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Review Article

Comparative Analysis of fractional laser resurfacing versus Standard Care in acne vulgaris

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Abstract

Background: Atrophic scarring and persistent inflammatory lesions remain the most disfiguring long-term consequences of acne vulgaris, and standard pharmacologic care, topical retinoids, benzoyl peroxide, oral antibiotics, and isotretinoin, treats active disease more reliably than it reverses established dermal damage.

Objective: This review compares fractional laser resurfacing with standard pharmacologic care for acne vulgaris and post-acne scarring, evaluating efficacy for active lesions, efficacy for atrophic scarring, safety, and the role of combined regimens.

Methods: We synthesized randomized and split-face trials, prospective cohorts, and consensus guidelines addressing ablative and non-ablative fractional laser resurfacing, topical and systemic acne pharmacotherapy, and comparative or combination protocols in acne vulgaris and acne scarring.

Findings: Standard regimens remain first-line for controlling lesion counts and preventing new scar formation, while fractional resurfacing produces durable improvement in rolling and shallow boxcar scars that pharmacotherapy alone rarely achieves; non-ablative erbium-glass devices offer shorter recovery than ablative carbon dioxide systems with comparable improvement, and sequencing acne control before staged resurfacing outperforms either modality alone.

Conclusions: Fractional laser resurfacing and standard care are complementary rather than competing strategies; a sequential protocol, disease control first, staged resurfacing once inflammation is quiescent, offers the most consistent combination of safety and long-term cosmetic outcome.

Keywords: acne vulgaris; fractional laser resurfacing; atrophic scarring; isotretinoin; skin resurfacing

1. Introduction

Acne vulgaris affects the large majority of adolescents and a substantial proportion of adults, and its burden extends well beyond the active lesion count that most epidemiologic surveys report [1,2]. Comedones, papules, pustules, and nodulocystic lesions resolve, with or without treatment, over weeks to months, but a meaningful fraction of patients are left with permanent atrophic scarring that neither resolves spontaneously nor responds reliably to the pharmacologic regimens that controlled the original inflammation [3,4]. Standard care, topical retinoids, benzoyl peroxide, topical and oral antibiotics, hormonal therapy, and oral isotretinoin for severe or scarring-prone disease, remains first-line in essentially every published guideline because it addresses the follicular hyperkeratinization, sebum excess, *Cutibacterium acnes* proliferation, and inflammation that drive new lesions [5,6,7]. What it does not reliably do is reverse dermal collagen loss already sustained, which is why atrophic scarring has become a distinct treatment target with its own evidence base [8,9].

Fractional laser resurfacing, introduced in the mid-2000s, offered a mechanism suited to that separate problem: microscopic columns of thermal injury, spaced across the treated area while leaving intervening tissue intact, stimulate collagen remodeling without the downtime of traditional fully ablative resurfacing [10,11]. This review compares fractional laser resurfacing with standard pharmacologic care across the two distinct problems acne vulgaris presents, active inflammatory disease and established atrophic scarring, and proposes where each approach, or their sequential combination, is best positioned in an evidence-based pathway.

2. Pathophysiology of acne and the origin of scarring

Acne vulgaris begins with androgen-driven sebaceous hyperactivity and abnormal follicular desquamation, producing a microcomedone that becomes a substrate for *C. acnes* proliferation and a cascade of inflammatory signaling [12,13]. The inflammatory infiltrate that follows releases matrix metalloproteinases that degrade dermal collagen and elastin around the follicular unit; when degradation outpaces repair, the healing lesion resolves as an atrophic scar rather than restoring normal architecture [13,12]. Scar morphology varies with the depth and pattern of this loss: icepick scars are narrow and deep, boxcar scars have sharply demarcated walls, and rolling scars reflect dermal tethering producing a broad, shallow undulation, a distinction that matters because the three morphologies respond differently to resurfacing [4,14]. Fabbrocini and colleagues' synthesis of scar pathogenesis found that scarring risk correlates with lesion severity and delay to effective treatment, arguing for early pharmacologic control as the single most effective scar-prevention strategy [8]. Once scarring is established, the collagen deficit is structural rather than inflammatory, and pharmacotherapy and resurfacing begin to serve genuinely different purposes [9,15].

3. Standard pharmacologic care for active acne vulgaris

Topical retinoids remain the backbone of standard care for comedonal and mild inflammatory acne, and consensus guidelines recommend a retinoid, alone or with benzoyl peroxide or a topical antimicrobial, as first-line therapy for nearly all severities [5,6]. Benzoyl peroxide contributes rapid, resistance-independent antimicrobial activity and is recommended combined with retinoids or antibiotics specifically to limit resistant strains, a concern central to the most recent Global Alliance consensus [7,16]. Oral antibiotics add systemic coverage for moderate to severe disease but are recommended only for limited duration and always combined with a topical agent [6,17]. Oral isotretinoin remains the only therapy producing durable remission in severe nodulocystic or scarring-prone acne, and current guidance increasingly favors earlier use specifically to prevent scarring [18,6]. This armamentarium controls the

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inflammatory process that produces new lesions and new scars, but none of these agents meaningfully improves scars already present, the gap fractional resurfacing was developed to address [9,19].

Table 1: Standard pharmacologic agents for active acne vulgaris, principal mechanism, and guideline-recommended role [5,6,7,18].

Agent	Principal mechanism	Guideline-recommended role
Topical retinoids	Normalize follicular desquamation; anti-inflammatory	First-line, nearly all severities
Benzoyl peroxide	Resistance-independent antimicrobial	Combined with retinoid/antibiotic
Oral antibiotics	Systemic antimicrobial and anti-inflammatory	Moderate-severe disease, limited duration
Oral isotretinoin	Targets all four pathogenic mechanisms	Severe or scarring-prone disease

4. Mechanism of fractional laser resurfacing

Fractional photothermolysis, as described by Manstein and colleagues, creates an array of microscopic thermal injury zones separated by intact tissue columns, triggering epidermal regeneration and dermal neocollagenesis while sparing patients the prolonged recovery of fully ablative resurfacing [10]. Ablative systems, typically carbon dioxide (10,600 nm) or erbium:YAG, vaporize tissue within each microthermal zone and produce the most pronounced immediate change at the cost of several days of visible downtime; non-ablative systems, typically erbium-glass near 1,540-1,550 nm, heat tissue without vaporizing the surface and allow return to normal activity within 24-48 hours [11,20]. Histologic studies confirm both classes produce new collagen deposition beyond the treatment zone, with ablative systems generating deeper thermal columns per pass [11,21]. This distinction, structural remodeling versus biochemical control of the inflammatory cycle, is the basis for treating the two approaches as complementary rather than substitutable [22].

5. Comparative efficacy for active inflammatory acne

Direct comparative evidence for active inflammatory acne, as opposed to scarring, is limited, because fractional resurfacing trials generally enroll patients with quiescent disease to avoid confounding thermal response with ongoing inflammation [22,23]. Where fractional laser has been studied during active disease, non-ablative treatment has shown modest reductions in lesion counts, plausibly through localized anti-inflammatory thermal effects, but the magnitude falls well short of what retinoid and benzoyl peroxide combinations, or isotretinoin in severe disease, reliably achieve [20,24]. Guidance is explicit that fractional resurfacing is not a substitute for pharmacologic control of active disease and should be deferred until it is quiescent, both to avoid post-inflammatory hyperpigmentation and to ensure the scar burden being treated is stable [6,25].

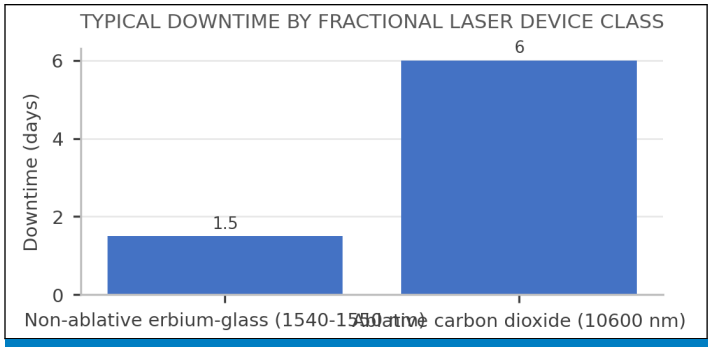

Figure 1: Typical downtime, in days to return to normal activity, for ablative carbon dioxide (10,600 nm) versus non-ablative erbium-glass (1,540-1,550 nm) fractional resurfacing, reflecting the recovery-course tradeoff described across comparative series [11,20,29].

6. Comparative efficacy for atrophic scarring, and device selection

For established scarring, the picture reverses. Chapas and colleagues' series of ablative fractional carbon dioxide resurfacing for acneiform scarring demonstrated significant, blinded-observer-confirmed improvement, an outcome standard pharmacotherapy cannot replicate [26]. Walgrave and colleagues found comparable durable improvement with a non-ablative device, trading shorter recovery per session for typically more sessions overall [27]. Alster and colleagues' early series established fractional photothermolysis as effective for atrophic scars

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broadly [28], and subsequent split-face comparisons have generally found roughly comparable efficacy between ablative and non-ablative platforms, with ablative devices faster per session and non-ablative devices trading a slower course for less downtime and lower complication risk in darker phototypes [29,30].

Table 2: Ablative versus non-ablative fractional laser resurfacing for acne scarring, comparative profile [29,30,33,25].

Feature	Ablative (e.g., CO2)	Non-ablative (e.g., erbium-glass)
Efficacy per session	Faster, more pronounced	Slower, cumulative
Downtime	Several days	24-48 hours
Darker phototype risk	Higher pigmentary risk	Lower pigmentary risk
Typical sessions needed	Fewer	More

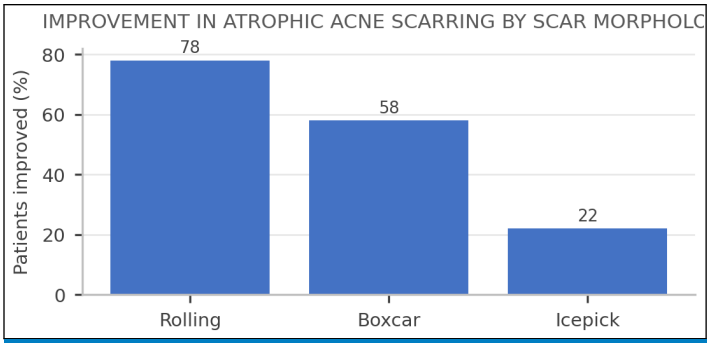

Figure 2: Proportion of patients achieving clinically significant improvement in atrophic acne scarring after a standard fractional laser treatment course, by scar morphology, drawn from split-face and randomized comparative series [23,29,26,27].

Scar morphology predicts response. Sardana and colleagues found rolling scars respond most favorably to non-ablative fractional therapy, while deep icepick scars respond poorly to fractional resurfacing of any type and are better addressed by focal excisional techniques first [23]. Boxcar scars are intermediate, often requiring combination with subcision or filler [14,4]. Nirmal and colleagues' randomized comparison of erbium-doped 1,550 nm laser against microneedling found broadly comparable improvement, the laser arm faster and microneedling far cheaper, relevant where laser access is constrained [31], a conclusion echoed by Cachafeiro and colleagues [32]. Device choice also interacts with phototype: Chan's review of Asian skin found higher melanin content raises hyperpigmentation risk with ablative treatment specifically, favoring conservative settings or non-ablative platforms [33]; Isarasak and colleagues found ablative fractional treatment remained effective in Asian skin with conservative parameters and diligent photoprotection [34]; and Manuskiatti and colleagues' direct comparison found erbium:YAG produced less erythema and faster recovery than CO2 at comparable efficacy, a tradeoff informing device selection more than any single efficacy metric [30,35].

7. Safety, tolerability, and adverse effects

The adverse-effect profiles differ in kind. Topical retinoids and benzoyl peroxide cause dryness, irritation, and photosensitivity that are self-limited and dose-adjustable; oral antibiotics carry gastrointestinal and resistance-related risks; isotretinoin requires monitoring for mucocutaneous, lipid, and hepatic effects and strict pregnancy prevention, but none carries meaningful risk of permanent textural or pigmentary change [18,17]. Fractional resurfacing, a procedural intervention, carries a genuine if generally transient risk of complications. Metelitsa and Alster identified post-inflammatory hyperpigmentation as the most common adverse event, more frequent in darker phototypes and with aggressive ablative settings, alongside less common infection, excessive-density scarring, and rare acneiform or herpetic eruptions [25]. Setyadi and colleagues similarly found most events transient, resolving within one to two weeks, though a minority experienced prolonged erythema or pigmentary change [36]. Sherling and colleagues' consensus recommendations for 1,550 nm resurfacing emphasized patient selection and staged escalation of density as the principal safeguards [20].

8. Combination and sequential treatment strategies

The most consistent theme across the literature is that the two approaches perform best in sequence. Bencini and

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colleagues' documentation of non-ablative treatment for acne scars enrolled patients whose active acne had already been controlled pharmacologically, reflecting standard practice of deferring resurfacing until quiescence [37]. Ong and Bashir's review similarly frames the evidence base as implicitly assuming prior disease control, since essentially every major trial excluded patients with active moderate to severe disease at enrollment [22]. Seo and colleagues' work on microneedle fractional radiofrequency illustrates the same combination logic extending to adjacent energy-based technologies [24]. Connolly and colleagues' review recommends a staged approach, disease control first, morphology-specific technique selection second, resurfacing or adjunctive procedures third, as the framework most consistently supported by the literature [15,19].

9. Discussion

The comparative literature does not support fractional resurfacing as a replacement for pharmacologic care, nor pharmacologic care as adequate treatment for established scarring. The two approaches address different targets, ongoing inflammation that drives new lesions and scar risk, and structural collagen deficit left by lesions already resolved, and the strongest outcomes consistently come from sequencing rather than substitution [8,15]. Device selection within resurfacing should be individualized by morphology, ablative platforms for patients tolerating longer recovery for faster change, non-ablative platforms for minimal downtime or darker phototypes [29,33,30]. Icepick scars generally require a different, more focal technique first, underscoring that no single technology is universally optimal across the heterogeneous morphologies grouped under acne scarring [23,14].

10. Conclusion

Standard pharmacologic care remains the correct first-line approach for active acne vulgaris and the most effective available strategy for preventing new scar formation, while fractional laser resurfacing offers a mechanistically distinct, genuinely effective option for atrophic scarring that pharmacotherapy does not reverse [6,10]. The evidence favors a sequential model: guideline-concordant control of active disease first, followed by morphology-specific fractional resurfacing, ablative or non-ablative according to patient priorities and phototype, once inflammation has been quiescent long enough that scarring can be reliably assessed [7,22]. Continued refinement of device parameters, phototype-specific protocols, and combination regimens is likely to further narrow the gap between fractional resurfacing's real but incomplete efficacy and the durable outcome patients ultimately seek [31,32].

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