Tabari Biomedical Student Research Journal

OPEN ACCESS

ORIGINAL: Identification of novel lncRNAs and potential role in cervical cancer

Faezeh Rafiei
Hossein Maghsoudi

Payame noor university, Tehran, Iran

ARTICLE INFO

Submitted: 05 Oct 2025
Accepted: 11 Nov 2025
Published: 21 Dec 2025

Keywords:
Cervical cancer
lncRNAs
Lnc2Cancer

Correspondence:
Hossein Maghsoudi
Payame noor university, Tehran, Iran
Email: Hossein_m2002@yahoo.com
ORCID: 0000-0002-7173-7863

Citation:
Rafiei F, Maghsoudi H. Identification of novel lncRNAs and potential role in cervical cancer. Tabari Biomed Stu Res J. 2025;7(4): 8- 16.

ABSTRACT

Background: Cervical cancer, considered as common cancer in females and in this case uterus cells begins to grow and reproduce out of control. It is obvious that knowing the molecular factors involved in progression of cervical cancer the diagnosis and treatment process can be more efficient, so in this study, we aimed to investigate LncRNAs related database to provide a collection of LncRNAs in this article for researchers.

Methods: In this research the data related to LncRNAs associated with cervical cancer were retrieved from the global databases. The database that was examined in this research include: Lnc2Cancer.

Results: Expression pattern of about 369 lncRNA and circRNA genes involved in cervical cancer were obtained.

Conclusion: These were only a few examples of the types of lncRNAs that have been collected by bioinformatics databases and it is possible to design new treatment processes for cervical cancer by identifying them.

Introduction

Cervical cancer, considered as common cancer in females and in this case uterus cells begins to grow and reproduce out of control[1]. Although the causes of this cancer have not been fully understood, scientists still do not know how the cancer grows and develops. Before the growth of cancer cells, the tissue undergoes changes that are different from the normal tissue[2, 3]. In the past several years, many studies have been conducted to find the molecular mechanisms and regulatory networks that play a role in tumorigenesis. Cervical cancer is the most important disease caused by human papillomavirus, which is a major public health problem worldwide[4]. HPV infection can be the origin of pre-neoplastic lesions known as cervical intraepithelial neoplasia. E6 and E7 oncoproteins, which are encoded by high-risk HPVs, affect infected cervical cells, they are able to inhibit P53 and PRb repressors, respectively, and with a large number of cell signaling factors regulating cell cycle, genomic stability and epigenetic changes interact [5, 6]. The gradual accumulation of genetic and epigenetic changes in HPV-infected cells is also very important for the final progression to cervical cancer[7, 8]. The mutational profile of cervical cancer, the presence of a series of non-synonymous somatic nucleotide changes in TP53, PTEN, PIK3CA, STK11 and KRAS genes shows that epigenetic changes, including microRNA, LncRNA and circRNA deregulation, play an important role in cell transformation during specific stages[9]. Long non-coding RNAs are associated with a large number of cellular functions. Long non-coding RNAs that are more than 200 nucleotides in length are a new and emerging class of transcripts that are encoded by the genome but are not usually translated into protein and play a key role in gene expression, growth, development, differentiation, and in regulating chromatin

dynamics[10]. According to the ENCODE project, the human genome encodes approximately 28,000 LncRNAs, many of which have not yet been discovered, and their expression is inappropriate in various cancers[11]. LncRNAs are important and necessary regulators of gene expression and play a role in all cellular processes, gene expression through expansion or inhibition of translation of mRNA to protein, expansion or inhibition of DNA transcription, affecting mRNA splicing, controlling chromatin density and determines the stability of regulated mRNA[12]. They can bind to proteins, DNA or even messenger and small RNAs. According to recent studies, lncRNAs use a wide range of mechanisms to affect their targets[13]. The mechanism of lncRNAs' effect is on two levels: transcriptional regulation and post-transcriptional regulation. LncRNAs have wide expression patterns in different types of cancer. Dys-regulation of lncRNAs is related to human diseases and cancers[14]. According to the studies conducted, their expression changes are involved in metastasis, tumor formation and tumor progression[15]. LncRNAs can also play a tumor suppressor role and by reducing their expression, they play a role in cancer progression, such as GAS5 and MEG3, and they can also play an oncogenic role, in which case, by increasing their expression, they stimulate cancer conditions, such as HOTAIR and MALAT[16, 17]. It is obvious that knowing the molecular factors involved in progression of cervical cancer the diagnosis and treatment process can be more efficient, so in this study, we aimed to investigate LncRNAs related database to provide a collection of LncRNAs in this article for researchers.

Material and Methods

Lnc2Cancer

The updated Lnc2Cancer database is a manually entered, laboratory-validated database. This database presents the relationship between lncRNAs and human cancers. The new version of this database also provides the relationship between circRNAs and human cancers because circRNAs are a unique type of lncRNAs and are importance in many human cancers. In the new version, there are 10,303 associations between 2,659 human lncRNAs, 734 CircRNAs and 216 human cancer subgroups, which are the result of the review of more than 15,000 published articles. In this database, regulatory mechanism (enhancer, transcription factor, methylation, miRNA, diversity), biological function (coding, EMT, apoptosis, autophagy, immune ability, cell growth) and clinical application (circulation, metastasis, recurrence, survival) are presented for lncRNAs and circRNAs in human cancers. To discover and identify the level of expression of circRNAs and lncRNAs in human cancers in this database, RNAi, in vitro knockdown, Western blot, Luciferase reporter assay and scRNA-seq techniques are also used in this database using molecular biology techniques and analyzed lncRNAs and circRNAs associated with cancers based on biological functions, regulatory mechanisms and clinical applications[18]. The Lnc2Cancer v3.0 database is available at http://bio-bigdata.hrbmu.edu.cn/lnc2cancer/. In the menu bar of the home page of this database, by clicking on the "Browse" icon, a new window will open. You can browse by lncRNA/circRNA and select any of the 3 subgroups of regulatory mechanism, biological function and application. Clinical information related to lncRNA and circRNA can be retrieved in different cancers. Also, in the browse by tissue section, lncRNAs and circRNAs related to that cancer can be found by selecting the desired tissue. Also, on the right side of the page, there are three boxes, Cancer, lncRNA, and circRNA. By clicking on cancer and selecting the desired cancer, the lncRNAs and circRNAs of that cancer are retrieved. Also, by clicking on the lncRNA or circRNA box and selecting the desired lncRNA or circRNA, information including the name of the cancer, the identification method, and the expression pattern are retrieved. In this research, by clicking on the "Browse" icon and selecting cervical cancer, cervical intraepithelial neoplasia, and cervical

squamous cell carcinoma, a new window will open and lncRNAs and circRNAs along with the expression pattern in cervical cancer will be shown.

Results

The Lnc2Cancer database is a laboratory-approved and updated database that examines the relationship between lncRNAs and circRNAs in various cancers and also interprets their mechanisms. Table 1 showed expression pattern of about 369 lncRNA and circRNA genes involved in cervical cancer. In Table 2, a lncRNA gene involved in cervical intra-epithelial neoplasia is shown along with the expression pattern.

Table1. List of lncRNAs and circRNAs involved in cervical cancer retrieved from Lnc2Cancer database

Expression pattern	lncRNA/circRNA	Expression pattern	lncRNA/circRNA
up-regulated	ARAP1-AS1	up-regulated	AB073614
up-regulated	ASB16-AS1	down-regulated	AC017078.1
down-regulated	ASK00420	differential expression	AC024560.2
up-regulated	AX748340	up-regulated	AFAP1-AS1
up-regulated	BC200	up-regulated	AL592284.1
up-regulated	BCAR4	differential expression	ANRASSF1
up-regulated	BCAR4	up-regulated	ANRIL
up-regulated	BCYRN1	up-regulated	ANRIL
up-regulated	BDLNR	up-regulated	ANRIL
up-regulated	BLACAT1	up-regulated	ANRIL
up-regulated	BLACAT1	up-regulated	C5orf66-AS1
up-regulated	BLACAT1	up-regulated	CALML3-AS1
differential expressed	circ_103519	up-regulated	CAR10
up-regulated	circ_103973	up-regulated	CASC11
differential expressed	circ_104315	up-regulated	CASC15
differential expressed	circ_400068	up-regulated	CASC15
up-regulated	circAGFG1	down-regulated	CASC2
up-regulated	circAKT1	down-regulated	CASC2
up-regulated	circAMOTL	down-regulated	CASC9
up-regulated	circATP8A2	up-regulated	CCAT1
up-regulated	circCLK3	up-regulated	CCAT1
up-regulated	circCSPP1	up-regulated	CCAT1
up-regulated	circEIF4G2	up-regulated	CCAT1
down-regulated	circFoxO3a	down-regulated	CCAT2
down-regulated	circITCH	up-regulated	CCEPR
up-regulated	circMYBL2	up-regulated	CCHE1
up-regulated	circSLC26A4	up-regulated	CCHE1
down-regulated	circSMARCA5	up-regulated	CCHE1
up-regulated	CRNDE	up-regulated	CDKN2B-AS1
up-regulated	CRNDE	up-regulated	circ8924
up-regulated	CRNDE	up-regulated	circ_0000263
up-regulated	CRNDE	up-regulated	circ_0000285
up-regulated	CSMARCA5	up-regulated	circ_0000388
up-regulated	DANCR	up-regulated	circ_0000515
up-regulated	DANCR	up-regulated	circ_0000745
up-regulated	DDN-AS1	up-regulated	circ_0001038
down-regulated	DGCR5	up-regulated	circ_000284
up-regulated	DLEU1	up-regulated	circ_0005576
up-regulated	DLG1-AS1	differential expressed	circ_000596
up-regulated	DLX6-AS1	up-regulated	circ_0007534
up-regulated	DLX6-AS1	up-regulated	circ_0018289
up-regulated	DLX6-AS1	up-regulated	circ_0023404
up-regulated	DSCAM-AS1	up-regulated	circ_0031288
up-regulated	ENST00000413430	up-regulated	circ_0067934
up-regulated	ENST00000420168	up-regulated	circ_0075341
up-regulated	ENST00000430751.1	differential expressed	circ_101958
down-regulated	ENST00000447565	up-regulated	circ_101996
up-regulated	ENST00000456944	up-regulated	ENST00000551152
up-regulated	ENST00000474768	up-regulated	ENST00000554055.1
up-regulated	ENST00000503812	up-regulated	ENST00000564977
up-regulated	ENST00000531193	up-regulated	ENST00000570282

up-regulated	ENST00000531702.1	up-regulated	ENST00000572284.1
differential expression	HOTAIR	up-regulated	FALEC
down-regulated	HOTAIR	up-regulated	FAM83H-AS1
up-regulated	HOTAIR	up-regulated	FEZF1-AS1
up-regulated	HOTAIR	up-regulated	FEZF1-AS1
up-regulated	HOTAIR	up-regulated	FOXD2-AS1
up-regulated	HOTAIR	up-regulated	FOXD2-AS1
up-regulated	HOTAIR	up-regulated	FOXP4-AS1
up-regulated	HOTAIR	up-regulated	FTH1P3
down-regulated	HOTAIR	down-regulated	GAS5
down-regulated	HOTAIR	down-regulated	GAS5
differential expression	HOTAIR	down-regulated	GAS5
up-regulated	HOTAIR	down-regulated	GAS5
up-regulated	HOXA11-AS	up-regulated	GAS5
up-regulated	HOXD-AS1	down-regulated	GAS5
up-regulated	HOXD-AS1	down-regulated	GAS5
up-regulated	HOXD-AS1	down-regulated	GAS5-AS1
up-regulated	HULC	up-regulated	GHET1
up-regulated	HULC	up-regulated	GHET1
up-regulated	LINC-POU3F3	up-regulated	GIHCG
up-regulated	LINC-ROR	up-regulated	GPC3-AS1
up-regulated	LINC-UFC1	down-regulated	H19
up-regulated	LINC-UFC1	up-regulated	H19
down-regulated	LINC00037	down-regulated	H19
down-regulated	LINC00052	down-regulated	H19
up-regulated	LINC00152	down-regulated	HAND2-AS1
up-regulated	LINC00152	down-regulated	HAND2-AS1
down-regulated	LINC00277	up-regulated	HCP5
up-regulated	LINC00319	up-regulated	HIPK1-AS
up-regulated	LINC00473	up-regulated	HNF1A-AS1
up-regulated	LINC00483	up-regulated	HOST2
up-regulated	LINC00511	up-regulated	HOTAIR
up-regulated	LINC00511	up-regulated	HOTAIR
up-regulated	LINC00511	up-regulated	HOTAIR
up-regulated	LINC00518	NA	HOTAIR
up-regulated	LINC00675	up-regulated	HOTAIR
up-regulated	LINC00958	up-regulated	HOTAIR
up-regulated	LINC00958	up-regulated	HOTAIR
down-regulated	LINC01101	up-regulated	HOTAIR
up-regulated	LINC01133	up-regulated	HOTAIR
differential expression	LINC01139	up-regulated	HOTAIR
up-regulated	LINC01305	up-regulated	HOTAIR
differential expression	LINC01503	differential expression	HOTAIR
up-regulated	LINC01535	up-regulated	HOTAIR
up-regulated	LINC01783	up-regulated	HOTAIR
up-regulated	LINCRNA-p21	down-regulated	HOTAIR
up-regulated	PVT1	up-regulated	LINCRNA-p21
up-regulated	PVT1	up-regulated	LINP1
up-regulated	PVT1	up-regulated	LINP1
up-regulated	PVT1	up-regulated	lnc-CC3
up-regulated	PVT1	down-regulated	lnc-CCDST
up-regulated	PVT1	up-regulated	lnc-LIF-AS
up-regulated	PVT1	up-regulated	lncRNA-ATB
up-regulated	PVT1	up-regulated	lncRNA-ATB
up-regulated	RHPN1-AS1	up-regulated	lncRNA-CTS
up-regulated	ROR1-AS1	up-regulated	lncRNA-EBIC
up-regulated	RP1-93H18.6	down-regulated	lncRNA-LET
down-regulated	RP11-381N20.2	differential expression	lncRNA-MIF
down-regulated	RP11-381N20.2	up-regulated	lncRNA799
up-regulated	RP11-396F22.1	up-regulated	LOC105374902
up-regulated	RP11-480I12.5	up-regulated	LOC554202
up-regulated	RP11-552M11.4	up-regulated	LUCAT1
up-regulated	Rs11655237	up-regulated	LUCAT1
up-regulated	RSU1P2	up-regulated	LUCAT1
up-regulated	SBF2-AS1	up-regulated	MALAT1
up-regulated	SNHG1	up-regulated	MALAT1
up-regulated	SNHG12	up-regulated	MALAT1

up-regulated	SNHG12	up-regulated	MALAT1
up-regulated	SNHG14	up-regulated	MALAT1
up-regulated	SNHG14	differential expression	MALAT1
up-regulated	SNHG16	up-regulated	MALAT1
up-regulated	SNHG16	up-regulated	MALAT1
up-regulated	SNHG20	down-regulated	MALAT1
up-regulated	SNHG4	down-regulated	MALAT1
up-regulated	SNHG7	up-regulated	MALAT1
up-regulated	SNHG7	down-regulated	MALAT1
up-regulated	SNHG7	up-regulated	MALAT1
up-regulated	SNHG8	differential expression	MALAT1
up-regulated	SOX21-AS1	up-regulated	MALAT1
differential expressed	SOX2OT	up-regulated	MALAT1
up-regulated	SOX2OT	up-regulated	MALAT1
up-regulated	SPRY4-IT1	up-regulated	MALAT1
up-regulated	SPRY4-IT1	down-regulated	MEG3
up-regulated	SRA	down-regulated	MEG3
down-regulated	STXBP5-AS1	down-regulated	MEG3
down-regulated	TCONS_00001368	down-regulated	MEG3
down-regulated	TCONS_00010232	up-regulated	MEG3
up-regulated	TCONS_00026907	down-regulated	MEG3
up-regulated	TDRG1	down-regulated	MEG3
up-regulated	TDRG1	down-regulated	MEG3
up-regulated	TDRG1	down-regulated	MEG3
down-regulated	TI09485	up-regulated	MFI2
up-regulated	TI10124	up-regulated	MIAT
up-regulated	TI13831	differential expression	MIR100HG
up-regulated	TI18318	up-regulated	MIR205HG
down-regulated	TI21327	up-regulated	MIR205HG
down-regulated	TI22687	up-regulated	MIR210HG
up-regulated	TMPO-AS1	down-regulated	MIR22HG
up-regulated	TMPO-AS1	up-regulated	MNX1-AS1
up-regulated	TMPOP2	up-regulated	NCK1-AS1
down-regulated	TOB1-AS1	up-regulated	NCK1-AS1
up-regulated	TP73-AS1	up-regulated	NCK1-AS1
up-regulated	TP73-AS1	up-regulated	ncRNA-CCND1
up-regulated	TPT1-AS1	up-regulated	NEAT1
up-regulated	TTN-AS1	up-regulated	NEAT1
up-regulated	TUG1	up-regulated	NEAT1
up-regulated	TUG1	up-regulated	NEAT1
up-regulated	TUG1	up-regulated	NEAT1
down-regulated	TUSC8	up-regulated	NEAT1
up-regulated	uc021zkp.1	up-regulated	NEAT1
up-regulated	UCA1	up-regulated	NNT-AS1
up-regulated	UCA1	up-regulated	NOC2L-4.1
up-regulated	UCA1	up-regulated	NORAD
up-regulated	UCA1	up-regulated	NR_003679.1
up-regulated	UCA1	up-regulated	NR_024008.1
up-regulated	UCA1	up-regulated	NR_034077.1
up-regulated	UCA1	up-regulated	NR_037645.1
up-regulated	UICC	up-regulated	NR_037793.1
down-regulated	WT1-AS	up-regulated	NR_038940.1
down-regulated	WT1-AS	up-regulated	NR_047677.1
down-regulated	WT1-AS	up-regulated	OGFRP1
up-regulated	XIST	up-regulated	OIP5-AS1
up-regulated	XIST	up-regulated	OIP5-AS1
up-regulated	XIST	up-regulated	OIP5-AS1
up-regulated	XLOC_000303	differential expression	OIP5-AS1
up-regulated	XLOC_006390	up-regulated	PANDAR
up-regulated	XLOC_006390	up-regulated	PARROT
up-regulated	XLOC_008466	down-regulated	PCAT1
down-regulated	XLOC_010588	up-regulated	PCAT6
down-regulated	XLOC_011152	up-regulated	PCAT6
up-regulated	ZEB1-AS1	up-regulated	PCGEM1
up-regulated	ZEB1-AS1	down-regulated	PTCSC3
up-regulated	ZFAS1	down-regulated	PTCSC3
		down-regulated	ZNF667-AS1

Table2. lncRNA involved in cervical intraepithelial neoplasia retrieved from LncRNA2Cancer databas

Expression pattern	lncRNA
up-regulated	**lnc-IL7R**

Table3. List of lncRNA/circRNA involved in cervical squamous cell carcinoma retrieved from LncRNA2Cancer database

Expression pattern	LncRNA/circRNA	Expression pattern	LncRNA/circRNA
up-regulated	GATA6-AS	up-regulated	BLACAT1
down-regulated	HAND2-AS1	up-regulated	CCAT2
down-regulated	lncRNA-NEF	up-regulated	circ_0101119
down-regulated	lncRNA-SRA1	up-regulated	circ_0101996
up-regulated	loc285194	up-regulated	FAL1
down-regulated	NR2F1-AS1	up-regulated	MACC1-AS1
up-regulated	PVT1	up-regulated	MAGI2-AS3
up-regulated	SNHG12	down-regulated	MCM3AP-AS1
up-regulated	TINCR	down-regulated	MIR503HG
up-regulated	TINCR	down-regulated	NKILA
		down-regulated	WT1-AS

Discussion

MALAT1 was retrieved from lncCeRBase, CLING, LncTarD, Lnc2Cancer, LncRNADisease, LncRNAWiki, GeneCards databases. The results from the LncTarD and Lnc2 Cancer databases show the increased pattern of expression, and the LncRNAWiki database shows the increased and differentially expression pattern. Liang and his colleagues reported in 2021 that MALAT1 increased the proliferation of cervical tumor cells by its potential target, miR-124, thus increasing its expression in cervical cancer[19]. In fact, they found that MALAT1 inhibited the expression of miR-124 and lead to progression of cervical carcinoma Also, MALAT1 suppresses apoptosis and promotes cell proliferation and cell cycle regulation in cervical cancer by sponging miR-145[20]. MALAT1 has been identified as a potential target for improving clinical effects in cervical carcinoma progression[21]. Jiang and colleagues reported in 2014 that MALAT1 is not expressed in normal cells but is expressed in cervical cancer[22]. Sun and colleagues reported in 2016 that the expression level of MALAT1 was significantly increased in cervical cancer cells and tissues and promoted cervical cancer invasion and metastasis through the induction of EMT [23]. In 2021, Wang and colleagues reported that MALAT1 is highly expressed in cervical cancer cells and is associated with cervical cancer metastasis. They also showed that the viral E7 oncoprotein targets the MALAT1 promoter and induces MALAT1 transcription[24]. Liu and colleagues reported in 2016 that MALAT1 can sponge with miR-124 and increase GRB2, which is a target gene of miR-124, thereby increasing the growth and invasion of cervical cancer cells. Also, MALAT1 interacts with miR-375, and miR-375 can target the E6 gene of HPV16 and inhibit its translation, and on the other hand, E6 of HPV16 increases the expression of MALAT1 in cervical cancer cells[25]. Xia and colleagues reported in 2018 that the expression of MALAT1 is increased in cervical cancer and that miR-142-3p inhibits cell proliferation and invasion of cervical cancer cells by targeting FZD7 in cervical cancer, and the expression of miR- 142-3p has decreased expression in cervical cancer, also MALAT1 acts as a sponge with miR-142-3p

and miR-142-3p decreases and cancer cells proliferate[26]. According to these studies, the results are consistent with the data retrieved from the databases. CDKN2B is another important lncRNA in cervical cancer, which is retrieved from Lnc2Cancer databases[27]. In 2016, Zhang and colleagues reported that CDKN2B-AS1 contributes to tumor progression in a variety of cancers. According to their findings, CDKN2B-AS1 is increased in cervical cancer cell lines and tissues after inhibiting CDKN2B-AS1. The PI3K/AKT pathway is inactivated. By inhibiting CDKN2B-AS1, the phosphorylated levels of PI3K and AKT significantly decrease in cervical cancer, which indicates that the PI3K/AKT pathway is involved in the proliferation and metastasis of cervical cancer cells caused by CDKN2B-AS1. The PI3K/AKT pathway in proliferation and EMT is an essential intracellular signaling pathway that plays a role during cancer processes. By removing CDKN2B-AS1 in laboratory conditions, cervical cancer metastasis is prevented and leads to inactivation of the PI3K/AKT pathway[28]. These studies are consistent with the data retrieved from the databases. These were only a few examples of the types of lncRNAs that have been collected by bioinformatics databases and it is possible to design new treatment processes for cervical cancer by identifying them. In cervical cancer, apart from lncRNAs and miRNAs, other RNAs have been proven to exist, and researches on them are very limited, including snoRNAs, piRNAs, tRNAs, circRNAs, etc. in cervical cancer. It is suggested that more research should be done on them to use them in the treatment, diagnosis and prognosis of cancer. Some examples of circRNAs predicted in cervical cancer include circ_103519, circ_103973, circ_104315, circ_400068, circAGFG1, circAKT1, circAMOTL.

Conclusion

In summary, the diverse lncRNAs cataloged within bioinformatics databases represent a rich and rapidly expanding resource for understanding cervical cancer. While only a select few examples have been highlighted here, their documented roles in tumor progression, metastasis, and therapy resistance underscore a significant opportunity. The systematic identification and functional characterization of these molecules is a critical step toward a new frontier in oncology. By leveraging this knowledge, the future holds promising potential for designing novel, targeted treatment strategies that move beyond conventional approaches, ultimately paving the way for more precise and effective interventions against cervical cancer.

Acknowledgments

Not declared.

Authorship

All authors contributed to the study's conception and design, conducted the literature review, and participated in drafting and critically revising the manuscript. All authors reviewed and approved the final version for submission.

Conflicts of interest

No potential conflict of interest relevant to this article was reported.

Funding

The authors received no financial support for this article's research, authorship, and publication.

References

1. Ahmadi M, Rasi H, Mostafazadeh M, Hajazimian S, Maroufi NF, Nahaei MR, et al. Analysis of cervical lesions for presence of HSV-2 and HPV-16 and HPV-18 in Iranian patients by PCR. Horm Mol Biol Clin Investig. 2017;31(3): 20170019.

2. Maroufi NF, Vahedian V, Hemati S, Rashidi M-R, Akbarzadeh M, Zahedi M, et al. Targeting cancer stem cells by melatonin: Effective therapy for cancer treatment. Pathol Res Pract. 2020;216(5):152919.

3. Maroufi NF, Rashidi MR, Vahedian V, Akbarzadeh M, Fattahi A, Nouri M. Therapeutic potentials of Apatinib in cancer treatment:

Possible mechanisms and clinical relevance. Life Sci. 2020;241:117106.

4. Wang X, Huang X, Zhang Y. Involvement of human papillomaviruses in cervical cancer. Front Microbiol. 2018;9:2896.

5. Schettino M, Mainini G, Ammaturo F, De Franciscis P, Torella M, Grauso F, et al. The role of human papillomavirus in cervical pre-neoplastic lesions: the relationship between virus genotype and persistence or clearance of the infection. Eur J Gynaecol Oncol. 2018;39(4):564-8.

6. Yuan Y, Cai X, Shen F, Ma F. HPV post-infection microenvironment and cervical cancer. Cancer Lett. 2021;497:243-54.

7. Sun Q, Wang L, Zhang C, Hong Z, Han Z. Cervical cancer heterogeneity: a constant battle against viruses and drugs. Biomark Res. 2022;10(1):85.

8. Akbarzadeh M, Maroufi NF, Tazehkand AP, Akbarzadeh M, Bastani S, Safdari R, et al. Current approaches in identification and isolation of cancer stem cells. J Cell Physiol. 2019;234(9):14759-72.

9. Liu J, Li Z, Lu T, Pan J, Li L, Song Y, et al. Genomic landscape, immune characteristics and prognostic mutation signature of cervical cancer in China. BMC Med Genomics. 2022;15(1):231.

10.Mattick JS, Amaral PP, Carninci P, Carpenter S, Chang HY, Chen L-L, et al. Long non-coding RNAs: definitions, functions, challenges and recommendations. Nat Rev Mol Cell Biol. 2023;24(6):430-47.

11.Ladomery M, Pisignano G, editors. Post-transcriptional Regulation through Long Non-coding RNAs (lncRNAs). Basel: MDPI; 2021.

12.Sebastian-delaCruz M, Gonzalez-Moro I, Olazagoitia-Garmendia A, Castellanos-Rubio A, Santin I. The role of lncRNAs in gene expression regulation through mRNA stabilization. Non-coding RNA. 2021;7(1):3.

13.Gil N, Ulitsky I. Regulation of gene expression by cis-acting long non-coding RNAs. Nat Rev Genet. 2020;21(2):102-17.

14.García-Padilla C, Dueñas Á, García-López V, Aránega A, Franco D, Garcia-Martínez V, et al. Molecular mechanisms of lncRNAs in the dependent regulation of cancer and their potential therapeutic use. Int J Mol Sci. 2022;23(2):764.

15.Statello L, Guo C-J, Chen L-L, Huarte M. Gene regulation by long non-coding RNAs and its biological functions. Nat Rev Mol Cell Biol. 2021;22(2):96-118.

16.Ahadi A. Functional roles of lncRNAs in the pathogenesis and progression of cancer. Genes Dis. 2021;8(4):424-37.

17.Taefehshokr S, Taefehshokr N, Derakhshani A, Baghbanzadeh A, Astamal RV, Safaei S, et al. The regulatory role of pivotal microRNAs in the AKT signaling pathway in breast cancer. Curr Mol Med. 2022;22(3):263-73.

18.Gao Y, Shang S, Guo S, Li X, Zhou H, Liu H, et al. Lnc2Cancer 3.0: an updated resource for experimentally supported lncRNA/circRNA cancer associations and web tools based on RNA-seq and scRNA-seq data. Nucleic Acids Res. 2021;49(D1):D1251-D8.

19.Liang T, Wang Y, Jiao Y, Cong S, Jiang X, Dong L, et al. LncRNA MALAT1 Accelerates Cervical Carcinoma Proliferation by Suppressing miR-124 Expression in Cervical Tumor Cells. J Oncol. 2021;2021:8836078.

20.Lu H, He Y, Lin L, Qi Z, Ma L, Li L, et al. Long non-coding RNA MALAT1 modulates radiosensitivity of HR-HPV+ cervical cancer via sponging miR-145. Tumour Biol. 2016;37:1683-91.

21.Qiao F-H, Tu M, Liu H-Y. Role of MALAT1 in gynecological cancers: Pathologic and therapeutic aspects. Oncol Lett. 2021;21(4):316.

22.Jiang Y, Li Y, Fang S, Jiang B, Qin C, Xie P, et al. The role of MALAT1 correlates with HPV in cervical cancer. Oncol Lett. 2014;7(6):2135-41.

23.Sun R, Qin C, Jiang B, Fang S, Pan X, Peng L, et al. Down-regulation of MALAT1 inhibits cervical cancer cell invasion and metastasis by inhibition of epithelial–mesenchymal transition. Mol Biosyst. 2016;12(3):952-62.

24.Wang T, Zhang W, Huang W, Hua Z, Li S. LncRNA MALAT1 was regulated by HPV16 E7 independently of pRB in cervical cancer cells. J Cancer. 2021;12(21):6344-55.

25.Liu S, Song L, Yao H, Zhang L, Xu D, Gao F, et al. MiR-375 is epigenetically downregulated by HPV-16 E6 mediated DNMT1 upregulation and modulates EMT of cervical cancer cells by suppressing lncRNA MALAT1. PLoS One. 2016;11(9):e0163460.

26.Xia C, Liang S, He Z, Zhu X, Chen R, Chen J. Metformin, a first-line drug for type 2 diabetes mellitus, disrupts the MALAT1/miR-142-3p sponge to decrease invasion and migration in cervical cancer cells. Eur J Pharmacol. 2018;830:59-67.

27.de Oliveira AL, de Almeida VD, Pereira TP, Carvalho AE, Wojcieszyn VSN, Andrade MF, et al. Role of lncRNA alterations in cervical oncogenesis. Eur J Med Oncol. 2020;4(3):162-9.

28.Zhang D, Sun G, Zhang H, Tian J, Li Y. Long non-coding RNA ANRIL indicates a poor prognosis of cervical cancer and promotes carcinogenesis via PI3K/Akt pathways. Biomed Pharmacother. 2017;85:511-6.