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The Role of Prebiotics in the Prevention and Management of Breast Cancer: A Comprehensive Review of Preclinical and Clinical Evidence

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A B S T R A C T

Objective: Breast cancer is among the most commonly diagnosed malignancies in women worldwide. Increasing evidence suggests that the gut microbiota and nutrition-related interventions, including prebiotics, may influence biological pathways involved in cancer development and progression. This review aimed to synthesize the available preclinical and clinical evidence concerning the potential role of prebiotics in the prevention and management of breast cancer.

Methods: This narrative review synthesized evidence from preclinical studies, including cellular and animal models, as well as clinical and observational studies. Relevant literature published between 2010 and 2025 was identified through major scientific databases, including Google Scholar, Web of Science, PubMed, Scopus, the Scientific Information Database (SID), and the Islamic World Science Citation Center (ISC). The review considered randomized controlled trials, case–control studies, animal experiments, and systematic reviews investigating prebiotics and related microbiota-targeted interventions in the context of breast cancer.

Results: Preclinical and emerging clinical evidence indicates that prebiotics may influence the gut–estrogen axis through alterations in gut microbial composition and metabolic activity, including changes in the production of short-chain fatty acids (SCFAs). These microbiota-mediated effects may influence inflammatory and immune responses, metabolic homeostasis, and signaling pathways implicated in tumor development and progression. Some experimental studies have also reported effects on cancer-cell proliferation and mechanisms potentially associated with endocrine therapy resistance. In humans, prebiotic or microbiota-targeted interventions have been associated with improvements in selected metabolic parameters and changes in certain inflammatory biomarkers. However, the clinical evidence remains limited and heterogeneous, and the extent to which these biological effects translate into meaningful improvements in breast cancer outcomes remains uncertain.

Conclusion: Current evidence suggests that prebiotics may modulate the gut microbiota and metabolic, inflammatory, and hormonal pathways relevant to breast cancer. However, the available evidence remains insufficient to establish their preventive or therapeutic efficacy. Larger, well-designed, standardized clinical trials are needed to determine their clinical benefits and to establish optimal prebiotic formulations, doses, and treatment durations.

Keywords: Prebiotics, Breast cancer, Gut microbiota, Gut–estrogen axis, Short-chain fatty acids, Prebiotic interventions

➢ **How to cite this paper**

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Introduction

Breast cancer remains a major global public health concern and is the most commonly diagnosed malignancy among women worldwide [1]. Its clinical presentation may include a palpable breast or axillary mass, changes in breast size or shape, skin alterations such as dimpling or peau d'orange, abnormal nipple discharge, and, in some cases, nipple retraction or inversion. Enlargement or involvement of the axillary lymph nodes may also occur [2,3]. The etiology of breast cancer is multifactorial and reflects complex interactions among genetic, hormonal, environmental, and lifestyle-related factors. Genetic susceptibility, including pathogenic variants in *BRCA1* and *BRCA2*, as well as prolonged exposure to estrogen, obesity, and certain dietary and lifestyle patterns, may contribute to breast cancer development. These factors can influence cellular homeostasis, genomic stability, and signaling pathways involved in malignant transformation [4]. At the molecular level, breast cancer develops through the accumulation of genetic and epigenetic alterations that disrupt pathways regulating cell proliferation, differentiation, survival, and apoptosis. Progressive changes within the tumor microenvironment, including altered angiogenesis, immune responses, and hormonal signaling, may subsequently facilitate tumor growth, local invasion, and metastatic dissemination [5].

Despite substantial advances in early detection and the development of surgical, chemotherapeutic, endocrine, and targeted therapies, breast cancer remains a major cause of cancer-related morbidity and mortality among women [6]. Consequently, increasing attention has been given to preventive strategies and adjunctive approaches, particularly dietary and lifestyle interventions, as complementary components of comprehensive cancer prevention and management [7].

Within this context, the gut–tumor axis has emerged as an important area of cancer research, providing new perspectives on the potential relationship between the intestinal microbiota and the development and progression of malignancies, including breast cancer [8]. The gut microbiota participates in diverse physiological processes and may influence tumor-related pathways through modulation of immune responses, hormone metabolism, production of bioactive metabolites, and regulation of systemic inflammatory activity [9]. In particular, alterations in microbial composition and function, commonly referred to as dysbiosis, have been associated with changes in metabolic and inflammatory signaling that may contribute to conditions relevant to carcinogenesis [8,9].

Against this background, microbiota-targeted interventions, including probiotics, prebiotics, and synbiotics, have attracted increasing research interest because of their potential to influence intestinal microbial composition and host–microbiota interactions [10–12]. Probiotics are live microorganisms that, when administered in adequate amounts, confer a health benefit on the host and may influence intestinal microbial ecology, barrier function, and immune responses [13]. Prebiotics are substrates that are selectively utilized by host microorganisms and confer a health benefit; commonly studied examples include inulin-type fructans and fructooligosaccharides, which can be metabolized by intestinal microorganisms and contribute to the production of metabolites such as short-chain fatty acids (SCFAs) [14]. Synbiotics combine live microorganisms with substrates selectively utilized by host microorganisms and are designed to provide complementary or synergistic effects on the host–microbiota system [15].

The interaction between the gut microbiota and the host hormonal and immune systems may be particularly relevant to hormone-responsive malignancies such as breast cancer [16]. Alterations in microbial composition and metabolic activity may

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affect estrogen metabolism and enterohepatic estrogen circulation, systemic inflammatory responses, and immune functions involved in tumor surveillance. In this context, prebiotics have been proposed as one potential means of modifying the intestinal microbial environment and its metabolic output. Through these effects, prebiotics may influence inflammatory, metabolic, and hormonal pathways that are potentially relevant to breast cancer biology [17]. However, the extent to which these biological effects translate into clinically meaningful outcomes remains uncertain.

Given the growing body of preclinical and clinical research examining the relationship between prebiotics, the gut microbiota, and breast cancer, a critical synthesis of the available evidence is warranted. This review therefore aims to evaluate the current evidence regarding the potential role of prebiotics in breast cancer prevention and management, summarize the proposed biological mechanisms underlying their effects, distinguish findings derived from preclinical studies from those supported by human evidence, and identify key limitations and knowledge gaps that should be addressed in future research.

Methods

This study was conducted as a narrative review to examine and critically synthesize the available evidence on the potential role of prebiotics in the prevention and management of breast cancer. The review considered evidence from *in vitro* studies and animal models alongside clinical and observational research, with particular attention to the proposed interactions among prebiotics, the gut microbiota, and breast cancer–related biological processes.

Relevant literature published between 2010 and 2025 was identified through searches of PubMed, Web of Science, Scopus, Google Scholar, the Islamic World Science Citation Center (ISC), and the Scientific Information Database (SID). The search strategy combined terms related to prebiotics and breast cancer, supplemented by additional keywords addressing the gut microbiota, inflammation, estrogen metabolism, short-chain fatty acids, and related biological mechanisms. The reference lists of relevant articles and previously published reviews were also examined manually to identify additional studies that might not have been retrieved through the database searches.

Studies were considered for inclusion based on their relevance to the primary objectives of the review and the type and quality of evidence they provided. Evidence from cellular and molecular experiments, animal studies, clinical trials, observational studies, and systematic reviews was considered and interpreted according to the nature of the underlying evidence. The narrative synthesis focused on the type of prebiotic or microbiota-targeted intervention, study model or population, proposed mechanisms of action, breast cancer–related outcomes, and the consistency of findings across different experimental and clinical settings.

Because this was a narrative review, no formal meta-analysis or statistical pooling of results was performed. Instead, the available evidence was synthesized descriptively and critically, with explicit distinction between findings derived from preclinical models and those supported by human studies. Results from cellular and animal experiments were not considered direct evidence of clinical efficacy in humans. Particular attention was given to sources of heterogeneity, including differences in prebiotic type, formulation, dose, and duration of administration; experimental models and participant characteristics; outcome measures; and the relatively limited number of controlled human studies. These factors were taken into account when assessing the consistency, strength, and potential clinical relevance of the available evidence.

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Results

The available evidence suggests that prebiotics and related microbiota-targeted interventions may influence biological processes relevant to breast cancer, including intestinal barrier integrity, microbial composition and activity, inflammatory responses, and immune regulation (Table 1). In preclinical studies, commonly reported effects included improvements in intestinal barrier function, attenuation of inflammatory and oxidative responses, and modulation of host immune activity (Table 1).

Several preclinical studies have also reported potential antitumor and antimetastatic effects of prebiotic- or synbiotic-based interventions, including in experimental models of triple-negative breast cancer (Table 1). However, these findings are primarily derived from cellular and animal models and therefore cannot be directly interpreted as evidence of clinical anticancer efficacy. Human studies have reported changes in selected metabolic and inflammatory parameters, gastrointestinal symptoms, and quality-of-life measures following probiotic-, prebiotic-, or combined microbiota-targeted interventions (Table 1). Nevertheless, epidemiological evidence has not consistently demonstrated a significant association between the consumption of specific prebiotic-rich dietary fibers and a lower risk of breast cancer.

The magnitude and direction of the reported effects varied according to the type, composition, and dose of the intervention, duration of exposure, baseline characteristics of the gut microbiota, study population, and breast cancer subtype (Table 1). Overall, the available findings indicate that prebiotics may modulate biological pathways relevant to breast cancer; however, the current evidence is insufficient to establish their clinical efficacy for breast cancer prevention or treatment or to define optimal formulations, doses, and treatment durations. Further well-designed and adequately powered clinical trials are required to determine whether the effects observed in preclinical and early clinical studies translate into meaningful improvements in breast cancer-related outcomes. A detailed summary of the available evidence is provided in Table 1

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Table 1: Preclinical and clinical evidence on prebiotics and related gut microbiota-targeted interventions in breast cancer and other relevant cancer settings

Study type and model	Sample and intervention	Main findings	Overall interpretation	Reference
Animal model (DMBA-induced rat model)	Fucoidan, 200–400 mg/kg	Improved intestinal barrier integrity, increased expression of tight-junction proteins, and favorable changes in gut microbial diversity	Suggests potential effects on intestinal barrier function and gut microbial composition; direct relevance to breast cancer prevention remains uncertain	Xue et al., 2018 [18].
Review of evidence across multiple cancer types	Probiotics and prebiotics, administered individually or in combination	Reported effects included improved mucosal barrier function and immune responses, attenuation of treatment-related adverse effects, and improvements in selected metabolic parameters	Suggests potential supportive roles for microbiota-targeted interventions in cancer care, although evidence varies substantially across cancer types and interventions	Jampílek et al., 2022 [19].
Translational review and animal model	Exercise combined with prebiotic fiber in a mouse model	Favorable changes in gut microbial composition and potential effects on physiological and treatment-related outcomes	Indicates that prebiotic fiber combined with lifestyle interventions may influence gut microbiota and host responses; breast cancer-specific clinical relevance remains to be established	Sampsell et al., 2022 [20].
Systematic review and meta-analysis of RCTs	Probiotics (*Lactobacillus*, *Bifidobacterium*) with or without FOS;	Reported reductions in selected obesity-related parameters, dyslipidemia, and TNF-α, together with improvements in quality of life and lymphedema-related outcomes;	Indicates potential benefits of microbiota-targeted interventions for selected metabolic and inflammatory outcomes in cancer populations	Thu et al., 2023 [21].

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	approximately 10^9 CFU for 10 weeks	some effects were greater with probiotic–prebiotic combinations		
Case–control study (Italy)	Dietary intake of fiber sources related to prebiotic substrates, including inulin, FOS, and GOS	Most examined fiber types were not significantly associated with breast cancer risk; associations with ovarian cancer risk were reported for some fiber types	Provides limited evidence for a consistent association between specific prebiotic-related fibers and breast cancer risk	Turati et al., 2024 [22].
Preclinical and human evidence in HPV infection and cervical cancer	Probiotics, prebiotics, and synbiotics	Reported reductions in selected HPV-related outcomes, inflammatory and oxidative responses, and cellular proliferation	Provides indirect evidence that microbiota-targeted interventions may influence mechanisms relevant to cancer; applicability to breast cancer remains uncertain	Gutiérrez Salmeán et al., 2024 [23].
Preclinical and clinical studies	Prebiotics and synbiotics at different doses and intervention durations	Reported modulation of the gut–estrogen axis, inflammatory responses, immune and metabolic parameters, and selected tumor-related pathways	Supports a potential biological link between microbiota-targeted interventions and pathways relevant to breast cancer, but clinical efficacy remains insufficiently established	Sabit et al., 2025 [24].
Mouse model of triple-negative breast cancer (4T1)	BMPE and the prebiotic LevAE in BALB/c mice	Antitumor and antimetastatic effects accompanied by changes in immune responses and oxidative stress	Demonstrates potential anticancer activity in a preclinical model of triple-negative breast cancer; clinical relevance requires validation in human studies	Shawky et al., 2025 [25].
Scoping review of RCTs	33 studies involving patients with cancer	Reported improvements in gastrointestinal symptoms, quality of life, anxiety, and	Indicates potentially beneficial effects on selected supportive-care outcomes, but heterogeneity limits	Belloni et al., 2025 [26].

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		depression, although findings were heterogeneous	firm conclusions regarding clinical efficacy	

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Discussion

The available evidence suggests that the gut microbiota and microbiota-targeted nutritional interventions may influence several biological processes relevant to breast cancer. Preclinical studies indicate that prebiotics and related compounds may improve intestinal barrier integrity, enhance tight-junction protein expression, reduce intestinal permeability, and modify the composition and metabolic activity of the gut microbiota. These changes may, in turn, influence inflammatory and metabolic pathways associated with cancer biology. In an animal model of breast cancer, fucoidan was associated with improved intestinal barrier function, increased microbial diversity, and favorable changes in gut microbial composition, supporting the potential relevance of the gut–microbiota axis to breast cancer biology [18]. However, because fucoidan is not a conventional prebiotic substrate, these findings should be interpreted as evidence from a related microbiota-targeted intervention rather than direct evidence for prebiotic efficacy.

The potential biological effects of prebiotics are unlikely to depend solely on changes in microbial composition. Fermentation of prebiotic substrates can generate metabolites, particularly short-chain fatty acids (SCFAs), that may influence inflammatory signaling, immune responses, epithelial barrier function, and host metabolic processes. The gut–estrogen axis has also emerged as a potentially relevant pathway in breast cancer. Changes in microbial composition and metabolic activity may influence estrogen transformation and enterohepatic circulation, thereby potentially affecting systemic estrogen exposure and hormone-responsive tumor biology [24]. Nevertheless, much of the mechanistic evidence supporting these associations remains derived from experimental models, and the extent to which these mechanisms operate in patients with breast cancer remains incompletely established.

Human studies of probiotic and probiotic–prebiotic interventions have reported improvements in selected metabolic and inflammatory parameters, quality of life, and some treatment-related outcomes among patients with breast cancer. In some studies, combined probiotic–prebiotic interventions produced more favorable effects than probiotic administration alone [27]. However, these findings have primarily involved supportive, metabolic, or treatment-related outcomes and should not be interpreted as evidence of direct antitumor activity or reduced breast cancer recurrence in humans. Moreover, the contribution of the prebiotic component cannot always be separated from the effects of the probiotic component in combination interventions.

Epidemiological evidence also does not demonstrate a consistent or generalized protective association between dietary intake of prebiotic-related fibers and breast cancer risk. In a large case–control study, most examined fiber types, including those related to commonly studied prebiotic substrates, were not significantly associated with breast cancer risk [22]. These findings suggest that any potential association may depend on the specific fiber source, amount and duration of consumption, overall dietary pattern, individual metabolic characteristics, and the baseline composition and functional activity of the gut microbiota. Accordingly, dietary fiber intake should not currently be considered equivalent to a standardized prebiotic intervention.

In experimental models of triple-negative breast cancer (TNBC), certain microbiota-targeted compounds, including bacterial levan, have demonstrated antitumor and antimetastatic effects accompanied by changes in immune responses and oxidative–reductive status [25]. Although these findings provide mechanistic evidence supporting further investigation of microbiota-targeted strategies in TNBC, they remain restricted to preclinical models and cannot be directly extrapolated to therapeutic efficacy in patients. Similarly, reviews of clinical trials suggest that microbiota-targeted interventions may improve selected supportive-care outcomes, including gastrointestinal symptoms, mucositis, fatigue, and quality of life, in some cancer populations. However, substantial heterogeneity in interventions, cancer types, study populations, and outcome measures limits the certainty and generalizability of these findings [27].

Taken together, the available evidence provides a biologically plausible rationale for investigating prebiotics and other gut microbiota-targeted nutritional strategies in breast cancer. Potentially relevant pathways include modulation of microbial composition and function, production of metabolites such as SCFAs, maintenance of intestinal barrier integrity, regulation of inflammatory and immune responses, and alterations in host metabolic processes. Microbiota-mediated effects on estrogen metabolism and enterohepatic estrogen circulation may represent an additional pathway of interest, particularly in hormone-responsive breast cancer. Nevertheless, the current

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evidence remains heterogeneous, and direct evidence demonstrating clinically meaningful antitumor effects of prebiotics in humans is limited [28–30]. Future research should therefore prioritize well-designed clinical trials that evaluate clearly defined prebiotic interventions, standardized doses and treatment durations, microbiota-related biomarkers, and clinically relevant breast cancer outcomes.

Conclusion

Overall, current evidence suggests that prebiotics and related gut microbiota-targeted interventions may influence biological processes relevant to breast cancer through interconnected effects on intestinal barrier integrity, microbial composition and function, inflammatory and immune responses, host metabolism, and potentially the gut–estrogen axis. However, the available evidence currently supports a potential adjunctive and supportive role rather than an established preventive or therapeutic effect of prebiotics in breast cancer. The predominance of preclinical evidence, heterogeneity in intervention characteristics and study populations, and the limited number of well-controlled human studies preclude definitive conclusions regarding their clinical efficacy. Larger, well-designed, standardized, and adequately powered clinical trials with long-term follow-up are needed to determine whether specific prebiotic interventions can meaningfully influence breast cancer risk, treatment outcomes, recurrence, and survival.

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Competing interests

The authors have no competing interests to declare that are relevant to the content of this article.

Ethics approval

This study was performed in line with the principles of the Declaration of Helsinki.

Consent to participate

Informed consent was obtained from all individual participants included in the study.

Author contributions

SK and MA: Conceptualization, the original draft writing, investigation, writing including reviewing and editing and investigation and formal analysis; SK: Conceptualization, supervision, and project administration; SK and MA Conceptualization, the original draft writing, investigation, writing including reviewing and editing.

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