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COMPREHENSIVE REVIEW ON ACNE VULGARIS AND ITS TREATMENT

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ABSTRACT: Acne vulgaris is a multifactorial skin disorder of pilosebaceous unit which majorly affects teenagers, but it can continue into adulthood and cause aesthetical as well as psychological problems. This review article enlightens the knowledge related to cause and treatment of acne vulgaris. Several factors which are involved in the development of acne vulgaris are hormonal activity, sebum production, follicular epidermal hyperproliferation, colonization with Propioni-bacterium acnes (*P. acnes*) and inflammation. There are several treatment methods available for acne, although some new therapeutic agents have been introduced during the last few years; synthetic drugs still remain the best option in the treatment of acne. However, rapid appearance of antibiotic resistance and high re ocurrence rates have led to a search for alternatives in nature. The use of herbal drugs in prevention and treatment of acne vulgaris nowadays increased because of their efficiency, availability and lesser side effects than synthetic drugs. Some common used topical herbal remedies with significant anti-inflammatory activities antimicrobial activities have mentioned in this review article include tea tree oil (*Melaleuca alternifolia*), turmeric (*Curcuma longa*), neem (*Azadirachta indica*), licorice (*Glycyrrhiza glabra*), lavender (*Lavandula angustifolia*), green tea (*Camellia sinensis*) and basil (*Ocimum basilicum*). Other commonly utilized effective synthetic drugs discussed in this include benzoyl peroxide, azelaic acid and retinoids *etc*. This review article provides complete knowledge about cause factors and treatment options which are available for acne vulgaris patients.

INTRODUCTION: Acne is an inflammatory condition of the pilosebaceous unit. These include overproduction of oil, abnormal thickening in the skin lining a hair follicle, inflammation and proliferation of *Cutibacterium acnes* [1] Acne affects nearly 90% of adolescents and approximately have symptoms until they are adults. About 1% of men and about 5 % in women have acne lesions by the age at which most people live until they are officials [2]. This condition commonly affects sebaceous rich areas of skin such as the face, upper chest and back.

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The onset is common in adolescence, but it can occur at any age. This can be in the form of non-inflammatory comedones *i.e.* blackheads and whiteheads as well as inflammatory papules, nodules, pustules, cysts or a combination of these types of lesions [3]. For young teens, acne has a lot of drawbacks. It results in physical deformity, discomfort, emotional strain, and possibly long-lasting skin scars. Patients may experience fear and humiliation as well as a decline in their social and physical well-being [4].

By classifying acne into two categories non-inflammatory acne, which is characterized by closed and open come rational treatment modality selection can be considerably improved dones (blackheads and whiteheads, which are compact lumps of microorganisms, keratin, and sebum dilating the follicular duct), and Inflammatory acne

is defined by the appearance of inflammation as nodulocystic lesions, papules, and pustules on an ascending degree of devastation and likelihood of lasting damage [5].

Pathogenesis of Acne: There are four main pathogenic causes associated with acne, however the exact mechanisms are unknown:

1. Overproduction of sebum.
2. Hyperkeratinization of the pilosebaceous duct.
3. Aberrant bacterial function.
4. Development of inflammation [6].

Increased Sebum Production: In the formation of acne the overproduction of sebum is the main factor. Together, acne patients secrete more sebum than average people, depending on the severity of their condition and the Sebum is primarily associated role in the etiology of acne. Sebum plays an indisputable role in providing the substrate for *P. acnes* growth. Specifically, triglycerides are acted upon by *P. acnes* lipase to form diglycerides, monoglycerides, and free fatty acids, which in turn form glycerol, the utilizable moiety for *P. acnes* metabolism [7].

Hyperkeratinization of Pilosebaceous Duct: Healthy follicles frequently shed keratinocytes with just one cell into the lumen, which are then at last wiped out. Nonetheless, in Keratinocytes in acne patients hyperproliferate and do not shed into the lumen, which prompts the collection of sporadic desqua mated corneocytes in the pilosebaceous follicles combined with lipids what's more, monofilaments [8].

Abnormal Bacterial Function: *P. acne* playing a major role here the bacterium thrives in clogged, oxygen deprived pores, breaking down sebum into free fatty acids, potentially leading to acne formation. However, it remains uncertain whether these microorganisms trigger acne, simply thrive in lesions or maintaining a microbial balance within the follicle is key to acne development [9].

Development of Inflammation: If bacteria Propionibacterium acnes (*P. acnes*) makes its way into your immune system, it responds with the inflammation boundaries. *P. acnes* itself is also very proinflammatory and will stimulate the production of chemotactic agents that summon immune cells like lymphocytes, neutrophils and macrophages to the affected skin site.

It not only gives a collagen boost its also aid in wound healing by controlling the immune response that often occurs when there is an impaired keratin confinement around hair follicle and this help to damage it leading to rupture and releasing of bacteria fat acids lipids into the dermis. This leads to the formation of inflammatory lesions such as pustules, cysts, nodules, and papules. Non-inflammatory lesions, on the other hand, are smaller and contain less pus than inflammatory ones. The delivery of ROS by neutrophils contributes in the breakdown of follicular epithelium and uncompleted acne inflammation and leucotrine B4 and IL-1alsoresponsible for inflammation and come do genesis [10, 11].

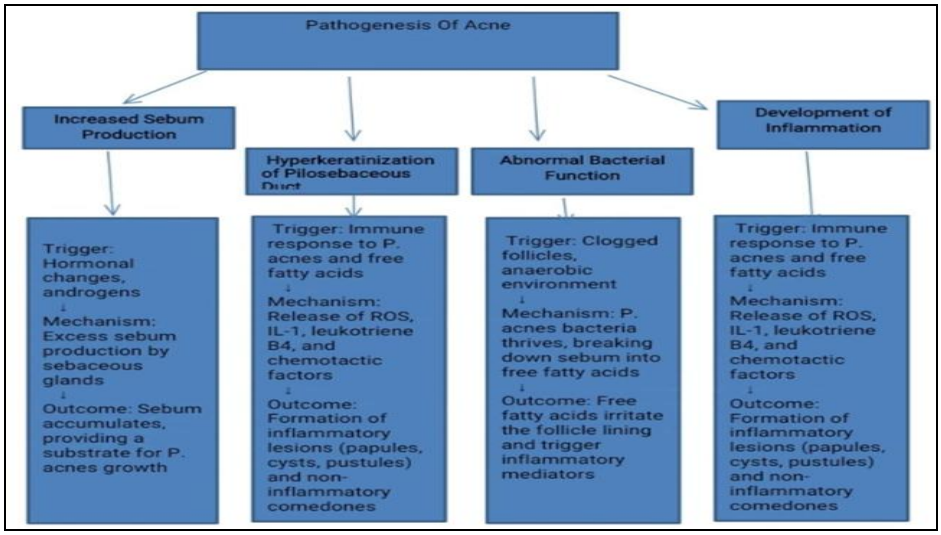


FIG. 1: PATHOGENESIS OF ACNE VULGARIS

Etiology of Acne: Follicle obstruction, hyperkeratinization, keratin plug production, and sebum (microcomedo) all contribute to the development of acne. Increased androgen production causes the sebaceous glands to expand and produces more sebum. An opencomedo which is known as blackheads or closed comedo might develop from enlargement of the microcomedo. These comedones are mainly caused due to obstruction of sebaceous gland or may be due to overproduction of sebum or oil [12]. In the dermis surrounding the microcomedo or comedone, the naturally existing commensal bacteria Propionibacterium acnes can induce inflammatory lesions such as infected pustules, nodules, and papules, resulting in redness and hyperpigmentation [13, 14].

Environmental Factors: Dirt, cook oil vapours humidity (atmosphere should not be too oily or sweating), if your skin is more hydration that may also develop acne and chemicals including petroleum derivatives can cause an increase in the incidence of pimples on face and back [15].

Drugs: Some medications can cause acne, including phenobarbital, lithium, steroids and rifampin [15].

Hormonal Factors: Puberty, menstrual cycles, PCOS or menopause (acne climacterica) mediated changes in hormones culminating into acne [16].

Genetics: The risk for acne is polygenic with different genes including TNF-α and I1α referenced to have a relationship (independent variable) (never linked before) [17].

Psychological Factors: Stress associated with stress and acne severity (studies from Singapore and USA) [18].

Microorganisms or Bacteria: Propioni-bacterium acnes is a fundamental bacterium that causes acne, and resistance to antibiotics is on the rise [19].

Diet: Nutrition although a high-glycemic diet is linked to acne exacerbation, the exact association between food and acne is yet unknown Increases in the prevalence of acne are positively correlated with milk consumption Acne development is not linked to eating chocolate or salt, according to reports Chocolate has a high glycemic load due to its high sugar content. Acne may have anything to do with insulin metabolism and fat.

Parasites: Although the parasite mite Demodex and acne are related, it is unclear if the effects are caused by Demodex itself or by bacteria associated with Demodex [20].

TABLE 1: CLASSIFICATION OF ACNE 21

Acne	Non inflammatory acne	Inflammatory
Type of lesion	Pustules	Cystscomedons
Papules		
Locations	Central face less common and on chest and back	Face in any region

Treatment of Acne Vulgaris: Some common herbal plants used in treatment of acne vulgaris.

***Melaleuca alternifolia* (Tea Tree Oil):**

Mechanism of Action: Shows potent antibacterial qualities against the bacteria which cause acne.

Evidence: Compared to benzoyl peroxide, 5% tea tree oil gel had fewer side effects and a substantial reduction in acne lesions after 12 weeks [22].

Overall, 44% (27/61) of the patients treated with tea tree oil experienced adverse effects during the trial, compared to 79% (50/63) of the patients treated with benzoyl peroxide who reported similar issues. There was a substantial difference ($P<0.001$) [23].

Chemical Constituent: Terpinen-4-ol is an important oil component which has capability to inhibit microbial growth as well as reduce inflammation and is responsible in the treatment of acne [24].

***Curcuma longa* (Turmeric):**

Mechanism of Action: The stronger superoxide anion, hydroxyl radical, singlet oxygen, peroxynitrite, and nitric oxide found in the rhizomes of *Curcuma longa* (Zingiberaceae) have anti-inflammatory and antioxidant properties, and are thus employed in treatment of acne vulgaris and for other skin diseases also [25].

Evidence: Mate *et al* in 2021 developed polyherbal gel against acne.

The result of their study showed significant inhibition of bacterial growth with no irritation. Also this study showed that this gel has a additive effect when compared to the individual extract having good stability [26].

Chemical Constituent: Curcumin is responsible for the treatment of acne vulgaris and it also inhibits MCP-1 production from adipocytes and hence decreases the formation or activation of macrophages in adipose tissue and decrease the inflammatory reactions occurred by obesity. It may help obese people with long-term inflammatory disorders [27].

Azartica Indica (Neem):

Mechanism of Action: The antibacterial activity of neem oil is due to Margolone and Mahmoodin which shows strong inhibitory effect on Cutibacterium acnes.

It contains salicylic acid-like compounds which reduce redness and infla-mmation and active ingredient that are similar to several of the compounds found in common acne medications like salicylic acid as well making neem really suitable for treating all types of skin conditions related with acnes. It inhibits the bacterial growth and avoid the reoccurance of lessions of skin [28].

Evidence: Molecular docking was done for prediction of affinity of binding prior to pathway and when investigations of protein protein interaction occur. I was examined that Neem oil can potentially target STAT1, CSK, CRABP2, and SYK genes in the treatment of *AV* [29, 30].

Chemical Constituents: Chemical constituents such as Nimbidin, Nimbin, Nimbolide, Gedunin, Azadirachtin, Mahmoodin, Cyclic trisulphide and others are used as anti-inflammatory, antibacterial, antigastric ulcer, antiarthritic, spermicidal antifungal, antimalarial, hypoglycemic, immunomodulatory, diuretic and antitumour [31, 32].

***Lavandula angustifolia* (Lavender):**

Mechanism of Action: Lavender oil has the ability to kill bacteria and has bactericidal effect on some microorganisms who are antibiotic-resistant in natutre. This is essential in the case of long-term acne treatment, and it also inhibits Gram -positive and Gram-negative bacteria [33, 34].

Evidence: *In-vivo* studies had been done by Kim and Shin in which a mixture of TTO (3%) and LAO (2%) was used. This mixture was applied topically for 4 weeks, and it results in a reduction in acne and inflammation [35]. These studies give a solid proof that the essential oils has microbial inhibiting activity and can improve acne lesions and can be used as an alternative treatment for those patients who do not want or are not able to use antibiotics fir the treatement of acne [36].

Chemical Constitutions: Due to the large content of linalool and linalyl acetate in the lavender oil, primarily responsible for its antibacterial property [37, 38, 39].

***Camellia sinensis* (Green Tea):**

Mechanism of Action: It inhibits the growth of bacteria, is to prevent the adhesion of bacteria to mammalian cells [40, 41]. EGCG also induces cytotoxic effect of SEB-1 sebocytes *via* cell death and decreases the count of *P. acnes* and also targets the other features of acne [42].

Evidence: Current studies suggest higher phosphoinositide 3-kinase-Akt-mTORC1 activity in the skin of acne vulgaris patients resulting in overproduction of sebum [43, 44, 45]. Green tea-derived plant sterols have been suggested to be potential therapeutic agents for acne vulgaris, and an experimental study showed a significant inhibitory effect of EGCG (epigallocatechin gallate) on mTORC1 due to the reduction in activation of mTOR, as a consequence of decreased phosphorylation levels relative to IGF-1 stimulated sebocytes. Moreover, EGCG also behaves as an ATP-competitive inhibitor of PI3K and mTOR by binding to the active site of molecular docking studies of EGCG with PI3K demonstrated that EGCG bound effectively in the active side of PI3K. Enhanced PI3K-Akt-mTORC1 signaling, due to increased insulin-IGF-1 signaling in acne especially in those with prepubescent or western diet, green tea polyphenols, especially EGCG, may help suppress this pathway. Green tea contains the highest polyphenolic content, with EGCG and EGC being the most prominent components [46, 47, 48].

Chemical Constituents: Green tea contains several polyphenols of the catechin family, EGCG, EGC, ECG and EC [49].

***Glycyrrhiza glabra* (Licorice):** Mechanism of action. It inhibits all the four mechanisms that are responsible for the pathogenesis of AV. Its anti-inflammatory activity, anti-androgenic, antimicrobial activity is responsible for showing antagonistic effect against AV and Glycerin is majorily responsible for anti androgenic effect [50, 51, 52].

Evidence: Methanolic extract of *Glycyrrhiza glabra* (licorice) has shown good *in-vitro* antimicrobial activity: MIC 1.25 mg/ml for Propionibacteriumacnes; MIC 2.5 mg/ml for *Staphylococcus aureus* and *S. epidermidis*. Another study reported that licorice has antibacterial activity on two different strains of P. acnes, with MIC values of 200 µg/ml for ATCC 6919 and 100 µg/ml for ATCC 11827. We also found the MICs of licorice extract is 0.25 mg/ml for methicillin-susceptible *S. aureus* and 2.5 mg/ml for methicilin-resistant *S. aureus*, respectively [53, 54, 55].

Chemical Constituents: Licochalcone A, licochalcone E, Glycyrrhizin and the total extract of licorice are examined and identified as bioactive compounds against bacteria which cause acne [56, 57, 58].

Basil (*Ocimum basilicum*):

TABLE 2: COMPONENTS AND THEIR ROLE IN ACNE VULGARIS [59, 60, 61]

Chemical Constituents	Mechanism of action or role in treatment
Polyphenolic Flavonoids	Scavenge free radicals and it prevents the water loss and acne
Lutein, carotene, vitamin A, vitamin K, oxygen and Zeaxanthin	Shows protective roles against derived free radicals and ROS
Citronellol, limonene, eugenol, linalool, and terpineol	Anti-inflammatory and antibacterial action.

Evidence: Viyoch concluded that upon testing activity against P. acne the sweet basil oil shows high susceptibility at conc. 2.0%v/v and Holybasil oil inhibited *P. acnes* at 3.0%v/v Lertsatitthanakorn P, also concluded that holy basil and sweet basil oils showing an inhibition zone were 1.00% v/v against P. acne [62].

Synthetic Drugs Used in Treatment of Acne vulgaris:

Benzoyl Peroxide: Benzoyl peroxide (BPO) is well-known for its anti-inflammatory qualities; however, it may also harm host cells in addition to killing bacteria like P. acnes. (65,74) BPO indirectly reduces tissue inflammation by inhibiting human polymorph nuclear leukocytes' (PMNL) production ROS [63]. Moreover, BPO has comedolytic and keratolytic properties that decrease comedone size and accelerate sebum excretion rates. Longer head-to-head studies are required to make an accurate comparison, but a 2006 study found that BPO was more effective than retinoic acid (RA) and salicylic acid (SA) at disrupting stratum corneum cohesion [64].

Properties: It is of lipophilic nature and hence it can easily cross the stratum corneum[65].

It is Soluble: Organic solvent

Insoluble: Water

Route of Administration: Topical [65].

Combinational Therapy:

TABLE 3: BPO IN COMBINATION WITH ERYTHROMYCIN

Study Year	Study Type	Intervention	Results	References
1983	Double-blind clinical trial	5% Benzoyl Peroxide Gel vs. 1.5% Erythromycin Lotion	For the treatment of small, inflamed lesions both were equally effective but in case of non inflamed lesions only benzoyl peroxide effective.	Burke B *et. al* 66
1983	Double-blind, controlled clinical trial	3% Erythromycin + 5% Benzoyl Peroxide Gel	For reducing papules and pustules the combination was more effective, and the adverse effect is similar as benzoyl peroxide given alone.	Chalker DK *et. al* 67.
1992	Observational study	Topical Erythromycin vs. Erythromycin + Benzoyl	By using Erythromycin it leads to antibiotic resistant Staphylococcus	Harkaway KS *et. al* 68.

		Peroxide vs. Benzoyl Peroxide alone	epidermis.however in combination or given alone the benzoyl peroxide decreases the bacterial populations.	
2003	Randomized, double-blind, multicenter, parallel group study	3% Erythromycin + 5% Benzoyl Peroxide Gel vs. 0.025% Tretinoin + 4% Erythromycin	When benzoyl peroxide given in combination it was more effective in reducing the acne symptoms as early	Gupta, A.K. *et. al* 69.

TABLE 4: BPO IN COMBINATION WITH CLINDAMYCIN

Study Year	Study Type	Intervention	Results	References
1988	Randomized, investigator-blind clinical trial	1% Clindamycin vs. 5% Benzoyl Peroxide Gel	Clindamycin was effective in reducing acne lessions but less effective as compared to benzoyl peroxide and it was better tolerated.	Swinyer LJ *et. al* 70.
2001	Open labeled study and multicenter, double-blind trial	5% Benzoyl Peroxide + 1% Clindamycin vs. 1% Clindamycin alone	Improvement in efficacy was by combination therapy as resulted in a 99.8% (>2 logs) reduction in total number of P. bacterium after 1 week of therapy.	Leyden J *et. al* 71. And leyden JJ *et.al* 72.
2001	randomized, 10-week,multicenter, single-blind trial	Benzoyl Peroxide + Clindamycin vs. benzoyl Peroxide + Erythromycin	Upon comparison Benzoyl peroxide/clindamycin shows improved efficacy and similar tolerability to benzoyl peroxide when used alone.	Leyden JJ *et. al* 73

Azelic Acid: Azelaic acid is an acid analogue of dicarboxylic acid which naturally occurs in nature. Compared to vinegar, it is a far weaker acid. Certain whole grains contain it, and the human body has trace quantities of it with plasma levels typically ranging from 20 to 80 ng/ml [74, 75].

TABLE 5: STUDIES OF AZELIC ACID

Study year	Study type	Intervention	Results	References
1986	double-blind study	Topical azelaic acid and oral tetracycline	Only minor side effects were produced and oral tetracycline was more effective than other one.	Bladon PT *et. al* 76.
2010	multicenter, randomized double blind study	(AA) 5% and erythromycin 2% gel compared with AA 20% or erythromycin 2% gels	AA 5% and erythromycin 2% gel were more effective with fewer side effects.	Pazoki-Toroudi H *et. al* 77
2011	multicenter, randomized, and double-blind study	(AA) 5% and clindamycin (Clin) 2%and combination (AA-Clin)	The combinational therapy did not produce any adverse effects as individual shows.	Pazoki-Toroudi H *et. al* 78.

Retinoids: Retinoids affect cell division and proliferation they also counteract aberrant desquamation by speeding up the shedding of corneocytes and follicular epithelial turnover, which leads to the expulsion of comedones and the inhibition of microcomedone formation.

The retinoid molecules that are employedin the topical management of acne are:

Tretinoin: Tretinoin increases turnover of follicular epithelial cells and enhances desquamation of corneocytes, leading to improved keratinization. This process allows the excreting comedone to drain and new ones from forming. Thus, the paste dilates the previously hindered follicles that become less anaerobic and in turn antimicrobial agents can penetrate better. This would also help minimize the colonisation from Propionibacterium acnes with their pro-inflammatory mediators.

Of course, these are all data from topical retinoids (which have been shown to reduce *P. acnes* counts) since that's your active ingredient in the Differin, but we also know from recent chromatographic analyses of microcomedone lipid contents, that retinoids reduce free fatty acids within them as well [79].

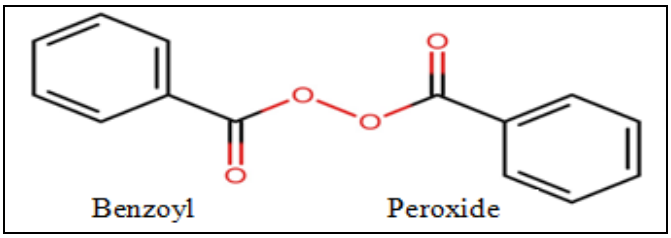
Adaplene: It effectively regulates cellular keratinization and inflammatory processes hence it is highly comedolytic and anti-inflammatory. It inhibits the lipooxygenase activity and the oxidative metabolism of Arachidonic acid which helps in reduction of skin irritation and causes law risk of erythema. Adapalene has low percutaneous absorption, allows it to remain concentrated in the epidermis and in the hair follicles [80].

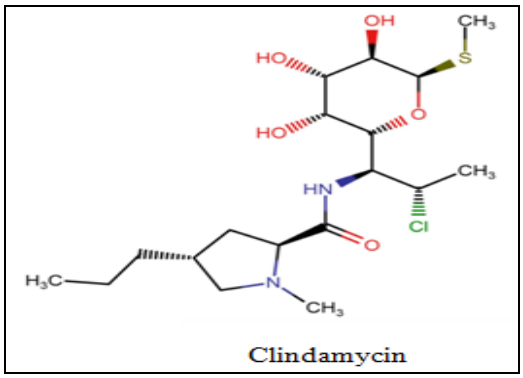
Isotretinoin: Isotretinoin affects follicular epithelialization through its anticomedonal action. Additionally, it suppresses sebum production by impeding retinol dehydrogenase-4's oxidative 3-alpha hydroxysterol dehydrogenase activity, which transforms androsterone and 3-androstanediol into dihydrotestosterone and androsterone, so promoting sebum secretion and this is the unique property of Isotretinoin. with oral isotretinoin only the sebum suppressive effect occurred [81].

Tazarotene: Like all retinoids, tazarotene works against a number of the variables that cause acne lesions when applied topically as an aqueous gel or cream. It makes the keratinization normal. Pattern and reduces the coherence of follicular keratinocytes, which causes comedones to burst and stops new ones from forming.

Moreover, by preventing the expression of migratory inhibitory factor-related protein type 8 in skin grafts, tazarotene may directly reduce inflammation. Localized cutaneous irritation that ranges from mild to moderate, accompanied by burning, itching, erythema, peeling, and/or dryness.

In case of oral isotretinoin the current dosage iss 0.5–2.0 mg/kg/ day, administered over 4–6 months and this drug is potent tetratogen causes strict contraception in females [82].





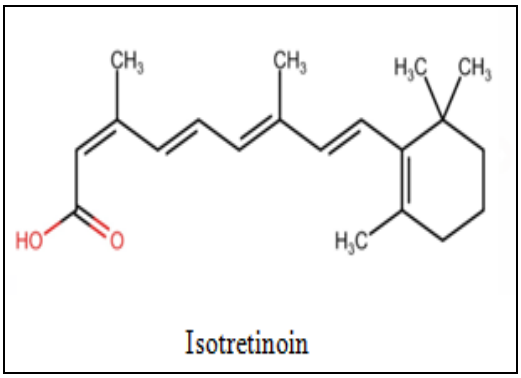


FIG. 2: SOME SYNTHETIC COMPOUNDS USED IN THE TREATMENT OF ACNE VULGARIS

Futuristic Approach: The mounting evidence for the integration of personalized medicine, advanced biotechnology and the holistic paradigm into the future of acne vulgaris management is promising. Recently some research has emerged about skin microbiomes, and new therapeutic strategies are being envisioned for modulating the microbial communities. Further progression in genomics and proteomics could also allow for personalized treatments corresponding to each person's genetic predisposition and skin type, as well improve efficiency while minimizing possible side-effects. The penetration and bioavailability of both herbal and synthetic agents into or through the skin can be enhanced by incorporating new dermal delivery systems including nanoparticles and microneedles, which may lead to a more efficient way of using topicals. Additionally, the investigation of lifestyle determinants, such as diet and stress coping strategies, may allow the development of integrative care schemes covering acne-related psychosocial dimensions.

If inter-departmental collaboration among Dermatologists, Nutritionists and Psychiatrists is handled then it will lead to patient-centered frameworks, ultimately improving outcomes and quality of life.

CONCLUSION: The present review is aimed to focus on the complexity of factors contributing to the pathophysiology and etiology with special emphasis on the knowledge of these interactions for effective management the treatment of Acne. Emerging research will increasingly focus on the interplay between personalized medicine and novel therapeutic modalities to refine treatment efficacy and Exploration of both the synthetic and herbal treatment options revealed various therapeutic targets with specific benefits. More comprehensive management strategies will be developed including consideration of individual patient needs, lifestyle factors, and emerging scientific insights. The new treatment guidelines literally broaden clinicians' horizons in treating patients affected by acne vulgaris for optimal health status.

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