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Nutritional modulation of immune response in colorectal cancer: a mini review

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Abstract

The intricate relationship between diet, specific nutrients, and the immune system may play a key role in both the prevention and management of various diseases, including colorectal cancer. Chronic inflammation and a protracted immune activity, driven by elements such as lymphocytes, macrophages, and pro-inflammatory cytokines, can contribute to tumor progression and changes in the tumor microenvironment. As immune responses are critical to block cancer initiation and progression, enhancing immune function and regulating inflammation through stress management and the use of immunonutrients has been proposed as a promising approach to support the clinical management of colorectal cancer patients.

Keywords Immune nutrition, Colorectal cancer, Immune response, Tumor microenvironment, Immunonutrients

1 Introduction

Around one third of cancer deaths are related to modifiable risk factors, for example sedentary lifestyle, obesity, tobacco and alcohol intake, and a diet poor in fruit and vegetables [1]. Diet and nutrients are closely connected to immune function, and this relationship may influence both the prevention and management of various illnesses, including colorectal cancer (CRC); in fact, chemical substrates originated from nutrient metabolism could play a role as either enhancers or inhibitors of enzymatic processes, impacting key molecular mechanisms involved in inflammation, response to microorganisms, and oxidative stress [2, 3]. Chronic inflammation is a recognized factor in CRC development, largely due to disruptions in immune signaling pathways. A sustained immune response, characterized by the activity of immune cells like lymphocytes and macrophages, as well as inflammatory mediators such as tumor necrosis factor (TNF)-α, interleukin (IL)−6, and IL-1β, can contribute to tumor growth and alter the tumor microenvironment (TME). Additionally, chronic inflammation may trigger signaling cascades such as NF-κB and MAPK, which are known to facilitate genetic instability and promote the emergence of more aggressive cancer cell types [4]. Immune responses are critical to block cancer initiation and progression; nonetheless, further studies are needed to investigate how nutritional factors can contribute to balance immune responses as regards prevention and treatment of CRC. The aim of this paper is to offer a brief overview of the current literature regarding

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the interaction between nutrients and the immune system, which could potentially have an effect on the management of CRC.

2 Search strategy

A comprehensive literature search was conducted to identify relevant studies exploring the impact of nutrition on immune responses in CRC. Electronic databases including PubMed, Google Scholar and Scopus were searched for peer-reviewed articles published in English between January 2000 and January 2025. The search strategy combined keywords and MeSH terms such as "colorectal cancer," "immune response," "nutrition," "dietary modulation," "nutrients," "immunonutrition," and "tumor microenvironment." Studies were selected based on their relevance to the topic, with a focus on experimental, clinical, and review articles addressing the interaction between specific nutrients or dietary patterns and immune mechanisms in CRC. Articles not directly related to colorectal cancer or immune modulation through nutrition were excluded. Reference lists of key studies were also screened to identify additional relevant publications. Moreover, a manual search was conducted on textbooks of medicine, surgery, immunology and gastroenterology. Limitations of this review included the only inclusion of published studies, articles published in English and the lack of systematic comparison between studies (Table 1).

3 Potential therapeutic strategies

Currently, the definition of immune nutrition (IN) is not globally accepted, but it generally refers to the intake of specific bioactive nutrients in doses exceeding those found in standard diets. Common IN formulations often include combinations of arginine (Arg), omega-3 polyunsaturated fatty acids (PUFAs), nucleotides, glutamine (Glu), and antioxidants [5, 6]. Recent strategies aimed at improving immune defenses and controlling inflammatory responses to physiological stress. The use of immunonutrients has also been proposed as a beneficial intervention in CRC patients undergoing surgery [7]. A retrospective study by Ambrosio et al. highlighted that IN administered to subjects undergoing elective surgery for CRC, could improve immune function by favorably influencing the TME, lowering the incidence of postoperative complications, shortening length of stay (LOS), and potentially improving overall survival [8]. These promising results have been supported by various systematic reviews and meta-analyses, suggesting that IN may play a valuable role in optimizing clinical outcomes and reducing healthcare costs [9]. Moreover, IN has shown benefits in other critical conditions such as sepsis, acute pancreatitis, acute respiratory distress syndrome (ARDS), and critical illness [10, 11]. IN has been demonstrated to have the potential to enhance both humoral and T-cell immune response [12]. The European Society for Clinical Nutrition and Metabolism (ESPEN) recommends IN for malnourished patients, and it is incorporated into Enhanced Recovery After Surgery (ERAS) protocols [13]. Malnutrition, immune dysregulation, and gastrointestinal impairment are commonly observed in CRC patients, often as a result of prolonged disease progression, reduced nutrient intake, and surgical stress. Studies report that 15–40% of oncology patients present with malnutrition at diagnosis, with prevalence increasing to 80–90% in advanced stages [14]. Among CRC patients, malnutrition rates range from 45 to 60%, and these numbers tend to rise following radical surgery [15]. Furthermore, alterations in immune function or immunosuppression due to surgery represent a leading cause of post-operative complications; in fact, a number of studies found a link between post-operative complications (i.e. surgical site infection,

Table 1 Screening criteria for study selection

Criteria Type	Inclusion Criteria	Exclusion Criteria
Population	Human subjects diagnosed with colorectal cancer	Animal studies, in vitro studies not involving human-derived immune cells
Intervention	Nutritional factors (e.g., specific nutrients, dietary patterns, supplements)	Non-nutritional interventions (e.g., drugs, surgery, chemotherapy alone)
Outcome	Effects on immune response (e.g., cytokine levels, immune cell profiles, inflammation)	Studies without immune-related outcomes
Study Design	Clinical trials, cohort studies, case–control studies, systematic reviews, narrative reviews, meta-analyses	Editorials, commentaries, abstracts without full texts, non-peer-reviewed sources
Language	English	Non-English publications
Publication Date	January 2000 – January 2025	Studies published outside this range
Relevance	Direct relevance to interaction between nutrition and immune response in CRC	Studies focusing on nutrition in CRC but without immune endpoints

intra-abdominal abscess, anastomotic leakage, bleeding and intestinal obstruction) and immune dysfunction or malnutrition [16].

4 Glutamine

Glutamine is an important element of IN, representing a fundamental fount of energy for the gut and being involved in the improvement of intestinal function. Numerous studies have highlighted the beneficial role of glutamine in CRC patients who have undergone radical surgery. Elevated serum glutamine levels have been associated with improved overall survival and progression-free survival, suggesting its potential as a prognostic marker in CRC cases [17, 18]. A meta-analysis of 31 randomized, prospective clinical trials found that glutamine supplementation enhanced immune response and reduced post-surgical complications in CRC patients following surgery. Specifically, significant increases in humoral immunity markers, such as IgA, IgM, and IgG, were observed in patients receiving glutamine compared to controls. Furthermore, the analysis revealed that glutamine influenced T-cell immune activity, with a notable rise in CD4 + cells and a decrease in CD8 + cells, indicating a regulatory effect on T-cell dysfunction. The incidence of post-operative complications was lower among patients who received glutamine. The combined data demonstrated significant reductions in surgical site infections (RR = 0.48, 95% CI: 0.30–0.75), anastomotic leakage (RR = 0.23, 95% CI: 0.09–0.61), and LOS (SMD = −1.13, 95% CI: −1.68 to −0.58) compared to the control group. Overall, the evidence supports the use of glutamine as a promising IN therapy following radical surgery in CRC patients [12].

5 Omega-3 polyunsaturated fatty acids

PUFAs represent an important component of IN for patients due to their anti-inflammatory effects, which could enhance immune response. A meta-analysis of 20 studies found that omega-3 PUFAs significantly enhanced humoral immunity indicators, including IgA and IgG, as well as T-cell immunity markers such as CD3 +, CD4 +, and the CD4 +/CD8 + ratio. In contrast, levels of CD8 + cells were notably decreased following omega-3 PUFA supplementation. Additionally, the analysis revealed improvements in nutritional status indicators, including total protein, albumin, and prealbumin levels. These findings support the potential role of omega-3 PUFAs as an effective immunonutritional therapy for CRC patients post-surgery [19]. Another meta-analysis demonstrated that omega-3 PUFA supplementation significantly lowered inflammatory markers like IL-6, C-reactive protein, and TNF-α, though it had no significant impact on IL-1β. Also, it has been observed a slight increase in serum albumin levels and a decrease in post-operative infectious complications and LOS [20].

6 Arginine

A systematic review of nine studies found a positive association between intake of Arg and CRC [21]. These protective effects could be mainly due to the increase of inducible nitric oxide (NO) synthase expression and consequently NO levels after Arg administration. Furthermore, Arg intake could decrease cell growth in CRC and it could improve immune function after surgery [22, 23]. However, although the positive effects of Arg in subjects with CRC were described in many studies, these findings need to be confirmed in further well-designed trials before drawing firm conclusions.

7 Antioxidants

Recently, exogenous antioxidants have been widely investigated in both preclinical and clinical settings due to their potential as therapeutic agents for CRC [24]. These compounds fall into three main categories: vitamins and their derivatives, minerals, and polyphenols. Among them, polyphenols represent the largest group and are further divided into flavonoids and phenolic acids. Flavonoids, such as anthocyanins, apigenin, quercetin, and epigallocatechin-3-gallate, are abundant in fruits, vegetables, and various medicinal plants. Plant derived antioxidants are largely studied for different potential anti-cancer treatments including those for CRC [25]. Numerous studies have indicated that diets rich in flavonoids may lower the risk of developing CRC and mitigate disease severity [26]. The protective effects of flavonoids in the colon are believed to stem from their ability to influence key cellular processes, such as apoptosis, cell proliferation, and inflammation, as well as their role in modulating gut microbiota [27]. Further studies are needed to assess the individual impacts of specific flavonoids and their metabolites on CRC development. Such insights could help inform the creation of targeted nutritional strategies aimed at maximizing the intake of the most effective bioactive compounds.

8 Vitamin D

Vitamin D plays a crucial role in regulating gene expression by activating the nuclear vitamin D receptor (VDR), which contributes to its wide-ranging anticancer effects. These effects include inhibiting cell proliferation, promoting cell differentiation and programmed cell death, limiting the formation of new blood vessels, and reducing inflammation. Through the VDR, vitamin D influences both innate and adaptive immune responses, modulating the balance between Th1 and Th2 cells, affecting dendritic cell development, decreasing the proliferation

Table 2 Immunomodulatory effects of selected nutrients in colorectal cancer

Nutrient	Immunomodulatory effects	Key immune mechanisms	Stage of evidence
Glutamine	Supports immune cell metabolism; maintains intestinal barrier; reduces treatment-related immune dysfunction	Fuel for lymphocytes and macrophages; preservation of gut-associated lymphoid tissue; cytokine regulation	Preclinical; limited clinical
Omega-3 PUFAs (EPA/DHA)	Reduces tumor-associated inflammation; promotes antitumor immune responses	↓ COX-2/PGE_2; macrophage polarization (M2 → M1); modulation of T-cell signaling	Clinical and preclinical
Arginine	Enhances T-cell–mediated immunity; improves immune competence in cancer patients	↑ T-cell proliferation; nitric oxide–dependent immune signaling	Preclinical; early clinical
Antioxidants (vitamins C, E, carotenoids, polyphenols)	Reduces oxidative stress–induced immune suppression; modulates inflammatory signaling	↓ ROS; inhibition of NF-κB and pro-inflammatory cytokines; enhanced NK and T-cell activity	Primarily preclinical; limited clinical
Vitamin D	Regulates innate and adaptive immunity; may enhance immune surveillance in CRC	Modulation of dendritic cells; ↓ Th17 inflammation; improved T-cell balance	Clinical and preclinical
Dietary fiber	Promotes microbiota-driven immune regulation; reduces chronic inflammation	SCFA (butyrate) production; ↑ $CD8^+$ T-cell function; Treg homeostasis	Clinical and preclinical

of regulatory T cells (Tregs), and regulating T-cell receptor signaling. This suggests that vitamin D is essential for maintaining immune balance. Higher pre- and post-diagnosis levels of circulating 25-hydroxyvitamin D have been consistently associated with better survival outcomes in CRC patients across various stages [28]. This link is further supported by a phase II randomized controlled trial, which demonstrated improved progression-free survival in metastatic CRC patients receiving high doses of vitamin D [29]. Additionally, one study found that the protective effect of elevated vitamin D levels was particularly evident in CRC cases characterized by strong immune responses and high infiltration of CD3 + T cells, indicating a potential role for vitamin D in enhancing cancer immunoprevention through its interaction with the immune system [30].

9 Fiber

Studies have shown that a higher intake of dietary fiber, especially from cereal sources and whole grains, after a CRC diagnosis is linked to improved survival outcomes. Patients who increased their fiber consumption following diagnosis, compared to their intake prior, demonstrated significantly better survival rates [31]. These results indicate that the benefits of fiber may go beyond just reducing the risk of developing CRC and may also play a role in enhancing outcomes after diagnosis. In addition to its positive effects on metabolism, such as improving insulin sensitivity and lipid levels, fiber also influences both systemic and gut-related immune responses. Diets rich in fiber or whole grains have been associated with reductions in circulating inflammatory markers. Furthermore, soluble fiber is fermented by gut microbiota into short-chain fatty acids (SCFAs), which are known to help regulate intestinal immune function and may offer protection against CRC [32]. Fiber intake has also been found to counteract damage to the colonic mucus layer caused by a Western-style diet, partly through promoting the growth of Bifidobacteria, gut microbes with anti-inflammatory and potential anticancer effects [33] (Table 2).

10 Changes in tumor microenvironment

Diet and gut microbiota can influence the tumor microenvironment (TME), potentially shifting intra-tumoral balance toward cancer cell death by activating immune-mediated antitumor inflammation [34]. Indeed, bioactive dietary compounds warrant further investigation for their ability to enhance anti-cancer immune responses. Some studies have explored the role of oral IN in modulating the TME. For instance, a prospective study by D'Ignazio et al. involving 24 patients with gastric or colorectal cancer receiving IN showed several immunological changes: increased levels of T-helper and cytotoxic T cells, reduced populations of exhausted and regulatory T cells, a shift toward M1 macrophage polarization, a reduced number of CD163 + macrophages, and suppression of the PD-1/PD-L1 immune checkpoint pathway. Additionally, IN may help reduce treatment-related side effects, particularly mucositis, in patients undergoing chemoradiotherapy [35]. Boucher et al. further suggested that dietary bioactive components play a key role in shaping gut microbiota composition, which in turn can influence immune responses against tumors. Their findings demonstrated that a diet enriched with inulin, a prebiotic that promotes immunostimulatory bacterial growth, enhanced Th1-polarized CD4 + and CD8 + αβ T-cell responses and slowed tumor progression [36].

11 Conclusions

A number of studies have shown that IN was able to improve the immune response against CRC by beneficially altering the TME, lowering postoperative complication rates and LOS, and potentially extending overall survival. These findings may play an important role in improving clinical management and reducing healthcare expenditures in CRC surgical care. Also, promising results showed that dietary components may influence gut microbiota composition, thereby impacting immune function in CRC patients. Nonetheless, more research is necessary to fully understand the impact of IN on immune regulation, patient survival, and quality of life in the context of CRC.

Abbreviations

ARDS	Acute respiratory distress syndrome
CRC	Colorectal cancer
ERAS	Enhanced Recovery After Surgery
ESPEN	European Society for Clinical Nutrition and Metabolism
IL	Interleukin
IN	Immune nutrition
LOS	Length of stay
NO	Nitric oxide
PUFAs	Polyunsaturated fatty acids
SCFAs	Short-chain fatty acids
TME	Tumor microenvironment
TNF	Tumor necrosis factor
VDR	Vitamin D receptor

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