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A COMPREHENSIVE STUDY FOR ACNE MODELS: A REVIEW

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ABSTRACT

Acne vulgaris (AV), a widespread chronic inflammatory skin condition that mostly affects the pilosebaceous unit, affects a considerable portion of the population. The Gram-positive anaerobic bacterium Propionibacterium acnes, which is a normal part of the skin microbiota but plays a significant role in causing inflammation and the development of acne lesions, is a major contributor to this complex disorder. The development of the condition is also influenced by other elements such immunological reactions, follicular hyperkeratinization, excess sebum production, and hormonal imbalance. The current in vitro, ex vivo, and in vivo models used to assess pharmaceutical and cosmetic formulations created for the treatment and prevention of acne are highlighted in this paper. Ex vivo models use removed skin to examine medication penetration and retention, while in vitro models use microbe culture experiments to evaluate antibacterial efficacy. Understanding the overall therapeutic efficacy and safety of formulations is aided by in vivo models, which frequently involve animal experiments or clinical trials. These experimental methods offer dependable platforms for the screening of new drug delivery systems like nanoformulations, herbal extracts, and biologically active chemicals. As a result, they are vital to the advancement of acne research and the creation of safer, more efficient, and focused treatments for better acne control.

KEYWORDS: nanoformulations, chemicals, population.

1. INTRODUCTION

The pilosebaceous unit (PSU), which consists of the skin's sebaceous gland, hair follicle, and hair shaft, is where acne, a complicated, long-term inflammatory condition, first appears. Blackheads (open comedones) and whiteheads (closed comedones) are examples of non-inflammatory lesions; papules, pustules, nodules, and cysts are examples of inflammatory lesions. Propionibacterium acnes, a Gram-positive anaerobic bacterium that causes inflammation by producing hyaluronidases, lipases, and proteases, is closely linked to the illness. About 90–95% of teenagers, 20–40% of adults, and a sizable percentage of women including those who have adult female acne after the age of 25 are affected by acne. Increased sebum production, follicular hyperkeratinization, microbial colonization, and inflammation are the four main causes that cause acne. Its development and severity may also be influenced by a number of contributing factors, including food, hormonal imbalance, genetic predisposition, and other microbial species. Due to inherited genetic characteristics, people who have a family history of acne are more likely to get the disorder. Hormones, especially androgens, are crucial because they increase sebum output and stimulate sebaceous gland activity, which can clog pores and encourage the development of lesions.[1]

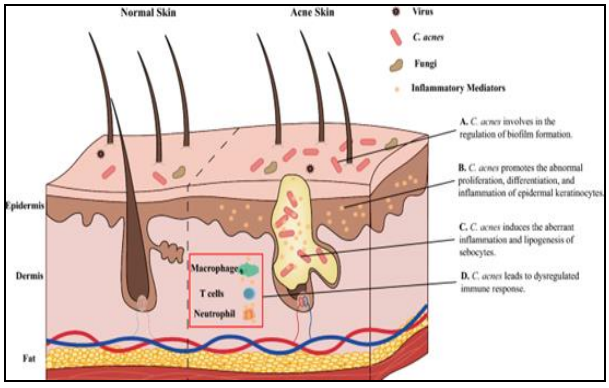

Fig 1: Acne on Face.

Because they use a holocrine secretion process to produce sebum, a complex mixture of lipids that includes triglycerides, wax esters, and squalene, sebaceous glands are essential to the pathophysiology of acne. While the anatomy of sebaceous glands is similar in all areas of the skin, their regulation and activity differ. While certain sebaceous glands occur separately in specific skin regions, the majority are connected to hair follicles to create pilosebaceous units. Microcomedones are the first lesions that might develop into more severe inflammatory forms due to excess sebum production and aberrant keratinocyte shedding. In addition to Propionibacterium acnes, additional microbes like Klebsiella pneumoniae, Streptococcus agalactiae, Staphylococcus epidermidis, and Staphylococcus aureus may also aid in the advancement of the illness. P. acnes is the most important of these because it stimulates androgen receptor activity, increases sebum production, encourages keratinization, and sets off immunological reactions. Through chemotactic factors and Toll-like receptors, it initiates inflammatory pathways that eventually result in the distinctive inflammation observed in acne lesions.[2]

2. Mechanism of acne formation

Sebaceous gland (SG) hyperactivity, follicular hyperkeratinization, immunological changes, and follicular infundibulum obstruction are the first steps in the development of acne. Propionibacterium acnes colonization and the ensuing inflammation are made possible by these alterations. A major factor in the development of acne is increased sebum production, which acts as a source of nutrients for bacterial growth. Designing and assessing successful anti-acne products requires an understanding of this pathogenic mechanism. The etiology of acne is influenced by a number of important aspects, such as aberrant keratinization, excessive sebum secretion, pilosebaceous unit dysfunction, and the release of inflammatory mediators.[3]

The follicular wall and adjacent dermal tissue become irritated when Propionibacterium acnes breaks down sebaceous triglycerides into free fatty acids. Apart from P. acnes, additional microorganisms like Klebsiella pneumoniae, Streptococcus agalactiae, Staphylococcus epidermidis, and Staphylococcus aureus may also be involved in the development of acne. Lesions like papules and pustules are caused by these microorganisms' production of virulence factors and enzymes that harm tissues, encourage infection, and heighten inflammatory reactions. Anti-acne medications and delivery methods can be assessed using a variety of experimental models, however there aren't many thorough resources that only concentrate on these models. Thus, the purpose of this review is to gather and provide comprehensive data on in vitro, ex vivo, and in vivo models used to investigate acne and evaluate the effectiveness of cosmetic and therapeutic approaches.[4]

3. In vitro models for acne

Organ culture, monolayer culture, follicular models, and testosterone-induced models are among the in vitro models frequently employed in acne research. Nevertheless, the intricate process of acne lesion formation cannot be completely replicated by a single in vitro system. The organ culture model allows for the study of an organ's structure, function, and response to treatments in a nearly physiological setting by maintaining whole tissues or portions of an organ under controlled conditions.[5]

Cells are cultivated in monolayer culture as a single layer atop an appropriate culture medium, which is usually made by enzymatically dissociating tissue pieces. The substrate must be biologically inert, non-toxic, and compatible with the cells in order for growth to be effective; otherwise, cell survival is jeopardized, resulting in aberrant morphological alterations. Sebocytes, the epithelial cells that produce sebum, are used in a specific type of monolayer cultivation. These cells break and degrade their cell membranes to liberate their contents. Sebocytes proliferate and differentiate quickly in culture, but their capacity to accurately replicate natural circumstances is limited because they produce less lipids, such as squalene and wax esters, than newly isolated cells or in vivo sebaceous glands.[6]

a. 3.1. SG organ culture

These models are essential for comprehending the pathophysiology of sebaceous gland (SG)-related skin conditions such acne, seborrhea, and sebostasis. They are also useful instruments for the creation and assessment of pharmaceutical and cosmetic products. Human SG organ culture models are especially helpful because they provide advantages over sebocyte monolayer cultures and demonstrate the functional involvement of sebaceous glands in preserving skin homeostasis. This method uses dissection or microdissection techniques to separate sebaceous glands from human skin taken from cadavers, donors, or surgical waste. The separated tissues are quickly moved into appropriate culture media, like Dulbecco's Modified Eagle's Medium, where the dermal portion is preserved for additional research while the epidermis is separated.[7]

Lipogenesis and the impact of different substances on lipid synthesis are measured using intact SGs, with or without related dermal or epidermal components. In order to observe cell proliferation and lipid synthesis simultaneously, SG-derived cells are seeded onto feeder layers (such as 3T3 cells) and cultured under controlled conditions. Drug transport to the gland is unrestricted in SG organ culture, which is a significant advantage over animal models and allows for precise assessment of biological efficacy. Nevertheless, the process requires a lot of time and technical expertise. Anti-acne research also uses in situ SG models with glands taken from animals. For instance, it has been demonstrated that neuropeptides such substance P promote lipid production

and cell proliferation in sebaceous glands, indicating a connection between stress and the development of acne.[8]

b. Sebocyte monolayer culture

Sebaceous gland (SG) organ culture is a time-consuming method that depends on the constant supply of viable tissue samples, despite its obvious benefits. As an alternative, cell culture techniques offer more scalability and flexibility, enabling the processing, storage, and reuse of a large number of cells from the same cell lineage for numerous investigations. Human sebocyte (HS) cultures with fibroblast support have been successfully grown in a number of laboratories in serum-free conditions, allowing for a thorough assessment of the effects of growth factors and serum components. Compared to organ culture, HS culture provides a well-defined paradigm for altering local environmental circumstances, researching cell metabolism, and evaluating lipogenesis inhibitors in a higher-throughput manner.[9]

It is especially helpful for studying lipid production and androgen metabolism, two important processes in the development of acne. Since lipid production in sebocytes is usually dose-dependent, this model is appropriate for pharmacological research. Another benefit of sebocyte monolayer culture is the production of a high number of cells that may be stored and utilized repeatedly. These cultures are created using two primary techniques: the digestion method, in which isolated SGs are treated with enzymes like trypsin to release cells that are then cultured on a fibroblast feeder layer, and the explant outgrowth method, in which isolated SGs are placed on culture plates and allowed to produce proliferative sebocytes over time. These cells multiply into colonies that can be used for future growth and research.[10]

i. Rat preputial sebocyte monolayer culture

The human sebaceous gland (SG) is frequently modeled in experiments using the mouse preputial gland. These specialized glands release secretions involved in mating behavior and territorial marking when they open close to the urethral meatus. In order to create monolayer cell cultures, rat preputial glands are separated, enzymatically broken down, and cultivated on a fibroblast feeder layer. These preputial cell cultures are a valuable model for researching how hormones affect the growth and operation of sebaceous glands.[11]

They have been extensively employed to study the effects of androgens and estrogens, as well as the function of peroxisome proliferator-activated receptors (PPARs) in controlling lipid metabolism and cell proliferation. Preputial cells go through similar differentiation processes to human sebocytes, however the degree of differentiation is typically less. Furthermore, there are insufficient endogenous PPAR ligands in preputial cells cultivated in vitro, which are required to induce lipid droplet formation. They can therefore only partially replicate the lipid-producing activities of human sebaceous glands. The preputial gland model is nevertheless a useful and approachable tool for researching sebaceous gland biology and hormone regulation in spite of these variations.[12]

c. Follicle model

Propionibacterium acnes, keratinocytes, and sebocytes are studied using the follicular model. Usually, this bacteria colonizes both the pilosebaceous unit (PSU) and the outer layers of the skin. Preserved sebocytes (SZ95) and keratinocytes (HPV-KER) are cultivated in a dual-chamber system divided by a permeable polyester membrane in order to examine the interaction between these cell types. Selective contact is made possible by this membrane, which also keeps soluble substances from uncontrollably mingling between compartments. Propionibacterium acnes is mainly found in the PSU's hair follicle region during normal physiological settings, which makes this model useful for simulating in vivo conditions and researching the pathophysiology of acne.[13]

d. Squalene oxidation model

For the in vitro assessment of acne, squalene oxidation models have been created as sophisticated skin tissue engineering systems. These models are utilized to investigate the composition of sebum and comprehend the part oxidative derivatives of squalene play in the pathophysiology of acne. Oxygen exposure, UV radiation, and controlled incubation conditions are typically used to oxidize squalene. Analytical methods including nuclear magnetic resonance (NMR) and gas chromatography–mass spectrometry (GC–MS) are then used to characterize and quantify the resultant oxidized compounds. Furthermore, models of recreated human epidermis (RHE) are created using oxidized squalene derivatives. These models enable the measurement of certain biomarkers, such as inflammatory cytokines, and the evaluation of morphological changes in the epidermis. These systems are useful for assessing the in vitro effectiveness of medications and formulations intended to stop or lessen the development of acne by focusing on oxidative stress and inflammation since they closely resemble human skin conditions.[14]

e. Testosterone-induced acne model (TIAM)

One steroid hormone that is important in the development of acne is testosterone. It is transformed into its more potent form, dihydrotestosterone (DHT), which uses follicular receptors to promote keratin synthesis. Both DHT and testosterone increase the size and activity of sebaceous glands, which results in an excess of sebum production and acne. Because it closely resembles hormone-driven acne disorders, the Testosterone-Induced Acne Model (TIAM) is frequently used to assess the effectiveness and biocompatibility of different anti-acne medications.[15]

4. In vivo models for acne

These models are frequently used to forecast the therapeutic effectiveness of medications used to treat acne. Because in vivo models accurately mimic real physiological settings, including the processes involved in acne production, they have been increasingly important in medication research in recent years. Because they replicate both healthy and unhealthy human skin, these models are very useful for assessing the anti-acne potential of formulations. The Mexican hairless dog, rabbit ear assay, and rhino mouse model are popular models that closely mimic comedone production. Acne-related skin irritation is a complicated inflammatory reaction that includes endothelial cells, fibroblasts, keratinocytes, and infiltrating leukocytes. Early symptoms include redness, swelling, and itching, which may progress to scaling and erythema. Histologically, the dermis and epidermis are also affected, and different cell types release inflammatory mediators. In addition to serving as a physical barrier, keratinocytes actively contribute to inflammation by producing cytokines. Immune responses are further triggered by bacterial participation, especially by Propionibacterium acnes.[16]

Lesion count, bacterial activity, comedogenicity, histological alterations, and overall anti-acne efficacy can all be assessed using in vivo acne models. Acute models created by topical medicines and chronic models created by intradermal bacterial injection are the two primary forms of inflammatory animal models. These systems aid in evaluating pro-inflammatory and immunostimulatory reactions. Propionibacterium acnes and other microorganisms create cytotoxins that promote chemotaxis, drawing neutrophils and triggering immune cells like natural killer cells and macrophages. Histamine, leukotrienes, interleukins, prostaglandins, and neuropeptides are among the inflammatory mediators that are released as a result. Additionally, neutrophils cause follicular damage by producing enzymes and reactive oxygen species. Papules, pustules, and nodules are examples of inflammatory lesions that arise when comedones burst and release their contents into the dermis. In order to quantify cytokines like TNF-α and IL-1β using immunoassays and get insight into the inflammatory response and treatment success, experimental evaluation frequently entails injecting live bacterial strains into animal tissue.[17]

5. Skin penetration studies

Although this may change their natural physiological state, ex vivo conditions enable the study of cells or tissues outside the living organism in more controlled settings than in vivo investigations. When assessing the penetration of substances through biological membranes, such animal or human skin, these models are especially helpful. Ex vivo investigations are frequently used to evaluate formulations' drug diffusion and penetration properties.[18]

They provide benefits like affordability, easy access to tissue samples, and a comparatively straightforward experimental setting. They do not, however, completely reproduce in vivo settings, including elements like blood circulation and heat sink effects, and their capacity to replicate long-term impacts is restricted. Membranes like egg membranes are occasionally employed in place of biological skin models for in vitro permeation investigations to assess the dispersion of drug-loaded carriers. All things considered, ex vivo models offer a practical and effective initial platform for topical and transdermal formulation assessment.[19]

6. Estimation of antimicrobial (ATM) activity

Finding the minimum inhibitory concentration (MIC) is a typical way to assess the activity of antimicrobial (ATM) drugs. The minimum inhibitory concentration (MIC) is the lowest concentration of an antimicrobial that, following subculture onto an antibiotic-free medium, prevents a particular microbe from growing visibly. It is frequently used to evaluate the in vitro potency of novel antimicrobial drugs, confirm resistance, and gauge microbial susceptibility. MIC is regarded as the gold standard for assessing the efficacy of antimicrobial agents against infections because of its precision and dependability. There are several standardized techniques for calculating MIC, which are covered in the sections that follow.[20]

a. ATM assay

Propionibacterium acnes (MTCC 1951) and Staphylococcus aureus (MTCC 96) are two frequent test organisms utilized in antimicrobial (ATM) assays. Agar plate methods are used to assess the antibacterial activity of compounds and formulations against P. acnes and S. aureus. Initially, microbial cultures are kept at 4 °C on suitable agar slants and utilized as stock cultures for research. In the assay, sterile paper discs with a diameter of about 8 mm are coated with about 60 µL of the test sample and deposited on the surface of agar plates that have been heated beforehand. Under ideal circumstances, the plates are incubated for 24 to 48 hours at 37 °C. The zone of inhibition (ZOI) surrounding the discs, which represents the sample's capacity to impede microbial growth, is measured after incubation to assess the test compound's antimicrobial activity.[21]

b. Tube dilution technique (test tube method)

A common approach for assessing microbial resistance to a particular antimicrobial (ATM) treatment is the tube dilution method. This method involves preparing the test chemical in various concentrations in a liquid growth medium. The microbial culture is then placed in a number of test tubes along with these dilutions. The tubes are checked for turbidity after being incubated under the right circumstances. while turbidity is absent, microbial growth is inhibited; while turbidity is present, growth is continuing. This technique aids in figuring out the lowest concentration needed to stop the organism.[22]

c. Agar diffusion method

The minimum inhibitory concentration (MIC) of antimicrobial (ATM) drugs is frequently discovered using this technique. nutrition broth and nutrition agar are both used to cultivate microorganisms and assess antibiotic efficacy. Using this method, agar plates that have already been inoculated with the microbial culture are covered with filter paper discs that have been impregnated with the test substance. A zone of inhibition (ZOI) forms around the disc as a result of the substance diffusing from the disc into the agar during incubation. The diameter of this inhibitory zone is then measured to determine the agent's antibacterial efficacy.[23]

d. Anti-androgen hypersecretion of sebum

Sebaceous gland (SG) hyperplasia and excessive sebum production are frequently assessed using the fuzzy rat paradigm. This model is a genetically altered breed that was created by crossing hairless and hairy albino rats. It is especially helpful for researching how medications or bioactive substances can lessen the glandular and ductal hyperplasia linked to acne. With the exception of the control and vehicle-treated groups, the test formulation is mixed in an appropriate vehicle, such as water or alcohol, and applied topically to the dorsal skin of male rats for about two months. Both epidermal sheet preparations and frozen skin slices are used to quantify the size of sebaceous gland lobules and ducts after therapy. In order to see sebaceous structures, the tissues are stained with an osmium–potassium dichromate solution after around eight weeks. By counting the amount of bromodeoxyuridine (BrdU)-positive cells using immunohistochemical labeling, the proliferation of cells within the sebaceous glands is evaluated. By assessing test chemicals' capacity to decrease sebaceous gland size and activity, this model offers important insight into their potential to prevent acne.[24]

e. Ex vivo pig skin colonization

After being sterilized with 70% alcohol, hairless sections of pig skin are cut and infected with a suspension of bacteria or fungi. Approximately 100 µL of the antimicrobial (ATM) agent is applied to the prepared skin samples (usually 10 per group). Following treatment, the samples are humidified and incubated for 24 hours at 35 ± 2°C. After incubation, a sterile cotton applicator dipped in one milliliter of sterile saline containing an antimicrobial inhibitor is used to swab the skin's surface to measure bacteria growth. Colony counts are then calculated by diluting and plating the obtained samples onto agar media. If no live colonies are found, the test agent's significant antimicrobial action is indicated by the result being below the detectable limit.[25]

7. CONCLUSION

The creation and improvement of experimental models for acne research has advanced significantly in recent years. Among these, models of human sebaceous gland (SG) cell culture offer important chances to comprehend the function of SGs in the pathophysiology of acne. These models are useful instruments for assessing the pharmacological activity of novel anti-acne medications. Sebum-based models also aid in reproducing important aspects of the pilosebaceous unit (PSU) and promote Propionibacterium acnes development and biofilm formation. There are currently numerous in vitro, ex vivo, and in vivo models available to help find and create efficient acne treatments. Researchers can examine the mechanism of action of possible medicinal substances and evaluate their effectiveness thanks to these systems. To further replicate actual skin conditions, sophisticated culture methods, such as three-dimensional skin models, are being created. All things considered, these methods are essential for choosing prospective medication candidates and developing cutting-edge drug delivery systems for better acne treatment.

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