Re-evaluating omega-3 (EPA/DHA) in cancer prevention and management

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Purpose of review
This review summarizes the most recent evidence on the role of omega-3 polyunsaturated fatty acids (PUFAs) in oncology, focusing on cancer prevention, cachexia and body composition, treatment-related toxicities, and therapeutic response.

Recent findings
Recent large-scale epidemiological and biomarker-based studies confirm a consistent, dose-dependent inverse association between eicosapentaenoic acid (EPA) and colorectal cancer risk, while evidence for docosahexaenoic acid (DHA) remains less consistent. Observational data reinforce the protective role of omega-3-rich dietary patterns, such as the Mediterranean and MIND diets, in reducing cancer risk. In clinical settings, omega-3 PUFA supplementation has shown modest but significant clinical benefits on body weight, inflammation, and quality of life in cancer cachexia, though effects on lean mass are variable. Promising data also support a reduction in severe oral mucositis during chemotherapy, whereas trials on chemotherapy-induced peripheral neuropathy and chemosensitization have produced inconclusive results, mainly due to heterogeneity in design and adherence.

Summary
Omega-3 fatty acids exert measurable biological and clinical effects in oncology, however, their benefits appear context-dependent. Future studies should focus on standardized interventions, patient stratification based on molecular and inflammatory profiles, and integration within immunonutrition frameworks to enhance therapeutic precision and clinical outcomes.

Keywords
cancer risk and prevention, docosahexaenoic acid, eicosapentaenoic acid, inflammation, omega-3 fatty acids

INTRODUCTION

Over the last decade, research on omega-3 polyunsaturated fatty acids (PUFAs) in oncology has expanded considerably, reflecting the growing recognition of the complex interplay between nutrition, inflammation, and cancer progression. We previously focused on the evidence supporting the potential of omega-3 fatty acids – particularly eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) – as modulators of tumor-associated inflammation, contributors to the prevention and management of cancer cachexia, and possible enhancers of chemotherapy response, while reducing treatment-related toxicity [1]. At that time, although preliminary results were promising, evidence remained fragmented and limited by studies conducted on limited cohorts of patients, heterogeneous clinical settings, and methodological challenges.

In recent years, advances in both preclinical and clinical research have provided new insights into the mechanisms and potential therapeutic strategies of omega-3 fatty acids in oncology. Novel data have emerged regarding their role in modelling the tumor microenvironment, influencing immune and inflammatory pathways, and modulating skeletal muscle and adipose tissue metabolism. In addition, their integration with anticancer therapies – including chemotherapy, immunotherapy, and targeted agents – has gained renewed attention, supported

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KEY POINTS

- Recent studies strengthen the mechanistic rationale for omega-3 fatty acids supplementation as modulators of inflammation, metabolism, and treatment response in cancer.
- Omega-3 fatty acids are linked to reduced cancer risk in observational studies, but clinical trials remain inconclusive, underscoring the dominant role of dietary patterns in cancer prevention.
- In cancer cachexia, omega-3 supplementation modestly improves body weight, inflammatory status, and quality of life with uncertainty on the effect on body composition, in particular lean body mass.
- Omega-3 fatty acids may reduce the severity of chemotherapy-induced oral mucositis, while evidence for preventing peripheral neuropathy or enhancing chemotherapy efficacy remains unclear.
- Future research should focus on molecular and inflammatory patient stratification and standardized immunonutrition strategies to optimize omega-3 therapeutic benefits in oncology.

by early-phase clinical studies and translational research.

Beyond their anti-inflammatory properties, EPA and DHA are now recognized as precursors of specialized pro-resolving mediators (SPMs) – including resolvins, protectins, and maresins – which actively promote the resolution of inflammation. These lipid mediators modulate leukocyte migration, cytokine production, macrophage polarization, and tissue repair, thereby restoring immune homeostasis within the tumor microenvironment. In cancer, SPMs have been shown to limit chronic inflammation, influence angiogenesis, and potentially enhance responsiveness to chemotherapy and immunotherapy.

For these reasons, the aim of this review is to summarize the most relevant evidence published in the last two years on omega-3 fatty acids in cancer, with a focus on their impact on cancer prevention, cachexia, treatment-related toxicities, and therapeutic response. By comparing the latest data with the conclusions of previous data, we seek to provide an integrated perspective on the current status and future directions of omega-3 fatty acid research in oncology.

OMEGA-3 FATTY ACIDS IN THE PREVENTION OF CANCER

Omega-3 fatty acids, particularly EPA and DHA, have attracted increasing attention for their potential role in cancer prevention (Fig. 1), as well as in cancer journey including during anticancer treatments and protection from complications (Table 1). These long-chain polyunsaturated fatty acids counterbalance the pro-inflammatory actions of omega-6 fatty acids and modulate several biological pathways implicated in carcinogenesis, including prostaglandin E2 synthesis, aromatase activity, apoptosis, and insulin sensitivity [1]. However, the largest randomized, placebo-controlled trial conducted to date on marine omega-3 fatty acids supplementation, the VITAL study [2], which enrolled over 25 000 participants, did not demonstrate a reduction in the incidence of invasive cancer [hazard ratio (HR) 1.03; 95% confidence interval (CI), 0.93–1.13] [2]. However, the VITAL trial showed a nonsignificant

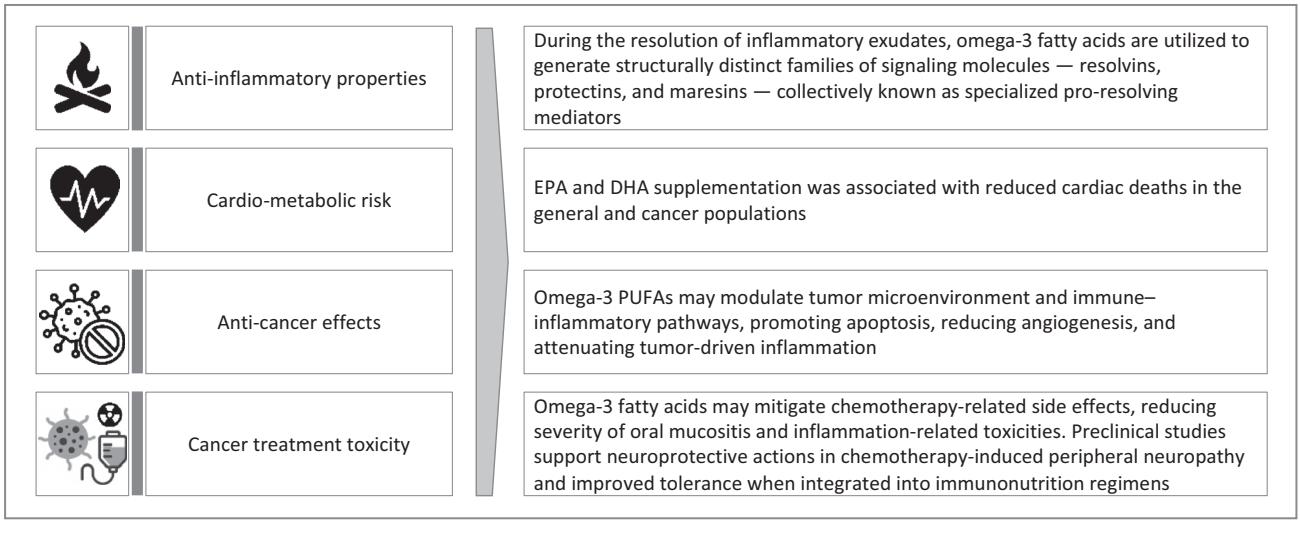

FIGURE 1. Biological and clinical effects of omega-3 fatty acids in cancer.

Table 1. Potential mechanisms and clinical impact of omega-3 fatty acids from cancer prevention to cachexia management

	Healthy At risk for cancer	Cancer diagnosis: treatment toxicity	Response to anti-cancer treatment	Cancer cachexia
Protective biological mechanisms	• Modulation of inflammation and insuline resistance • Increased cancer cell apoptosis • Reduced angiogenesis	• In *vivo* studies showed protective effect against chemotherapy-induced peripheral neuropathy and oral mucositis	• Omega-3 fatty acids can promote DNA damage in cancer cells • Inhibit the activation of various signaling pathways involved in chemoresistance	• Modulation of inflammation may increase the efficacy of nutritional intervention
Clinical effects	• Epidemiological data showed protective effects against the development of breast and colon cancer	• The findings partly confirm a protective effect against oral mucositis in patients undergoing anticancer treatments	• No clinical trial has conclusively demonstrated an improved response to anticancer therapy	• Omega-3 fatty acids can reduce inflammation, promote weight gain, and improve quality of life in cachexia, although evidence of their impact on lean body mass is still limited.

reduction in the composite endpoint of total invasive cancer and nonfatal myocardial infarction, but some follow-up studies have suggested possible benefits for specific subgroups or cancer types (e.g., reduced mortality). These findings suggest that, despite strong mechanistic plausibility and supportive preclinical and observational evidence, clinical benefits of omega-3 fatty acid supplementation in cancer prevention remain unproven, underscoring the need for further targeted trials in at-risk populations or specific cancer types. Indeed, more recent epidemiological investigations on large cohorts have provided additional insights, particularly in colorectal cancer. A case-control study in Korea demonstrated that higher dietary intake of alpha-linolenic acid (ALA), the main plant-derived omega-3 fatty acid, was inversely associated with colorectal cancer risk [adjusted odds ratio (OR) = 0.58; 95% CI, 0.45–0.75], with the protective effect being more evident in individuals carrying the MUC4 rs2246901 major allele, suggesting an interaction between genes and diet [3]. Biomarker-based analyses from the UK Biobank showed that higher plasma concentrations of total omega-3 PUFAs and DHA were associated with a reduced colorectal cancer risk (HR 0.88 and 0.89 for intermediate tertiles), with evidence of a nonlinear "plateau effect" and stronger associations for proximal colon cancers, for males but not for females [4▪]. Interestingly, a recent meta-analysis of 26 prospective cohorts found that higher EPA exposure was consistently associated with reduced colorectal cancer risk, with each 100 mg/day dietary increase linked to a modest 5% risk reduction and circulating EPA showing a stronger, dose-dependent inverse association [relative risk (RR) 0.86; 95% CI 0.80–0.92]. By contrast, DHA showed weaker and less consistent associations [5]. The roles of DHA and EPA in cancer prevention are likely distinct, reflecting their differing functions in inflammation, lipid metabolism, and as precursors of SPMs.

A large metanalysis conducted on 21 prospective cohort studies demonstrated that higher dietary intake of marine omega-3 polyunsaturated fatty acids was associated with a 14% reduction in breast cancer risk [6]. However, to our knowledge, no recent data are present in intervention trials aimed at confirming the reduction of cancer risk.

In cancer prevention, growing evidence highlights the importance of overall dietary patterns, rather than isolated nutrients, in shaping long-term risk and protective effects. A pivotal study [7▪▪] based on more than 180 000 UK Biobank participants demonstrated that adherence to a Mediterranean or MIND dietary pattern was consistently associated with reduced risk of overall and multiple site-specific cancers, an effect partly mediated by specific circulating metabolites. This observation emerged also in other clinical settings. For instance, evidence from a large cohort of older adults indicates that adherence to healthy dietary patterns such as the MIND, Alternative Healthy Eating Index, and Mediterranean diet is associated with a slower accumulation of multimorbidity, particularly in cardiovascular and neuropsychiatric domains, whereas pro-inflammatory diets accelerate disease clustering [8▪]. These data reinforce the concept that overall diet quality, rather than single-nutrient supplementation, is a central determinant of long-term disease trajectories and healthy aging [8▪].

OMEGA-3 FATTY ACIDS AND CANCER CACHEXIA

Cancer cachexia represents a complex metabolic syndrome in which tumor-driven alterations profoundly reshape host metabolism, leading to muscle wasting, fat redistribution, and systemic inflammation [9]. In this setting, omega-3 PUFAs have emerged as promising candidates, with preclinical and early clinical studies suggesting their potential to attenuate systemic inflammation, modulate resting energy expenditure, and preserve muscle mass, thereby mitigating the detrimental host–tumor metabolic interplay [10▪].

Impact on body weight and quality of life

Over the last two years, evidence on omega-3 PUFAs for cancer cachexia can be considered inconsistent. A dose–response meta-analysis of RCTs specific to cachexia [11] found small weight gains with supplementation, particularly in older patients and with lower body weight, though overall certainty of the evidence was low and effects on body composition were inconsistent [11]. A multicenter phase II study in advanced gastric cancer patients with cachexia showed that adherence to an oral EPA-enriched nutritional supplement was associated with longer time to treatment failure and improved overall survival, despite the primary endpoint (i.e., time to treatment failure in patients adhering to $\geq$25% of the planned ONS-EPA dose in the first two weeks) was not reached [12]. Importantly, patients consuming at least 0.5 g/day of EPA demonstrated better inflammatory and nutritional profiles, highlighting the role of adherence and baseline inflammatory burden in affecting outcomes [12]. Similarly, a 2024 meta-analysis in patients with advanced non-small cell lung cancer (NSCLC) demonstrated that omega-3 supplementation led to significant increases in body weight and improvements in health-related quality of life scores, though without measurable gains in lean body mass [13]. Conversely, a 2025 double-blind RCT in locally advanced head-and-neck cancer did not demonstrate significant advantages of EPA over placebo for weight, BMI, or skeletal muscle, with notable difficulties in adherence to the supplementation [14].

Immunonutrition in cancer cachexia

These findings align with the field of immunonutrition, where combinations of omega-3 fatty acids, arginine, and nucleotides have been investigated for their capacity to modulate host immune and inflammatory responses. In fact, in surgical oncology, preoperative immunonutrition has shown promise in reducing postoperative infectious complications and supporting recovery [15]. For instance, the Korean multicenter randomized clinical trial (K-CROSS) specifically aims to target malnourished colorectal cancer patients (NRS 2002 score 3–5) to evaluate whether a 7-day preoperative regimen of arginine- and omega-3 enriched oral supplements can reduce infectious complications and improve perioperative nutritional and immune profiles [16].

In summary, recent evidence confirms the potential of omega-3 fatty acids to modulate inflammation, body weight, and quality of life in cancer cachexia, although effects on lean body mass remain inconsistent. This evidence is in line with previous observations [1], where the importance of integrating chemotherapy and nutritional strategies as a combined host- and tumor-targeted approach was emphasized. Importantly, in the latest ESPEN guidelines of Clinical Nutrition in cancer there is recommendation for patients with advanced cancer undergoing chemotherapy and at risk of weight loss or malnourished, to use supplements containing long-chain omega-3 fatty acids or fish oil in order to stabilize or improve appetite, food intake, lean body mass, and body weight [17]. Up to date, the field of immunonutrition extends this concept, where omega-3 fatty acids are not used as isolated agents but as components of formulations aimed at modulating inflammation, immunity, and treatment tolerance. Future research should possibly focus on patient stratification, standardized supplementation protocols, and combination strategies, to finally establish the clinical role of omega-3 fatty acids in the setting of immunonutrition for cancer cachexia management.

OMEGA-3 FATTY ACIDS, CANCER TREATMENT TOXICITY AND RESPONSE TO TREATMENT

Impact on treatment toxicity

Omega-3 fatty acids have been widely investigated for their potential role in preventing cancer treatment toxicity. Preclinical studies demonstrated their antitumor activity and their ability to mitigate chemotherapy-related side effects, possibly contributing to improved clinical outcomes [18▪▪]. Moreover, omega-3 PUFAs may influence cancer development and progression through their enzymatic conversion into bioactive endogenous proresolving metabolites [19]. All these protective mechanisms found strong rationale in several preclinical studies. Recently, Melato *et al.* [20▪], in mice tested EPA/DHA-enriched

fish oil (~55% EPA, 37% DHA) administered for 30 days in models of oxaliplatin and paclitaxel neuropathy. Interestingly, omega-3 supplementation prevented the development of neuropathic pain behaviors. Also, in oxaliplatin-treated mice, omega-3 blocked acute cold hypersensitivity and chronic pain, correlating with reduced spinal cord microglial activation and lower pro-inflammatory cytokine levels in nervous tissue, providing a biological basis for the clinical trials of omega-3 in chemotherapy-induced peripheral neuropathy [20▪]. However, in humans the trials of omega-3 fatty acids for mitigating chemotherapy-induced peripheral neuropathy obtained mixed results. For instance, a pilot study on 60 breast cancer patients on weekly paclitaxel reported no benefit of high-dose omega-3 (4 g/day ethyl esters; 38% DHA and 47% EPA) for preventing acute paclitaxel-associated pain syndrome or subsequent peripheral neuropathy [21]. In particular, the incidence of neuropathic pain and sensory neuropathy was similar in omega-3 vs. placebo arms (84% vs. 88%) and patients receiving omega-3 used slightly more analgesics and experienced higher grade of peripheral neuropathy even if not statistically significant [21]. Recently, a study protocol was published of the OxaNeuro trial to test whether high-dose fish oil rich in EPA and DHA can prevent oxaliplatin-induced peripheral neuropathy in colorectal cancer patients. This randomized, placebo-controlled study may provide information on neuroinflammatory and biomarker mechanisms to improve treatment tolerance and quality of life in this setting [22].

Intensive research was also conducted on the role of omega-3 in gastrointestinal protection during chemotherapy. A recent meta-analysis including five studies and 337 patients evaluated the efficacy of omega-3 fatty acids for preventing and treating oral mucositis in cancer patients undergoing anticancer therapy. Although omega-3 supplementation (both EPA and DHA) did not significantly reduce the overall incidence of oral mucositis (RR 0.50, 95% CI 0.25–1.01), it markedly decreased the incidence of severe oral mucositis (RR 0.31, 95% CI 0.17–0.56) and significantly alleviated associated pain (SMD −1.61, 95% CI −2.79 to −0.43), with no heterogeneity across studies [23]. These findings suggest that omega-3 fatty acids may exert clinically meaningful protective effects on mucosal integrity, likely mediated through their anti-inflammatory and pro-resolving properties [23].

Impact on response to treatment

Interesting findings are related on the combination of omega-3 in association with chemotherapy in cancer. The DHA-WIN phase II randomized trial aimed to evaluate whether high-dose docosahexaenoic acid (4.4 g/day) administered during neoadjuvant chemotherapy could enhance treatment efficacy in breast cancer patients by reducing tumor proliferation (Ki-67) and improving pathological complete response (pCR). The study found that while DHA supplementation was safe and well tolerated, it did not significantly affect Ki-67, pCR, or survival, though a trend toward greater Ki-67 reduction emerged in the HER2-positive subgroup [24].

These findings are particularly interesting given the potential subtype-specific benefits observed in HER2-positive breast cancer. In this light, in a cohort of breast cancer patients naïve to any anticancer treatment, we detected differential plasma levels of D-series Resolvins – bioactive metabolites derived from DHA – according to BRCA1/2 mutation status and tumor immunohistochemical profile [25]. We also observed in a similar cohort that DHA oral supplementation (2 g/day for 10 days) significantly increased plasma Resolvin D1 and D2 in BRCA1/2-mutated patients, while familial breast cancer patients showed a decrease in Resolvin D1 levels, suggesting divergent inflammation-resolving capacities. Moreover, patients with low Ki-67 expression exhibited a greater increase in Resolvin D2 levels, suggesting that the modulation of DHA-derived pro-resolving mediators may partly depend on the intrinsic biological characteristics of the disease [26▪].

Overall, these data highlight the heterogeneity of response to omega-3 supplementation across cancer settings and suggest that the clinical efficacy of DHA and its bioactive metabolites may depend on specific tumor and host characteristics. Therefore, precise phenotyping – integrating molecular, genetic, and inflammatory profiles – appears essential to identify patients who are most likely to benefit from omega-3 supplementation during cancer. Future studies should aim to standardize key methodological aspects to enhance comparability and clinical translation of findings. In particular, the dose and formulation of omega-3 fatty acids should be clearly defined, including the relative proportions of EPA and DHA, to allow consistent evaluation of their biological and clinical effects. Also, the duration of intervention should be aligned with the clinical context – distinguishing short-term supplementation protocols from long-term strategies for chronic cachexia management. Finally, the use of the omega-3 index – quantifying red blood cell EPA+DHA content – should be encouraged as a standardized biomarker of adherence and biological exposure.

CONCLUSION

Over the past two years, evidence on omega-3 fatty acids in oncology has evolved toward a deeper and mechanistic understanding. Omega-3 fatty acids found potential biological mechanisms and clinical effects across different phases of the cancer journey – from primary prevention in at-risk individuals to modulation of treatment toxicity, enhancement of therapeutic response, and management of cancer cachexia.

On the positive side, consistent epidemiological and biomarker-based data confirm inverse associations between EPA and the risk of colorectal cancer, with dose-dependent effects supporting a true biological gradient. Similarly, observational evidence highlights the role of omega-3-rich dietary patterns, such as the Mediterranean and MIND diets, in lowering overall and site-specific cancer risk. In the clinical setting, several trials in patients with cancer cachexia demonstrated benefits on body weight, inflammation, and quality of life, particularly when adherence was adequate and inflammatory burden was high at baseline. Moreover, meta-analyses in supportive care suggest that omega-3 fatty acids can reduce the severity of chemotherapy-induced oral mucositis, supporting their role in maintaining mucosal integrity and improving tolerance to treatment.

Conversely, large interventional studies, including the VITAL trial, have not confirmed a preventive effect of omega-3 supplementation on overall cancer incidence, emphasizing the limitations of generalized supplementation approaches. Similarly, despite promising preclinical findings, randomized clinical trials assessing omega-3 in chemotherapy-induced peripheral neuropathy and as chemosensitizing agents have yielded inconclusive results, often limited by heterogeneity in formulations, doses, and compliance.

Taken together, these findings suggest that omega-3 fatty acids exert measurable biological effects in oncology, yet their clinical efficacy remains context-dependent. Further research should prioritize patient stratification based on molecular and inflammatory phenotypes, standardized intervention designs, and integration within multimodal immunonutrition strategies to unlock their full therapeutic potential in cancer prevention and management.

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Conflicts of interest

There are no conflicts of interest.

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