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REVIEW ARTICLE

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Nutrology of probiotics and nutrients in cancer patients: a systematic review

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Abstract

Introduction: In the scenario of immunomodulation by regulatory T cells (Tregs), studies show that the increase in Bifidobacterium probiotics, in addition to colonizing the intestine, alters the intestinal microbiota, activating Treg cells, which produces anti-tumor effects. **Objective:** This was to establish, through a systematic review study, the main clinical studies that show the positive effects of probiotics and nutrients in stimulating regulatory T cells in cancer patients, to enable immunotherapy for the control, reduction, or even elimination of cancer cells. **Methods:** The systematic review rules of the PRISMA Platform were followed. The search was carried out from April to May 2025 in the Scopus, PubMed, Science Direct, Scielo, and Google Scholar databases. The quality of the studies was based on the GRADE instrument and the risk of bias was analyzed according to the Cochrane instrument. **Results and Conclusion:** 141 articles were found. A total of 40 articles were fully assessed and 31 were included and developed in the present systematic review study. Considering the Cochrane tool for risk of bias, the overall assessment resulted in 26 studies with a high risk of bias and 24 studies that did not meet GRADE and AMSTAR-2. Most studies showed homogeneity in their results, with X^2=67.8%>50%. It was concluded that probiotics, together with intestinal microbiota, have been increasingly proposed to improve immune checkpoint blockade treatments against cancer by activating Treg cells. Oral administration of butyrate, propionate, and acetate, individually or in combination, led to an increase in the number of Treg cells in the colon. Also, all-trans retinoic acid (atRA), a bioactive form of vitamin A, stimulates Treg cells in the human intestine.

Keywords: Probiotics. Nutrients. Regulatory T cells. Cancer.

Introduction

In the context of immunomodulation by regulatory T cells (Tregs), studies show that increasing Bifidobacterium probiotics not only colonizes the gut but also alters the gut microbiota, thereby activating Tregs. This treatment has been found to activate the anti-CTLA-4 antibody - a checkpoint inhibitor that

frequently causes autoimmunity in humans undergoing cancer treatment. This effect stems from the probiotic's impact on CD4+ regulatory cells, altering their metabolic and immunosuppressive functions. These CD4+ regulatory T cells are a key mechanism for controlling immune system

autoreactivity in humans **[1]**.

In this context, immunotherapy boosts the host immune system to generate anti-tumor effects; specifically, immune checkpoint blockade has led to remarkable advances in the treatment of a wide range of malignancies **[2,3]**. This treatment can use monoclonal antibodies to block negative immune regulatory factors within the tumor microenvironment, thereby enhancing tumor immunosurveillance and boosting host immune activity against tumors **[4,5]**.

The most promising targets in both scientific research and clinical applications include cytotoxic T-lymphocyte-associated protein 4 (CTLA-4), programmed cell death protein 1 (PD-1), and programmed cell death ligand 1 (PD-L1) **[6-8]**. However, only a small percentage of patients exhibit significant responses to immune checkpoint blockade therapy **[9]**. Therefore, novel strategies, such as using probiotics to activate and regulate Tregs, are needed to improve host responses and outcomes associated with immune checkpoint inhibitor therapy.

Emerging evidence indicates that the efficacy of immune checkpoint blockade therapy is strongly correlated with the recipients' baseline gut microbiota **[10-13]**. Interestingly, dysbiotic gut microbiota has been implicated in ineffective cancer therapies **[14,15]**, whereas "healthy" gut microbiota often leads to beneficial immunotherapy outcomes **[16,17]**.

Consequently, modulating gut microbiota in combination with immune checkpoint inhibition could be a promising and powerful strategy for developing next-generation anti-tumor treatments **[18,19]**. Probiotics have garnered significant attention due to their potent ability to modulate gut microbiota and promote gut or host health. Exogenous probiotics can directly or indirectly produce beneficial metabolites *in vivo* that activate and promote anti-tumor immune responses **[20]**. For instance, enrichment or supplementation with Bifidobacterium species (such as *B. longum*, *B. breve*, and *B. bifidum*) enhanced dendritic cell (DC) antigen presentation, further promoting the infiltration and activation of cytotoxic T lymphocytes (CTLs) within tumors, thereby increasing the efficacy of PD-1/PD-L1 blockade-based immunotherapies **[21,22]**.

Although these studies have demonstrated a role for microbiota in anti-tumor immunity, the underlying mechanisms related to autoimmunity induced by checkpoint-blocking antibodies remain unclear. Optimizing gut microbiota depends on the presence of Tregs **[1]**. The present study conducted a systematic review to identify key clinical studies demonstrating the positive effects of probiotics and nutrients on stimulating regulatory T cells in cancer patients, with the aim of facilitating immunotherapy to control, reduce, or even eliminate cancer cells.

Methods

Study Design

This study followed an international systematic review model, adhering to PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. Available at: http://www.prisma-statement.org/?AspxAutoDetectCookieSupport=1. Accessed on: April 7, 2025. It also followed the AMSTAR-2 (Assessing the Methodological Quality of Systematic Reviews) standards for methodological quality. Available at: https://amstar.ca/. Accessed on: April 7, 2025.

Data Sources and Search Strategy

The literature search process was conducted from April to May 2025 using Scopus, Embase, PubMed, LILACS, EBSCO, SciELO, and Google Scholar, covering scientific articles from various periods up to the present. The following descriptors (MeSH terms) were used: *Probiotics, Nutrients, Regulatory T cells, and Cancer*, employing the Boolean operator "AND" between MeSH terms and "OR" between historical findings.

Study Quality and Risk of Bias

Quality regarding risk of bias, sample size, methodology, clarity of comparisons, precision, and consistency of analyses was classified as high, moderate, low, or very low. Priority was given to systematic reviews or meta-analyses of randomized clinical trials, followed by randomized clinical trials. Evidence quality was rated as low for case reports, editorials, and brief communications, in accordance with the GRADE instrument. Risk of bias was assessed using the Cochrane tool, while funnel plot analysis (sample size versus effect size) and Cohen's d test were used to examine publication bias and effect estimates.

Results and Discussion

Summary of Findings

A total of 141 articles were identified and assessed for eligibility, and 31 were ultimately included in this systematic review. The included studies showed

moderate to high quality (Figure 1), based on study designs such as meta-analyses, consensus statements, and randomized clinical, prospective, and observational studies. According to the GRADE instrument, the results were largely homogeneous, with X^2 = 67.8% (>50%). Based on the Cochrane risk-of-bias tool, 26 studies were rated as having a high risk of bias, and 24 studies did not meet GRADE and AMSTAR-2 criteria.

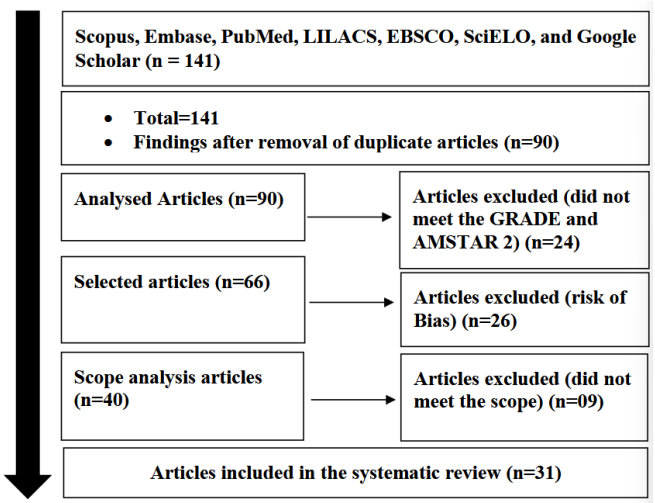

Figure 1. Flowchart showing the article selection process. Source: Own authorship.

Figure 2 presents the results regarding the risk of bias across the studies using a funnel plot, showing the calculated effect size (magnitude of difference) based on Cohen's d. Precision (sample size) was determined indirectly as the inverse of the standard error (1/Standard Error). The plot exhibited a symmetrical pattern, suggesting no significant risk of bias among either studies with small sample sizes (lower precision)—shown at the bottom of the plot—or studies with large sample sizes—shown at the top.

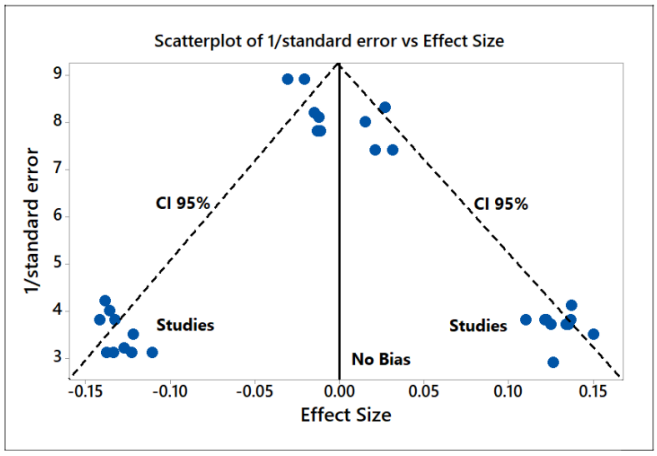

Figure 2. The symmetrical funnel plot suggests no risk of bias among studies with small sample sizes, which are shown at the bottom of the plot. Studies with high confidence and high recommendation levels are shown at the top of the plot (n = 31 studies). Source: Own authorship.

Probiotics and Immunomodulation by Tregs

The specific mechanism by which symbionts stimulate Treg accumulation is not fully understood. It is believed that *Clostridia spp*. may act synergistically to stimulate Treg induction **[23]**. One proposed mechanism of action is the cooperative production of short-chain fatty acids (SCFAs) such as butyrate, propionate, and acetate through the fermentation of dietary fiber by commensals, including Clostridium and Bacteroides spp., in the colon. SCFAs are absorbed via passive diffusion and H+-coupled active transport through MCT1 (SLC16A1) and MCT4 (SLC16A3), two electroneutral monocarboxylate transporters, or via Na+-coupled electrogenic monocarboxylate transporters SMCT1 (SLC5A8) and SMCT2 (SLC5A12) **[23]**.

Oral administration of butyrate, propionate, and acetate, either individually or in combination, led to an increase in the number of colonic Treg cells **[24]**. Butyrate is known to participate in Treg differentiation by facilitating histone H3 acetylation at the promoter region and at CNS1 and CNS3 of the Foxp3 gene **[25]**. Recognition of butyrate by G protein-coupled receptors, such as GPR43 and GPR15 expressed by colonic Treg cells, and GPR109A expressed by DCs and macrophages **[23-25]**, also promotes Treg differentiation. Interestingly, SCFA treatment increased the number of Helios+ Tregs, indicating that SCFAs also promote the expansion of tTregs **[24]**.

The aryl hydrocarbon receptor (AhR) is a nuclear sensor expressed by Tregs that helps them detect and respond to compounds acting as AhR ligands. Intestinal pTregs exhibit higher AhR expression than Tregs in any other tissue. Dietary tryptophan, an essential amino acid, is metabolized by IDO (indoleamine 2,3-dioxygenase) and TDO (tryptophan 2,3-dioxygenase). Kynurenine enhances the generation of Foxp3+ Tregs *in vitro* in the presence of TGFβ1. *Lactobacillus spp*. can also metabolize tryptophan into various AhR ligands **[26]**.

Another dietary metabolite that drives Treg cell development in the intestine is all-trans retinoic acid (atRA), a bioactive form of vitamin A. Dietary vitamin A, specifically retinol, is absorbed by intestinal epithelial cells via passive diffusion. Although epithelial cells are capable of synthesizing atRA from dietary vitamin A, a process regulated by commensal bacteria, the vast majority of studies to date have focused on dendritic cells, macrophages, and eosinophils as sources of atRA for iTreg induction **[27]**.

Furthermore, atRA has been reported to suppress the IL-6-induced conversion of Foxp3+ Tregs into inflammatory Th17 cells, a mechanism likely mediated by the retinoic acid receptor α. While vitamin A plays

the most prominent role in Treg development, other vitamins also affect this lymphocyte population. Dietary vitamin D3 is metabolized into 1,25-dihydroxyvitamin D3, which binds to the vitamin D receptor element (VDRE) within the non-coding CNS region (spanning nucleotides +1714 to +2554) of the human *Foxp3* gene. FR4, the receptor for vitamin B9 (folic acid), is highly expressed by Tregs and is known to promote intestinal Treg survival through the upregulation of the anti-apoptotic factor BCL2 **[27]**.

In this context, mucosal surfaces are distinct sites exposed to environmental, dietary, and microbial antigens. Particularly in the intestine, the host adapts continuously and actively through complex interactions involving the microbiota, dietary compounds, immune cells, and other tissues. Treg cells are essential for fine-tuning the intestinal immune response to both self and non-self antigens. Their importance in intestinal homeostasis is illustrated by the onset of overt inflammation resulting from deficiencies in Treg generation, function, or stability within the gut. A substantial imbalance in Treg populations has been observed in intestinal tissue during pathological conditions, where a tightly regulated and balanced system becomes dysregulated, leading to chronic and unchecked immune responses **[28]**.

Human CD4+ Treg cells are characterized by the expression of the forkhead box P3 (FOXP3, or scurfin) transcription factor, high surface expression of CD25, and low or absent expression of CD127. Tregs express high levels of CD25 (the IL-2 receptor αchain) due to their strong dependence on interleukin-2 (IL-2) for development, maintenance, and peripheral homeostasis **[1]**.

In addition, the systematic analysis of tumor-infiltrating lymphocytes is crucial for the development of immunotherapies and the prediction of clinical responses in cancer. Zheng et al. (2017) **[29]** performed single-cell RNA sequencing on 5,063 individual T cells isolated from peripheral blood, tumor tissue, and adjacent normal tissue from six patients with hepatocellular carcinoma. The transcriptional profiles of these individual cells, combined with assembled T-cell receptor (TCR) sequences, enabled the identification of 11 T-cell subsets based on their molecular and functional properties and the delineation of their developmental trajectories. Specific subsets, such as exhausted CD8+ T cells and Tregs, are preferentially enriched and potentially clonally expanded in hepatocellular carcinoma (HCC), and signature genes have been identified for each subset. One of these genes, Leilina, is upregulated in activated CD8+ T cells and Tregs and represses CD8+ T cell functions *in vitro*. This compendium of transcriptome data has provided valuable insights and a rich resource for understanding the immunological landscape of cancer.

Against this backdrop, probiotics have increasingly been proposed to enhance cancer treatments involving immune checkpoint blockade. However, their causal relationship with immunotherapeutic efficacy remains unclear, prompting Gao et al. (2023) **[30]** to investigate whether and how the probiotic *Lacticaseibacillus rhamnosus* Probio-M9 manipulates the gut microbiota to achieve the desired outcomes. The effects of Probio-M9 on anti-PD-1 treatment for colorectal cancer in mice were evaluated using a multi-omics approach. The results indicated that the Probio-M9 intervention enhanced anti-PD-1-mediated tumor inhibition. Both prophylactic and therapeutic administration of Probio-M9 demonstrated remarkable performance in controlling tumor growth during ICB treatment. Probio-M9 supplementation modulated the enhanced immunotherapeutic response by promoting beneficial microbes (e.g., Lactobacillus and *Bifidobacterium animalis*) and the production of beneficial metabolites, including butyric acids in the gut, and by leading to the accumulation of blood-derived α-ketoglutaric acid, N-acetyl-L-glutamic acid, and pyridoxine; these factors promoted the infiltration and activation of cytotoxic T lymphocytes (CTLs) and suppressed Treg function within the tumor microenvironment.

Finally, Tregs play important roles in the tumor microenvironment, particularly in inducing immune evasion. To identify the underlying mechanism of Treg dysregulation in breast cancer tissues, Moallemi-Rad et al. (2023) **[31]** evaluated the expression of five Treg-related long non-coding RNAs (lncRNAs), namely FLICR (FOXP3 Regulating Long Intergenic Non-Coding RNA), NEST (IFNG-AS1), RMRP (RNA Component of Mitochondrial RNA Processing Endoribonuclease), MAFTRR (MAF Transcriptional Regulatory RNA), and TH2-LCR (Th2 Cytokine Locus Control Region), in paired breast cancer and adjacent non-cancerous tissues. Expression levels of RMRP, TH2-LCR, MAFTRR, and GATA3-AS1 were significantly higher in breast cancer samples compared to non-tumor tissues. Significant positive associations were observed between RMRP gene expression levels in tumor tissues and nuclear grade, tubule formation, and tumor size. Furthermore, there was a significant positive association between MAFTRR gene expression levels in tumor tissues and nuclear grade. Additionally, FLICR expression levels differed among tumors with varying HER2/neu receptor levels. Therefore, Treg-associated lncRNAs may contribute to the pathogenesis of breast cancer.

Conclusion

It was concluded that probiotics, in conjunction with the gut microbiota, have increasingly been proposed to enhance cancer immune checkpoint blockade treatments through the activation of Treg cells. Oral administration of butyrate, propionate, and acetate, either individually or in combination, led to an increase in the number of Treg cells in the colon. Additionally, all-trans retinoic acid (atRA), a bioactive form of vitamin A, also stimulates Treg cells in the human intestine.

CrediT

Author contributions: Conceptualization-; Data curation-; Formal Analysis-; Investigation; Methodology; Project administration; Supervision; Writing - original draft; Writing-review & editing- All authors.

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Informed Consent

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No additional data are available.

Conflict of Interest

The authors declare no conflict of interest.

Similarity Check

It was applied by Ithenticate®.

Application of Artificial Intelligence (AI)

Not applicable.

Peer Review Process

It was performed.

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