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Fractional picosecond laser-induced optical breakdown in dermatology: from controlled microinjury and molecular repair pathways to clinical endpoints and post-procedural care

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ABSTRACT

Fractional picosecond lasers have expanded the clinical use of ultrashort-pulse devices from tattoo and pigment removal to non-ablative rejuvenation, scar remodeling, enlarged pores, dyschromia and selected photoaging indications. Their distinctive tissue effect is not conventional fractional coagulation or ablation, but focal photomechanical disruption mediated by laser-induced optical breakdown (LIOB) and associated laser-induced cavitation (LIC), with vacuolization in epidermal and dermal tissue planes.. This review synthesizes the translational evidence linking fractional optical delivery systems, histologic microinjury patterns, non-invasive imaging correlates, molecular repair pathways, clinical endpoints, safety across skin types and post-procedural care. PubMed-indexed literature was reviewed using search blocks for fractional picosecond lasers, LIOB/LIC, microlens and diffractive optical systems, optical coherence tomography (OCT), reflectance confocal microscopy (RCM), multiphoton and harmonic generation microscopy (HGM), wound healing, barrier repair, photoprotection and post-inflammatory hyperpigmentation (PIH). Current evidence shows that fractional picosecond systems can generate epidermal and dermal vacuoles, shockwave-associated optical signatures, melanin-dependent thresholds and remodeling responses involving matrix metalloproteinases (MMP), collagens, heat shock proteins (HSPs), cytokines, chemokines and barrier-related proteins. Clinical evidence is strongest for atrophic acne scars, photoaging, pores and selected non-acne scars, with increasing data in Asian and darker skin phototypes. However, the extent to which clinical outcomes are directly attributable to LIOB rather than combined photomechanical, photoacoustic and limited photothermal effects remains incompletely defined. Evidence-based aftercare remains underdeveloped. Conservative barrier support, strict photoprotection and avoidance of irritants are currently the most defensible post-procedural principles.

Key words: picosecond laser; laser-induced optical breakdown; fractional laser; wound healing; post-inflammatory hyperpigmentation.

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Introduction

Picosecond lasers were introduced into dermatology primarily to improve tattoo and pigment clearance by delivering ultrashort pulses with high peak power and limited thermal diffusion. With the addition of fractional optical delivery systems, however, these devices acquired a second and mechanistically distinct role: the creation of controlled microscopic zones of photomechanical injury in the epidermis and dermis without continuous surface ablation. This has broadened clinical applications toward atrophic acne scars, photoaging, enlarged pores, wrinkles, dyspigmentation and selected non-acne scars.[1-13]
The central tissue event of fractional picosecond treatment is laser-induced optical breakdown (LIOB), commonly described as plasma-mediated and cavitation-associated microinjury produced when local irradiance exceeds the optical breakdown threshold. Depending on wavelength, pulse duration, fluence, spot architecture, melanin density and focal depth, LIOB may be followed by cavitation and may manifest histologically as intraepidermal vacuoles or dermal cavities.. These events differ from the thermal coagulation columns of non-ablative fractional lasers and the vaporization columns of ablative fractional lasers.[2,14-24]
Several reviews have addressed the clinical uses of picosecond lasers, but most treat picosecond lasers as a broad device category and focus on clinical indications. A clinically useful synthesis requires a narrower translational approach: how fractional beam delivery generates LIOB/laser-induced cavitation (LIC), how these lesions are visualized, which molecular repair pathways are plausibly involved, how clinical endpoints differ across skin types, and how post-procedural care should be aligned with the specific microinjury model. This review therefore focuses on fractional picosecond LIOB as a controlled microinjury paradigm rather than on picosecond lasers as a general treatment class.

Search strategy and evidence selection

A structured narrative search was performed in PubMed through 18 June 2026. Search blocks included fractional picosecond lasers, LIOB, LIC, optical breakdown, laser-induced cavitation, picosecond Nd:YAG, picosecond alexandrite, 755 nm, 785 nm, 532 nm, 1064 nm, diffractive lens array (DLA), micro-lens array (MLA), diffractive optical element (DOE), holographic optics, optical coherence tomography (OCT), reflectance confocal microscopy (RCM), multiphoton microscopy (MPM), harmonic generation microscopy (HGM), histology, wound healing, collagen, matrix metalloproteinase (MMP), heat shock proteins (HSPs), cytokines, chemokines, dexpanthenol, sunscreen, photoprotection, barrier repair and PIH. Additional citation discovery was used only to identify candidate references; inclusion in the manuscript required PubMed verification or a PubMed-indexed source with a verifiable DOI.
Eligible articles included reviews, clinical trials, split-face studies, observational studies, histologic investigations, animal and *ex vivo* models, imaging studies, molecular studies and post-procedural care studies relevant to fractional picosecond treatment, LIOB/LIC or laser-related wound healing. Tattoo-only literature, melasma-only literature, non-dermatologic plasma physics and non-picosecond energy-based device literature were included only when they clarified mechanism, safety or comparator biology. This is a narrative review rather than a systematic review or meta-analysis; no pooled effect size was calculated. The search logic is summarized in Table 1.

Physical and optical basis of fractional picosecond LIOB

The defining optical property of picosecond pulses is the delivery of high peak power within an ultrashort pulse duration. In fractional modes, this energy is spatially redistributed by optical systems such as DLA, MLA, DOE or holographic beam-splitting optics. These systems create microspots with locally high fluence surrounded by lower-fluence areas, allowing microscopic photomechanical injury while preserving intervening tissue.[2,15,19-24]
The tissue endpoint is determined by both device and patient variables. Device-dependent factors include wavelength, pulse duration, beam profile, spot size, focal depth, pulse energy, fluence, repetition rate, number of passes and cooling. Tissue-dependent factors include melanin density, epidermal thickness, hydration, anatomical site, scar architecture and skin phototype. Direct experimental evidence indicates that 1064 nm picosecond LIOB thresholds depend on skin type, supporting the need for conservative parameter selection in darker phototypes.[15] *Ex vivo* pigmented skin models also show wavelength-dependent immediate reactions after 532 and 1064 nm picosecond-domain irradiation.[16]
Optical design is clinically relevant. Micro-lens arrays and diffractive optical elements do not simply “fractionate” the beam; they alter local irradiance distribution and thus the

Table 1. Search strategy and evidence-selection logic.

Search block	Key terms	Use in the review
Fractional picosecond platforms	Fractional picosecond, fractionated picosecond, 755 nm, 785 nm, 532 nm, 1064 nm, Nd:YAG, alexandrite	Identification of clinical and translational picosecond studies
LIOB and LIC	Laser-induced optical breakdown, LIOB, optical breakdown, laser-induced cavitation, LIC	Mechanistic and histologic core evidence
Optical delivery	Diffractive lens array, micro-lens array, diffractive optical element, holographic optic, beam splitter	Device and optics stratification
Imaging and histology	OCT, RCM, multiphoton microscopy, harmonic generation microscopy, SHG, THG, histology, histopathology	Validation of microscopic tissue endpoints
Molecular and aftercare	Wound healing, collagen, MMP, HSP, cytokine, dexpanthenol, sunscreen, PIH, barrier repair	Repair-pathway and post-procedural care synthesis

LIOB, laser-induced optical breakdown; LIC, laser-induced cavitation; OCT, optical coherence tomography; RCM, reflectance confocal microscopy; SHG, second harmonic generation; THG, third harmonic generation; MMP, matrix metalloproteinases; HSP, heat shock protein; PIH, post-inflammatory hyperpigmentation.

probability, depth and uniformity of LIOB formation. Comparative optical studies demonstrate different LIOB patterns between MLA and DOE systems after 1064 nm picosecond irradiation, while newer diffractive micro-lens arrays aim to improve uniformity and selectivity.[19,20] Clinical and histologic studies using 755 nm alexandrite DLA, 532/1064 nm Nd:YAG DOE and 1064 nm holographic optics illustrate that the optical delivery system must be reported alongside wavelength and fluence.[21-24]

Histologic and imaging evidence of controlled microinjury

The strongest direct evidence for fractional picosecond LIOB comes from histology and non-invasive imaging. Early clinical and histologic work with 755 nm alexandrite fractional lens systems demonstrated intraepidermal cavities interpreted as areas of LIOB, with the number and size of cavities influenced by delivered energy and melanin index.[25,26] Subsequent studies using 1064 nm Nd:YAG fractional picosecond devices reported epidermal and dermal vacuoles, pigment accumulation, inflammatory cell infiltration and dose-dependent tissue effects *in vivo* and *ex vivo*.[27,28]

Non-invasive imaging has become essential because visible clinical endpoints are not reliable surrogates for subclinical LIOB. OCT can identify epidermal LIOB morphology and has been proposed as a method for determining LIOB thresholds in relation to melanin content.[29] RCM can visualize hyporeflective spherical structures consistent with vacuoles and correlate these with histopathology in Fitzpatrick III-IV volunteers.[30] Multiphoton microscopy provides label-free longitudinal imaging of epidermal microinjury, necrotic debris, inflammatory cells and exfoliation over weeks.[25] HGM adds second harmonic generation (SHG) and third harmonic generation (THG) contrast, showing epidermal vacuoles, shockwave-associated concentric THG patterns and loss of SHG signal in papillary dermis beneath epidermal vacuoles, supporting the concept that superficial LIOB may be coupled to deeper collagen remodeling.[31] RCM has also been used to evaluate treatment effects in photoaged skin.[32]

Melanin-dependent imaging data are particularly important for skin-type-specific safety. A 755 nm picosecond-domain study using dermoscopy, line-field confocal OCT, conventional OCT, *ex vivo* confocal microscopy and histology demonstrated that vacuole formation varied by pigmentation level. Darker porcine skin showed more superficial epidermal vacuoles, whereas medium-pigmented samples showed vacuoles near or below the dermal-epidermal junction, and light-pigmented samples showed no vacuoles under the tested conditions.[26] These observations reinforce that "same parameter" does not mean "same tissue endpoint" across skin types. Porcine *in vivo* work has additionally characterized microlesion healing dynamics after 1064 nm picosecond-domain treatment.[33] The core mechanistic, optical-delivery and imaging evidence is summarized in Table 2.

Table 2. Core mechanistic, optical-delivery and imaging evidence.

Evidence domain	Representative references	Main contribution	Interpretive caution
LIOB/controlled microinjury	14-18	Defines optical breakdown, cavitation and skin-type dependence of LIOB thresholds	Physics and ex vivo findings should not be overgeneralized to all clinical settings
Optical delivery systems	19-24	Compares MLA, DLA, DOE and holographic beam-splitting systems	Device-specific results are not interchangeable
OCT/RCM/MPM/HGM	25,26,29-32	Visualizes vacuoles, thresholds, shockwave patterns and collagen-related optical changes	Small samples and experimental sites predominate
Histology and healing kinetics	27,28,33	Shows dose-dependent epidermal and dermal vacuoles and microlesion healing dynamics	Animal, *ex vivo* and human data must be separated

LIOB, laser-induced optical breakdown; MLA, micro-lens array; DLA, diffractive lens array; DOE, diffractive optical element; OCT, optical coherence tomography; RCM, reflectance confocal microscopy; MPM, multiphoton microscopy; HGM, harmonic generation microscopy.

Molecular repair pathways and wound-healing biology

The molecular evidence base is emerging and remains less mature than the histologic evidence. The most directly relevant study used a melanocyte-containing human 3D skin model exposed to DOE-assisted fractional 1064 nm Nd:YAG picosecond irradiation at 0.2 J/cm^2. Histology showed intraepidermal vacuoles with intact surrounding tissue immediately after irradiation and at 24 hours. Next-generation sequencing demonstrated upregulation of MMPs, collagens, HSPs, cytokines and chemokines, supporting the interpretation that LIOB initiates repair and remodeling programs rather than causing simple destructive injury.[34]

Topical dexpanthenol in the same model accelerated visible repair, with vacuoles no longer visible after 24 hours, while initial laser-induced stimulation was maintained.[34] This is a key translational finding because it suggests that post-procedural barrier-supportive care may accelerate recovery without necessarily suppressing the desired remodeling signal. It should nevertheless be interpreted cautiously: the model is standardized and mechanistically informative, but it is not equivalent to human facial skin after multi-session clinical treatment.

The translational interpretation of these experimental findings requires caution. A standardized melanocyte-containing three-dimensional skin model provides valuable mechanistic insight into LIOB-induced vacuolization and repair-associated gene expression, but it does not fully reproduce the clinical microenvironment of intact human skin. In particular, it lacks the complete barrier architecture, pilosebaceous and sweat gland function, native vascular dynamics, neuroimmune responses and patient-specific inflammatory reactivity that influence post-procedural recovery. Therefore, accelerated vacuole resolution *in vitro* should not be equated with an automatically superior clinical aftercare strategy. In darker skin phototypes, especially Fitzpatrick III to VI, the dominant practical objective after fractional picosecond treatment is often not only rapid morphologic repair, but also tight control of transient inflammation to reduce the risk of post-inflammatory hyperpigmentation (PIH). Accordingly, dexpanthenol-supported repair can be discussed as a biologically plausible and experimentally supported approach, but clinical recommendations should remain conservative and prioritize barrier protection, bland moisturization, strict photoprotection, avoidance of irritants and early recognition of exaggerated inflammatory responses.

Additional molecular evidence comes from animal and clinical-adjacent studies. A 2026 porcine study reported pigment reduction, macrophage-associated melanin clearance, reduced tyrosinase expression, increased collagen I and III by day 30, and changes in transforming growth factor (TGF)-β1, growth differentiation factor (GDF)11, filaggrin and claudins after picosecond irradiation.[35] A recent photoaging model reported molecular changes involving collagen synthesis, MMP-9, TGF-beta/Smad, barrier-related proteins and inflammatory signaling after consecutive 755 nm picosecond DLA treatments.[36] Immunohistochemical and clinical work in wrinkled skin adds further evidence that picosecond treatment can be associated with tissue remodeling markers.[37]

Comparator literature from ablative fractional lasers is use-

ful but should not be overextended. Transcriptomic studies after ablative fractional injury and 3D organotypic skin models after fractional Er:YAG treatment show broad inflammatory, barrier and remodeling programs.[38,39] Earlier work on fractional photothermolysis and Hsp70 expression provides a useful framework for understanding how fractional laser microinjury can trigger repair biology.[40-42] However, these sources involve predominantly thermal or ablative mechanisms and cannot be cited as direct proof of LIOB-specific molecular signaling. Similarly, mechanotransduction pathways such as YAP/TAZ and Piezo signaling are biologically plausible links between photomechanical stress and extracellular matrix remodeling, but direct PubMed-indexed evidence connecting fractional picosecond LIOB to YAP/TAZ activation in human skin is currently lacking. These pathways are best presented as future research targets rather than established mechanisms.

Clinical endpoints and applications

Clinical evidence is strongest for atrophic acne scars and photorejuvenation endpoints. Early work with 755 nm picosecond lasers and specialized optics showed improvement in facial acne scarring, supporting the transition of picosecond devices from pigment removal to remodeling indications.[5] Subsequent studies demonstrated efficacy for wrinkles, non-ablative rejuvenation and photoaging in Asian cohorts, including long-term follow-up after 755 nm alexandrite DLA treatment.[6-8,43] Fractional 1064 nm picosecond treatment with diffractive optics has also shown clinical improvement in wrinkles and acne scars.[9] For atrophic acne scars, meta-analytic evidence suggests that fractional picosecond lasers may provide clinical improvement broadly comparable to other fractional laser modalities, with potentially lower pain and PIH risk in susceptible patients, although pinpoint bleeding may occur more often.[10] Randomized split-face studies comparing 1064 nm picosecond MLA or holographic systems with fractional Er:YAG or fractional CO_2 lasers provide a clinically important message: fractional picosecond treatment may offer favorable tolerability and downtime, but it should not be assumed to outperform ablative fractional systems in all scar phenotypes.[11,12]

Emerging evidence extends beyond acne scars. Fractional 1064 nm picosecond treatment has been evaluated for enlarged pores, Asian skin rejuvenation, non-acne atrophic scars and scar erythema, and postmastectomy scars.[44-47] A broader systematic review has also examined laser treatment across atrophic, hypertrophic and keloid scars.[48] A 2025 split-face study comparing fractional 1064 nm picosecond Nd:YAG with fractional Q-switched 1064-nm Nd:YAG for photoaging found improvement with both modalities and no significant difference in several clinical outcomes, but higher pain on the picosecond side.[49] These findings support clinical utility but also argue against simplistic claims that picosecond LIOB is universally superior to other fractional or photoacoustic approaches. The clinical evidence and translational endpoints are summarized in Table 3.

Table 3. Clinical evidence and translational endpoints.

Indication	Representative references	Typical endpoints	Clinical interpretation
Atrophic acne scars	5,10-12,22,51	Scar scales, physician assessment, patient satisfaction, downtime, pain, PIH	Most mature clinical indication; picosecond devices may reduce pain/PIH but are not always superior to ablative fractional lasers
Photoaging, wrinkles, pores, texture	6-9,13,32,43-45,49	Wrinkles, pores, texture, VISIA metrics, RCM/histology, satisfaction	Growing evidence across Asian cohorts and split-face designs
Non-acne and postsurgical scars	46-48	mVSS, scar roughness, erythema, patient satisfaction	Promising but heterogeneous and indication-specific
Pigmentary disorders and melasma	52-55	MASI, pigmentation indices, VISIA, PIH, recurrence	Relevant to safety and skin-of-color discussion; not the main remodeling endpoint

PIH, post-inflammatory hyperpigmentation; RCM, reflectance confocal microscopy; mVSS, modified Vancouver Scar Scale; MASI, Melasma Area and Severity Index.

Safety and skin-type considerations

Safety interpretation must be skin-type specific. The LIOB threshold is influenced by melanin content, and epidermal melanin can shift the balance between desired focal vacuolization and excessive superficial pigment-related injury.[15,26,29] In Asian and Fitzpatrick III-IV cohorts, fractional picosecond studies generally report transient erythema, mild pain, edema, pinpoint bleeding, crusting or scaling as common short-term events, with PIH rates varying by indication, parameter selection and aftercare.[7-13,44,45,50-55]

The emerging literature in Fitzpatrick V-VI is small but important. A report on 755 nm picosecond laser with DLA for acne scars in Fitzpatrick V and VI provides targeted evidence for darker skin types, an area often underrepresented in laser trials.[51] A broader systematic review of PIH prevention in skin of color emphasizes the need for conservative endpoints, pigment-risk assessment, strict photoprotection and avoidance of unnecessary inflammation.[56] These principles should be integrated into fractional picosecond practice, particularly for melasma-prone patients and those with a history of PIH.

Melasma and pigmentary disorders remain complex because the same photomechanical properties that allow pigment disruption may also provoke inflammatory relapse or PIH. Trials in Asian melasma cohorts using picosecond alexandrite DLA and other picosecond platforms suggest potential benefit with generally tolerable adverse-event profiles, but recurrence, heterogeneity of protocols and combination regimens limit generalizability.[52-55] In a review centered on LIOB and remodeling, pigmentary disorders should therefore be discussed as secondary safety-relevant indications rather than as the primary clinical endpoint.

Post-procedural care after fractional picosecond LIOB

Evidence-based aftercare after fractional picosecond LIOB remains underdeveloped. The available literature supports a conservative framework rather than a product-specific algorithm. The mechanistic rationale is straightforward: focal epidermal or dermal microinjury, transient inflammation, barrier stress and melanin-dependent PIH risk require short-term protection while the desired repair cascade proceeds. Direct post-LIOB data are limited, with the strongest mechanistic support coming from the dexpanthenol-treated 3D skin model.[34]

Post-laser barrier care can be informed by adjacent evidence. A randomized trial after fractional ablative CO_2 resurfacing found accelerated wound healing with a dexpanthenol-containing ointment, and a review of dexpanthenol supports its role in postprocedure wound healing.[57,58] Sunscreen data are directly relevant to picosecond treatment: in a randomized double-blinded split-face study in phototypes III-IV after picosecond laser, sunscreen containing anti-inflammatory ingredients showed a tendency toward reduced post-laser pigmentation and also improved acne-related parameters.[50] Evidence from 532-nm Q-switched treatment of solar lentigines further supports structured assessment and prevention of post-inflammatory hyperpigmentation after pigment-targeting laser procedures.[59] General expert recommendations support bland, barrier-oriented skincare around dermatologic procedures.[60] Epidermal growth factor (EGF)-containing topical products and recombinant EGF ointment have also been studied after laser procedures, but most evidence derives from ablative or non-picosecond contexts.[61,62]

A practical post-procedural approach after fractional picosecond LIOB should therefore emphasize brief post-treatment cooling for comfort, when clinically appropriate, gentle cleansing, bland moisturization or barrier-supportive repair, strict broad-spectrum photoprotection, avoidance of retinoids, acids, scrubs and other irritants during early recovery, and early reassessment if pain, persistent crusting, blistering, erosions or hyperpigmentation occur. In patients with darker phototypes, melasma tendency or prior PIH, pre-treatment counseling, conservative fluence escalation, test spots where appropriate and reinforced photoprotection are clinically prudent. Any claim that a specific active topical enhances LIOB-induced remodeling should be framed as preliminary unless supported by direct controlled data. Post-procedural care and safety principles are summarized in Table 4.

Practical synthesis for clinical reporting and research

The literature indicates that fractional picosecond treatment should be reported with greater technical precision than many existing clinical papers provide. Minimum reporting should include device type, wavelength, pulse duration, spot size, pulse energy, fluence, optical delivery

Table 4. Post-procedural care and safety principles.

Care domain	Evidence base	Practical principle	Level of certainty
Barrier support	34,57,58,60	Gentle cleansing, bland moisturization and barrier repair during early recovery	Moderate rationale, limited direct post-LIOB clinical data
Photoprotection and PIH prevention	45,50,56,59	Strict broad-spectrum sunscreen, UV/visible-light avoidance and anti-inflammatory photoprotection where appropriate	Strong clinical rationale, especially in Fitzpatrick III-VI
Active topicals	61,62	EGF or other actives should be considered investigational or adjunctive unless supported by indication-specific trials	Limited and mostly non-picosecond evidence
Abnormal healing recognition	48,56	Early review for persistent pain, erosions, blisters, prolonged crusting or worsening pigmentation	Prudent safety recommendation based on laser-complication principles

LIOB, laser-induced optical breakdown; PIH, post-inflammatory hyperpigmentation; EGF, epidermal growth factor.

system, focal setting, repetition rate, number of passes, cooling, interval between sessions, total number of sessions, skin phototype, anatomical site, clinical endpoint and post-procedural regimen. Without these details, results cannot be compared across platforms and the term "fractional picosecond laser" becomes too broad for meaningful interpretation.

From a translational perspective, the most robust model is not that LIOB alone explains all observed clinical improvement. A safer interpretation is that fractional picosecond systems produce a spectrum of tissue effects in which LIOB/LIC, photoacoustic shockwaves, pigment interactions and limited photothermal modulation vary by wavelength and optical delivery. Clinical outcomes may result from a combination of epidermal renewal, dermal remodeling, pigment clearance, vascular or inflammatory modulation and barrier recovery. The relative contribution of each mechanism remains a key research question.

Limitations

This review has the limitations of a structured narrative synthesis. The literature is heterogeneous in devices, wavelengths, optics, pulse durations, fluence reporting, treatment intervals, patient populations, endpoints and follow-up duration. Preclinical, *ex vivo*, porcine, murine, 3D skin model and human clinical data cannot be merged into a single evidentiary tier. Many studies are small, retrospective, split-face or pilot in design. Histologic and imaging endpoints are often obtained in non-facial or experimental sites, whereas clinical use commonly involves the face. Aftercare studies frequently involve ablative or non-picosecond laser contexts, limiting direct transferability. Finally, emerging molecular pathways such as YAP/TAZ-mediated mechanotransduction remain hypothesis-generating rather than LIOB-proven.

These data support dexpanthenol as a biologically plausible adjunct for post-LIOB repair, but they should not be overextended into a universal clinical protocol. *In vitro* vacuole resolution represents only one dimension of recovery. In clinical practice, particularly in Fitzpatrick III to VI skin, post-procedural care must also address erythema, inflammatory persistence, barrier fragility and PIH risk. The translational challenge is therefore to support repair without amplifying irritation or suppressing the controlled remodeling response that fractional picosecond LIOB is intended to induce.

Future directions

Future studies should use standardized parameter reporting and combine clinical outcomes with OCT, RCM, MPM, HGM or histologic validation. Trials should stratify by Fitzpatrick phototype, melanin index and indication, and should distinguish 532 nm, 755 nm, 785 nm and 1064 nm platforms as well as MLA, DLA, DOE and holographic optics. Molecular studies in human skin are needed to test whether LIOB induces reproducible signa-

tures involving MMP regulation, collagen I/III synthesis, HSP expression, cytokines, chemokines, barrier proteins and mechanotransduction pathways. Post-procedural care should be studied in randomized designs comparing bland barrier repair, dexpanthenol, sunscreen strategies, anti-inflammatory photoprotection and other topical regimens using both visible recovery and objective barrier or pigment endpoints.

Conclusions

Fractional picosecond LIOB represents a controlled photomechanical microinjury model with growing relevance in dermatologic laser medicine. Current evidence supports visible and histologic vacuolization, melanin-dependent tissue thresholds, non-invasive imaging correlates and emerging molecular repair signals. Clinical data are strongest for acne scars, photoaging, enlarged pores and selected scars, with increasing but still incomplete safety evidence in darker skin types. Post-procedural care remains a major gap and should currently be based on conservative barrier support, strict photoprotection and early recognition of abnormal wound-healing responses. The next phase of research should connect parameter-specific LIOB formation to molecular repair signatures, validated clinical endpoints and standardized aftercare protocols.

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