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Review

A concise review on the role of LINC00324 in different cancers

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ABSTRACT

Long intergenic non-protein coding RNA 324 (LINC00324) is an example of lncRNAs whose roles in the carcinogenesis is being elucidated. This lncRNA is encoded by a gene located on 17p13.1. It has been shown to be over-expressed in a variety of cancer cell lines and tumoral tissues. However, there are few reports showing down-regulation of LINC00324 in cancer cell lines and tissues. miR-615–5p/AKT1, miR-139–5p/IGF1R, miR-769–5p/STAT3, miR-3200–5p/BCAT1 and miR-214–5p/CDK6/CCND1/MDM2/MDM4 are examples of miRNA/mRNA axes being influenced by LINC00324. LINC00324 can be regarded as a promising candidate for development of diagnostic and prognostic panels. Moreover, it can be used a therapeutic target for a wide range of cancers. The current review summarizes the evidence regarding the impact of lINC00324 in the carcinogenic processes.

1. Introduction

Non-coding RNAs that are more than 200 nt are known as long non-coding RNAs (lncRNAs). LncRNAs affect gene expression at the epigenetic, transcriptional, post-transcriptional, translational, and post-translational levels by interacting with mRNA, DNA, proteins, and miRNAs [10,11,14]. Long intergenic non-coding RNAs (lincRNAs) are a group of long non-coding RNAs (lncRNAs) that do not have overlap with annotated coding genes [17,27]. These transcripts share several features with lncRNAs and include more than half of lncRNAs in humans [9,21]. They have been firstly identified through application of tiling array techniques across genomic sequences, which showed persistent transcriptions from genomic areas having no recognized coding genes [1,15,22]. The main feature of lincRNAs that distinguishes them from other lncRNAs is lack of shared sequences with coding loci. Identification of lincRNAs in gene poor areas of the genome may decline the load of evolutionary conservation, thus permitting rapid functional divergence of lincRNA loci [21]. LincRNAs can influence activity of chromatin modification complexes or RNA binding proteins, thus changing gene expressing programs [25]. Most notably, they exhibit distinctive gene signatures between primary tumor samples and metastatic ones [25]. In addition to RNA-dependent activities, lincRNA loci have been suggested to serve as DNA elements, mediating RNA-independent functions [26]. It has been estimated that the number of lincRNAs in human genome exceeds 15,000 [6]. Recently, several lincRNAs have been identified that contribute in the pathogenesis of human disorders, particularly cancers through acting as either tumor suppressors or oncogenes [5,12].

Long intergenic non-protein coding RNA 324 (LINC00324) is an example of this group of lncRNAs whose roles in the carcinogenesis is being elucidated. This lncRNA is encoded by a gene located on 17p13.1. Being alternatively named as Chromosome 17 Open Reading Frame 44 (HGNC), Non-Protein Coding RNA 324 (HGNC), NONHSAG020759.2 (NONCODE), HSALNG0114408 (LncBook) and HSALNG0114404 (LncBook), it has three exons and 2100 bp. Currently, no splice variant has been identified for LINC00324. Recent studies have assessed expression of LINC00324 in different cancers revealing a possible oncogenic role for it in most of assessed tissues. However, in some tissues, it has the opposite effect. The current review summarizes the

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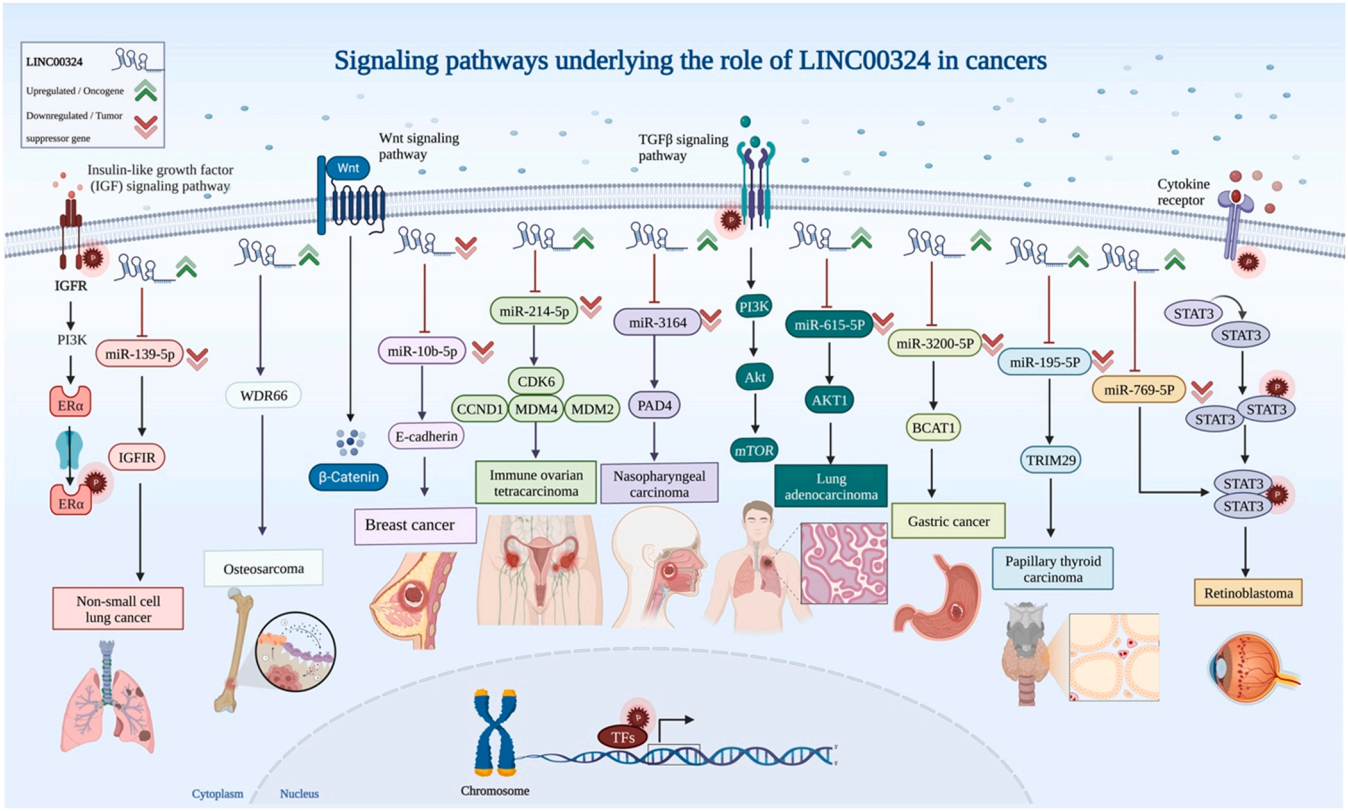

Fig. 1. Cancer-related functions of LINC00324. LINC00324 has been found to be highly expressed in different types of cancer. Up-regulation of LINC00324 promotes cancer cell proliferation and migration via sponging miRNAs or activating oncogenic proteins and oncogenic signaling pathways, while LINC00324 silencing is linked to the opposite effects.

evidence regarding the impact of lINC00324 in the carcinogenic processes.

2. Role of LINC00324 in cancers

LINC00324 has been found to be highly expressed in lung adenocarcinoma tissues compared with the adjacent non-tumoral samples. Similarly, expression of this lincRNA has been elevated lung adenocarcinoma cell lines. Up-regulation of LINC00324 has enhances proliferation and reserved apoptosis of these cells, while LINC00324 silencing has been associated with opposite effects. Moreover, LINC00324 could accelerate cell migration and invasion. Mechanistically, LINC00324 sponges miR-615–5p to increase AKT1 expression, revealing the importance of LINC00324/miR-615–5p/AKT1 axis in the progression of lung adenocarcinoma [19]. LINC00324 has also been among 11 oncosis-related lncRNAs that exhibit association with the prognosis of lung cancer. These lncRNAs could classify lung adenocarcinoma patients into distinct clusters and distinct risk groups. Notably, this expression signature has been strictly associated with immune activity. In fact, patients attributed to the low-risk group had a tendency to have more immune and stromal cells in tumor niche and elevated levels of PD-1, CTLA-4 and HAVCR2, endowing them a better response to immune checkpoint inhibitors [3]. LINC00324 has also been among ferroptosis and iron-metabolism related lncRNAs used for assessment of overall survival of patients with lung cancer [33].

Similarly, over-expression of LINC00324 in non-small cell lung cancer tissues has been correlated with poor prognosis. Mechanistically, LINC00324 sponges miR-139–5p to elevate IGF1R levels in these cells and promote their proliferation and invasive abilities. Therefore, LINC00324 can be a potential diagnostic and therapeutic target for this type of lung cancer [34].

LINC00324 has also been up-regulated in osteosarcoma tissues and cell lines in association with progression of this type of cancer and its metastatic ability. Notably, LINC00324 has a prognostic value in this type of cancer. Knock-in and knock-down studies have shown the role of LINC00324 in acceleration of cell proliferation and migration in osteosarcoma. From a mechanistical point of view, LINC00324 enhances stability of WDR66 mRNA via interacting with Hu antigen R (Fig. 1). Moreover, WDR66 has a critical role in the regulation of oncogenic effects of LINC00324 effects in osteosarcoma cells [31].

Over-expression of LIN00324 in colorectal cancer cells has been correlated with down-regulation of miR-214–3p. Moreover, LIN00324 silencing has inhibited proliferation, migratory aptitude and invasive properties of SW620 and HCT15 cells. Similar effects have been reported when miR-214–3p has been over-expressed. Cumulatively, LINC00324 has been shown to regulate proliferation, migration and invasive features of colorectal cancer cells through sponging miR-214–3p [18].

Consistent with above-mentioned oncogenic roles of LINC00324, this lincRNA has been found to be over-expressed in retinoblastoma tumors and cell lines compared with control samples. Most notably, up-regulation of LINC00324 has been correlated with the TNM stage, optic nerve invasion, and shorter overall survival of these patients. LINC00324 silencing has suppressed proliferation, colony forming ability, migration, and invasive properties of retinoblastoma cells, and stimulated apoptosis and cell cycle arrest. These effects have also been verified in animal models of retinoblastoma. Mechanistically, LINC00324 sponges miR-769–5p in retinoblastoma cells to increase expression of STAT3 [7].

Similar to the findings from retinoblastoma samples, over-expression of LINC00324 in gastric cancer tissues has been correlated with advanced TNM stage, larger tumor dimensions, and presence of lymph node metastases, indicating a poor prognosis. LINC00324 silencing has been shown to reduce proliferation of these cells and affect expression of FAM83B as revealed by RNA sequencing technique. Mechanistically,

Table 1
Overview of the role of LINC00324 in cancers.

Cancer Type	Expression / Role	Samples / Assessed Cell Lines	Pathways	Targets / Regulators	Function	Ref.
Lung adenocarcinoma	Upregulated / Oncogene	A549, PC-9, H1650, SPCA1, H1299	miR-615–5p/AKT1 axis	miR-615–5p / AKT1	The biological action of LINC00324 is to encourage LAC cell growth. The importance of this lncRNA in LAC formation makes it a viable treatment option.	[19]
	Upregulated / Oncogene	Bioinformatics methods	–	–	The innovative oncosis-based methodology looked at the tumor immunity and prognosis of LUAD sufferers, which might theoretically assist individualized treatment plans for LUAD patients.	[3]
	No significant difference	Bioinformatics methods, A549, H1299, 16HBE	Ferroptosis and iron-metabolism	–	The clinical outlook and immunotherapeutic actions in LUAD sufferers could be assessed by this risk signature based on the ferroptosis-related lncRNAs.	[33]
Non-small cell lung cancer cell (NSCLC)	Upregulated / Oncogene	48 NSCLC tissues, 18 adjacent normal Tissues, A549, H1299, H460, SK-MES-1, SPC-A-1, BEAS-2B, H1299, H460, SK-MES-1, SPC-A-1, BEAS2B	miR-139–5p/IGF1R axis	miR-139–5p / IGF1R	Enhanced production of LINC00324 in NSCLC boosted IGF1R production, and aided cell growth and infiltration. LINC00324 has been shown to be a useful treatment option.	[34]
Osteosarcoma	Upregulated / Oncogene	86 osteosarcoma tissues and corresponding non-tumoral tissues, 143B, MG-63, Saos-2, HOS, 143B	–	WDR66	Through controlling WDR66, LINC00324 promotes growth and motility osteosarcoma cells, making it a unique predictive target and treatment option.	[31]
Colorectal cancer (CRC)	Upregulated / Oncogene	NCM460, SW620, HCT15, SW480, HCT116	–	miR-214–3p	LINC00324 controls CRC cell growth and motility, indicating that it would be an efficient treatment target for CRC therapy.	[18]
Retinoblastoma	Upregulated / Oncogene	47 tumor samples and 13 normal retinal tissue specimens, Y79, SO-RB50, WERI-RB-1, ARPE-19	LINC00324–miR-769–5p–STAT3 pathway	miR-769–5p / STAT3	Blocking this lncRNA could be a viable method for treating people with RB, because it is involved in the diseases progression.	[7]
Gastric cancer (GC)	Upregulated / Oncogene	66 paired GC and adjacent tissues, SGC7901, BGC823, MGC803, AGS, GES-1	–	HuR - FAM83B	A putative indicator for the detection of GC could be LINC00324 that operated as an oncogene throughout the tumor formation.	[35]
	Upregulated / Oncogene	60 GC patients, AGS, MGC803, MKN-45	miR-3200–5p/BCAT1 axis	miR-3200–5p / BCAT1	By controlling the miR-3200–5p/BCAT1 axis, LINC00324 silencing prevented GC cells from proliferating, migrating and invading.	[30]
Papillary thyroid carcinoma (PTC)	Upregulated / Oncogene	42 pairs of PTC tissue and adjacent non-tumoral tissues, B-CPAP, KTC-1, TPC1, K1, Nthy-Ori 3–1,	–	miR-195–5p / TRIM29	By reducing TRIM29 transcription while increasing miR-195–5p production, LINC00324 silencing prevents PTC cells from proliferating and invading.	[32]
	Upregulated / Oncogene	60 pairs of PTC and normal tissues, Nthy-ori 3–1, K1, TPC-1, BCPAP, KTC-1	Notch signaling pathway	–	By blocking the notch signaling, silencing LINC00324 may prevent PTC cell growth, stop the cell cycle in the G1/G0 stage and boost cell death.	[28]
Nasopharyngeal carcinoma (NPC)	Upregulated / Oncogene	42 paired human NPC samples and paracarcinoma samples, 5–8 F, 6–10B, NP69	PI3K/AKT signaling pathway	miR-3164 / PAD4	Via the enhanced production of PAD4 and stimulation of the PI3K/AKT pathway, LINC00324 boosts NPC malignancy. This study might provide a strategy for NPC therapy.	[2]
Immune ovarian teratocarcinoma (IOT)	Upregulated / Oncogene	45 pairs of IOT tissues and mature ovarian teratocarcinoma (MOT) (benign) tissues, Hs 38. T, 293 T, PA-1	P53 signaling pathway, LINC00324-miR-214–5p-CDK6/CCND1/MDM2/MDM4 ceRNA network	miR-214–5p	LINC00324 acts as a ceRNA to encourage the growth of IOT cells, offering a potential new treatment target for IOT.	[4]
De novo acute myeloid leukemia (De novo AML)	Upregulated / Oncogene	–	–	–	In de novo AML sufferers, LINC00324 hypomethylation is a frequent biological occurrence. In leukemia cells, the unusually elevated LINC00324 encourages proliferation.	[24]
Acute myeloid leukemia (AML)	Downregulated / Tumor suppressor gene	peripheral blood leukocytes, KG-1, THP-1, U937	–	–	LINC00324 may have an impact on controlling immune cell formation, development and activity, offering a fresh approach to the creation of targeted medications of AML.	[16]
Breast cancer		45 paired breast cancer tissue samples and adjacent	–	miR-10b-5p / E-cadherin	LINC00324 may serve as a breast cancer treatment option.	[29]

(*continued on next page*)

Table 1 (*continued*)

Cancer Type	Expression / Role	Samples / Assessed Cell Lines	Pathways	Targets / Regulators	Function	Ref.
	Downregulated / Tumor suppressor gene	normal tissues, MCF-10A, MDA-MB-231, MCF-7				
Kidney Renal Clear Cell Carcinoma (KIRC)	Upregulated / Oncogene	OSRC2, ACHN	–	–	In KIRC, LINC00324 has both diagnostic and prognostic relevance, and this investigation proposes fresh treatment options for KIRC therapy.	[13]
Hepatocellular carcinoma (HCC) and liver cancer stem cell (LCSC)	Upregulated / Oncogene	182 tumor tissue samples and paired adjacent normal tissue samples, THLE-3, HepG2, Hep3B, HEP-1	–	FasL, PU.1	The biological properties of LCSCs are inevitably maintained by LINC00324 via boosting the production of FasL. This highlights linc00324 as a potential treatment option for HCC.	[8]
Esophageal squamous cell carcinoma (ESCC)	Upregulated / Oncogene	–	–	hsa-miR-493–5p	Although multi - center researches also are required, circulating LINC00324 may be employed as a potential indicator of ESCC.	[23]
Melanoma	Upregulated / Oncogene	Bioinformatics Methods	Autophagy pathway	–	The risk model created utilizing the 15 lncRNAs linked with autophagy, amongst them LINC00324, may have significant predictive significance for melanoma.	[20]

LINC00324 interacts with human antigen R (HuR) and increase stability of FAM83B. Besides, down-regulation of FAM83B has reversed the promoting effect of LINC00324 up-regulation on growth of gastric cancer cells [35]. miR-3200–5p/BCAT1 is another route that mediates the oncogenic role of LINC00324 in gastric cancer [30].

Experiments in papillary thyroid carcinoma cells and tissues have verified up-regulation of LINC00324. LINC00324 silencing has repressed proliferation and invasion of these cells. The oncogenic effect of LINC00324 is exerted through adsorbing miR-195–5p and subsequent up-regulation of TRIM29 [32]. Moreover, siRNA-mediated silencing of LINC00324 has led to cell cycle arrest in G1/G0 phase and induction of apoptosis in papillary thyroid carcinoma cells. Since this intervention has resulted in alterations in the levels of downstream Notch signaling markers, the oncogenic effects of LINC00324 has been attributed to its regulatory role on Notch signaling pathway [28].

In nasopharyngeal carcinoma cells, LINC00324 has been found to up-regulate expression of PAD4 through interaction with miR-3164 and recruitment of HuR protein. Moreover, LINC00324/miR-3164/PAD4 axis could modulate activity of PI3K/AKT signaling in these cells [2]. Similarly, LINC00324 has a promoting effect on proliferation of immature ovarian teratocarcinoma cells via sponging miR-214–5p [4].

In acute myeloid leukemia (AML), hypomethylation of LINC00324 has been reported to be a frequent molecular event. In addition, a negative correlation has been detected between expression level of LINC00324 and the methylation status of the promoter region of this lincRNA. Besides, lower expression of LINC00324 has been associated with longer overall survival of these patients. Notably, up-regulation of LINC00324 in leukemia cell lines has enhanced cell proliferation and inhibited cell apoptosis [24]. Contradictory to this report, Liu et al. have reported down-regulation of LINC00324 in peripheral blood leukocytes of AML patients in correlation with immunophenotype CD33. Moreover, expression of this lincRNA has been negatively correlated with percentage of peripheral blood blasts and white blood cell counts in these patients [16]. In addition to latter study, a single study in breast cancer has demonstrated a tumor suppressor role for LINC00324. This effect has been shown to be mediated through modulation of expression of miR-10b-5p [29] (Table 1).

3. Discussion

LINC00324 is an example of lincRNAs with dual functions in the carcinogenesis. It has been shown to participate in the construction of ceRNA networks that affect this process. miR-615–5p/AKT1, miR-139–5p/IGF1R, miR-769–5p/STAT3, miR-3200–5p/BCAT1 and miR-214–5p/CDK6/CCND1/MDM2/MDM4 are examples of miRNA/mRNA axes being influenced by LINC00324. In addition to regulation of autophagy and ferroptosis, it has regulatory effect on PI3K/AKT and Notch signaling pathways.

LINC00324 can be regarded as a promising candidate for development of diagnostic and prognostic panels. Moreover, it can be used a therapeutic target for a wide range of cancers. Based on the heterogeneity of cancer cells in terms of gene signature, multi-lncRNA panels are more promising than individual lncRNAs in both biomarker and therapeutic target development strategies. Thus, high throughput strategies that combine transcriptome data and single nucleotide variations with clinical data of patients are expected to find clinically relevant candidates for development of biomarker panels.

An important issue about LINC00324 is the fact that it can affect several types of cell death including apoptosis, autophagy, oncosis and ferroptosis. This unique feature of LINC00324 provides an opportunity of development of effective therapeutic modalities for cancers. Moreover, the observed association between expression of LINC00324 and presence of immune cells in the tumor microenvironment suggests that this lincRNA might be used as a candidate for combating immune evasion of cancer cells.

The exact mechanism of up-regulation of LINC00324 in cancer tissues has not been identified yet. However, a single study in AML patients has suggested hypomethylation of LINC00324 as a possible mechanism for its up-regulation [24]. The importance of other regulatory mechanisms including the effects of transcription factors on its expression should be evaluated.

The impact of LINC00324 on induction of chemo-/radio-resistance in cancer cells should be assessed in future studies. Moreover, the possible effects of single nucleotide variants within or near *LINC00324* gene on its activity, interactions with miRNAs and risk of different cancers should be evaluated in upcoming investigations.

Taken together, LINC00324 can be a possible therapeutic target in different cancers. However, future studies are required for identification of the mechanisms of its dysregulation and the consequences of abnormal expression of this lncRNA in different tissues.

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Ethics approval and consent to participate

Not applicable.

CRediT authorship contribution statement

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

Conflict of interest

The authors declare they have no conflict of interest.

Data availability

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

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Consent of publication

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