm and BMI <20 kg/m²,⁴⁴ compensating the limitations of the prepectoral plane while preserving the benefits of prepectoral augmentation.⁴⁶

Future Perspectives

It is equally relevant to recognize the contribution of immune reactivity to tissue injury. Well recognized elements such as intraoperative bleeding or residual blood within the pocket create an environment conducive to inflammation and subsequent fibrosis. Optimal outcomes therefore depend not only on device design but also on the surgeon's ability to minimize tissue trauma, avoid devitalization, bacterial mitigation efforts, and reduce postoperative inflammatory triggers that can drive different patterns of scarring. Tissue preservation techniques have spotlighted the advantages of less traumatic procedures, with reduced thermal damage from electrocautery pocket dissection, shorter surgical time and thus, avoiding additional inflammation while obtaining breast stability without the need of slings, multi-plane techniques or external tissue support.^{34,35,47}

Collectively, these converging lines of evidence point toward a broader conceptual framework in which the FBR is not simply an inevitable consequence of implantation but a biologically modifiable process. By understanding and ultimately modulating this response with a 4 μm surface topography, it becomes possible to cultivate an optimal interface between the device and native breast tissue—one that supports stable low-inflammatory biointegration, minimizes adverse outcomes, and aligns more closely with physiologic healing.

The evidence reviewed in this manuscript suggests that the biologic behavior of the periprosthetic capsule is determined not simply by its thickness, but by its structural organization, elasticity, and underlying immune profile. Implants with this specific 4 μm surface consistently demonstrated thinner yet well-organized and compliant capsules associated with reduced proinflammatory signaling, increased regulatory immune activity, and favorable tissue adaptation in both preclinical and translational studies. Consistent with this, available clinical data demonstrate low rates of capsular contracture; moreover, they have not shown increased rates of implant malposition or instability with these devices when appropriate implant selection and contemporary surgical techniques are used. These findings suggest that concerns regarding displacement attributable solely to reduced capsular thickness may instead reflect the learning curve experience by surgeons transitioning away from high roughness implant surfaces.^{40,48} Rather than relying on excessive fibrotic adhesion for stability, this low-inflammatory capsule phenotype appears capable of supporting physiologic implant integration while preserving tissue compliance and more natural biomechanical behavior. This type of structured capsule may exhibit greater elasticity and compliance, allowing the biomechanical properties of the implant to interact more naturally with surrounding breast tissue and reducing implant rippling.^{34,49} Elastographic studies have suggested

that reduced capsular stiffness may improve tissue dynamics and contribute to more physiologic breast movement.⁵⁰ These findings align with evolving surgical goals centered on patient comfort, the sense of naturalness, and responsive device biomechanics.

Limitations

This review article has several limitations. First, it is a narrative synthesis that reflects the scientific and clinical interpretation of previously published works, curated to provide context and perspective. Second, some comparative outcome data derive from studies performed across different decades, during which implant design, surgical technique, perioperative protocols, and definitions of complications have evolved substantially. These temporal differences may introduce bias and limit direct comparison between historical and contemporary cohorts. Third, the clinical evidence included is inherently heterogeneous, encompassing aesthetic augmentation, breast reconstruction, explanted capsule analyses, and both prepectoral and subfascial approaches, which may limit the generalizability of conclusions across all patient populations.

CONCLUSIONS

Emerging evidence indicates that implant surface microtopography plays an important role in modulating the host foreign body response and the characteristics of the periprosthetic capsule. Surface with roughness on the order of approximately 4 μm has been associated in preclinical and translational studies with reduced inflammatory signaling, enhanced regulatory immune activity, and reduced fibrotic capsule formation and more compliant capsules. These biologic effects translate into clinically relevant differences in tissue behavior, patient comfort, and implant dynamics. As surgical techniques increasingly incorporate breast tissue-preserving prepectoral approaches, the biologic interaction between implant surfaces and surrounding tissues becomes an important determinant of long-term outcomes supported by low inflammatory breast stability. Continued integration of biomaterial science, immunologic insights, and clinical data will be essential to further refine implant design and optimize surgical strategies in breast augmentation and reconstruction.

Disclosures

Drs Chacon-Quiros and Teitelbaum are consultants for Establishment Labs (Alajuela, Costa Rica) and own shares of the company. Dr Munhoz is a member of the Medical Advisory Board of Establishment Labs and serves as medical educator for Establishment Labs. Dr Calobrace is a consultant and medical educator for Establishment Labs. Drs Doloff and Veisheh have received funding for research projects from Establishment Labs. No compensation was provided to the authors for research and writing of this manuscript. The other authors declared no potential conflicts of interest with respect to the research, authorship, and publication of this article.

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