

Potential Applications of Chitosan in Seborrheic Dermatitis and Other Skin Diseases: A Comprehensive Review

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Abstract: This review article explores the potential applications of chitosan, a natural polysaccharide derived from crustacean shells, in the treatment of seborrheic dermatitis (SD) and other skin diseases. SD is a common chronic inflammatory skin condition characterized by erythema, scaling, itching, and an oily appearance, predominantly affecting areas rich in sebaceous glands. Current treatments, including antifungal agents, corticosteroids, and calcineurin inhibitors, offer symptomatic relief but have limitations in long-term use due to side effects and resistance issues. Chitosan exhibits excellent biocompatibility, biodegradability, and broad-spectrum antibacterial properties, making it a promising candidate for SD treatment. This review highlights chitosan's multifunctional properties such as antimicrobial, anti-inflammatory, sebum-regulating, and barrier-enhancing effects, which are closely related to the pathogenesis of SD. Additionally, the article summarizes the applications of chitosan in other skin conditions, including wound healing, infectious skin diseases, and atopic dermatitis, demonstrating its broad therapeutic potential. Through this comprehensive evaluation, the review aims to provide a theoretical foundation for clinical research on chitosan in SD and support the development of new, safer, and more effective treatment options for various skin conditions.

Keywords: chitosan, seborrheic dermatitis, other skin diseases, potential application

Introduction

Seborrheic dermatitis (SD) is a common chronic and/or recurrent inflammatory skin condition, typically characterized by erythema, scaling, itching, and oily appearance. It most frequently occurs in areas rich in sebaceous glands, such as the scalp, face, chest, and back.¹ While the exact etiology of SD is not completely understood, it is generally believed to result from a combination of factors, including increased sebum production, *Malassezia* yeast infection, immune system abnormalities, and impaired skin barrier function. SD is a globally prevalent skin disease with an overall prevalence of 4.38%, but this proportion varies significantly by region and age group. A subgroup analysis of the study conducted by Polaskey MT et al showed that the prevalence in adults was 5.64%, significantly higher than in children (3.7%) and newborns (0.23%). Geographically, the highest prevalence is observed in South Africa at 8.82%, while the lowest is found in India at 2.62%.² Due to its frequent occurrence on the face and other visible areas, SD not only negatively impacts patients' daily lives but also severely affects their self-confidence and quality of life, causing great distress. Women, younger patients, and those with higher educational levels are especially affected.³

Current treatments for SD primarily include topical antifungal agents (such as ketoconazole, and miconazole nitrate), corticosteroids (such as hydrocortisone, and fluocinolone), and calcineurin inhibitors (such as pimecrolimus, tacrolimus). Other widely used methods include cleansers containing lithium gluconate/succinate, selenium sulfide, coal tar, tea tree oil, or salicylic acid, as well as phototherapy.^{4,5} Although these methods can effectively control symptoms, reduce inflammation, and

alleviate itching in the short term, there are many limitations in long-term treatment. For instance, antifungal agents may lead to resistance and could cause irritant contact dermatitis, burning, and stinging. Long-term use of corticosteroids may result in side effects such as telangiectasia, skin atrophy, folliculitis, hypertrichosis, and hypopigmentation. Calcineurin inhibitors are not approved for SD treatment in the United States but are often used as a second-line treatment. Their efficacy is typically insufficient, and long-term use poses risks of skin malignancies and lymphoma.^{6,7} Furthermore, existing treatments are mostly symptomatic and do not cure the condition, leading to recurrent symptoms. Therefore, finding safer, more effective, and long-term treatment options remains an urgent issue to be addressed.

Chitosan, also known as deacetylated chitin and soluble chitin, is a product of partial deacetylation of natural polysaccharide chitin and is the only naturally abundant cationic alkaline polysaccharide. Chitosan is highly regarded in the biomedical and cosmetic industries due to its excellent biocompatibility, biodegradability, cell affinity, safety, non-toxicity, broad-spectrum antibacterial, hemostatic, anti-inflammatory, wound healing, and other biological properties.⁸

Research indicates that although the application of chitosan in the treatment of seborrheic dermatitis has not been widely studied or verified, its various biological functions in skin care are closely related to the main pathogenesis of seborrheic dermatitis, demonstrating its potential therapeutic value. This review aims to explore the potential application of chitosan in seborrheic dermatitis and further introduce its broad application in the treatment of other skin diseases, highlighting its multifunctional properties. Through this comprehensive evaluation, we hope to provide a theoretical foundation for clinical research on chitosan in seborrheic dermatitis and support the development of new, safer, and more effective treatment options for seborrheic dermatitis and other skin diseases.

Etiology and Pathogenesis of SD

The pathogenesis of SD is complex and diverse, involving multiple interconnected internal and external factors that work together to cause the onset and progression of the condition. The main pathogenic mechanisms include:

Microbiome Dynamics

Numerous studies have indicated that the lipophilic yeast *Malassezia* is the primary pathogenic microbe on the skin of patients with SD.^{9–11} As a common skin commensal microorganism, *Malassezia* exhibits a significant increase in quantity and activity among patients with SD. Research has shown that *Malassezia* possesses lipase characteristics, capable of hydrolyzing triglycerides to release unsaturated fatty acids such as oleic acid and arachidonic acid. These metabolic products may lead to abnormal keratinization and inflammatory reactions. Additionally, *Malassezia* can activate the skin's immune response through pattern recognition receptors, inflammasomes, NF- κ B, and IL-1 β , thereby exacerbating inflammation and worsening symptoms.^{6,12}

Apart from *Malassezia*, dysbiosis of the bacterial microbiome has also been confirmed in the lesions of SD. Studies have found that the genera *Staphylococcus* and *Pseudomonas* are potential biomarkers for SD. Compared to healthy subjects, patients with SD show a relative increase in the abundance of *Staphylococcus* and *Corynebacterium*, while the abundance of *Cutibacterium/Staphylococcus* decreases.^{11,13}

Inflammation and Immune Dysregulation

The higher incidence of SD in immunosuppressed patients (such as those with HIV, chronic alcoholic pancreatitis, and hepatitis B) suggests that immunological mechanisms play a crucial role in its pathogenesis.¹⁴ In SD, elevated levels of various inflammatory markers have been observed, including IL-1 α , IL-1 β , IL-2, IL-4, IL-6, IL-8, IL-10, IL-12, TNF- α , β -defensin, IFN- γ , nitric oxide, and histamine. Innate immunity is essential in the initial immune response against *Malassezia*. In susceptible individuals, when epidermal cells interact with *Malassezia*, they activate pattern recognition receptors, such as Toll-like receptor-2, NOD-like receptors, and C-type lectin receptors, further promoting the inflammatory response.^{15,16}

Sebaceous Glands and Lipids

The amount of sebum and/or the composition of skin surface lipids may play an important role in the etiology of SD. There is a strong correlation between SD and the distribution and activity of sebaceous glands, and excessive sebum provides an ideal environment for the growth of pathogenic microorganisms such as *Malassezia*. However, the role of

sebaceous glands remains controversial. Some studies indicate that SD patients may have normal sebum secretion levels, and individuals with excessive secretion may not necessarily develop SD. Therefore, although sebaceous glands are closely related to the condition, sebum itself is not the decisive factor.^{16,17} Moreover, while changes in the sebum composition of SD patients remain unclear, the role of *Malassezia* does lead to changes in the lipid composition of the skin surface. *Malassezia* can convert saturated fatty acids into irritating unsaturated fatty acids, thereby increasing an individual's sensitivity to irritating free fatty acids and the inflammatory response they trigger. This sensitivity ultimately affects an individual's susceptibility to SD and may also increase the risk of fatty acid-induced skin barrier disruption.¹⁸

Epidermal Barrier

Recent evidence indicates a strong correlation between the integrity of the epidermal barrier and the severity of SD. A healthy skin barrier effectively prevents moisture loss and the intrusion of harmful external substances. However, in patients with SD, epidermal differentiation is abnormal, keratinocyte morphology changes, desmosomes are reduced, and the composition of intercellular lipids is altered (such as ceramide subclass disorder, impaired chain length, and increased chain unsaturation). SD often presents with significant erythema, neovascularization, increased epidermal thickness, and aggravated surface roughness, as well as increased transepidermal water loss (TEWL). These findings suggest that impaired epidermal barrier function plays a role in the exacerbation of dandruff. Recent genetic studies indicate that compromised barrier function may even directly lead to seborrheic dermatitis-like conditions.^{19,20}

Other Factors

Environmental and genetic factors also significantly impact the occurrence and development of SD. Environmental factors, such as climate changes (particularly dry and cold seasons), dietary patterns (such as Western diets), psychological stress, and lifestyle, may trigger or worsen symptoms.²¹ Genetic factors render some individuals more susceptible to SD, potentially influencing the activity of sebaceous glands, immune system function, or the integrity of the skin barrier through various mechanisms. In some families, the occurrence of SD shows a certain genetic predisposition.²²

The pathophysiology of SD is illustrated in [Figure 1](#). Multiple factors contribute to the onset and progression of SD. While the proliferation of *Malassezia* yeast on the skin has traditionally been considered the primary cause, recent research highlights the potential central role of immune system dysregulation and alterations in skin barrier function in the disease's pathogenesis.²³

Characteristics of Chitosan

Chitosan is a linear polysaccharide obtained through the deacetylation of chitin, primarily found in the shells of crustaceans and the cell walls of certain fungi. In many physiological conditions, chitosan can be protonated and carry a positive charge ([Figure 2](#)).²⁴ As one of the few naturally occurring cationic polyelectrolytes, chitosan is soluble in acidic aqueous solutions and exhibits unique physicochemical and biological properties. It demonstrates excellent biocompatibility and biodegradability, does not accumulate in the human body, and is non-irritating, non-toxic, and non-immunogenic. The cationic nature of chitosan imparts significant bioactivity in skin applications, promoting wound healing and exhibiting antibacterial, anti-inflammatory, immunomodulatory, antioxidant, film-forming, and moisturizing effects. Thus, it holds substantial application value in the fields of medicine, cosmetics, and biomaterials.^{25,26}

Antimicrobial Activity of Chitosan

Chitosan is a natural broad-spectrum antimicrobial agent that effectively inhibits the growth of various pathogenic microorganisms, such as *Malassezia*,²⁷ *Candida albicans*,²⁸ *Staphylococcus aureus*,²⁹ *Cutibacterium acnes*,³⁰ and *Pseudomonas aeruginosa*,³¹ among other bacteria, fungi, and viruses. The current hypotheses regarding chitosan's antimicrobial mechanisms mainly include the following: ① Disruption of cell walls or membranes: Positively charged chitosan can interact with negatively charged substances in cell membranes and walls, leading to the rupture of bacterial biofilms or cell walls, causing leakage of cellular components such as proteins and resulting in the death of bacteria and fungi. ② Targeting intracellular substances: Low molecular weight chitosan or its hydrolysates can enter the microbial nucleus and inhibit the synthesis of DNA/RNA and proteins, leading to the death of microorganisms. ③ Chelation of

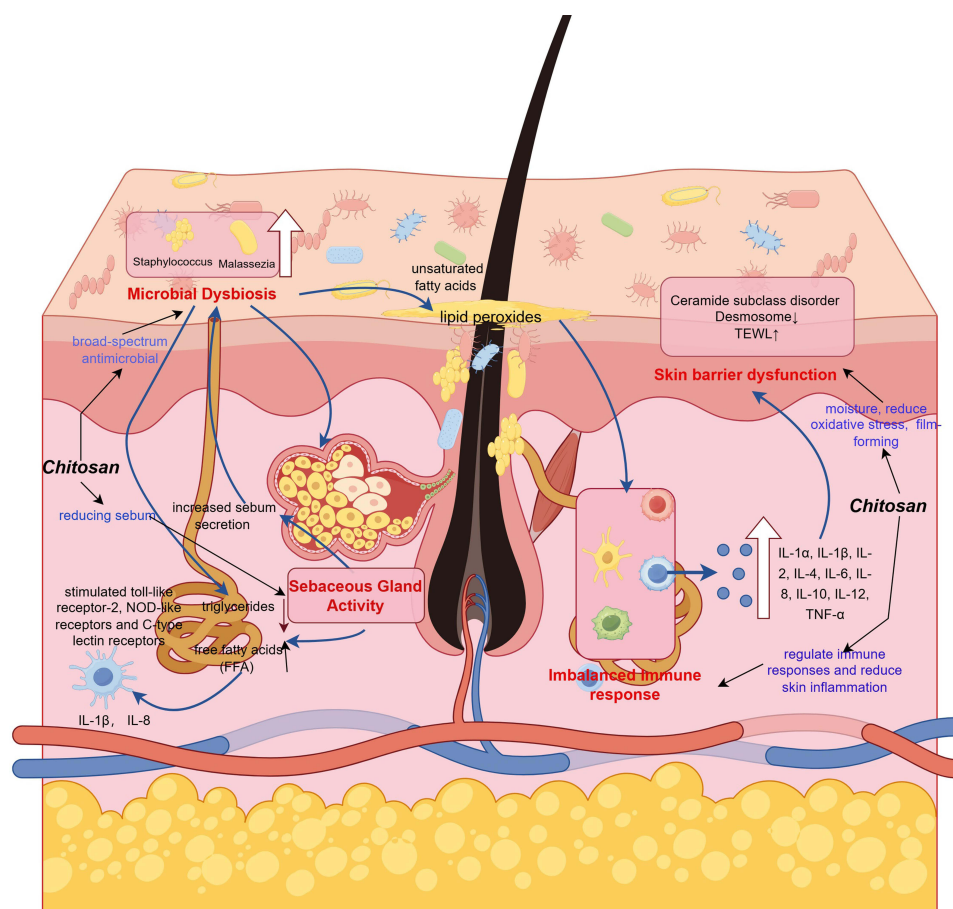


Figure 1 Pathophysiology of SD (By Figdraw). This figure illustrates the main pathological mechanisms of SD.

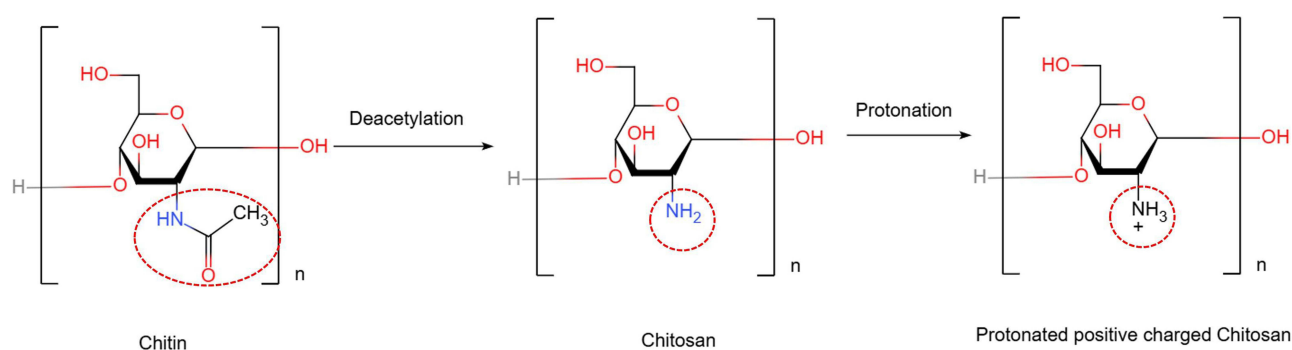


Figure 2 The conversion of chitin to chitosan. This figure illustrates the conversion of chitin into chitosan through deacetylation. Under various physiological conditions, chitosan can become protonated and carry a positive charge.

essential nutrients: Chitosan can selectively chelate metal ions that play a key role in microbial growth, thereby leading to toxin production and inhibiting microbial growth. ④ Formation of a dense polymer membrane on the cell surface: High molecular weight chitosan can form a dense polymer layer on the surface of bacteria, preventing the intake of nutrients or the excretion of metabolites, leading to metabolic disruption and bacterial death.^{32–34}

Anti-Inflammatory and Immune-Regulating Effects of Chitosan

Chitosan can regulate immune responses and reduce skin inflammation. It can enhance dendritic cell activation and T-cell responses by participating in the cGAS-STING pathway and NLRP3 inflammasome activation. The cGAS-STING pathway is activated in response to double-stranded DNA (dsDNA), where cGAS binds to host nucleic acids to produce cyclic dinucleotides, which subsequently bind to STING in the endoplasmic reticulum. STING then further interacts with IKK or TBK-1 and relocates to the perinuclear Golgi apparatus, ultimately activating the transcription factors NF- κ B and IRF-3. Chitosan has been shown to activate this downstream pathway, characterized by the absence of pro-inflammatory cytokine production and the induction of type I interferons (IFN). The activation of the NLRP3 inflammasome requires two steps: first, the assembly of the NLRP3 complex and the upregulation of pro-IL-1 β expression; second, caspase-1 cleaves pro-IL-1 β into its bioactive form. Research indicates that chitosan activates the NLRP3 inflammasome through mtROS-dependent mechanisms, potassium ion (K) efflux, and lysosomal destabilization.^{35–37}

Anti-Sebum Effects of Chitosan

Chitosan is a positively charged cationic polymer that can effectively bind with lipids, reducing sebum secretion from the skin and forming a protective film to prevent sebum accumulation. A clinical trial conducted by Tangkijngamvong N et al involving 40 patients with seborrhea over four weeks demonstrated that chitosan particles can reduce facial sebum secretion.³⁸ Theerawattanawit C et al also confirmed that chitosan significantly lowers sebum levels.³⁹

Effects of Chitosan on Improving the Epidermal Barrier

Chitosan has a significant effect on improving the epidermal barrier. Firstly, chitosan can adsorb onto the negatively charged skin surface, where its molecular chain contains numerous hydrophilic groups such as hydroxyl and amino groups, which help increase the moisture content of the stratum corneum. Its excellent moisturizing properties assist the skin in retaining water, preventing dryness and dehydration, thereby enhancing the skin's barrier function.⁴⁰ Secondly, chitosan has a notable ability to scavenge various free radicals, reduce oxidative stress, protect skin cells, and improve the damage resistance of the epidermal barrier.⁴¹ Additionally, chitosan promotes wound healing by accelerating hemostasis, inhibiting bacteria, stimulating granulation tissue growth, and regulating cytokines, acting at various stages of wound healing to expedite the restoration of epidermal integrity.⁴² It is noteworthy that chitosan also possesses excellent film-forming properties, capable of forming a breathable protective film on the skin surface. This protective film not only locks in moisture to prevent water loss but also defends against external irritants and pollutants, further strengthening the skin barrier function.⁴³

Applications of Chitosan and Its Derivatives in Other Skin Diseases

Chitosan is a natural polysaccharide extracted from crustacean shells. In addition to its use in seborrheic dermatitis, it has been widely applied in treating other skin conditions due to its biocompatibility, biodegradability, and antibacterial properties. Below are its key applications in the skin area, with relevant studies summarized in [Table 1](#).

Promoting Wound Healing

Skin wound healing is a complex process involving tissue regeneration after trauma, typically progressing through four stages: hemostasis, inflammation, proliferation, and remodeling. Chitosan supports all these stages, enhancing healing. Clinical trials show that chitosan dressings reduce healing time, improve wound color match, and better manage exudate compared to traditional dressings.⁵⁷

Infectious Skin Diseases

Infectious skin diseases are caused by pathogens like viruses, bacteria, and fungi. These include conditions like folliculitis, athlete's foot, ringworm, and scabies. The main goal in treatment is to eliminate the pathogens.⁴⁷ Chitosan has broad-spectrum antibacterial properties, good biocompatibility, biodegradability, and non-toxicity. Chitosan and its derivatives are used as excipients, drug delivery systems, or therapeutic agents, and various formulations have been developed, including gels, membranes, microspheres, nanofibers, nanoparticles, and sponges.^{48,58}

Table 1 Applications of Chitosan and Its Derivatives in Other Skin Diseases

Application Area	Author(s)	Study Type	Findings	Reference
Wound Healing	Haque AE et al, 2020	A Comparative Clinical Study	Sixty patients participated in the study. The results indicate that Chitosan enhances soft tissue healing, improves color matching, and reduces scarring more effectively than Collagen.	[44]
	Mo X et al, 2015	An open multicenter comparative randomized clinical study	Seventy-nine patients completed the study. After 4 weeks, the chitosan group showed a greater wound area reduction ($65.97 \pm 4.48\%$) compared to the vaseline gauze group ($39.95 \pm 4.48\%$). Pain levels were lower in the chitosan group (1.12 ± 0.23 vs 2.30 ± 0.23), and wound depth was shallower (0.30 ± 0.48 cm vs 0.54 ± 0.86 cm)	[45]
	Hu J et al, 2023	A prospective, randomised, single-blind, positive control clinical trial	A total of 80 patients with second-degree deep burns were enrolled, with 40 in the study group (chitosan wound dressing combined with wet compress) and 40 in the control group (wet compress alone). The study group had a significantly shorter wound healing time compared to the control group (19.53 ± 2.74 days vs 24.78 ± 4.86 days). On day 14, the wound healing percentage was higher in the study group (65.00%) than in the control group (37.50%) ($P = 0.014$). After 3 months, the study group had a lower scar score (6.00 ± 0.98) compared to the control group (8.77 ± 1.19) ($P = 0.031$).	[46]
Infectious skin diseases	Mohite P et al, 2023	A review	This review collects and analyzes recent research data on chitosan-based hydrogels for skin infection treatment, evaluating their ability to inhibit microbial growth and alleviate infection-related symptoms. In addition, the review will explore the safety of chitosan-based hydrogels, particularly any potential adverse effects associated with their use.	[47]
	Dilnawaz F et al, 2023	A review	The review explores the use of chitosan nanoparticles (CNPs) in preventing infectious diseases.	[48]
	Banche G et al, 2015	Through microbiological, biochemical, and sonophoresis assays	Chitosan-shelled oxygen-loaded nanodroplets (OLNs) and oxygen-free nanodroplets (OFNs) showed cytostatic activity against MRSA and Candida albicans. OLN were non-toxic to keratinocytes. Ultrasound (US) treatment improved OLN transdermal delivery.	[49]
	Friedman AJ et al, 2013	Vitro studies	Chitosan- and alginate-based NPs demonstrated direct antimicrobial activity against Propionibacterium acnes in vitro, along with anti-inflammatory properties, which are beneficial for treating skin conditions with both infectious and inflammatory components.	[50]
	Donalisio M et al, 2018	Vitro studies	The chitosan nanoparticles enhanced the skin permeability of acyclovir, and the chitosan nanoparticles loaded with acyclovir exhibited higher antiviral activity against HSV-1 and HSV-2 strains compared to free acyclovir.	[51]
	Sinani G et al, 2024	A review	This review examines recent applications of chitosan in the prevention and treatment of infectious diseases, highlighting both the possibilities and limitations related to technical and regulatory aspects	[52]
	Meng Q et al, 2021	A review	This review summarizes the application and design considerations of chitosan-based systems for the treatment of infectious diseases, aiming to offer insights into future therapeutic approaches	[53]
Atopic Dermatitis	Wu Y et al, 2023	A review	Hollow manganese dioxide-chitosan hydrogel effectively alleviated AD symptoms by reducing epidermal thickness, mast cell count, and allergen antibody levels through inflammation inhibition and reactive oxygen species (ROS) clearance	[54]
	Lopes C et al, 2015	A Randomized Controlled Trial	In an 8-week study of 78 AD patients, those wearing chitosan-coated cotton pajamas showed a 43.8% improvement in SCORAD ($P = 0.01$), compared to 16.5% in the placebo group ($P = 0.02$). The chitosan group also had a significant improvement in quality of life ($P = 0.02$) and a notable increase in Coagulase-negative Staphylococci ($P = 0.02$).	[55]
	Chuah LH et al, 2023	A review	This review summarizes the recent advancements in chitosan-based drug delivery systems for AD treatment, focusing on studies published from 2012 to 2022.	[56]

Atopic Dermatitis (AD)

Atopic dermatitis (AD) is a prevalent, chronic inflammatory skin condition marked by dry skin, persistent eczematous lesions, and itching. Its pathogenesis is multifactorial, involving disrupted skin barrier function, skin microbiome dysbiosis, and immune system dysregulation.⁵⁹ Chitosan, with its powerful antibacterial, antioxidant, immunomodulatory, moisturizing, and barrier-repairing properties, has emerged as a promising therapeutic agent for AD.

In summary, chitosan and its derivatives show significant potential in treating a wide range of skin diseases beyond seborrheic dermatitis. Its multifunctionality as an antibacterial, anti-inflammatory, and healing-promoting agent makes it a valuable tool in skin care and medical treatments. Further research and clinical trials will be essential to fully harness its therapeutic potential.

Exploration of Chitosan's Relationship with SD

Chitosan has emerged as a promising ingredient in the management of Seborrheic Dermatitis (SD), a common chronic inflammatory skin condition that is primarily characterized by erythema, scaling, and itching. SD is often associated with an imbalance in the skin microbiome, particularly with an overgrowth of *Malassezia* yeast, which is a key contributor to the inflammatory process. The condition is further influenced by sebaceous gland activity, immune system dysregulation, and the integrity of the epidermal barrier. Given these multifactorial causes, treatments targeting one or more of these pathways could be beneficial in managing SD.

Chitosan, a biopolymer derived from chitin, exhibits a broad spectrum of biological activities that are particularly relevant to SD management.⁶⁰ Its antibacterial properties are especially important in controlling the overgrowth of pathogens such as *Malassezia* and *Staphylococcus aureus*, which play a significant role in the development and exacerbation of SD. By inhibiting the proliferation of these microorganisms, chitosan helps restore a more balanced skin microbiome, a key factor in the pathogenesis of SD.

In addition to its antimicrobial effects, chitosan exerts powerful anti-inflammatory actions.^{61,62} It modulates several key inflammatory pathways, including the cGAS-STING pathway and the NLRP3 inflammasome. By regulating these pathways, chitosan helps to reduce the inflammatory cytokine release that contributes to the erythema and discomfort commonly seen in SD. This modulation of the immune response not only alleviates existing symptoms but also prevents future flare-ups, providing long-term relief to affected individuals.

Chitosan's ability to regulate sebaceous gland activity is another crucial factor in its potential to manage SD.^{38,39} Excessive sebum production is often seen in SD patients and contributes to the exacerbation of the condition. Chitosan helps balance sebum secretion, reducing the greasy appearance of affected skin while also preventing clogged pores, which can further aggravate the condition. This sebum-controlling effect is particularly beneficial for individuals with the oily skin type, which is more prone to SD flare-ups.

Furthermore, chitosan forms a protective film on the skin's surface, which serves multiple purposes.⁶³ This film provides an antioxidant effect, which helps neutralize free radicals that may contribute to oxidative stress and skin aging.⁶⁴ By enhancing the skin's barrier function, chitosan helps prevent moisture loss, ensuring that the skin remains hydrated and less prone to irritation and damage. This barrier reinforcement is vital for individuals with SD, whose skin barrier function is often compromised, leading to increased sensitivity and susceptibility to external irritants.

Given these combined effects—antimicrobial, anti-inflammatory, oil-regulating, moisturizing, and barrier-protective—chitosan holds significant promise as a therapeutic agent for managing SD. Its versatile biological activities suggest that it could be integrated into both topical treatments and long-term skin care regimens aimed at reducing flare-ups and improving the overall health of the skin. As such, chitosan warrants further investigation in clinical studies to confirm its efficacy and safety, and to explore its potential for use in combination with other treatment modalities for SD.

Challenges and Limitations of Chitosan Use

Despite the promising potential of chitosan in the treatment of skin diseases, its application in the skin still faces several challenges and limitations. Firstly, its limited solubility in neutral and alkaline solutions restricts its application in aqueous formulations. Additionally, chitosan is not a single polymer but a family of molecules with varying sizes, molecular weights, concentrations, and monomer distributions. This heterogeneity profoundly affects its biological and technical performance,

leading to batch-to-batch variability.^{65,66} Moreover, as chitosan is derived from natural sources, it may contain impurities such as endotoxins and allergens, which can affect its purity and biological safety. Therefore, ensuring the purity, sterility, and quality control of chitosan and its derivatives is crucial. Strict purification and sterilization techniques must be employed to remove potential contaminants and ensure its safety for clinical use.⁴⁷ Another significant limitation is its poor permeability; chitosan has minimal penetration through the skin, which confines its mechanism of action to the skin/external environment interface, limiting its deep therapeutic effects.²⁶ Therefore, improving chitosan's solubility and permeability to enhance its efficacy in skincare products remains a critical area of future research.

Conclusion

Seborrheic Dermatitis (SD) is a complex and chronic inflammatory skin condition that, while manageable with traditional treatments, presents certain limitations, particularly in long-term use due to side effects and resistance issues.⁶⁷ Chitosan, a natural polysaccharide derived from crustacean shells, exhibits excellent antibacterial, anti-inflammatory, moisturizing, and wound-healing properties, making it a promising candidate for the treatment of SD. It not only alleviates the symptoms and signs of SD but also aids in restoring the normal structure and function of the skin.

However, clinical studies on the efficacy and safety of chitosan in the treatment of SD are still relatively limited. This highlights the need for more high-quality randomized controlled trials and long-term observational studies to further validate its clinical application value. Future research should focus on exploring the mechanisms of action of chitosan in SD, including its regulation of sebum secretion, improvement of skin barrier function, and impact on the dynamic changes of the skin microbiome.

Additionally, with the integration of modern technologies such as nanotechnology and biomaterials engineering, new chitosan formulations can be developed to optimize its release characteristics and biocompatibility, thereby enhancing therapeutic effects.⁶⁸ Large-scale clinical trials are also needed to assess the efficacy and safety of these new formulations, laying the foundation for the widespread application of chitosan.

This comprehensive review also underscores the potential applications of chitosan in other skin conditions, including wound healing, infectious skin diseases, and atopic dermatitis, demonstrating its broad therapeutic potential. These studies can not only provide new insights into the treatment of SD but also offer valuable references for the management of various skin conditions, supporting the development of new, safer, and more effective treatment options.

Abbreviations

SD, Seborrheic dermatitis, AD, Atopic dermatitis, CNPs, Chitosan nanoparticles, OLN, Chitosan-shelled oxygen-loaded nanodroplets, OFNs, Oxygen-free nanodroplets, US, Ultrasound, ROS, Reactive oxygen species.

Disclosure

The authors report no conflicts of interest in this work.

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