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A review on the role of LINC00173 in human cancers

Soudeh Ghafouri-Fard [a], Arash Safarzadeh [b], Bashdar Mahmud Hussen [c], Mohammed Fatih Rasul [d], Mohammad Taheri [e,f,*], Nader Akbari Dilmaghani [g,**]

[a] Department of Medical Genetics, School of Medicine, Shahid Beheshti University of Medical Sciences, Tehran, Iran
[b] Phytochemistry Research Center, Shahid Beheshti University of Medical Sciences, Tehran, Iran
[c] Department of Pharmacognosy, College of Pharmacy, Hawler Medical University, Kurdistan Region, Erbil, Iraq
[d] Department of Medical Analysis, Faculty of Applied Science, Tishk International University, Kurdistan Region, Erbil, Iraq
[e] Institute of Human Genetics, Jena University Hospital, Jena, Germany
[f] Urology and Nephrology Research Center, Shahid Beheshti University of Medical Sciences, Tehran, Iran
[g] Skull Base Research Center, Loghman Hakim Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran

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ABSTRACT

Long intergenic non-protein coding RNA 173 (LINC00173) is a long non-coding RNA with especial function in the tumorigenic process. Studies in different types of cancers support an oncogenic role for LINC00173 except for studies in B-cell precursor acute lymphoblastic leukemia, cervical cancer, pancreatic cancer and gastric cancer. In breast and lung cancers, both oncogenic and tumor suppressor roles have been reported for LINC00173. LINC00173 can serve as a molecular sponge for miRNAs. miR-218/Etk, miR-511–5p/VEGFA, miR-182–5p/AGER, miR-765/NUTF2, miR-765/PLP2, miR-182–5p/FBXW7, miR-338–3p/Rab25, miR-641/RAB14 and miR-1275/BCL2 are examples of the miRNA/mRNA axes being regulated by LINC00173 in the context of cancer. The current review provides a summary of different studies on the role of LINC00173 in these cancers.

1. Introduction

Long non-coding RNAs (lncRNAs) are transcripts with sizes more than 200 nucleotides that have regulatory effects on expression of several genes. They not only affect chromatin configuration, but also regulate transcription and translation of genes [21]. The important effects of lncRNAs on these processes as well as their contribution in splicing, protein localization, integrity of cellular structures, imprinting processes, cell cycle transition and apoptosis and stem cells function [16], endow them the potential to contribute in several physiological and pathological processes. Recent studies have shown their imperative functions in the development of cancer [8,9,14] and other disorders [3]. Despite these findings, inter-species investigations have shown poor conservation of lncRNAs compared with other types of RNAs [4,19,25]. Therefore, they might be involved in the determination of species-specific features. Moreover, lncRNAs are usually expressed at low levels [1,24].

Long intergenic non-protein coding RNA 173 (LINC00173) is an example of lncRNAs with especial function in the tumorigenic process. Alternatively named as NCRNA00173, this lncRNA is encoded by a gene on 12q24.22. This gene has four exons. In addition to the LINC00173–202 transcript which has retained intron, four other transcripts have been identified for this lncRNA with sizes ranging from 435 bp (ENST00000470091.1, LINC00173–201) to 1597 bp (ENST00000480237.1, LINC00173–203). LINC00173 is expressed in several tissues, particularly skin and endometrium [6]. The role of this lncRNA has been investigated in the pathogenesis of breast cancer, different types of lung cancer, as well as other malignancies. The current review provides a summary of different studies on the role of LINC00173 in these cancers.

2. Role of LINC00173 in cancers

2.1. Breast cancer

Two independent studies have investigated the role of LINC00173 in breast carcinogenesis. This lncRNA has been found to be over-expressed in triple negative breast cancer (TNBC) tissues compared to normal breast tissues. Over-expression of LINC00173 in tumoral tissues has been associated with low recurrence-free and overall survival rates of

* Corresponding author at: Institute of Human Genetics, Jena University Hospital, Jena, Germany.
** Corresponding author.
E-mail addresses: mohammad.taheri@uni-jena.de (M. Taheri), nadakbari@sbmu.ac.ir (N. Akbari Dilmaghani).

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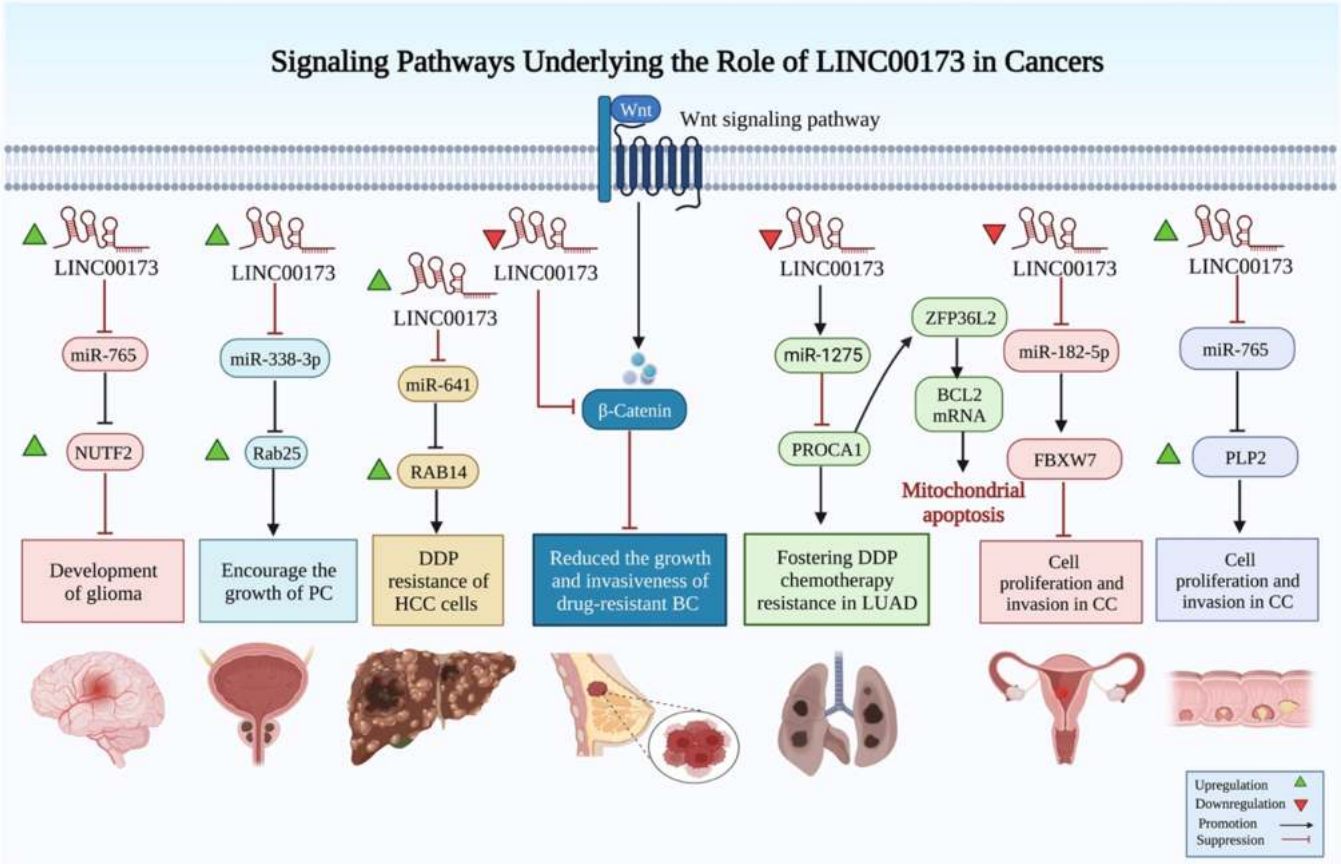

Fig. 1. An illustration of how LINC00173 works in different types of cancer. By sponging miRNAs, LINC00173 causes the up- or down-regulation of certain genes which makes cancer progression or drug resistance.

these patients. In vitro assays have shown that LINC00173 silencing inhibits proliferation, colony forming ability, and invasion of TNBC cells, while up-regulation of LINC00173 has led to opposite effect. More importantly, LINC00173 depletion has resulted in the tumor growth in animal models. Mechanistically, LINC00173 suppresses expression of miR-490–3p to enhance aggressive phenotypes of this type of breast cancer [7]. Contrary to this report, another study has shown that over-expression of LINC00173 suppresses proliferation and invasive properties of drug-resistant mammary tumor cells through suppression of β-catenin expression leading to induction of sensitivity to albumin paclitaxel in these cells [23].

2.2. Lung cancer

LINC00173 has also been shown to contribute to the chemo-resistance phenotype and progression of small-cell lung cancer. It has been shown to be over-expressed in chemoresistant cell lines of this histological type of lung cancer, and promote their chemoresistance, proliferation, and invasiveness. These effects of LINC00173 have also been verified in animal studies. Mechanistically, LINC00173 up-regulates expression of Etk via sponging miR-218. Moreover, its role in up-regulation of GSKIP and NDRG1 results in the translocation of β-catenin. Over- expression of LINC00173 in clinical samples of this type of lung cancer has been strictly correlated with chemoresistance, advanced stage, and shorter survival of patients [32]. Another study in this type of lung cancer has revealed that the impact of LINC00173 in the chemoresistance phenotype is mediated through binding with hnRNPA2B1 and hnRNPI and further regulating CHK2 levels [10]. Similarly, in squamous cell carcinoma of lung, LINC00173.v1 has ben shown to promote angiogenic process and cancer progression through sequestering miR-511–5p and regulating expression of VEGFA [2].

On the other hand, another study has reported down-regulation of LINC00173 in sera and tissues of patients with non-small cell lung cancer. Down-regulation of this lncRNA leads to up-regulation of miR-182–5p and subsequent enhancement of proliferation and migration and blockage of apoptotic processes through modulation of AGER/NF-κB pathway [29].

The potential of LINC00173 as a diagnostic marker in non-small cell lung cancer has been the subject of another study as well [28]. However, contrary to the latter study, this lncRNA has been found to be over-expressed in the sera of aptients with this istological type of lung cancer [28]. Serum levels of LINC00173 could differentiate between these patients and healthy donors with AUC value of 0.809. Yet, for small-cell lung cancer, the AUC value has been 0.730. The proposed diagnostic model consisting of LINC00173, CEA and Cyfra21–1 levels has even higher diagnostic efficiency. Notably, over-expression of LINC00173 has been correlated with histological typing of tumors, the presence of metastasis and serum levels of Cyfra21–1. Most remarkably, serum levels of LINC00173 have been reduced following chemotherapy and increased in patients following tumor recurrence [28].

Another study in lung adenocarcinoma has suggested a tumor suppressor role for LINC00173 and shown that downregulation of this lncrNA enhances stability of BCL2 transcript through the miR-1275/PROCA1/ZFP36L2 axis and leads to induction of resistance to cisplatin (Fig. 1) [22].

2.3. Other cancers

LINC00173 has an oncogenic role in glioma. Mechainistically, it increases expression of NUTF2 via adsorbing miR-765 [5]. This lncRNA

Table 1
Oncogenic role of LINC00173 in cancers.

Cancer Type	Expression / Role	Samples / Assessed Cell Lines	Pathways	Targets / Regulators	Function	Ref
Breast Cancer (BC)	Upregulated / Oncogene	84 patients with TNBC / MDA-MB-231, MDA-MB-468, BT-549	-	miR-490–3p	Via antagonizing miR-490–3p, LINC00173 triple-negative breast cancer (TNBC) cell growth. In order to combat TNBC, aiming LINC00173 could be a viable therapeutic approach.	[7]
Small cell lung cancer (SCLC)	Upregulated / Oncogene	9 formalin-fixed, paraffin-embedded (FFPE) normal lung tissues, 60 FFPE primary cancerous tissues and 40 blood samples / NCI-H69, NCI-H446, H69AR, H446DDP	-	miR-218/ Etk	Chemoresistance and SCLC advancement are boosted by the LINC00173-mediated procedure that may offer a viable treatment option and therapeutic approach.	[32]
	Upregulated / Oncogene	60 SCLC patient samples	-	CHK2	It was discovered that LINC00173 encouraged SCLC growth. A biomarker for a poor prognosis in SCLC sufferers is upregulation of LINC00173.	[10]
	Upregulated / Oncogene	60 SCLC patient samples	-	miR-218/ Etk	LINC00173 increases SCLC growth by serving as miR-218 s sponger, It controls Etk levels. These genes could serve as fresh markers for the clinical diagnosis of SCLC tumor development.	[31]
Lung squamous cell carcinoma (LSQC)	Upregulated / Oncogene	10 lung ADC tissues, 10 lung SQC tissues and the 20 corresponding adjacent tumor normal tissues/ HUVEC, BEAS-2B, Calu-3, NCI-H1395, NCI-H1975, NCI-H2228, NCI-H2347, NCI-H520, A549, Calu-1, NCI-H292, NCI-H460, NCI-H596, NCI-H661, NCI-H1299, NCI-H209, NCI-H446, MRC-5, WI-38, HFL1	-	miR-511–5p/ VEGFA	Because LINC00173.v1 encourages SQC carcinogenesis, it is possible to treat SQC with a sensible treatment regimen that includes cisplatin and a LINC00173.v1-targeted medication.	[2]
Non-small-cell lung cancer (NSCLC)	Upregulated / Oncogene	Serum samples were obtained from 108 NSCLC patients, 37 SCLC patients, 91 healthy donors and 79 BPD (benign pulmonary disease) patients / 16HBE, A549, NCI-H1299, SPC-A1	-	-	An elevated blood level of LINC00173 could act as a diagnostic marker for NSCLC and a tool for tracking tumor dynamics.	[28]
Glioma	Upregulated / Oncogene	37 glioma tissues and normal tissues / NHA, glioma cell lines	miR-765/NUTF2 Pathway	miR-765/ NUTF2	MiR-765 was absorbed by LINC00173 to increase NUTF2 production. Likewise, the LINC00173/miR-765/NUTF2 axis is crucial in fostering the development of gliomas.	[5]
Colorectal Cancer (CC)	Upregulated / Oncogene	FHC, CRC cell lines	miR-765/PLP2 axis	miR-765/ PLP2	Via the sponging of miR-765 from PLP2 mRNA, LINC00173 suppression lowered the expansion of CRC cells. The LINC00173/miR-765/PLP2 axis could be a viable treatment option.	[30]
Prostate Cancer (PCa)	Upregulated / Oncogene	124 PCa patients	LINC00173/miR-338–3p/Rab25 pathway	miR-338–3p/ Rab25	In PCa, LINC00173 could act as an oncogene. The LINC00173/miR-338–3p/ Rab25 pathway's revelation led to new approaches to treat PCa.	[12]
Hepatocellular carcinoma (HCC)	Upregulated / Oncogene	30 pairs of HCC and adjacent-normal tissues / Huh7, Hep3B,	microRNA-641/ RAB14 axis	miR-641/ RAB14	By sponging miR-641 to upregulate RAB14, LINC00173 encourages the HCC cisplatin [DDP] resistance of HCC cells, offering a unique therapeutic target for overcoming DDP resistance in HCC.	[34]
Wilms' tumor (WT)	Upregulated / Oncogene	-	-	MUC3A	Through MGAT1-mediated MUC3A N-glycosylation, LINC00173 accelerated WT evolution, providing fresh information on the mechanism underpinning WT advancement.	[35]
Melanoma	Upregulated / Oncogene	163 paired cancerous and noncancerous specimen samples	-	-	When compared to nearby non-neoplasm samples, the expression of LINC00173 in melanoma specimens is noticeably greater. Based on this research, LINC00173 might be employed as a bad prognostic marker for patients.	[26]
Esophageal squamous cell carcinoma (ESCC)	Upregulated / Oncogene	Bioinformatics methods	-	-	Ehe ESCC cell growth is influenced by LINC00173. The ESCC cells cell cycle and growth are aided by LINC00173 knockdown.	[17]
Nasopharyngeal Carcinoma (NPC)	Upregulated / Oncogene	16 normal nasopharynx tissues and 20 freshly frozen NPC tissues / NP69, HK1, SUNE1, CNE1, HNE1, HONE1, 5–8 F, 6–10B, S18, S26	LINC00173-RAB1B-PA2G4/SDF4 axis	PA2G4 and SDF4	A possible treatment target for NPC sufferers might be the LINC00173-RAB1B-PA2G4/SDF4 axis, which is a critical driver of NPC development.	[11]

Table 2
Tumor suppressive role of LINC00173 in cancers.

Cancer Type	Expression / Role	Samples / Assessed Cell Lines	Pathways	Targets / Regulators	Function	Ref
Breast Cancer (BC)	Downregulated / Tumor Suppressor Gene	26 BCE tissues and adjacent normal tissues / SK-BR-3	Wnt signaling pathway	β-catenin	The elevated expression of LINC00173 may prevent drug-resistant breast cancer cells from proliferating, invading and migrating. This study will aid in the creation of novel breast cancer treatments.	[23]
Non-small-cell lung cancer (NSCLC)	Downregulated / Tumor Suppressor Gene	Serum samples from 67 NSCLC patients, 39 healthy donors and 13 benign pulmonary diseases patients/ A549, NCI-H1299	AGER/NF-κB pathway	miR-182–5p/ AGER	In NSCLC cells, reduced LINC00173-induced miR-182 accumulation led to accelerated cell growth, and suppression of apoptosis via the AGER/NF-B axis.	[29]
Lung adenocarcinoma (LUAD)	Downregulated / Tumor Suppressor Gene	H1299, H1650, H1975, A549, SPCA1, PC9	miR-1275/PROCA1/ZFP36L2 axis	miR-1275/ BCL2	The poor survival result of LUAD sufferers receiving cis-diamminedichloroplatinum (DDP) therapy is attributed to down-regulation of LINC00173. In LUAD, LINC00173 is down-regulated which prevents cell apoptosis and increases DDP chemotherapy resistance.	[22]
Cervical Cancer (CC)	Downregulated / Tumor Suppressor Gene	30 pairs of CC and matched tumor-adjacent tissues / HeLa, SiHa, CaSki, C33A, Ect1/E6E7	miR-182–5p/ FBXW7 axis	miR-182–5p/ FBXW7	To control the production of FBXW7, LINC00173 served as a molecular sponge for miR-182–5p. As a possible treatment target for CC, our findings pointed to LINC00173/ miR-182–5p/FBXW7.	[33]
B-Cell Precursor Acute Lymphoblastic Leukemia (BCP-ALL)	Downregulated / Tumor Suppressor Gene	A total of 83 Bone marrow (BM) samples / HL60, K652, REH, SUPB15, MOLT	arachidonic acid metabolism, SNARE interactions in vesicular transport and lysosome pathways, integrins, IL3, IL6, and PTEN pathways	-	The level of LINC00173 is dysregulated in BCP-ALL, indicating that this gene is crucial in the overall progression of cancer.	[18]
Pancreatic cancer	Downregulated / Tumor Suppressor Gene	120 Pancreatic Cancer Samples / MIA PaCa-2, SW1990, PANC-1	NF-κB signaling pathway	SPHK1	Due to its ability to suppress SPHK1 transcription, LINC00173 upregulation may slow the spread of pancreatic cancer. For the treatment of pancreatic cancer, improving LINC00173 looks promising.	[15]
Gastric cancer (GC)	Downregulated / Tumor Suppressor Gene	Bioinformatics methods	Cell adhesion molecules (CAMS), cytokine-cytokine receptor interaction, the chemokine signaling pathway and leukocyte transendothelial migration	-	The best risk score system could be used to assess a prognosis for GC. In the etiology of GC, LINC00173 may play significant roles.	[13]

also increases growth of colorectal cancer cells, their invasive properties and chemoresistance phenotype via influencing activity of miR-765/PLP2 axis [30]. In prostate cancer cells, LINC00173 exerts its oncogenic role through sponging miR-338–3p and regulating expression of Rab25 [12]. Moreover, LINC00173 has an essential role in resistance of hepatocellular carcinoma cells to the anti-cancer drug cisplatin through influencing miR-641/RAB14 axis [34].

On the other hand, LINC00173 is regarded as a tumor suppressor lncRNA in cervical cancer through modulation of miR-182–5p/FBXW7 axis [33]. Similarly, LINC00173 down-regulation in TCF3/PBX1-positive cases of acute lymphoblastic leukemia has been found to be an indicator of poor prognosis [18]. Finally, LINC00173 has been shown to inhibit tumor growth and promote apoptosis of pancreatic cancer cells through suppression of expression of sphingosine kinase 1 protein expression [15]. Tables 1 and 2 summarize the role of LINC00173 in cancers.

3. Discussion

LINC00173 has diverse roles in the pathogenesis of different types of cancers. The impact of this lncRNA in the carcinogenesis is mostly assessed in the context of cancer. Except for two studies in lung adenocarcinoma [22] and non-small cell lung cancer [29], other studies have reported an oncogenic role for this lncRNA in small cell lung cancer [32], squamous cell carcinoma [2] and non-small cell carcinoma of lung [29]. Studies in other types of cancers are also in favor of an oncogenic role for LINC00173 except for studies in B-cell precursor acute lymphoblastic leukemia, cervical cancer, pancreatic cancer and gastric cancer. In breast cancer, both oncogenic and tumor suppressor roles have been reported for LINC00173. Therefore, it is possible that LINC00173 exerts tissue-specific or stage-specific functions in the carcinogenesis.

The most important route of its participation in the carcinogenesis is its sequestering effect on certain miRNAs. Through this rout, LINC00173 regulates expression of miRNA targets. miR-218/Etk, miR-511–5p/ VEGFA, miR-182–5p/AGER, miR-765/NUTF2, miR-765/PLP2, miR-182–5p/FBXW7, miR-338–3p/Rab25, miR-641/RAB14 and miR-1275/ BCL2 are examples of the miRNA/mRNA axes being regulated by LINC00173 in the context of cancer.

Additionally, LINC00173 participates in several axes in various cancers. We can specifically mention the LINC00173-RAB1B-PA2G4/ SDF4 axis in NPC, LINC-00173/miR-765/NUTF2 axis in glioma, LINC00173/miR-338–3p/Rab25 axis in prostate cancer, LINC00173/ miR-641/RAB14 axis in hepatocellular carcinoma and LINC00173/miR-1275/PROCA1/ZFP36L2 axis in lung adenocarcinoma.

Competing endogenous RNA (ceRNA) networks have been recently attracted attention of researchers in cancer biology [20,27]. Thus, it is possible that with the advent of high throughput sequencing methods and help of bioinformatics tools, researchers find other LINC00173-rgulated miRNA/mRNA axes in different types of cancers. These findings can facilitate identification of molecular aspects of cancer development and recognition of more sensitive and specific markers for early detection of cancer. Moreover, these molecular axes are putative targets for therapeutic interventions.

The diagnostic impact of LINC00173 in patients with different types of cancers is another aspect that should be assessed in future studies.

These studies should particularly focus on biofluids to find its importance in non-invasive methods of cancer detection. By focusing on the pathway in which LINC00173 participates, it is possible to stop the progression of cancer in malignancies where it acts as an oncogene. In order to treat certain tumors, this may be a target.

Finally, the mechanism of dysregulation of LINC00173 in different cancers should be unraveled. The presence of single nucleotide polymorphisms that affect its expression or interactions with miRNAs should also been assessed. This information would help in design of personalized methods of cancer treatment.

Ethics approval and consent to participant

Not applicable.

Consent of publication

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CRediT authorship contribution statement

MT designed and supervised the study. SGF wrote the draft and revised it. AS, NAD 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.

Availability of data and materials

The analyzed data sets generated during the study are available from the corresponding author on reasonable request.

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