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The Influence of Autophagy on the Onset of Acne

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Abstract

This article reviews the role of autophagy in the pathogenesis of acne. Acne is a common skin disease with a complex etiology, involving multiple contributing factors such as excessive sebum production, hyperkeratinization of the pilosebaceous duct, Propionibacterium acnes infection, and inflammatory responses. Autophagy, as an intracellular degradation and recycling mechanism, plays a crucial role in maintaining cellular homeostasis and regulating metabolic processes. Recent studies have indicated that autophagy also participates in the development and progression of acne. Modulating autophagy can influence sebum production, keratinization, microbial colonization by P. acnes, and inflammatory signaling pathways, thereby offering potential therapeutic benefits for acne. This article summarizes the fundamental concepts and biological functions of autophagy, discusses its specific involvement in acne pathogenesis, and presents current applications of autophagy-targeted therapies, including autophagy activators, anti-androgen treatments, and autophagy inducers. Furthermore, the article outlines the current challenges and future perspectives in autophagy research, suggesting potential directions for further investigation and integrated therapeutic strategies. A deeper understanding of the mechanisms underlying autophagy's role in acne may provide novel insights and therapeutic approaches for the management of this prevalent skin condition.

Keywords

Autophagy; Acne; Pathogenesis; Sebum Production; Hyperkeratinization of the Pilosebaceous Duct

1. Introduction

Acne is a prevalent skin disease worldwide, particularly among adolescents, with substantial effects on physical appearance and psychological well-being [1]. Its complex pathogenesis involves the interplay of multiple factors, including excessive sebum secretion caused by dysregulated sebaceous gland activity, pore obstruction due to abnormal keratinization of the pilosebaceous ducts, and inflammatory responses triggered by the proliferation of Propionibacterium acnes [2-4]. In recent years, with advances in cell biology, an intracellular process known as "autophagy" has attracted increasing attention and has been implicated in the development and progression of various diseases [4-6].

As a cellular self-degradation mechanism, autophagy plays an essential role in maintaining cellular homeostasis, facilitating organelle renewal, and regulating metabolic processes. It achieves this by forming autophagosomes that engulf and degrade damaged organelles, proteins, or other cellular components [7-9]. Autophagy also contributes significantly to skin health and pathology, and dysfunction in this process may directly or indirectly affect multiple aspects, including skin barrier integrity, inflammatory responses, and cell differentiation [10].

Given the multifactorial nature of acne pathogenesis and the central role of autophagy in cellular regulation, investigating the specific mechanisms by which autophagy contributes to acne development may enhance our understanding of its pathophysiology. Moreover, it could provide a scientific foundation for the development of novel and effective therapeutic strategies [11, 12]. Therefore, this article aims to review the impact of autophagy on acne

pathogenesis and explore its potential regulatory roles in sebum secretion, keratinization of the pilosebaceous ducts, P. acnes infection, and inflammatory responses. The goal is to offer new insights and perspectives for the clinical management of acne [13, 14].

2. Basic Concepts and Functions of Autophagy

Autophagy is a cellular process first described by Ashford and Porter in 1962, following their observation of intracellular "self-eating" phenomena [15]. It involves the formation of autophagosomes, which originate from the non-ribosome-associated regions of the rough endoplasmic reticulum. These structures encapsulate intracellular components such as organelles and proteins marked for degradation, and subsequently fuse with lysosomes to form autophagolysosomes, where the enclosed materials are broken down to meet the cell's metabolic needs and support organelle renewal [16, 17].

Autophagy occurs in both physiological and pathological conditions, and its functional role—whether protective or detrimental—remains incompletely understood, particularly in the context of cancer research [18]. It plays a crucial role in processes such as the cellular response to starvation, maintenance of normal cellular activity, and tissue-specific functions [19]. For instance, during nutrient deprivation, amino acid deficiency triggers autophagy to degrade macromolecules and generate metabolic intermediates. In normal cellular function, autophagy facilitates the turnover of cellular proteins to maintain homeostasis. In specific tissues, such as dopaminergic neurons of the substantia nigra, autophagosome formation is essential for the synthesis of neuromelanin, which requires the sequestration of dopamine quinone within autophagic vesicles.

The formation of autophagosomes begins with the enclosure of cytoplasmic components, including organelles such as mitochondria, by an isolation membrane, resulting in a double-membrane autophagosome. This structure then fuses with endosomes to form intermediate autophagic vesicles and ultimately merges with lysosomes to generate degradative autophagosomes. Autophagosomes typically range from 300 to 900 nm in diameter, with an average size of approximately 500 nm. Their luminal contents commonly include cytoplasmic material and organelles such as mitochondria, endosomes, and peroxisomes. Compared to other organelles, autophagosomes have a very short half-life of about 8 minutes, highlighting the dynamic and responsive nature of autophagy to environmental changes [20].

Autophagy is categorized into three main types based on the mechanisms by which cellular materials are delivered to lysosomes: macroautophagy, microautophagy, and chaperone-mediated autophagy [21]. Macroautophagy involves the enclosure of cytoplasmic cargo by membranes derived from the endoplasmic reticulum, Golgi apparatus, or plasma membrane, forming autophagosomes that later fuse with lysosomes for degradation. Microautophagy occurs when the lysosomal membrane directly engulfs cytoplasmic components or organelles through invagination. In chaperone-mediated autophagy, cytoplasmic proteins bind to molecular chaperones and are transported into the lysosomal lumen for enzymatic degradation. This review focuses exclusively on the role of macroautophagy in acne pathogenesis and will refer to it simply as autophagy in subsequent discussions.

3. The Role of Autophagy in the Pathogenesis of Acne

In recent years, accumulating evidence has demonstrated that autophagy, as a critical intracellular clearance and recycling mechanism, plays an increasingly recognized role in the development of acne. This article aims to provide a comprehensive review of the diverse roles autophagy may play in the pathogenesis of acne.

3.1 Autophagy and Sebum Secretion

The fundamental pathological basis of acne is excessive sebum secretion. During adolescence, elevated androgen levels stimulate increased activity of sebaceous glands, leading to excessive oil production. When this excess sebum combines with shed epidermal cells, it can clog pores, form comedones, and ultimately progress into acne. Among androgens, testosterone and its derivative dihydrotestosterone (DHT) have particularly significant effects on sebaceous gland function [22].

Studies suggest that androgens may promote the development of acne by inhibiting autophagy and impairing sebum degradation. Specifically, androgens stimulate the proliferation and secretory activity of sebaceous gland cells while simultaneously suppressing autophagosome formation and downregulating key autophagy-related genes such as LC3 and Beclin-1. This suppression prevents the normal degradation and excretion of lipids within sebaceous cells, resulting in the accumulation of excessive sebum. The accumulated sebum, together with desquamated epidermal cells, forms keratin plugs that obstruct the pilosebaceous duct, leading to comedone formation [23-25].

Further studies have shown that male groundhogs exhibit more developed sebaceous glands, higher androgen levels, and reduced autophagic activity compared to females. This observation suggests that androgens may be a key factor in suppressing autophagy within sebaceous glands. Therefore, interventions that inhibit androgen secretion or block androgen receptor activity may alleviate autophagy inhibition and reduce abnormal sebum production, offering a potential therapeutic strategy for acne.

3.2 Autophagy and Keratinization of Follicular Sebaceous Gland Ducts

Follicular blockage is primarily caused by excessive keratinization of the stratum corneum at the follicular orifice. This obstruction impedes sebaceous secretion and excretion, not only intensifying sebum accumulation but also promoting the proliferation of microorganisms such as *Propionibacterium acnes*, thereby further exacerbating acne development [26].

Autophagy plays a crucial role in maintaining organelle renewal and tissue homeostasis, and may therefore regulate keratinocyte differentiation and metabolism. Experimental evidence has shown that autophagy activators can increase the expression of differentiation marker proteins in cultured keratinocytes, suggesting that autophagy contributes to the normal differentiation and function of keratinocytes. Impaired autophagy may lead to dysregulated keratinocyte differentiation, resulting in follicular occlusion and the progression of acne.

Specifically, autophagy may preserve the morphology and function of keratinocytes by modulating lipid metabolism and facilitating organelle turnover. When autophagy is suppressed, lipid metabolism becomes dysregulated, leading to hyperkeratinization of the stratum corneum. Concurrently, impaired organelle renewal disrupts keratinocyte function, further aggravating follicular blockage and acne formation [27, 28].

3.3 Autophagy with Propionibacterium Acnes Infection

Propionibacterium acnes is a primary pathogenic factor in acne. It is a lipophilic, Gram-positive anaerobic bacterium predominantly localized in human pilosebaceous units. *P. acnes* infection stimulates sebaceous gland secretion, promotes hyperkeratinization of the sebaceous ducts, and creates an anaerobic environment that facilitates bacterial proliferation. This process activates the host immune system, induces inflammatory responses, and leads to the production of inflammatory mediators such as tumor necrosis factor α (TNFα) and interleukins (ILs), thereby contributing to the development of acne [29].

Autophagy not only supports cellular energy metabolism and the biosynthesis of structural proteins but may also modulate inflammatory responses by regulating the production of inflammatory mediators and clearing damaged organelles. During acne-related inflammation, the infiltration and activation of various immune cells—such as T cells, macrophages, and neutrophils—at the lesion site result in the release of inflammatory mediators. These mediators recruit additional immune cells, amplifying the inflammatory cascade and perpetuating a self-sustaining cycle [30].

Autophagy may mitigate inflammation by eliminating damaged organelles and modulating the production of inflammatory cytokines. On one hand, autophagy degrades damaged organelles and misfolded proteins, reducing cellular stress and limiting inflammatory signaling. On the other hand, autophagy can regulate the intensity and duration of inflammation by influencing the production and release of inflammatory mediators. For example, autophagy can inhibit the activation of inflammatory signaling pathways such as NF-κB, thereby reducing the synthesis and secretion of pro-inflammatory cytokines like TNFα and IL-1β. These effects collectively contribute to the alleviation of acne-related inflammation and clinical symptoms [31, 32].

3.4 Autophagy and Immune Response

Immunological factors also play a crucial role in the pathogenesis of acne. The inflammatory process of acne involves the expression of various immune-related molecules, including Toll-like receptors (TLRs) and antimicrobial peptides such as LL-37. These molecules participate in local immune responses and regulate the progression of inflammation. In addition, sebaceous gland cells contribute to acne-related inflammation by secreting lipid mediators such as lipoxins and arachidonic acid derivatives, which modulate the activity and differentiation of inflammatory cells [33-35].

As an intracellular clearance and recycling mechanism, autophagy may regulate immune responses. On one hand, autophagy can eliminate damaged organelles and proteins within cells, thereby reducing the accumulation of immunogenic substances and alleviating both immune and inflammatory responses. On the other hand, autophagy can also influence the direction and intensity of immune responses by modulating the activation and differentiation of immune cells. For example, autophagy promotes the maturation of dendritic cells and enhances their antigen-presenting function, which in turn boosts T cell activation and differentiation, thereby shaping the immune response [35].

Autophagy may alleviate acne symptoms by modulating immune and inflammatory responses during disease progression. Specifically, autophagy can remove damaged sebaceous gland cells and keratinocytes, reducing the buildup of immunogenic materials and thus dampening immune activation and inflammation. Additionally, autophagy can regulate the extent and duration of inflammation by influencing immune cell function. For instance, autophagy promotes the production and release of anti-inflammatory cytokines while suppressing the synthesis and secretion of pro-inflammatory cytokines, thereby mitigating inflammatory responses and alleviating acne symptoms.

4. Innovative Applications of Autophagy in the Treatment of Acne

Research on the role of autophagy in acne has been rapidly expanding, revealing its significant potential in the treatment of this condition. By modulating autophagy, multiple pathological processes involved in acne development can be influenced, including sebum production, follicular keratinization, inflammation, and immune responses. The following section will provide an in-depth discussion of the therapeutic applications of autophagy in acne management.

4.1 Innovative Applications of Autophagy Activators

In recent years, with the deepening understanding of autophagy mechanisms, researchers have uncovered that autophagy signaling plays a critical role in sebaceous gland adipogenesis and epidermal differentiation. Therefore, autophagy activators show promising therapeutic potential in the management of acne [36-38].

A clinical study investigating the effects of autophagy activators on acne-prone skin demonstrated that an 8-week double-blind, randomized controlled trial revealed significant reductions in the number of closed comedones, decreased cutaneous lipid content, and improved epidermal barrier function as indicated by reduced transepidermal water loss. These results provide robust evidence supporting the therapeutic application of autophagy activators in acne treatment [39, 40].

Further investigations have also shown that after 8 weeks of autophagy activator use, patients exhibited decreased levels of squalene and increased cholesterol levels on the skin surface. These changes suggest that autophagy activators can modulate the composition of cutaneous lipids, offering new insights into their role in regulating sebum secretion and acne pathogenesis [41-44].

4.2 Synergistic Effects of Anti-androgen Therapy with Autophagy

Anti-androgen therapy, a widely used strategy in acne management, alleviates acne symptoms by reducing abnormal sebum secretion through the suppression of androgen production. However, recent studies have revealed that anti-androgen therapy not only directly inhibits sebum secretion but may also further improve acne pathology by modulating the autophagy process [45-47].

Research has shown that following anti-androgen treatment, levels of dihydrotestosterone (DHT) and sebum secretion are significantly reduced, while autophagy markers such as LC3-II and P62 are markedly elevated. These findings suggest that anti-androgen treatment effectively lowers androgen levels and concurrently enhances cellular autophagy activity. This upregulation of autophagy may be linked to the relief of androgen-mediated suppression of autophagy. Therefore, anti-androgen therapy may exert its therapeutic effects on acne through a dual mechanism: reducing abnormal sebum production and restoring autophagy function [48-50].

4.3 Development and Frontier Applications of Autophagy Inducers

Autophagy inducers are a class of compounds capable of stimulating autophagy in cells, thereby reducing inflammatory responses and alleviating acne symptoms through the clearance of damaged organelles and abnormally accumulated proteins. A variety of autophagy inducers have been identified and reported to exhibit therapeutic potential in the treatment of multiple diseases [51].

The application of autophagy inducers in acne therapy shows considerable promise. By promoting autophagy, these inducers can remove damaged sebaceous gland cells and keratinocytes, thereby reducing follicular obstruction and inflammatory reactions. Additionally, autophagy inducers may modulate the composition of cutaneous lipids and enhance skin barrier function, further contributing to the alleviation of acne symptoms [52].

Future research is expected to focus on the development of autophagy inducers specifically targeting acne. Scientists may design novel autophagy inducers with improved specificity and reduced toxicity through structural optimization and activity screening. Moreover, combination therapies involving autophagy inducers and other treatment modalities—such as topical agents, oral medications, or phototherapy— could be explored to evaluate potential synergistic effects and enhance overall acne treatment outcomes.

5. Challenges and Prospects of Autophagy Research

Although the role of autophagy in acne pathogenesis has attracted growing attention, significant challenges remain. First, the precise mechanisms underlying autophagy are not yet fully understood, and further research is needed to clarify its functional dynamics. Second, the specific involvement of autophagy in acne development requires in-depth investigation, particularly how autophagy regulates sebum production, keratinization of the follicular sebaceous ducts, *Propionibacterium acnes* infection, and inflammatory responses [53].

Future research directions may include the following areas: 1. In-depth exploration of autophagy mechanisms: Strengthening studies on the conditions under which autophagy occurs, its classification, and methodological approaches to better understand its role in cellular metabolism, organelle turnover, and tissue homeostasis. 2. Investigation of autophagy's specific roles in acne development: Utilizing cell culture models, animal experiments, and clinical studies to elucidate how autophagy influences sebum secretion, follicular keratinization, microbial infection, and inflammation, thereby identifying novel therapeutic targets for acne treatment.

6. Discussion

Autophagy, as a fundamental cellular self-degradation mechanism, plays a vital role in maintaining cellular homeostasis and responding to environmental stress. Dysregulation of autophagy has been implicated in key pathological processes of acne, a common dermatological condition, including excessive sebum production, follicular hyperkeratinization, and inflammatory responses. Therefore, modulating autophagy has emerged as a promising therapeutic strategy for acne.

First, the application of autophagy activators offers novel opportunities for acne treatment. By inducing autophagy, these agents can reduce the number of closed comedones, decrease cutaneous lipid content, and enhance skin barrier function, thereby alleviating acne symptoms. This finding not only provides a solid theoretical foundation for the use of autophagy activators in acne therapy but also opens new avenues for the development of next-generation acne treatments.

Second, the synergistic interaction between anti-androgen therapy and autophagy further broadens the therapeutic options for acne. Anti-androgen therapy suppresses androgen production and reduces abnormal sebum secretion, while autophagy activation may further eliminate damaged sebaceous gland cells and keratinocytes, thereby decreasing follicular obstruction and inflammation. This dual mechanism enhances the efficacy of anti-androgen therapy and lays the groundwork for more targeted and effective treatment strategies in the future.

In addition, the development and application of autophagy inducers have introduced new possibilities for acne management. By promoting autophagy, these inducers can remove damaged organelles and misfolded proteins, reduce inflammatory responses, and improve acne symptoms. Numerous autophagy inducers have been identified and demonstrated therapeutic potential in various diseases. Future research is expected to focus on the development of acne-specific autophagy inducers, which may offer safer and more effective treatment options for acne patients.

However, the application of autophagy in acne therapy still faces several challenges. These include how to precisely regulate autophagy to avoid potential cellular damage caused by excessive activation, how to identify autophagy modulators with high efficacy and low toxicity, and how to integrate autophagy-based therapies with existing treatment modalities to develop comprehensive and personalized therapeutic strategies. Addressing these questions will require continued scientific investigation to advance the development of safer and more effective autophagy-targeted therapies.

7. Conclusion

In summary, autophagy demonstrates significant potential in the treatment of acne. By modulating the autophagy process, multiple pathological features of acne—such as excessive sebum production, follicular hyperkeratinization, and inflammatory responses—can be effectively ameliorated, offering patients a safer and more effective therapeutic option. With further elucidation of autophagy mechanisms and the development of novel autophagy-targeted therapies, autophagy is expected to play an increasingly important role in acne management. Meanwhile, interdisciplinary collaboration and the integration of comprehensive treatment strategies will be crucial for advancing the clinical application of autophagy-based therapies in dermatology. It is anticipated that autophagy therapy will become a key therapeutic approach in the treatment of acne in the near future, providing new hope for a broader patient population.

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