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DERMOCOSMETICS IN THE MANAGEMENT OF ACNE VULGARIS

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Abstract

Introduction:

Treating acne vulgaris remains a significant challenge for dermatologists. Dermocosmetics are becoming an important component of acne management. The active ingredients in dermocosmetics help support dermatological treatments, while gentle formulations help maintain the skin's lipid barrier, reduce transepidermal water loss, and minimize the risk of irritation. Our objective was to review the active ingredients and various preparations used in dermocosmetics for acne, and to emphasize the clinical evidence supporting their effectiveness.

Aim of study:

The aim of the study is to summarize the available knowledge about the active ingredients used in acne management. The epidemiology, etiology, of acne and and methods of treatment were summarized and described.

Materials and methods:

The literature available in PubMed database was reviewed using following keywords: “Acne vulgaris”, “Dermocosmetics”.

Conclusion:

Dermocosmetics are essential in managing acne, either as maintenance therapy or as a complement to pharmacological treatments. They contain various active ingredients and formulations that address critical acne pathways, including inflammation, abnormal keratinization, excessive sebum production, and C. acnes colonization. Furthermore, dermocosmetics help alleviate the side effects of dermatological treatments, such as compromised skin barriers, increased transepidermal water loss and irritation.

Key words: Acne Vulgaris; Dermocosmetics, active ingredients in dermocosmetics for acne

Introduction

Definition

Acne is a condition affecting the pilosebaceous unit, which consists of hair follicles connected to oil glands in the skin. Clinically, acne presents with excess oil production (seborrhoea), noninflammatory lesions (open and closed comedones), inflammatory lesions (papules and pustules), and varying degrees of scarring [1, 2]. It typically appears in areas with a high density of pilosebaceous units, such as the face, neck, upper chest, shoulders, and back.

Severe acne includes nodules and cysts, known as nodulocystic acne [3, 4]. Acne adversely impacts various aspects of health-related quality of life (HRQoL) for both adolescents and adults. It affects emotional and social functioning, relationships, leisure and daily activities, sleep, and performance at school or work [5].

Disease mechanisms

Four key processes contribute significantly to the formation of acne lesions: changes in the keratinisation process resulting in comedones; increased and modified sebum production influenced by androgens (or heightened sensitivity of androgen receptors); the release of inflammatory mediators into the skin; and the colonization of hair follicles by P. acnes [6]. The precise sequence of these events and their interactions with other factors are still not fully understood. Increasing evidence suggests that individuals with acne do not have a higher quantity of C. acnes in their follicles compared to those without acne, but they do have different strains of the bacteria. This has led to the hypothesis that acne may result from an imbalance in the C. acnes phylotypes within the skin microbiota, particularly the relative increase of the acne-associated phylotype IA1. In general, the loss of skin microbial diversity along with the activation of innate immunity are believed to be key factors driving this chronic inflammatory condition [7,8].

Epidemiology

The risk factors and genes related to acne prognosis and treatment remain uncertain. Twin studies have highlighted the significant role of genetic factors in severe, scarring acne. A study of 1002 Iranian 16-year-olds [9] found that having a family history of acne doubled the risk of developing significant acne and a large study of Chinese [10] undergraduates reported a 78% heritability rate of acne among first-degree relatives of those affected. Acne tends to appear earlier in girls, though more boys are affected during mid-adolescence [11]. Black children may develop acne at a younger age and it is often more comedonal compared to white children, likely due to earlier puberty onset [12].

Causes

Earlier observational studies indicated an inverse relationship between smoking and acne, more recent research shows that severe acne increases with smoking [13]. Increased insulin resistance and high serum dehydroepiandrosterone may explain acne in polycystic ovary syndrome [14]. Acne can worsen due to the occlusion of the skin surface with greasy products (pomade acne), clothing, and sweating. Certain drugs, such as anti-epileptics, typically cause monomorphic acne, and acneiform eruptions have been linked to anti-cancer drugs like gefitinib [15]. The use of anabolic steroids to increase muscle mass, which might be underestimated, can lead to severe acne forms [16]. Diet, sunlight, and skin hygiene have all been considered factors in acne [17]. One systematic review suggested that dairy products, especially milk, increase acne risk, though the included observational studies had significant limitations [18]. Previous studies that involved giving young people large amounts of chocolate to provoke acne were too small and too brief to draw definitive conclusions. A randomized controlled trial indicated that a low glycaemic load diet might improve acne, providing preliminary support for this theory [19].

Symptoms

Acne causes physical symptoms such as soreness, itching, and pain, but its primary impact is on quality of life. Psychological issues are significant and are exacerbated by several factors: acne affects visible skin, a crucial aspect of social interaction; societal and cultural pressures demand flawless skin; healthcare professionals often dismiss acne as a minor, self-limiting condition; and acne typically peaks during teenage years, a critical period for developing confidence and selfesteem [20]. In the UK, teenagers with acne were twice as likely to score in the borderline or abnormal range on an age-appropriate emotional wellbeing

questionnaire compared to those without acne, and they exhibited higher levels of behavioral difficulties [21]. A case-control study found that the presence of acne was associated with unemployment among young men and women [22]. However, a community study of 14- to 17-year-old Australian students found no link between acne and later psychological or psychiatric issues, a surprising result that may be due to effective treatments or personality traits [23].

Acne exposome factors

The acne exposome refers to the totality of environmental factors that affect the occurrence, duration, and severity of acne. These factors influence treatment response and relapse frequency by interacting with the skin barrier, sebaceous glands, innate immunity, and skin microbiota [24, 25]. The main categories of these factors are nutrition, medication, occupational factors (including cosmetics), pollutants, climatic factors, and psychological and lifestyle factors [26]. Currently, the primary food groups believed to potentially trigger acne include dairy products, especially skim milk, and high-glycaemic carbohydrates [27]. Nutritional supplements like whey proteins containing leucine, commonly used by athletes, might also provoke or exacerbate acne [28]. Evidence suggests that certain oral contraceptives, particularly first- and second-generation ones, can lead to metabolites of testosterone that worsen acne, especially in adolescent and adult females.

However, oral contraceptive pills containing chlormadinone acetate, dienogest, drospirenone, and norgestimate have been noted to have beneficial effects in acne treatment [29]. Anabolic steroids trigger acne by targeting androgen receptors on sebocytes and keratinocytes [30]. Various substances such as corticosteroids, halogens, isoniazid, lithium, vitamin B12, immunosuppressants, certain anti-cancer agents, and radiotherapy have been reported to cause acneiform eruptions [31]. Aggressive skincare routines and inappropriate cosmetics can exacerbate acne by altering the skin barrier and the balance of skin microbiota, particularly in the sebaceous areas, thereby activating innate immunity and causing inflammation. Mechanical factors such as rubbing, scrubbing, and the use of home or medical devices like sonic brushes, dermarollers, or microneedling systems can also trigger acne flare-ups [32]. Air pollutants can harm the skin by increasing oxidative stress, leading to significant disruptions in lipid, DNA, and/ or protein functions in the skin [33]. Tobacco and cannabis consumption may also contribute to acne as they act as pollutants that affect human health. Climatic conditions and seasonal changes, particularly combinations of heat, humidity, and intense UV radiation, may induce inflammatory acne flare-ups, a phenomenon known as acne tropicana, acne majorca, or tropical acne [34].

Dermocosmetics

Dermocosmetics are skincare products formulated with advanced, dermatologically active ingredients designed to directly address or alleviate symptoms of various skin conditions, beyond what a simple base product could achieve [35]. In the article "Dermocosmetics in dermatological practice. Recommendation of the Polish Dermatological Society part1." Barbara Zegarska, Lidia Rudnicka, et al. explain the differences between cosmetics and dermocosmetics. They note that historically, topical products for skin application were divided into cosmetics and drugs, according to the American definition adopted in the Federal Food, Drug, and Cosmetic Act of 1938. Cosmetics were defined as substances intended for "cleansing, caring for, beautifying, and improving the appearance of the skin." In contrast, drugs were defined as "articles intended to affect the structure or any function of the body or articles intended for use in diagnosing, treating, mitigating, curing, or preventing disease in humans". Another significant difference is that cosmetics do not require approval before being marketed. Regulations only specify the list of substances that can be used in cosmetics and their concentrations. On the other hand, to market a drug, its efficacy and safety must be proven through numerous clinical trials [36]. Dermocosmetics occupy an ambiguous area between

cosmetics and drugs. These products are designed with active ingredients intended to provide beneficial physiological effects through enhanced pharmacological action, but from a legal standpoint, they remain cosmetics [37]. The active cosmetic ingredients influence [38] these four pathogenic pathways: changes in the keratinisation process resulting in comedones; increased and modified sebum production influenced by androgens; the release of inflammatory mediators into the skin; and the colonization of hair follicles by P. acnes [6]. Increased and altered sebum production is a major factor in the development of acne, but only a few topical products have been proven to effectively target this abnormal sebum production [39]. Currently, masks and day creams that work on the skin's surface are used to absorb skin-surface lipids and reduce the appearance of oiliness [40]. Several active ingredients in dermocosmetics have demonstrated sebo-suppressive properties, and there is a growing interest in the use of topical antioxidants. Additionally, topical niacinamide has been shown to increase desquamation and potentially reduce sebum production. A study by Biedermann et al. [41] found that topical niacinamide had a dose-dependent sebosuppressive effect in a cell culture of human sebocytes. Niacinamide is also known to target inflammation. In individuals prone to acne, excess keratin causes dead skin cells to clog the hair follicle, leading to blocked pilosebaceous glands and the formation of microcomedones. It is now believed that inflammation precedes ductal hyperkeratinization, which may be caused by an increased rate of keratinocyte proliferation, reduced separation of ductal corneocytes, and increased cohesion between keratinocytes [1]. This theory supports the use of acidic formulations, such as acid peels, in acne scar therapy.

Alpha hydroxy acids

AHAs thin the stratum corneum, increase epidermal thickness, disperse basal layer melanin, and boost collagen synthesis within the dermis [42]. Glycolic acid peels, the most common type of AHA peel, target corneosomes by reducing their cohesiveness, promoting their breakdown, and causing desquamation [43]. Low concentrations of AHAs (5-10%) act on the skin's superficial layers by enhancing the healing response through subcorneal epidermolysis, opening comedones, and unroofing pustules. Consequently, many dermatologists believe that products containing AHAs should not be classified as cosmetics [44]. However, various studies have demonstrated the safety and efficacy of preparations combining glycolic acid and retinaldehyde (a form of vitamin A) in treating acne and post-inflammatory hyperpigmentation [45]. Furthermore, a recent trial showed that 10% glycolic acid monotherapy significantly improved mild acne compared to a placebo after 90 days of treatment [46].

Nicotinamide

Nicotinamide, also known as niacinamide, is a form of vitamin B3, an essential water-soluble nutrient found in various foods [47]. Topically applied nicotinamide not only has sebostatic effects but is also an effective anti-inflammatory agent. Several double-blind studies comparing nicotinamide gel to clindamycin gel in acne patients have demonstrated that nicotinamide significantly reduces inflammatory papules and acne lesions, showing comparable results to clindamycin gel [48,49]. Nicotinamide may also be effective in combination treatments to reduce inflammation. A pilot study using skin biopsies from 16 patients found that a combination of nicotinamide, retinol, and 7-dehydrocholesterol had an anti-inflammatory effect, lowering levels of pro-inflammatory molecules associated with acne [50]. Nicotinamide offers potent antiinflammatory properties without the risk of bacterial resistance or systemic side effects, making it a promising treatment option for acne vulgaris.

Zinc

Zinc, a divalent cation, is an essential micronutrient necessary for various bodily processes. It has been found to play a role in several skin disorders, including acne vulgaris. The benefit of zinc for acne was first identified in the 1970s when Fitzherbert (1977) observed improvements in acne among zinc-deficient patients with acrodermatitis enteropathica.

Subsequent research found that individuals with acne had significantly lower zinc levels compared to controls [51]. A small in vitro study investigated zinc's mechanism on inflammatory acne lesions and discovered that zinc possesses strong anti-inflammatory properties by inhibiting leukocyte chemotaxis [52]. Additionally, a recent in vitro study on zinc calx, a mineral used in traditional medicine, demonstrated an inhibitory effect on both P. acnes growth and P. acnes-induced IL-8 and TNFα signaling in a monocyte cell line [53]. Further in vitro and in vivo studies have shown that zinc affects various pro-inflammatory signaling pathways involved in acne and comedo formation [54]. These preliminary findings suggest that further research into the anti-inflammatory effects of topical zinc is warranted.

Azelaic acid

Azelaic acid is a naturally occurring, plant-derived saturated dicarboxylic acid that has shown effectiveness both as a standalone treatment and in combination therapies for rosacea, acne vulgaris (both inflammatory and comedonal), and various hyperpigmentation disorders such as melasma and post-inflammatory hyperpigmentation [55]. Azelaic acid possesses antibacterial and antiinflammatory properties, inhibiting mitochondrial metabolism and microbial protein synthesis, thereby exhibiting antimicrobial activity [56]. While a 20% concentration has been used for treating acne, it has mostly been replaced by a 15% concentration due to its lower irritancy. In two randomized controlled trials, azelaic acid was found to be more effective than a placebo, especially for treating the inflammatory aspects of acne [57].

Salicylic acid

Salicylic acid has an anti-inflammatory mechanism in acne treatment. Shao et al. [58] reported that supramolecular salicylic acid treatment increased the expression of caveolin-1 and decreased the expression of interleukin IL-1a, IL-6, IL-17, transforming growth factor beta, and toll-like receptor 2 in skin tissue after supramolecular SA treatment. Additionally, Klebeko et al. found that salicylic acid could inhibit the production of the proinflammatory cytokine IL-6 in LPSstimulated keratinocytes and suggested that novel salicylic acid agents could be used for chronic skin diseases, including acne vulgaris. Recent studies have also indicated that salicylic acid affects keratinocytes and sebocytes, which are involved in acne pathogenesis [58, 59]. Furthermore, in a crossover study, 30 patients using a 2% salicylic acid cleanser for two weeks showed significant improvement in their acne, evidenced by a reduction in comedones. However, their acne worsened during the subsequent use of a benzoyl peroxide (BPO) wash, a commonly used first-line bactericidal treatment [60, 61].

Retinoids

Topical corneolytics, such as retinaldehyde and retinol found in low concentrations in a wide variety of over-the-counter formulations, have comedolytic and skin-lightening effects. These can help facilitate skin absorption of topical medications enhancing patient satisfaction [62, 63]. According to El-Samahy et al. [64] in the article, namely the effect of topical application of nano retinol on mild to moderate acne vulgaris stated that topical retinol preparations have a mechanism of action able to reduce the number of acnes on facial skin caused by Propionibacterium acne (P. acne) bacteria by inhibiting excess oil production (micro blackheads) and able to disguise acne lesions. In the study The Antibacterial Activity of Topical Retinoids: The Case of Retinaldehyde M. Pechere et al. have showed that RAL demonstrated notable antibacterial activity against grampositive bacteria. The minimum inhibitory concentration (MIC) was 4 mg/l for Staphylococcus aureus, Micrococcus flavus, and P. acnes CIP179 [65].

Antioxidants

Antioxidants are increasingly significant in acne treatment [36]. For example, epigallocatechin-3gallate (EGCG) has demonstrated anti-inflammatory properties by

suppressing the NF-κB and activator protein 1 (AP-1) pathways and helps control sebum production [66]. This makes EGCG a promising therapeutic option for acne [66]. Similarly, fullerene is another antioxidant potentially useful in dermocosmetics. Using fullerene gel twice daily for eight weeks reduced the number of pustules in acne patients, and in vitro studies showed it inhibits sebum production and reduces neutrophil infiltration [67]. Ascorbic acid, or vitamin C, is a well-known antioxidant with antiinflammatory effects. It prevents sebum oxidation, thereby reducing inflammation and follicular keratinization. Research indicates that vitamin C can reduce UVAinduced sebum oxidation by up to 40% [68].

Protection of the skin barrier

Maintaining the lipid barrier at an appropriate level and decreasing TEWL is an important mechanism whereby dermocosmetics can improve acne management. Dysfunction of the epidermal barrier can be a feature of the disease and can also occur as a result of acne treatments, including over the counter (OTC) products, ethical prescription products, and procedures such as peeling [69]. Clinically, barrier dysfunction manifests as dry skin, irritation in the form of stinging/burning/ tingling, tightness, pain, or irritant dermatitis [69]. These are thought to be related to TEWL and can be at least partially relieved with use of moisturizers [69, 70]. As early as 1995, Yamamoto et al. [71] demonstrated that Japanese acne patients had increased TEWL compared with control subjects. The differences were significant even in patients with mild and moderate acne. These patients also had lower levels of ceramides, which correlated with water barrier function. The authors speculated that the decreased ceramides may contribute to hyperkeratotic barrier dysfunction and formation of comedones [71].

Cleansers

Acne can worsen with aggressive cleansing or using a cleanser with an unsuitable pH. A dermocosmetic cleanser with a pH similar to that of normal skin is less irritating and may improve patient adherence to treatment [36]. Alkaline soaps raise the skin's surface pH, potentially impairing the skin barrier's repair mechanisms [72, 73] and causing irritation [74]. They can also alter the skin surface and increase transepidermal water loss [73]. Compared to acidic syndet bars, soap can cause peeling, dryness, and burning [75,76]. One study evaluated the degreasing effect and skin tolerability of a botanical face cleanser containing hops, willow bark extract, and disodium cocoyl glutamate, a mild cleansing agent, against a standard cleanser with sodium laureth sulfate (SLES) [77]. Both cleansers were used by 21 healthy volunteers with normal to oily skin, applied twice daily for 15 days in a split-face manner. The botanical cleanser significantly reduced sebum levels [77] and maintained a degreasing effect even after a 48-hour treatment break, whereas the SLES cleanser saw an increase in sebum levels. Although neither cleanser caused skin irritation, those without SLES may be more suitable for sensitive skin [77].

Moisturizers

Ceramide-containing moisturizers should be considered for acne dermocosmetics. When the skin barrier function is compromised, there is a reduction in skin surface ceramides, leading to increased transepidermal water loss. Applying moisturizers with ceramides can help improve skin dryness and irritation [78]. This, in turn, may enhance adherence to existing treatment regimens by alleviating the symptoms and side effects, such as skin dryness and irritation, that often lead to nonadherence [78]. Draelos reported that non-comedogenic, non-acnegenic moisturizer selection is important to counteract the drying effect of some acne medications [79]. Topical emollient compounds can help reduce skin irritation by enhancing the stratum corneum barrier function. In an open-label, randomized study involving 30 patients receiving either oral isotretinoin or topical tretinoin, a simple emollient cream used as an adjunctive treatment significantly improved skin dryness, roughness, and desquamation [80].

UV exposure

UVA and UVB rays affect acne differently. UVA rays, specifically UVA1 and blue light (400nm) may have anti-inflammatory effects [81]. In contrast, UVB rays cause inflammation, and increase sebum production and proliferation of keratinocytes [82,83]. Patients should be aware that not using UV protection on their skin during the summer may not help their acne and could actually worsen the condition in the following months [84]. A prospective open-label study on acne patients undergoing various treatments found that daily use of a cleanser and SPF 30 sunscreen improved skin tolerability, reduced transepidermal water loss, and helped patients maintain consistent application of their therapies [85]. Another study demonstrated that using a sunscreen containing anti-inflammatory agents reduced inflammatory facial acne lesions within two weeks [86]. The need for UV protection is increased in patients with acne as the skin barrier integrity is reduced leading to increased photosensitivity, which may be exacerbated by treatments such as BPO or retinoids [87, 88].

Make up

Corrective makeup is designed to conceal disfiguring skin lesions and enhance the skin's appearance. It can correct pigmentation issues, control oil, moisturize, protect against UV light, enhance the absorption of acne treatments, strengthen the skin barrier, and boost personal wellbeing [89,90]. An ideal acne camouflage should have a natural appearance, be nongreasy, noncomedogenic, and can be easily applied. The preferred product is largely determined by patient preference, market availability, the range of available shades, and the presence of specific ingredients [91]. Some products might include botanical agents with natural beta-hydroxy acids reputed for their anti-inflammatory and antimicrobial properties, or vitamins (like Vitamin E) that may serve as antioxidants [92]. In the conducted study Monfrecola et al. reported that one hundred percent of patients reported satisfaction with a face compact cream (FCC) containing selective photofilters, Salix alba, 1,2-decanediol, soy isoflavones, and vitamins B3, C, and E, after applying it once daily for 28 days. Additionally, 80% of the patients observed an improvement in their skin. The application of the FCC significantly reduced the number of comedones by 16% from baseline to Day 28 ($p < .001$). The cream was well-tolerated, with no skin reactions such as erythema, edema, dryness, desquamation, tightness, itching, or burning reported at any time points (Days 0, 14, and 28) [93].

Conclusion

Dermocosmetics play a vital role in acne management, serving as maintenance therapy or complementing pharmacological treatment. Various active ingredients and formulas can target key acne pathways, including inflammation, abnormal keratinization, excessive sebum production, and C. acnes colonization. Additionally, dermocosmetics aid in mitigating the side effects of dermatological treatments, such as compromised skin barrier, increased transepidermal water loss, and irritation. Dermocosmetics have been linked to excellent patient adherence and high levels of satisfaction. Consciously incorporating them into dermatological treatment under the supervision of a dermatologist allows for achieving satisfactory results of therapy. Newly emerging dermocosmetics on the market require proper research and informed selection in dermatological treatment.

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Autor's contribution:

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