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Research Progress on the Pathogenesis, Immune and Inflammatory Mechanisms and Treatment of Acne

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Abstract: ***Acne is a chronic inflammatory skin disease involving the pilosebaceous unit. Based on studies published from 2021 to 2025, this review summarizes recent progress in acne pathogenesis, immune inflammation, pharmacological treatment, and light- and energy-based therapies. It also briefly reviews the pathogenesis and external therapies of traditional Chinese medicine (TCM). Recent evidence suggests that excessive sebum production, altered lipid composition, follicular keratinization, skin barrier dysfunction, microbial dysbiosis, and immune responses promote one another. The strain types, biofilms, and metabolites of Cutibacterium acnes may better explain differences in inflammatory activity than the total bacterial load. TLR/NF-κB signaling, the NLRP3 inflammasome, neutrophil extracellular traps, and Th1/Th17 responses all contribute to acne inflammation. Topical retinoids, benzoyl peroxide, and appropriate antimicrobial therapy remain the main treatments. Oral isotretinoin may be considered for severe acne or patients at high risk of scarring. The 1726 nm laser, intense pulsed light, and photodynamic therapy are mainly used for active acne, whereas fractional lasers, microneedle radiofrequency, and fractional picosecond lasers are mainly used for atrophic scars. Treatment should be selected according to lesion type, disease severity, scarring risk, and patient tolerance.***

Keywords: Acne, Skin microbiome, Immune inflammation, Retinoids, Light- and energy-based therapy.

1. Introduction

Acne mainly affects adolescents but may persist into adulthood. Its lesions include comedones, papules, pustules, nodules, and cysts, and it may leave erythema, pigmentation, or scars. Traditional theories emphasize increased sebum production, abnormal follicular keratinization, C. acnes colonization, and inflammation. Studies from the past five years have further clarified the links among lipid composition, bacterial strain differences, the skin barrier, and immune pathways. This review summarizes these findings to provide a reference for the comprehensive management of acne.

2. New Insights into the Pathogenesis of Acne

2.1 Sebum, Follicular Keratinization, and the Skin Barrier

Acne is not caused by a single factor. It results from the combined effects of genetic background, hormones, sebum, keratinization, microorganisms, and immune responses. Androgens promote sebaceous gland growth and sebum production. Sebum retention and abnormal shedding of keratinocytes at the follicular opening contribute to microcomedone formation. Abnormal lipids and bacterial products then stimulate keratinocytes and immune cells, allowing keratinization, microbial colonization, and inflammation to reinforce one another [1]. These processes therefore do not occur in a fixed order, but form a positive cycle during disease progression. In addition to the amount of sebum, its composition and oxidation status are also important. Free fatty acids, squalene, and oxidized lipids may promote oxidative stress, abnormal keratinization, and inflammation, while altering the growth environment of skin microorganisms [2]. Thus, reducing sebum alone cannot fully prevent acne, and treatment should also address keratinization and inflammation. Patients with acne often show increased transepidermal water loss, altered stratum corneum lipids, dryness, and stinging, which indicate skin barrier dysfunction [3]. Retinoids, benzoyl peroxide, and excessive cleansing may further increase irritation. Gentle cleansing, moisturization, and sun protection should therefore be included to improve treatment tolerance and adherence.

2.2 Bacterial Strain Types, Biofilms, and Microbial Dysbiosis

C. acnes is a normal resident of the skin and can be detected in both healthy individuals and patients with acne. Acne therefore cannot be explained simply by an increase in bacterial numbers. Recent studies have focused more on microbial community structure and functional differences among bacterial strains. In inflammatory lesions, microbial diversity may decrease, whereas the proportion of inflammation-associated types such as IA1 may increase. Strains also differ in adhesion, biofilm formation, metabolite release, and tolerance to antimicrobial drugs [4-7]. Biofilms help bacteria persist in follicles and may make antimicrobial treatment less effective. Porphyrins produced by acne-associated strains may also induce potassium efflux from keratinocytes, activate the NLRP3 inflammasome, and promote IL-1β release [8]. These findings show that acne microbiome research has shifted from asking whether bacteria are present to examining strain structure, metabolic activity, and host responses. Future therapies may focus less on broadly removing resident bacteria and more on controlling inflammatory strains, biofilms, and microbial imbalance.

3. Immune and Inflammatory Mechanisms

Inflammation is involved in microcomedone formation, the development of papules and pustules, and the later

development of pigmentation and scarring. It is therefore not limited to visibly red or swollen lesions. Keratinocytes, sebocytes, macrophages, neutrophils, and T lymphocytes all contribute to the inflammatory network [9]. After C. acnes and its products are recognized by pattern-recognition receptors such as TLR2 and TLR4, the NF-κB and MAPK pathways may be activated, increasing the expression of IL-1β, IL-6, IL-8, and TNF-α. In vitro studies show that particulate matter can enhance C. acnes-induced TLR4/NF-κB signaling, suggesting that air pollution may aggravate local inflammation [10]. TLR2 stimulation may also promote inflammatory mediators and keratinization-related proteins through calcium-dependent and calcium-independent pathways [11]. This finding supports a close link between abnormal keratinization and immune inflammation. Early control of inflammation may therefore help prevent the progression of active lesions and reduce the risk of scarring.

The NLRP3 inflammasome has become an important focus of recent acne research. Once activated, it promotes the maturation and release of IL-1β through caspase-1 and amplifies local inflammation [12]. In addition to bacterial components, porphyrins produced by acne-associated strains may activate this pathway [8]. A 2024 study found that the NLRP3 inflammasome and neutrophil extracellular traps can promote each other and maintain an inflammatory cycle. In animal models, inhibiting extracellular trap formation reduced acne-like inflammation [13]. However, most current evidence comes from cell studies, animal models, and lesion tissues, and cannot yet be directly translated into routine drug treatment. Th1 and Th17 responses also help maintain inflammation. IL-17 promotes neutrophil recruitment, whereas IFN-γ contributes to cell-mediated immunity [4,9]. However, a placebo-controlled pilot trial found that anti-IL-17A treatment did not significantly reduce inflammatory lesions in moderate-to-severe acne. This suggests that common acne is driven by several pathways and may not respond sufficiently to the blockade of a single cytokine [14]. Current evidence also does not support the routine use of biologics for acne. Their possible role is mainly limited to rare severe cases, autoinflammatory syndromes, or cases overlapping with hidradenitis suppurativa [15]. In addition, extracellular vesicles released by C. acnes can deliver proteins, lipids, and other active substances to skin cells and activate MAPK and NF-κB signaling. Cannabidiol reduced this response through the CB2 receptor in vitro, but this evidence remains preclinical [16].

4. Advances in Pharmacological Treatment

4.1 Topical and Antimicrobial Therapies

The 2024 American Academy of Dermatology guidelines continue to recommend topical retinoids and benzoyl peroxide as basic treatments for acne [17]. Topical retinoids regulate follicular keratinization, reduce microcomedones, and provide anti-inflammatory effects. They can be used for both initial and maintenance treatment. Benzoyl peroxide reduces the C. acnes burden and has a low risk of resistance. When combined with a topical retinoid, it can improve both comedonal and inflammatory lesions. Dryness, peeling, and stinging are common during early treatment. Starting on alternate days or at a lower frequency, applying a small amount, and using a moisturizer may improve tolerance. Trifarotene is a selective topical retinoid. A randomized, double-blind, split-face study showed that 24 weeks of treatment reduced both active lesions and the number of atrophic scars [18]. This result suggests that early control of follicular keratinization and inflammation may help prevent scarring.

Topical antibiotics should not be used alone for long periods. They should be combined with benzoyl peroxide to reduce the selection of resistant strains [17]. Topical 4% minocycline foam delivers the drug directly to the pilosebaceous unit and results in lower systemic exposure than oral preparations. It has shown efficacy in moderate-to-severe inflammatory acne, but the treatment duration should still be limited and adjusted according to lesion changes [19]. Oral tetracyclines may be used for moderate-to-severe inflammatory acne, and doxycycline is commonly prescribed. Sarecycline is a narrow-spectrum tetracycline that can be dosed by body weight. Pharmacokinetic and clinical studies indicate that a high-fat meal may alter drug exposure but has little effect on its overall clinical efficacy [20]. Narrow-spectrum antibiotics may reduce effects on non-target microorganisms, but they do not replace appropriate antimicrobial stewardship. Oral antibiotics should be combined with a topical retinoid or benzoyl peroxide, should not duplicate topical antibiotic therapy, and should be discontinued once the disease is stable.

4.2 Isotretinoin and Antiandrogen Therapy

Oral isotretinoin reduces sebum production, improves follicular keratinization, and suppresses inflammation. It is an important option for severe acne, acne with a high risk of scarring, or disease that responds poorly to standard treatment [17]. A systematic review and meta-analysis showed that low-dose regimens can improve mild-to-moderate acne and reduce some dose-related adverse effects, such as cheilitis and dry skin. However, conventional doses may provide more durable remission [21]. Treatment should therefore not focus only on a low dose or a fixed cumulative dose. The dose and duration should be determined according to disease severity, body weight, relapse risk, tolerance, and laboratory findings. Women of childbearing potential must use strict contraception during treatment. Isotretinoin is contraindicated during pregnancy and in women planning pregnancy.

Antiandrogen therapy is suitable for some patients with hormonally related acne, especially adult women with recurrent lesions on the jawline or around the mouth, or with menstrual flares. Clascoterone is a topical androgen receptor antagonist that blocks androgen signaling in the pilosebaceous unit. Its systemic antiandrogen effect is relatively low, and it can be used in both male and female patients [22]. Spironolactone is mainly used in adult women. The multicenter phase III SAFA randomized controlled trial included 410 women and found that spironolactone was more effective than placebo, with greater improvement at 24 weeks than at 12 weeks [23]. This suggests that the drug requires time to take effect and may reduce the need for long-term oral antibiotics. Before treatment, clinicians should assess the possibility of pregnancy, underlying diseases, and concomitant medications. After inflammation is controlled, topical retinoids, benzoyl peroxide, or azelaic acid may be

continued as maintenance therapy. Skin barrier care may further reduce relapse and irritation [3,17].

5. Advances in Light- and Energy-Based Therapies

5.1 Active Acne

Light- and energy-based therapies may improve active acne by affecting sebaceous gland activity, bacterial porphyrins, and local inflammation. However, they are generally used as an addition to drug treatment rather than as the first choice for all patients. The 1726 nm laser has high absorption in sebum and can act relatively selectively on sebaceous glands. A prospective single-arm study of 104 patients with moderate-to-severe acne across different skin types showed that inflammatory lesions continued to improve for 26 weeks after three treatments, with no serious treatment-related adverse events [24]. Because the study lacked a control group, its long-term efficacy and optimal parameters still require confirmation in randomized trials. A small randomized controlled study found that intense pulsed light combined with minocycline was more effective than minocycline alone in reducing inflammatory lesions and disease severity, and it also improved erythema [25]. Photodynamic therapy uses a topical photosensitizer followed by irradiation at a specific wavelength. The resulting photochemical reaction affects sebaceous glands and inflammation. A 2025 systematic review of 82 studies involving 4, 340 patients found that photodynamic therapy generally improved acne. However, photosensitizers, light sources, energy settings, and treatment courses varied widely. Pain, erythema, crusting, and pigmentary changes were common, and no standard protocol has been established [26].

5.2 Acne Scars

Fractional CO_2 laser treatment creates microscopic thermal injury zones and promotes collagen remodeling. It is commonly used for atrophic acne scars. Microneedle radiofrequency delivers radiofrequency energy into the dermis and causes less thermal injury to the epidermis. A randomized split-face study found that a single fractional CO_2 laser treatment and microneedle radiofrequency produced similar improvements in scar texture. However, fractional CO_2 laser caused more erythema and barrier damage, whereas microneedle radiofrequency caused more pain during treatment [27]. Fractional picosecond lasers promote tissue remodeling mainly through photomechanical effects. A meta-analysis showed that their overall efficacy was similar to that of other fractional lasers, but the risks of pain and post-inflammatory hyperpigmentation may be lower [28]. A meta-analysis comparing fractional CO_2 and Er: YAG lasers suggested that fractional CO_2 laser may provide better overall efficacy, but it also causes more treatment-related pain and longer-lasting erythema [29]. Fractional CO_2 laser combined with a hyaluronic acid dressing may improve scar scores, crust recovery, and patient satisfaction, and may reduce some pigmentary changes. However, the overall quality of evidence is low to moderate [30]. Device and parameter selection should therefore consider scar type, skin color, pain tolerance, downtime, and the risk of post-inflammatory hyperpigmentation.

6. Traditional Chinese Medicine Perspectives and Treatment

Traditional Chinese medicine (TCM) commonly explains acne in terms of external pathogens, damp-heat, heat in the blood, and impaired movement of qi. The chapter Sheng Qi Tong Tian Lun in Su Wen states that “stagnation gives rise to acne.” Recent studies have further discussed the correspondence between facial regions and the zang-fu organs and meridians, the ascending and descending movement of qi, and the theory of regulating the Liver. Damp-heat in the Lung and Stomach, constrained Liver qi transforming into fire, and the combination of phlegm and blood stasis are considered possible causes [31-34]. Common TCM patterns include wind-heat in the Lung meridian, damp-heat in the Spleen and Stomach, phlegm-blood stasis, and disharmony of the Chong and Ren vessels. Treatment mainly aims to clear heat, resolve dampness, remove toxins, and disperse nodules. According to the pattern, treatment may also strengthen the Spleen, transform phlegm, activate blood, or regulate the Chong and Ren vessels [35]. Studies of the gut-skin axis suggest that Chinese herbal medicine may improve acne by regulating gut microorganisms, sebum production, and local immune responses, although direct clinical evidence remains limited [36]. Modified Xiaoyao San combined with pulsed dye laser may improve moderate-to-severe acne and regulate Th1/Th2-related factors, while collagen dressings may support skin barrier repair [37,38]. Current TCM studies often have small samples, inadequate control groups, and short follow-up periods. Their efficacy should therefore be interpreted with caution.

7. External Therapies of Traditional Chinese Medicine

External TCM therapies include medicinal washes, wet dressings, masks, fire needling, bloodletting, cupping, and auricular therapy [39]. Prescription data mining shows that commonly used herbs include Baizhi, Jinyinhua, Huangqin, Danshen, Dahuang, Lianqiao, and Huangbai. Their main functions are described as clearing heat and toxins, resolving dampness and swelling, and activating blood [40,41]. Basic research suggests that baicalein may reduce IL-1β, IL-6, and TNF-α by regulating the NLRP3 inflammasome. Tea-derived active compounds may also influence sebum production, abnormal keratinization, and local microorganisms [42,43]. These studies provide preliminary support for the mechanisms of external herbal therapy, but effective doses, formulation stability, and long-term safety require clinical confirmation. Clinicians should also monitor adverse reactions such as erythema, itching, and contact dermatitis.

Recent clinical studies have often used combined external therapies. Fine fire needling combined with the Quzhi Xiaocuo formula improved lesion color, number, type, swelling, and pain. A Chinese herbal cold wet dressing combined with fire needling also showed benefits for mild-to-moderate acne [44,45]. Auricular bloodletting at different frequencies combined with auricular acupressure, bloodletting with cupping, and fire needling combined with auricular acupressure have shown some effects in reducing inflammation and improving lesions [46-48]. However, most

studies were single-center trials with small samples. Blinding was difficult, and outcome measures and follow-up periods were inconsistent. Current evidence is therefore insufficient to compare the relative value of these methods, and well-designed randomized controlled trials are still needed.

8. Conclusion

The understanding of acne pathogenesis has moved beyond four separate processes—sebum production, keratinization, bacteria, and inflammation—to an integrated model involving lipid composition, the skin barrier, bacterial strain types, biofilms, and immune responses. Current treatment remains based on topical retinoids, benzoyl peroxide, appropriate antimicrobial therapy, and oral isotretinoin. Antiandrogen drugs and newer topical preparations provide additional options for individualized treatment. Light- and energy-based therapies should be selected differently for active acne and atrophic scars, with attention to skin color, recovery time, and adverse effects. TCM may be included as part of comprehensive treatment, but more rigorous studies are required. Future research should better connect mechanistic findings with clinical outcomes, use consistent efficacy measures, and give greater attention to relapse, scarring, pigmentation, and quality of life.

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