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Mini-Review

NOTCH1 gene signaling pathway in the development and progression of carcinomas: an integrative review in cellular and molecular contexts

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Abstract

The *NOTCH1* gene and its signaling pathway play a critical role in the oncogenesis and progression of various carcinomas through complex cellular and molecular mechanisms. This integrative literature review examines 21 selected studies across multiple carcinoma types, including head and neck, prostate, penis, breast, and hepatocellular carcinomas, to elucidate the functional impact of notch1 alterations on tumor behavior and clinical outcomes. *NOTCH1* functions as a context-dependent regulator, acting either as an oncogene or tumor suppressor according to cellular environment and molecular context. Aberrations in notch1 signaling influence cell proliferation, differentiation, apoptosis, and angiogenesis, thereby contributing to cancer initiation and progression. Despite some variability in findings, the majority of studies indicate that notch1-related molecular changes have significant implications for prognosis and potential therapeutic targeting. This review highlights the importance of understanding notch1 signaling pathways in the cellular and molecular biology of carcinomas, aiming to pave the way for novel diagnostic and treatment strategies.

Keywords: *NOTCH1*, Signaling pathway, Carcinogenesis, Tumor progression, Oncogenic pathway.

1. Introduction

Cancer poses a challenge to public health, being one of the leading causes of death and morbidity globally. In most countries, this disease is one of the leading causes of death before the age of 70, posing a challenge to public health and medical research [1,2].

Cancer is characterized by the dysregulation of cell signaling mechanisms, causing uncontrolled cell growth and proliferation. Among the various molecular factors associated with this process, the *NOTCH1* gene plays a crucial role in several cell signaling pathways. This gene is part of a gene family of four genes responsible for encoding transmembrane receptors. These receptors are involved in the process of transcriptional regulation, differentiation, angiogenesis, apoptosis, stem cell maintenance, and cell proliferation through their activation by cleavage, after which the cleaved portion is translocated to the cell nucleus, forming the transcriptional activation complex together with the DNA-binding protein (CSL) + transcriptional coactivator such as MAML1 + recombination signal binding protein Jκ (RBP-J), thereby initiating transcription and activation of target genes such as: HES1, HEY, BCL-1, MYC (c-myc), CCND1, DLL1, PTEN, P53, among others [3-6].

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Mutations in this gene have been frequently reported in various types of cancer, especially in carcinomas [7]. The different mutations that occur can have different impacts on the behavior of this gene. Furthermore, the function of *NOTCH1* in tumorigenesis varies according to the cell type analyzed, acting both as a tumor suppressor gene and as an oncogene, and has been presented as a potential therapeutic target [8-10].

This functional duality of *NOTCH1* requires specific therapeutic strategies for each type of cancer, aiming to modulate its signaling and suppress tumor growth [11-13].

Currently, there are ongoing clinical trials with two main approaches to inhibit the *NOTCH1* pathway using monoclonal antibodies: these antibodies bind to the extracellular part of the *NOTCH1* receptor, preventing natural ligands from activating it. They are more targeted than gamma-secretase inhibitors (GSIs) because they do not impact other pathways that depend on this enzyme. Several antibodies are being evaluated for solid tumors with *NOTCH1* pathway overactivation. The other option is Gamma-Secretase Inhibitors (GSIs), which block the cleavage and activation of the *NOTCH1* receptor, interrupting the gene transcription process. Although promising in preclinical models of lymphoblastic leukemia, they exhibit considerable toxicity due to inhibition of the *NOTCH* pathway in healthy cells, particularly in the intestine [14-17].

Given the facts listed above, to better elucidate the role of *NOTCH1* and its signaling pathway in the progression of carcinomas, this study aimed to describe the role of the *NOTCH1* gene and its signaling pathway in different carcinomas. Understanding the role of this gene and its signaling pathway is essential for identifying new biomarkers and developing more effective and accurate therapeutic strategies.

2. Materials and Methods

This integrative literature review was conducted to examine the role of *NOTCH1* gene and its signaling pathway in the oncogenesis and progression of various carcinomas. A comprehensive and systematic search of relevant studies was performed across multiple scientific databases, including PubMed, Scopus, and Web of Science, covering publications up to the year 2024.

The inclusion criteria were original research articles and reviews focusing on *NOTCH1*gene alterations and related signaling pathways in carcinomas, specifically head and neck, prostate, penis, breast, and hepatocellular carcinoma. Studies providing molecular, cellular, or clinical data regarding notch1 function or expression were considered. Papers not published in English or lacking sufficient methodological details were excluded.

A total of 21 studies meeting the criteria were selected after screening titles, abstracts, and full texts. Data extracted included study type, carcinoma subtype, *NOTCH1* molecular findings, impact on cellular processes (such as proliferation, apoptosis, differentiation), and clinical outcomes when available.

The collected data were synthesized using qualitative analysis to identify common patterns, discrepancies, and biological implications of notch1 signaling in cancer development and progression. The review emphasized cellular and molecular mechanisms supported by experimental and clinical evidence to provide an integrative understanding of notch1's dual role as oncogene or tumor suppressor.

3. Results

The initial search in the databases retrieved 22,839 articles. Of these, some were excluded due to duplication and ineligibility, resulting in only 21 articles that met the established criteria. Figure 1 presents the flowchart used for the search and selection of articles in the bibliographic databases.

Table 1 of the supplementary material presents the data from the 21 articles analyzed, including authors, title, journal, carcinoma types, database, and year of publication. Table 2 of the supplementary material presents the objective, study type, and results related to the topic, as found in the studies selected for this integrative review.

Table 3 presents the 21 selected articles according to the type of carcinoma evaluated.

Table 4 demonstrates the number of studies that suggested a tumor suppressor role, oncogene or bimodal action for the *NOTCH1* gene in relation to its activation or inhibition, as well as the type of carcinoma in which these functions were observed.

Table 5 describes the number of studies according to the type of carcinoma and the impact of molecular changes in *NOTCH1* on the clinical outcome of the disease. In 9 of the 21 studies analyzed, there was no information about the patients' clinical outcome.

4. Discussion

4.1. Head and Neck Carcinoma

A total of four articles on *NOTCH1* in head and neck carcinoma (HNC) were highlighted. In HNC, the function of *NOTCH1* was shown to vary, acting as an oncogene, a tumor suppressor, or having a bimodal role. In the study

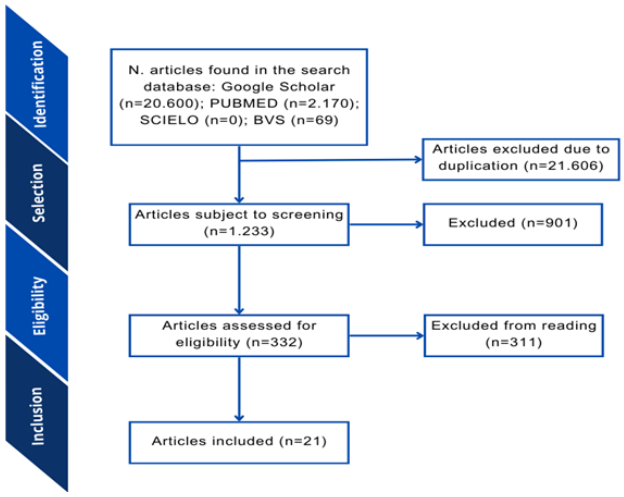


Fig. 1. Flowchart of the process of identification, screening, eligibility, and inclusion of articles in the literature review.

Table 3. Selected articles for each type of carcinoma discussed in this review.

Type of Carcinoma	№ selected articles
Head and neck	4
Prostate	4
Penis	5
Breast	4
Hepatocellular	4
Total =	21

Table 4. The role of NOTCH1 in carcinomas observed in this study.

Type of carcinomas	Oncogene		Tumor suppressor		Bimodal action	Total number of articles
	Notch1 activated	Notch1 inhibited	Notch1 activated	Notch1 inhibited		
Squamous head and neck	0	1	1	1	1	4
Prostate	3	0	0	1	0	4
Penis	5	0	0	0	0	5
Breast	4	0	0	0	0	4
Hepatocellular	2	0	0	2	0	4
Total	14		5		1	21

Table 5. Association of NOTCH1 with unfavorable or favorable prognosis according to studies highlighted in this review.

Carcinomas	Favorable prognosis	Unfavorable prognosis	No information	Total number of articles
Head and neck	1	0	3	4
Prostate	0	2	2	4
Penis	0	4	1	5
Breast	0	2	2	4
Hepatocellular	2	1	1	4
Total	3	9	9	21

by Grilli et al. (2020) [11], Notch1 receptor expression was active in 98% of HNC samples and was associated with non-recurrent disease and better patient prognosis, including higher disease-free and overall survival rates ($p < 0.001$). Based on these findings, the authors suggested a tumor suppressor role for *NOTCH1* and its signaling pathway in this carcinoma type.

In contrast, in the study conducted by Nyman et al. (2018) [18], the Notch1 signaling pathway was inhibited in HNC. The authors used genetically modified mice susceptible to HNC development and inhibited Notch1 signaling via DNMAML1 (Dominant Negative Mastermind-Like 1), a dominant-negative inhibitor designed to interfere with the Notch pathway. Loss of canonical Notch1 signaling favored tumorigenesis through increased nuclear expression of β-catenin. This study showed that inhibition of *NOTCH1* signaling could induce in vivo tumorigenesis, independently of HPV infection status, a major risk factor for the disease.

These two studies complement each other, suggesting that active Notch1 is associated with tumor suppressor function, whereas its absence may lead to enhanced tumorigenesis in HNC.

In contrast to the previous studies, Mao et al. (2017) [19] found that Notch1 signaling was active in HNC and associated with oncogenic behavior. In vivo experiments using transgenic rats showed that inhibition of *NOTCH1* via γ-secretase inhibitors (e.g., DAPT) significantly reduced tumor burden. Additionally, DAPT treatment reduced the infiltration of immune cells, especially macrophages and regulatory T cells. The authors proposed that targeting Notch1 could be beneficial in treating HNC. These findings were not observed by Nyman et al. (2018) [18].

Fukusumi and Califano (2018) reported that *NOTCH1* is the second most frequently mutated gene in HNC, after TP53. Their findings support a bimodal role for *NOTCH1*—as both an oncogene and a tumor suppressor—unlike other frequently mutated genes such as TP53 and PTEN, which are established tumor suppressors.

4.2. Prostate Carcinoma

Prostate carcinoma (PC) is among the cancers with the highest mutation rates in the *NOTCH1* gene. According to Rice et al. (2019) [21], Notch1 activation is elevated in PC, and loss of its function reduces proliferation, invasion, and tumor formation. In this study, inhibition of Notch1 using γ-secretase inhibitors or DAPT significantly suppressed the proliferative and metastatic potential of PC cells in both in vitro and in vivo assays. Thus, the presence of *NOTCH1* activity is linked to tumor progression and poor prognosis.

Cheng et al. (2021) [22] corroborated these findings, reporting that inhibition of the Notch1 pathway—especially in association with its ligand Jagged1—significantly reduced tumor growth. These results support the notion that *NOTCH1* acts as an oncogene in PC when activated.

Bayramoğlu et al. (2020) [23] detected *NOTCH1* expression in 17 (28.3%) of 60 PC patients. High NOTCH1 expression was found in metastatic tumors from nine different organs, showing its association with poor prognosis. Similarly, Shi et al. (2022) [13] observed that increased activity of γ-secretase and MAML2 enhanced *NOTCH1* expression, leading to increased tumor cell proliferation and invasion. These findings further reinforce the oncogenic role of *NOTCH1* in PC.

4.3. Penile Carcinoma

Penile carcinoma (PeCa) is a rare cancer type that also exhibits mutations in the *NOTCH1* gene. Signoretti and Aster (2018) [24] identified two gain-of-function mutations—originally described in T-cell acute lymphoblastic

leukemia/lymphoma—that result in ligand-independent activation of the Notch1 pathway in a metastatic PeCa case: F1592S (T-to-C point mutation at nucleotide 4775, found in 5% of 321 reads) and L1678P (T-to-C point mutation at nucleotide 5033, found in 14% of 339 reads). This case report suggests that mutated *NOTCH1* may act as an oncogene in PeCa. The patient showed high tumor proliferation and recurrence, with metastases to bones, lungs, and liver.

Wang et al. (2019) [25] reported that among the nine main signaling pathways altered in PeCa, the Notch1 pathway showed the highest alteration rate (30% of samples). They suggested that Notch1 plays roles in inflammation and tumor angiogenesis, promoting tumorigenesis. Similarly, Canto et al. (2022) [26] and Chahoud et al. (2021) [27] identified *NOTCH1* and its pathway among the most frequently mutated in PeCa. Patients with mutations in this pathway showed worse overall survival.

Supporting these findings, Cao et al. (2021) [28] also showed that recurrent *NOTCH1* mutations are associated with penile carcinogenesis. Mutations were found in patients with and without lymph node metastasis. However, patients with nodal metastases had lower overall survival regardless of mutation status.

4.4. Breast Carcinoma

The Notch1 signaling pathway plays a fundamental role in the development of breast carcinoma (BC). Koury et al. (2017) [29] reported that abnormal activation of this pathway promotes tumorigenesis in BC. High Notch1 expression correlated with lower differentiation in 80% of usual-type epithelial hyperplasias, 67% of ductal carcinoma in situ cases, 89% of invasive ductal carcinomas, and 57% of invasive lobular carcinomas. Notch1 was also critical in the epithelial–mesenchymal transition (EMT).

Jiang et al. (2020) [30] further reported that endothelial Jagged1, the primary Notch1 ligand, activates Notch1 and increases ZEB1 expression, promoting EMT. This activation is associated with BC progression, tumor aggressiveness, and poor prognosis.

Kałafut et al. (2022) [31] confirmed that Notch1 is involved in cell proliferation and chemotherapy resistance in BC. Consistent with these findings, Miao et al. (2020) [32] identified *NOTCH1* as an oncogene that suppresses BRCA1-deficiency-induced apoptosis and promotes BC through EMT pathway activation.

4.5. Hepatocellular Carcinoma

In hepatocellular carcinoma (HCC), aberrant Notch1 activation has been linked to metastasis. Zhang (2019) [33] showed that patients with high Notch1 expression had lower disease-free survival compared to those with low expression. The study also identified RNF187 as a key mediator of Notch1-induced invasion and metastasis via EMT induction. Notch1-RNF187 axis activation was associated with worse prognosis in HCC patients.

In another study, Wang et al. (2020) [34] reduced *NOTCH1* expression in HCC cells and observed significant reductions in cell proliferation. In contrast, Hany et al. (2018) found that *NOTCH1* expression was higher in low-grade HCC than in high-grade cases ($p = 0.036$). *NOTCH1* expression was inversely correlated with mitotic count ($p = 0.008$) and necrosis ($p = 0.005$). Patients with lower *NOTCH1* expression had reduced disease-free survival ($p = 0.045$), while overall survival did not differ significantly ($p = 0.220$), though it was slightly longer in patients with higher expression. These findings suggest a tumor suppressor role for Notch1 and that Notch1 agonists could improve HCC outcomes.

Li et al. (2023) [36] also observed a tumor suppressor role for *NOTCH1* in HCC, mainly through crosstalk with other signaling pathways. In their study, high *NOTCH1* expression was associated with better survival. However, in some contexts, *NOTCH1* may also promote tumorigenesis through EMT activation.

5. Conclusion

This review revealed that the *NOTCH1* gene and its signaling pathway play different roles depending on the type of carcinoma and the nature of the mutation involved. In certain carcinomas, such as head and neck carcinoma, *NOTCH1* functioned as a tumor suppressor. However, in penile carcinoma, due to gain-of-function mutations, *NOTCH1* and its signaling pathway were predominantly identified as oncogenic in most of the evaluated studies. Furthermore, *NOTCH1* activation appears to promote tumor metastasis, as observed in penile carcinoma (PeCa). Therefore, inhibition of the Notch1 signaling pathway emerges as a potential therapeutic option in tumors harboring gain-of-function *NOTCH1* mutations. In addition, *NOTCH1* has shown promise as a potential biomarker.

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Conflict of Interests

The author has no conflicts with any step of the article preparation.

Consent for publications

The author read and approved the final manuscript for publication.

Ethics approval and consent to participate

No human or animals were used in the present research.

Informed Consent

The authors declare that no patients were used in this study.

Availability of data and material

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Authors' contributions

Leticia Milene Silva da Silva: conceptualization, writing, research; Ana Gabrielly de Melo Matos: support in the selection of articles, data curation; Kwang II Marciaga Teófilo: Review; João Victor Carvalho: Review; Denner Rodrigo Diniz Duarte: Review; Bruna Larissa Nolêto Sousa: Review; Juliana Martins da Guia Ribeiro do Carmo: Review; Joyce Santos Lages: Review; Rita da Graça Carvalhal Frazão Corrêa: Review; Antonio Augusto Lima Teixeira Júnior: Review, supervision, and critical analysis

of the content; Jaqueline Diniz Pinho: Conception, writing supervision, and critical analysis of the content; Gyl Eanes Barros Silva: Conception and writing supervision, critical analysis of the content.

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