REVIEW

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Role of SNHGs in Adverse Prognostic Factors in Cervical Cancer: A Systematic Review

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Abstract

Objective: This study aims to summarize the main findings in the literature regarding the family of long non-coding RNAs (LncRNAs), specifically SNHGs, in cervical cancer. **Methods:** The study was conducted following the PRISMA protocol, using the PICOS framework for the search strategy. The research sources included PubMed, ScienceDirect, Lilacs, and Medline. Inclusion and exclusion criteria were applied, and data from each article were extracted, including: clinicopathological characteristics, biological function of SNHGs, clinical indicators, and diagnostic and prognostic markers. **Results:** Out of a total of 3.803 studies, 12 were selected, encompassing 8 SNHGs (GAS5, SNHG5, SNHG7, SNHG12, SNHG14, SNHG16, SNHG17, and SNHG20) associated with cervical cancer. All, except for GAS5, showed increased expression. In the literature review, SNHG expression was linked to adverse prognostic factors in cervical cancer, such as proliferation, migration, invasion, lymph node metastasis, and apoptosis. **Conclusion:** Although further studies are needed, these data highlight the significant role of SNHGs in tumor biology and the promising potential of this class of transcripts as tools in the clinical management of cervical cancer.

Keywords: Cervical cancer- lymph node metastasis- long non-coding RNA- prognosis.

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Introduction

Cervical cancer (CC) is the fourth most common cancer among women globally [1]. In Brazil, it ranks as the third most prevalent cancer in women and, in some regions, remains one of the most frequently diagnosed cancers [2,3]. The occurrence of metastases is significantly associated with reduced survival rates in CC patients. However, the precise mechanisms underlying metastasis development are not yet fully understood. A comprehensive understanding of these carcinogenic pathways is essential to improving survival outcomes [4, 5].

One potential element involved in these processes is the family of long non-coding RNAs (lncRNAs), which have emerged as potential biomarkers for more effective prognosis and treatment strategies [6–8]. LncRNAs are endogenous, single-stranded RNA molecules longer than 200 nucleotides that do not encode proteins. Instead, they are involved in various biological processes at transcriptional and post-transcriptional levels. Additionally, lncRNAs can interact with other regulatory molecules, such as proteins and microRNAs, functioning as molecular sponges that influence the expression of target genes [9, 10].

Some lncRNAs can host other non-coding RNAs within their sequences, including small nucleolar RNAs (snoRNAs), which are relatively small, ranging from 70 to 140 nucleotides in length [11]. Small Nucleolar RNA Host Genes (SNHGs) are a subgroup of lncRNAs that contain both introns and exons in their sequences and produce snoRNAs through alternative splicing. The SNHG family currently comprises 22 members, from

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SNHG1 to SNHG22, all of which play significant roles in cellular processes such as proliferation, apoptosis, invasion, and migration [12, 13].

Dysregulated expression of SNHG family members is commonly observed in various cancer types, including thyroid [14, 15], breast [16], pancreatic [17, 18], and prostate cancer [19, 20]. Collectively, these studies suggest that SNHGs could serve as promising oncological biomarkers. However, research on SNHGs in CC remains limited and has predominantly focused on specific SNHG members. Therefore, this systematic review aims to consolidate and summarize key findings regarding this specific class of lncRNAs in the context of CC.

Materials and Methods

Study Design and Protocol Registration

This study is a systematic literature review registered in the International Prospective Register of Systematic Reviews (PROSPERO) (https://www.crd.york.ac.uk/PROSPERO/) under the registration number CRD42022356239. The review was conducted in accordance with the guidelines of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) (https://www.prisma-statement.org/).

Study Question

This study addresses the following research question: "What is the association between SNHGs and adverse prognostic factors in cervical cancer?" To answer this question, the PRISMA protocol was employed, and the PICOS framework was defined as follows: the population consisted of patients with cervical cancer; the intervention involved the effectiveness of SNHG biomarkers; no comparison group was applicable; the outcome focused on the relationship with biomarker expression; and the study design was restricted to quasi-experimental studies.

Eligibility criteria

The inclusion criteria were as follows: (1) articles written in English, (2) published between 2017 and 2022, (3) experimental studies involving patients diagnosed with cervical cancer, and (4) studies providing detailed descriptions of the techniques used. The exclusion criteria were: (1) abstracts, reports, reviews, monographs, and dissertations, and (2) studies that utilized data from The Cancer Genome Atlas Program (TCGA).

Selection of studies

Titles and abstracts were reviewed to exclude studies that were clearly irrelevant to the review. Full texts of the remaining studies were retrieved, and eligible studies for inclusion in this systematic review were identified.

The selection process was conducted independently by two reviewers, based on the eligibility criteria. Any disagreements were resolved by a third reviewer.

Data Sources and Search Strategies

We conducted searches in the following electronic databases: U.S. National Library of Medicine (PubMed), ScienceDirect, Lilacs, and Medline. The search strategy incorporated the following descriptors: "Uterine Cervical Neoplasms"[Mesh] AND "RNA, Long Noncoding"[Mesh]; "Uterine Cervical Neoplasms"[Mesh] AND "long noncoding RNA SNHG8, human" OR "long non-coding RNA SNHG1, human" OR "long non-coding RNA GAS5, mouse" OR "long non-coding RNA SNHG5, human" OR "long non-coding RNA SNHG6, human" OR "SNHG12 long non-coding RNA, human" OR "SNHG16 lncRNA, human" OR "long non-coding RNA SNHG20, human"; as well as keywords such as "LncRNAs and cervical cancer," "LncRNAs and uterine cancer," "SNHG1 cervical cancer," "SNHG5 cervical cancer," "SNHG6 cervical cancer," "SNHG4 cervical cancer," "SNHG7 cervical cancer," "SNHG12 cervical cancer," "SNHG16 cervical cancer," and "GAS5 cervical cancer."

Study Selection and Strategies

We removed duplicate studies and those that did not meet the inclusion criteria. Information from each article was recorded in a Microsoft Excel 2019 table. For each study, we documented the following information: a) Clinico pathological characteristics: TNM staging, lymph node metastasis, cancer stage, degree of cellular differentiation, and type of expression; b) Biological Function of SNHGs: roles in proliferation, migration, invasion, cell cycle, apoptosis, colony formation, involvement in epithelial-mesenchymal transition, and whether these lncRNAs target microRNAs and pathways; c) Clinical Indicators: diagnostic and prognostic indicators, and biomarkers.

Assessment of Methodological Quality of Included Studies

The methodological quality of the 12 included studies was independently evaluated by researchers EAA, AGDMM, and JDP. The Joanna Briggs Institute (JBI) Critical Appraisal Tools (JBI, 2020) were employed for this assessment. Each criterion was rated as "yes," "no," "unclear," or "not applicable" for each study. The risk of bias classification was determined based on the following scoring system: a) 1 to 3 "yes" responses indicated a high risk of bias; b) 4 to 6 "yes" responses indicated a moderate risk of bias; c) 7 to 8 "yes" responses indicated a low risk of bias (Table 1).

Results

Literature Search

From the initial pool of 3.803 studies identified across the four databases, 1.543 records were excluded due to duplicate removal, leaving 2.260 articles for title and abstract screening. During this process, 84 articles were identified as reviews, and 82 were excluded for not addressing the research question or being published in languages other than English. After applying the inclusion criteria, a total of 12 studies were included. A detailed overview of the article screening process is provided in Figure 1.

Among the 12 selected articles, findings reveal that most SNHGs are significantly overexpressed and play critical regulatory roles in invasion, cell proliferation, apoptosis, epithelial-mesenchymal transition, and migration.

Table 1. Assessment of Methodological Quality in Included Studies Using Joanna Institute Critical Appraisal Tools (JBI) - Yes; No; Unclear (U); Not Applicable (NA).

	Quality Assessment										
Reference	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Total (YES)	Bias Level
Zhang et al. [33]	YES	YES	NO	YES	U	YES	YES	YES	YES	7	Low
Zeng et al. [22]	YES	YES	NA	YES	NO	YES	NO	YES	YES	6	Moderate
Dong et al. [28]	YES	YES	U	YES	NA	YES	YES	YES	YES	7	Moderate
Zhang et al. [25]	YES	YES	U	YES	NO	YES	YES	YES	YES	7	Low
Ji t al. [29]	YES	YES	NA	YES	U	YES	NA	YES	YES	6	Moderate
Zhu et al. [34]	YES	YES	NA	YES	NO	YES	NA	YES	YES	6	Moderate
Tao et al. [27]	YES	YES	NO	YES	NA	YES	YES	YES	YES	7	Low
Wu et al. [19]	YES	YES	U	YES	NO	YES	NA	YES	YES	6	Moderate
Cao et al. [32]	YES	YES	U	YES	U	YES	YES	YES	YES	7	Low
Guo et al. [30]	YES	YES	U	YES	NA	YES	YES	YES	YES	7	Low
Li et al. [23]	YES	YES	NO	YES	NO	YES	NO	YES	YES	6	Moderate
Fang et al. [24]	YES	YES	U	YES	NO	YES	YES	YES	YES	7	Low

Table 2 provides an overview of the methodologies used, biological sample characteristics, clinical parameters, and histopathological information related to CC.

Analysis of the expression types, target genes, and signaling pathways of the eight studied SNHGs reveals that seven (SNHG5, SNHG12, SNHG14, SNHG16, SNHG17, SNHG20, and GAS5) act as molecular sponges for miRNAs, contributing to key biological processes such as cell proliferation, apoptosis, migration, and invasion (Table 3).

Discussion

SNHGs and Their Biological Function in the Context of Cervical Cancer

SNHGs have been extensively studied as potential biomarkers for cancer due to their role in regulating critical biological pathways [11, 21]. In the context of In the context of CC, investigating SNHGs is particularly significant as it provides valuable insights into the biological mechanisms underlying tumor progression.

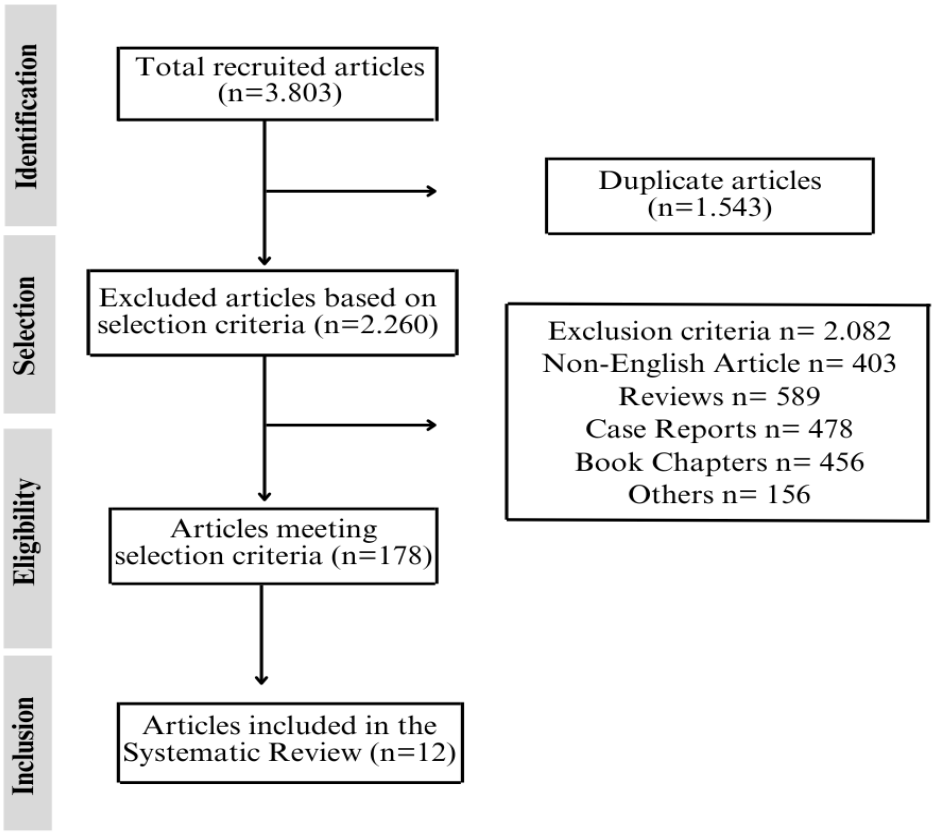

Figure 1. Flowchart Depicting the Selection and Identification of Studies, Following the Methodological Steps Outlined in the PRISMA Guidelines.

Table 2. Functional Characterization and Clinical Significance of lncRNAs in Cervical Cancer

LncRNA	Expression Level	Samples	Techniques	Biological Function	Clinical Significance	Reference
SNHG5	Overexpressed	tumor tissue and cell lineage	qRT-PCR	Proliferation, migration, and invasion.	FIGO II and lymph node metastasis.	Zhang et al. [33]
SNHG7	Overexpressed	tumor tissue and cell lineage	qRT-PCR	Proliferation, invasion, and EMT.	TNM III-IV, lymph node metastasis.	Zeng et al. [22]
SNHG12	Overexpressed	tumor tissue and cell lineage	qRT-PCR	Proliferation, migration, and invasion.	FIGO II, lymph node metastasis.	Dong et al. [28]
SNHG14	Overexpressed	tumor tissue and cell lineage	qRT-PCR	Proliferation, promotion of apoptosis.	FIGO III-IV, tumor size ≥4 cm.	Zhang et al. [25]
	Overexpressed	tumor tissue and cell lineage	qRT-PCR	Cell proliferation, migration, invasion, and promotion of apoptosis.	FIGO III-IV, lymph node metastasis.	Ji t al. [29]
SNHG16	Overexpressed	tumor tissue and cell lineage	qRT-PCR	Proliferation, migration, and EMT.	FIGO IIb-III, lymph node metastasis, poorly differentiated tumor, and tumor size ≥5 cm.	Zhu et al. [34]
	Overexpressed	tumor tissue and cell lineage	qRT-PCR	Proliferation, migration.	TNM IIIa, tumor size >4 cm, worse survival, and poorly differentiated tumor.	Tao et al. [27]
	Overexpressed	tumor tissue and cell lineage	qRT-PCR	Proliferation, EMT, migration, and invasion.	TNM III-IV, tumor size ≥5 cm.	Wu et al. [19]
SNHG17	Overexpressed	blood samples	qRT-PCR	Proliferation, migration, and invasion.	FIGO III-IV, lymph node metastasis, tumor diameter ≥4 cm.	Cao et al. [32]
SNHG20	Overexpressed	tumor tissue and cell lineage	qRT-PCR	Proliferation, migration.	FIGO IIb-IIa, lymph node metastasis, tumor size ≥4 cm.	Guo et al. [30]
GAS5	Decreased	tumor tissue and cell lineage	qRT-PCR	Proliferation, invasion.	FIGO IIa, lymph node metastasis.	Li et al. [23]
	Decreased	tumor tissue and cell lineage	qRT-PCR	Promotion of apoptosis.	FIGO III-IV, lymph node metastasis.	Fang et al. [24]

Table 3. LncRNAs and microRNAs associated with Cervical Cancer

LncRNA	MicroRNAs	Expression	Target gene	Signaling Pathways	Reference
SNHG5	miR-132	down-regulated.	*SOX4*	-	Zhang et al. [33]
SNHG12	miR-424-5p	down-regulated.	-	-	Dong et al. [28]
SNHG14	miR-206	down-regulated.	*YWHAZ*	-	Ji t al. [29]
SNHG16	miR-216-5p	down-regulated.	*ZEB1*	-	Zhu et al. [34]
	miR-128	down-regulated.	*GSPT1 e WNT3A*	WNT/β-catenina	Ji t al. [29]
SNHG17	miR-375-3p	down-regulated.	-	-	Zhu et al. [34]
SNHG20	miR140-5p	down-regulated.	*ADAM10*	MEK/ERK	Cao et al. [32]
GAS5	miR-21	down-regulated.	*PDCD4*	-	Fang et al. [24]

This systematic review highlights that dysregulation of these biomolecules in CC influences key tumorigenic processes. Notably, the study by Zeng et al. [22] demonstrates that SNHG7 plays a pivotal role in tumor progression by promoting processes such as increased cell proliferation, enhanced invasion capacity, and migration of cancer cells.

Conversely, the lncRNA GAS5 exhibited decreased expression in cervical cancer tissue. Experimental studies assessing the impact of this lncRNA revealed that overexpression of GAS5 inhibited cell proliferation in vitro in CC cells [23]. Fang et al. [24] further support these findings, emphasizing the reduced expression of GAS5 and its association with resistance to cisplatin, a chemotherapeutic agent widely used in CC treatment.

These findings underscore the complex regulatory functions of SNHGs in CC, affecting processes such as cell proliferation, invasion, migration, apoptosis, and drug resistance. A deeper understanding of the mechanisms by which SNHGs influence these processes could facilitate the development of targeted therapies and improve prognostic accuracy for CC patients. Further research is essential to fully realize the potential of SNHGs as diagnostic and therapeutic tools in CC [25].

SNHGs as Endogenous Competitors of microRNAs

SNHGs can act as endogenous competitors of microRNAs (miRNAs) due to their potential sequence complementarity, functioning as molecular "sponges." By binding to miRNAs, SNHGs inhibit their interaction with specific target genes, thereby altering the availability of miRNAs for other mRNAs [26]. This interaction significantly influences the expression of genes involved in biological processes such as cell proliferation, invasion, metastasis, and treatment resistance [25].

In the context of colorectal cancer (CC), Wu et al. [19] suggested that SNHG16 may function as an oncogene by acting as a sponge for miR-128, thereby suppressing the activity of the WNT/β-catenin signaling pathway. This study also reported that aberrant SNHG16 expression is correlated with tumor stage and size. The WNT/β-catenin signaling pathway is widely studied in cancer due to its potential as a therapeutic target [26]. Additionally, SNHG16 overexpression was associated with a more aggressive tumorigenic phenotype and demonstrated the ability to interact with the transcription factor SPI1, recruiting it to specific regions of the PARP9 gene [27]. These findings highlight the intricate regulatory roles of SNHG16, which interacts with both miRNAs and proteins.

SNHG12 expression in CC was significantly upregulated, promoting cell growth and invasion. SNHG12 binds to miR-424-5p, preventing its interaction with target genes, which activates pathways that enhance cell proliferation and invasion in CC cells [28]. Similarly, Ji et al. [29] found that overexpression of SNHG14 is associated with a higher histological grade of CC. SNHG14 interacts with miR-206, reducing its intracellular levels and impairing its regulation of the zeta 14-3-3 protein (YWHAZ).

SNHG20, when overexpressed, was shown to regulate the miR-140-5p/ADAM10 axis and the MEK/ERK signaling pathway in CC [30]. The MEK/ERK signaling pathway is a critical intracellular signaling cascade involved in various biological processes, including gene expression regulation. Aberrant MEK/ERK signaling is associated with apoptosis resistance and angiogenesis [31].

SNHGs as Potential Biomarkers

SNHGs have been extensively studied for their differential expression across various cancer types, including CC. Abnormal levels of SNHGs provide valuable insights for early diagnosis and cancer monitoring [25]. The expression of SNHG17 was significantly elevated at FIGO stage II, when the carcinoma extends beyond the uterus but not to the pelvic wall, and was also associated with lymph node metastasis. Notably, altered expression of this lncRNA was detected in serum samples from patients [32].

In another study, SNHG20 expression was significantly increased in CC tumors with larger size, advanced FIGO stage, and lymph node metastasis [30]. Similarly, elevated SNHG14 expression in CC patients was linked to worse prognostic factors, particularly the presence of metastasis [26].

SNHG12 expression was notably higher at FIGO stage II, indicative of invasion beyond the uterus but without extension to the lower third of the vagina or pelvic wall [29]. This lncRNA was also associated with lymph node metastasis in another study, further emphasizing its role in tumor progression [33].

In conclusion, this systematic review underscores the importance of SNHGs in translational cancer research, particularly for CC. The therapeutic modulation of

lncRNAs, as explored in RNA-based therapies, offers significant potential to improve outcomes in CC patients. Understanding the contributions of SNHGs to drug resistance and their complex interactions with other RNAs is pivotal for developing more effective therapeutic approaches.

Nonetheless, further research is needed to fully elucidate the functions and mechanisms of SNHGs to enable the translation of these findings into meaningful clinical applications.

Author Contribution Statement

EAA, JDP, and MSDA acquired the data and had full access to all the data in the study, being responsible for the integrity and accuracy of data analysis. EAA analyzed and interpreted the data. EAA, JDP, and MSDA reviewed the data and applied the protocol. EAA, FJLL, MDSDSC, GEBS, JMCDS, ASK, FCM, AGCO, AGDMM, JDP, and MSDA conducted a critical review of the entire manuscript regarding relevant intellectual content. EAA, JDP, and MSDA developed the concept and design of the study.

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Study Registration

This study was registered in the International Prospective Register of Systematic Reviews under the number CRD42022356239.

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