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Bioinformatics Analysis

RESEARCH ARTICLE

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LINC00511 in Breast Cancer: A Bioinformatics Exploration of Its Prognostic Value, Functional Mechanisms, and Role in Tumor Immunity

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Abstract

Objective: This study preliminarily examines the clinical value and molecular mechanisms of *LINC00511* in breast cancer (*BRCA)* through bioinformatics analysis. **Methods:** Expression data of *LINC00511* and related information for *BRCA* samples were collected from the TCGA website. The levels of *LINC00511* in cancerous and normal tissues were compared using the Wilcoxon rank-sum test. The relationship between *LINC00511* levels and clinicopathological features was examined using the chi-square test. Additionally, Kaplan-Meier and Cox regression analyses were employed to evaluate the prognostic significance of *LINC00511*. Finally, to determine the biological functions and molecular mechanisms of *LINC00511*, functional enrichment and immune infiltration analyses were performed using the Xiantao Academic website. **Results:** *LINC00511* was found to be upregulated in *BRCA*. *LINC00511* expression was closely related to T stage, pathological stage, race, estrogen receptor (ER), and progesterone receptor (PR). For BRCA patients, independent prognostic factors included older age (≥65 years), advanced pathological stages (III and IV), and elevated *LINC00511* levels. Enrichment analysis demonstrated the close association of *LINC00511* with the IL-17 signaling pathway, estrogen signaling pathway, chemical carcinogenesis, and drug metabolism. Furthermore, *LINC00511* expression was significantly related to the level of immune cell infiltration. **Conclusion:** *LINC00511* is significantly overexpressed in *BRCA*, and elevated levels of this factor are associated with a reduction in overall survival in *BRCA* patients. *LINC00511* not only possesses a high degree of clinical value in the diagnosis and prognosis of *BRCA* but also has the potential to serve as a target for immunotherapy.

Keywords: *LINC00511- BRCA*- diagnosis- prognosis- immunotherapy

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Introduction

BRCA is the primary factor in cancer-related fatalities among women, with both its incidence and mortality rates rising each year [1]. A 2020 data study revealed that *BRCA* incidence among women accounted for 11.7% of all cancer incidences, surpassing lung cancer as the most prevalent cancer for the first time [2]. The International Agency for Research on Cancer (IARC) forecasts that *BRCA* incidence will continue to grow, anticipating over 3 million new cases globally by 2040 [2]. This indicates that *BRCA* poses a significant threat to human health, particularly affecting women. Despite some therapeutic success in early-stage *BRCA* with conventional treatments such as surgery, radiotherapy, and pharmacological approaches (including chemotherapy, targeted therapy, endocrine therapy, and immunotherapy), tumor recurrence and metastasis remain challenging issues in clinical treatment [3]. For advanced *BRCA* patients, targeted therapy based on the expression of key genes has become an important treatment method in addition to systemic chemotherapy. However, drug resistance often leads to unsatisfactory therapeutic outcomes for advanced *BRCA*. Therefore, it is imperative to discover reliable prognostic biomarkers to enable the implementation of personalized treatment strategies.

lncRNAs are transcripts containing over 200 bases that do not have protein-coding ability. However, in-depth research has found that some lncRNAs possess a certain protein-coding ability [4-7]. This significant discovery has garnered widespread attention for lncRNAs. Most lncRNAs are located in the cytoplasm or nucleus, where they can influence gene expression and function by regulating processes such as epigenetics, transcription,

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and post-transcription [8], thereby contributing to physiological processes like cell growth and programmed cell death [9, 10]. *LINC00511* belongs to the lncRNA category and was discovered by Cabanski et al. in 2015 as an RNA gene primarily associated with lung cancer [11]. *LINC00511* is located in the q24.3 region of chromosome 17, with a full length of 1716 nucleotides [12]. Previous research has demonstrated that LINC00511 is abnormally expressed in several cancers, including colorectal, non-small cell lung, cervical, gastric, and hepatocellular cancers [13-17]. These studies have shown that aberrant expression of *LINC00511* affects the biological functions of these cancer cells, indicating that *LINC00511* is a crucial molecule influencing the progression and survival outcomes of malignant tumors. Although previous studies have found that *LINC00511* is overexpressed in *BRCA* [18], the sample size in those studies was very limited. This conclusion needs further substantiation, and the clinical significance and mechanism of action of *LINC00511* in *BRCA* remain to be fully elucidated, necessitating further comprehensive research.

This research examines the expression levels of *LINC00511* in *BRCA* patients using bioinformatics and investigates its correlation with clinical pathology and prognosis. Furthermore, a nomogram model is developed to estimate the prognosis of *BRCA*. Finally, we preliminarily explore the mechanism of *LINC00511* in *BRCA*. Our research indicates that *LINC00511* holds significant value in both the diagnosis of breast cancer and the prediction of prognosis for *BRCA* patients.

Materials and Methods

Collection of data

In April 2024, clinical information and *LINC00511* expression data (TPM format) of *BRCA* samples were collected from the TCGA (https://cancergenome.nih.gov/). All patients had not received anti-tumor treatment prior to registration. We extracted 1,231 samples from the TCGA breast cancer project. After eliminating 23 duplicate cases, we finally retained 1,208 independent samples, including 1,095 tumor samples and 113 normal control samples. Subsequently, we excluded patients with missing clinical information from the tumor cohort to ensure the reliability of the data and the rigor of the analysis. The download and processing procedures of the TCGA data are shown in Figure 1.

Nomogram Construction and Validation

Univariate COX analysis was performed to screen for potential prognostic factors. Variables with a P value < 0.05 in univariate analysis were further incorporated into multivariate COX regression analysis to identify the prognostic factors of *BRCA* patients. In addition, we established a nomogram model based on these prognostic factors using the "*rms*" package to predict the prognosis of *BRCA* patients. ROC curves were plotted using the "*timeROC*" package, and the accuracy of the model was evaluated by the area under the curve (AUC), and the model performance was assessed through calibration.

Analysis of function, pathways and immune infiltration

Using the GEPIA database [19], we evaluated the relationship between LINC00511 levels and various immune checkpoints. Differentially expressed genes (DEGs) were analyzed using Xiantao tools (https://www.xiantao.love/) [20], with genes having a p-value < 0.05 and |log2-fold-change (FC)| > 1 included as DEGs. A functional enrichment analysis was conducted, encompassing both Gene Ontology (GO) and Kyoto Encyclopedia of Genes and Genomes (KEGG) analyses. Furthermore, this study also explored the correlation between the level of LINC00511 and the degree of immune infiltration through the "*ssgsea*" algorithm.

Statistical Analysis

R software (version 4.3.3) was used for statistical analysis and graphical presentation in this study. To compare *LINC00511* expression levels between tumor and non-tumor samples, the Wilcoxon rank-sum test and Wilcoxon signed-rank test were utilized. The diagnostic performance of *LINC00511* for *BRCA* was evaluated by plotting receiver operating characteristic (ROC) curves using the pROC package. The chi-square test was employed to evaluate the correlation between *LINC00511* expression and clinical pathological characteristics, while the Kaplan-Meier method was used to analyze survival prognosis. To identify independent factors influencing the survival prognosis of *BRCA* patients, both univariate and

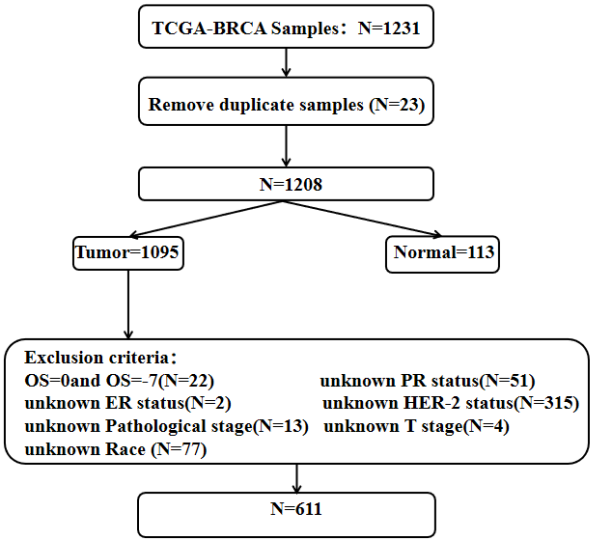

Figure 1. Flowchart of TCGA-BRCA Sample Selection and Exclusion Criteria. A flow diagram illustrating the sample inclusion/exclusion process for the TCGA-BRCA cohort. A total of 1,231 TCGA-BRCA samples were initially collected, and 23 duplicate samples were removed, leaving 1,208 unique samples. These samples were classified into tumor (n = 1,095) and normal (n = 113) groups. Tumor samples were further filtered according to the following exclusion criteria: overall survival (OS) = 0 or OS = −7 (n = 22), unknown ER status (n = 2), unknown pathologic stage (n = 13), unknown race (n = 77), unknown PR status (n = 51), unknown HER2 status (n = 315), and unknown T stage (n = 4). After applying these criteria, 611 tumor samples with complete clinical information were retained for subsequent analyses.

multivariate Cox regression analyses were conducted. Correlations were assessed using Spearman correlation. All hypothesis tests were two-sided, with a p-value of less than 0.05 deemed statistically significant.

Results

LINC00511 is Upregulated in BRCA

We downloaded 113 normal and 1,095 cancer tissue samples from the TCGA website. The Wilcoxon rank-sum test revealed a significant elevation of *LINC00511* expression in cancer tissues compared to normal tissues (Figure 2A). Similarly, the Wilcoxon signed-rank test showed that *LINC00511* expression was significantly higher in 113 cancer tissue samples compared to their corresponding normal tissue counterparts (Figure 2B). Additionally, the ROC curve indicated that the area under the curve (AUC) was 0.844 (95% CI: 0.792–0.896), suggesting that *LINC00511* could effectively distinguish between cancerous and non-cancerous tissues, making it a valuable diagnostic biomarker for *BRCA* (Figure 2C).

Clinical data and pathological characteristics of BRCA patients

We collected clinical pathological characteristics of 1,095 *BRCA* samples from the TCGA database, including race, age, T stage, N stage, pathological stage, ER status, PR status, HER2 status, primary tumor location, and *LINC00511* expression. After excluding samples with missing clinical information, 611 samples were included in the subsequent analysis (Table 1).

Table 1. Clinicopathological Information of 611 Breast Cancer Patients

Characteristics	level	Overall (N=611)
Race (%)	Asian	43 (7.0)
	Black	89 (14.6)
	White	479 (78.4)
Age (%)	<65	443 (72.5)
	≥65	168 (27.5)
T_stage (%)	T1+T2	518 (84.8)
	T3+T4	93 (15.2)
N_stage (%)	N0	304 (49.8)
	N1+2+3	307 (50.2)
Pathologic_stage (%)	I+II	466 (76.3)
	III+IV	145 (23.7)
PR_status (%)	Negative	208 (34.0)
	Positive	403 (66.0)
ER_status (%)	Negative	144 (23.6)
	Positive	467 (76.4)
HER2_status (%)	Negative	482 (78.9)
	Positive	129 (21.1)
Primary_tumor_location (%)	Left	329 (53.8)
	Right	282 (46.2)
LINC00511 (%)	High group	306 (50.1)
	Low group	305 (49.9)
Status (%)	Alive	544 (89.0)
	Dead	67 (11.0)

Relationship Between LINC00511 and Clinical Pathological Characteristics

The median *LINC00511* expression was used as the dividing line, and the study population was divided into two groups: the high expression group and the low expression group. The chi-square test was then applied to analyze the correlation between *LINC00511* expression and clinical pathological characteristics. The findings indicate that *LINC00511* expression is significantly associated with five factors: T stage (P=0.017), pathologic stage (P=0.010), Race (P=0.048), ER status (P<0.001), and PR status (P<0.001) (Table 2).

The Relationship Between LINC00511 and the OS of BRCA Patients

Kaplan-Meier curves were plotted to investigate the correlation between the various factors and the OS of *BRCA* patients (Figure 2). The findings indicated that *BRCA* patients with high levels of *LINC00511* expression had significantly worse OS (P=0.007). Additionally, younger age (<65 years), early T stage T1/

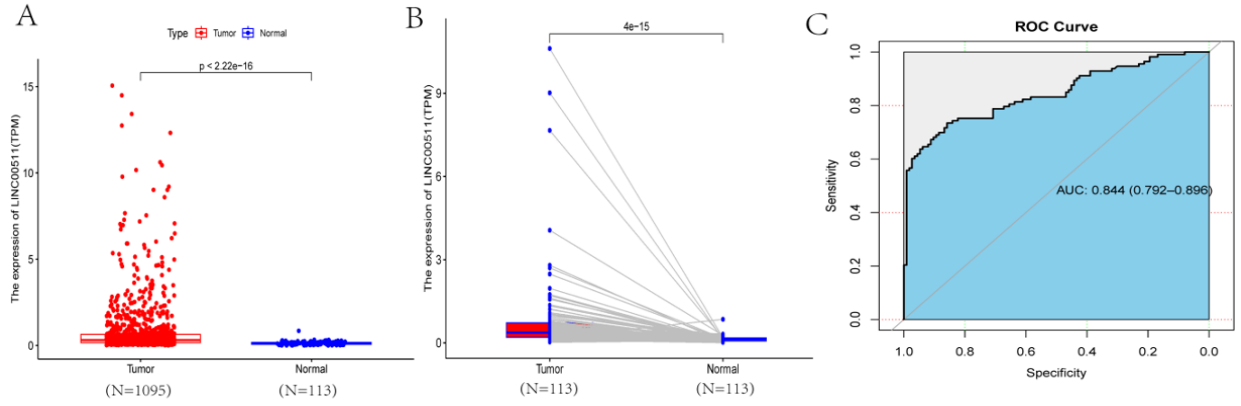

Figure 2. Evaluation of *LINC00511* Expression and Its Diagnostic Efficacy in *BRCA*. (A) Comparison of *LINC00511* level in 1095 cancer tissues and 113 normal tissues. (B) Comparison of LINC00511 level in 113 cancer tissues and paired normal tissues. (C) ROC curve showing *LINC00511*'s ability to distinguish between cancerous and normal tissues in *BRCA*.

Table 2. Correlation between *LINC00511* Expression and Clinicopathological Features

Characteristics	Low group	High group	P value
n	305	306	
Race, n (%)			0.004
Black	34 (5.6%)	55 (9%)	
White	256 (41.9%)	223 (36.5%)	
Asian	15 (2.5%)	28 (4.6%)	
Age, N (%)			0.699
<65	219 (35.8%)	224 (36.7%)	
≥65	86 (14.1%)	82 (13.4%)	
T_stage, N (%)			0.017
T1+T2	248 (40.6%)	270 (44.2%)	
T3+T4	57 (9.3%)	36 (5.9%)	
N_stage, N (%)			0.544
N1+2+3	157 (25.7%)	150 (24.5%)	
N0	148 (24.2%)	156 (25.5%)	
Pathologic_stage, N (%)			0.01
I+II	219 (35.8%)	247 (40.4%)	
III+IV	86 (14.1%)	59 (9.7%)	
ER_status, N (%)			< 0.001
Positive	279 (45.7%)	188 (30.8%)	
Negative	26 (4.3%)	118 (19.3%)	
PR_status, n (%)			< 0.001
Positive	244 (39.9%)	159 (26%)	
Negative	61 (10%)	147 (24.1%)	
*HER2*_status, N (%)			0.782
Positive	63 (10.3%)	66 (10.8%)	
Negative	242 (39.6%)	240 (39.3%)	
Primary_tumor_location, N (%)			0.774
Right	139 (22.7%)	143 (23.4%)	
Left	166 (27.2%)	163 (26.7%)	
Status, N (%)			0.003
Alive	283 (46.3%)	261 (42.7%)	
Dead	22 (3.6%)	45 (7.4%)	

T2, early pathological stage (I and II), and No lymph node metastasis were associated with longer overall survival (Figure 3).

To verify the results obtained from the K-M survival analysis, we conducted univariate and multivariate Cox proportional hazards regression analyses. Consistent with the observations from the K-M curve, the univariate Cox analysis indicated that age, T stage, N stage, pathological stage, and *LINC00511* expression were all significantly associated with OS. Multivariate COX regression analyses of the above four factors were performed and the results showed that age [HR=3.272 (1.987 - 5.386), P<0.001], pathological stage [HR=3.351 (1.584 - 7.087), P=0.002], and *LINC00511* expression [HR=2.063 (1.172 - 3.633), P=0.012] were independent prognostic factors for *BRCA* patients (Table 3).

In conclusion, both Kaplan-Meier curves and Cox regression analysis indicate that *LINC00511* expression in *BRCA* is closely related to patient prognosis.

Development and Validation of Nomogram

A nomogram was constructed to assess the one-year, three-year, and five-year OS probabilities of *BRCA* patients (Figure 4), providing a theoretical reference for clinicians to evaluate the OS of *BRCA* patients.

Moreover, we evaluated the performance of the nomogram model using ROC and calibration curves. The ROC curves demonstrated that the AUCs for one-year, three-year, and five-year OS were 0.854, 0.778, and 0.744, respectively (Figures 5A-C), indicating good discriminatory ability of the nomogram prediction model. The calibration curves showed high consistency between observed and predicted results (Figures 5D-F), suggesting good calibration ability of the nomogram.

Biological Function and Signalling Pathway Analysis

Biological function analysis indicated that gene sets regulated by *LINC00511* are primarily associated with biological processes such as skin development, keratinization, passive transmembrane transporter activity, and gated channel activity (Figure 6A). KEGG pathway analysis results showed that gene sets regulated by *LINC00511* are closely related to multiple conduction pathways, including chemical carcinogenesis, cytokine-cytokine receptor interaction, estrogen signaling pathway, IL-17 signaling pathway, cytochrome P450 metabolism of xenobiotics, and drug metabolism (Figure 6B). These findings suggest that *LINC00511* may impact *BRCA* development by mediating the aforementioned signaling pathways.

Relationship Between LINC00511, Immune Cell Infiltration, and Immune Checkpoints

The present study assess the relationship between the immune infiltration in the tumour microenvironment and the expression of *LINC00511*, utilising the Xiantao academic website. The findings suggest that *LINC00511* levels were negatively correlated with the infiltration levels of certain immune cells, especially eosinophils, Th17 cells, mast cells, and NK CD56 Bright cells. Conversely, there is a positive correlation between levels of *LINC00511* and the degree of infiltration of Th1 cells, macrophages and Tgd cells. (Figure 7A). Additionally, Our study revealed a significant positive correlation between *LINC00511* expression levels and the expression of immune checkpoints PD1 (*PDCD1*), *CTLA4*, and PD-L1 (CD274). (Figures 7B-D). Overall, our study indicates that *LINC00511* may influence the occurrence and development of *BRCA* by interacting with immune cells.

Discussion

BRCA has become the number one "killer" of women's health [21]. Despite significant improvements in 5-year survival rates for *BRCA* patients due to advances in science and technology and continuous improvement in treatment methods, more satisfactory therapeutic effects have yet to be achieved [22]. Therefore, identifying diagnostic and prognostic factors and potential mechanisms for *BRCA*

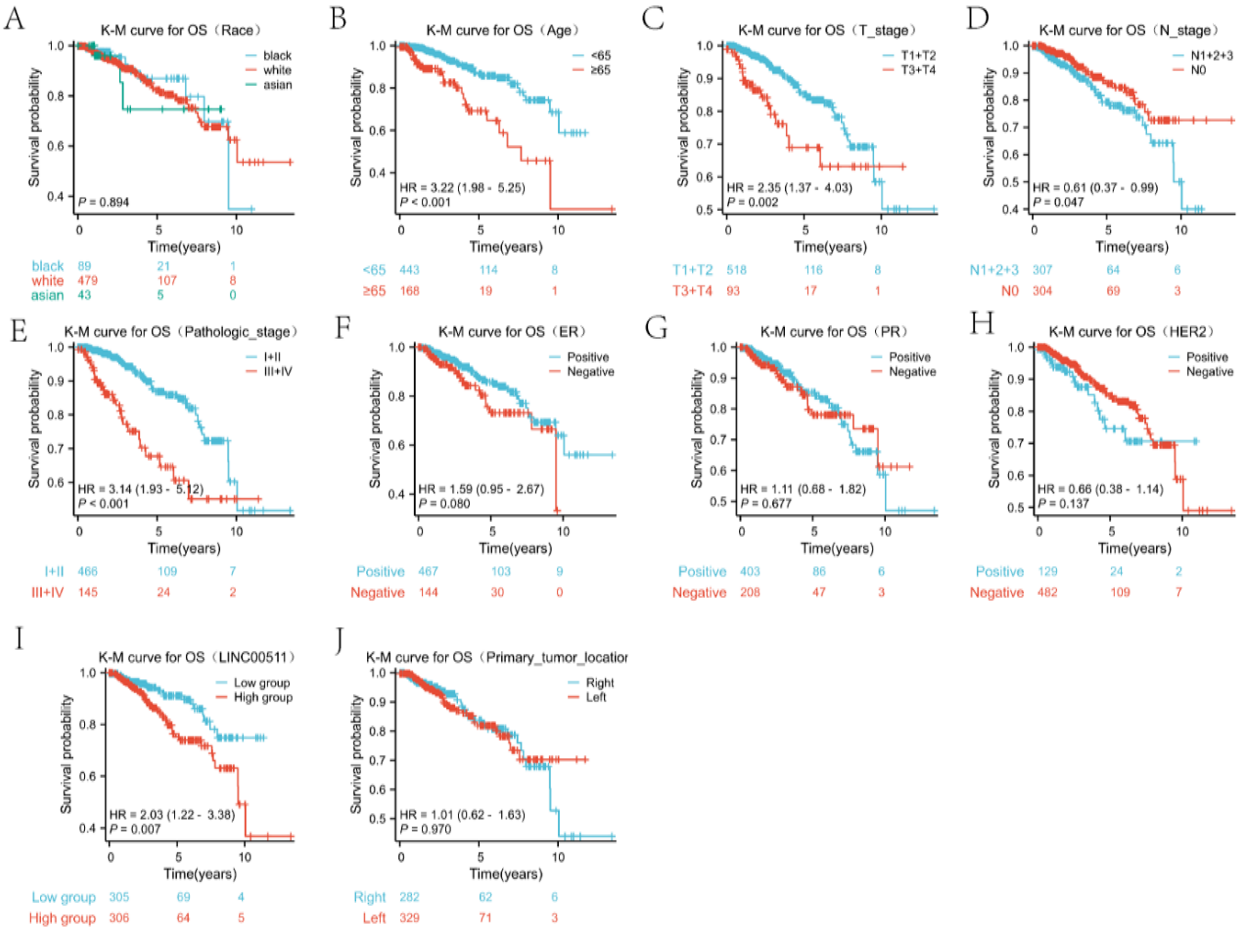

Figure 3. Kaplan-Meier Curves Assessing the Correlation between Race(A), Age(B), T stage(C), N stage(D), Pathological stage(E), ER status(F), PR status(G), *HER2* status(H) , *LINC00511* expression(I), Primary tumor location (J) and OS of patients.

is crucial for early diagnosis and the implementation of precise individualized treatment. The dysregulation of lncRNAs levels has been widely reported in the literature as a significant factor in the induction of various cancers [23]. Some lncRNAs have been shown to facilitate cancer screening and diagnosis, as well as predict survival outcomes in cancer patients. For example, lncRNA DARS-AS1 has high predictive value for the prognosis of gastric cancer, hepatocellular carcinoma, and lung adenocarcinoma [24], while lncRNA PCAT19 is downregulated in colorectal cancer and serves as a novel indicator for diagnosing colorectal cancer [25]. In this study, we focus on a novel *BRCA*-promoting gene, *LINC00511*.

Previous research has confirmed that LINC00511 is upregulated in *BRCA* [18]. Our study additionally revealed that *LINC00511* expression is markedly elevated in *BRCA* tissues relative to normal tissues, indicating that LINC00511 may be a pivotal factor in the predisposition to *BRCA*. Moreover, ROC analysis shows that LINC00511

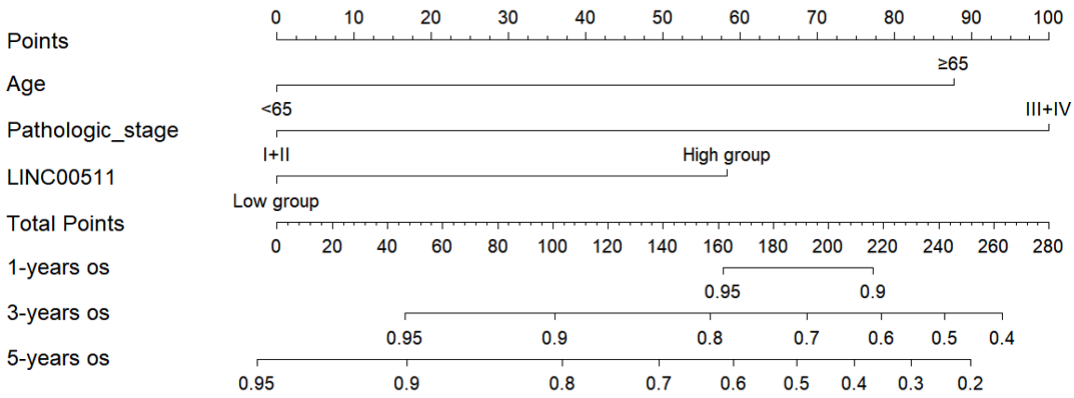

Figure 4. Nomogram Prediction Model for Prognostic Factors of *BRCA* Patients

Table 3. Cox Regression Analysis of *LINC00511* and Clinicopathological Features

Characteristics	Total(N)	Univariate analysis		Multivariate analysis	
		Hazard ratio (95% CI)	P value	Hazard ratio (95% CI)	P value
Race	611				
Black	89	Reference			
White	479	1.176 (0.581 - 2.381)	0.652		
Asian	43	1.227 (0.331 - 4.553)	0.76		
Age	611				
<65	443	Reference		Reference	
≥65	168	3.220 (1.975 - 5.250)	< 0.001	3.272 (1.987 - 5.386)	< 0.001
T_status	611				
T1+T2	518	Reference		Reference	
T3+T4	93	2.348 (1.367 - 4.035)	0.002	1.566 (0.809 - 3.031)	0.183
N_status	611				
N1+2+3	307	Reference		Reference	
N0	304	0.607 (0.371 - 0.994)	0.047	1.131 (0.586 - 2.183)	0.715
Pathologic_stage	611				
I+II	466	Reference		Reference	
III+IV	145	3.144 (1.932 - 5.116)	< 0.001	3.351 (1.584 - 7.087)	0.002
ER_status	611				
Positive	467	Reference		Reference	
Negative	144	1.590 (0.946 - 2.670)	0.08	1.361 (0.774 - 2.394)	0.284
PR_status	611				
Positive	403	Reference			
Negative	208	1.111 (0.677 - 1.824)	0.677		
*HER2*_status	611				
Positive	129	Reference			
Negative	482	0.657 (0.378 - 1.143)	0.137		
Primary_tumor_location	611				
Right	282	Reference			
Left	329	1.009 (0.624 - 1.634)	0.97		
LINC00511	611				
Low group	305	Reference		Reference	
High group	306	2.031 (1.219 - 3.383)	0.007	2.063 (1.172 - 3.633)	0.012

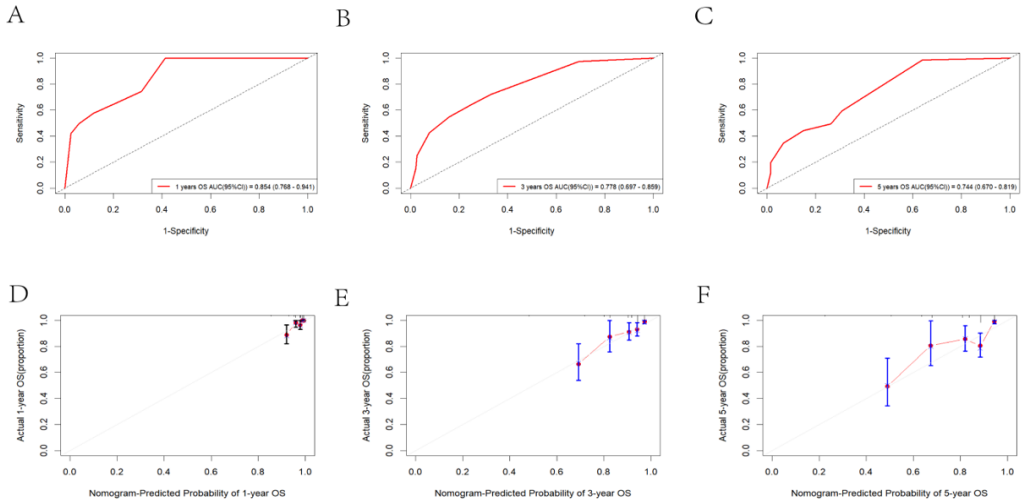

Figure 5. ROC and Calibration Curves of the Nomogram. (A)ROC curves of one-year OS for *BRCA* patients. (B) ROC curves of three-year OS for *BRCA* patients. (C) ROC curves of five-year OS for *BRCA* patients. (D)Calibration curves of one-year OS for *BRCA* patients. (E)Calibration curves of three-year OS for *BRCA* patients.(E)Calibration curves of five-year OS for *BRCA* patients.

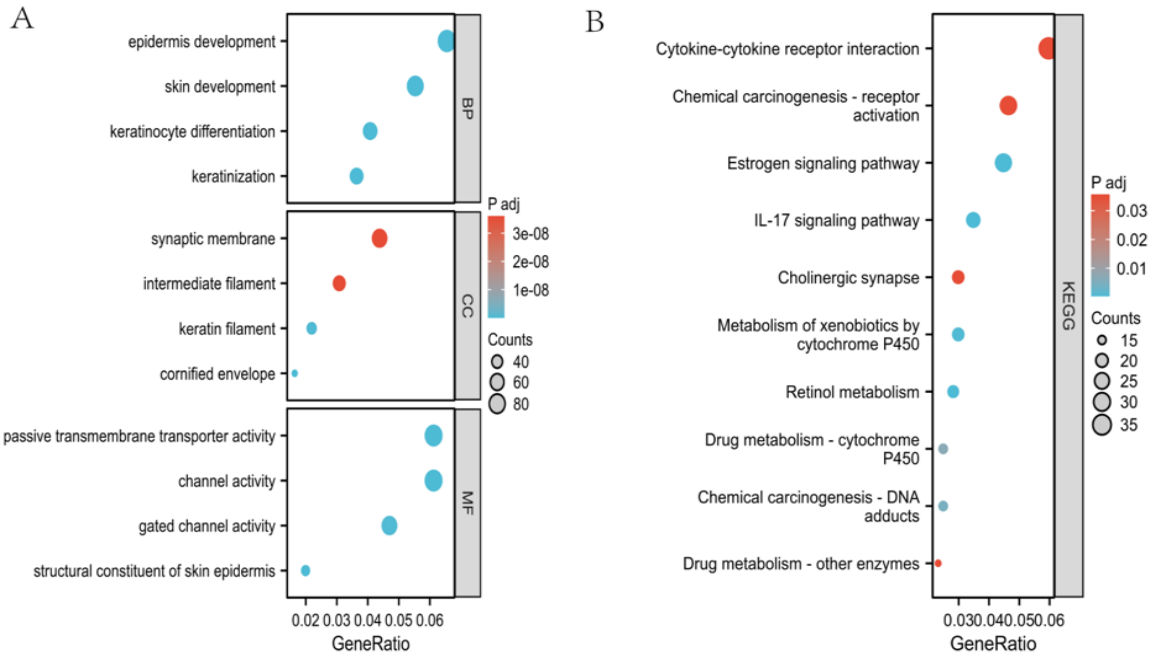

Figure 6. GO and KEGG Enrichment Analyses of Differentially Expressed Genes. (A) Gene Ontology (GO) enrichment dot plot of the differentially expressed genes (DEGs), shown for the three GO categories: Biological Process (BP), Cellular Component (CC), and Molecular Function (MF). The x-axis indicates the GeneRatio (the proportion of DEGs annotated to each term). Dot size represents the gene count (Counts) mapped to each term, and dot color denotes the adjusted P value (P adj).(B) Kyoto Encyclopedia of Genes and Genomes (KEGG) pathway enrichment dot plot of the DEGs. The x-axis indicates the GeneRatio. Dot size represents the gene count (Counts) in each pathway, and dot color denotes the adjusted P value (P adj).

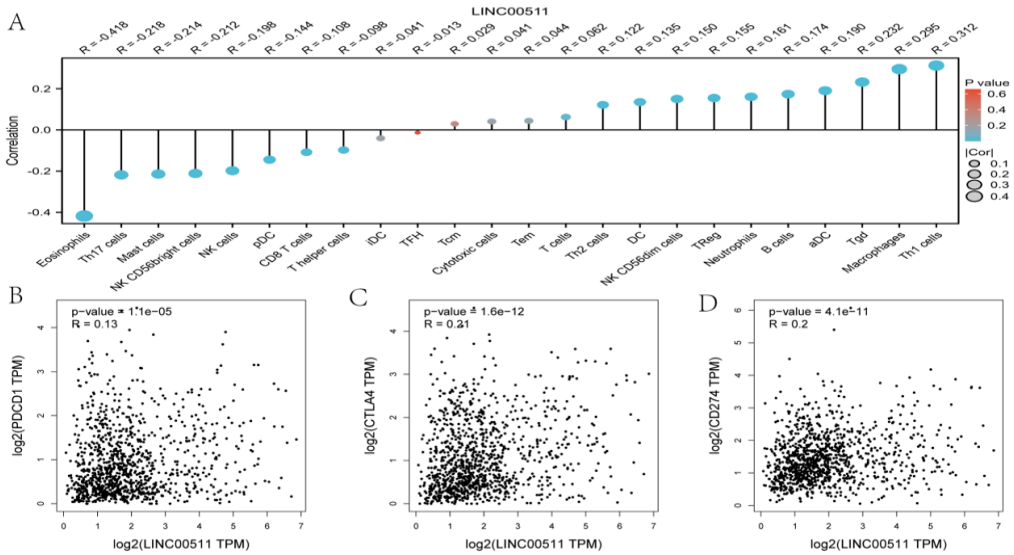

Figure 7. Correlation of *LINC00511* Expression with Immune Cell Infiltration and Immune Checkpoint Genes. (A) Lollipop plot showing the correlations between *LINC00511* expression and the estimated infiltration levels of multiple immune cell types. The x-axis lists immune cell types and the y-axis indicates the correlation coefficient. Dot color represents the P value, and dot size represents the magnitude of the correlation (|correlation|). (B–D) Scatter plots depicting correlations between *LINC00511* and immune checkpoint–related genes: PDCD1 (B), CTLA4 (C), and CD274 (D). The x-axis shows log2(*LINC00511* TPM) and the y-axis shows log2(gene TPM). The correlation coefficient (R) and P value are indicated in each panel.

has strong discriminatory ability for cancerous versus non-cancerous tissues, and Mahmoud's study on the serum of Egyptian *BRCA* patients also suggests that *LINC00511* possesses high diagnostic value in *BRCA* [12]. Further analysis reveals that *LINC00511* expression was significantly associated with ER status, PR status, T stage, and pathological stage, whereas no meaningful relationship was observed with tumor laterality, N stage, or HER2 status. These findings differ from those reported in the study by Lu, which identified no significant correlations between *LINC00511* expression and ER status, PR status, or HER2 status, but demonstrated strong associations with pathological stage and lymph node status [18]. The contradictory results can be attributed to two possible

reasons. First, the sample size difference: our study is a large-scale data study, while Lu's study included only 39 patients, possibly leading to errors; second, differences in patient populations: our study collected patients from around the world, while Lu's study included only Chinese patients. Therefore, our study's conclusions should be more convincing and require further validation with more *BRCA* tissue samples.

Survival analysis shows that both Kaplan-Meier and Cox regression results indicate that *BRCA* patients with high *LINC00511* expression have shorter OS. These findings indicate that *LINC00511* is highly valuable for assessing the prognosis of *BRCA* patients. Additionally, this study found that age, pathological stage, and *LINC00511* are independent prognostic factors for *BRCA*. Using these four prognostic factors identified through multifactorial analysis, we developed a clinical prediction model to estimate the OS of *BRCA*. Both ROC and calibration curves indicate that the clinical prediction model has good predictive value. In clinical practice, healthcare professionals can use the nomogram model to accurately and efficiently estimate the OS of *BRCA* , thereby enhancing work efficiency. The aforementioned studies clearly demonstrate the significance of *LINC00511* in *BRCA*. Therefore, a thorough exploration into the mechanisms of *LINC00511* in *BRCA* is essential.

Biological function and signaling pathway enrichment analyses, revealing that *LINC00511* is closely related to multiple conduction pathways, including IL-17 signaling pathway, chemical carcinogenesis, estrogen signaling pathway, and drug metabolism. IL-17 (interleukin-17) is an important cytokine involved in inflammatory response and immune regulation [26]. It has been reported in the literature that that IL-17 and its receptor are expressed in *BRCA* cells and the tumor microenvironment, influencing *BRCA*'s biological behavior through various mechanisms. The IL-17 signaling pathway can regulate *BRCA* cell proliferation, invasion, and metastasis by activating downstream signaling pathways such as the ERK (extracellular signal-regulated kinase) [27] and NF-κB (nuclear factor-κB) pathways [28], leading to an imbalance between cell proliferation and apoptosis, increasing tumor growth and metastasis capacity. Additionally, the IL-17 signaling pathway is related to tumor immune evasion, as *BRCA* cells can inhibit immune cell activity by expressing IL-17 receptors, thus evading immune surveillance and attack [29]. Therefore, *LINC00511* may participate in *BRCA* progression by regulating the IL-17 signaling pathway. Furthermore, the estrogen signaling pathway is another critical pathway in the context of *BRCA*. Estrogen is an important hormone that exerts its effects by binding to estrogen receptors (ER). In *BRCA*, *ER* can be divided into two types: *ERα* and *ERβ*, with *ERα* expression being more closely related to *BRCA* occurrence and development [30, 31]. When estrogen binds to ERα, it triggers a series of signal transduction pathways, including the Ras/MAPK (Ras/mitogen-activated protein kinase) and PI3K/AKT (phosphoinositide 3-kinase/protein kinase B) pathways, leading to cell proliferation, differentiation, and survival, thereby promoting *BRCA* cell growth and metastasis [32]. Our study indicates that *LINC00511* is related to the estrogen signaling pathway, and previous studies also suggest that *LINC00511* expression is closely related to ER status. These findings imply that *LINC00511* may exert its effects by regulating the estrogen signaling pathway.

A substantial body of research has demonstrated that the tumour microenvironment plays a pivotal role in regulating the growth and migration of tumour cells, as well as influencing the response of tumour cells to treatment [33]. This evidence indicates a strong correlation between the tumour microenvironment and the process of cancer development. Signalling pathway enrichment analysis showed that *LINC00511* was closely associated with the IL-17 pathway, and previous studies have indicated that the IL-17 pathway plays a pivotal role in the immune evasion process of tumour cells [29]. Consequently, it can be postulated that *LINC00511* may have a correlation with immune cells within the tumour microenvironment. To substantiate our suspicions, we sought to determine the relationship between *LINC00511* levels and the degree of infiltration of immune. In a surprising discovery, *LINC00511* showed a strong association with the infiltration degree of various immune, suggesting that *LINC00511* may influence *BRCA* development and prognosis through interactions with immune cells. As an emerging cancer treatment method, tumor immunotherapy is an important part of treating advanced malignant tumors. Research shows that two conditions need to be met for immunotherapy to be effective. Firstly, The tumour microenvironment must be adequately infiltrated by immune cells. Secondly, immune checkpoints must be expressed at an adequate leve [34]. Therefore, this study also assessed the correlation between *LINC00511* and three immune checkpoints. The findings indicated that *LINC00511* is closely related to PD1 (PDCD1), CTLA4, and PD-L1 (CD274), this suggests that *LINC00511* could potentially be targeted for future immunotherapy.

Although previous studies in *BRCA* and other malignancies have suggested that *LINC00511* may contribute to tumor initiation and progression, most available reports focus on a single aspect (e.g., differential expression or one specific pathway). A comprehensive study integrating expression patterns, prognostic value, potential mechanistic insights, and the tumor immune microenvironment within a unified framework remains limited. Building on the existing literature, our study advances current knowledge in the following ways:

Systematic clinical relevance: We validated the differential expression of *LINC00511* in *BRCA* cohorts and evaluated its association with overall survival, suggesting that *LINC00511* may serve as a potential prognostic biomarker.

Novel mechanistic clues: Through functional enrichment analyses (GO/KEGG) and correlation analyses, we identified that *LINC00511* may be involved in processes such as the IL-17 signaling pathway and the estrogen signaling pathway, providing testable hypotheses for subsequent mechanistic investigations.

An immune-related perspective: We further demonstrated significant associations between *LINC00511*

expression and immune cell infiltration, as well as the expression of immune checkpoint genes (e.g., PDCD1, CTLA4, and CD274), implying a potential link to immune microenvironment regulation and/or immunotherapy response. This complements prior studies in which immune-related aspects have been less thoroughly discussed.

Taken together, our findings provide a more integrated view of the role of *LINC00511* in *BRCA* and offer a rationale for future studies exploring its potential utility in patient stratification and therapeutic targeting.

Nevertheless, this study is primarily a retrospective analysis based on public databases and lacks systematic experimental validation at the cellular and animal levels. Future work will include in vitro gain- and loss-of-function assays, mechanistic experiments (e.g., RIP, ChIRP, dual-luciferase reporter assays, and ChIP-based analyses), and in vivo tumorigenesis/metastasis models to validate our observations and further elucidate the underlying molecular mechanisms.

In conclusion, *LINC00511* holds promise as a future biomarker for diagnosing, prognosticating, and guiding immunotherapy in *BRCA*. Nevertheless, the specific mechanisms of *LINC00511* in *BRCA* require further elucidation.

Author Contribution Statement

Sangita Biswas and Min Liu ; Conceptualization; Methodology; Investigation; Resources; Data Curation. Lifeng Zhao; writing-original draft preparation, writing-review and editing. Caixin Li; writing-review and editing. All authors have read and agreed to the published version of the manuscript.

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Data Availability Statement

All raw data for this study were obtained from the Public database, Further inquiries can be directed to the corresponding author.

Ethics Statement

All raw data for this study were obtained from the public database. Ethical review and approval were not required for the study on human participants in accordance with the relevant ethical standards.

Competing interests

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

References

1. Siegel RL, Miller KD, Wagle NS, Jemal A. Cancer statistics, 2023. CA Cancer J Clin. 2023;73(1):17-48. https://doi.org/10.3322/caac.21763.
2. Sung H, Ferlay J, Siegel RL, Laversanne M, Soerjomataram I, Jemal A, et al. Global cancer statistics 2020: Globocan estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin. 2021;71(3):209-49. https://doi.org/10.3322/caac.21660.
3. Isaac-Lam MF, DeMichael KM. Calorie restriction and breast cancer treatment: A mini-review. J Mol Med (Berl). 2022;100(8):1095-109. https://doi.org/10.1007/s00109-022-02226-y.
4. Statello L, Guo CJ, Chen LL, Huarte M. Gene regulation by long non-coding rnas and its biological functions. Nat Rev Mol Cell Biol. 2021;22(2):96-118. https://doi.org/10.1038/s41580-020-00315-9.
5. Yao RW, Wang Y, Chen LL. Cellular functions of long noncoding rnas. Nat Cell Biol. 2019;21(5):542-51. https://doi.org/10.1038/s41556-019-0311-8.
6. Zhao Y, Teng H, Yao F, Yap S, Sun Y, Ma L. Challenges and strategies in ascribing functions to long noncoding rnas. Cancers (Basel). 2020;12(6). https://doi.org/10.3390/cancers12061458.
7. Kopp F, Mendell JT. Functional classification and experimental dissection of long noncoding rnas. Cell. 2018;172(3):393-407. https://doi.org/10.1016/j.cell.2018.01.011.
8. Xia Y, Pei T, Zhao J, Wang Z, Shen Y, Yang Y, et al. Long noncoding rna h19: Functions and mechanisms in regulating programmed cell death in cancer. Cell Death Discov. 2024;10(1):76. https://doi.org/10.1038/s41420-024-01832-8.
9. Wang Y, Mei X, Song W, Wang C, Qiu X. Lncrna linc00511 promotes col1a1-mediated proliferation and metastasis by sponging mir-126-5p/mir-218-5p in lung adenocarcinoma. BMC Pulm Med. 2022;22(1):272. https://doi.org/10.1186/s12890-022-02070-3.
10. Mao BD, Xu P, Xu P, Zhong Y, Ding WW, Meng QZ. Linc00511 knockdown prevents cervical cancer cell proliferation and reduces resistance to paclitaxel. J Biosci. 2019;44(2).
11. Cabanski CR, White NM, Dang HX, Silva-Fisher JM, Rauck CE, Cicka D, et al. Pan-cancer transcriptome analysis reveals long noncoding rnas with conserved function. RNA Biol. 2015;12(6):628-42. https://doi.org/10.1080/15476286.2015.1038012.
12. Mahmoud MM, Sanad EF, Elshimy RAA, Hamdy NM. Competitive endogenous role of the linc00511/mir-185-3p axis and mir-301a-3p from liquid biopsy as molecular markers for breast cancer diagnosis. Front Oncol. 2021;11:749753. https://doi.org/10.3389/fonc.2021.749753.
13. Sun S, Xia C, Xu Y. Hif-1α induced lncrna linc00511 accelerates the colorectal cancer proliferation through positive feedback loop. Biomed Pharmacother. 2020;125:110014. https://doi.org/10.1016/j.biopha.2020.110014.
14. Sun CC, Li SJ, Li G, Hua RX, Zhou XH, Li DJ. Long intergenic noncoding rna 00511 acts as an oncogene in non-small-cell lung cancer by binding to ezh2 and suppressing p57. Mol Ther Nucleic Acids. 2016;5(11):e385. https://doi.org/10.1038/mtna.2016.94.
15. Lu M, Gao Q, Wang Y, Ren J, Zhang T. Linc00511 promotes cervical cancer progression by regulating the mir-497-5p/mapk1 axis. Apoptosis. 2022;27(11-12):800-11. https://doi.org/10.1007/s10495-022-01768-3.
16. Wang D, Liu K, Chen E. Linc00511 promotes proliferation and invasion by sponging mir-515-5p in gastric cancer. Cell Mol Biol Lett. 2020;25:4. https://doi.org/10.1186/s11658-020-0201-x.
17. Wang RP, Jiang J, Jiang T, Wang Y, Chen LX. Increased long noncoding rna linc00511 is correlated with poor prognosis

and contributes to cell proliferation and metastasis by modulating mir-424 in hepatocellular carcinoma. Eur Rev Med Pharmacol Sci. 2019;23(8):3291-301. https://doi.org/10.26355/eurrev_201904_17691.
18. Lu G, Li Y, Ma Y, Lu J, Chen Y, Jiang Q, et al. Long noncoding rna linc00511 contributes to breast cancer tumourigenesis and stemness by inducing the mir-185-3p/e2f1/nanog axis. J Exp Clin Cancer Res. 2018;37(1):289. https://doi.org/10.1186/s13046-018-0945-6.
19. Tang Z, Li C, Kang B, Gao G, Li C, Zhang Z. Gepia: A web server for cancer and normal gene expression profiling and interactive analyses. Nucleic Acids Res. 2017;45(W1):W98-w102. https://doi.org/10.1093/nar/gkx247.
20. Zhu Y, Chen Y, Xie D, Xia D, Kuang H, Guo X, et al. Macrophages depletion alleviates lung injury by modulating akt3/gxp4 following ventilator associated pneumonia. Front Immunol. 2023;14:1260584. https://doi.org/10.3389/fimmu.2023.1260584.
21. Anastasiadi Z, Lianos GD, Ignatiadou E, Harissis HV, Mitsis M. Breast cancer in young women: An overview. Updates Surg. 2017;69(3):313-7. https://doi.org/10.1007/s13304-017-0424-1.
22. Siegel RL, Miller KD, Jemal A. Cancer statistics, 2020. CA Cancer J Clin. 2020;70(1):7-30. https://doi.org/10.3322/caac.21590.
23. Cui M, Liu Y, Cui L. Long non-coding rna linc02613 is a prognostic biomarker for breast cancer and correlates with the cell cycle and immune infiltration based on tcga data. Transl Cancer Res. 2022;11(4):615-28. https://doi.org/10.21037/tcr-21-2479.
24. Shu J, Xia K, Luo H, Wang Y. Dars-as1: A vital oncogenic lncrna regulator with potential for cancer prognosis and therapy. Int J Med Sci. 2024;21(3):571-82. https://doi.org/10.7150/ijms.90611.
25. Masoud A, Mohamadynejad P. Identification of lncrna pcat19 as potential novel biomarker for colorectal cancer. Gene. 2024;891:147828. https://doi.org/10.1016/j.gene.2023.147828.
26. Zhang Q, Liu S, Parajuli KR, Zhang W, Zhang K, Mo Z, et al. Interleukin-17 promotes prostate cancer via mmp7-induced epithelial-to-mesenchymal transition. Oncogene. 2017;36(5):687-99. https://doi.org/10.1038/onc.2016.240.
27. Laprevotte E, Cochaud S, du Manoir S, Lapierre M, Dejou C, Philippe M, et al. The il-17b-il-17 receptor b pathway promotes resistance to paclitaxel in breast tumors through activation of the erk1/2 pathway. Oncotarget. 2017;8(69):113360-72. https://doi.org/10.18632/oncotarget.23008.
28. Huang CK, Yang CY, Jeng YM, Chen CL, Wu HH, Chang YC, et al. Autocrine/paracrine mechanism of interleukin-17b receptor promotes breast tumorigenesis through nf-κb-mediated antiapoptotic pathway. Oncogene. 2014;33(23):2968-77. https://doi.org/10.1038/onc.2013.268.
29. Coffelt SB, Kersten K, Doornebal CW, Weiden J, Vrijland K, Hau CS, et al. Il-17-producing γδ t cells and neutrophils conspire to promote breast cancer metastasis. Nature. 2015;522(7556):345-8. https://doi.org/10.1038/nature14282.
30. Gopinath P, Oviya RP, Gopisetty G. Oestrogen receptor-independent actions of oestrogen in cancer. Mol Biol Rep. 2023;50(11):9497-509. https://doi.org/10.1007/s11033-023-08793-8.
31. Moisand A, Madéry M, Boyer T, Domblides C, Blaye C, Larmonier N. Hormone receptor signaling and breast cancer resistance to anti-tumor immunity. Int J Mol Sci. 2023;24(20). https://doi.org/10.3390/ijms242015048.
32. Clusan L, Ferrière F, Flouriot G, Pakdel F. A basic review on estrogen receptor signaling pathways in breast cancer. Int J Mol Sci. 2023;24(7). https://doi.org/10.3390/ijms24076834.
33. Deng M, Xiong C, He ZK, Bin Q, Song JZ, Li W, et al. Mcts1 as a novel prognostic biomarker and its correlation with immune infiltrates in breast cancer. Front Genet. 2022;13:825901. https://doi.org/10.3389/fgene.2022.825901.
34. Lou W, Wang W, Chen J, Wang S, Huang Y. Ncrnas-mediated high expression of sema3f correlates with poor prognosis and tumor immune infiltration of hepatocellular carcinoma. Mol Ther Nucleic Acids. 2021;24:845-55. https://doi.org/10.1016/j.omtn.2021.03.014.