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Fucoidan as a Marine-Derived Therapeutic: From Molecular Mechanisms to Clinical Applications

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ABSTRACT

Fucoidan is a heterogeneous group of sulfated, fucose-rich polysaccharides predominantly isolated from brown seaweeds, including Fucus vesiculosus, Undaria pinnatifida, Cladosiphon okamuranus, Sargassum spp., and Laminaria spp. During the last several decades, fucoidan has attracted considerable interest in biomedical research because of its broad range of biological activities, including anti-inflammatory, immunomodulatory, anticoagulant, antithrombotic, antioxidant, antiviral, cardioprotective, metabolic, and anticancer effects. Importantly, fucoidan should not be regarded as a single chemically defined active pharmaceutical ingredient. Rather, the term describes a structurally diverse family of sulfated polysaccharides whose biological properties vary according to algal source, extraction and purification procedures, molecular weight, monosaccharide composition, degree and pattern of sulfation, and glycosidic linkage. This structural heterogeneity represents one of the principal challenges in translating experimental findings into clinical applications. Preclinical investigations have demonstrated that fucoidan can modulate several signaling pathways involved in inflammation, tumor growth, apoptosis, angiogenesis, metastasis, oxidative stress, immune responses, and coagulation. In oncology, experimental evidence is particularly extensive, with animal and cell-based studies suggesting inhibitory effects on tumor proliferation, angiogenesis, invasion, and metastatic dissemination. However, clinical evidence remains considerably more limited. A double-blind randomized controlled trial in 54 patients with metastatic colorectal cancer found a significantly higher disease-control rate when low-molecular-weight fucoidan was administered as a supplement to chemotherapy and targeted therapy, but no significant improvement in overall survival, progression-free survival, objective response rate, quality of life, or adverse-event outcomes was demonstrated. A systematic review of fucoidan as supplemental therapy in cancer patients identified only four eligible clinical studies involving 118 patients and concluded that the clinical evidence remained inconsistent and was limited by small sample sizes and methodological heterogeneity. Human studies investigating immunomodulatory effects, osteoarthritis, metabolic disease, and liver dysfunction have similarly produced preliminary but insufficient evidence for routine therapeutic use. Fucoidan appears to be generally well tolerated in short-term human studies, although potential anticoagulant effects warrant caution in patients receiving anticoagulant or antiplatelet medications. Contemporary research increasingly focuses not only on fucoidan as a direct therapeutic agent but also on its incorporation into nanoparticles, hydrogels, films, and other drug-delivery platforms. Thus, as of 2026, fucoidan should be considered a promising marine-derived bioactive polymer and investigational therapeutic platform rather than an established treatment for cancer or other major diseases. Future clinical development will require rigorous structural characterization, pharmaceutical standardization, pharmacokinetic studies, well-designed randomized controlled trials, and clear differentiation between specific fucoidan preparations.

KEYWORDS: *Fucoidan, phaeophyta, cancer, inflammation, pharmacology, clinical trials.*

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1. INTRODUCTION

Marine organisms represent an increasingly important source of biologically active compounds with potential pharmaceutical applications. Among marine-derived biomolecules, sulfated polysaccharides isolated from brown seaweeds have received particular attention because of their

structural diversity, biological compatibility, and ability to interact with multiple cellular and molecular targets. Fucoidan is one of the most extensively investigated members of this group and has been studied in relation to cancer, inflammation, immunity, coagulation, cardiovascular disease, metabolic disorders, viral infections, tissue repair, and drug delivery [1–6]. Contemporary reviews continue to identify fucoidan as a multifunctional marine polysaccharide with potential applications ranging from nutritional supplements to advanced biomedical materials.

A fundamental issue in the interpretation of the fucoidan literature is that fucoidan does not represent a single chemically homogeneous compound. Instead, it is a family of structurally heterogeneous sulfated polysaccharides whose composition depends strongly on the algal species, geographical and seasonal origin, extraction method, purification procedures, molecular weight, monosaccharide composition, sulfation degree, and distribution of sulfate groups. Fucoidan preparations may contain predominantly L-fucose together with varying amounts of galactose, xylose, mannose, glucose, uronic acids, and other monosaccharides [2–4,7]. Consequently, biological findings obtained using one preparation cannot automatically be extrapolated to all commercially available or experimentally prepared fucoidans. This distinction is particularly important when discussing fucoidan as a potential "active ingredient," because pharmaceutical development requires reproducible chemical identity and batch-to-batch consistency.

The pharmacological interest in fucoidan derives from its ability to influence several biological pathways simultaneously. Experimental studies have reported modulation of NF-κB, MAPK, PI3K/AKT, apoptosis-related pathways, angiogenic signaling, inflammatory cytokines, leukocyte adhesion, coagulation factors, and immune-cell activity [2–6]. In cancer research, fucoidan has attracted particular attention because of its reported effects on proliferation, apoptosis, angiogenesis, epithelial–mesenchymal transition, invasion, metastasis, and tumor-associated immune responses. Nevertheless, the distinction between preclinical efficacy and demonstrated clinical benefit remains essential. The purpose of this review is therefore to evaluate the medical potential of fucoidan by integrating mechanistic, preclinical, pharmacokinetic, safety, and clinical evidence, with particular emphasis on the extent to which current evidence supports therapeutic use in humans.

2. Sources, Chemical Structure, and Structure–Activity Relationships

Fucoidan is primarily obtained from brown seaweeds belonging to the Phaeophyceae class. Frequently investigated sources include *Fucus vesiculosus, Undaria pinnatifida, Cladosiphon okamuranus, Sargassum* species, and *Laminaria* species [2–4]. Their backbone is generally enriched in fucose residues and sulfate ester groups, although the precise structural organization varies considerably. Some preparations contain substantial quantities of uronic acids and neutral sugars, and the glycosidic linkage patterns may differ between species and extraction procedures. These differences are not merely chemical curiosities; they can substantially modify biological activity, solubility, molecular interactions, pharmacokinetics, and toxicity.

Molecular weight is another major determinant of fucoidan behavior. Native high-molecular-weight fucoidans may display high viscosity and limited gastrointestinal absorption, whereas low-molecular-weight fractions generated through controlled hydrolysis or enzymatic processing may possess different solubility, biological distribution, and cellular interactions. However, "low molecular weight" does not inherently mean "more effective." Rather, the optimal molecular size appears to depend on the intended biological activity. Recent structure–activity research emphasizes that biological effects cannot be predicted solely from total sulfate content; molecular size, sulfation position, monosaccharide composition, linkage pattern, and three-dimensional conformation must be considered together. A 2025 study involving a library of 58 synthetic fucoidans demonstrated particularly clearly that anticoagulant activity varied substantially according to molecular architecture and sulfation pattern [8]. Such findings reinforce the need for chemically characterized and standardized preparations in future clinical trials.

From a pharmaceutical-development perspective, these structural characteristics create both opportunities and challenges. Structural heterogeneity may explain why different studies report apparently contradictory results, while controlled chemical modification may allow researchers to optimize fucoidan derivatives for specific biological targets. Therefore, future clinical publications should report the algal species, extraction process, molecular-weight distribution, sulfate content, monosaccharide profile, purity, and relevant structural characteristics rather than referring simply to fucoidan.

3. Pharmacological Activities and Mechanisms

3.1. Anti-inflammatory and Immunomodulatory Effects

The anti-inflammatory activity of fucoidan is among its most consistently investigated biological properties. In cellular and animal models, fucoidan has been reported to inhibit activation of NF-κB and MAPK signaling pathways and to modify the production of pro-inflammatory cytokines such as tumor necrosis factor-α, interleukin-1β, and interleukin-6 [2,5]. These effects have generated interest in inflammatory disorders, metabolic disease, cancer-associated inflammation, and tissue injury. Human evidence, however, remains considerably less robust than experimental evidence. In an exploratory study involving 20 patients with advanced metastatic cancer, oral fucoidan administration was associated with reductions in IL-1β, IL-6, and TNF-α, particularly during the first two weeks of treatment [10]. Because the study was small, open-label, and uncontrolled, these observations should be interpreted as pharmacodynamic signals rather than evidence of clinical efficacy.

Fucoidan also appears capable of modulating immune-cell function. A randomized, double-blind, placebo-controlled pilot study involving 40 healthy adults evaluated 3 g/day of Cladosiphon okamuranus-derived fucoidan for 12 weeks. No clinically significant adverse events were observed, while NK-cell activity increased significantly at week 8 compared with baseline; a significant difference was also observed in male participants compared with placebo [12].

These findings indicate that fucoidan can influence measurable immune parameters in humans, but they do not demonstrate that supplementation prevents infection, treats cancer, or produces clinically meaningful immunological benefits. The term "immune boosting" should therefore be avoided as an oversimplification; fucoidan is more appropriately described as an immunomodulatory compound whose effects may vary according to molecular structure, dose, host condition, and immune-cell population.

3.2. Anticancer Activity

The anticancer properties of fucoidan have been investigated extensively in vitro and in vivo. Proposed mechanisms include inhibition of cancer-cell proliferation, induction of apoptosis, cell-cycle arrest, modulation of PI3K/AKT and MAPK signaling, suppression of HIF-1α/VEGF-mediated angiogenesis, inhibition of epithelial–mesenchymal transition, reduction of tumor-cell migration and invasion, and modulation of the tumor microenvironment [13–17]. Experimental studies have reported activity against breast, colorectal, gastric, hepatocellular, melanoma, hematological, and other malignancies. A systematic review and meta-analysis of 23 animal studies found that fucoidan significantly reduced tumor weight, tumor volume, and tumor number across several experimental models, although the magnitude of the effect varied according to cancer type, dose, and route of administration [18].

These findings provide a strong biological rationale for clinical investigation but cannot by themselves establish therapeutic efficacy in humans. The most important translational limitation is that concentrations capable of producing cytotoxic effects in cultured tumor cells may not be achievable in human tissues following oral administration. Furthermore, tumor biology, drug metabolism, immune responses, and pharmacokinetics are substantially more complex in humans than in experimental systems. Contemporary reviews published in 2025 and 2026 continue to describe fucoidan as a promising candidate for oncology while emphasizing unresolved problems involving standardization, bioavailability, and clinical validation.

3.3. Anticoagulant and Antithrombotic Effects

The negatively charged sulfate groups of fucoidan confer heparin-like biochemical properties and have stimulated extensive investigation of its anticoagulant and antithrombotic effects. Depending on structural characteristics, fucoidan can influence thrombin, factor Xa, antithrombin-dependent pathways, platelet interactions, and P-selectin-mediated cellular adhesion. Human data remain limited. A pilot clinical study involving 10 volunteers who received 3 g/day of fucoidan for 12 days demonstrated an increase in activated partial thromboplastin time, although the authors considered the systemic anticoagulant effect after oral administration to be limited [27]. More recently, systematic synthesis of structurally defined fucoidans demonstrated substantial variation in anticoagulant potency depending on molecular size and sulfation pattern [8]. These observations have two implications: fucoidan derivatives may represent a useful platform for development of novel anticoagulant agents, while certain preparations may theoretically increase bleeding risk when combined with anticoagulant or antiplatelet medications. Consequently, clinical evaluation of fucoidan should include coagulation parameters and systematic assessment of concomitant medications.

3.4. Antioxidant, Antiviral, Cardiovascular, Renal, and Tissue-Protective Effects

Fucoidan has also demonstrated antioxidant and cytoprotective properties in experimental systems, including modulation of oxidative-stress pathways and enhancement of endogenous antioxidant defenses. Its antiviral activity has attracted considerable attention because sulfated polysaccharides may interfere with viral attachment and entry into host cells. Studies involving several viruses, including SARS-CoV-2-related experimental models, have reported inhibitory activity in vitro. However, these findings do not establish efficacy as an antiviral treatment in humans, and fucoidan is not currently a standard treatment for COVID-19, influenza, herpesvirus infection, or other viral diseases. Similar considerations apply to cardiovascular and renal applications: experimental studies suggest effects on inflammation, thrombosis, oxidative stress, fibrosis, and vascular biology, but clinical evidence remains insufficient for routine therapeutic use. Fucoidan has additionally been investigated for wound healing and tissue regeneration. Experimental studies indicate that it may influence inflammation, cell migration, proliferation, angiogenesis, and extracellular-matrix remodeling. This has encouraged development of fucoidan-containing hydrogels, films, nanofibers, and other biomaterials. Such approaches may ultimately prove more clinically feasible than systemic administration because they permit localized delivery and may overcome some limitations associated with oral bioavailability.

4. Clinical Evidence and Medical Applications

4.1. Cancer and Supportive Oncology

The most clinically developed application of fucoidan is cancer supportive therapy. The strongest randomized evidence currently available comes from a prospective double-blind controlled study of 54 patients with metastatic colorectal cancer receiving chemotherapy and targeted therapy. Patients were randomized to receive low-molecular-weight fucoidan or placebo in addition to standard treatment. The disease-control rate was 92.8% in the fucoidan group compared with 69.2% in the control group, representing a statistically significant difference (p=0.026) [19].

However, importantly, the study did not demonstrate significant advantages in overall survival, progression-free survival, objective response rate, quality of life, or adverse-event outcomes. Therefore, the findings should be interpreted as preliminary evidence of a possible adjunctive effect rather than proof that fucoidan has independent anticancer efficacy. A systematic review specifically evaluating fucoidan as supplemental therapy in cancer patients identified only four eligible clinical studies, including one randomized controlled trial and three quasi-experimental studies, with a total sample of 118 participants. The review concluded that clinical outcomes were inconsistent and that small sample sizes, methodological heterogeneity, and differences in outcome measurement prevented firm conclusions [25].

This conclusion remains important because the extensive preclinical literature can otherwise create an impression that clinical efficacy has already been established. As of 2026, there is no convincing evidence that fucoidan can replace chemotherapy, targeted therapy, immunotherapy, radiotherapy, surgery, or other established cancer treatments. An additional clinical question concerns interaction with anticancer drugs. A small study involving 20 patients with breast cancer receiving tamoxifen or letrozole found no clinically significant changes in the pharmacokinetics of these drugs after short-term supplementation with Undaria pinnatifida-derived fucoidan [20]. Although reassuring, these findings cannot be generalized to all anticancer agents or all fucoidan preparations. The clinical role of fucoidan in oncology should therefore presently be framed as investigational supportive therapy rather than established anticancer treatment.

4.2. Metabolic Disease, Liver Disease, and Osteoarthritis

Human studies in metabolic disorders provide preliminary but insufficient evidence. In patients with type 2 diabetes, a randomized placebo-controlled crossover study investigated a high-molecular-weight fucoidan preparation at approximately 1.62 g/day for 12 weeks. No major adverse events were observed, and exploratory changes in stool frequency, taste sensitivity, HbA1c, and GLP-1 were reported in selected subgroups [14]. These findings are hypothesis-generating rather than definitive evidence of antidiabetic efficacy.

Fucoidan has also been investigated in nonalcoholic fatty liver disease. A double-blind randomized trial involving 42 patients evaluated a combination of low-molecular-weight fucoidan and high-stability fucoxanthin. The intervention was associated with a significant reduction in serum alanine aminotransferase, although objective measures of hepatic steatosis did not demonstrate a corresponding significant improvement [30]. Because fucoidan and fucoxanthin were administered together, the independent contribution of fucoidan cannot be

determined. Accordingly, fucoidan cannot currently be considered an established treatment for NAFLD.

Osteoarthritis provides another example of the distinction between biological plausibility and clinical efficacy. A randomized placebo-controlled study of 122 patients with mild-to-moderate hip or knee osteoarthritis evaluated 300 mg/day of an Fucus vesiculosus-derived preparation for 12 weeks. Although the preparation was generally well tolerated, the study did not demonstrate a significant improvement in osteoarthritis symptoms compared with placebo [31]. Therefore, current clinical evidence does not support routine use of fucoidan as an analgesic or disease-modifying osteoarthritis treatment.

5. Pharmacokinetics, Bioavailability, Safety, and Drug Interactions

One of the major obstacles to therapeutic development is the uncertain pharmacokinetic behavior of orally administered fucoidan. Because native fucoidans are large polysaccharides, extensive systemic absorption might not be expected. Nevertheless, human studies have detected fucoidan-related material in blood or urine following oral ingestion, indicating that at least some components may cross the gastrointestinal barrier or undergo partial degradation and absorption. In a study of 396 Japanese volunteers, fucoidan-derived material was detected in urine after ingestion of 3 g of fucoidan in most participants. These findings suggest that oral fucoidan is not completely biologically inert, but they do not establish that intact high-molecular-weight fucoidan reaches systemic tissues at pharmacologically relevant concentrations [40].

An alternative explanation for some biological effects is action within the gastrointestinal tract. Fucoidan may interact with intestinal epithelial cells, digestive processes, or the gut microbiota, with subsequent indirect systemic effects. This possibility is particularly relevant to immunological and metabolic outcomes and suggests that future pharmacokinetic research should incorporate microbiome, metabolomic, and degradation-product analyses in addition to conventional plasma concentration measurements. Short-term human studies generally suggest good tolerability. In a randomized study of Cladosiphon okamuranus-derived fucoidan administered at 3 g/day for 12 weeks, no clinically significant adverse events attributable to the intervention were identified [12].

Earlier human safety studies similarly reported acceptable tolerability at gram-level doses over short periods [41]. Gastrointestinal effects, including changes in bowel movements or diarrhea at high intake, may occur in some individuals. However, long-term safety data, particularly in elderly populations, patients with multiple comorbidities, and individuals receiving multiple medications, remain limited. Drug interactions represent a clinically relevant area requiring further investigation. The most important potential pharmacodynamic interaction is with anticoagulant and antiplatelet drugs because of fucoidan's sulfated structure and potential effects on coagulation. Patients receiving warfarin, direct oral anticoagulants, heparins, aspirin, clopidogrel, or combinations of antithrombotic drugs should therefore not assume that fucoidan is pharmacologically inert. Similarly, patients undergoing major surgery may require discontinuation or medical supervision because of the theoretical risk of altered hemostasis. These considerations do not necessarily establish absolute contraindications, but they justify caution until stronger clinical pharmacovigilance data become available.

6. Standardization and Pharmaceutical Development

The greatest challenge in translating fucoidan research into clinical medicine is arguably standardization rather than lack of biological activity. A clinically meaningful fucoidan product should be characterized according to its algal source, extraction procedure, purification level, molecular-weight distribution, total sulfate content, monosaccharide composition, degree and pattern of sulfation, protein and polyphenol contamination, heavy-metal content, and microbiological quality. Without these parameters, comparison between studies is difficult and reproducibility is compromised. The structural heterogeneity of fucoidan also complicates dose interpretation. Reporting that a patient received “1 g of fucoidan” does not provide sufficient pharmacological information if the molecular weight, sulfate content, and composition are unknown. Two products administered at the same mass dose may possess substantially different biological activities. The emergence of synthetic and semi-synthetic fucoidan derivatives may therefore represent an important transition from traditional nutritional preparations toward defined pharmaceutical candidates. Recent research demonstrating structure-dependent anticoagulant activity provides a clear example of how rational molecular design could potentially produce more predictable pharmacological effects. This principle should also guide clinical trial design. Future randomized studies should ideally use chemically characterized preparations from a single manufacturing process, predefine molecular and pharmacological specifications, investigate dose-

response relationships, and include clinically meaningful endpoints. For oncology studies, overall survival, progression-free survival, objective response, treatment completion, patient-reported outcomes, and clinically relevant toxicity should be prioritized over isolated laboratory biomarkers [8].

7. Fucoidan as a Drug-Delivery and Biomedical Platform

An increasingly important direction in fucoidan research is its use as a biomaterial rather than simply as an orally administered supplement. The sulfate groups and polysaccharide structure of fucoidan permit interactions with proteins, other polymers, nanoparticles, and cell-surface molecules. Consequently, fucoidan has been incorporated into nanoparticles, liposomes, hydrogels, films, nanofibers, and controlled-release systems for biomedical applications [37,38]. Recent 2026 literature highlights fucoidan-based nanoparticles, liposomes, and hydrogels as promising approaches for cancer drug delivery and other biomedical applications.

In oncology, fucoidan-based delivery systems may provide several theoretical advantages. Fucoidan can function as a structural component of a delivery vehicle while potentially contributing its own biological activity. In addition, its interactions with P-selectin and other molecular targets may offer opportunities for selective targeting of tumor-associated or inflamed tissues. Nanostructured fucoidan derivatives may also improve solubility, protect active compounds from degradation, and permit controlled or localized drug release [39-42]. Nevertheless, most of these approaches remain preclinical, and considerable work is required before their safety, manufacturing feasibility, biodistribution, and therapeutic advantages can be established in humans.

The development of fucoidan-based biomaterials may ultimately represent a more realistic pharmaceutical pathway than attempting to use native fucoidan as a conventional systemic drug. The combination of defined molecular structure, targeted delivery, controlled release, and mechanistically selected therapeutic cargo could potentially overcome some of the limitations associated with the oral administration of large heterogeneous polysaccharides [43].

8. Current Clinical Evidence: Critical Assessment

The overall evidence base for fucoidan is characterized by a marked discrepancy between extensive preclinical research and limited clinical validation. Cell and animal studies provide substantial evidence for anti-inflammatory, immunomodulatory, antitumor, antioxidant, anticoagulant, antiviral, and tissue-protective effects. However, these findings do not automatically translate into clinical efficacy. Differences in pharmacokinetics, tumor biology, dose exposure, metabolism, immune regulation, and disease complexity can substantially modify therapeutic effects in humans.

Clinical studies are generally limited by small sample sizes, short follow-up periods, heterogeneous fucoidan preparations, variable doses, lack of standardized pharmacokinetic measurements, and differences in patient populations and concomitant treatments. The systematic review of cancer studies is particularly informative because it identified only four eligible human studies and concluded that available clinical outcomes were inconsistent [44].

Thus, the current evidence hierarchy can reasonably be summarized as follows: preclinical evidence is substantial and biologically persuasive; small human studies demonstrate feasibility and potential pharmacodynamic effects; randomized clinical evidence remains limited; and evidence supporting fucoidan as an independent treatment capable of improving major clinical outcomes is insufficient.

Importantly, the clinical development of fucoidan is not stagnant. Current research increasingly investigates combination strategies, supportive oncology, inflammatory diseases, and advanced drug-delivery systems. Recent 2026 reviews emphasize combination therapy and precision-oriented applications rather than positioning fucoidan as a universal standalone treatment.

This represents a scientifically more defensible direction because fucoidan's multitarget biological profile may be particularly useful as an adjunctive modulator rather than as a replacement for highly specific modern therapies [45, 46].

9. Future Research Priorities and Conclusion

Several priorities should guide the future clinical development of fucoidan. First, structural and pharmaceutical standardization must be improved so that clinical trials evaluate chemically reproducible products. Second, dose-finding and pharmacokinetic studies should establish relationships between molecular characteristics, systemic exposure, gastrointestinal metabolism, and biological activity. Third, large, adequately powered randomized controlled trials are needed, particularly in oncology and inflammatory diseases. Fourth, studies should distinguish between fucoidan monotherapy and adjunctive treatment, because the existing evidence is more compatible with the latter approach. Fifth, clinically meaningful endpoints rather than isolated

biochemical changes should constitute the primary outcomes of efficacy trials. Sixth, drug–drug interaction studies should systematically evaluate anticoagulants, antiplatelet agents, chemotherapeutic drugs, targeted therapies, and immunotherapies. A further priority is the development of structure–activity relationships. Instead of treating all fucoidans as one therapeutic entity, future research should identify which molecular features are associated with specific effects. For example, a fucoidan optimized for anticoagulant activity may have substantially different structural requirements from one designed to modulate immune function or serve as a drug-delivery material. The development of synthetic and semi-synthetic libraries provides a promising strategy for addressing this problem. Finally, future clinical research should pay greater attention to the gut microbiome and the possibility that some biological effects occur through intestinal rather than systemic mechanisms. This may be particularly relevant to immunological and metabolic applications. Integration of pharmacokinetics, microbiome analysis, metabolomics, structural characterization, and clinical outcomes could substantially improve understanding of the relationship between fucoidan ingestion and therapeutic effects.

Fucoidan is a structurally diverse family of sulfated polysaccharides derived predominantly from brown seaweeds and represents one of the most extensively investigated marine-derived bioactive polymers. Its broad pharmacological profile includes anti-inflammatory, immunomodulatory, anticancer, antioxidant, anticoagulant, antithrombotic, antiviral, metabolic, cardiovascular, renal, and tissue-regenerative activities. The mechanistic and preclinical evidence is substantial, particularly regarding modulation of inflammation, apoptosis, angiogenesis, immune responses, oxidative stress, and coagulation.

Nevertheless, the clinical evidence remains considerably weaker than the preclinical literature. The available randomized and observational studies suggest that certain fucoidan preparations can be administered safely for relatively short periods and may influence selected biological or clinical parameters. The randomized metastatic colorectal cancer trial is particularly noteworthy because it demonstrated an improvement in disease-control rate when low-molecular-weight fucoidan was added to standard therapy; however, the absence of significant improvements in overall survival, progression-free survival, objective response, quality of life, or toxicity means that the study cannot establish fucoidan as an effective anticancer drug. The current evidence therefore does not justify replacing established medical treatments with fucoidan. Rather, fucoidan should be considered an investigational marine-derived bioactive polymer with potential roles in supportive oncology, immunomodulation, inflammatory disease, antithrombotic drug development, and advanced drug-delivery systems. Its future clinical relevance will depend on overcoming the major limitations of existing research, particularly structural heterogeneity, inadequate standardization, uncertain bioavailability, insufficient pharmacokinetic characterization, and the small size of human clinical trials.

As of 2026, the most scientifically appropriate conclusion is that fucoidan is promising but not yet an established therapeutic agent. Its biological activity is sufficiently compelling to justify continued translational research, but definitive claims regarding disease prevention or treatment require substantially stronger clinical evidence. The central question for future research should therefore not simply be whether "fucoidan works," but rather which structurally defined fucoidan preparation, at what dose, through which mechanism, in which patient population, and for which clinically meaningful endpoint can provide a reproducible therapeutic benefit. Answering this question will determine whether fucoidan ultimately progresses from a nutraceutical and experimental marine polysaccharide to a clinically validated pharmaceutical or biomedical platform.

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