Research Article

Structure, Function, and Quality Control of Chondroitin Sulfate: Its Potential as a Functional Food Ingredient

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Abstract: Osteoarthritis (OA) is a progressive degenerative joint disease with a prevalence that increases markedly with age. Although current treatments primarily provide symptomatic relief, symptomatic slow-acting drugs for osteoarthritis (SYSADOAs) have attracted attention for their potential to alleviate pain and slow structural deterioration. Among these agents, chondroitin sulfate (CS) is widely used as both a pharmaceutical agent and a dietary supplement. However, its clinical efficacy remains controversial, largely because CS exhibits substantial structural heterogeneity according to animal origin, sulfation pattern, molecular weight, and manufacturing process. These variations complicate the establishment of consistent structure–activity relationships and highlight the need for rigorous analytical and quality control approaches. This review summarizes the chemical diversity of CS, evaluates analytical methods used to determine its identity, purity, molecular weight, and disaccharide composition, and discusses regulatory challenges associated with its use in pharmaceuticals and functional foods. Particular attention is given to salmon nasal cartilage-derived proteoglycans (SPG), which contain densely clustered CS chains and exhibit biological activities distinct from those of CS alone. Emerging evidence suggests that orally administered CS and SPG may act as matrikines, extracellular matrix-derived signaling molecules that modulate intestinal immunity and may contribute indirectly to cartilage repair. By integrating recent advances in glycan analysis with current biological findings, this review provides a framework for understanding the structure–function relationships of CS and SPG and outlines future directions for improving their quality control and application as functional food ingredients.

Keywords: chondroitin sulfate, salmon cartilage-derived proteoglycans, quality control, matrikines, intestinal immunity, tissue regeneration

1. Introduction

Osteoarthritis (OA) is a major public health concern, affecting tens of millions of individuals in Western countries and an estimated 10 million symptomatic patients in Japan, with the total number approaching 30 million when potential cases are included. Its prevalence increases with age and leads to chronic pain, restricted joint mobility, and reduced quality of life. Conventional treatment strategies rely primarily on symptomatic management using nonsteroidal anti-inflammatory drugs (NSAIDs), and few options are available to fundamentally halt disease progression. Against this backdrop, symptomatic slow-acting drugs for osteoarthritis (SYSADOAs), which provide components of the extracellular matrix (ECM), have gained increasing attention. Among these, chondroitin sulfate (CS) has long been used as both a pharmaceutical and a nutraceutical ingredient; however, its clinical efficacy remains controversial, with conflicting positive and negative reports.

Recent advances have highlighted proteoglycans (PGs) derived from salmon nasal cartilage as promising new functional food ingredients. These PGs, composed primarily of high-molecular-weight aggrecans bearing numerous CS chains, exhibit biological activities distinct from those of CS alone. This review provides a comprehensive overview of the structure, function, and quality control of CS and PGs and discusses their potential applications as functional food materials.

2. Fundamentals of CS: Structure, Origin, and Diversity

2.1 Chemical Structure

Osteoarthritis (OA) is a major public health concern, affeChondroitin sulfate is a glycosaminoglycan (GAG) that is widely present in the extracellular matrix and on the cell surface of cartilage tissues in animals, including humans. It is composed of repeating disaccharide units consisting of glucuronic acid (GlcA) and *N*-acetylgalactosamine (GalNAc) (**Figure 1**).[4] CS is a linear acidic polysaccharide that exhibits structural diversity depending on the position of the sulfate group esterified to the hydroxy groups of each constituent monosaccharide. The sulfation pattern, degree of sulfation, and mean molecular weight (Mw) of CS reportedly vary depending on the tissue of origin, animal species, and age.[5] In general, CS extracted from the cartilage of terrestrial animals differs from CS derived from marine animal cartilage. Terrestrial animal-derived CS is rich in disaccharide units sulfated at C-4 of GalNAc (A units), whereas marine animal-derived CS is rich in disaccharide units sulfated at C-6 of GalNAc (C units), often accounting for 50% or more of total disaccharides. Although relatively few studies have investigated human cartilage, available data indicate that A units predominate in childhood and shift toward C units with age.[6]

CS chains derived from fish cartilage, such as those from shark, ray, and salmon, characteristically contain D units, which are disulfated disaccharides bearing sulfate esters at C-6 of GalNAc and C-2 of GlcA. In addition, CS derived from squid cartilage is rich in E units, which contain sulfate esters at C-4 and C-6 of GalNAc.[5,6] The reasons for this structural diversity remain unknown, and glycan sequencing, particularly of sulfated disaccharides, remains insufficient despite its importance for elucidating biological activity. CS also varies in molecular weight, with CS derived from mammals such as swine (Mw, 14.0–26.0 kDa) generally being smaller than that derived from marine animals such as sharks (Mw, 30–70 kDa). Commercially available CS is mainly extracted from porcine and bovine tracheal cartilage, chicken and squid cartilage, and shark fins. However, regulations on raw material labeling are often lacking, and intentional or accidental mixing of CS of different origins during the manufacturing process may complicate the evaluation of biological activity.[4,5]

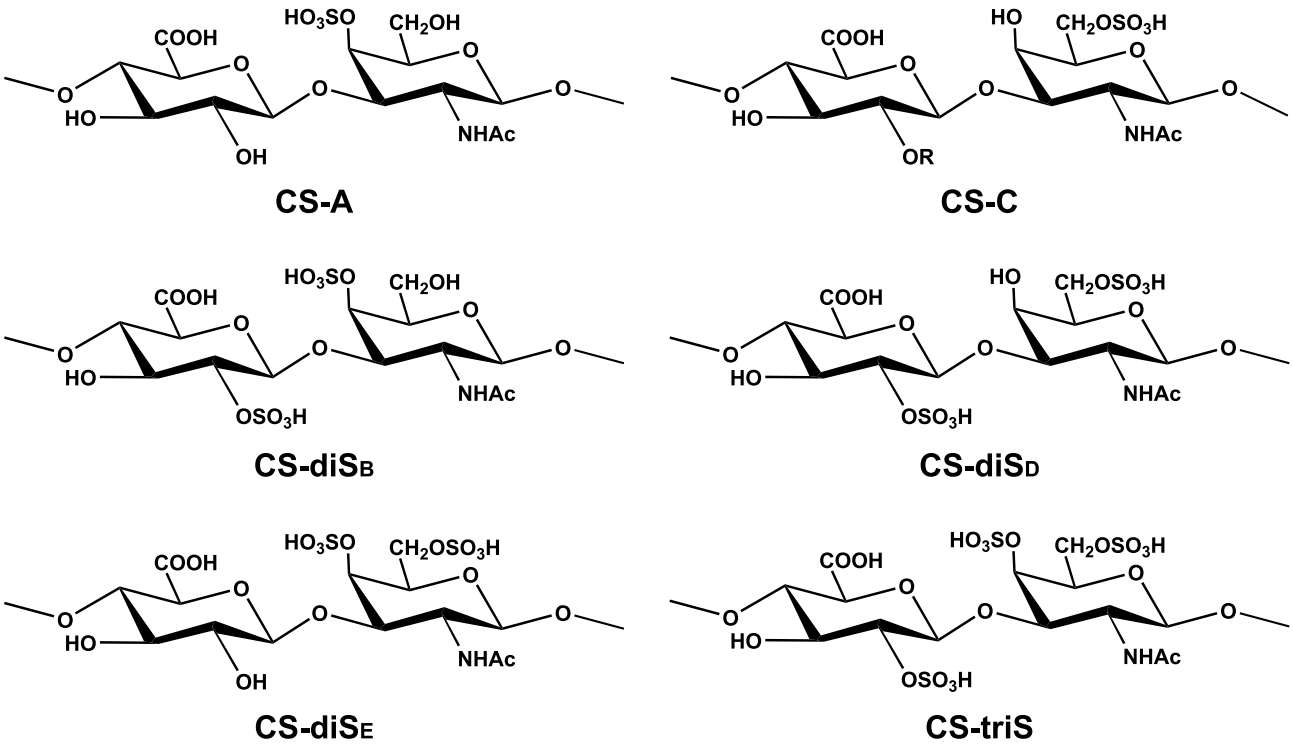

Figure 1. Representative disaccharide units of chondroitin sulfate.

CS exhibits diverse biological activities, including anti-inflammatory, antioxidant, antitumor, anticoagulant, neuroprotective, and immunomodulatory effects.[5] In recent years, however, the substantial structural diversity of CS, despite its long-standing use as a pharmaceutical and nutraceutical ingredient, has emerged as a critical issue for ensuring consistent efficacy, particularly as structure–activity relationships become increasingly well defined.[5] It is now recognized that the biological activity of CS is influenced not only by dosage but also by molecular weight, sulfation degree and pattern, purity, and the presence of other glycosaminoglycans introduced during manufacturing.[4,5] Consequently, clear identification of the origin of CS used in pharmaceuticals, nutraceuticals, and dietary supplements is essential for evaluating its functional effects.

2.2 Structural Diversity According to Origin

Key analytical challenges in characterizing CS include standardizing analytical procedures, accurately assessing CS purity, and elucidating correlations between structural features and biological activity. Each of these issues is inherently complex. First, the structural heterogeneity of CS (**Table 1**) makes it difficult to obtain universally applicable CS reference standards for quantitative analysis.[4,5] As a result, official compendia such as pharmacopoeias specify only certified CS standards for quantitative testing.[5] The European Pharmacopoeia recommends CS standards derived from both terrestrial and marine sources, whereas the United States Pharmacopeia endorses proprietary standards isolated from bovine, porcine, and avian tissues.[7] Notably, recent evaluations have revealed that even these standards may contain keratan sulfate as an impurity.[5]

This challenge is also recognized by the Association of Official Analytical Chemists (AOAC), which requires pharmaceutical manufacturers to use raw materials accompanied by results from multiple analytical methods to confirm CS identity, molecular weight, the ratio of 4-sulfated to 6-sulfated disaccharides, and overall purity.[7] Nevertheless, variations in manufacturing processes, limited reproducibility of extraction and purification protocols, and intrinsic structural diversity mean that even pharmaceutical-grade CS may differ in oral absorption and biological activity.[5] Indeed, clinical trials evaluating CS for osteoarthritis have produced inconsistent and sometimes contradictory outcomes, which have been attributed largely to differences in structural uniformity and purity among CS preparations.[5]

Accordingly, pharmaceutical-grade CS is now subject to increasingly stringent quality control measures and regulatory oversight. In contrast, CS used in health foods is not regulated to the same degree, despite similar concerns regarding variability. To address this gap, the AOAC has recently proposed the adoption of high-performance liquid chromatography (HPLC)-based disaccharide composition analysis, molecular weight determination, and proton nuclear magnetic resonance (^{1}H-NMR) spectroscopy as quality control methods for CS intended for use as a functional food ingredient.[8]

species	tissue	ΔDi0S	ΔDi4S	ΔDi6S	ΔDi2,4diS	ΔDi2,4diS	Δdi-4,6diS
porcine	trachea	3.0%	82.0%	15.0%	0.0%	0.0%	0.0%
bovine	trachea	9.0%	51.0%	40.0%	0.0%	0.0%	0.0%
shark	cartilage	5.2%	29.6%	37.2%	23.1%	0.0%	4.4%
ray	cartilage	3.0%	43.0%	39.0%	13.0%	1.0%	1.0%
chicken	tracheal	12.6%	68.8%	18.5%	0.0%	0.0%	0.0%
whale	cartilage	1.6%	76.2%	19.3%	2.7%	0.0%	0.3%
squid	cartilage	5.9%	22.9%	9.6%	0.0%	0.0%	61.5%
mouse	articular cartilage	–	75-90%	10-25%	–	–	–
human	articular cartilage	2-5%	5-40%	60-85%	–	–	–

Table 1. Diversity of chondroitin sulfate disaccharide composition

3. Quality Control and Techniques for Chondroitin Sulfate Analysis

Quality control requirements for CS differ markedly between pharmaceuticals and nutraceuticals. Whereas pharmaceutical-grade CS is subject to strict regulatory standards, food-grade CS often has inadequate labeling, insufficient purity control, inaccurate content claims, and potential vulnerability to adulteration. The major issues are summarized below.

3.1 Verification of Identity

As noted earlier, European and United States pharmacopoeias recommend multiplexed analytical approaches for CS identity testing, including molecular weight estimation based on infrared absorption spectroscopy, optical rotation, and viscosity measurements.[5] However, accurate qualitative and quantitative analysis of CS in complex mixtures, such as industrial raw materials or health food products, remains challenging. For example, although matching the infrared absorption spectrum of an isolated CS fraction with that of a reference standard can confirm its identity, this method cannot reliably determine the degree of sulfation or the distribution of sulfation positions.

NMR spectroscopy provides a robust qualitative and quantitative tool for CS, provided that the analyst is experienced in spectral interpretation. Nevertheless, NMR offers limited information on molecular weight and requires long acquisition times to detect impurities below 5%. Overall, HPLC analysis of unsaturated disaccharides generated by chondroitinase digestion remains the most reliable method for determining CS origin. The resulting 4-sulfated and 6-sulfated disaccharide profiles serve not only as qualitative markers of CS origin but also as quantitative indicators when high-purity, origin-defined CS standards are used.[4-6]

3.2 Quantitative Methods

Accurate quantification of CS in pharmaceuticals and health foods is essential for establishing correlations between CS dosage, biological activity, and clinical outcomes. The European and United States pharmacopoeias recommend determining CS content using photometric titration with cetylpyridinium chloride (CPC), combined with agarose gel or cellulose acetate electrophoresis.[5] Although simple, these methods lack sufficient analytical reliability for complex samples.

Colorimetric assays, such as the carbazole method for GlcA and the Elson–Morgan method for GalNAc, remain widely used due to their convenience.[5] However, these assays lack specificity, as other polysaccharides containing uronic acid or amino sugars interfere with quantification. Quantitative NMR spectroscopy, recently endorsed by pharmacopoeias, offers improved specificity but is costly, requires expert data processing, and is not yet widely adopted. In contrast, HPLC analysis of CS disaccharides generated by enzymatic degradation has been firmly established as a reliable qualitative and quantitative method.[4,5] This approach is expected to become the standard quantitative technique for CS analysis.

3.3 Impurities and Related Substances

Cellulose acetate membrane electrophoresis and gel-filtration HPLC are convenient methods for detecting mixtures of CS with other GAGs.[5] Additionally, ^{1}H-NMR spectroscopy is effective for identifying organic impurities and quantifying the presence of other GAGs.[4,5]

Pharmaceutical-grade CS typically contains only glycan chains without peptides at the reducing end. In contrast, food-grade CS often retains peptide fragments, as purification processes used for pharmaceutical-grade CS cannot be applied to food materials.

3.4 Molecular Weight Determination

Traditionally, the average molecular weight of CS has been estimated using intrinsic viscosity measurements or electrophoretic mobility on polyacrylamide gels.[4,5] However, limitations of these methods have become increasingly apparent, and more precise and sensitive analyses now combine gel permeation or size-exclusion chromatography with detectors such as differential refractometers and UV detectors. Size-exclusion chromatography is widely recommended by the AOAC for CS molecular weight determination.[8]

A major limitation of size-exclusion and gel-permeation chromatography is their inability to distinguish CS from other GAGs, including hyaluronic acid, dermatan sulfate, and heparan sulfate. Moreover, average molecular weight, including polydispersity, is an important structural characteristic that reflects CS origin, as terrestrial and marine CS exhibit distinct molecular weight ranges. Thus, combining disaccharide composition analysis with gel-filtration HPLC improves the accuracy of origin determination. This is particularly important for functional foods containing CS, for which the mechanism of action may depend on structural attributes. Unfortunately, origin labeling is not mandatory, and mixtures of CS from different sources are frequently encountered.[4,5]

3.5 Analysis of CS Disaccharide Composition

The United States Pharmacopeia recently adopted disaccharide composition analysis as an official method for determining CS origin.[8] Disaccharide profiles vary depending on the enzyme used for digestion, as well as on the animal species and tissue source.[4,5] Microbial chondroitinase AC, which preferentially degrades chondroitin sulfate A and C, cleaves the GalNAcβ1→4GlcA linkage to generate unsaturated disaccharides, which are then separated and quantified by HPLC and widely used for CS origin analysis (**Figure 2**).[4] Chondroitinase ABC degrades CS almost completely into unsaturated disaccharides and can also act on hyaluronan and dermatan sulfate, necessitating confirmatory analysis by electrophoresis and HPLC.

Because CS from different origins exhibits distinct disaccharide compositions (**Table 1**), the origin of CS can be determined by comparison with well-characterized CS standards. Pharmaceutical products generally use a single, well-defined CS source, and mixing of CS from different origins has not been reported. In contrast, food products frequently contain mixtures of CS from multiple origins.[4,5]

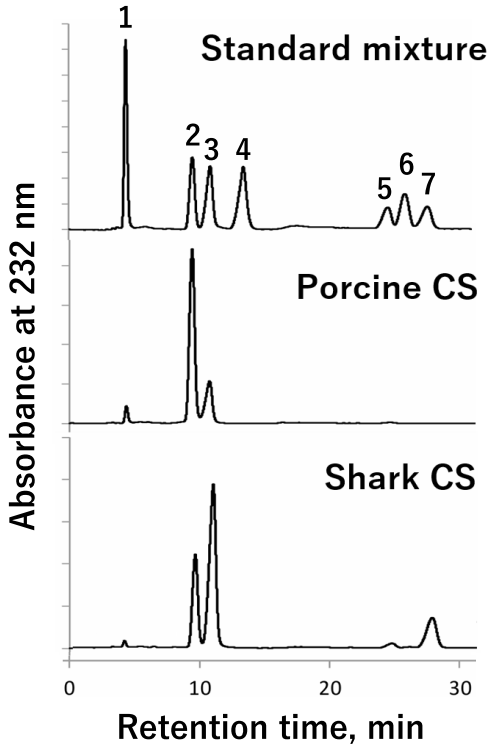

Figure 2. HPLC analysis of CS-derived unsaturated disaccharides

Figure legend: Chromatograms of a standard mixture of seven unsaturated disaccharides generated by chondroitinase digestion (top), porcine cartilage–derived CS (middle), and shark cartilage–derived CS (bottom). Peaks correspond to ΔDi-0S (1), ΔDi-4S (2), ΔDi-6S (3), ΔDi-2S (4), ΔDi-S_E (5), ΔDi-S_B (6), and ΔDi-S_D (7). Differences in peak distribution reflect origin-specific disaccharide compositions that allow CS origin to be determined and sample purity to be assessed.

3.6 Other Analytical Methods

NMR spectroscopy using both ^{1}H and ^{13}C nuclei has long been used to characterize the structural features and origin of CS.[4] In recent years, NMR has also been applied to confirmatory and quantitative testing of certain pharmaceutical preparations.[5] As NMR instrumentation continues to advance in scale, automation, and analytical precision, it is anticipated that NMR alone may eventually serve as a comprehensive platform for CS quality control.

Mass spectrometry (MS), another major analytical platform that has advanced substantially, remains under development as a routine quality control method for CS. Although MS has been successfully applied in specific cases, such as the structural analysis of bikunin, a chondroitin sulfate proteoglycan (CSPG), its broader application to CS quality control will require further methodological refinement.[10]

4. Biological Activity of CS: Structure Dependence and Clinical Debat

4.1 Basic Research

CS exhibits a wide range of biological activities, including anti-inflammatory, antioxidant, antithrombotic, anticoagulant, and immunomodulatory effects.[4,5] Consequently, extensive basic research continues to explore its potential roles in mitigating cardiovascular, neurological, oncological, and joint-related disorders. The market for CS is expanding both as a dietary supplement and as an over-the-counter therapeutic for OA, underscoring the urgent need to clarify its mechanisms of action.[4,5]

Numerous animal studies have demonstrated that orally administered CS exerts anti-inflammatory effects by reducing levels of interleukin-1β (IL-1β), nuclear factor-κB (NF-κB), and matrix-degrading enzymes.[5] CS has also been reported to possess antitumor activity.[11] Collectively, these findings indicate that the biological activity of CS is strongly dependent on its structural characteristics. Further elucidation of CS structure–activity relationships is therefore needed.

4.2 Inconsistencies in Clinical Research on OA

As noted in the introduction, OA is a globally prevalent disease. CS has been used for approximately 40 years as one of the SYSADOAs. Although various mechanisms have been proposed, direct absorption of intact CS in the gastrointestinal tract is considered unlikely. Current evidence suggests that its anti-inflammatory effects may be mediated through modulation of the intestinal immune system and gut microbiota.[4,5]

As discussed in section 5.4, one hypothesis is that orally ingested CS acts as a matrikine, a degradation-derived signal associated with ECM damage, thereby influencing intestinal immune responses and contributing indirectly to cartilage repair.[6] Nevertheless, clinical outcomes with commercially available CS products remain inconsistent, largely because of variations in structural uniformity and purity.[4,5] In particular, the Glucosamine/chondroitin Arthritis Intervention Trial (GAIT) conducted by the U.S. National Institutes of Health failed to reach a conclusion because "the quality and origins of CS samples were unknown."[12] Subsequent clinical studies have produced both positive and negative results, with the primary source of inconsistency attributed to differences in CS quality, including purity and structural uniformity, such as molecular weight and disaccharide composition.[4,5] Although pharmaceutical-grade CS cannot legally be used in nutraceuticals, it is increasingly evident that CS intended for functional food applications also requires rigorous quality control.

5. Salmon Nasal Cartilage–Derived Proteoglycans (SPG)

Since Kakizaki and colleagues first characterized SPG in detail,[13] this material has gained attention as a functional food ingredient. Both basic and clinical studies have demonstrated that SPG exerts beneficial effects not only on OA but also on gut microbiota, inflammation, and other biological processes. Based on these findings, SPG has increasingly attracted global interest. The following sections summarize its characteristics and compare its effects with those of CS.

5.1 Structure of SPG

SPG is a high-molecular-weight proteoglycan (>1000 kDa) composed primarily of aggrecan and is estimated to contain approximately 40–50 CS chains (Mw 20–30 kDa).[13] Notably, the disaccharide composition of CS within SPG closely resembles that of CS in aggrecan derived from adult human cartilage.[6]

Given that SPG contains densely packed CS chains, its affinity for CS-binding receptors such as L-selectin is greater than that of single CS chains, potentially inducing stronger biological responses.[14] Furthermore, when administered orally as a complex with type II collagen, SPG appears to exert stronger effects on the intestinal immune system than when administered alone.[15]

5.2 Quality Control of SPG

Quality control of SPG typically involves determining its molecular weight by gel-filtration HPLC and analyzing the disaccharide composition of its CS chains using HPLC of unsaturated disaccharides generated by enzymatic digestion. Because SPG is not approved as a pharmaceutical, no certified reference standard is available. Consequently, research-grade SPG reagents are used as *de facto* standards for quality assessment.[16]

When the authors analyzed three commercially available SPG reagents—evaluating molecular weight, CS disaccharide composition, and ^{1}H-NMR spectra—results were broadly similar, although minor differences were observed, likely reflecting variations in purification methods or raw materials. As with other natural polymers, careful selection of appropriate reference materials is essential for reliable SPG quality control.

5.3 Biological Activity and Mechanism of Action of SPG

The biological activities of SPG often parallel those of CS. For example, oral administration of SPG to mice and rats reduces circulating markers of type II collagen degradation and enhances markers of collagen synthesis, thereby supporting balanced cartilage metabolism.[17] In a cumulative ultraviolet B irradiation model using hairless mice, SPG administration reduced erythema, improved transepidermal water loss, maintained stratum corneum hydration, suppressed dermal thickening, and contributed to the preservation of skin barrier function and collagen content.[3]

SPG has also been shown to promote chondrocyte proliferation and improve motor function in animal studies. Given its large molecular size, direct absorption of intact SPG is highly unlikely, suggesting that its effects occur indirectly through modulation of the intestinal immune system and gut microbiota.

5.4 CS and SPG as Matrikines

Although various hypotheses have been proposed regarding CS absorption, uptake of intact CS with molecular weights in the tens of thousands is now widely regarded as unlikely. Since the early 2000s, when the authors demonstrated the involvement of CS in intestinal immune responses at the molecular level, this view has gained broad acceptance.[4,5] The mechanism of action of SPG, a macromolecule, should likewise be considered in the context of intestinal immune modulation. Receptors for CS, including Toll-like receptiors (TLRs), CD44, and L-selectin, are expressed not only on immune cells but also on epithelial cells and fibroblasts, indicating that the immune system is central to the biological activities of both CS and SPG.

Matrikines were originally defined as bioactive peptides derived from partial degradation of ECM proteins, but the term has been expanded to include degradation products of ECM glycosaminoglycans, such as hyaluronan, CS, and PG.[18] These molecules act as indicators of ECM damage and regulate cytokine production in immune and epithelial cells, thereby contributing to tissue repair.

It is therefore reasonable to infer that orally administered CS and PG, or their degradation products, may act as matrikines within the intestinal immune system and participate in the repair of damaged cartilage. **Figure 3** summarizes the hypothetical mechanism by which orally administered CS and SPG act as matrikines to modulate intestinal immunity and contribute to cartilage repair. In this model, CS and SPG are considered ECM-derived matrikines that signal tissue damage and activate immune-mediated repair pathways.

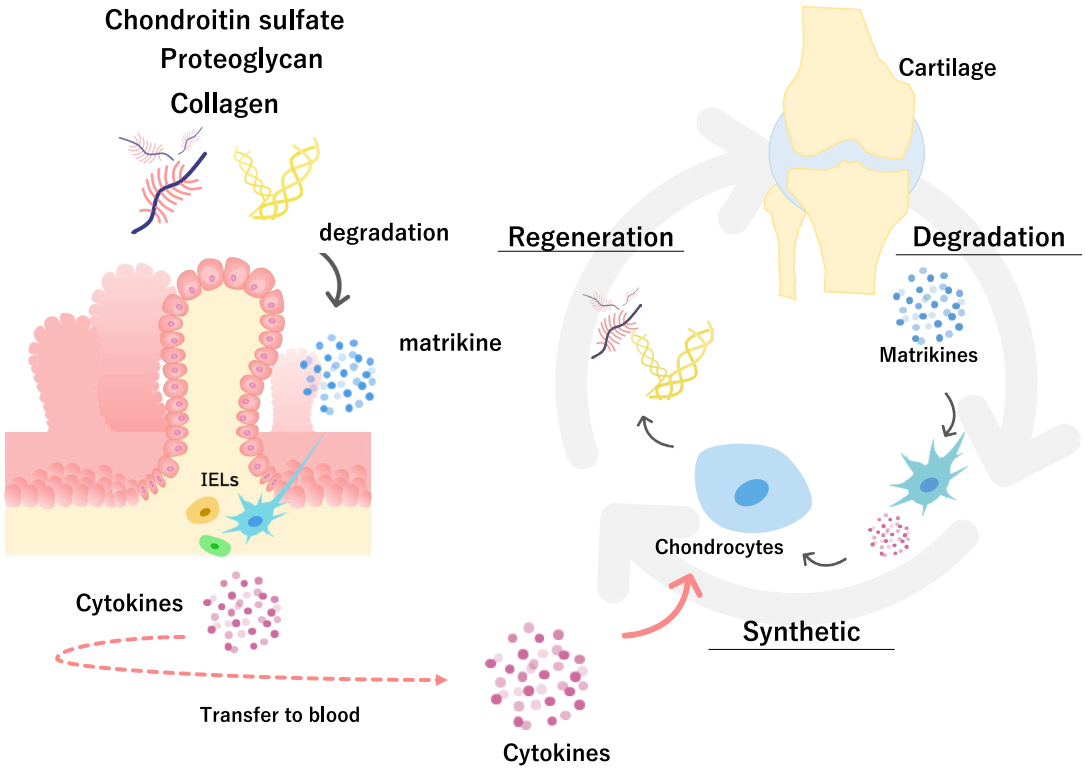

Figure 3. CS and SPG as matrikines: a hypothetical model

Figure legend: Conceptual model showing how CS, SPG, or their degradation products may act as matrikines by interacting with receptors expressed on intestinal epithelial and immune cells, including Toll-like receptors (TLRs), CD44, and L-selectin. These interactions may modulate cytokine production, activate regulatory immune pathways, and contribute indirectly to cartilage repair. Several steps in this model remain hypothetical and require further experimental validation. Modified from Figure 8 in Reference 6.

6. Future Challenges and Prospects

Addressing the challenges associated with reliable quality control of CS and SPG, elucidating the relationships between their structural properties and biological activities, and ultimately enhancing their potential for pharmaceutical and medical applications will require a multifaceted and integrated analytical approach. This approach should include the development of highly accurate analytical techniques and the strategic combination of complementary methodologies.

Securing appropriate reference standards for natural polymers such as CS and SPG remains a significant obstacle. For CS, the situation is relatively favorable because pharmaceutical-grade CS is already commercially available, allowing access to reference materials with guaranteed purity. In contrast, no publicly certified standard exists for SPG. As a result, quality control efforts rely on research-grade SPG products, whose physical and chemical properties can only be verified after purchase. Even among commercially available SPG reagents, substantial variability exists in molecular weight and CS disaccharide composition, limiting their use as reliable standards.

Despite these limitations, previous studies suggest that the structural characteristics and biological activities of CS and SPG are closely related. This relationship underscores the importance of rigorous quality control, particularly for functional foods, which are subject to less stringent regulatory requirements than pharmaceuticals. Ensuring the safety and functional integrity of such products will become increasingly important as their use expands.

Advances in glycan analytical technologies, an area that has progressed remarkably in the 21st century, are expected to play a central role in overcoming these challenges. Continued development and application of these technologies will substantially improve product quality and, ultimately, promote human health.

References

1. J. L. Bowden, D. J. Hunter, L. A. Deveza, V. Duong, K. S. Dziedzic, K.D. Allen, P.-K. Chan, J. P. Eyles, *Nat. Rev. Rheumatol*. **2020**, *16*, 434.
2. K. E. R. Baden, S. L. Hoeksema, N. Gibson, D. N. Gadi, E. Craig, J. A. Draime, S. M. Tubb, A. M. H. Chen, *Nutrients* **2025**, *17*, 2093.
3. X. D. Bai, Y. C. Liu, S. Y. Ge, W. C. Fei, *J. Cosmet. Dermatol*. **2025**, *24*, e70218.
4. S. Sakai, E. Otake, T. Toida, Y. Goda, *Chem. Pharm. Bull*. **2007**, *55*, 299.
5. N. Volpi, *Molecules* **2019**, *24*, 1447.
6. T. Toida, *NATPRO J*. **2025**, *2*, 13.
7. D. T. Burns, M. J. Walker, C. Mussell, *J. AOAC Int*. **2018**, *101*, 196.
8. S. L. Brunelle, *J. AOAC Int*. **2016**, *99*, 53.
9. K. Adels, G. Elbers, B. Diehl, Y. Monakhova, *Anal. Sci*. **2024**, *40*, 101.
10. T. N. Laremore, F. E. Leach 3rd, I. J. Amster, R. J. Linhardt, *Int. J. Mass Spectrom*. **2011**, *305*, 109.
11. R. Lin *et al*., *Mol. Cell* **2018**, *69*, 923.
12. D. O. Clegg *et al*., *N. Engl. J. Med*. **2006**, *354*, 795.
13. I. Kakizaki, Y. Tatara, M. Majima, Y. Kato, M. Endo, *Arch. Biochem. Biophys*. **2011**, *506*, 58.
14. Y. Okamoto, K. Higashi, T. Toida, *Jpn. J. Food Chem. Safety* **2021**, *28*, 9.
15. I. Jeong, J. Park, S. Park, T. Wada, D. S. Lim, O.-K. Kim, *Mar. Drugs* **2024**, *22*, 507.
16. D. Hashimoto, Y. Okamoto, S. Onishi, K. Higashi, T. Wada, T. Toida, *Jpn. J. Food Chem. Safety* **2022**, *29*, 104.
17. T. Wada, Y. Kobuna, T. Toida, *Jpn. J. Food Chem. Safety* **2023**, *30*, 165.
18. A. E. Rapp, F. Zaucke, *Am. J. Physiol. Cell Physiol*. **2023**, *324*, C377.

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Related Product

Proteoglycan from Salmon nasal cartilage	10mg	P3423