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REVIEW ARTICLE

Galectin-3 and galectin-1 interactions in breast cancer therapy

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ABSTRACT

Galectins, a family of β-galactoside-binding proteins, play critical roles in tumor progression, angiogenesis, and immune evasion, making them significant therapeutic targets in cancer treatment. By binding β-galactoside-containing glycoconjugates, galectins modulate immune responses, apoptosis, and tumor development. The increasing recognition of their oncogenic roles has led to the development of carbohydrate- and peptide-based inhibitors that competitively bind to the carbohydrate recognition domain (CRD), disrupting galectin-mediated immune evasion, T-cell apoptosis, and angiogenesis. Given their intricate functions in the tumor microenvironment, a comprehensive evaluation of galectin inhibitors is warranted. This review synthesizes recent advancements in galectin-targeted therapies, including their mechanisms of action, efficacy in preclinical models, and potential synergy with chemotherapeutic agents and monoclonal antibodies. Despite promising developments, challenges remain in optimizing treatment regimens, overcoming resistance mechanisms, and identifying predictive biomarkers for patient stratification. Patient stratification, based on molecular or genetic profiles, is essential for enhancing therapeutic efficacy and ensuring personalized treatment approaches. A systematic literature search (2014–2024) was conducted using Google Scholar, ProQuest, Science Direct, and Scopus databases, with key terms including galectin inhibitors, cancer therapy, tumor microenvironment, immune evasion, and targeted therapy. This review highlights the role of galectin-1 and galectin-3 in breast cancer therapy, emphasizing their impact on tumor progression, immune modulation, and resistance to conventional treatments. Further translational research is necessary to refine clinical applications, optimize combination strategies, and establish biomarkers that enhance the integration of galectin inhibitors into existing treatment paradigms.

Keywords: Galectins, cancer therapy, galectin inhibitors, tumor microenvironment, immune evasion, personalized treatment

INTRODUCTION

Cancers of the stomach, colon, prostate, breasts, and lungs are the most common malignancies worldwide.[1] Breast cancer is the most common cause of cancer-related mortality, whereas tracheal, lung, and bronchial malignancies make up the bulk of cancer-related deaths.[2] The incidence of colorectal cancer fatalities has more than doubled in the last 30 years.[3] Lifestyle factors such as diet, lack of physical activity, and obesity contribute to an increase in colorectal cancer diagnoses, but the overall survival rate remains low.[4] Public health initiatives and early detection can reduce colorectal cancer prevalence, while genetic abnormalities such as breast cancer 1, early onset (BRCA1) and breast cancer 2, early onset (BRCA2) increase breast cancer risk, accounting for 25% of cases.[5] Similarly, CDH1 mutations affect cell adhesion, contributing to cancer spread and metastasis.[6] Genetic vulnerabilities and family history can significantly influence the likelihood and aggressiveness of breast cancer, emphasizing the need for personalized screening and prevention strategies due to these genetic and familial factors.[7] Healthcare providers recommend early breast cancer screenings and genetic counseling for individuals with a family history of tumors or with known gene mutations, enabling informed decision-making about preventive measures.[8]

Breast cancer risk is significantly influenced by DNA repair gene mutations, such as CHEK2, BRIP1, and ATM, which interact with BRCA1 and BRCA2 pathways, leading to impaired DNA repair mechanisms, increased susceptibility to cancerous changes, genomic instability, tumor progression, and therapeutic resistance.[9] Genetic testing and personalized medicine have advanced to understand genetic risks, with galectin-3, a beta-galactoside-binding protein, playing a significant role in breast cancer progression and metastasis.[10] Galectin-3, a key player in cell adhesion, migration, and immune response, is linked to increased metastatic potential in breast cancer, making it a promising biomarker for advanced disease.[11] The role of galectin-3 in the cellular stress response and apoptosis resistance complicates treatment outcomes, highlighting the need for personalized intervention strategies in breast cancer management. This review aimed to explore and summarize the recent scientific findings on the interactions of galectin-3 and galectin-1 with key signaling pathways, tumor microenvironment components, and their crosstalk with immune cells, epithelial-mesenchymal transition (EMT), and extracellular matrix remodeling in breast cancer. Galectin-3, a key cancer cell survival factor, regulates autophagy, antioxidant defenses, and hypoxia-inducible factor-1α (HIF-1α) stability under metabolic stress. It interacts with Beclin-1, preventing apoptosis and enhancing autophagic flux. Under hypoxic conditions, galectin-3 enhances HIF-1α stability, facilitating angiogenesis and metabolic adaptation. High Gal-3 expression correlates with poor prognosis and resistance to therapy, making it a potential biomarker for personalized treatment. This review summarizes 11 years of research on galectin-3's effects on tumor progression, metastasis, chemoresistance, immune evasion, and therapeutic targeting in breast cancer, that was retrieved from various sources, including Science Direct, PubMed, Nature, and Biomedicine.

Research methods

A systematic search was conducted on electronic databases such as PubMed, Science Direct, and Google Scholar to find English publications on galectin-3 and galectin-1 interactions in breast cancer therapy. Fifty publications were selected from 230, excluding duplicates and non-compliant articles (Figure 1). The selected articles were analyzed and reviewed (Table 1).

A member of the galectin family that has been extensively examined is galectin-3, which was initially discovered as a 32 kDa antigen on the surfaces of murine macrophages,[12] has a key immune cell function and has gained significant research due to its unique structure and functional dynamics. Its N-terminal part has 14 amino acid repetitions, while its C-terminal part has a single carbohydrate recognition domain (CRD). This suggests a specialized cell signaling and molecular interaction function.[13] Galectin-3's oligomerization upon ligand binding enhances its interactions with other cellular components, increasing its role in cellular communication and allowing it to engage in various pathways.

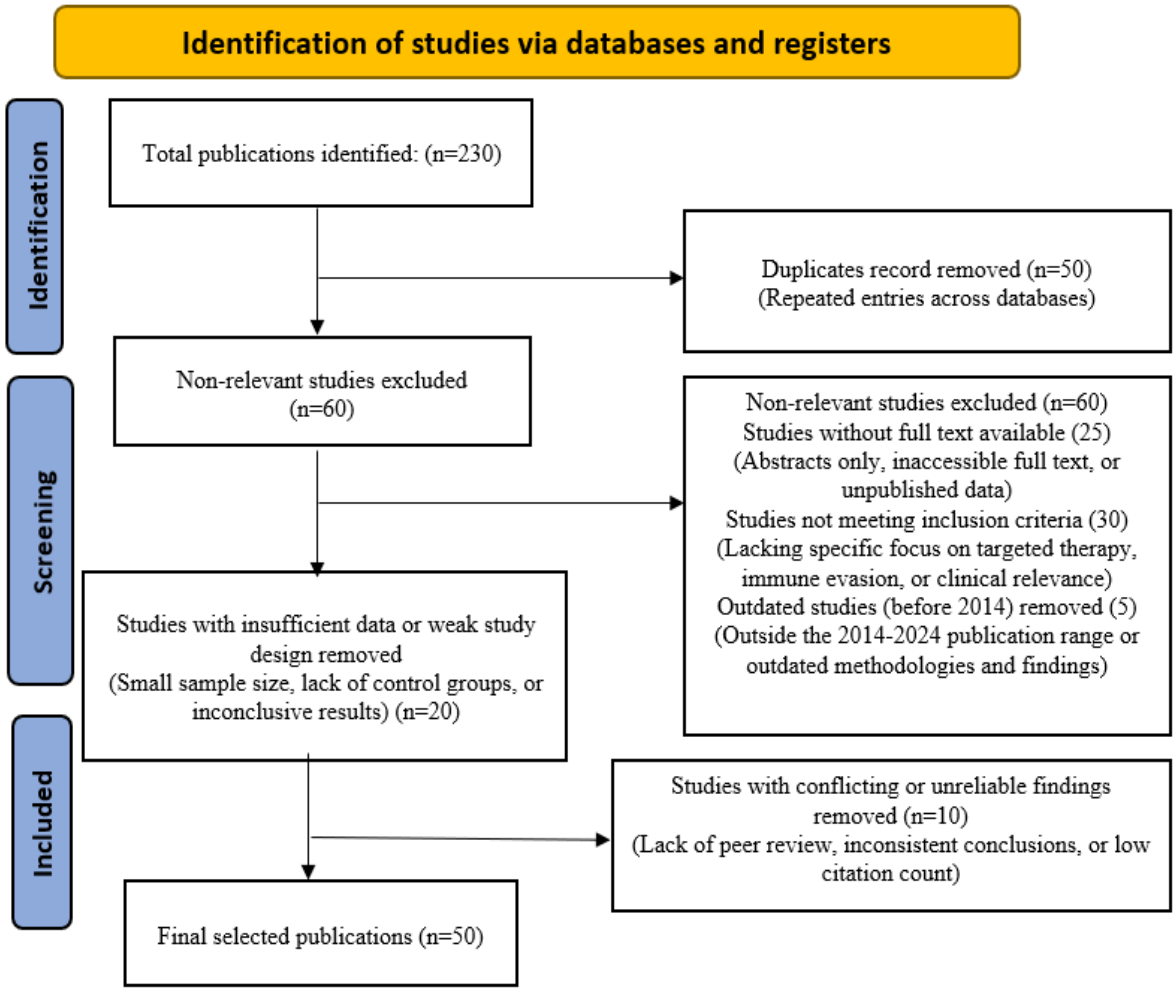

Figure 1. Flow chart of selection of publications

Table 1. Studies on galectin-3 and its role in breast cancer progression, diagnosis, and therapeutics

Reference	**Key focus area**	**Study type/methodology**	**Key findings**	**Potential application in review**
Cao et al. [1]	Global cancer burden trends	Secondary analysis	Rising cancer incidence	Background on cancer burden
Bashar & Begam [2]	Breast cancer prevalence	Review	Breast cancer surpassing lung cancer	Cancer epidemiology discussion
Morgan et al.[3]	Cancer incidence	GLOBOCAN data	Projected increase in cases	Cancer trends & implications
Adebayo et al.[4]	Cancer treatments	Review	Future therapeutic outlook	Galectin-3 in colorectal cancer
Nathan et al. [5]	TP53 mutations in cancer	Molecular study	Mutational impact on tumor growth	Genetic factors linked to galectin-3
Aitchison et al.[6]	CDH1 mutations & cancer	Case study	Genetic predisposition	Role of mutations in cancer progression
Parsons [7]	PTEN tumor suppressor function	Review	PTEN-PI3K-AKT pathway	Connection to galectin-3 function

Rashidi et al.[8]	Breast disease risk factors	Comparative study	Risk factors for malignancy	Breast cancer context
Singh et al.[9]	Breast cancer awareness	Survey	Gaps in awareness	Public health relevance
Boutas et al.[10]	Galectin-3 in breast cancer	Systematic review	Association with metastasis	Core discussion on galectin-3 in cancer
Hara et al.[11]	Galectin-3 as a biomarker	Review	Early disease detection	Diagnostic potential of galectin-3
Di Gregoli et al.[12]	Galectin-3 & macrophages	Experimental study	Role in atherosclerosis	Immune response in cancer
Ahmed et al.[13]	Galectin-3 antagonists	Pharmaceutical review	Therapeutic development	Potential treatment targeting galectin-3
Wang et al. [14]	Galectin-3 & HIV	Cellular study	Membrane lipid raft alterations	Mechanistic insights on galectin-3 binding
Capone et al. [15]	Galectin-3 binding protein	Review	Impact on tumor progression	Galectin-3's role in metastasis
Miller et al.[16]	Targeting galectin-3	Drug study	Allosteric modulation	Drug discovery implications
Ernur, 2024 [17]	Serum galectin-3 in breast cancer	Clinical study	Pre-/post-op level variations	Biomarker potential
Guo et al., 2020 [18]	Galectin-3 & tumor microenvironment	Experimental study	Impact on metabolism	Tumor growth and progression
Suthahar.[19]	Galectin-3 in cardiovascular disease	Scholarly Research study	Activation/inhibition roles	Broader implications of galectin-3
Setayesh et al.[20]	Galectin-3 in liver cancer	Experimental study	Overexpression in HCC	Role in hepatocellular carcinoma
Pang. [21]	Galectin-3-ligand interaction and cancer cell behavior	Scholarly Research Study	Explores ligand binding and cancer progression	Supports 'Ligand Affinity of galectin-3'
Sasaki et al.[22]	Galectin-2 interactions with pathogens	Experimental study	Identifies galectin-mediated bacterial aggregation	Provides insight into galectin-3's structural makeup
Wu et al.[23]	Full-length galectin-3 in microbial interactions	Experimental study	Confirms necessity of full-length galectin-3 for binding	Helps explain ligand affinity and tumor progression
Goud et al.[24]	Role of galectins in multiple cancers	Review	Galectin-1 as a molecular target in oncology	Supports 'Galectin-3 Throughout Cancer'
Kim et al.[25]	Non-classical role of galectin-3	Experimental study	Nuclear translocation of galectin-3 independent of carbohydrate binding	Supports 'The Structural Makeup of galectin-3'
Li et al.[26]	Diagnostic value of galectin-3 in thyroid carcinoma	Clinical study	Combination with lncRNA HOTAIR improves diagnosis	Supports potential clinical applications
Pokharel et al.[27]	Galectin-3 in lung cancer	Clinical study	Higher expression in squamous cell carcinoma	Supports 'Growth of Tumors due to galectin-3'
Chang et al. [28]	Galectin-3 and epithelial-mesenchymal transition	Translational research	Galectin-3 regulates EMT via AMPK/TGF-β signaling	Explains galectin-3 in tumor progression

Srejovic et al.[29]	Galectin-3 in immune responses	Immunology study	Modulates T-cell-mediated inflammation	Links galectin-3 to cancer-related immune responses
Mayo et al. [30]	Structural interactions in galectin-3	Biochemical study	Phosphorylation-dependent interaction with N-terminal tail	Supports 'The Structural Makeup of Galectin-3'
Dimitrijevic et al.[31]	Galectin-3 in pancreatic adenocarcinoma	Review	Highlights complex galectin-3 interactions in pancreatic cancer	Supports 'Galectin-3 Throughout Cancer'
Setayesh et al.[32]	Galectin-3 in liver cancer	Experimental study	Overexpression in hepatocellular carcinoma	Supports 'Growth of Tumors due to Galectin-3'
Wan et al.[33]	Galectin-3 in tumor microenvironment	Experimental study	Regulates tumor cell and macrophage interactions	Supports 'Galectin-3 Throughout Cancer'
Thijssen.[34]	Role of Galectins in angiogenesis	Review	Regulates endothelial functions in tumor growth	Supports tumor development mechanisms
Hoffmann et al.[35]	Galectin-3 binding to TF-antigen	Glycoconjugate study	Examines glycan interactions with galectin-3	Supports 'Ligand Affinity of Galectin-3'
Grazier et al.[36]	Galectins in breast cancer metastasis	Review	Role of galectin-3 in cancer spread	Supports 'Growth of Tumors due to Galectin-3'
Abourehab et al.[37]	Chondroitin sulfate-based biomaterials	Materials chemistry	Explores biomedical applications of glycan interactions	Provides insights into galectin-3-based therapeutics
Moure et al.[38]	Protein binding to galectin-3	NMR spectroscopy study	Reveals selective binding epitopes	Supports 'Ligand Affinity of Galectin-3'
Vicente et al.[39]	Glycans and immune system reprogramming	Immunology study	Impacts T-cell development and diversity	Links galectin-3 to immune system interactions
Sendid et al.[40]	Mannan immune response and Galectins	Immunology study	Identifies protective glycan epitopes	Supports Galectin-3 in immune modulation
Ghanbari-Movahed et al.[41]	Camptothecin nano-formulations in cancer treatment	Systematic Review	Improved efficacy of camptothecin through nano-drug delivery	Provides insights into targeted drug delivery mechanisms
Ballari et al. [42]	Lipophilic Salirasib analogs and tumor proliferation	Experimental Study	Enhanced antiproliferative activity of Salirasib analogs	Potential role of galectin-3 in modulating tumor suppression
Pang et al.[43]	Transcriptomic analysis of tocotrienols in chondrosarcoma cells	Experimental Study	Identifies molecular changes in chondrosarcoma treated with tocotrienols	Explains galectin-3 involvement in tumor response to treatment
Aldabaan .[44]	EMT reversal in TNBC by γ-Tocotrienol	Scholarly Research Study	γ-Tocotrienol inhibits EMT and androgen receptor expression	Supports role of galectin-3 in metastasis
Funkhouser et al.[45]	Genetic mutations and galectin-3 levels in breast cancer	Correlation Analysis	Links genetic mutations to galectin-3 expression in breast cancer	Demonstrates galectin-3's structural influence on cancer growth

Iwamoto et al.[46]	Galectin-3 phosphorylation in metastasis	Experimental Study	Galectin-3 modulates E-cadherin downregulation and tumor metastasis	Supports galectin-3's role in EMT and metastasis
Chen et al.[47]	Tim-3/Galectin-9 in T-cell function and cervical carcinoma	Immunology Study	Galectin-3 interactions affect immune evasion in cancer	Highlights galectin-3's role in tumor immune modulation
Vrbata et al.[48]	Glycopolymers as inhibitors of galectin-3	Medicinal Chemistry Study	Development of galectin-3 inhibitors	Highlights ligand-binding potential of galectin-3
Mechahougui et al.[49]	Advances in personalized oncology	Review	Discusses biomarker-based cancer treatments	Supports role of galectin-3 in precision medicine
Markalunas et al.[50]	Genetic mutations and galectin-3 in breast cancer	Correlation Analysis	Reinforces galectin-3's structural and functional role in oncogenesis	Supports structural and ligand-binding properties

Galectin-3, a soluble S-type lectin, is primarily present in the cytoplasm and released extracellularly through non-classical mechanisms. It bypasses the conventional secretion pathway, allowing it to quickly respond to cellular signals and influence cell behavior at multiple levels. Being a surface localized protein, galectin-3 is essential for cell-environment interactions and regulation of processes such as inflammation, apoptosis, cell division, and immunological responses. Its involvement in these processes may be linked to cancer progression and immune modulation, highlighting its versatility in both intracellular and extracellular communication. This multifunctional protein influences membrane dynamics, cell signaling, and tissue remodeling, thereby impacting health and disease. It plays a significant role in tumor development and metastasis, particularly in cancer biology. Galectin-3's binding properties make it a potential target for therapeutic interventions. Its structural flexibility allows it to adapt to various molecular partners, amplifying its regulatory impact. Understanding galectin-3's roles may provide insights into new therapeutic strategies for managing cancer, inflammation, and immune disorders.[14,15]

Galectin-3 is the only chimera-type galectin with a C-terminal CRD and flexible N-terminal domain for oligomerization. Galectins can dimerize and interact with glycopeptides at each CRD,[16] identifying carbohydrates, particularly N-acetyllactosamine residues, influenced by glycosylation in glycoproteins and glycans.

Ligand affinity of galectin-3

Galectin-3's interactions with cellular glycans, including matrix and cell surface glycoproteins, suggest it may play a crucial role in cellular communication and signaling pathways, opening new therapeutic research avenues.[17,18]

Galectin-3 interacts with growth factor receptors and integrins, regulating tumor cell adhesion, migration, and invasion, highlighting its role in cancer metastasis and enhancing or reducing integrin activity.[19,20] Galectin-3's interaction with CD98 on the cell membrane initiates integrin-mediated cell attachment, influencing cancer progression and therapeutic approaches. It also interacts with EGFR in lung cancer CL1-5 cells, increasing EGFR homodimers.[21] Galectin-3, a bacterial glycan binder, plays a crucial role in promoting hematogenous metastasis by stabilizing tumor cells within the vascular system. Targeting galectin-3 can prevent cancer cell spread to new sites.[22] Galectin-3's functional versatility and role in immune responses against microbial infections, including interactions with diverse glycans, may relate to inflammatory conditions and identify carbohydrates in *Neisseria meningitidis* lipopolysaccharides.[23] The mechanism of galectin-3 interaction with microbial glycans remains unclear, but is crucial for understanding host-pathogen interactions and immune modulation, as well as for developing targeted antimicrobial therapies.[24] Galectin-3 fusion proteins have potential for cancer treatment, offering personalized treatments through medication delivery and imaging in theranostics, highlighting their potential in precision medicine.

Growth of breast cancer due to galectin-3

Overexpression of galectin-3, often found in malignancies such as breast cancer, may serve as a biomarker for cancer diagnosis and prognosis, as demonstrated in human papillary thyroid carcinoma.[25] Galectin-3's potential role in cancer cell survival, particularly in breast cancer, is evident in its ability to inhibit apoptosis and to cause a serum-independent growth of cDNA-transfected normal thyroid follicular cells.[26] Galectin-3's resistance indicates its ability to promote cancer cell proliferation, including breast cancer, through various molecular pathways, including its interaction with K-Ras at the plasma membrane.[27] Galectin-3 plays a crucial role in cancer signaling cascades, promoting tumor proliferation. It increases cyclin D and c-MYC production through β-catenin interaction, accelerating cell division and causing aggressive tumors. Understanding these mechanisms could lead to new therapeutic targets and treatments for galectin-3-related cancers, including breast cancer. Interventions targeting galectin-3's role in cell cycle progression could also be explored.[28] Serum-independent in aggressive cancers such as breast cancer can lead to new ways of retarding tumor growth and spread. By halting galectin-3interactions, targeted medicines could be developed, focusing on specific cancer cell mechanisms. This could reduce side effects associated with broad-spectrum treatments and be a major advancement in cancer therapy (Figure 2).

Galectin-3-induced apoptosis

Galectin-3, a member of the lectin family, can either stimulate or inhibit apoptosis, depending on its location inside the cell. This lectin facilitates the movement of the annexin family member to the mitochondria by binding to synexin in the cytoplasm and prevents apoptosis in cells by preventing cytochrome C release. Its apoptotic activity may be due to interaction with Nucling protein. Galectin-3 is involved in growth, adhesion, proliferation, and apoptosis. Being a protein in tumor cells, this lectin is a diagnostic marker for differentiated thyroid carcinoma due to its high expression and its ability to promote cellular transformation, suppress apoptosis, and increase cell viability through pro-survival pathways.[29-31]

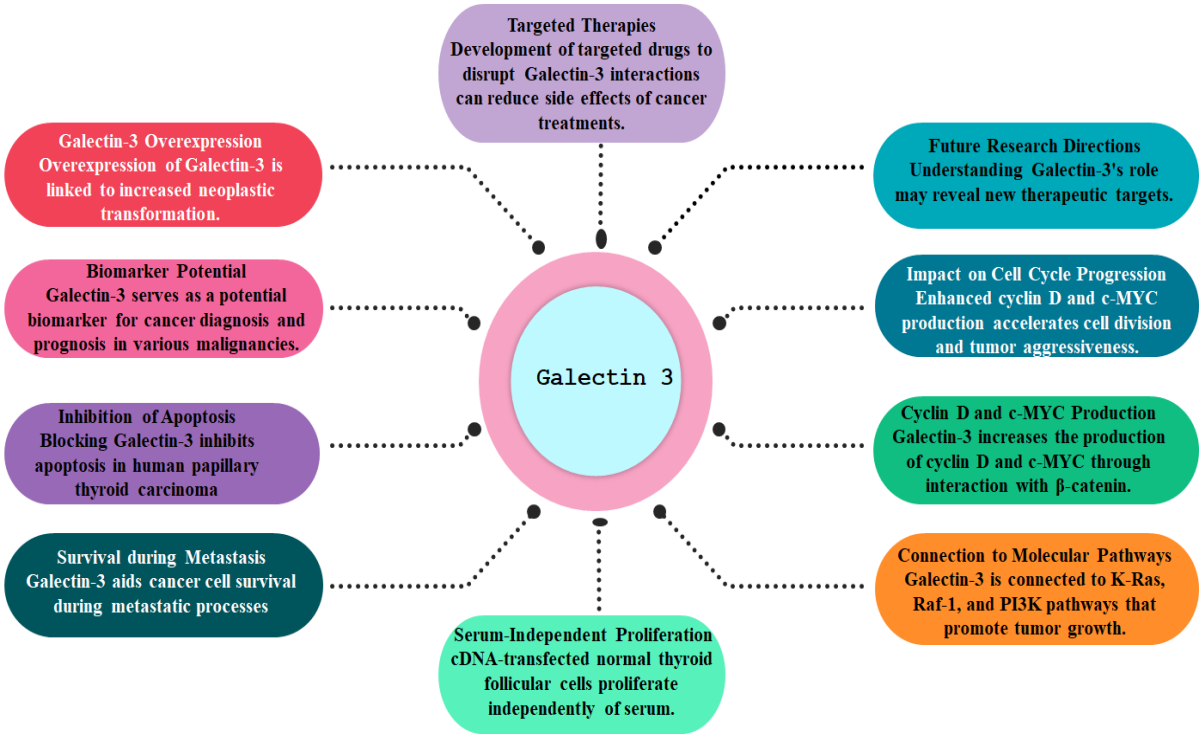

Figure 2. Roles of galectin-3 in breast cancer progression, metastasis, apoptosis, molecular pathways, and targeted therapies

Galectin-3 in breast cancer metastasis and angiogenesis

Tumor cells can spread by penetrating endothelium and entering lymphatic or circulatory systems, complicating treatment and leading to poorer prognoses. Overexpression of galectin-3 improves cell-ECM adhesion and encourages tumor cell migration.[32] Galectin-3 enhances tumor cell invasiveness by promoting aggregation in circulation and avoiding anoikis during metastasis. It also increases cancer cell adherence to endothelial cells by interacting with surface glycans such as MUC1 and Galβ1-3GalNAc-.[33] Oncofetal antigens, produced during fetal development, are crucial for cancer prognosis, diagnosis, and therapeutic monitoring, enhancing patient care and treatment personalization. Galectin-3, through binding with integrins and activating focal adhesion kinase (FAK), ,significantly influences neovascularization, stimulating angiogenesis by controlling VEGF and βFGF, and accelerating this process through interaction with aminopeptidase N (CD13).[34] Galectin-3 is crucial for cell aggregation, tumor angiogenesis, and cancer spread. It triggers angiogenesis and metastasis in various cancers. Galectin-3's role in tumor-endothelial cell interactions and early metastasis interactions is significant. External Thomsen-Friedenreich (TF) antigen could inhibit galectin-3-mediated tumor development.[35] Galectin-3-TF interactions could be a promising therapeutic strategy for aggressive cancers, limiting tumor proliferation and reducing metastatic potential.

Functions of galectins in glycan biology

The Golgi apparatus, which contains glycosyltransferases, plays a role in post-translational modification of glycoproteins, and the N-glycan content in SKBR-3 breast cancer cells reduces doxorubicin sensitivity.[36] The glycocalyx, a structural barrier in cancer cells, can evade therapeutic targeting, highlighting the complexity of cancer treatment. When treated with tunicamycin, glycocalyx cells were less responsive to mitogenic growth factors and more sensitive to doxorubicin, suggesting potential targets for improved treatment outcomes.[37] Glycan degradation decreases cell adhesion, potentially limiting cancer spread. The glycocalyx regulates integrin activity, guiding integrins to adhesion sites and forming a barrier across epithelial cells.[38] Structural integrity is crucial for maintaining epithelial cell cohesion, which can impact tumor progression. Galectins and glycans interact through complex mechanisms, such as oligomeric structure and multivalency. Galectin-3 plays a role in T-cell activation, possibly by interacting with N-glycans on T-cell receptors (TCR). MGAT5, a branching enzyme, influences immune receptor accessibility and activation potential, limiting TCR clustering.[39] Glycan structure influences immune cell responsiveness and autoimmunity. Galectin-3 prevents IL-5 production and interacts with pathogen glycans, promoting antigenic properties in macrophages. It eradicates *Candida albicans* by binding to β1-2 oligomannosyl residues.[40] Studies on genetically engineered mice deficient in certain galectins highlight their importance in the innate immune response to microbial infections, offering potential therapeutic targets for infectious disease control.

Galectin-3 as a therapeutic target

Galectin inhibitors, such as G3-C12, have shown promising results in preclinical research, preventing breast cancer cells from spreading to the lungs in athymic nude mice. [41] Recent research has successfully inhibited metastasis in mucin-secreting breast cancer cells using a high-affinity antibody. Salirasib, a derivative of salicylic acid, is being studied as a potential galectin inhibitor, which prevents farnesylated Ras activation and Ras isoform aggregation in cancer cells.[42] Tocotrienol, a natural vitamin E variant, may enhance the anticancer effects of γ-tocotrienol in metastatic breast cancer cells due to its hydrophobic and hydrophilic properties.[43] γ-tocotrienol significantly induces apoptosis and hinders metastasis in breast cancer cell lines by inhibiting processes such as epithelial-to-mesenchymal transition (EMT), lipid raft stability, and invasion, while minimizing normal mammary epithelial cell survival.[44] MDA-MB-231 human breast cancer cells produce large, metastatic polyploid cancer cells, which can be reduced by antioxidant therapy due to decreased mitochondria and reactive oxygen species levels.

Galectin-3's function in the biology of breast tumors

Galectin-1 increases Foxp3+ Treg cells in breast cancer tumors, aiding immune evasion and modulating immune tolerance. It interacts with neuropilin-1 to initiate signaling pathways, stimulating hepatic stellate cell migration and activation.[45] Galectin-1 promotes tumor growth and invasion, while galectin-3 prevents human

breast cancer cells from apoptosis due to nitric oxide.

Galectin-3's role in tumor cell survival and cancer persistence may be linked to chemotherapy resistance. Its knockdown in breast cancer cells improves treatment efficacy, suggesting it could be a target for enhancing treatment efficacy in resistant cancers.[46] Galectin-3 disrupts cell adhesion, suggesting potential tumor dissemination. Galectin-9, an anti-tumor and tumor-promoting protein, may cause apoptosis in colon cancer by increasing receptor tyrosine kinase phosphorylation, suppressing the immune system, and causing death.[47] Galectin-9's interaction with immune cells makes it a promising target for immunotherapy, potentially suppressing or boosting immune responses in cancer treatment, while galectins are intriguing candidates for therapeutic suppression.

Inhibitors of galectin

Galectin inhibitors, discovered decades ago, are being researched to slow or halt cancer growth and spread. Two main types are carbohydrates-based and non-carbohydrate-based, designed to mimic natural sugar-binding sites for targeted interactions with galectins.

Thiodigalactoside, a carbohydrate-based inhibitor of galectin-1, has shown promise in mouse models of breast cancer by causing apoptosis and reducing tumor development and metastasis.[48] Research shows that reducing angiogenesis by means of anginex, a peptide-based inhibitor of galectin-1, can impede tumor growth and cell proliferation, potentially slowing or reversing cancer spread. Modified citrus pectin, a carbohydrate-based inhibitor, targets galectin-3 and has antimetastatic properties, preventing metastasis and enhancing T-cell immune surveillance. These findings highlight the importance of these inhibitors in cancer treatment.

Galectin inhibitors are being studied in clinical trials, both alone and in combination with chemotherapeutic medications or monoclonal antibodies, to maximize effectiveness and reduce side effects, a strategy influenced by gene-targeting therapies.[49] Olaparib targets specific gene mutations, demonstrating precision medicine's potential for better patient outcomes. Alpelisib is approved for breast cancer treatment in PIK3CA mutant breast cancer.[49]

Alpelisib's approval highlights genetic marker-based cancer treatment. A phase I clinical trial tested the galectin-1 and -3 inhibitor GM-CT-01 in advanced solid tumors, including breast cancer, to evaluate disease impact and chemotherapy response.[50] The trial demonstrates the potential of galectin inhibitors in integrating into existing treatment regimens, potentially leading to new therapeutic protocols. The ATM inhibitor AZD1930 and a galectin-9 inhibitor showed synergistic properties in a mouse model, allowing for better-targeted therapy and personalized treatment, potentially improving patient survival and quality of life.[50]

CONCLUSION

Galectin inhibitors are promising in cancer therapeutics, targeting angiogenesis, metastasis, and immune surveillance. Both carbohydrate-based and peptide-based inhibitors show potential in preclinical models. However, challenges remain in optimizing dosing strategies, understanding resistance mechanisms, and identifying biomarkers. Future research should focus on elucidating pathways, refining inhibitor development, and conducting larger trials to establish safety and efficacy.

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Author Contributions

VIR and ALM were responsible for the conception and design of the study. UM contributed to the acquisition of data. MI and MAC were involved in the analysis and interpretation of data, as well as drafting the article. Critical revisions were made by VIR and MAC. All authors have read and approved the final manuscript.

Ethical Statement and Informed Consent

Not applicable.

Data Availability Statement

There were no new data generated, data sharing is not applicable.

Conflict of Interest

The authors declare that no conflicts exist.

Financial Disclosure

The authors declared no financial support.

Declaration the Use of AI in Scientific Writing

Nothing to declare.

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