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# Sulfated Hyaluronan in Dermatology: From Molecular Insights to Disease-Specific Evidence

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**ABSTRACT:** A chemically altered form of natural hyaluronic acid (HA) with enhanced stability and biological activity is called sulfated hyaluronic acid (sHA). Glycosaminoglycans (GAGs), especially HA, are essential for immunological response, skin moisture, and structure. Information about GAGs and their dermatological uses in ailments like psoriasis, acne, and atopic dermatitis is compiled in this review. According to a narrative assessment of research published in PubMed and Scopus up until June 2025, sHA outperforms HA in terms of enzymatic resistance, charge density, water-binding, and antioxidant capabilities. These characteristics help to regulate oxidative stress and inflammation as well as restore the function of the epidermal barrier. Preclinical research shows that sHA is effective in reducing dryness and irritation in skin disorders, with possible advantages for skin restoration and wound healing.To validate these results and create standardized treatment procedures for sHA in dermatology and cosmetology, however, more controlled clinical trials are required.

**KEYWORDS:** Sulfated hyaluronic acid, Dermatology, Skin care, Glycosaminoglycans

## I. INTRODUCTION

A vital glycosaminoglycan in the extracellular matrix, hyaluronic acid (HA) is essential for tissue hydration, flexibility, and cellular communication. It is especially important for preserving the integrity of the skin barrier and promoting wound healing. However, its efficacy is limited by its short half-life and quick disintegration. Chemically modified HA derivatives, such as cross-linked HA for dermal fillers, acetylated HA for increased resistance, carboxymethylated HA for enhanced mucoadhesion, and benzyl ester HA for tissue engineering, have been created to solve these issues and improve stability and performance. Notably, because it resembles naturally occurring sulfated glycosaminoglycans, sulfated hyaluronic acid (sHA) has drawn attention.[1, 2]

Hyaluronic acid (sHA) gains better water-binding ability, antioxidant potential, and a higher negative charge density when sulfate groups are added. Its potential as a humectant and bioactive molecule that can control oxidative stress, inflammation, and tissue regeneration is suggested by these characteristics. However, the majority of the evidence now available about its dermatological applications is preclinical, indicating the need for a comprehensive synthesis of its processes, therapeutic potential, and research gaps. The structural, biochemical, and functional characteristics of sHA are assessed in this review, with a special emphasis on its applicability to inflammatory and barrier-related skin conditions. HA's application in topical preparations and tissue healing matrices is supported by sulfation, which lessens its vulnerability to degradation and enhances interactions with growth factors.[3-5]

## II. BIOSYNTHESIS AND ROLE OF HYALURONIC ACID IN SKIN

A crucial structural component of the extracellular matrix (ECM), hyaluronic acid (HA) is mostly found as a high-molecular-weight polymer (HMW-HA) composed of D-glucuronic acid and N-acetylglucosamine units connected by β-1,3 and β-1,4 glycosidic linkages. Low-

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molecular-weight fragments (LMW-HA) with distinct biological functions can be produced via enzymatic division of HA. In contrast to other glycosaminoglycans (GAGs) made in the Golgi apparatus, three hyaluronan synthase (HAS) isoenzymes—HAS1, HAS2, and HAS3—synthesize HA at the plasma membrane, enabling the formation of massive polymers while preserving cellular integrity.[6]

In addition to interacting with receptors like CD44 and RHAMM to control keratinocyte proliferation and fibroblast migration, hyaluronic acid (HA) is crucial for preserving the structure, hydration, and viscoelasticity of the extracellular matrix (ECM). Molecular weight affects its characteristics; 50–500 kDa HA exhibits superior permeability and stability. While dermal HA fragmentation compromises the integrity of the extracellular matrix, aging reduces epidermal HA, resulting in dryness. While high-molecular-weight HA offers structural integrity and anti-inflammatory advantages in wound healing, low- to medium-molecular-weight HA promotes re-epithelialization and collagen remodeling. However, both forms rapidly breakdown in vivo, limiting therapy efficacy.[7, 8]

The stability, biological activity, and resistance to degradation of hyaluronic acid (HA) have all been improved by chemical modification techniques, especially sulfation. Sulfation imitates the characteristics of naturally sulfated glycosaminoglycans (GAGs), such as heparan and chondroitin sulfate, by increasing HA's negative charge density and affinity for bioactive chemicals.

**Table 1: A comparison between LMW-HA and HMW-HA**

| Property/Function | LMW-HA | HMW-HA |
|---|---|---|
| Molecular Weight | 50-500 kDa | >1000 kDa |
| Enzymatic Degradation | Fast | Slow |
| Water-binding | Moderate | Very high |
| Diffusion through epidermis | High | Low |
| Therapeutic Action | Pro-healing, pro-angiogenic | Anti-inflammatory, space-filling |
| Applications | Healing gels | Moisturizers |

## III. BIOSYNTHESIS AND ROLE OF SULFATED GAGS IN SKIN

The Golgi apparatus uses a multi-step process to manufacture sulfated glycosaminoglycans (GAGs), including chondroitin sulfate, dermatan sulfate, heparan sulfate, and keratan sulfate. The first step in this process is for core proteins to produce a tetrasaccharide linker, which is necessary for GAG chain elongation. Glycosyltransferases add hexosamine and uronic acid residues to form polymers, which are then sulfated by sulfotransferases using 3′-phosphoadenosine-5′-phosphosulfate (PAPS). The degree and pattern of sulfation that results affects how well sGAGs bind to growth factors, cytokines, and extracellular matrix proteins, hence dictating their biological functions.[9, 10]

Sulfated glycosaminoglycans (sGAGs) are structurally connected to core proteins to produce proteoglycans, which are essential components of the extracellular matrix (ECM). They work with collagen and elastin to give the dermal matrix elasticity and mechanical stability. Their negatively charged polysaccharide chains facilitate the passage of nutrients by creating a moist microenvironment. The ECM's capacity to hold onto water and growth factors is diminished as we age due to changes in composition and a decrease in the overall amount of sGAG, which jeopardizes the structure and healing of the skin. Important biological pathways are also hampered by changes in sulfation patterns and chain lengths, which have an adverse effect on collagen synthesis and wound healing. This ultimately leads to ECM disarray and a loss of skin suppleness.[11, 12]

These results demonstrate how crucial sulfation is to the dermatological functions of glycosaminoglycans (GAGs). Sulfate groups improve charge density, molecular stability, and GAGs' capacity to bind signaling molecules. By providing hydration, antioxidant protection, and biochemical signaling modulation, sulfated hyaluronic acid (sHA) is thought to replicate these

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qualities and restore essential functions of native hyaluronic acid (HA), demonstrating its potential for use in dermatological applications as a next-generation bioactive polymer.[13]

## IV. PROPERTIES OF SULFATED HYALURONAN

Due to their low molecular weight (<0.1 MDa) and moderate sulfation (DS 1-2), sGAGs can perform a variety of physicochemical and biological processes. Hyaluronic acid (HA), the only non-sulfated GAG with a greater molecular weight (0.2–8 MDa), has better lubricating and viscoelastic qualities than heparin, which has a higher sulfation (DS 2–3) and is a powerful anticoagulant. HA and chondroitin sulfate (CS), which were formerly sourced from animal tissues, are now mostly made via microbial fermentation, which increases their purity and repeatability. To improve its stability and performance, HA can also be chemically altered.[14]

As a derivative of hyaluronic acid (HA), sulfated hyaluronic acid (sHA) is created by sulfating its hydroxyl groups, which increases its water solubility and negative charge density. The sodium salt of HA can be used in acidic aqueous circumstances, or the tetrabutylammonium salt of HA can be reacted with a sulfating agent in an acidic environment to create sHA. The sulfating reagent's molar ratio can be changed to regulate the degree of sulfation. Usually, dialysis and lyophilization or ethanol precipitation are used to purify the finished product. The major hydroxyl group of the N-acetylglucosamine residue is preferentially sulfated by sHA, which has an average molecular weight of 15 to 45 kDa and a sulfation degree of 1 to 3 mol. The other hydroxyl groups are sulfated more randomly.[15, 16]

Sulfated hyaluronic acid (sHA) is structurally identical to heparan sulfate but differs in glycosidic connections, acetylation patterns, and sulfation sites. Highly sulfated HAs, such as sHA3, have less anticoagulant action because they lack the particular 3-O-sulfation site, although having other physicochemical characteristics in common with heparin. According to kinetic studies, sHA3 is roughly thirty times weaker than heparin, while sHA1 has no anticoagulant effect. As a biocompatible extracellular matrix component, HA showed minimal cytotoxicity; the IC50 for sHA2 was 7.7 mg/mL. Studies reveal that sulfated HA depolymerizes more slowly than native HA, indicating that chemical sulfation greatly increases HA's resistance to enzymatic degradation. Sulfation degree and enzyme inhibition are directly correlated, according to inhibitory studies, indicating improved therapeutic durability of sHA formulations.[17]

**Table 1: A Comparison between Native HA and Sulfated HA (sHA)**

| Property | Native HA | Sulfated HA |
|---|---|---|
| Charge density | Negative ($COO^-$) | More negative ($COO^- + SO3^-$) |
| Affinity for receptor | Moderate | Depends on sulfation process |
| Stability | Low | High |
| Antioxidant capacity | Limited | Improved |
| Applications | Hydration, barrier support | Barrier repair, anti-inflammatory effects, ECM remodeling |

Beyond its stability, sulfated hyaluronic acid (sHA) offers functional advantages in tissue regeneration and inflammation. It promotes fibroblast adhesion by binding fibronectin and maintaining its structural integrity. The added sulfate groups facilitate interactions with positively charged proteins, aiding in their concentration modulation and protecting against degradation. The presence of β(1→4) and β(1→3) glycosidic linkages enhances sHA's water retention and selective binding to proteins, distinguishing it from heparan sulfate and chondroitin sulfate. This unique structural organization supports sHA's superior hydration and substrate-binding properties, highlighting its potential in dermatology, wound repair, and regenerative medicine.[18, 19]

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## V. USE OF SULFATED HYALURONAN IN DERMATOLOGY

Due to its size, high-molecular-weight hyaluronic acid (HMW-HA) has limited epidermal penetration but aids in the formation of a protective skin barrier. Low-molecular-weight hyaluronic acid (LMW-HA), on the other hand, penetrates more effectively but may trigger inflammatory reactions. LMW-HA's anti-inflammatory properties are strengthened when it is sulfated to produce sulfated hyaluronic acid (sHA), which makes it a useful component for topical dermatological treatments. Because of its interaction with heparin-binding epidermal growth factor (HB-EGF), which promotes keratinocyte and fibroblast activity during repair processes, sHA has been shown to be helpful in wound healing and tissue regeneration. Furthermore, it has been discovered that sHA inhibits the TGF-β1 signaling pathway, which may assist regulate collagen synthesis and fibroblast activation, thus providing antifibrotic advantages.In skin models, sHA exhibits resilience to degradation and encourages advantageous angiogenesis and inflammatory regulation. It is a promising therapeutic option in dermatology for the treatment of chronic skin conditions because of its safety, biocompatibility, and capacity to lower inflammation and improve skin moisture.[20,21]

### i. Psoriasis

About 2% of people worldwide suffer from psoriasis, a chronic inflammatory skin condition that primarily manifests as erythematous plaques with a white scale. Genetic and environmental variables frequently impact the illness, resulting in a Th1-driven immune response involving cytokines, specifically TNF-α, IL-17, and IL-23. Along with additional types like guttate, erythrodermic, and pustular psoriasis, the most prevalent type, psoriasis vulgaris, accounts for 80–90% of cases and usually affects the knees, elbows, and scalp. Topical therapies, phototherapy, and systemic medications, including conventional and biologic alternatives, are all part of management, which is customized according on the kind and severity of psoriasis.[22, 23]

Research showing altered sGAG metabolism in psoriasis patients and the potential to restore skin balance while reducing side effects of systemic therapies like corticosteroids supports the use of sulfated glycosaminoglycans (sGAGs) in topical formulations for psoriasis treatment. Increased sGAG excretion and a link between heparinase overexpression and the development of psoriasis plaque have been demonstrated in studies. Furthermore, sGAGs with antioxidant qualities, like sodium hyaluronate (sHA), are intriguing for topical adjuvant therapy and wound healing because oxidative stress is a major component of psoriasis.[24, 25]

### ii. Atopic Dermatitis

Over 230 million people worldwide suffer from atopic dermatitis (AD), a chronic inflammatory skin illness that affects 11–20% of children and 5–8% of adults. Its symptoms, which include itching, redness, dryness, and recurring eczema, have a major impact on mental health and quality of life. The severity of AD varies, and it frequently coexists with asthma and other allergy disorders. Microbial dysbiosis and filaggrin gene mutations are among the genetic, environmental, and immunological interactions that contribute to its pathogenesis. By causing inflammation and damage to the skin barrier, oxidative stress makes AD worse by increasing itching and lesions. Furthermore, excessive high molecular weight hyaluronic acid in the epidermis caused by HA-synthase overexpression may compromise the function of the skin barrier by altering cell junctions and water retention in afflicted skin.[20, 21]

By reducing symptoms in mice models and promoting wound healing, LMW HA and its derivatives exhibit potential as an adjuvant treatment for atopic dermatitis (AD). Heparinoid polysulfated mucopolysaccharide (MPS) improves tight junction integrity and barrier function while acting as a moisturizing agent for xerosis in AD patients. Conversely, the effects of chondroitin sulfate (CS) and traditional HA on barrier characteristics were negligible. Furthermore, because of its decreased molecular weight, which enhances skin penetration and hydration along with antioxidant qualities, sulfated hyaluronic acid (sHA) is thought to have advantages comparable to those of MPS.[26, 27]

### iii. Acne

Comedones and inflammatory lesions like papules and pustules are the hallmarks of acne vulgaris, an inflammatory condition of the pilosebaceous unit. Increased sebum production, follicular hyperkeratinization, colonization by Cutibacterium acnes and Staphylococcus species, and immune response activation are all associated with its development. Acne primarily affects teenagers, but it can also develop or persist in

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adults, particularly in women who have diseases like polycystic ovarian syndrome. Topical retinoids, antibiotics, and isotretinoin are examples of conventional therapies, however they frequently have adverse effects and increase antibiotic resistance. Because of its moisturizing qualities, capacity to regulate inflammation and sebum production, restore the function of the skin barrier, and encourage skin cell regeneration, hyaluronic acid is becoming a useful complement to acne treatment.[28, 29]

Because C. acnes expresses two hyaluronate lyase (HYL) variants—HYL-IA, which is weakly active and linked to surface strains, and HYL-IB/II, which is highly active and linked to deeper tissue invasion—the interaction between the bacterium and hyaluronic acid (HA) is critical in the context of treating acne. While the breakdown products may facilitate bacterial colonization, these enzymes break down HA in the extracellular matrix, which may increase inflammation and tissue injury. By preventing bacterial adherence, exogenous sulfated or modified HA derivatives may lessen inflammation. Furthermore, by inhibiting FGFR2 and androgen receptor signaling, reducing sebum production, and avoiding compensatory pathways that are often triggered by FGFR inhibitors, HA-based nanoconjugates such as HA-FGF2-derived peptide bioconjugates (HA-P5) demonstrate anti-acne effects.Since oxidative stress has also been connected to the pathophysiology of acne, low-sulfated HA may help retain moisture and restore redox balance while reducing discomfort from conventional therapies.[30, 31]

**iv. Other Conditions**

Beyond psoriasis,atopic dermatitis and acne, research on sulfated hyaluronic acid (sHA) suggests that it may be beneficial for wound healing and persistent skin lesions such pressure ulcers and diabetic wounds. Because of its higher negative charge, which helps bind growth factors, it seems to promote fibroblast proliferation, angiogenesis, and collagen organization while regulating inflammation. Through growth-factor sequestration and hydration, the administration of sHA creates an environment that is favorable for tissue healing. Nevertheless, there is still little proof of clinical efficacy, and more investigation is required, especially to assess sHA's capacity to prevent bacterial infections like impetigo by lowering bacterial adhesion. All things considered, the field appears promising for further research to ascertain its effectiveness in treating persistent wounds and skin diseases.[32, 33]

## VI. CONCLUSION

Glycosaminoglycans (GAGs) are essential for skin health and the extracellular matrix (ECM). Aging and inflammatory skin conditions cause changes in GAG expression, which result in problems including wrinkles and dryness. Although hyaluronic acid (HA) is important for skin healing and hydration, its therapeutic application is constrained by its quick degradation. Sulfated derivatives (sHA), which are produced when HA is chemically sulfated, enhance water retention and resistance to deterioration. According to preliminary data, sHA may improve the restoration of the skin barrier and reduce inflammation. To establish clinical efficacy and standards, more research is necessary. Although sodium sulfated hyaluronate is already utilized in cosmetics, there is little clinical proof of its efficacy.Overall, sulfated HA has potential in dermatology, but more study is required to confirm its therapeutic uses.

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