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Review

An update on the role of long non-coding RNAs in the pathogenesis of breast cancer

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ABSTRACT

Breast cancer is the most frequent female malignancy. This malignancy has diverse clinical and molecular subtypes with different prognoses. Dysregulation of long non-coding RNAs (lncRNAs) not only participates in the development of breast cancer, but also affects the clinical course and prognosis of this type of cancer. Hundreds of studies have shown up-regulation or down-regulation of lncRNAs in breast cancer samples or serum samples of affected individuals suggesting these RNA molecules as diagnostic markers for breast cancer. Different anticancer agents such as trastuzumab, lapatinib, doxorubicin, hydroxyurea, docetaxel, 5-fluorouracil and 6-thioguanine affect expression profile of lncRNAs. In the present article, we review the results of investigations about the role of lncRNAs in the evolution of breast cancer.

1. Introduction

Breast cancer is the foremost common type of cancer in female subject accounting for about one third of cancers in this subgroup [150]. Statistics have shown a slight upsurge in the incidence of this cancer since 2004 [137]. Among different molecular mechanism underlying development of breast cancer, dysregulation of long non-coding RNAs (lncRNAs) has much attracted the attention of investigators [137]. These large-sized transcripts are involved in a broad diversity of biological processes, including gene imprinting, developmental processes and immune responses through different mechanisms. The most appreciated mechanisms of function of lncRNAs are their interplay with chromatin-modifying proteins, regulation of configuration of nuclear domains, interference with transcription system, maintenance of the arrangement of nuclear speckles or their roles as transcriptional enhancers [2,122]. Besides, a number of lncRNAs regulate gene expression at post-transcriptional levels through modulating splicing, transcripts degradation, protein translation or stability, or sponging microRNAs (miRNAs) [122]. Several studies have shown aberrant profile of lncRNAs in breast cancer tissues or peripheral blood of affected persons. Moreover, the molecular mechanisms of lncRNAs partake in breast cancer have been appraised by several groups. In the present article, we review the results of investigations about the role of lncRNAs in the evolution of breast cancer. To avoid redundancy with formerly published review articles in this filed, we have only included publications in the period of 2018-2020.

2. Up-regulated lncRNAs in breast cancer

At least 195 distinct articles have reported up-regulation of certain lncRNAs in breast cancer. Most of these studies have been conducted on both cell lines and clinical specimens, thus appraising the molecular routes of involvement of lncRNAs in this kind of cancer. Chen et al. have shown over-expression of the nucleus-localized lncRNA Linc00839 in the chemoresistant breast cancer cells and clinical samples in association with poor prognosis. Mechanistical studies have demonstrated the impact of this lncRNA in the enhancement of proliferation, invasiveness, and migratory potential and resistance to paclitaxel both in vitro and in the xenograft model. Notably, expression of this lncRNA is enhanced by Myc oncogene. Linc00839has functional interaction with Lin28B and is implicated in the induction of PI3K/AKT signaling pathway [13]. Mitobe et al. have reported over-expression of TMPO-AS1 in basal-like

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breast cancer samples. Functional studies have demonstrated the role of TMPO-AS1 in the stimulation of proliferation and migration of triple negative breast cancer (TNBC) cells. Notably, TMPO-AS1 has a role in modulation of expression of TGF-β and proliferative E2F signaling pathways. TMPO-AS1 silencing has compromised the growth of primary and metastatic TNBC in the animal models [123]. Moreover, TMPO-AS1 expression has been induced by estrogen and has been increased endocrine therapy-resistant breast cancer cells compared the original cells. This lncRNA has a fundamental role in the enhancement of the proliferation of estrogen receptor (ER) positive breast cancer cells [124]. Among two alternative splice forms of NEAT1, NEAT1_2 expression has been correlated with the development of HER2-positive breast cancers and advanced pathological grades. Notably, NEAT1_1 and NEAT1_2 have different expression patterns among distinct subtypes of breast cancer [73]. MALAT1 is another up-regulated lncRNA in breast cancer whose silencing can suppress proliferation and migration of these cells. In breast cancer cells, MALAT1 has served as a molecular sponge for miR-145 to regulate expression of vascular endothelial growth factor [58].

Fig. 1 illustrates the role of some lncRNAs in the breast carcinogenesis. These lncRNAs mostly affect expression of miRNAs. For instance, the up-regulated lncRNAs SPRY4-IT1 and LUCAT1 inhibit expressions of miR-6882-3p and miR-5582-3p, respectively, thus enhancing expression of TCF7L2. This factor in cooperation with β-catenin increases expression of genes which participate in the breast carcinogenesis [154, 214].

Table 1 gives a comprehensive summary of the oncogenic roles of up-regulated lncRNAs in breast cancer.

3. Down-regulated lncRNAs in breast cancer

Totally, we identified 49 articles that reported down-regulation of lncRNAs in breast cancer samples in the mentioned time period (2018–2020). Among down-regulated lncRNAs in breast cancer is NKILA. Forced up-regulation of NKILA has suppressed the phosphorylation of IκBα and the nuclear transport of p65, thus inducing expression of apoptotic proteins and suppressing expression of those related with epithelial-mesenchymal transition. It has an inhibitory effect on IL-6 production through NF-κB signaling route [110]. GAS5 is a down-regulated lncRNA in TNBC subtype of breast cancer. Up-regulation of GAS5 has inhibited the progression of TNBC and stimulated chemosensitivity and apoptosis in these cells [81]. The tumor suppressor lncRNA LINC01355 has been shown to induce cell cycle arrest at the G0/G1 phase through suppressing CCND1. Furthermore, LINC01355 has a role in the enhancement of stability of FOXO3 protein, further suppressing CCND1 expression [3]. Being down-regulated in TNBC cells, WT1-AS has a regulatory role on expression of TGF-β1. Thus, forced over-expression of this lncRNA might suppress migration and

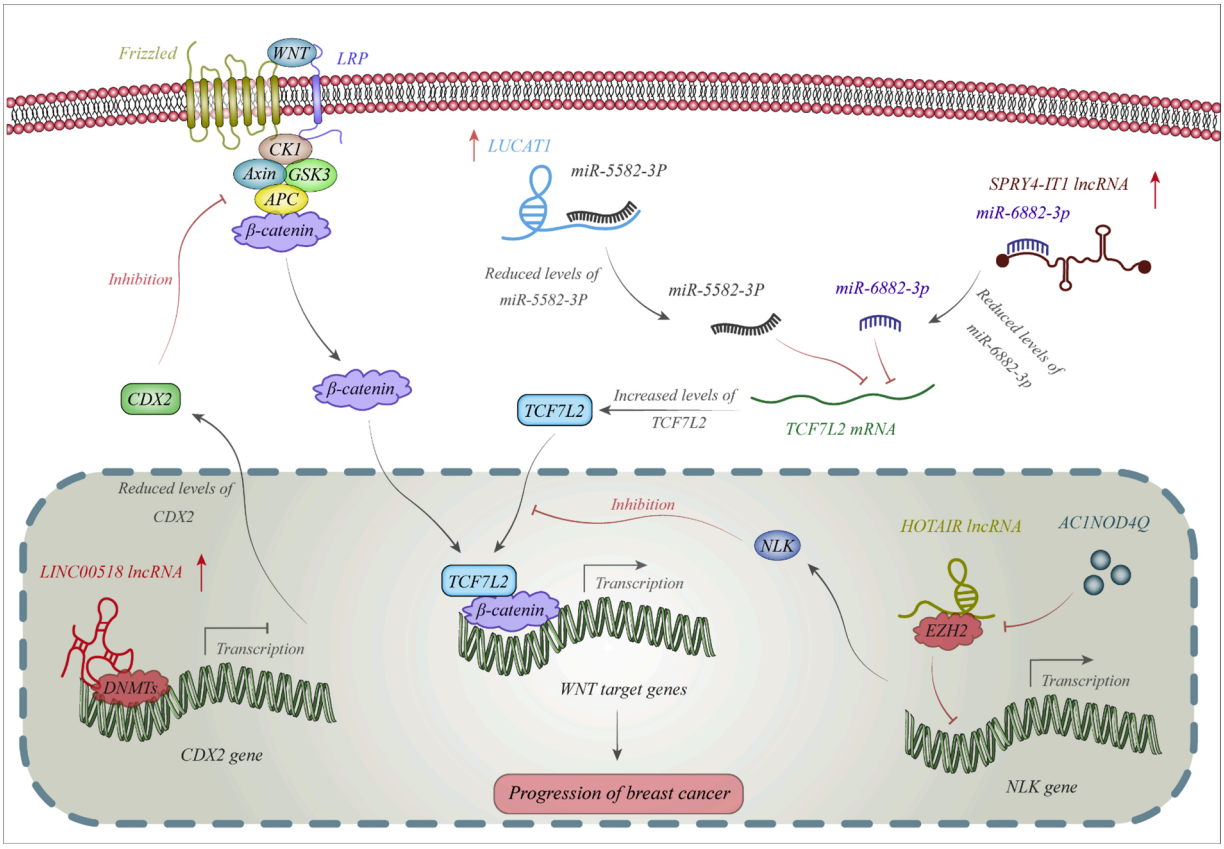

Fig. 1. SPRY4-IT1 and LUCAT1 are two up-regulated lncRNAs in breast cancer. These lncRNAs inhibit expressions of miR-6882-3p and miR-5582-3p, respectively. Down-regulation of these miRNAs leads to up-regulation of TCF7L2. This factor in cooperation with β-catenin increases expression of genes which participate in the breast carcinogenesis [154,214]. Expression of LINC00518 is also increased in breast cancer. This lncRNA recruits DNMT to the promoter of *CDX2* and represses its expression. CDX2 is involved in the suppression of Wnt/ β-catenin pathway [54]. In addition, HOTAIR recruits EZH2 to the promoter of *NLK* to suppress its expression. NLK is another gene which participates in the inhibition of Wnt/ β-catenin pathway. AC1NOD4Q substance can inhibit interaction between HOTAIR and EZH2 [144].

Table 1
Up-regulated lncRNAs in breast cancer (BC: breast cancer, ANTs: adjacent non-cancerous tissues).

lncRNA	Clinical Samples	Classification	Assessed Cell Lines	Targets / Regulators	Signaling Pathways	Description	Reference
Linc00839	32 BC and ANTs	ER positive: 15 PR positive: 19 HER2 positive: 14	MCF7, BT549, MDA-MB-231	Lin28B / Myc	PI3K/AKT	Linc00839 increases proliferation, invasion, migration, Paclitaxel resistance, and tumor growth. Linc00839 correlates with lymph node metastasis, TNM stage, and Ki67 levels.	[13]
TMPO-AS1	–	–	MDA-MB-231, MDA-MB-468, TNBC-PDC	MCM6, E2F, TMPO, MAD2L1, CHEK1, TGFBR1, TGFBR2	TGF-β	TMPO-AS1 improves the proliferation rate, tumor growth, and migration of TNBC cells.	[123]
	115 BC cases	HER2 positive: 7	MCF7, T47D	ESR1, GREB1, WISP2, MCM6, CDC6, MAD2L1 / estrogen	–	TMPO-AS1, induced by estrogen, enhances the proliferation, cell cycle progression, tumor growth, Tamoxifen resistance, and viability of ER^+ BC cells. TMPO-AS1 conforms with tumor stage, histological grade, and HER2 status.	[124]
lincRNA-p21	–	–	4T1, LLC	MDM2, p53	NF-κB, STAT	LincRNA-p21 down-regulation results in increased macrophage polarization into pro-inflammatory M1 in the tumor microenvironment. LincRNA-p21 knockdown increases apoptosis but decreases cell migration and invasion.	[222]
NEAT1_2	74 BC cases, 27 normal samples, 8 lactating females, and one pregnant woman sample	ER positive: 21 PR positive: 19 HER2 positive: 3	MCF7, T47D, BT474, HCC1569, SK-BR-3, BT549, Hs 578 T, MDA-MB-231, MDA-MB-468	ERBB2	–	NEAT1_2 levels correlate with tumor grade and HER2 status and are increased during lactation. NEAT1_2 increases ERBB2 mRNA expression.	[73]
NEAT1	–	–	MCF10A, MCF7, MDA-MB-231, 293	miR-107, CPT1A, BAD, CASP9, COL18A1, TIMP-1, PDGF-A, SERPINB2, cyclin D1, CDK4	–	NEAT1 promoted BC cells progression by diminishing apoptosis and enhancing cell cycle progression and invasion.	[190]
	–	–	MDA-MB-231, BT-549, MDA-MB-468, BT-549, MF-7	miR-21, RRM2	–	This lncRNA, like an oncogene, up-regulates proliferation and migration.	[142]
	–	–	MCF7, MDA-MB-453, MDA-MB-231, SKBR3, MCF10A	miR-448, ZEB1	–	NEAT1 enhances growth, migration, and the invasion capacity.	[67]
	40 BC and ANTs	ER positive: 8 PR positive: 4 HER2 positive:14	MCF7, MDA-MB-468, SKBR-3, HEK293 T	miR-133b, TIMM17A	–	NEAT1 improves cell migration and invasion.	[171]
	31 BC and ANTs and 5 normal breast tissues	–	MCF7, T47D, MCF10A	miR-124, STAT3	–	NEAT1 increases proliferation and cell cycle progression in BC cells.	[135]
	Blood and tissue samples of 179 BC and 192 healthy subjects	TN: 11 HER2: 17 Luminal: 154 NA: 4	MDA-MB-231	cyclin E1, cyclin D1, SOX2, ALDH	–	NEAT1 increases chemoresistance, cell stemness, and cell cycle progression while reducing the apoptosis rate.	[149]
GACAT3	20 BC and ANTs	–	MCF10A, MCF7, HEK293 T	miR-497, Caspase-9, Bcl-2	–	BC cells with a higher GACAT3 have an increased rate of cell proliferation and reduced apoptosis.	[56]
	41 BC and ANTs	–	MCF7, MDA-MB-231, MDA-MB-468, MDA-MB-453, T47D, SK-BR-3, MCF10A	miR-497, CCND2	–	GACAT3 evidently increases the proliferation rate.	[220]
	20 BC and 20 normal cases	–	MCF7, HUVEC	miR-145	–	MALAT1 knockdown markedly prohibits cell proliferation, migration, and tube formation.	[58]
MALAT1	7 primary BC and ANTs, TCGA dataset: 1086 BC cases	ER positive: 801 PR positive: 693 HER2 positive: 164	MCF7	miR-339−5p, BLCAP	–	MALAT1 matches ER status, PR status, and diagnosis ages.	[215]
	127 invasive ductal BC and ANTs	–	MCF12A, MCF7, MDA-MB-231	miR-216b-5p, PNPO	–	MALAT1 down-regulation reduces PNPO, inhibiting cell proliferation, migration, invasion, cell cycle progression, colony formation, and increasing the apoptosis rate.	[143]
BACH1	240 TNBC cases	TN	–	–	–		[131]

(*continued on next page*)

Table 1 (*continued*)

lncRNA	Clinical Samples	Classification	Assessed Cell Lines	Targets / Regulators	Signaling Pathways	Description	Reference
						The overexpression of MALAT1 and BACH1 in BC patients relates to large tumor size, lymph node metastasis, and advanced TNM stage.	
RAB11B-AS1	–	–	MDA-MB-231, MDA-MB-468, SUM159, MCF7, T47D, BT474, ZR-75-1, HCC1954, HeLa, HEK293FT, HEK293 T, HUVEC	VEGFA, ANGPTL4 / HIF2	–	RAB11B-AS1 expands migration, invasion, tumor angiogenesis, and distant metastasis.	[40]
CYTOR	40 BC and ANTs	Tamoxifen-resistant: 12 No treatment: 28	MCF7	miR-125a-5p, TAZ, p-ERK1/2	Hippo, MAPK	CYTOR enhances Tamoxifen resistance in BC patients.	[103]
LINC02273	319 BC cases	ER positive: 114 PR positive: 118 HER2 positive: 57	HEK293 T, MDA-MB-231, BT549	AGR2	PI3K-Akt, MAPK	This lncRNA promotes metastasis and correlates with lymph nodes metastasis.	[191]
BCLIN25	301 BC cases	–	MDA-MB-231, MDA-MB-453, MDA-MB-468, Hs-578 T, MCF7, UACC812, SKBR3, T47D, BT-549, MCF10A	miR-125b, ERBB2	–	BCLIN25 is related to HER2 status, and extends the level of migration and invasion.	[193]
HOXC-AS3	TCGA dataset: 1089 BC cases	–	MCF10A, MDA-MB-231, MCF7, T47D, HCC-1954, SK-BR-3, MDA-MB-468	miR-3922-5p, PPP1R1A	–	HOXC-AS3 hightens lung metastasis, cell invasion, EMT, and migration.	[198]
DLX6-AS1	45 BC and ANTs	ER positive: 27 PR positive: 19 HER2 positive: 30	MDA-MB-231, MCF7, MDA-MB-468, T47D, BT-4, MCF10A	miR-505-3p, RUNX	–	DLX6-AS1 elevates cell proliferation, metastasis, invasion, migration, and suppresses apoptosis. High DLX6-AS1 expression associates with tumor size, lymph node metastasis, and TNM stage.	[212]
	47 TNBC and 28 ANTs	TN	CCD-1095Sk, HCC1599, MDA-MB-231, HCC1806, HS578 T	miR-199b-5p, PXN	–	DLX6-AS1 down-regulation curtails cell proliferation, Cisplatin resistance, EMT, but promotes apoptosis.	[25]
FBXL19-AS1	–	–	MCF7, BT-549, MDA-MB-231, SKBR3, MCF10A	miR-876-5p, FOXM1	–	FBXL19-AS1 raises cell proliferation and restricts apoptosis.	[21]
	80 BC and ANTs	Luminal B: 63 TN/basal-like: 64 HER2 positive: 22	MCF7, MDA-MB-231	miR-204/211, miR-148a, miR-152, ANXA2, TCF4, β-catenin, DNMT1, FAT4	Wnt/ β-catenin	LncCCAT1 strengthens cell proliferation, stemness, migration, and invasion, and correlates with tumor grade, tumor histological, tumor size, and lymph node metastasis.	[167]
CCAT1	42 radio-sensitive BC tissues and 23 BC patients with no previous exposure	–	MCF7, MDA-MB-231	miR-148b		CCAT1 enhances the radio-resistance of BC cells.	[77]
	10 TNBC and ANTs	TN	MDA-MB-231, MDA-MB-436, MDA-MB-468, MCF10A	miR-218, ZFX	–	This oncogenic lncRNA improves TNBC cell proliferation, migration, tumor growth, and invasion.	[49]
linc02095	42 TNBC primary tumor samples as opposed to 21 normal tissues adjacent	TN	MCF10A, MCF10CA1h, MCF10CA1a.cl1	SOX9	–	Linc02095 expands the cell proliferation rate.	[170]
PSMG3-AS1	33 BC and ANTs	–	MCF10A, MDA-MB-468, MDA-MB-231, MCF7	miR-143-3p, COL1A1, PCNA	–	PSMG3-AS1 up-regulation results in inhibiting miR-143-3p, which escalates the proliferation and migration	[17]
BC200	Three breast core needle biopsies from 8 parous and 8 nulliparous women and another 10 BC and ANTs		MCF10A, MCF10 F, MCF7, T47D, MDA-MB-231, SK-BR-3, HEK293 T	–	–	BC200 overexpression increases cell survival and proliferation, cell migration and invasion, suppressing apoptosis, enhances tumor growth.	[8]
	Blood samples from 55 BC patients	Hormone receptor-positive: 29 HER2 positive: 15 TN: 11	–	–	–	H19 expression is related to a younger age, TN tumors, and Ki-67 index.	[132]
H19	60 TNBC and ANTs	TN	MCF7, SK-BR-3, BT-549, MDA-MB-231, MDA-MB-468, MCF10A	p53, TNFAIP8	–	H19 knockdown diminishes cell proliferation, migration, EMT, cell cycle progression, tumor growth, and invasion.	[195]
	48 BC cases	Hormone receptor-positive: 23	SKBR3 and HCC1954, MDA-MB-231,	–	–	H19 increases Trastuzumab resistance in BC cells and correlates with the TNM stage and Ki67 index.	[160]
		–	MCF7	Beclin1	–	H19 increases Tamoxifen resistancy by promoting the autophagy rate.	[205]

(*continued on next page*)

Table 1 (*continued*)

lncRNA	Clinical Samples	Classification	Assessed Cell Lines	Targets / Regulators	Signaling Pathways	Description	Reference
	14 tamoxifen-sensitive and 23 tamoxifen-resistant BC tissues						
	–	–	LCC2, LCC9, MCF7, T47D	ERα / Notch, c-MET	Notch and HGF	H19 augments the Tamoxifen and Fulvestrant resistance rate.	[9]
	–	–	MCF7, MDA-MB-231, HEK293 T	miR-93-5p, STAT3	–	Overexpression of H19 promoted proliferation, migration, and invasion.	[82]
	–	–	MDA-MB-453, MDA-MB-157, MDA-MB-231, Hs578Bst, MCF10A	Akt, Bax, Bcl-2 and cleaved caspase-3	AKT	H19 raises Paclitaxel-resistant in TNBC cells by increasing tumor growth and decreasing apoptosis.	[50]
GHSROS	176 BC and 16 normal breast tissues	ER positive: 74 PR positive: 67 HER2 positive: 42 TN: 33	MDA-MB-231, MDA-MB-468, MDA-MB-453, T47D, MCF7, MCF10A	HTR1F, EPHA3, TENM1, TBX3, HLA-DRB3, HLA-DRA, HLA-DPB1	–	GHSROS up-regulates cell migration and tumor growth, but not the cell proliferation rate.	[171]
	63 BC and ANTs	–	MCF7, MDAMB-231, SKBR3, T47D, MDA-MB-453, MCF10A	miR-7-5p, SOX2	–	lnc-LUCAT1 broadens cell proliferation, migration, and invasion	[89]
LUCAT1	94 TNBC and ANTs	TN	MCF10A, MDA-MB-231, TB-549, MDA-MB-453, MDA-MB-468	miR-5702	–	LUCAT1 improves cell proliferation, EMT, cell cycle progression, and metastasis, while restricting cell apoptosis. LUCAT1 associates with tumor grade, lymph node metastasis, and distant metastasis.	[126]
	26 BC and ANTs and another 151 BC cases	ER positive: 58 PR positive: 59 HER2 positive: 101	MCF7, T47D, MCF10A	MiR-5582-3p, TCF7L2	Wnt/ β-catenin	LUCAT1 expression is related to tumor size, lymph node metastasis, and TNM stage. LUCAT1 up-regulation up-regulates proliferation and self-renewal.	[214]
LINC00673	80 BC and ANTs	ER positive: 51 PR positive: 32 HER2 positive: 36	MDA-MB-231, MDA-MB-453, MDA-MB-468, Hs-578 T, MCF7, T47D, BT-549, HEK293 T, MCF10A	miR-515-5p, MARK4 / YY1	Hippo	LINC00673 intensifies proliferation by diminishing the cell cycle arrest and apoptosis. LINC00673 is related to tumor size and Ki67 status.	[140]
LINC02582	2 sample series: 136 paraffin-embedded BC cases and 42 fresh BC tumor tissues	–	MDA-MB-231, MCF7, BT549, SKBR3, T47D, BT474, MCF10A, 293 T	USP7, CHK1 / miR-200c	–	LINC02582 improves radio-resistance in BC cells and tissues.	[174]
SNHG3	60 BC and ANTs	ER positive: 45 PR positive: 37 HER2 positive: 36	MDA-MB-231, MCF7, MCF10A	miR-384, HDGF	–	SNHG3, like an oncogene, extends tumor growth, metastasis, proliferation, and invasion. It also associates with histological grade, lymph node metastasis, advanced TNM stage, ER, and HER2 status.	[118]
	48 BC and ANTs	–	MCF7, MDA-MB-231, MDA-MB-468, BT-474, MCF10A	miR-326	–	This lncRNA improves proliferation, colony formation, migration, invasion, cell cycle proliferation, and tumor growth	[15]
lnc-SLC4A1-1	66 BC and ANTs and blood samples	ER positive: 43 PR positive: 48 HER2 positive: 38	MCF7, MDA-MB-231	p65, CXCL8	NF-κB	Lnc-SLC4A1-1 boosts cell viability, proliferation, migration, and invasion but suppresses apoptosis.	[201]
UFC1	76 ductal BC and ANTs	Ductal	MDA-MB-231, SKBR-3,MDA-MB-453, BT-474, MCF7, HBL-100	miR-34a, CXLC10	–	UFC1 knockdown results in overexpression of miR-34a, down-regulated cell growth, proliferation, EMT, invasion, migration and up-regulated apoptosis.	[188]
	39 BC and ANTs	ER positive: 19 PR positive: 25 HER2 positive: 12	MDA-MB-468, MDA-MB-231, MDA-MB-453, MCF7, MCF10A	miR-185-3p, E2F1, Nanog	–	LINC00511 increases the proliferation, invasion, tumor stemness, and tumor growth.	[106]
LINC00511	98 BC and ANTs	ER positive: 48 PR positive: 62 HER2 positive: 34	MDA-MB-231, MDA-MB-436, MDA-MB-361, MCF7, MCF10A	miR-185, STXBP4	–	LINC00511 escalates tumor proliferation, radio-resistance tumor growth and reduces apoptosis. It also correlates with tumor size and recurrence.	[101]
	70 BC and ANTs and 3 non-BC tissues	ER positive: 19 PR positive: 14 HER2 positive: 37	MCF7, UACC-812, MDA-MB-231	PRC2, CDKN1B / TFAP-2	–	LINC00511 increases tumor growth, G1/S transition, proliferation and decreases apoptosis. LINC00511 correlates with tumor size, ER, PR, Ki-67, and p53 status.	[175]
	21 BC and ANTs	HER2 positive: 2 TN: 3	MDA-MB-231, MCF7, Hs-578 T, T47D, MCF10A	miR-29c, CDK6	–	LINC00511 promotes Paclitaxel resistance.	[72]

(*continued on next page*)

Table 1 (*continued*)

lncRNA	Clinical Samples	Classification	Assessed Cell Lines	Targets / Regulators	Signaling Pathways	Description	Reference
LINC01096	60 TNBC and ANTs	TN	T47-D, BT-549	miR-3130-3p	–	LINC01096 knockdown lowers cell viability, migration, and invasion, as well as promoting apoptosis.	[181]
HEIH	60 TNBCs, 60 ANTs, and 30 non-TNBCs	TN	MDA-MB-436, BT549	miR-4458, SOCS1	–	HEIH extends the cell proliferation rate and curtails apoptosis. Tumor size and an advanced clinical stage correlate with HEIH expression.	[181]
NONHSAT101069	–	–	MCF7, T47D, MDA-MB-231, SUM1315, ZR-75-1, SK-Br-3	miR-129-5p, Twist1	–	NONHSAT101069 up-regulates the Epirubicin resistance, migration, invasion, and EMT.	[200]
lncRNA-HAL	80 TNBC and normal breast tissues	TN	MCF7	ALDOC, CD151, CRIP2, PRSS8, SELENBP1	TGFβ	lncRNA-HAL raises proliferation, migration, invasion, cell cycle progression, and cell survival rates.	[35]
MIR210HG	45 invasive BC and ANTs	–	MDA-MB-231, MCF7	miR-1226-3p, MUC1-C	–	MiR210HG down-regulation impedes BC cell proliferation, invasion, EMT, tumor growth, and metastasis. Tumor metastasis and TNM stage associate with MIR210HG expression.	[80]
SNHG6	20 BC and ANTs	–	EFM192A, AU565, UACC893, MDA-MB-415, HS742 T, MDA-MB-231, MCF7, HEK293 T	miR-26a, VASP	–	SNHG6 enhances proliferation, cell cycle progression, migration, and invasion.	[161]
	45 BC and ANTs	ER positive: 23 PR positive: 18 HER2 positive: 10	SK-BR-3, MCF7, MDA-MB-231, NCCIT, DPSC, Hela, AGS, SW480, SW1116, U-87MG, SH-SY5Y, PC3, LNCaP, HepG2, Jurkat, HEK239	–	–	SNHG6 promotes proliferation, migration, EMT, cell cycle progression, and restricts apoptosis and senescence. SNHG6 expression is related to higher tumor grade, size, and PR^+ status.	[60]
	60 BC and ANTs	ER positive: 33 PR positive: 26 HER2 positive: 22	MCF10 A, BT-474, MDA-MB-231, ZR-75-30, MDA-MB-468, T47D	miR-26a-5p, MAPK6	–	SNHG6 upgrades BC cell proliferation, migration, and invasion. Moreover, it correlates with tumor size, TNM stage, and distant metastasis.	[114]
TATDN1	24 BC and ANTs	–	Hs578Bst, MCF7, MDA-MB-231, and BCap-37	miR-140-3p, NOVA1	–	TATDN1 increases proliferation and cell cycle progression.	[178]
ADPGK-AS1	74 BC and ANTs	Hormone receptor-positive: 14 HER2 positive: 20	MDA-MB-436, MDA-MB-453, MCF7, MDA-MB-231, MCF10A	miR-3196, OTX1	–	ADPGK-AS1 enhances cell proliferation, migration, EMT, and lowers cell apoptosis.	[68]
PAPAS	78 TNBC and ANTs and blood samples from each patient	TN	MDA-MB-157, BT-549	miR-34a, lnc-OC1	–	PAPAS soars migration and invasion rate but does not affect proliferation.	[76]
CDR1as	90 BC patients	–	MCF7, SKBR-3, MDA-MB-231, MDA-MB-468, HCC-1937, MCF10A	miR-7, REGγ	–	CDR1as lessens Cisplatin sensitivity in BC cells.	[178]
HCP5	30 cases of invasive ductal carcinomas and 30 ANTs	–	MCF10A, MCF7, T47D, SK-BR-3	miR-219a-5p, BIRC3	–	HCP5 expression correlates with the TNM stage and TNBC, increases tumor growth, cell proliferation, and reduces the apoptosis rate.	[133]
	–	–	MDA-MB-231, MDA-MB-157, MDA-MB- 468, HCC1806, HEK293 T	PTEN	PTEN/Akt	Overexpression of HCP5 increases cisplatin resistance.	[133]
LINC00473	60 BC and ANTs	ER positive: 30 PR positive: 32 HER2 positive: 19	MDA-MB-231, MCF7, SK-BR-3, MDA-MB-453, MCF10A	miR-198, MAPK1	–	LINC00473 raises the proliferation, invasion, migration, and tumor growth of BC cells. LINC00473 expression is related to tumor size, lymph node metastasis, and TNM stage.	[129]
	122 BC and ANTs	ER positive: 58 PR positive: 66 HER2 positive: 54	MDA-MB-231, MDA-MB-453, MCF7, MDA-MB-468, MCF10A	miR-497	–	LINC00473 correlates with lymph node metastasis and clinical stage. LINC00473 down-regulation weakens cell proliferation, metastasis, and cell viability.	[7]
LINC01287	112 BC and ANTs	ER positive: 61 PR positive: 64 HER2 positive: 63	MDA-MB-468, MDA-MB-453, MDA-MB-231, MCF7, MCF10A	β-catenin, cyclin D1, c-myc	Wnt/β-catenin	This lncRNA conforms with the TNM stage and lymph node metastasis. LINC01287 enhances proliferation and metastasis and restricts apoptosis.	[151]
ATXN8OS	120 BC and ANTs	ER positive: 84 PR positive: 78 HER2 positive: 36	MCF7, MDA-MB-231, MCF10A	miR-204, JAK2, FOXA1, ANGPT1, TGFβR2	–	ATXN8OS elevates proliferation, cell cycle progression, viability, and invasion.	[19]
SNHG7	37 BC and ANTs	–	MCF7, MDA-MB-231, MDA-MB-157, MDA-MB-435, MCF10A, T47D, SK-BR-3	miR-34a, CyclinD1, survivin	Notch-1	SNHG7 evidently improves cell proliferation, tumor growth, EMT, and invasion.	[158]
	72 BC and ANTs	–	MCF7, MDA-MB-231, SKBR3, MCF10A	miR-186	–		[113]

(*continued on next page*)

Table 1 (*continued*)

lncRNA	Clinical Samples	Classification	Assessed Cell Lines	Targets / Regulators	Signaling Pathways	Description	Reference
						SNHG7 was positively associates with tumor stage, lymph node and distant metastasis. SNHG7 knockdown restricts proliferation, invasion, and migration.	
PANDAR	65 BC and ANTs	ER positive: 31 PR positive: 42	MCF7, MDA-MB-231, MDA-MB-436, SK-BR-3, MCF10A	Vimentin, MMP2, MMP9	–	PANDAR expression is parallel to lymph node metastasis and advanced clinical stage. PANDAR down-regulation diminishes proliferation, EMT, colony formation, and invasion.	[119]
lncUSMycN	52 BC and ANTs	ER positive: 17 PR positive: 16 HER2 positive: 15	ZR-75-1, MCF7, MDA-MB-231	–	–	lncUSMycN expression correlates with the TNM stage. lncUSMycN knockdown restricts EMT and up-regulates the apoptosis rate.	[36]
MIF-AS1	82 BC and ANTs	Hormone receptor positive:19 HER2 positive: 47	MCF7, MDA-MB-231, MDA-MB-468, MCF10A	miR-1249-3p, HOXB8	–	MIF-AS1 increases BC cell proliferation, migration, and EMT.	[20]
RHPN1-AS1	30 BC and ANTs	–	MCF7	miR-4261, P53, P21, MDM2 / c-Myc	–	RHPN1-AS1 up-regulates cell proliferation and colony formation.	[226]
RP1	54 BC and ANTs, and another 97 BC cases	–	MCF7, T47D, SKBR3, MDA-MB-231, BT549, HCC38, HCC1937, HEK293 T	p27kip1, eIF4E, eIF4G, Snail1 / KLF5, p300	–	RP1 increases the proliferation rate, stemness, EMT, and metastasis. It also correlates with tumor size, node status, TNM stage, and distant metastasis.	[88]
HOXC13-AS	100 BC and ANTs	–	MCF7, MDA-MB-468, 293 T	miR-497-5p, PTEN, cyclin D1, p21, PCNA	–	This lncRNA enhances cell proliferation and tumor growth in BC cells and tissues.	[130]
AWPPH	Plasma samples from 72 TNBC and 44 healthy cases	TN	MDA-MB-231, BT-20	miR-21	–	AWPPH escalates cell proliferation and chemoresistance.	[98]
FOXD3-AS1	19 BC and ANTs, TCGA dataset: 966 BC and 94 normal tissues	–	MDA-MB-231, BT-549	–	–	FOXD3-AS1 down-regulation is related to smaller tumor size and less distant metastasis. FOXD3-AS1 knockdown restricts cell proliferation, migration, and invasion.	[44]
MAFG-AS1	42 BC and ANTs	–	MDA-MB-231, HCC1937, MCF7, MDA-MB-468, MCF10A	miR-339-5p, MMP15	–	EMT, migration, invasion, tumor growth, and metastasis are all promoted after MAFG-AS1 up-regulation.	[206]
	186 primary BC cases	–	MDA-MB-231, MDA-MB-468	CHST15	–	HOTAIR increases migration and invasion rate in BC cells and tissues.	[206]
	63 BC and 23 normal subjects	ER positive: 17 HER2 positive: 18	MCF7, MDA-MB-231, HEK293	JMJD6	–	This lncRNA enhances the tumor growth.	[12]
HOTAIR	10 BC and ANTs	–	MCF7, SKBR3, MDA-231, MCF10A	miR-218	–	HOTAIR down-regulation lowers cell survival and enhances cell apoptosis, DNA damage, and cell cycle arrest.	[57]
	–	–	HBL-100, MCF7, MDA-MB-231, SKBR-3	EZH2, PTEN	–	Silencing HOTAIR represses the proliferation, invasion, and migration while promoting the apoptosis of BC cells.	[52]
	20 BC and ANTs	–	MCF7, SKBR3, MDA-MB-231, MCF10A	miR-20a-5p, HMGA2	–	HOTAIR augments cell proliferation, migration, invasion, and metastasis and diminishes cell apoptosis.	[213]
NAMPT-AS	64 BC cases, TCGA dataset: 746 BC cases	–	MDA-MB-231, MDA-MB-468, MCF7, SKBR3, MCF10A	NAMPT, POU2F2, miR-548b-3p	PI3K/Akt/mTOR	Tumor proliferation, metastasis, tumor growth are all enhanced by NAMPT-AS expression. NAMPT-AS overexpression is associated with younger age, larger tumor size, lymph node involvement, negative ER and PR status, TNBC subtype, metastasis, and advanced stage. It also diminishes apoptosis.	[169]
LINC01433	–	–	MDA-MB-231, MDA-MB-453, MCF7, MDA-MB-436, MCF10A	miR-2116-3p, MYC	–	LINC01433 heightens cell proliferation, migration, and EMT, but reduces cell apoptosis.	[184]
ES1	45 BC and ANTs	ER positive: 30 PR positive: 21 HER2 positive: 10	MCF7, MDA-MB-231, SKBR3	Oct4, Sox2, miR-302, miR-106b	–	ES1 expands proliferation, migration, EMT, cell cycle progression, and induces apoptosis. ES1 expression is correlated with ER and PR status and tumor grade.	[70]
AGAP2-AS1	–	–	SKBR-3, BT474, MCF10A	hnRNPA2B1	–	AGAP2-AS1 increases Trastuzumab resistance in BC cells.	[219]

(*continued on next page*)

Table 1 (*continued*)

lncRNA	Clinical Samples	Classification	Assessed Cell Lines	Targets / Regulators	Signaling Pathways	Description	Reference
DANCR	42 primary BC and ANTs	–	SKBR-3, BT474	MyD88, CBP / SP1	NF-κB	AGAP2-AS1 extends the cell growth and Trastuzumab resistance and lowers apoptosis.	[23]
	57 TNBC and ANTs	TN	MCF7, MDA-MB-231, MCF10A	miRNA-216a-5p, Nanog, SOX2, OCT4	–	DANCR improves cell proliferation, tumor growth, migration, and invasion.	[169]
	60 TNBC and 10 normal tissues, TCGA dataset: 19 BC and ANTs	TN	BT549, MCF7, T47D, MDA-MB-231, MDA-MB-453, MDA-MB-468, MCF10A	RXRA, GSK3β, PIK3CA	PI3K/AKT	DANCR enhances TNBC proliferation and tumor growth.	[163]
ZEB2-AS1	98 BC and ANTs	–	MCF10A, T47D, MDA-MB-435, MDA435, MCF7, MDA-MB-231	ZEB2, EGF, F-actin	PI3K/Akt/ GSK3β	ZEB2-AS1 increases proliferation, EMT, and metastasis. It is also related to tumor differentiation, lymph node metastasis, and distant metastasis.	[41]
CASC9	17 BC and ANTs	–	MDA-MB-231, MDA-MB-468, MCF7, MDA-MB-415, MCF10A	miR-195/497, CHK1	–	CASC9 promotes cell proliferation, tumor growth, cell cycle progression, and suppressed cell apoptosis	[146]
	48 BC and ANTs	ER positive: 34	MCF7, MDA-MB-231, MDA-MB-157, MDA-MB-468	EZH2, MDR1	–	Silencing CASC9 abates the growth and metastasis of chemo-resistant BC cells.	[63]
LincK	79 BC cases and 8 healthy tissues	–	MCF7, 293 T, MDA-MB-453, MDA-MB-231	miR-200 s, ZEB1, PBK	MAPK	LincK knockdown lessens the growth, colony formation, migration, EMT, metastasis, and invasion.	[30]
HOXD-AS1	107 BC and ANTs	ER positive: 53 PR positive: 47 HER2 positive: 49	MCF7, MDA-MB-435, MDA-MB-231, MDA-MB-468, MCF10A	miR-421, SOX4	–	HOXD-AS1 enhances cell proliferation, EMT, cell cycle progression, migration, and invasion. high HOXD-AS1 correlates with distant metastasis and advanced TNM stage.	[64]
LOL	21 luminal BC and ANTs, and another 20 luminal BC and 20 non-luminal BC, and a 374 luminal BC cohort	Luminal	MCF7, BT-474, LCC-1, LCC-2, ZR-75-1, T-47D, hTERT-HME-1, MCF10A	Let-7 / ZMYND8, BRD4, ERα, Estrogen	Wnt/ β-catenin	LOL strengthens tumor proliferation, G1/S progression, and Tamoxifen resistance.	[157]
HOTTIP	84 BC and ANTs	ER positive: 23 PR positive: 16 HER2 positive: 12	MDA-MB-231, MDA-MB-468	β-catenin, HOXA13	Wnt/ β-catenin	Migration, invasion, metastasis, EMT are all improved after HOTTIP overexpression. It is also correlated with TNM stage and lymph node metastasis.	[53]
	–	–	MCF7, T47D, MCF10A, HEK293 T	miR-148a-3p, Wnt1	Wnt/ β-catenin	HOTTIP promotes stemness and tumor growth of BC cells.	[51]
	–	–	MCF7, MCF10A	HOXA11	–	HOTTIP or HOXA11 knockdown lower cell proliferation and migration but promote cell apoptosis.	[159]
	70 BC and ANTs	ER positive: 36 PR positive: 42 HER2 positive: 27	MCF10A, SKBR3, MDA-MB-231, MCF7	Cyclin D1, PCNA	PI3K/AKT	HOTTIP improves cell cycle, proliferation, colony formation, and invasion while reducing the apoptosis rate.	[32]
HIF1α-AS2	86 TNBC, 30 non-TNBC, and 30 ANTs	–	MCF10A, DU4475, HCC1806, MDA-MB- 468	–	–	HIF1α-AS2 relates to lymph node metastasis, distant metastasis, and histological grade in TNBC patients. This lncRNA increases migration and invasion rates.	[189]
FOXD2-AS1	34 BC and ANTs	ER positive: 23 PR positive: 19 HER2 positive: 20	MCF7, MCF10A, MDA-MB-231, MDA-MB-453, MDA-MB-468	miR-150-5p, PFN2	–	FOXD2-AS1 increases cell proliferation, stemness, EMT, migration, tumor growth, and invasion. FOXD2-AS1 up-regulation is associated with ER status, HER2 status, distant metastasis, lymph node metastasis.	[22]
UCA1	10 hormone receptor-positive and 10 normal cases	Hormone receptor-positive	T47D, MCF7, LCC2, LCC9	EZH2, p21, CREB	PI3K/AKT	UCA1 induces Tamoxifen resistance by increasing cell cycle arrest and apoptosis.	[92]
	–	–	MCF7, MCF10AT	Merlin, HK2, AKT, STAT3	TGF-β, Hippo	HK2 enhances the aerobic glycolysis rate in BC cells.	[125]
	–	–	MCF10A, MDA-MB-231, JIMT1, MDA-MB-468, HCC1937, MCF7	- / ARID1A, CEBPα	–	Proliferation and migration rate are improved due to UCA1 up-regulation.	[47]
	–	–	SKBR-3	miR-18a, YAP1	–	Silencing UCA1 raises Trastuzumab resistance.	[224]
LINC01857	67 BC and ANTs	ER positive: 32 PR positive: 38 HER2 positive: 16	MCF7, BT-20, T47D, SKBR3, MCF10A	CREB1, CREBBP	–	LINC01857 expands proliferation, migration, invasion rate, and restricts apoptosis of BC cells. LINC01857 overexpression is related to lymph node metastasis, large tumor size, and advanced clinical stage.	[189]

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Table 1 (*continued*)

lncRNA	Clinical Samples	Classification	Assessed Cell Lines	Targets / Regulators	Signaling Pathways	Description	Reference
LINC00461	15 BC and ANTs	ER positive: 6	MDA-MB-468, MDA-MB-231, MDAMB- 415, MCF7	miR-30a-5p, ITGB3	MAPK/ ERK, PI3K/ AKT	LINC00461 up-regulation conforms with the TNM stage and differentiation. LINC00461 boosts BC cell migration and invasion rate.	[22]
TINCR	60 HER2-positive BC cases	HER2 positive	SKBR-3, BT474, MCF10A	miR-125b, HER2, Snail-1 / CBP	–	TINCR increases the trastuzumab resistance, migration, invasion, and EMT in BC cells. Lymph node and distant metastasis and TNM stage are related to TINCR expression.	[22]
	24 BC and ANTs		MDA-MB-231, MDA-MB-435, MDA-MB-453, MDA-MB-468, MCF7, MCF10A	miR-7, KLF4 / SP1		Elevated cell proliferation, migration, invasion, tumor growth, and suppressed cell apoptosis result from TINCR expression.	[102]
LINC00518	60 BC and ANTs, TCGA dataset: 1102 BC and 113 normal cases	–	HBL-100, MCF7, MDA-MB-231, MDA-MB-4359	CDX2	Wnt/ β-catenin	LINC00518 escalates proliferation, invasion, metastasis, migration, and EMT of BC cells.	[54]
	52 BC and ANTs, TCGA dataset: 572 BC cases	ER positive: 447 PR positive: 471 HER2 positive: 513	MDA-MB-468 and MDA-MB-231, MCF10A, MCF7, BT474, T47D	PTEN, NEDD4-1 / YY1	–	LINC00152 overexpression is related to ER or PR negative expression, late TNM stage, and lymph node metastasis.	[147]
LINC00152	31 TNBC and ANTs, and 31 non-TNBC samples	TN	MDA-MB-231, MDA-MB-469, MDAMB- 361, MCF7, T47D, MDA-MB-435, BT474, BT20, BT483, 184A1, MCF10A	BRCA1, PTEN	–	LINC00152 improves invasion, tumor, and colony growth and reduced apoptosis rate.	[99]
	40 BC and ANTs	–	SKBR3, MCF10A, MDA-MB-231, MCF7, MCF7/ADR	–	–	Silencing LINC00152 conspicuously lowers cell viability, chemo-resistance, EMT, growth, invasion, and migration.	[99]
PRNCR1-2	20 BC and ANTs	–	HS-578 T, MCF7, MDA-MB-468, MDA-MB-231, BT-549, MCF10A	CHK2, AKT	–	Like an oncogene, this lncRNA improves proliferation, migration, cell cycle progression, and invasion, though it didn't affect the apoptosis rate.	[134]
	183 BC and ANTs	ER positive: 93 PR positive: 105 HER2 positive: 112	–	–	–	LINP1 up-regulation correlates with advanced TNM stage, lymph node metastasis, and poor pathological differentiation.	[100]
LINP1	TCGA dataset: 364 BC patients	ER positive: 235	MCF7, T47D, TAMR	ER, GREB1, PGR / estrogen	–	LINP1 enhances proliferation, migration, invasion, EMT, chemo-resistance, and increases apoptosis.	[120]
	67 BC patients	ER positive: 56 PR positive: 26 HER2 positive: 1	MDA-MB-231, MDA-MB-468, MCF7	- / p53	–	This lncRNA promotes migration, invasion, EMT, chemo-resistance, and correlated with distant metastasis and advanced clinical stage.	[94]
RUSC1-AS-N	100 BC and ANTs	–	T47D, MCF7, MDA-MB-231, MDA-MB-468, SK-BR-3, MCF10A	Wnt1, β-catenin	Wnt/ β-catenin	RUSC1-AS-N heightens proliferation, colony formation, and metastasis.	[223]
ZFHX4-AS1	62 BC and ANTs	–	MDA-MB-231	FAT4, YAP, TAZ	Hippo	ZFHX4-AS1 knockdown diminishes BC cell proliferation, migration, invasion metastasis, tumor growth, cell cycle progression while increasing cell apoptosis.	[85]
lncATB	131 primary BC and 16 normal breast tissues	–	MCF7, T47D, MDA-MB-231, BT-549, BT-20 MDA-MB-436, MDA-MB-435, SKBR3, MCF10A	miR-200, Twist1 / TGF-β	–	lncATB promotes cell migration, EMT, invasion, and colony formation. Higher lncATB expression associates with tumor grade, Ki-67 expression, and distant metastasis.	[84]
	10 invasive BC and 10 normal breast tissues	–	MDA-MB-231, BT549, MCF10A	miR-141-3p, ZEB1, ZEB2	–	lnc-ATB raises cell migration, EMT, and invasion.	[210]
HOST2	98 BC and ANTs	–	MDA-MB-231, MDA-MB-468, SK-BR-3, MCF7, MCF10A	let-7b	–	HOST2 improves cell motility, migration, and invasion, thus enhancing BC tumors' growth. Its expression correlates with lymph node metastasis and TNM stage.	[176]
DSCAM-AS1	40 BC and ANTs	–	HCC1937	miR-204-5p, RRM2	–	DSCAM-AS1 escalates the proliferation, metastasis, tumor growth, invasion of BC cells, and reduced apoptosis.	[93]
	21 BC and ANTs	Luminal	MCF7, T47D		–		[156]

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Table 1 (*continued*)

lncRNA	Clinical Samples	Classification	Assessed Cell Lines	Targets / Regulators	Signaling Pathways	Description	Reference
				BCL2, CDC6, E2F7, ESR1, FEN1, TOP2A		DSCAM-AS1 elevates cell proliferation, colony formation, G1/S transition, DNA replication, sister chromatid cohesion, chromosome segregation, and DNA recombination. This lncRNA also improves DNA replication, cell cycle, pyrimidine metabolism, mismatch repair.	
	30 BC patients divided into two groups: tamoxifen-resistant and responsive	–	MCF7, T47D, SK-BR-3, MDA-MB-31, MCF7	miR-137, EPS8	–	DSCAM-AS1 increases Tamoxifen resistance, cell cycle promotion, and cell proliferation.	[121]
CASC15	32 BC and ANTs	–	MDA-MB-468, MCF7, MDA-MB-231, MDA-MB-453, MCF10A	KLF5 / miR-153-3p, KLF5	–	CASC15 improves cell proliferation, invasion, and tumor growth.	[203]
TALNEC2	20 BC and ANTs	–	MCF7, MDA-MB-231, SK-BR-3, BT-474, MCF10A	EZH2, p57, KIP2, p-p38, RelA, RelB	MAPK and NF-κB	TALNEC2 boosts cell viability, colony formation, G0/G1 transition, and restricts apoptosis.	[139]
lncRNA-CDC6	837 BC and A105 normal tissues	–	MCF-10A, MCF-10AT, MCF-10CA1A, MCF-10CA1H, MDA-MB-231, MDA-MB-468, MCF7, MDA-MB-453, ZR-75-1, Hs578 T	miR-215, CDC6	–	lncRNA-CDC6 soars proliferation and migration.	[75]
BLAT1	11 normal and 19 breast tumor tissues	–	HMEC, 184A1, AU-565, CAMA-1, DU-4475, HCC-1500, HCC-1569, HCC-1806, HCC-1937, HCC-202, HCC-70, Hs578 T, MCF7, MDA-MB-157, MDA-MB-175V11, MDA-MB-468, T47D, UACC-3199, ZR-75-30	γ-H2AX	–	BLAT1 knockdown improves the apoptosis rate due to DNA damage accumulation.	[182]
AWPPH	68 TNBC and ANTs and 64 healthy controls	TN	MDA-MB-231, BT-20	FZD7	–	AWPPH extends cell proliferation and corresponds with tumor size and TNM stage.	[24]
TP73-AS1	25 TNBC and ANTs	TN	MDA-MB-231	miR-490-3p, TWIST1	–	TP73-AS1 restricts vasculogenic formation.	[168]
	86 BC and ANTs	ER positive: 48 PR positive: 47 HER2 positive: 37	MCF7, MDA-MB-231, HEK293	miR-200a, ZEB1, E-cadherin, Twist / ZEB1	–	TP73-AS1 enhances the invasion and migration and is correlated with tumor size, TNM stage, and lymph node metastasis. TP73-AS1 down-regulation axiomatically diminished the mtDNA copy number.	[228]
	36 BC and ANTs	ER positive: 17 PR positive: 17 TN: 20	Hs578Bst, BT474, MDA-MB-231, T47D, MCF7, MDA-MB-453	miR-200a, TFAM	–	TP73-AS1 decreases BC cell proliferation and conforms to an advanced TNM stage.	[65]
STAR1	30 BC and ANTs	–	MCF10A, MCF-12A, MDA-MB-231, MCF7	/ KLHDC7B	–	STAR1 improves G0/G1 transition and cell migration.	[62]
LincRNA-ROR	152 BC and ANTs	–	MCF7, MDA-MB-231, T47D, BCAP-37, ZK-75-1, MCF10A	p62	PI3K/Akt/mTOR	lincRNA-ROR elevates proliferation, Tamoxifen resistance, and dwindles apoptosis rate.	[108]
	30 TNBC and ANTs	–	MCF10A, MCF7, BT474, MDA-MB-453, MDA-MB-231	miR-145, MUC1	–	Invasion and metastasis rates are promoted after lincRNA-ROR expression.	[117]
SPRY4-IT1	–	–	SKBR-3, MCF7, MDA-MB-231	SKA2 / NT21MP, SDF-1α, CXCR4	–	SPRY4-IT1 intensifies proliferation, migration, invasion, cell cycle, and mitigates apoptosis rate.	[182]
SNHG14	36 BC and ANTs, and 62 HER2-positive BC patients treated with trastuzumab	HER2 positive	SKBR-3, BT474	PABPC1	Nrf2	SNHG14 grows cell proliferation, invasion, and Trastuzumab resistance.	[24]
MIR100HG	TCGA database: 360 BC patients	–	MDA-MB-231, 293 T, BT549	p27	–	Silencing MIR100HG lessens cell proliferation cell cycle progression.	[111]
LINC01638	54 BC and ANTs, and another 141 primary BC cases	TN: 37 (of second cohort)	MCF10A, T47D, MCF7, BT474, 293 T, SKBR3, ZR-75-30, ZR- 75-1, MDA-MB-231, BT549, HCC1937	SPOP, c-Myc, MTDH, Twist1	–	LINC01638 boosts EMT, stemness, proliferation, and metastasis.	[111]
	50 BC and ANTs	HER2 positive	MDA-MB-231, MCF7, T47D, BT549, BT483, BT20, BT474 and SKBR3, 184A, MCF10A	DNMT1, DNMT3a, DNMT3b, BRCA1, PTEN		LINC01638 down-regulation elevates cell apoptosis, as well as restricts the growth, invasion, and metastasis.	[104]
LINC00310	48 BC patients and 47 normal cases	–	MCF7, MDA-MB-231, HMLE, HEK-293 T, LM-4142	c-Myc	–	LINC00310 improves cell proliferation and tumor growth and matches an advanced BC stage.	[46]
LINC01296	55 BC and ANTs	ER positive: 35 PR positive: 34 HER2 positive: 17	MCF7, MDA-MB-231, SKBR3, BT-20, T47D, MDA-MB-436, MCF10A	–	–	LINC01296 conforms to larger tumor size, positive lymph node metastasis, and advanced TNM stage. LINC01296 knockdown mitigates BC cell growth and expands apoptosis.	[65]
AFAP1-AS1	160 BC and ANTs	ER positive: 73		–	–		[115]

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Table 1 (*continued*)

lncRNA	Clinical Samples	Classification	Assessed Cell Lines	Targets / Regulators	Signaling Pathways	Description	Reference
		PR positive: 140 HER2 positive: 113	MCF7, SK-RB-3, MDA-MB-231, MDA-MB-468, MCF10A			AFAP1-AS1 grows the proliferation, colony formation, and metastasis rates and lessens apoptosis. Its overexpression is consistent with TNM stage and lymph node metastasis.	
	104 BC patients	HER2 positive: 64	SKBR-3, BT474	AUF1, ERBB2	–	AFAP1-AS1 improves Trastuzumab resistance.	[209]
	–	–	MDA-MB-231, MDA-MB-468, MDA-MB-435S, HCC1937, MCF10A	miR-145, MTH1, ATF6	–	cell proliferation and invasion, cell viability and colony formation are raised by AFAP1-AS1 up-regulation.	[209]
FTH1P3	15 paclitaxel-sensitive and 15 -resistant patients	–	MCF7, MDA-MB-231, MDA-MB-468, MDA-MB-453, MCF10A	miR-206, ABCB1	–	FTH1P3 knockdown suppresses tumor growth and cell cycle progression of Paclitaxel-resistant BC cells.	[164]
lncRNA-BCHE	56 BC and ANTs	ER positive: 30 PR positive: 33 HER2 positive: 11	MDA-MB-231, MCF7, MDA-MB-468	ITGB1	–	lncRNA-BCHE matches with advanced clinical stage and lymph node metastasis. It also expands proliferation, tumor growth, migration, and invasion.	[199]
ITGB2-AS1	–	–	MDA-MB-231, MCF7	ITGB2, FAK, MMP9	–	This lncRNA escalates the migration and invasion rate of BC cells.	[105]
	128 BC and 16 normal tissues	–	MCF7, MDA-MB-231, Hs58T	OCT4	–	Up-regulated MIAT tallies with stages 1 and 2, cell cycle progression, tumor growth, and restricted apoptosis.	[5]
MIAT	84 BC and ANTs	HER2 positive: 16	MCF7, MDA-MB-231, T47D, MDA-MB-453, MCF10A	DNMT1, DNMT3A, DNMT3B, DLG3, MST2, LAST1, YAP	Hippo	MIAT inhibition results in increased DLG3, which reduces proliferation, migration, invasion, and enhances apoptosis.	[78]
	37 BC and ANTs	ER positive: 28 PR positive: 20 HER2 positive: 8	NCCIT, MCF7, SKBR-3	Cyclin D1, mir-302, mir-150, miR-29c, p16Ink4A, Cox2	–	MIAT expression corresponds to age, tumor size, and tumor grade. MIAT enhances cell cycle progression and migration but reduces apoptosis.	[4]
linc01561	–	–	MCF7, BT-20, ZR-75-1, MX-1, MCF10A	miR-145-5p, MMP11	–	Linc01561 inhibition diminishes cells proliferation and boosts the apoptosis rate.	[66]
ARNILA	88 TNBC	TN	MDA-MB-231, Hs578 T	miR-204, SOX4	–	EMT, invasion, migration, and metastasis are raised by ARNILA expression.	[107]
GHET1	60 BC and ANTs	–	SKBR3, ZR-75-1, BT-20, MCF7, MCF10A	–	–	GHET1 matches larger tumor size, advanced clinical stage, lymph node metastasis. GHET1 silencing drops proliferation, invasion, migration, EMT, and G0/G1 transition rates.	[153]
FEZF1-AS1	30 BC and ANTs	ER positive: 20 PR positive: 16 HER2 positive: 18	MCF7, MDA-MB-231, MDA-MB-468, MDA-MB-453, MCF10A	miR-30a, Nanog		FEZF1-AS1 extends stemness, proliferation, migration, tumor growth, and invasion.	[79]
AC026904.1	60 BC and normal tissues	Ductal and invasive ductal carcinoma	MDA-MB-231, luc-D3H2LN	Slug	TGF-β	EMT, invasion, and metastasis are intensified post AC026904.1 up-regulation.	[162]
PVT1	TCGA dataset: 19 TNBC and ANTs	TN	BT549, MCF7, T47D, MDA-MB-231, MDA-MB-453, MDA-MB-468, MCF10A, ZR-7530	KLF5, BAP1, β-catenin		PVT1 knockdown results in reduced cell proliferation, colony formation, and tumor growth.	[162]
	30 BC and ANTs	–	MCF10A, MCF7, MDA-MB-231, MDA-MB-436	miR-543, TRPS1	–	PVT1 promotes cell growth and motility, and lowers apoptosis.	[138]
linc-ZNF469-3	435 BC cases	TN: 212 Non-TN: 223	MDA-MB-361, MCF7, BT483, AU565, SKBR3, MDA-MB-157, MDA-MB-231, BT549, HCC1599, HCC1806, HS578 T	miR-574-5p, ZEB1	–	Linc-ZNF469-3 up-regulation elevates invasion, stemness, and lung metastasis.	[90]
NNT-AS1	64 BC and ANTs	Hormone receptor-positive: 32 HER2 positive: 33	MD-MB-231, MD-MB-468, MCF7, MCF10A	miR-142-3p, ZEB1	–	Knockdown of NNT-AS1 impedes proliferation, migration, and EMT of BC cells.	[105]
LINC01116	64 BC and 30 normal breast tissues	–	MCF7, MDA-MB-21, HCC38, MCF10A	miR-145, ESR1	–	LINC01116 expression associates with tumor size, TNM stage, and TNM stage in patients. LINC01116 escalates the viability and colony formation of BC cells.	[9]
BANCR	65 BC and ANTs	ER positive: 42 PR positive: 42 HER2 positive: 20	MCF7, MDA-MB-231, SKBR3, BT-20, MCF10A	BAX, PARP, MMP2, MMP9		BANCR increased cell proliferation, colony formation, invasion, metastasis, and EMT but plummeted the apoptosis. BANCR is related to TNM stage, tumor size, and lymph node metastasis.	[105]

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Table 1 (*continued*)

lncRNA	Clinical Samples	Classification	Assessed Cell Lines	Targets / Regulators	Signaling Pathways	Description	Reference
FOXC2-AS1	56 BC and ANTs	–	MDA-MB-468, MDA-MB-231, MDAMB- 436, MCF7, MCF10A	cyclin D1, cyclin D2, cyclin D3	–	FOXC2-AS1 boosts proliferation and cell cycle progression, but diminishes apoptosis. FOXC2-AS1 associates with tumor grade, lymph node metastases, HER2 status, and TNM stage.	[197]
MANCR	TCGA dataset of 960 BC patients	ER positive: 530 PR positive: 463	MCF10A, MDA-MB-231, MCF7	–	–	MANCR soars cell proliferation, cell cycle progression, and viability.	[172]
SNHG20	20 BC and ANTs	–	SK-BR-3, MDA-MB-453, MCF10A	miR-495, HER2	–	SNHG20 intensifies proliferation, invasion, and migration.	[45]
SNHG15	58 BC and ANTs	–	MCF7, BT-20, ZR-75-1, MDA-MB-231, MCF10A, SKBR3	miR-211-3p	–	SNHG15 expression is consistent with TNM stage, lymph node metastasis. SNHG15 expands the proliferation, migration, and EMT rate but lessens the apoptosis rate.	[74]
CAMTA1	–	–	MDA-MB-231	miR-20b, VEGF, MAPK, ERK, JAK, STAT1, STAT3	MAPK/ ERK, JAK/ STAT	lncCAMTA1 improves cell viability, migration, invasion, and mitigates apoptosis.	[107]
Z38	110 BC and ANTs	ER positive: 50 PR positive: 97 HER2 positive: 78	–	–	–	Z38 expression axiomatically conforms with TNM stage and lymph node metastasis.	[127]
BCRT1	68 BC patients	ER positive: 58 PR positive: 55 HER2 positive: 3	MCF10A, MCF7, MDA-MB-231, MDA-MB-468, HEK293 T, T47D, THP1	miR-1303, PTBP3 / HIF-1α	–	LncRNA BCRT1 increases tumor growth, proliferation, and metastasis. This lncRNA is related to distant metastasis and clinical stage.	[95]
ERINA	TCGA dataset: 559 BC patients	–	BT-20, HCC1937, HS578 T, MDA-MB-MB231, ZR-75-1, BT-474, MDA-MB-231, MCF7, T47D, HEK293 T, MCF10A	E2F1, RB1 / ER	–	ERINA up-regulates cell cycle progression, proliferation, Palbociclib resistance, and tumor growth.	[110]
LCPAT1	51 BC and ANTs	–	MCF10A, T47D, SKBR3, BT549, MCF7, MDA-MB-231	RBBP4, MFAP2	–	LCPAT1 raises BC cell proliferation, migration, tumor growth, and invasion while mitigating apoptosis.	[40]
ST8SIA6-AS1	138 BC and 73 ANTs	ER positive: 106 PR positive: 88	MDA-MB-231, ZR-75-30, Hs578 T, BT-549	AKT1, p38	MAPK	ST8SIA6-AS1 is consistent with ER-negative, PR-negative, advanced TNM stage. It also escalates proliferation, tumor growth, invasion, and migration of BC cells.	[110]
SPRY4-IT1	101 BC patients	–	MCF7, T47D	miR-6882-3p, TCF7L2	Wnt/ β-catenin	SPRY4-IT1 elevates proliferation and stemness in BC cells.	[154]
RP11-19E11.1	TCGA dataset: 1183 RNA-seq data	–	MDA-MB-231, Hs578 T, MDA-MB-157, MDA-MB-468, MCF7, SkBr3, BT474	PKC / E2F1	PI3K/AKT/ mTOR	This lncRNA improves BC cell proliferation, cell cycle progression, and survival and reduces apoptosis.	[39]
FAM83H-AS1	135 TNBC and 291 normal breast tissues	TN	DA-MB-231, MDA-MB-436, MDA-MB-468, MCF10A	miR-136-5p, MTDH	–	FAM83H-AS1 induces proliferation, migration, tumor growth, and invasion in BC cells	[207]
LINC00173	84 TNBC and ANTs	TN	MDA-MB-231, MDA-MB-468, BT-549	miR-490-3p	–	LINC00173 grows the proliferation, tumor growth, colony formation, and invasion rate of TNBC cells. The LINC00173 expression associates with lymph node metastasis and TNM stage.	[27]
SBF2-AS1	50 BC and ANTs	–	MCF7, MDA-MB-231, MCF10A	miR-143, RRS1	–	SBF2-AS1 promotes proliferation, viability, cell cycle, tumor growth, invasion, and migration but reduces apoptosis. SBF2-AS1 expression is related to LNM, tumor size, and clinical stage.	[187]
HOTAIRM1	60 paired primary and recurred tumors	–	MCF7, T47D, MCF7-TAMR, T47D-TAMR	HOXA1, EZH2, PRC2	–	HOTAIRM1 reduces sensitivity to Tamoxifen.	[71]
TTN-AS1	40 BC and ANTs	ER positive: 19 PR positive: 22 HER2 positive: 15	MCF7, MDA-MB-231, MDA-MB -453, BT474, ZR-75-30, MCF10A	miR-139-5p, ZEB1		TTN-AS1 promotes proliferation, migration, invasion, and EMT. It also correlates with higher TNM stage and lymph node metastasis.	[29]
BDNF-AS	250 BC samples	–	MCF7, T47D, MDA-MB-231	RNH1, TRIM21 / MEF2A	mTOR	BDNF-AS expression evidently correlates with Tamoxifen resistance, higher histopathological grading, larger tumor size, lymph node, distant metastasis, and advanced disease staging.	[96]
NONHSAT141924	35 BC and ANTs	ER positive: 15 PR positive: 18	BT-549, MDA-MB-231, Hs-578 T, ZR-75-30, T47D, MCF7, MCF10A	Bcl-2, p-CREB	–	NONHSAT141924 enhances proliferation and Paclitaxel resistance but not migration.	[18]

(*continued on next page*)

Table 1 (*continued*)

lncRNA	Clinical Samples	Classification	Assessed Cell Lines	Targets / Regulators	Signaling Pathways	Description	Reference
TROJAN	16 BC and ANTs, and another 92 BC tissues	HER2 positive: 21 ER positive: 92	MCF7, T47D, HEK293 T	NKRF, RELA, MKI67, CDK2	NF-κB	TROJAN promotes ER$^+$ BC resistance to Palbociclib and proliferation by inducing G1/S transition.	[69]
NONHSAT028712 (Lnc712)	10 BC and ANTs	–	MDA-MB-231, MCF7, MCF10A	HSP90, CDC37, CDK2	–	Cell proliferation rate is raised by Lnc712 up-regulation.	[18]
GAS6-AS1	60 BC and ANTs	ER positive: 15 PR positive: 17 HER2 positive: 20	MCF7, MDA-MB-231, SKBR-3, MDA-MB-468, MCF10A	miR-324 − 3p, SETD1A	PI3K/AKT	GAS6-AS1 intensifies proliferation, migration, tumor growth, and invasion and shortens the apoptosis rate. Its expression also matches tumor size, TNM stage, distant, and lymph node metastasis	[218]
NR2F1-AS1	–	–	MDA-MB-231, MCF7, HUVECs	IGF-1	ERK	NR2F1-AS1 boosts BC angiogenesis.	[208]
MCM3AP-AS1	–	–	MCF7, BT-549, MDA-MB-468, HS578 T, MCF10A	miR-28-5p, CENPF	–	Proliferation, migration, invasion, and tumor growth are induced by MCM3AP-AS1.	[14]
LOC645166	48 BC samples	–	MDA-MB-231, MCF7	GATA3, STAT3	NF-κB	LOC645166 promotes Adriamycin resistance, tumor growth, and metastasis.	[217]
DLG1-AS1	66 TNBC and ANTs	TN	BT-549, MDA-MB-157, MCF10A, MDA-MB-231, MDA-MB-468, BT20, CAL51	miR-203	–	DLG1-AS1 expression level correlates with clinical stages, and elevates migration and proliferation rate.	[110]
OIP5-AS1	70 BC and ANTs	ER positive: 40 PR positive: 35 HER2 positive: 27	MCF7, MDA-MB-231, T47D, SK-BR-3, BT-549, MCF10A	miR-129-5p, SOX2	–	OIP5-AS1 expression is consistent with tumor size, lymph node metastasis, pathological grading, and TNM stage. OIP5-AS1 knockdown reduces BC cell growth, proliferation, and migration, but promotes apoptosis.	[205]
	30 BC and ANTs	–	SK-BR-3, MDA-MB-231, MCF10A	miR-216a-5p, GLO1	–	OIP5-AS1 induces proliferation, migration, and invasion but diminishes the apoptosis rate.	[186]
CCAT2	25 BC and 10 normal breast samples	–	MDA-MB-231, MCF7, T47D, MDAMB- 231, Hs578t	miR-205, OCT4-PG1	Notch	CCAT2 improves stemness, cell proliferation, migration, and invasion.	[195]

Table 2
Down-regulated lncRNAs in breast cancer (BC: breast cancer, ANTs: adjacent non-cancerous tissues).

lncRNA	Clinical Samples	Classification	Assessed Cell Lines	Targets / Regulators	Signaling Pathways	Description	Reference
NKILA	–	–	MDA-MB-231, HUVEC	IL-6, IκBα, p65, VEGFA, VEGFR	NF-κB	NKILA mitigates cell proliferation, migration, EMT, angiogenesis and extends apoptosis of BC cells.	[110]
	164 BC patients	–	MCF7, BT474	TGF-β	NF-κB	NKILA up-regulation markedly restricts metastasis and EMT.	[185]
	–	–	MCF7, MDA-MB-231, HMEC	EZH2	NF-κB	migration and invasion are both promoted by NKILA expression.	[26]
LINC00899	26 BC and ANTs	–	BT549, T47D, MCF7, SKBR3, MDA-MB-231, MCF10A	miR-425, DICER1	–	LINC00899 overexpression reduces proliferation, migration, and invasion of BC cells.	[152]
WT1-AS	62 TNBC and ANTs	TN	BT-549	TGF-β1	TGF-β	WT1-AS plummets migration and invasion of TNBC cells and correlates with the clinical stage of BC cases.	[155]
LINC01133	74 BC and ANTs	ER positive: 40 PR positive: 30 HER2 positive: 47	MDA-MB-231, SKBR-3, MDA-MB-468, ZR-75-1, BT474, MCF7, T47D, MCF10A	EZH2, SOX4	–	LINC01133 up-regulation drops invasion, migration, and metastasis rates.	[155]
GAS5	98 TNBC and ANTs	TN	MDA-MB-231, MDA-MB-468, HCC1937, MDA-MB-453, MCF10A	–	–	GAS5 inhibits proliferation and tumor growth, but induces chemo-sensitivity and apoptosis.	[81]
	26 primary BC tissues	ER positive: 13 PR positive: 11 HER2 positive: 12	MCF7/ADR, MCF7	ABCB1, miR-221-3p, DKK2	Wnt/b-Catenin	GAS5 overexpression promotes Adriamycin sensitivity and apoptosis.	[15]
	103 TNBC and 50 ANTs	TN	MDA-MB-231, MDA-MB-468, MCF7, T47D, BT474, MCF10A	miR-196a-5p	FOXO1/ PI3K/ AKT	GAS5 expression correlates with tumor size, histological grade, increased apoptosis rate, lower proliferation rate, and lymph node metastasis.	[87]
	68 BC and ANTs	–	MCF7	miR-222, PTEN	–	GAS5 correlates with advanced TNM stage, Tamoxifen sensitivity, and tumor size.	[42]
	68 BC and ANTs	ER positive: 40 PR positive: 42 HER2 positive: 23	MDA-MB-231, MDA-MB-453, SK-BR-3, MCF10A, MCF7, BT549	miR-23a, ATG3	–	Lower GAS5 expression is consistent with larger tumor size, advanced TNM stage, and ER-negative BC tissues. GAS5 soars autophagy and autophagosome formation.	[43]

(*continued on next page*)

Table 2 (*continued*)

lncRNA	Clinical Samples	Classification	Assessed Cell Lines	Targets / Regulators	Signaling Pathways	Description	Reference
	156 BC and ANTs	–	MDA-MB-231, BT549	miR-378a-5p, SUFU	–	This lncRNA promotes apoptosis and sensitivity to Paclitaxel.	[218]
LINC01355	48 BC and ANTs	–	MCF7, MDA-MB-231, MCF10A, T47D, BT474	CCND1, FOXO3	–	LINC01355 up-regulation demonstrably restricts proliferation, cell cycle progression, colony formation, and tumor growth and correlates with tumor size and TNM stage.	[3]
AC073284.4	35 BC and ANTs	–	MCF7, SKBR3	miR-18b-5p, DOCK4	–	AC073284.4 attenuates invasion, paclitaxel-resistance, metastasis, and EMT.	[194]
LINC00968	52 BC and ANTs	ER positive: 31 PR positive: 35 HER2 positive: 24	BT-20, MCF7, MDA-MB-231, T47D, MCF10A	hsa-miR-423-5p, PROX1	–	LINC00968 reduces BC cell proliferation, migration, tumor growth, and tube formation. It also associates with histological grading, clinical grading, and lymph node metastasis of BC patients.	[72]
	42 BC and ANTs	–	MCF7, KPL-4	WNT2, HEY1, GSK3β	Wnt/ β-catenin	LINC00968 diminishes drug resistance, colony formation, EMT, tumor growth, migration, and invasion but improves apoptosis rate.	[192]
PTENP1	52 BC and ANTs	–	MDA-MB-231, T47D, MCF7, MCF10A	miR-20a, PTEN	PI3K/AKT	PTENP1 lessens cell proliferation, invasion, colony formation, tumor growth, and chemoresistance to Adriamycin.	[33]
TCONS_l2_00002973	96 TNBC and ANTs	TN	BT474, MCF7,MDA-MB-453, MDA-MB-231, MCF10A	Caspase-3, p38	–	Silencing this lncRNA is related to increased T stage, raised N stage, and advanced TNM stage. It also diminishes cell proliferation and enhances the cell apoptosis rate.	[196]
LIFR-AS1	30 BC and ANTs	–	Hs578Bst, MDA-MB-415, MDA-MB-231, MDA-MB-468, MCF7	miR-197-3p, Sufu	–	LIFR-AS1 plummets BC proliferation, colony formation, migration, and invasion.	[91]
MIR503HG	94 primary TNBC and 30 ANTs	TN	MDA-MB-231, TB549, MCF10A	miR-103, OLFM4	–	MIR503HG expression conforms with a late clinical-stage, lymph node metastasis, and distant metastasis. MIR503HG restricts cell migration and invasion.	[31]
PTCSC3	Blood and tissue samples of 69 TNBC cases	TN	BT-549, HCC70	H19	–	PTCSC3 suppresses proliferation rate, but not the migration and invasion rates.	[180]
EGOT	258 BC and ANTs	–	MCF7, T47D, UACC-812, SK-BR-3, MDA-MB-453, MDA-MB-231, Hs578 T, HCC70, BT549, MDA-MB-468	ITPR1 / Estrogen, NRIP1, AP-1	–	Hypoxia induces EGOT expression, which further promotes autophagy and Paclitaxel-sensitivity.	[211]
NRON	70 TNBC and ANTs	TN	Hs578 T, BT-549	snaR	–	NRON overexpression lowers cancer cell proliferation and correlates with clinical stages.	[128]
sONE	80 BC and ANTs	ER positive: 53 PR positive: 53 HER2 positive: 21 TN: 25	MDA-MB-231, MCF-7	eNOS, NO, TP53, c-Myc, miR-34a, miR-15, miR-16, let-7a	–	cellular viability, proliferation, colony-forming ability, migration, and invasion are decreased by sONE. Its expression associates with tumor size, lymph node metastasis, and age of BC patients.	[202]
NORAD	TCGA dataset: 58 BC and ANTs	–	293 T, 293FT, H460, MDA-MB-231, Hs578 T, T47D, ZR75	S100 P / YAP, TAZ-TEAD	Hippo	NORAD matches lymph node metastasis and inhibits migration and invasion.	[161]
LINC00472	525 BC patients	ER positive: 360 PR positive: 302	MCF7, T47D, MDA-MB-231, Hs578 T, SKBR3, ZR-75-1, 293 T	IKKβ, p65, IκBα / ERα	NF-κB	LINC00472 dwindles proliferation, migration, tumor growth, and invasion.	[181]
FGF13-AS1	60 BC and ANTs	Luminal like: 33 HER2 positive: 19 TN: 8	MCF10A, MCF7, T47D, MDA-MB-453, MDA-MB-468, MDA-MB-231, 293 T	c-Myc, IGF2BPs	–	FGF13-AS1 shortens the proliferation, migration, stemness, and invasion rate by impairing glycolysis. It is inversely correlated with lymph node metastasis and tumor stage.	[116]
TFAP2A-AS1	30 BC and ANTs	–	MCF7, MDA-MB-231, MDA-MB-435, T47D, SKBR-3	miR-933, SMAD2	–	TFAP2A-AS1 plummets cell cycle progression, tumor growth, and invasion, but promoted cell apoptosis. TFAP2A-AS1 tallies with tumor grade and TNM stage.	[152]
EPB41L4A-AS2	15 BC and ANTs and 3 normal breast tissues	–	UACC812, BT549, MDA-MB-453	MKI67, MYC / ZNF217, EZH2	TGF-β, BMP, Notch, MAPK	EPB41L4A-AS2 declines proliferation, migration, and invasion rate but raise cell apoptosis.	[133]
TUBA4B	38 BC and adjacent benign tissues	–	MCF7, ZR-75-1, MDA-MB-231, HCC-1937, MCF10A	miR-19	–	Proliferation and invasion are restricted TUBA4B expression.	[97]
LncKLHDC7B	156 TNBC	TN	MCF10A, BT20, MDA-MB-468, MDA-MB-231, Hs578-T, MCF7, HCC1187	KLHDC7B	–	LncKLHDC7B lessens migration and invasion, but elevate apoptosis rate.	[11]
MEG3	–	–	MDA-MB-231, MCF7		NF-κB	MEG3 up-regulation reduces tumor growth and promotes apoptosis.	[211]

(*continued on next page*)

Table 2 (*continued*)

lncRNA	Clinical Samples	Classification	Assessed Cell Lines	Targets / Regulators	Signaling Pathways	Description	Reference
				GRP78, IRE1, PERK, ATF6, CHOP, caspase-3, NF-κB, p53			
	20 primary BC and ANTs	–	MCF7, MDA-MB-231, MDA-MB-453, T47D, MCF10A	miR-21	PI3K/Akt	MEG3 overexpression suppresses cell proliferation, tumor growth, and glycolysis. MEG3 also correlates with the clinical stage of BC patients.	[225]
PDCD4-AS1	TCGA dataset: 814 BC and 105 normal cases	–	M1, M2, M3, M4	PDCD4	–	PDCD4-AS1 increases PDCD4 mRNA stability, a tumor suppressor, which inhibits migration.	[59]
NLIPMT	80 BC and ANTs	–	MDA-MB-231, SKBR3, MDA-MB-453, MCF7, BT474, BT549, MCF10A	GSK3β	Hedgehog	NLIPMT decreases proliferation, motility, metastasis, and tumor growth.	[68]
LINC01585	132 BC and ANTs	ER positive: 90 PR positive: 84 HER2 positive: 102	MCF7 cells, BCAP-37	CAMP, CREB / NONO	–	LINC01585 declines the proliferation and tumor growth rates.	[119]
LET	70 BC and ANTs	–	MCF10A, MDA-MB-231	–	–	LET inhibits cell proliferation, invasion, migration, EMT, but promotes cell apoptosis.	[221]
TUG1	20 TNBC and ANTs	TN	MCF7, T47D, MDA-MB-231, BT549, MCF10A	miR-197, NLK	WNT	TUG1 restricts cell viability in response to Cisplatin.	[166]
MAGI2-AS3	30 BC and ANTs	ER positive: 21 PR positive: 20 HER2 positive: 18	MDA-MB-231, MCF7, MCF10A	Fas, FasL	–	MAGI2-AS3 diminishes BC cell viability, tumor growth, and colony formation and increases apoptosis. MAGI2-AS3 knockdown inversely correlates with histological grade, TNM stage, ER expression, PR expression, and Her-2 expression.	[204]
PlncRNA-1	78 BC and ANTs and 45 healthy cases	–	MDA-MB-468, MCF7	TGF-β1, PHGDH	–	PlncRNA-1 suppresses the proliferation rate but elevates the apoptosis rate. It also matches with tumor diameter.	[83]
CTD-2108O9.1	97 BC and ANTs	–	MCF7, MDA-MB-231, ZR-75-1, MCF10A	LIFR	–	CTD-2108O9.1 reduces migration, invasion, and metastasis. Moreover, it is related to lymph node metastasis.	[179]
XIST	40 BC and ANTs	–	MCF7, ZR-75-1, HCC-1937, MDA-MB-231, MDA-MB-468, MDA-MB-453, MCF10A	miR-155, CDX1	–	XIST mitigates cell growth, migration, and invasion. XIST relates to lymph node metastasis and clinical stage.	[16]
ZFAS1	–	–	MDA-MB-231, MCF7, T47D, SK-BR-3, MCF10A	–	–	ZFAS1 mitigates proliferation cell cycle progression, migration, invasion, EMT, and excalates apoptosis in BC cells.	[28]
CASC2	35 BC and ANTs	ER positive: 22 PR positive: 18 HER2 positive: 12	MCF7, MDA-MB-231, MCF10A	miR-96-5p, SYVN1	–	CASC2 dwindles the viability, migration, invasion, tumor growth, and elevated apoptosis.	[34]
	52 BC and ANTs	–	HCC1937, LCC9, MDA-MB-231, MCF7	TGF-b, Smad2, a-SMA	TGF-β	CASC2 markedly shortens BC cell proliferation, cell cycle progression, migration, and metastasis.	[216]
RMST	48 TNBC and ANTs	TN	MCF7, AU565, BT-20, BT-549, MCF10A, HEK293, MB231, MB468	–	–	RMST reduces proliferation, migration, cell cycle progression, and invasion, but induces apoptosis.	[177]
PTENP1	20 BC and ANTs	–	MCF7, MDA-MB-231, MCF10A	miR-19b, PTEN, PDK-1	PI3k/Akt	PTENP1 lowers cell survival, colony formation, migration, and invasion but increases apoptosis.	[148]
SNORD3A	72 BC patients	–	MCF10A, MCF7, MDA-MB-231, T47D, SKBR3, ZR7530, BT549, HCC1937, BT474, HEK293 T	miR-185-5p, UMPS / Meis1	–	SNORD3A overexpression significantly sensitizes BC cells to 5-fluorouracil.	[112]
EGOT	50 BC and ANTs	–	BT549, MDA-MB-231, MCF7, SKBr3, HEK293	Gli1, HHIP, SMO, PTCH1	Hedgehog	EGOT diminishes cell viability, proliferation, and migration. EGOT correlates with higher tumor malignancy and Ki67 value.	[141]

Table 3
LncRNA polymorphisms and breast cancer (BC: breast cancer).

lncRNA	Number of Clinical Samples	Classification	SNP ID	Nucleotide change	Description	References
MEG3	144 blood samples from BC patients	ER positive: 102 PR positive: 114 HER2 positive: 52	rs10132552	T > C	Patients containing the T allele in rs10132552 have larger, more invasive tumors and a higher level of ki67. The TC + CC genotype associates with a higher pathological complete response rate compared with the TT genotype in BC patients.	[10]
	111 BC and 130 healthy subjects	–	rs2839698	C > T	T and TT alleles increase the BC risk.	[145]
			rs217727	C > T	T and TT alleles have protective roles against BC.	
H19	Blood samples of 150 BC and 100 healthy cases	–	rs217727	C > T	TT + TC/CC and CC + CT/TT did not significantly correlate with the risk of BC, ER, PR, or HER2 status, tumor stage, and age.	[1]
			rs3741219	T > C		
	2881 BC and 3220 healthy subjects	–	rs2071095	C > A	AA and CA + AA genotypes markedly reduce BC risk and correlate with BC risk in ER^+ patients, but not in ER^- cases.	[16]
Linc-ROR	484 BC and 484 healthy subjects	ER positive: 293 PR positive: 253 HER2 positive: 308	rs4801078	C > T	TT genotype axiomatically correlates with a higher risk of BC. Patients with CT + TT genotypes have increased BC risk in the participant with age >50 and age of menarche >13.	[109]
MALAT1	487 BC patients and 489 healthy controls	ER positive: 294 PR positive: 256 HER2 positive: 313	rs619586	A > G	Genotype AG diminishes the risk of BC. MALAT1 expression in BC patients with AG, GG, and AG + GG genotypes is markedly mitigated.	[136]
			rs3200401	C > T	Cases with CT genotype have a lower risk of BC.	

invasion of TNBC cells through inhibiting this cytokine [155]. Table 2 gives a summary of investigations which reported down-regulation of lncRNAs in breast cancer.

4. Role of lncRNA variants in breast cancer

Genomic variants within MEG3, H19, Linc-ROR and MALAT1 have been associated with risk of breast cancer in certain populations (Table 3). Notably, a number of these variants have been shown to affect certain subtypes of breast cancer. Moreover, genetic variants within lncRNAs might affect prognosis of breast cancer or response of the affected individuals to treatments. These variants may alter expression levels of the corresponding lncRNAs similar to what has been reported regarding the rs619586 MALAT1. However, the underlying mechanism of contribution of other lncRNAs variants in this process is not clear. Theoretically, these variants may affect the interaction between lncRNAs and their targets particularly miRNAs or transcription factors thus affecting carcinogenesis process without altering the expression levels of the related lncRNAs.

5. Interactions between lncRNAs and therapeutic agents in breast cancer

LncRNAs might affect the response of breast cancer cells to therapeutic agents. For instance, expression of CASC2 has been elevated in paclitaxel-resistant clinical specimens and cell lines. This chemotherapeutic agent has been shown to enhance CASC2 expression. CASC2 silencing has improved sensitivity to paclitaxel and diminished tumor growth in the animal model. CASC2 can sponge miR-18a-5p and enhance expression of CDK19 [216]. The oncogenic lncRNA BORG is highly expressed in TNBC cells exposed to environmental and chemotherapeutic stresses. This stress-associated activation of BORG expression raises the persistence of TNBC cells and makes them resistant to the anti-cancer impacts of doxorubicin. The effects of BORG on chemoresistance is mediated through induction of the NF-κB signaling pathway [41]. Table 4 summarizes the identified interactions between lncRNAs and therapeutic agents in breast cancer.

6. Prognostic/Diagnostic role of lncRNAs in breast cancer

Tissue or plasma levels of lncRNAs can be used for distinguishing breast cancer tissues/ patients from non-cancerous tissues/ healthy subjects. For instance, Zidan et al. have shown that circulating MALAT1 levels can differentiate breast cancer patients from controls with diagnostic sensitivity and specificity of 83.7 % and 81.2 %, respectively. Moreover, MALAT1 expression level has been correlated with lymph node positivity, ER status, clinical stage and histological grade [227]. Other lncRNAs namely H19, TATDN1, lncUSMycN, AWPPH and lncATB had diagnostic power of 69 %, 85 %, 70 %, 79 % and 85 %, respectively (Table 5). Expressions of different lncRNAs such as LUCAT1, LINC00673, LINC02582, lnc-SLC4A1-1, UFC1 and LINC00511 have been correlated with clinical outcome of patients (Table 5).

7. Discussion

LncRNAs have extremely heterogeneous functions in the development of breast cancer. These transcripts can adapt different structures and molecular interactions, thus having the ability to affect evolution of breast cancer from different aspects. Notably, a number of lncRNAs have specific expression in a certain subtype of breast cancer, based on the comparative expression profile studies. The functional association between lncRNAs and ESR1 gene as one of the most fundamental factors in the proliferation of breast cancer cells has been assessed by a number of studies, demonstrating possible interplay between TMPO-AS1, DSCAM-AS and LINC01116 lncRNAs and this nuclear factor. Therefore, aberrant expression of these lncRNAs might affect response of patients to anti-

Table 4
Interactions between lncRNAs and therapeutic agents in breast cancer (BC).

Drug	lncRNA	Expression pattern	Clinical Samples	Assessed Cell Lines	Targets / Regulators	Signaling Pathways	Description	Prognosis	Reference
Ginsenoside Rh2	C3orf67-AS1	↓	–	MCF10A, MCF-12A, MCF7, T47D, MDA-MB-231	C3orf67	–	Ginsenoside Rh2 downregulates C3orf67-AS1 via promoter hypermethylation and decreases tumor growth.	–	[61]
Paclitaxel	CASC2	↑	34 BC cases, half sensitive and half resistant to Paclitaxel	MCF7, MDA-MB-231, MCF10A	miR-18a-5p, CDK19	–	Paclitaxel enforces CASC2 expression in a concentration-dependent manner. CASC2 induces Paclitaxel resistance and tumor growth.	–	[216]
Delphinidin	HOTAIR	↓	–	MDA-MB-231, MCF7, MDA-MB-453,	miR-34a, β-catenin, Gsk3β, c-Myc, cyclin-D1, MMP-7	Wnt/ β-catenin	Delphinidin lowers HOTAIR mRNA level, proliferation, and migration rates.	–	[48]
Andes-1537	ASncmtRNA	↓	–	MDA-MB-231, MCF7, ZR-75-1	CDK1, cyclin B1, cyclin D1, hsa-miR-4485-3p, hsa-miR-5096, hsa-miR-3609	–	ASncmtRNA knockdown by Andes-1537 diminishes proliferation, cell cycle progression, tumor growth, and induces apoptosis.	–	[30]
AC1NOD4Q	HOTAIR	–	–	U87, LN229, MDA-MB-231	NLK, EZH2	Wnt/ β-catenin	AC1NOD4Q inhibits cell invasion, metastasis, tumor growth, and migration via up-regulating HOTAIR.	–	[144]
trastuzumab and lapatinib	BORG	↑	–	67NR, 4T1, 4T07	RPA1 / IKK, p65	NF-κB	Trastuzumab and Lapatinib, hypoxia, doxorubicin, hydroxyurea, docetaxel, 5-fluorouracil, 6-thioguanine, low glucose and glutamine induce BORG up-regulation. BORG protects against induced-apoptosis, DNA damage, and cell cycle arrest.	–	[41]
Bharangin	MEG-3 GAS-5 H19	↑ ↑ ↓	–	MCF7, MDA-MB-231, MDA-MB-453, MDA-MB-468, T47D	–	NF-κB	Bharangin reduces chemo-resistance and the expression of cell survival and invasive proteins.	–	[6]
Baicalein	PAX8-AS1-N	↑	76 BC and ANTs	MDA-MB-231, MCF7	miR-17-5p, PTEN, CDKN1A, ZBTB4		PAX8-AS1-N restricts cell viability, tumor growth, cell cycle progression and improves apoptosis of BC cells.	Lower OS rate	[204]

hormone therapies. A well-appreciated route of participation of lncRNAs in the pathogenesis of breast cancer is their sponging effect on miRNAs. GACAT3/miR-497/CCND2, MALAT1/miR-339-5p/BLCAP, LUCAT1/miR-5702, LUCAT1/miR-5582-3p/TCF7L2, LINC00673/ miR-515-5p/MARK4, LINC02582/miR-200c/CHK1, SNHG3/miR-384/hepatoma-derived growth factor, UFC1/miR-34a, LINC00511/miR-185-3p/E2F1/Nanog, LINC00511/miR-185/STXBP4 and LINC00511/miR-29c/CDK6 are among the molecular axes that affect development of breast cancer or response of patienst to chemotherapeutic substances.

Notably, most of dysregulated lncRNAs in breast cancer particularly MALAT1, NEAT1, HOTAIR, HOTTIP and H19 have been previously shown to be dysregulated in other cancer types [37,38], thus emphasizing on their oncogenic roles. Therefore, therapeutic intervention with their expression is expected to be a practical treatment method for several types of cancer.

A number of lncRNAs have alternative splice variants with different roles in the pathogenesis of breast acncer and distinctive signatures among breast cancer molecular subtypes, highlighting the neseccity for precise expression profiling using transcript-specific primers/ probes.

Breast cancer-associated lncRNAs have functional interpalys with Hippo, PI3K/AKT, TGF-β, NF-κB, STAT, Wnt/β-catenin and Notch pathwyas. Through modulation of these pathways, they regulate several aspects of invasiveness particularly epithelial-mesenchymal transition. A number of lncRNAs namely MAAT1, NR2F1-AS1 and RAB11B-AS1 and have been shown to affect tumor angiogenesis, further supporting tumor growth. On the other hand, NKILA and LINC00968 have anti-angiogenic properties in this kind of cancer.

Circulating levels of lncRNAs have the potential to be used as diagnostic markers for breast cancer. Moreover, tissue signature of lncRNAs have been correlated with clinical outcome of pateinst with braest cancer. Therefore, lncRNAs represent a valuable source for prediction of course of breast cancer and suitable targets for therapeutic interventions. Recent advances in monitoring lncRNAs levels in the peripheral blood raised hope for application of these markers for monitoring disease status in a non-ninvasive manner. Although therapeutic interventions with lncRNAs expressions have been promising in animal models, these results have not been tested in humans yet. As lncRNAs regulate expression of genes at almost all levels, such interventions are expected to combat carcinogenesis more efficiently compared with other targeted therapies. Therefore, future investigation of these results in human subjects is required.

Declaration of Competing Interest

The authors declare they have no conflict of interest.

Table 5
Prognostic/Diagnostic role of lncRNAs in breast cancer (BC: Breast Cancer, ANT: Adjacent Normal Tissue, TN: Triple Negative, TNBC: Triple Negative Breast Cancer, DFS: Disease-Free Survival, RFS: Relapse-Free Survival, DMFS: Distant Metastasis-Free Survival, PFS: Progression-Free Survival, PR: Progesterone Receptor, ER: Estrogen Receptor).

lncRNA and Clinical Cases	Area under curve	Sensitivity	Specificity	Kaplan-Meier Analysis	Univariate Cox Regression	Multivariate Cox Regression	Reference
626 BC cases expressing low (= 259) and high (= 367) levels of Linc00839	–	–	–	Linc00839 expression and OS rate are inversely correlated with each other.	–	–	[13]
TMPO-AS1 expression in BC cases: high = 32, low = 83	–	–	–	Lower TMPO-AS1 expression is related to higher OS, DFS, and RFS rates in BC patients.	Pathological T and N factors and TMPO-AS1 expression significantly correlate with OS and DFS rates.	The pathological N factor and TMPO-AS1 expression significantly correlate with OS and DFS rates. The pathological T factor only correlates with the DFS rate.	[124]
Kaplan Meier Plotter data of BC cases expressing NEAT1: high = 1332, low = 1187	–	–	–	NEAT1 expression in BC cases is negatively correlated with OS rate.	–	–	[171]
Low and high levels of LncRNA GACAT3 expression in BC cases, each containing 10 subjects	–	–	–	Higher expression levels of this lncRNA are associated with shorter OS rates.	–	–	[56]
41 BC patients with high and low amounts of GACAT3 expression	–	–	–	GACAT3 expression is inversely correlated to the OS rate of BC patients.	–	–	[220]
MALAT1 expression in BC cases aged less than 60: high = 94, low = 415, and in ductal cases: high = 12, low = 94	–	–	–	Lower MALAT1 expression is related to a higher OS rate in ductal BC cases and patients less than 60 years old.	–	Potential prognostic factors are classified as: Over 60-year-old: lymph node metastasis Below age 60: MALAT-1 expression and lymph node metastasis Lobular: age and lymph node metastasis Ductal: MALAT-1 expression, age, and lymph node metastasis	[215]
MALAT1 and BACH1 expression in 240 TNBC cases: both positive:88, one positive:87, both negative:65	–	–	–	patients overexpressing MALAT1 and BACH1 exhibit shorter OS and DFS	–	–	[131]
LINC02273 expression in BC cases: high = 150, low = 169	–	–	–	LINC02273 higher expression results in lower RFS rate.	LINC02273 expression and lymph node metastasis correlate with RFS rate.	LINC02273 expression and lymph node metastasis correlate with RFS rate.	[191]
BCLIN25 expression in BC cases: high = 160, low = 141	–	–	–	Higher BCLIN25 expression is related to a lower OS and DFS rate.	–	–	[193]
High (= 746) and low (= 343) HOXC-AS3 expressing BC subjects	–	–	–	HOXC-AS3 and OS rate of BC cases are inversely correlated with each other.	–	–	[198]
Two groups of DLX6-AS1 expression in BC patients: high = 23, low = 22	–	–	–	DLX6-AS1 expression is associated with a lower OS rate.	–	–	[212]
TCGA dataset: LncCCAT1 expression in BC cases divided into two groups of low and high, each containing 280 cases	–	–	–	Higher LncCCAT1 levels are related to lower OS rates.	–	–	[167]
H19 expression in response to neoadjuvant chemotherapy in blood samples of 55 BC cases	69 %	–	–	–	–	–	[132]
BC cases expressing H19: high = 21, low = 27	–	–	–	H19 expression and PFS rate are inversely associated with each other.	–	–	[160]
Higher and lower expression groups of LUCAT1 in TNBC patients, each containing 47 cases	–	–	–	LUCAT1 expression and OS rate of TNBC cases are negatively correlated with each other.	–	–	[126]
LUCAT1 expression in BC cases: high = 88, low = 63	–	–	–	Higher LUCAT1 level is associated with lower OS and DFS rates.	–	–	[214]
	–	–	–		–	–	[140]

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Table 5 (*continued*)

lncRNA and Clinical Cases	Area under curve	Sensitivity	Specificity	Kaplan-Meier Analysis	Univariate Cox Regression	Multivariate Cox Regression	Reference
Low and high expressing groups of LINC00673, each containing 40 cases				LINC00673 expression is inversely correlated with the OS rate.			
LINC02582 expression in BC cases: high = 71, low = 65	–	–	–	Lower LINC02582 levels are related to a higher RFS rate.	–	–	[174]
lnc-SLC4A1-1 expression in BC cases: high = 30, low = 36	–	–	–	Higher lnc-SLC4A1-1 expression is correlated with a lower OS rate.	–	–	[201]
High and low expression levels of UFC1 in ductal BC cases	–	–	–	UFC1 expression and survival rate are inversely related with each other.	–	–	[188]
LINC00511 expression in 39 BC cases: high = 23, low = 16	–	–	–	Patients with higher expression levels of LINC00511 have lower OS rates.	–	–	[106]
LINC00511 expression in BC cases divided into two groups of high and low, each with 49 patients	–	–	–	LINC00511 is highly expressed in BC cases with a shorter OS rate.	–	–	[101]
Higher (= 24) and lower (= 46) expression levels of LINC00511 in BC cases	–	–	–	Lower LINC00511 level results in a higher OS rate in BC patients.	–	–	[175]
High (= 20) and low (= 25) levels of MIR210HG in invasive BC cases	–	–	–	Lower MIR210HG expression is associated with higher OS and DFS rates.	–	–	[80]
TATDN1 expression in 24 BC and ANTs	0.8567	–	–	–	–	–	[178]
KM Plotter database: LINC01133 expression in 153 BC cases	–	–	–	Higher LINC01133 expression is correlated with a lower OS rate.	–	–	[173]
ADPGK-AS1 expression in two groups of high and low, each containing 37 cases	–	–	–	ADPGK-AS1 higher levels are related to lower OS rates.	–	–	[68]
LINC00473 expression in BC cases: high = 26, low = 34	–	–	–	Lower LINC00473 indicates a higher level of OS in BC patients.	–	–	[129]
BC cases with high (= 60) and low (= 62) expression levels of LINC00473	–	–	–	Higher LINC00473 expression is associated with a lower OS rate.	–	–	[7]
High (= 55) and low (= 57) expression levels of LINC01287	–	–	–	LINC01287 expression and OS rate are inversely correlated with each other.	Lymph node metastasis, TNM stage, and LINC01287 are potential prognostic factors for OS.	Lymph node metastasis, TNM stage, and LINC01287 significantly correlate with OS.	[151]
ATXN8OS expression in BC patients: high = 58, low = 62	–	–	–	Higher ATXN8OS expression is related to a lower OS rate.	–	–	[19]
High (= 31) and low (= 41) SNHG7 expressing BC patinets	–	–	–	Lower SNHG7 expression level is associated with a higher OS rate.	–	–	[113]
lncUSMycN expression in 52 BC and ANTs	0.70	0.85	0.55	–	–	–	[36]
Two groups of high and low expression of MIF-AS1 in BC, each with 41 subjects	–	–	–	MIF-AS1 and OS rate of BC subjects are negatively associated with each other.	–	–	[20]
TANCIR dataset of RHPN1-AS1 expression in BC subjects	–	–	–	OS rate in BC cases is lower when RHPN1-AS1 is highly expressed.	–	–	[226]
High (= 58) and low (= 39) expression levels of RP1 in BC subjects	–	–	–	Lower RP1 expression is associated with a higher OS rate.	–	–	[88]
AWPPH expression in plasma samples of 72 TNBC and 44 healthy cases	0.7980	–	–	–	–	–	[98]
TCGA dataset of 1108 BC samples for HOTAIR expression: high = 595, low = 332	–	–	–	An up-regulated HOTAIR expression is associated with a lower OS rate.	–	–	[12]
HOTAIR expression in BC cases	–	–	–	Higher HOTAIR expression is an indicator of lower OS rate.	–	–	[213]
NAMPT-AS expression in BC patients: high = 36, low = 28	–	–	–	NAMPT-AS expression is inversely correlated with OS and RFS rates.	Lymph node metastasis, TNBC, M1 stage, node metastasis, tumor grade,	NAMPT-AS is a potential prognostic factor for OS and RFS.	[169]

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Table 5 (*continued*)

lncRNA and Clinical Cases	Area under curve	Sensitivity	Specificity	Kaplan-Meier Analysis	Univariate Cox Regression	Multivariate Cox Regression	Reference
					clinical stage, and NAMPT-AS expression correlate with OS and RFS rates. HER2 status only correlates with the RFS rate.		
AGAP2-AS1 expression in trastuzumab responsive (= 33) and non-responsive (= 29) BC patients	0.753	78.7 %	63.7 %	–	–	–	[23]
High (= 25) and low (= 32) expression levels of DANCR in TNBC subjects	–	–	–	Lower DANCR expression in TNBC cases is related to a higher OS rate.	–	–	[169]
Two groups of 30 patients with high and low levels of DANCR expression	–	–	–	Higher DANCR expression is associated with a shorter OS rate.	–	–	[163]
ZEB2-AS1 expression in two groups of high and low, each with 49 cases	–	–	–	ZEB2-AS1 expression and survival rate are negatively associated with each other.	–	–	[41]
Low (= 183) and high (= 179) expression levels of LOL in luminal BC cases	–	–	–	Higher LOL expression indicates a lower OS and DFS rate.	Tumor size and grade, lymph node metastasis, Ki-67, and LOL expression are potential prognosis factors of the DFS rate.	Ki-67 and LOL expression, tumor size, tumor grade, and lymph node metastasis are prognosis indicators of DFS.	[157]
Two groups of BC patients expressing high and low amounts of HOTTIP, each with 35 subjects	–	–	–	Lower HOTTIP expression is an indicator of a higher OS rate.	–	–	[32]
Lower and higher expression groups for HIF1α-AS2, each with 43 cases	–	–	–	Patients with lower HIF1α-AS2 expression levels have higher rates of OS.	Clinical stage, distant and lymph node metastasis, and HIF1α-AS2 expression correlate with the OS rate.	HIF1α-AS2 expression and distant metastasis are possible prognostic factors for OS rate.	[189]
FOXD2-AS1 expression in BC cases: high = 19, low = 15	–	–	–	Higher FOXD2-AS1 expression in BC cases indicates a lower OS rate.	–	–	[22]
High (= 34) and low (= 33) LINC01857 expression levels in BC cases	–	–	–	Higher LINC01857 is associated with a lower OS rate.	–	–	[189]
TINCR expression in 60 primary BC cases and 30 trastuzumab responsive and 30 non-responsive cases:	0.833	76.7 %	70.0 %	Lower TINCR expression is an indicator of more desirable OS and PFS rates.	Lymph node and distant metastasis, TNM stage, and TINCR expression correlate with the PFS rate.	TNM stage, distant metastasis, and TNICR expression are associated with the PFS rate.	[22]
Two groups of 12 BC cases with high and low expression levels of TINCR	–	–	–	Up-regulated TINCR expression is associated with a lower OS rate.	–	–	[102]
LINC00152 expression in 572 BC cases from TCGA dataset	–	–	–	–	LINC00152 expression, age, ER and PR status, TNM stage, tumor size, and lymph node metastasis are correlated with OS rate.	TNM stage, tumor size, LINC00152 expression, PR status, and age are associated with the OS rate.	[147]
LINP1 expression in BC cases: high = 90, low = 93	–	–	–	LINP1 expression in inversely correlated with OS and DFS rates.	Lymph node metastasis, TNM stage, pathological differentiation, and LINP1 expression are correlated with OS and DFS rates.	Lymph node metastasis, TNM stage, pathological differentiation, and LINP1 expression are potential prognostic factors for OS and DFS rates.	[100]
Higher (= 33) and lower (= 34) expression levels of LINP1 in BC patients	–	–	–	Higher levels of LINP1 expression are related to lower OS and DFS rates.	LINP1 expression and lymph node metastasis correlate with the OS rate.	LINP1 expression and lymph node metastasis are possible prognostic factors for OS rate.	[94]
High (= 92) and low (= 39) expression levels of lncATB in BC cases	0.851	–	–	Lower lncATB expression is associated with a higher OS and DFS rate.	–	–	[84]
DSCAM-AS1 expression in TCGA dataset, BC cases: high = 239, low = 160	–	–	–	Higher DSCAM-AS1 expression is markedly related to a lower DFS rate.	Tumor size and grade, lymph node metastasis, Ki-67, and DSCAM-AS1 expression are associated with the DFS rate.	lymph node metastasis, Ki-67, and DSCAM-AS1 expression are potential prognostic factors for DFS rate.	[156]
TCGA dataset of 512 BC cases expressing BLAT1	–	–	–	BLAT1 expression and OS rate are negatively correlated with each other.	–	–	[182]
	0.8927	–	–		–	–	[24]

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Table 5 (*continued*)

lncRNA and Clinical Cases	Area under curve	Sensitivity	Specificity	Kaplan-Meier Analysis	Univariate Cox Regression	Multivariate Cox Regression	Reference
AWPPH expression in TNBC cases: high = 34, low = 34				Patients with a higher AWPPH expression have a lower OS rate			
Two groups of 18 BC patients with high and low amounts of TP73-AS1 expression	–	–	–	TP73-AS1 level is inversely related to OS.	Tumor size and TP73-AS1 expression correlate with OS.	TP73-AS1 is a promising prognostic factor for OS rate.	[65]
Expression of SNHG14 after trastuzumab treatment: responsive = 33, non-responsive = 29	0.796	69.8 %	81.4 %	–	–	–	[24]
MIR100HG expression in TCGA dataset of BC patients: high = 90, low = 270	–	–	–	Higher MIR100HG expression evidently results in a lower OS rate	–	–	[111]
LINC01638 expression in BC cases: high = 49, low = 92	–	–	–	Lower LINC01638 expression is associated with a higher OS rate.	–	–	[111]
TCGA dataset of LINC00310 expression in BC cases, and 48 BC cases and 47 healthy controls	0.828	77.08 %	87.23 %	Higher LINC00310 expression is related to lower OS and DFS rates.	–	–	[46]
LINC01296 expression in BC cases: high = 30, low = 25	–	–	–	LINC01296 expression is associated with a lower OS rate.	LINC01296 is associated with the OS rate of BC patients.	LINC01296 is a potential prognostic factor for OS rate.	[65]
Expression of AFAP1-AS1 in BC cases: high = 86, low=74	0.736	74 %	69 %	AFAP1-AS1 expression is negatively correlated with the OS rate.	Tumor grade, TNM stage, lymph node metastasis, and AFAP1-AS1 expression are associated with the OS rate.	TNM stage and AFAP1-AS1 expression are possible prognostic factors for OS rate in BC patients.	[115]
AFAP1-AS1 expression in $HER2^{+}$ BC patients: high = 32, low = 32	–	–	–	A lower AFAP1-AS1 expression is an indicator of a higher OS rate.	Clinical stage, AFAP1-AS1 expression, and distant or lymph node metastasis are correlated with the OS rate.	AFAP1-AS1 expression, TNM stage, and lymph node metastasis are promising prognostic factors for OS rate.	[209]
lncRNA-BCHE expression in BC patients: high = 298, low = 366	–	–	–	Higher lncRNA-BCHE levels are in line with a lower DMFS and RFS rates.	–	–	[199]
84 BC patients expressing different amounts of MIAT	–	–	–	MIAT expression positively conforms with higher OS rate.	–	–	[78]
ARNILA expression in BC patients: high = 49, low = 39	–	–	–	Lower ARNILA levels are associated with higher PFS rates.	–	–	[107]
BC patients with high and low expression levels of GHET1, each with 30 cases	–	–	–	GHET1 expression is inversely correlated with OS.	–	–	[153]
High (= 17) and low (= 13) expression levels of FEZF1-AS1 in BC patients	–	–	–	A lower OS rate is associated with a higher FEZF1-AS1 expression.	–	–	[79]
High and low expression levels of AC026904.1 in BC patients	–	–	–	Higher AC026904.1 expression is an indicator of shorter OS and DFS rates.	–	–	[162]
TCGA dataset of PVT1 expression in TNBC patients: high = 105, low = 104	–	–	–	A lower PVT1 expression in TNBC patients is related to a higher OS rate.	–	–	[162]
Lower (= 168) and higher (= 65) expression levels of linc-ZNF469-3	–	–	–	Increased linc-ZNF469-3 expression is associated with diminished OS and DFS rates.	–	–	[90]
Two groups of 32 patients with high and low expression levels of NNT-AS1	–	–	–	A down-regulated NNT-AS1 expression is related to a higher OS rate.	–	NNT-AS1 expression and hormone receptor status are promising prognostic factors.	[105]
LINC01116 higher and lower expression levels in two groups of BC cases, each with 32 subjects	–	–	–	Up-regulated LINC01116 expression is correlated with a shorter OS rate.	–	–	[9]
High (= 34) and low (= 31) expression of BANCR in BC subjects	–	–	–	A lower BANCR expression is evidently associated with higher rates of OS and DFS.	–	–	[105]
FOXC2-AS1 expression in BC cases: high = 23, low = 23	–	–	–	FOXC2-AS1 expression and OS rate of BC patients are	–	Up-regulated FOXC2-AS1 is an independent prognostic factor.	[197]

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Table 5 (*continued*)

lncRNA and Clinical Cases	Area under curve	Sensitivity	Specificity	Kaplan-Meier Analysis	Univariate Cox Regression	Multivariate Cox Regression	Reference
				negatively associated with one another.			
TCGA dataset of MANCR expression in BC patients: high = 56, low = 904	–	–	–	Decreased MANCR expression is related to a higher OS rate.	–	–	[172]
Two groups of high and low SNHG15 expression, each with 29 subjects	–	–	–	Higher SNHG15 expression is associated with a shorter OS rate.	–	–	[74]
Z38 expression in BC patients: high = 57, low = 53	0.758	78 %	70 %	A lower Z38 expression in BC patients manifests a higher OS rate.	TNM stage, tumor grade, lymph node metastasis, and Z38 expression correlate with the OS rate.	Z38 expression and TNM stage are potential prognostic factors for OS rate.	[127]
High (= 35) and low (= 33) BCRT1 expressing BC patients	–	–	–	Higher BCRT1 expression is associated with shorter OS and DFS rates.	BCRT1 expression and lymph node metastasis correlate with the OS rate.	BCRT1 expression and lymph node metastasis are promising prognostic factors for OS rate.	[95]
LCPAT1 expression in BC cases: high = 20, low = 31	–	–	–	LCPAT1 expression and the OS rate of BC patients are negatively correlated with one another.	–	–	[40]
ST8SIA6-AS1 expression in BC patients: high = 56, low = 82	–	–	–	Higher ST8SIA6-AS1 expression is associated with lower OS, RFS, and MFS rates.	–	–	[110]
SPRY4-IT1 expression in 101 BC patients	–	–	–	A lower SPRY4-IT1 expression is an indicator of higher OS and DFS rates.	–	–	[154]
High (= 100) and low (= 34) FAM83H-AS1 expressing BC patients	–	–	–	FAM83H-AS1 expression and OS rate are inversely related to one another.	–	–	[207]
LINC00173 expression in BC cases: high = 48, low = 36	–	–	–	A higher LINC00173 expression matches lower OS and RFS rates.	–	–	[27]
TTN-AS1 expression in BC patients: high = 24, low = 16	–	–	–	TTN-AS1 expression is negatively correlated with OS rate.	–	–	[29]
TCGA dataset of 100 TNBC patients	0.926	–	–	High levels of BDNF-AS expression conforms with lower OS and DFS rates.	–	BDNF-AS expression and clinical stage are potential prognostic factors.	[96]
TCGA and KMplot database of ER^+ BC cases expressing TROJAN	–	–	–	TROJAN higher expression matches with lower DFS and RFS rates.	–	TROJAN expression and lymph node metastasis are promising prognostic factors.	[69]
Two groups of 30 BC patients with low and high levels of GAS6-AS1	–	–	–	increased GAS6-AS1 expression is associated with a lower OS rate.	GAS6-AS1 expression is associated with a shorter OS rate.	–	[218]
DLG1-AS1 expression in TNBC patients: high = 37, low = 29	–	–	–	DLG1-AS1 expression is negatively correlated with OS rate in TNBC cases.	–	–	[110]
Two groups of 15 BC patients, each expressing high or low amounts of OIP5-AS1	–	–	–	A lower OIP5-AS1 expression conforms with a higher OS rate.	–	–	[186]
KM-Plotter data of LINC00899 expression in BC cases	–	–	–	Higher LINC00899 expression is positively associated with RFS rate in BC cases and HER2 positive, Luminal A and B subgroups.	–	–	[152]
LINC01133 expression in BC cases: high = 29, low = 45	–	–	–	LINC01133 expression is significantly related to a higher OS rate.	downregulation of LINC01133, together with lymph node metastasis, Tumor size, and TNM stage can be regarded as an independent prognostic indicator for BC patients.	downregulation of LINC01133, together with lymph node metastasis and TNM stage can be regarded as an independent prognostic indicator for BC patients.	[155]
High (= 50) and low (= 53) expression levels of GAS5 in BC patients	–	–	–	Higher GAS5 expression is evidently associated with a higher OS rate.	–	–	[87]
Different levels of GAS5 expression in 68 BC patients	–	–	–	GAS5 expression and OS rate of BC patients are markedly associated with one another.	–	–	[42]
GAS5 expression in BC patients: high = 69, low = 87	–	–	–	Higher GAS5 expression matches a higher OS rate.	–	–	[218]

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Table 5 (*continued*)

lncRNA and Clinical Cases	Area under curve	Sensitivity	Specificity	Kaplan-Meier Analysis	Univariate Cox Regression	Multivariate Cox Regression	Reference
High and low expression levels of LINC00968 in BC cases	–	–	–	Lower LINC00968 expression is associated with a lower OS rate.	–	–	[192]
Lower and higher expression levels of PTENP1 in BC cases	–	–	–	PTENP1 and OS rate in BC cases are positively correlated with each other.	–	–	[33]
TCONS_l2_00002973 expression in two groups of TNBC cases, each with 48 subjects	–	–	–	Higher TCONS_l2_00002973 expression level is markedly related with a higher OS rate.	–	–	[178]
MIR503HG expression in TNBC cases, low and high groups each with 47 cases	–	–	–	Lower MIR503HG expression is axiomatically related to a lower OS rate.	MIR503HG expression, clinical stage, N, M, and T classifications are related to OS rate.	M classification and MIR503HG expression are potential prognostic factors for TNBC patients' OS rate.	[31]
Two groups of EGOT expression in BC cases, each with 92 subjects	–	–	–	Higher EGOT expression evidently increases the OS and RFS rates.	–	–	[211]
High and low expression groups of NORAD, each containing 52 cases	–	–	–	NORAD higher level significantly indicates a higher OS rate.	–	–	[161]
KM plotter data of Lower and higher expression level of FGF13-AS1 divided to two BC groups	–	–	–	FGF13-AS1 is positively correlated with RFS rate.	–	–	[116]
TFAP2A-AS1 expression in BC cases: high = 10, low = 20	–	–	–	Higher TFAP2A-AS1 expression is related to a higher OS rate.	–	–	[152]
LncKLHDC7B expression in TNBC cases: high = 100, low = 61	–	–	–	LncKLHDC7B expression is significantly associated with a higher DFS rate.	–	–	[11]
TCGA dataset of 814 BC cases expressing high and low levels of PDCD4-AS1	–	–	–	PDCD4-AS1 and OS rate of BC cases are positively correlated with one another.	–	–	[59]
TCGA-BRCA dataset: 594 BC cases expressing TUG1	–	–	–	TUG1 expression and OS rate are significantly associated with each other.	–	–	[166]
PlncRNA-1 expression in 78 BC and 45 healthy cases	0.8994	–	–	–	–	–	[83]
SNORD3A expression in BC patients: high = 23, low = 49	–	–	–	Higher SNORD3A expression is associated with an increased level of OS rate.	–	–	[112]
144 cases expressing different amounts of MEG3 with three polymorphisms: rs7158663, rs941576, and rs10132552	–	–	–	Patients with rs7158663 AG + AA, rs941576 AG + GG, and rs10132552 CC + CT genotypes indicate higher rates of DFS.	MEG3 rs10132552 TT indicates a lower DFS rate in BC patients.	MEG3 rs10132552, High ki67 level, HER2 overexpression, younger age, and negative hormonal receptor correlate with a good response to chemotherapy.	[10]
PAX8-AS1-N expression in 76 BC subjects	–	–	–	PAX8-AS1-N expression is positively associated with OS rate.	–	–	[204]
HIT expression in plasma samples of BC cases	0.827	57.7 %	86.4 %	–	–	–	[55]
High (= 11) and low (= 9) expression levels of HOTAIR in BC patients	0.9178	–	–	Higher levels of HOTAIR correlate with lower OS and DFS rates.	–	–	[165]
Blood samples of 80 BC and 80 healthy controls expressing MALAT1	0.89	83.7 %	81.25 %	–	–	–	[227]
TINCR expression in 105 non-TNBC, 72 TNBC, and 86 healthy individuals	0.797	–	–	Higher TINCR expression is associated with lower OS and RFS rates in TNBC group, but not the non-TNBC group.	–	TNM stage and tumor grade are possible prognostic factors for both TNBC and non-TNBC patients. Lymph node stage and TINCR expression are prognostic factors for TNBC patients only.	[86]
TCGA database of 198 BC patients expressing TCL6	–	–	–	TCL6 up-regulated expression conforms with a down-regulated OS rate.	HER2 status, age, tumor grade, TNM stage, and TCL6 expression correlate with OS rate.	TCL6 expression is an independent prognostic factor for OS.	[183]

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