Pathology - Research and Practice 248 (2023) 154617

Contents lists available at ScienceDirect

Pathology - Research and Practice

journal homepage: www.elsevier.com/locate/prp

Review

Expression of LINC00174 in different cancers: Review of the literature and bioinformatics analyses

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ARTICLE INFO

Keywords:
LINC00174
LncRNA
Cancer

ABSTRACT

LINC00174 is an example of long intergenic non-coding RNAs with important functions in the development of human cancers. The gene encoding this lincRNA is located on 7q11.21. LINC00174 has been demonstrated to play an oncogenic role in a variety of cancers, including colorectal carcinoma, thymic carcinoma, glioma, glioblastoma, hepatocellular carcinoma, kidney renal clear cell carcinoma, breast cancer and non-functioning pituitary adenoma. In lung cancer, there is an obvious discrepancy between different studies regarding the role of this lincRNA. This lincRNA is also involved in the determination of prognosis of different cancers, particularly colorectal cancer. In the current review, we discuss the role of this lincRNA in human carcinogenesis based on the available data in the literature and bioinformatics tools.

1. Introduction

Long intergenic non-coding RNAs (lincRNAs) are a group of transcripts with sizes longer than 200 nucleotides, but lack of open reading frames for encoding proteins. Generally, they are transcribed by RNA polymerase II and are both spliced and poly-adenylated [4,5,7,20]. More than 15,000 lincRNAs have been identified in the human genome. As their names imply, they belong to the regions of the genome that do not comprise protein-encoding genes, or previously described as "junk" regions. However, they have functional roles in gene expression control, scaffold construction, and epigenetic mechanisms [4]. Most importantly, abnormal expression of lincRNAs can influence the occurrence and development of several cancers [18].

LINC00174 is an example of lincRNAs with important functions in the development of human cancers. The gene encoding this lincRNA is located on 7q11.21 (Fig. 1). RNAseq and microarray data have indicated expression of this lincRNA in a variety of immune, nervous, internal, secretory and reproductive tissues (https://www.genecards.org/cgi-bin/carddisp.pl?gene=LINC00174). Moreover, a bulk of evidence points to abnormal levels of LINC00174 in a variety of human cancers. In recent years, numerous studies have been conducted on the role of this gene in various diseases, including different types of cancers, revealing its significant involvement in the progression of these diseases. In the current review, we discuss the role of this lincRNA in human carcinogenesis based on the available data in the literature and bioinformatics tools.

2. Expression of LINC00174 in cancers

Expression and function of LINC00174 have been assessed in a variety of cancers, particularly colorectal and lung cancers.

3. Colorectal cancer

Shen et al. have demonstrated over-expression of LINC00174 in colorectal cancer tissues and cells compared to the corresponding control samples. Notably, Up-regulation of LINC00174 in this type of cancer has indicated poor prognosis. LINC00174 silencing could repress growth

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https://doi.org/10.1016/j.prp.2023.154617
Received 20 May 2023; Received in revised form 8 June 2023; Accepted 10 June 2023
Available online 11 June 2023

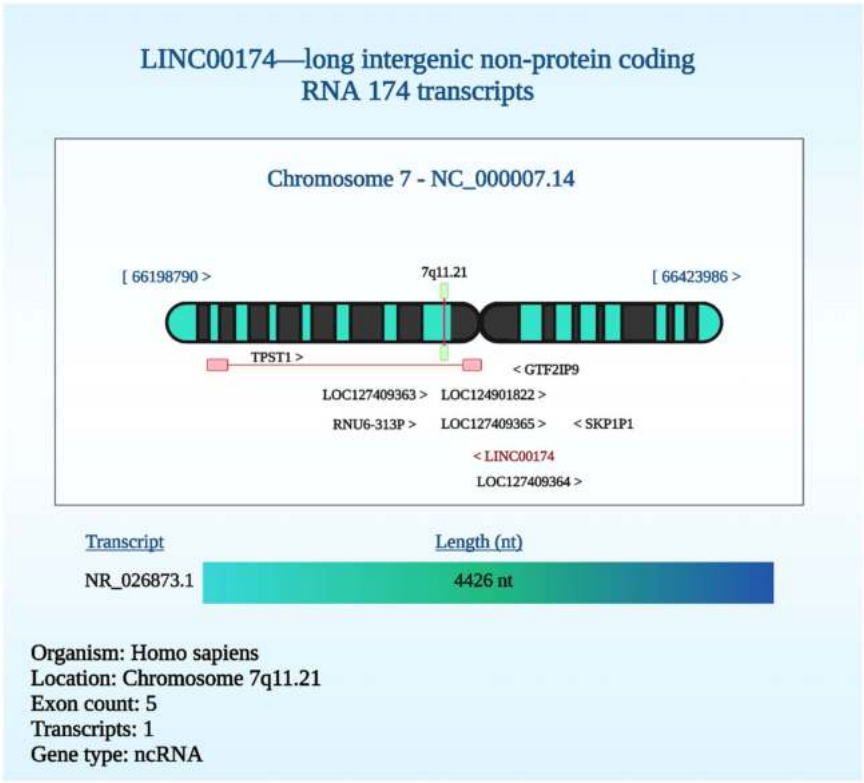

Fig. 1. Schematic representation of the LINC00174 transcript and its genomic location on chromosome 7q11.21.

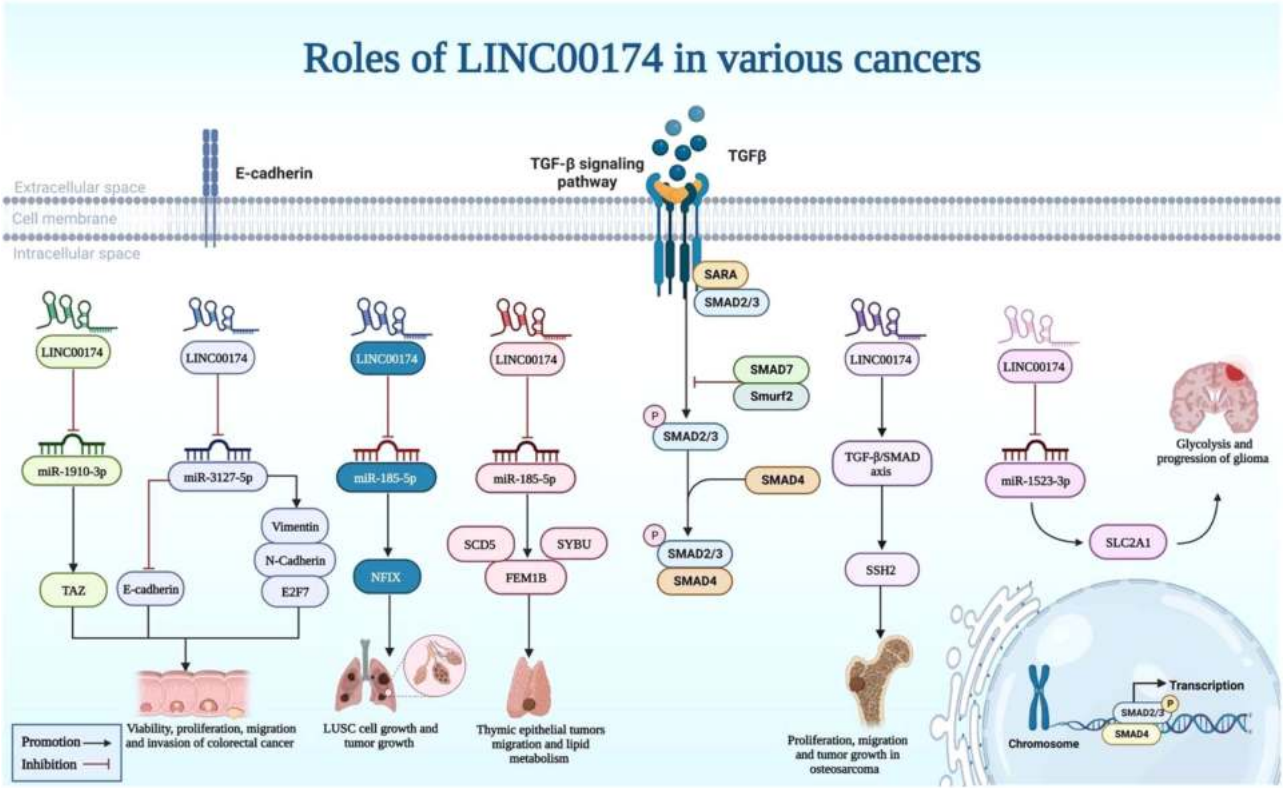

Fig. 2. Illustrates the essential functions of LINC00174 in cancer development and progression. LINC00174 serves as a crucial regulator in various cellular processes such as proliferation, migration, and invasion and leads to the promotion of cancer growth and metastasis in different types of cancer.

Table 1
Expression of LINC00174 in colorectal cancer.

Cancer Type	Expression/Role	Samples/Assessed Cell Lines	Pathways	Targets/ Regulators	Function	Ref
Colorectal Carcinoma (CRC)	Upregulated/ Oncogene	50 patients diagnosed as CRC/DLD1, HCT116, LOVO, RKO, LS174T, HCT8, HR28348, HT29, SW620, SW480, NCM460	LINC00174/miR-1910–3p/ TAZ signal pathway	miR-1910–3p/ TAZ	LINC00174 plays an oncogenic role through LINC00174/miR-1910–3p/TAZ pathway.	[21]
	Upregulated/ Oncogene	CRL-1459, CL-188	LINC00174/miR-3127–5p/ E2F7 axis	MiR-3127–5p/ E2F7	LINC00174 modified the miR-3127–5p/E2F7 axis, which in turn affected the biological properties of CRC cells. As a result, LINC00174 can be a viable treatment option for CRC.	[16]
	Upregulated/ Oncogene (Bioinformatics approach)	–	Hypoxia-related (Bioinformatics approach)	–	A reliable predictive signature for CRC, LINC00174 is a hypoxia-related lncRNA that serves as a therapeutic target and prognostic marker.	[10]
	Upregulated/ Oncogene	HT29, HCT116, RKO, NCM460	Autophagy-related (Bioinformatics approach), SRSF1/P53 signaling pathway	Mir-1910–3p/ TAZ, Mir-3127–5p/E2F7	Individuals with elevated expression of LINC00174 in their tissue samples have a bad prognosis. Via controlling the miR-1910–3p/ TAZ and miR-3127–5p/E2F7 pathways, LINC00174 upregulation encourages the growth and motility of CRC cells.	[26]

Table 2
Expression of LINC00174 in the different types of lung cancer.

Cancer Type	Expression/Role	Samples/Assessed Cell Lines	Pathways	Targets/ Regulators	Function	Ref
Lung Squamous Cell Carcinoma (LUSC)	Upregulated/ Oncogene	SK-MES-1, NCI-H226, SW900, NCI-H520, BEAS-2B	LINC00174/miR-185–5p/NFIX	miR-185–5p/ NFIX	By acting as a ceRNA to sponge miR-185–5p and eventually stimulate NFIX, LINC00174 accelerated the formation of LUSC and provided a viable target for LUSC treatment.	[8]
Lung Cancer (LC)	Upregulated/ Oncogene	–	LINC00174/miR-584–3p/ RPS24 axis	miR-584–3p/ RPS24	LINC00174 became tethered to the RPS24-specific miR-584–3p. Through altering the miR-584–3p/RPS24 axis, repressed LINC00174 can reduce the malignant phenotypes of LC cells.	[24]
Non-Small Cell Lung Cancer (NSCLC)	Downregulated/ Tumor suppressor gene	38 pairs of NSCLC tissues and adjacent tissues/95-D, H1299, A549, HBE	LINC00174/ miR-31–5p/LATS2 axis	MiR-31–5p/ LATS2	As a competitive endogenous RNA, LINC00174 increases the production of LATS2 and binds to miR-31–5p, which lowers the motility of NSCLC cells and encourages cell death.	[3]

of colorectal cancer cells in vitro and in animal models. Expression of LINC00174 has been induced by the transcription factor STAT1. Mechanistically, LINC00174 contributes to the progression of colorectal cancer through regulation of the miR-1910–3p/TAZ axis [21] (Fig. 2). Similarly, Ma et al. have shown up-regulation of LINC00174 in colorectal cancer clinical samples and cells in relation with the clinical features of this cancer. Over-expression of LINC00174 has been able to promote viability, proliferation, migration and invasion of colorectal cancer cells. Moreover, this lincRNA could up-regulate expression levels of N-Cadherin, Vimentin, E2F7, while inhibiting E-Cadherin expression. Additional studies have shown that LINC00174 affects the biological features of colorectal cancer cells through regulation of miR-3127–5p/ E2F7 axis [16]. Finally, assessment of expression profiles of colorectal cancer cases and further estimation of the hypoxia score using a single-sample gene set enrichment analysis algorithm have led to identification of LINC00174 among other prognostic lincRNAs in this type of cancer [10]. The expression of LINC00174 in colorectal cancer has been presented in Table 1.

4. Lung cancer

Gu et al. have shown high levels of LINC00174 expression in lung squamous cell carcinoma cells. LINC00174 silencing could confine proliferation, migration, and invasion of these cells while enhancing their apoptosis. Mechanical studies have revealed that LINC00174 interacts with miR-185–5p to increase levels of nuclear factor IX, which is directly targeted by miR-185–5p. Remarkably, NFIX up-regulation can rescue the suppressive effects of LINC00174 silencing on malignant behavior of lung squamous cell carcinoma cells. Therefore, LINC00174 enhances progression of this type of lung cancer through serving as a molecular sponge for miR-185–5p and subsequent up-regulation of NFIX [8] (Fig. 2). Similar to this study, Wang et al. have shown that down-regulation of LINC00174 suppresses malignant behavior of lung cancer cells through regulation of miR-584–3p/S24 axis [24]. On the other hand, Cheng et al. have shown down-regulation of LINC00174 in non small cell lung cancer tissues and cell lines compared with controls. LINC00174 levels have been negatively associated with the TNM stage. Functionally, LINC00174 up-regulation could inhibit multiplication and migration of these cells and induce apoptosis. These effects have been shown to be mediated through adsorbing miR-31–5p, thereby increasing LATS2 expression [3]. The expression of LINC00174 in the different types of lung cancer has been shown in Table 2.

5. Other types of cancers

LINC00174 has been demonstrated to play an oncogenic role in a variety of cancers, including thymic carcinoma, glioma, glioblastoma, hepatocellular carcinoma, kidney renal clear cell carcinoma, breast cancer and non-functioning pituitary adenoma.

LINC00174 has been shown to be up-regulated in thymic epithelial

Table 3
Expression of LINC00174 in other different types of cancers.

Cancer Type	Expression/Role	Samples/Assessed Cell Lines	Pathways	Targets/Regulators	Function	Ref
Thymic Carcinoma (TC)	Upregulated/Oncogene	4 thymomas of different histotypes and 2 thymi/ TC1889	LINC00174/miR-145–5p/SCD5 axis	miR-145–5p/ UBAC1, SYBU, FEM1B, and SCD5	SCD5, a crucial regulator of lipid metabolism, enhances the capacity of TET cells to migrate, establishing a connection between lipids and motility and identifying both pathways as pertinent targets for the creation of innovative treatment strategies for TET.	[23]
Glioma	Upregulated/Oncogene	45 paired brain glioma specimens and peritumoral brain edema (PTBE) tissues/ U251, LN229, H4, SW1783, A172, HEK-293 T	miR-152–3p/SLC2A1 pathway	miR-152–3p/ SLC2A1	By boosting the production of SLC2A1 and sponging miR-1523–3p, LINC00174 hastened the tumorigenesis of gliomas and may be used as a target for the diagnostics of gliomas.	[22]
	Upregulated/Oncogene	40 human glioma tissues (tumor) and adjacent control brain tissues (normal)/NHA, LN229, SHG-44, U118, U251, U87	LINC00174/miR-138–5p/SOX9 axis	miR-138–5p/SOX9	A freshly established regulatory mechanism of the LINC00174/miR-138–5p/SOX9 axis was thoroughly examined, and it may offer a fresh approach to the treatment of gliomas.	[12]
Glioma Endothelial Cells (GEC)	Upregulated/Oncogene	hCMEC/D3; ECs, U87MG, HEK293T	linc00174/miR-138–5p (miR-150–5p)/FOSL2 Feedback Loop	miR-138–5p and miR-150–5p/FOSL2	The endogenous expression of linc00174, miR-138–5p, miR-150–5p, and FOSL2 in glioma microvascular endothelium as well as their impact on BTB (Blood-tumor barrier) permeability were confirmed. The objective of this work is to identify a novel mechanism controlling BTB permeability and to provide novel therapeutic approaches for the treatment of gliomas as a whole.	[9]
Low-grade Glioma (LGG)	Upregulated in the high-risk group/ Oncogene (Bioinformatics approach)	–	Immune-associated (Bioinformatics approach)	–	As an immune-related lncRNA, LINC00174 may have predictive significance in low-grade gliomas, open up new research avenues, and provide management guidance.	[17]
Glioblastoma (GBM)	Upregulated/Oncogene	U87, U251	RNA transport 192, RNA degradation (Bioinformatics approach)	–	Suppression of LINC00174 production greatly diminished the growth of GBM cells. LINC00174 functions as an oncogene and could be a marker for cancer treatment.	[25]
Hepatocellular Carcinoma (HCC)	Upregulated/Oncogene	Hep3B, Huh7	LINC00174/miR-320/S100A10 axis	miR-320/S100A10	In HCC, LINC00174 acts as a sponge for miR-320 and raises the oncogene S100A10 expression level. This research reveals the function of LINC00174 in human illness and sheds light on the process of HCC carcinogenesis and development.	[28]
	Upregulated/Oncogene	Huh7, Huh7-H	ERK-JNK/c-JUN/ MYCBP/c-MYC signaling pathway	c-JUN	By facilitating EMT (epithelial-mesenchymal transition) via ERK-JNK/c-JUN/MYCBP/c-MYC signaling, exosomal LINC00174 offers a novel treatment option for HCC patients who, regrettably, got inadequate RFA.	[15]
Laryngeal Papilloma (LP)	Upregulated/Oncogene	–	LINC00174/miR-4500/BZW2 axis	miR-4500/BZW2	The modulation of the LINC00174/ miR-4500/BZW2 axis may be a useful method for treating laryngeal papillomatosis caused by the human papillomavirus.	[14]
Kidney Renal Clear Cell Carcinoma (KIRC)	Upregulated/Oncogene	42 cases of KIRC tissues	LINC00174/miR-612/FOXM1 axis	MiR-612/FOXM1	Through controlling the miR-612/ FOXM1 axis, LINC00174 promotes the metastatic ability of KIRC cells. As a result, this lncRNA could be a fresh marker for diagnosis of KIRC.	[13]
Breast Cancer (BCa)	Upregulated/Oncogene	64 paired tumor tissues and paracancerous tissues/MDA-MB-231, MDA-MB-453, MCF-7, SKBR3, ZR-75–30, MCF-10A	–	MiR1827	In BCa samples, LINC00174 is elevated. It is intimately related to the prognosis in BCa. BCa malignant growth is hastened by LINC00174, which adversely regulates the amount of miR1827.	[27]
Non-Functioning Pituitary Adenoma (NFPA)	Upregulated/Oncogene (Bioinformatics approach)	–	–	Has-let-7a-1 (Bioinformatics approach)	Being the regulator of Has-let-7a-1 and an lncRNA in the ceRNA network, LINC00174 could be one of the possible treatment options for NFPA.	[6]

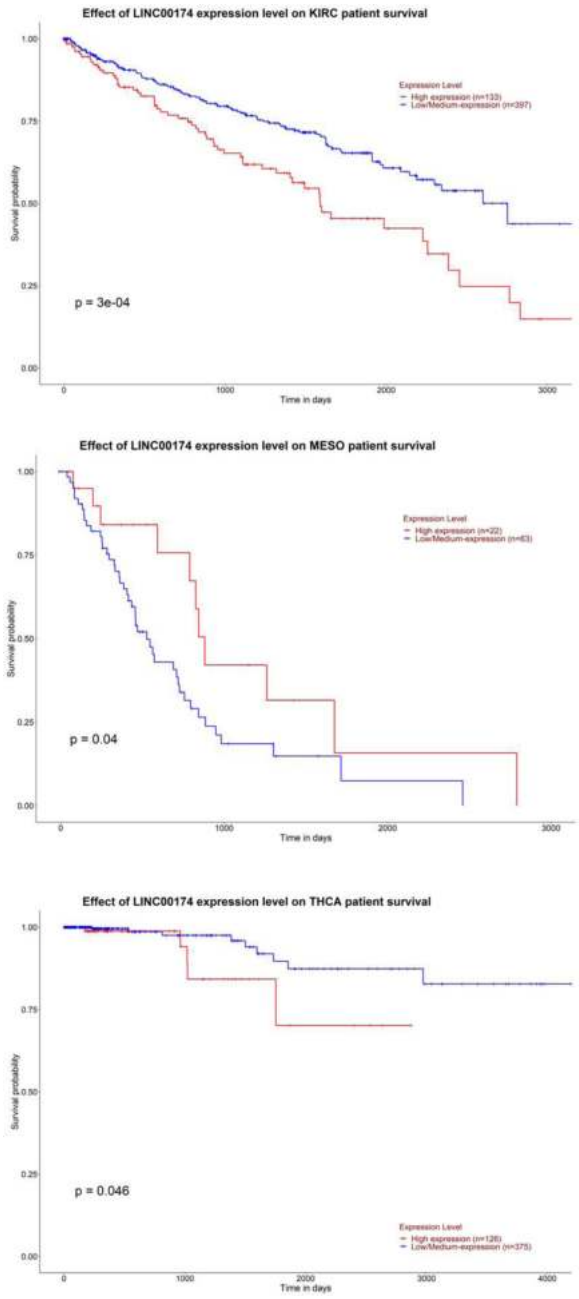

Fig. 3. LINC00174 survival analysis in malignancies with a significant p-value for the survival rate using TCGA datasets.

tumors. Notably, LINC00174 expression has been positively correlated with a 5-genes signature in these samples, in association with patients' prognosis. Mechanistically, LINC00174 increases expressions of SYBU, FEM1B, and SCD5 through adsorbing the tumor suppressor miRNA miR-145–5p. LINC00174 can also regulate migration and lipid metabolism [23].

This lincRNA can promote glycolysis and progression of glioma through regulation of miR-152–3p/SLC2A1 axis [22]. Moreover, it has a role in the determination of resistance to temozolomide through regulation of miR-138–5p/SOX9 axis [12]. Table 3 summarizes the impact of LINC00174 onprogression of other different types of cancers.

Table 4
Statistical significance of LINC00174 expression based on sample types in various cancers.

Cancer	Statistical significance of expression value*
BLCA	7.02234607788926E-07
BRCA	2.04388391291937E-06
CESC*	7.312763E-03
CHOL*	1.804917E-04
COAD	2.542198428286O1E-26
ESCA	2.220127E-03
GBM*	6.431244E-03
HNSC	1.83283946303052E-50
KIRC	6.4543697485498E-51
KIRP	4.96688509211345E-15
LIHC	4.44850038244708E-14
LUAD	1.31105965512479E-19
LUSC	1.68554751391523E-23
PRAD	7.23229695115504E-11
READ	1.55650370368292E-10
SARC*	1.08471784568946E-18
STAD	2.43015730990789E-18
THCA	1.481770E-04
LCEC	7.662646E-03

* Low number (<10) of normal samples considered.

6. Bioinformatics studies

Based on low- or high-throughput transcription factor functional studies from the CHEA Transcription Factor Targets dataset, ad ditional research by the Hormonizome database [19] revealed that 7 transcription factors (ELF1, GATA1, SPI1, STAT3, TCF4, TP63, and YY1) may bind to the promoter of LINC00174 gene.

LINC00174 is expected to be involved in three KEGG pathways, including glycerophospholipid metabolism (z-score = 2.239), notch signaling pathway (z-score = 2.097), and fanconi anemia pathway (z-score = 2.065), according to the lncHUB database (https://maayanlab.cloud/lnchub/). Additionally, LINC00511 can be associated with gene ontology (GO) entries, including regulation of histone deacetylation (GO:0031063), cochlea morphogenesis (GO:0090103), regulation of ribonucleoprotein complex localization (GO:2000197), regulation of mRNA export from nucleus (GO:0010793), regulation of histone modification (GO:0031056), positive regulation of histone deacetylation (GO:0031065), intrinsic apoptotic signaling pathway by p53 class mediator (GO:0072332), odontogenesis of dentin-containing tooth (GO:0042475), respiratory tube development (GO:0030323) and activation of GTPase activity (GO:0090630).

Using the ualcan database [2], we looked at the impact that LINC00511 had on cancer survival rates. We also used TCGA data for survival analysis. The difference had a log-rank P value below 0.05, indicating that it was statistically significant. The survival rate of individuals with thyroid cancer (THCA), kidney renal clear cell carcinoma (KIRC), and mesothelioma (MESO) is therefore impacted by LINC00511 (Fig. 3). Additionally, we investigated LINC00174 expression in different cancers. With p value less than 0.05, we found that LINC00174 was up-regulated in bladder urothelial carcinoma (BLCA), breast invasive carcinoma (BRCA), cervical squamous cell carcinoma (CESC), cholangiocarcinoma (CHOL), colon adenocarcinoma (COAD), esophageal carcinoma (ESCA), glioblastoma multiforme (GBM), head and neck squamous cell carcinoma (HNSC), kidney renal clear cell carcinoma (KIRC), kidney renal papillary cell carcinoma (KIRP), uterine corpus endometrial carcinoma (LCEC), liver hepatocellular carcinoma (LIHC), lung adenocarcinoma (LUAD), lung squamous cell carcinoma (LUSC), rectum adenocarcinoma (READ), sarcoma (SARC) and stomach adenocarcinoma (STAD) and was downregulated in thyroid carcinoma (THCA) Table 4 (Fig. 4).

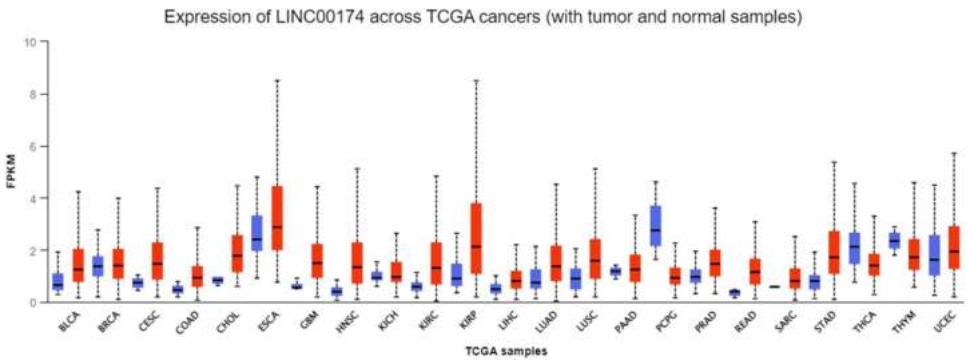

Fig. 4. Expression of LINC00174 in tumor samples compared to normal samples in different cancers based on TCGA datasets.

Table 5
Cell lines containing the highest LINC00174 copy number in different cancers.

Cancer	Cell line	Copy number
Chondrosarcoma	OUMS27	7
Invasive breast carcinoma	BT20	7
Diffuse glioma	LN382	7
Esophagogastric adenocarcinoma	NUGC2	7
Hepatocellular carcinoma	SNU423	7
Non-small cell lung cancer	LC1SQSF	7
Melanoma	SKMEL31	7
Head and neck squamous cell carcinoma	SNU899	6
Renal cell carcinoma	UMRC6NEO	6
Acute myloid leukemia	OCIM1	6
Pancreatic neuroendocrine tumor	QGP1	6
Rhabdomyosarcoma	SJRH30	6
Endometrial carcinoma	SNU685	6
Colorectal adenocarcinoma	SNUC1	5
Non-hodgkin lymphoma	MINO	5
Ovarian epithelial tumor	OC316	5
Anaplastic thyroid cancer	BHT101	5
Ampullary carcinoma	SNU478	4
Biliary tract	SNU245	4
Bladder urothelial carcinoma	5637	4
Neuroblastoma	KPNSI9S	4
Pleural mesothelioma	NCIH28	4
Prostate small cell carcinoma	NCIH660	4

Based on RNAInter (RNA Interactome Database) [11], LINC00174 has interactions with hsa-miR-1910–3p, hsa-miR-106a-3p, hcmv-miR-US25–1–5p, hsa-miR-1–3p, hsa-miR-335–5p, hsa-miR-106a-5p, RNF157-AS1, AC010913.1, CISTR, RXRB, AR and U1 snRNA with score ≥ 0.1.

Based on depmap database (https://depmap.org/portal/), we examined the copy number of LINC00174 in different cell lines and summarized the cell lines that contain the highest number of copies of this lncRNA in Table 5.

We explored LINC00174 alterations in different cancer based on ICGC/TCGA datasets using cBioPortal database [1]. As a result, we found out of a total of 2565 samples, 162 had mutations in LINC00174 (Fig. 5A) and this lncRNA has the most alterations in pancreatic neuroendocrine tumor (24 of 80 cases), melanoma (17 of 107 cases), breast invasive ductal carcinoma (16 of 180 cases), esophageal adenocarcinoma (11 of 97 cases) and hepatocellular carcinoma (9 of 315 cases) (Fig. 5B).

7. Discussion

LINC00174 has been demonstrated to play an oncogenic role in a variety of cancers, including colorectal carcinoma, thymic carcinoma, glioma, glioblastoma, hepatocellular carcinoma, kidney renal clear cell carcinoma, breast cancer and non-functioning pituitary adenoma.

Studies have demonstrated that LINC00174 exhibits upregulated expression in colorectal cancer (CRC), which consequently leads to growth and motility of CRC cells [10,16,21,26]. In lung cancer, there is an obvious discrepancy between different studies regarding the role of this lincRNA. LINC00174 exhibited a stimulating effect on the development of lung squamous cell carcinoma (LUSC), offering a promising target for therapeutic intervention in LUSC treatment. Similarly, the suppression of LINC00174 demonstrated a decrease in the malignant characteristics of lung cancer cells. Conversely, LINC00174 was found to hinder the motility of non-small cell lung cancer (NSCLC) cells while promoting cell death [3,8]. Furthermore, overexpression of this lincRNA leads to the progression of cancers such as glioma, hepatocellular carcinoma, breast cancer, thymic carcinoma, kidney renal clear cell carcinoma and non-functioning pituitary adenoma.

In addition to miRNAs that have been shown to be sponged by LINC00174 in different studies (Table 1), our *in silico* analyses have shown interactions between this lincRNA and a variety of miRNAs, including miR-1910–3p, hsa-miR-106a-3p, hcmv-miR-US25–1–5p, hsa-miR-1–3p, hsa-miR-335–5p and hsa-miR-106a-5p. Thus, several miRNA/mRNA axes are potentially affected by LINC00174.

The prognostic impact of LINC00174 has been verified in different cancers, particularly colorectal cancer [26]. Moreover, our bioinformatics analyses revealed that the survival rate of individuals with thyroid cancer (THCA), kidney renal clear cell carcinoma (KIRC), and mesothelioma (MESO) is influenced by LINC00511 expression. Thus, this lincRNA can be considered as a prognostic factor in a variety of cancers. The role of LINC00174 in the early diagnosis of human cancers, particularly through investigation of its levels in the biological fluids should be assessed in further studies.

This lincRNA has an impact of the determination of response of patients to temozolomide [12]. However, the effects of its up-regulation on response of patients to other therapeutic agents should be investigated in future. Since it affects both cell proliferation and apoptosis, it is expected that up-regulation of this lincRNA induces resistance to both chemotherapy and radiotherapy regimens.

Identification of LINC00174 mutations in different cancers, including pancreatic neuroendocrine tumors, melanoma, breast invasive ductal carcinoma, esophageal adenocarcinoma and hepatocellular carcinoma further implies important role of this lincRNA in the pathogenesis of human cancers. Therefore, this lincRNA is a putative target for design of novel therapeutic interventions.

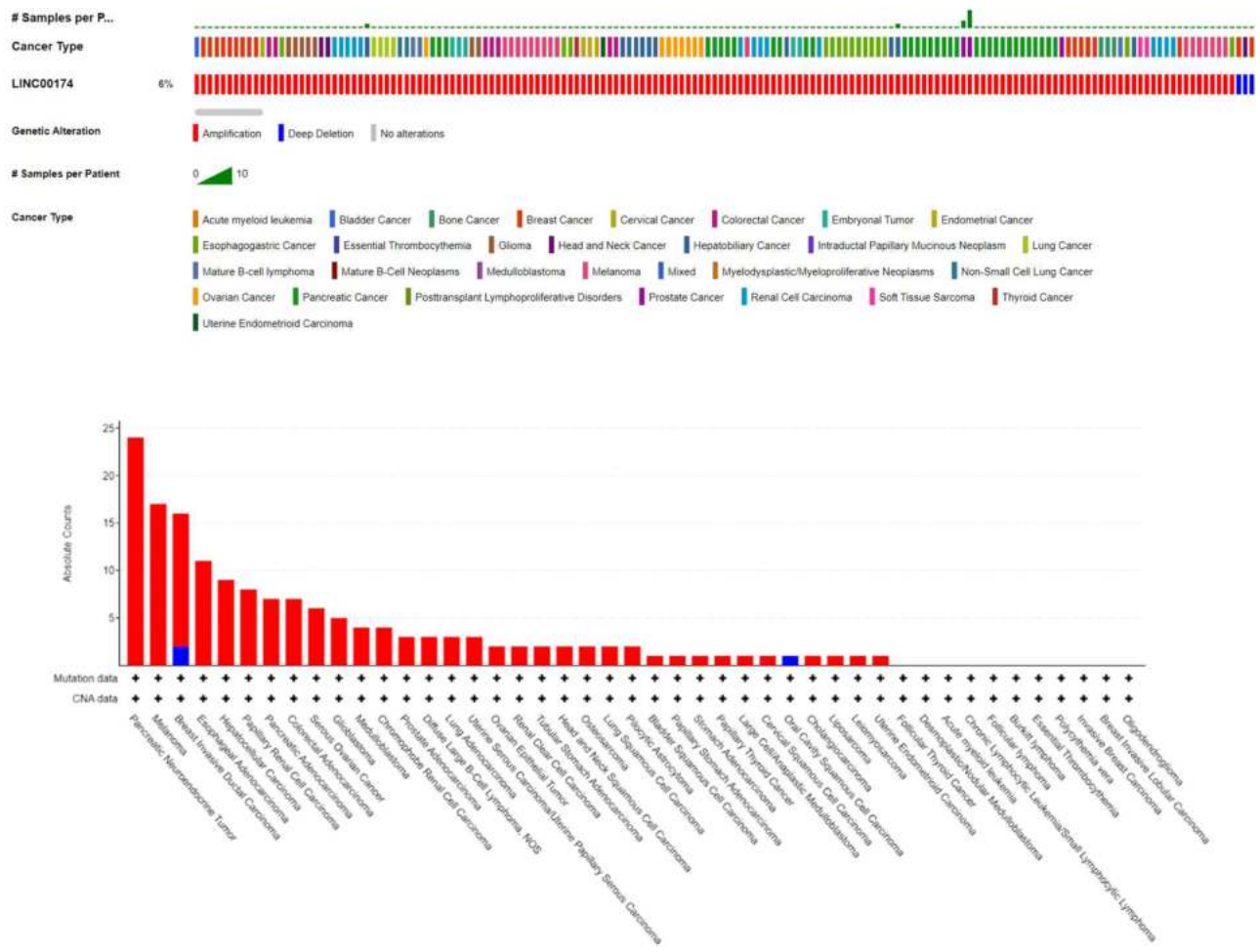

Fig. 5. TCGA datasets-based alterations in LINC00174 in various cancers. A) This graph displays all LINC00174 alterations in the TCGA cancer database. B) The LINC00174 alterations in each cancer are depicted in this plot.

Ethics approval and consent to Participant

Not applicable.

Funding

Not applicable.

CRediT authorship contribution statement

MT designed and supervised the study. **SGF** wrote the draft and revised it. **AS, AE and BMH** collected the data and designed the figures and tables. All the authors read the submitted version and approved it.

Declaration of Competing Interest

The authors declare they have no conflict of interest.

Acknowledgement

The authors would like to thank the Clinical Research Development Unit (CRDU) of Loghman Hakim Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran for their support, cooperation, and assistance throughout the period of study.

Consent of publication

Not applicable.

References

[1] E. Cerami, J. Gao, U. Dogrusoz, B.E. Gross, S.O. Sumer, B.A. Aksoy, A. Jacobsen, C. J. Byrne, M.L. Heuer, E. Larsson, Y. Antipin, B. Reva, A.P. Goldberg, C. Sander, N. Schultz, The cBio cancer genomics portal: an open platform for exploring multidimensional cancer genomics data, Cancer Discov. 2 (2012) 401–404.
[2] D.S. Chandrashekar, B. Bashel, S.A.H. Balasubramanya, C.J. Creighton, I. Ponce-Rodriguez, B. Chakravarthi, S. Varambally, UALCAN: a portal for facilitating tumor subgroup gene expression and survival analyses, Neoplasia 19 (2017) 649–658.
[3] X. Cheng, M. Sha, W. Jiang, L. Chen, M. Song, LINC00174 suppresses non-small cell lung cancer progression by up-regulating LATS2 via sponging miR-31-5p, Cell J. 24 (2022) 140–147.
[4] E. Deniz, B. Erman, Long noncoding RNA (lincRNA), a new paradigm in gene expression control, Funct. Integr. Genom. 17 (2017) 135–143.
[5] S. Ghafouri-Fard, S. Ahmadi Teshnizi, B.M. Hussen, M. Taheri, H. Zali, A review on the role of GHET1 in different cancers, Pathol. Res. Pract. 247 (2023).
[6] S. Ghafouri-Fard, A. Safarzadeh, M. Akhavan-Bahabadi, B.M. Hussen, M. Taheri, N. A. Dilmaghani, Expression pattern of non-coding RNAs in non-functioning pituitary adenoma, Front. Oncol. 12 (2022), 978016.
[7] S. Ghafouri-Fard, H. Shoorei, F.T. Anamag, M. Taheri, The role of non-coding RNAs in controlling cell cycle related proteins in cancer cells, Front. Oncol. 10 (2020).
[8] P. Gu, L. Lin, Long intergenic nonprotein coding RNA 00174 aggravates lung squamous cell carcinoma progression via microRNA-185-5p/Nuclear Factor IX axis, Biomed. Res. Int. 2022 (2022) 9490827.
[9] J. Guo, S. Shen, X. Liu, X. Ruan, J. Zheng, Y. Liu, L. Liu, J. Ma, T. Ma, L. Shao, D. Wang, C. Yang, Y. Xue, Role of linc00174/miR-138-5p (miR-150-5p)/FOSL2 feedback loop on regulating the blood-tumor barrier permeability, Mol. Ther. - Nucleic Acids 18 (2019) 1072–1090.
[10] H.U.A. Huang, S. Xu, Y. Li, Y. Gu, L. Ji, Identification of a hypoxia - related lncRNA signature for the prognosis of colorectal cancer, Res. Sq. (2021).

[11] J. Kang, Q. Tang, J. He, L. Li, N. Yang, S. Yu, M. Wang, Y. Zhang, J. Lin, T. Cui, Y. Hu, P. Tan, J. Cheng, H. Zheng, D. Wang, X. Su, W. Chen, Y. Huang, RNAInter v4.0: RNA interactome repository with redefined confidence scoring system and improved accessibility, Nucleic Acids Res. 50 (2022) D326–D332.
[12] B. Li, H. Zhao, J. Song, F. Wang, M. Chen, LINC00174 down-regulation decreases chemoresistance to temozolomide in human glioma cells by regulating miR-138-5p/SOX9 axis, Hum. Cell 33 (2020) 159–174.
[13] X. Li, W. Liu, W. Tao, LINC00174 promotes cell proliferation and metastasis in renal clear cell carcinoma by regulating miR-612/FOXM1 axis, Immunopharmacol. Immunotoxicol. 44 (2022) 746–756.
[14] J. Liu, T. Yang, Y. Zhang, S. Wang, Promotion of BZW2 by LINC00174 through miR-4500 inhibition enhances proliferation and apoptosis evasion in laryngeal papilloma, Cancer Cell Int. 20 (2020) 471.
[15] D. Ma, Exosomal LINC00174 facilitates epithelial-mesenchymal transition in residual hepatocellular carcinoma after insufficient radiofrequency ablation by regulating c-JUN/MYCBP/c-Myc axis, Ann. Oncol. 30 (2019) ix53–ix54.
[16] Y. Ma, Y. Li, Y. Tang, N. Tang, D. Wang, X. Li, LINC00174 facilitates proliferation and migration of colorectal cancer cells via MiR-3127–5p/E2F7 axis, 2021.
[17] A. Maimaiti, L. Jiang, X. Wang, X. Shi, Y. Pei, Y. Hao, H. Paerhati, Y. Zibibula, A. Abudujielili, M. Kasimu, Identification and validation of an individualized prognostic signature of lower-grade glioma based on nine immune related long non-coding RNA, Clin. Neurol. Neurosurg. 201 (2021), 106464.
[18] Y. Qian, L. Shi, Z. Luo, Long non-coding RNAs in cancer: implications for diagnosis, prognosis, and therapy, Front. Med. 7 (2020), 612393.
[19] A.D. Rouillard, G.W. Gundersen, N.F. Fernandez, Z. Wang, C.D. Monteiro, M. G. McDermott, A. Ma'ayan, The harmonizome: a collection of processed datasets gathered to serve and mine knowledge about genes and proteins, Database (2016) (2016).
[20] A. Safa, A. Abak, H. Shoorei, M. Taheri, S. Ghafouri-Fard, MicroRNAs as regulators of ERK/MAPK pathway: a comprehensive review, Biomed. Pharmacother. 132 (2020).
[21] Y. Shen, X. Gao, W. Tan, T. Xu, STAT1-mediated upregulation of lncRNA LINC00174 functions a ceRNA for miR-1910-3p to facilitate colorectal carcinoma progression through regulation of TAZ, Gene 666 (2018) 64–71.
[22] J. Shi, Y. Zhang, B. Qin, Y. Wang, X. Zhu, Long non-coding RNA LINC00174 promotes glycolysis and tumor progression by regulating miR-152-3p/SLC2A1 axis in glioma, J. Exp. Clin. Cancer Res. 38 (2019) 395.
[23] C. Tito, F. Ganci, A. Sacconi, S. Masciarelli, G. Fontemaggi, C. Pulito, E. Gallo, V. Laquintana, A. Iaiza, L. De Angelis, LINC00174 is a novel prognostic factor in thymic epithelial tumors involved in cell migration and lipid metabolism, Cell Death Dis. 11 (2020) 959.
[24] S. Wang, L. Xia, B. Zhang, H. Zhang, F. Lan, Downregulated long intergenic non-coding RNA 00,174 represses malignant biological behaviors of lung cancer cells by regulating microRNA-584-3p/ribosomal protein S24 axis, Funct. Integr. Genom. 22 (2022) 643–653.
[25] Z. Wang, Q. Wang, Z. Bao, L. Guo, H. Chen, T. Lv, T. Xu, X. Zhang, C. Zhou, L. Sun, LINC00174 is a favorable prognostic biomarker in glioblastoma via promoting proliferative phenotype, Cancer Biomark. 28 (2020) 421–427.
[26] G. Xu, M. Yang, Q. Wang, L. Zhao, S. Zhu, L. Zhu, T. Xu, R. Cao, C. Li, Q. Liu, W. Xiong, Y. Su, J. Dong, A. Novel, Prognostic prediction model for colorectal cancer based on nine autophagy-related long noncoding RNAs, Front. Oncol. 11 (2021).
[27] H. Xu, D. Han, K. Wang, T. Zhang, G. Gao, LINC00174 triggers the malignant development of breast cancer by negatively regulating miR-1827 level, Eur. Rev. Med. Pharmacol. Sci. 25 (2021) 6447–6453.
[28] J.T. Zhao, B.J. Chi, Y. Sun, N.N. Chi, X.M. Zhang, J.B. Sun, Y. Chen, Y. Xia, LINC00174 is an oncogenic lncRNA of hepatocellular carcinoma and regulates miR-320/S100A10 axis, Cell Biochem. Funct. 38 (2020) 859–869.