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Original Article

Subtype-specific lncRNA signatures: MALAT1 and XIST in breast cancer

Pelin Soytürk, Miray Acer

Department of Molecular Biology and Genetics, Demiroğlu Science University, Faculty of Arts & Science, İstanbul, Türkiye

ABSTRACT

Objectives: The objective of this study was to assess and compare metastasis-associated lung adenocarcinoma transcript 1 (MALAT1), and X-inactive specific transcript (XIST) expression among breast cancer molecular subtypes *in vitro*, and to evaluate their potential association with subtype-specific biological characteristics.

Materials and methods: The expression levels of MALAT1 and XIST were analyzed in breast cancer cell lines representing three molecular subtypes-luminal A (MCF-7), HER2-positive (MDA-MB-453), and triple-negative (MDA-MB-231)-as well as in non-tumorigenic MCF-10A cells, using quantitative real-time PCR. All experiments were performed in triplicate, and statistical analysis was conducted using one-way ANOVA followed by Dunnett tests.

Results: Both MALAT1 and XIST were significantly upregulated in all breast cancer cell lines compared with MCF-10A control ($p<0.001$). MALAT1 expression was highest in triple-negative MDA-MB-231 cells (6.23-fold), followed by luminal A MCF-7 (3.40-fold) and HER2-positive MDA-MB-453 (3.29-fold). Conversely, XIST showed the highest expression in luminal A MCF-7 cells (6.76-fold), with lower levels in HER2-positive (4.37-fold) and triple-negative breast cancer (TNBC) cells (4.06-fold). Notably, MALAT1 expression was significantly higher in TNBC than in other subtypes ($p<0.001$), whereas XIST expression in luminal A cells was significantly higher than in HER2+ and TNBC subtypes ($p<0.001$).

Conclusion: The findings demonstrate clear subtype-specific expression patterns of MALAT1 and XIST in breast cancer. Markedly elevated MALAT1 in TNBC corresponds to its aggressive phenotype and metastatic potential, while predominant XIST expression in luminal A cells may reflect estrogen receptor-related regulatory mechanisms. These differential expression profiles suggest potential roles for these long non-coding ribonucleic acids as subtype-specific biomarkers and therapeutic targets.

Keywords: Breast cancer, HER2-positive, long non-coding RNA, luminal A, MALAT1, molecular subtypes, triple-negative breast cancer, XIST.

Breast cancer, among female malignancies worldwide, continues to rank first in incidence and represents a major contributor to cancer mortality. GLOBOCAN 2022 data indicate roughly 2.3 million incident cases and approximately 670,000 fatalities attributable to this disease, comprising 11.6% of total cancer diagnoses globally.[1] This malignancy displays substantial biological diversity, presenting as molecularly distinct subtypes that differ in clinical manifestations, treatment sensitivity, and clinical outcomes.[2]

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Correspondence: Pelin Soytürk.
E-mail: psoyturk123@gmail.com

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The molecular stratification of breast cancer has transformed therapeutic decision-making via tumor categorization according to estrogen receptor (ER), progesterone receptor (PR), and human epidermal growth factor receptor 2 (HER2) status. Four primary categories emerge from this classification system: luminal A (ER+/PR+/HER2–, low Ki-67–), luminal B (ER+/PR+/HER2+ or HER2+, elevated Ki-67), HER2-enriched (ER–/PR–/HER2+), and triple-negative breast cancer (TNBC; ER–/PR–/HER2–).[3] Triple-negative breast cancer constitutes roughly 15–20% of breast malignancies and demonstrates particularly unfavorable characteristics, including aggressive behavior, frequent relapse, scarce targeted treatment options, and diminished survival owing to hormone receptor absence.[4]

Long non-coding ribonucleic acids (lncRNAs) constitute a heterogeneous transcript family characterized by lengths surpassing 200

nucleotides and absence of protein-coding potential, yet they substantially influence gene regulation via chromatin remodeling, transcriptional control, and post-transcriptional modifications.[5] Accumulating research has positioned lncRNAs as key molecular players governing breast cancer development, disease progression, metastatic spread, and drug resistance, rendering them valuable candidates for diagnostic and therapeutic applications.[6]

Metastasis-associated lung adenocarcinoma transcript 1 (MALAT1) is recognized as one of the most thoroughly characterized oncogenic lncRNAs across human malignancies. This evolutionarily conserved nuclear transcript influences gene expression through modulation of splicing patterns and transcriptional programs of downstream targets.[7] Within breast cancer, MALAT1 plays a role in increased proliferative capacity, migratory and invasive phenotypes, and epithelial-mesenchymal transition (EMT) via several signaling cascades such as phosphatidylinositol 3-kinase/protein kinase B (PI3K/AKT), Wnt/β-catenin, and diverse competing endogenous RNA regulatory circuits.[8] Elevated MALAT1 levels show association with nodal involvement, advanced disease stage, and unfavorable prognosis, with particularly strong correlation in TNBC.[9]

In breast cancer development and progression, besides MALAT1, other long non-coding RNAs have also been identified as influential, notably the X-inactive specific transcript (XIST), which plays a regulatory role that varies depending on the biological context. XIST, spanning approximately 17 kb, serves as the master regulator of X-chromosome inactivation (XCI) during female mammalian embryonic development. Apart from its established dosage compensation function, XIST has gained recognition as an important modulator in cancer biology, exhibiting context-specific activities. XIST demonstrates dual and occasionally opposing roles in breast cancer, functioning either as a tumor promoter or suppressor dependent on the cellular environment and molecular subtype.[10] Multiple investigations have revealed that reduced XIST expression activates the AKT signaling axis and augments cellular viability in breast cancer, pointing toward tumor-suppressive properties.[11] In contrast, alternative evidence shows XIST involvement in encouraging cancer stem cell characteristics via proinflammatory interleukin-6/signal transducer and activator of transcription 3 (IL-6/STAT3) pathway engagement.[12] Notably, aberrant XCI coupled with dysregulated XIST expression correlates with elevated TNBC incidence and shortened patient survival.[13]

The functional crosstalk between MALAT1 and XIST within breast cancer has attracted considerable research interest. Recent work established that both lncRNAs converge on the programmed death-ligand 1 (PD-L1) signaling network, wherein XIST predominantly suppresses MALAT1-driven oncogenic effects.[14] These observations imply that the relative abundance of these two regulatory RNAs may substantially impact tumor progression and immune evasion processes.

Although individual lncRNAs have been extensively investigated in breast cancer, systematic comparative assessments of MALAT1 and XIST expression profiles across molecular subtypes are notably lacking. Characterizing subtype-specific lncRNA signatures is important for deciphering the molecular basis of divergent subtype behaviors and discovering precision therapy opportunities.

This study examined MALAT1 and XIST levels in breast cancer cell models spanning three molecular categories-luminal A (MCF-7), HER2-positive (MDA-MB-453), and triple-negative (MDA-MB-231)-with MCF-10A non-malignant mammary epithelial cells as a reference. The aim was to define subtype-associated expression patterns for these lncRNAs and explore their relevance to breast cancer biology and clinical practice.

MATERIALS AND METHODS

Cell lines and culture conditions

All studies were conducted at the Demiroğlu Science University Research Laboratory. To examine lncRNA expression across breast cancer molecular subtypes, we selected representative cell lines for each classification: MCF-7 (HTB-22™), MDA-MB-453 (HTB-131™), and MDA-MB-231 (CRM-HTB-26™). All cell lines were purchased from the American

Type Culture Collection (ATCC, Manassas, VA, USA). The MCF-10A, a non-malignant mammary epithelial cell line, served as the reference control, and was cultured with Mammary Epithelial Growth Medium Bullet Kit (Lonza Group AG, Basel, Switzerland, Cat no: CC-3150). Cancer cell line cultures were maintained in RPMI-1640 (Gibco, Thermo Fisher Scientific, Grand Island, NY, USA, Cat no: A1049101) growth medium containing 10% heat-inactivated fetal bovine serum (Gibco, Thermo Fisher Scientific, Grand Island, NY, USA, Cat no: A5670801) and antibiotics (1% penicillin-streptomycin (100 U/mL penicillin and 100 µg/mL streptomycin; Gibco, Thermo Fisher Scientific, Grand Island, NY, USA, Cat no: 15140122). Incubation was at 37 °C with 5% CO_2 in a humidified environment. Cultures reaching 80-90% confluence were processed for downstream RNA isolation.

RNA isolation and complementary DNA synthesis

RNA extraction was performed using the miRNeasy Advanced Kit (Qiagen, Hilden, Germany, Cat no: 217684) according to manufacturer's guidelines. Spectrophotometric quality assessment was conducted on a NanoDrop spectrophotometer (Thermo Fisher Scientific, USA). Samples with A260/A280 ratios of 1.8-2.1 were deemed suitable for subsequent procedures. The A260/A230 ratio served as an additional quality metric for detecting organic contaminants; only samples with values of 1.8 or greater proceeded to analysis. Complementary DNA was generated using Qiagen's reverse transcription system (Qiagen, Hilden, Germany, Cat no: 330401) optimized for long transcript detection, following the manufacturer's protocol, with RNA input at a concentration of 500 ng/µL for cDNA synthesis.

Quantitative real-time PCR

Gene expression quantification used SYBR Green detection chemistry on a Rotor-Gene Q instrument (Qiagen, Hilden, Germany, RT^2 SYBR Green ROX FAST Mastermix Cat no: 330620). We used RT^2 lncRNA qPCR Assays (Cat: 330701) to determine the level of expression MALAT1 (GeneGlobe ID: LPH18065A) and XIST (GeneGlobe ID: LPH08103A). Thermal cycling parameters were: initial heat activation at 95 °C (5 min), followed by 40 amplification cycles consisting of denaturation (95 °C, 10 sec), primer annealing (60 °C, 20 sec), and elongation (72 °C, 20 sec).

Post-amplification dissociation analysis (65-95 °C gradient) validated product specificity, confirming single-amplicon generation without primer-dimer artifacts. Technical replicates (n=3) were run for each sample alongside negative controls to detect contamination. GAPDH (GeneGlobe ID: PPH00150F, Cat: 330001) was used for normalization. Expression fold-changes were determined using the comparative Ct approach ($2^{-\Delta\Delta Ct}$ method), with all values normalized against MCF-10A controls.

Statistical analysis

Experiments were conducted in triplicate, and results are expressed as mean ± standard deviation (SD). Group comparisons used one-way ANOVA with Dunnett's correction for multiple comparisons against the MCF-10A reference. Normality was verified using the Shapiro-Wilk test before parametric testing ($n<50$). Additionally, Tukey's honestly significant difference (HSD) post hoc test was applied for inter-group (inter-subtype) multiple comparisons among all experimental groups. GraphPad Prism was the statistical platform, with significance defined as $p<0.05$.

RESULTS

XIST expression across breast cancer subtypes

Similarly, XIST expression was significantly upregulated in all breast cancer cell lines compared with MCF-10A control, as determined by one-way ANOVA ($F=983.52$, $p<0.001$), as shown in Figure 1a. However, the expression pattern differed markedly from that of MALAT1. The highest XIST expression was observed in MCF-7 (luminal A) cells (7.77±0.15, 6.76-fold increase, $p<0.001$), followed by MDA-MB-453 (HER2+; 5.03±0.15, 4.37-fold increase, $p<0.001$) and MDA-MB-231 (TNBC; 4.67±0.15, 4.06-fold increase, $p<0.001$).

Inter-subtype analysis showed that XIST expression in MCF-7 cells was significantly higher than in both MDA-MB-453 ($p<0.001$)

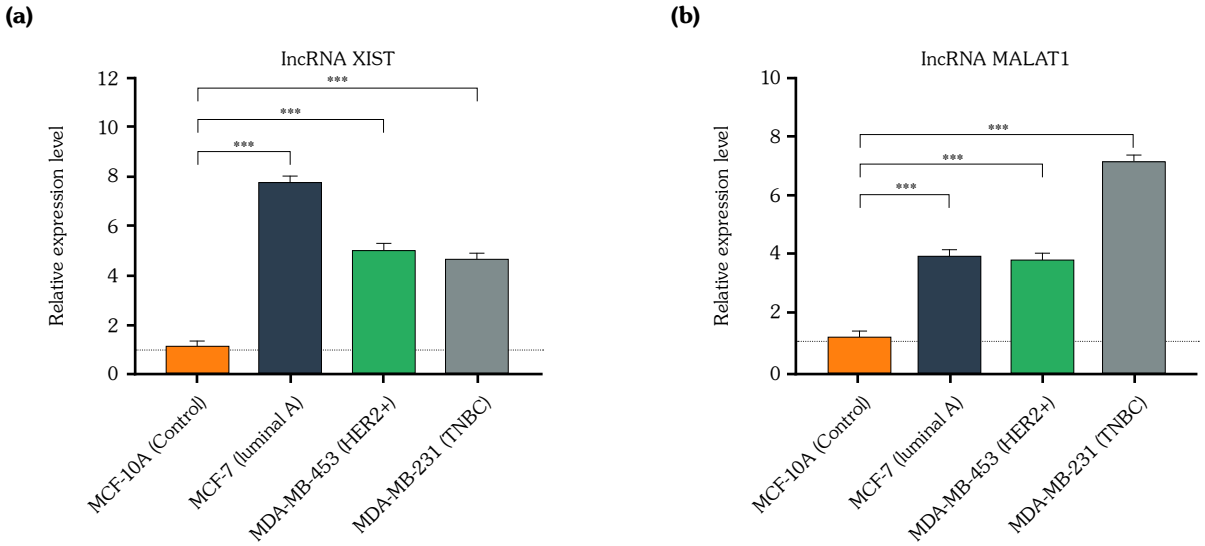

Figure 1. Expression levels of MALAT1 and XIST in breast cancer cell lines representing different molecular subtypes. **(a)** XIST expression and **(b)** MALAT1 expression were measured by qPCR in MCF-10A, MCF-7, MDA-MB-453, and MDA-MB-231 cells. Data are presented as mean ± SD of three independent experiments. Expression levels were normalized to the MCF-10A control. Statistical significance was determined by one-way ANOVA followed by Dunnett tests.

lncRNA: Long non-coding ribonucleic acid; MALAT1: Metastasis-associated lung adenocarcinoma transcript 1; XIST: X-inactive specific transcript; SD: Standard deviation; *** $p<0.001$ *vs.* MCF-10A control. The dashed line indicates the baseline expression level (1.0) in control cells.

and MDA-MB-231 ($p<0.001$) cells. Additionally, a modest but statistically significant difference was observed between MDA-MB-453 and MDA-MB-231 cells ($p=0.042$), with higher expression in the HER2-positive subtype.

MALAT1 expression across breast cancer subtypes

Quantitative real-time PCR analysis demonstrated notable upregulation of MALAT1 in all breast cancer cell lines compared with the

Table 1. Summary of statistical analyses for MALAT1 and XIST expression across breast cancer cell lines

	MALAT1 (*p*-value)	XIST (*p*-value)
Comparison		
One-way ANOVA	<0.001*	<0.001*
MCF-7 *vs.* control	<0.001*	<0.001*
MDA-MB-453 *vs.* control	<0.001*	<0.001*
MDA-MB-231 *vs.* control	<0.001*	<0.001*
Inter-subtype comparisons		
MCF-7 *vs.* MDA-MB-453	0.348 (ns)	<0.001*
MCF-7 *vs.* MDA-MB-231	<0.001*	<0.001*
MDA-MB-453 *vs.* MDA-MB-231	<0.001*	0.07 (NS)

MALAT1: Metastasis-associated lung adenocarcinoma transcript 1; XIST: X-inactive specific transcript; * $p<0.001$.

non-tumorigenic MCF-10A control, as shown in Figure 1b. One-way ANOVA demonstrated highly significant differences among groups ($F=810.13$, $p<0.001$). Post hoc analysis showed that MALAT1 expression was significantly elevated in MCF-7 (luminal A; 3.91±0.15, 3.40-fold increase, $p<0.001$), MDA-MB-453 (HER2+; 3.78±0.15, 3.29-fold increase, $p<0.001$), and MDA-MB-231 (TNBC; 7.17±0.15, 6.23-fold increase, $p<0.001$) cells relative to the MCF-10A control (1.15±0.15).

Notably, MALAT1 expression in the triple-negative MDA-MB-231 cell line was markedly higher than in other breast cancer subtypes. Inter-subtype comparison revealed no significant difference between MCF-7 and MDA-MB-453 cells ($p=0.348$), whereas MDA-MB-231 exhibited significantly elevated MALAT1 expression compared with both MCF-7 ($p<0.001$) and MDA-MB-453 ($p<0.001$). These findings indicate that MALAT1 overexpression is especially prominent in TNBC.

Distinct expression patterns of MALAT1 and XIST

Comparative evaluation demonstrated inverse expression patterns of MALAT1 and XIST across breast cancer subtypes. While MALAT1 expression increased progressively from hormone receptor-positive to hormone receptor-negative subtypes (luminal A ≈ HER2+ << TNBC), XIST showed the opposite trend, with the highest expression in luminal A (luminal A >> HER2+ > TNBC). This contrasting relationship suggests distinct regulatory mechanisms and potentially different functional roles of these lncRNAs in the pathogenesis of breast cancer subtypes. The statistical analysis is summarized in Table 1.

DISCUSSION

The present study demonstrates clear subtype-specific expression patterns of the lncRNAs MALAT1 and XIST across breast cancer molecular subtypes, providing important insights into their potential functions in subtype-specific pathogenesis. Although both lncRNAs are upregulated in breast cancer relative to normal mammary epithelial cells, their expression profiles diverge markedly across molecular subtypes, suggesting differential involvement in subtype-specific biological characteristics. Substantially elevated MALAT1 transcript levels in triple-negative MDA-MB-231 cells (6.23-fold) compared with hormone receptor-expressing subtypes merit particular attention. Triple-negative breast cancer exhibits clinically aggressive features, including heightened metastatic propensity, early disease recurrence, and restricted treatment modalities attributable to the lack of targetable hormone receptors and HER2 amplification.[4] Our data corroborate earlier reports linking MALAT1 to TNBC progression via different pathways, including EMT modulation through the MALAT1/miR-106a-5p/REEP5 regulatory circuit, PI3K-AKT signaling enhancement, and facilitation of metastatic dissemination.[8,9] Prior work identified MALAT1 as a standalone prognostic indicator in hormone receptor-negative, node-negative breast carcinoma, wherein increased transcript abundance correlated with diminished disease-specific survival. The selective MALAT1 enrichment in TNBC may underlie this subtype's aggressive biology through activation of prometastatic transcriptional networks and enhanced cellular adaptability.

The comparable MALAT1 abundance detected in MCF-7 versus MDA-MB-453 cell populations implies that hormone receptor presence by itself does not primarily govern MALAT1 transcription. Rather, the pronounced upregulation specific to TNBC points toward an association between MALAT1 elevation and basal-like or mesenchymal cellular characteristics typical of triple-negative malignancies. This observation is consistent with evidence implicating MALAT1 in sustaining stem-like cancer cell features and conferring therapeutic resistance-attributes especially pertinent to TNBC biology.[15]

In contrast to MALAT1, XIST shows peak expression in luminal A MCF-7 cells (6.76-fold), declining progressively across HER2-enriched and TNBC subtypes. This distribution raises compelling questions about XIST regulatory functioning within ER-expressing breast malignancies. The preferential XIST accumulation in ER-positive cells likely reflects the elaborate interrelationship between estrogen pathway activity and X-chromosome silencing.

Research has shown that ER-positive breast cancer cells maintain XCI via ER-dependent processes, whereas TNBC cells display disrupted XCI accompanied by attenuated XIST levels.[16] Such differential control may carry substantial biological consequences, given that compromised XCI correlates with increased TNBC prevalence and worse clinical outcomes.[13]

The biological ramifications of elevated XIST in luminal A breast malignancy appear multifaceted and situation-dependent. Certain investigations propose XIST as a tumor suppressor through AKT phosphorylation blockade mediated by HDAC3-dependent PHLPP1 transcriptional silencing.[11] Within this setting, XIST reduction in aggressive subtypes such as TNBC would facilitate oncogenic progression via unopposed AKT stimulation. Alternatively, documented XIST participation in breast cancer stemness regulation through inflammatory IL-6/STAT3 cascade activation.[12] The expression divergence we identified implies that XIST may execute subtype-dependent functions dictated by the specific molecular milieu.

The inverse expression patterns of MALAT1 and XIST across subtypes have potential clinical implications.[14] These lncRNAs engage within the PD-L1 regulatory signaling pathway, with XIST exerting a dominant inhibitory effect on MALAT1's oncogenic function. In luminal A cells with high XIST and relatively lower MALAT1 expression, this regulatory relationship may contribute to the less aggressive phenotype of hormone receptor-positive tumors. Conversely, the high MALAT1/low XIST ratio in TNBC may promote tumor progression by eliminating XIST-mediated suppression of MALAT1's oncogenic activity.

From a translational standpoint, these observations carry consequences for lncRNA-centered diagnostic and therapeutic development. The pronounced MALAT1 overexpression in TNBC identifies it as a leading candidate for antisense oligonucleotide intervention or alternative RNA-directed strategies against this challenging malignancy subtype. Published reports indicate that MALAT1 silencing attenuates TNBC cellular proliferation, motility, and invasive capacity, reinforcing its therapeutic promise.[7] Regarding luminal A tumors, elucidating XIST's contribution to hormone receptor-positive phenotype maintenance may clarify endocrine resistance pathways and inform rational combination treatment design.

Several limitations of the present study should be acknowledged. Reliance on cultured cell models, while permitting standardized experimental conditions, does not fully capture the complexity of *in vivo* tumor physiology and stromal interactions. Examining only one representative cell line per molecular category also overlooks substantial intra-subtype variation inherent to breast carcinoma. Additionally, this study focused on transcript quantification without functional mechanistic validation. Future research should corroborate these expression signatures in expanded patient tissue cohorts, delineate the molecular determinants underlying subtype-restricted lncRNA profiles, and assess the clinical relevance of MALAT1 and XIST as molecular subtype-tailored diagnostic or therapeutic tools.

In conclusion, this study demonstrates clear subtype-specific expression patterns of MALAT1 and XIST in breast cancer. The marked elevation of MALAT1 in TNBC is consistent with this subtype's aggressive phenotype and suggests a role in facilitating metastasis and treatment resistance. Predominant expression of XIST in luminal A cells may reflect ER-mediated regulatory mechanisms and potentially tumor-suppressive functions. These differential expression profiles provide a basis for further investigation of lncRNA-based diagnostic biomarkers and treatment targets tailored to specific breast cancer molecular subtypes.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: P.S., M.A.: Idea/concept, design, control/supervision, data collection and/or processing, analysis and/or interpretation, literature review, writing the article, references and fundings, materials. P.S.: Critical review.

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