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Research article

Unveiling the prognostic value of ARID3A in breast cancer through bioinformatic analysis

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ABSTRACT

Objective: Identifying reliable prognostic markers for breast cancer is crucial for improving survival rates and reducing mortality. Recent studies highlight the AT-rich interactive domain-containing protein (ARID) family, particularly ARID3A, as influential in cancer progression, though its specific role in breast cancer remains unclear. This study investigates ARID3A's expression, prognostic relevance, clinicopathological correlations, co-expression profiles, and protein-protein interactions in breast cancer.

Methods: ARID3A mRNA and protein expression levels were analyzed using UALCAN, GEPIA databases, and immunohistochemistry from our hospital samples. Clinical prognostic parameters and survival data were examined through bioinformatics tools, including GEPIA, Bc-GenExMiner, and BEST. Subtype-specific expression and co-expression, particularly with REXO1, were evaluated using LinkedOmics, TIMER, and bc-GenExMiner. Functional enrichment analysis was conducted *via* LinkedOmics. Protein-protein interactions (PPI) were established using GeneMANIA and STRING, with validation through molecular docking using Cluspro.

Results: Elevated ARID3A expression was associated with poor prognosis in breast cancer, particularly in Luminal and HER2-positive subtypes. A positive correlation with REXO1 was identified, and enrichment analysis emphasized ARID3A's involvement in immune-related pathways, such as "interferon gamma production" and "primary immunodeficiency." PPI network and docking studies identified TP53 as a potential binding partner, suggesting a novel interaction influencing tumor progression.

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Conclusion: These findings indicate that ARID3A may serve as a prognostic biomarker and therapeutic target in breast cancer, providing insights into its involvement in oncogenic pathways and interactions, particularly with TP53, that may drive cancer development and progression.

1. Introduction

Recent cancer statistics indicate that breast cancer is the most common and deadly cancer among women, accounting for approximately 31 % of all cancers [1]. While breast cancer incidence has increased annually, the five-year survival rate has increased from 75 % in 1975 to 91 % in 2018. Although the median age at diagnosis is declining among survivors receiving relatively limited treatment, such as radiotherapy, chemotherapy, and surgery, tumor metastasis rates are still rising. This indicate that breast cancer remains a challenging disease for patients and researchers, highlighting the urgent need for new diagnostic biomarkers and therapeutic targets [2].

The AT-rich interaction domain 3A (ARID3A) is a transcription factor that binds to specific DNA sequences, playing a crucial role in regulating gene expression. Evidence suggests that ARID3A is implicated in a range of diseases, including leukemia [3–5], systemic lupus erythematosus [6], and cholestasis [7]. Studies indicate that ARID3A often acts as a tumor-promoting factor. In pancreatic cancer [8], ARID3A enhances chemoresistance by inhibiting PTEN-induced ferroptosis, while in colorectal cancer [9], it drives tumor progression through the upregulation of AURKA. Interestingly, in colon cancer, ARID3A has also been reported to increase chemosensitivity by suppressing AKR1C3. Despite these findings, the precise role of ARID3A in human diseases, particularly breast cancer, remains insufficiently understood.

In this study, we explored alterations in ARID3A expression, assessed its prognostic significance, examined transcript interactions, and identified altered signaling pathways during the progression of breast cancer. We achieved this by leveraging online databases, with the goal of providing bioinformatic evidence for the significance of ARID3A as a novel biomarker and identifying potential therapeutic targets in breast cancer.

2. Material and methods

2.1. Differential ARID3A expression analysis

An analysis of ARID3A mRNA expression across cancers was conducted by Tumor IMmune Estimation Resource (TIMER) database (http://timer.cistrome.org/) and The University of ALabama at Birmingham CANcer database (UALCAN) databases (https://ualcan.path.uab.edu/). Additionally, the protein expression of ARID3A in human cancers was investigated using Clinical Proteomic Tumor Analysis Consortium (CPTAC) data from the UALCAN database. Then, ARID3A mRNA expression levels in breast cancer were validated utilizing data from GEPIA (http://gepia.cancer-pku.cn/), and UALCAN databases.

2.2. Survival analysis of ARID3A

Gene Expression Profiling Interactive Analysis (GEPIA) and bc-GenExMiner databases (http://gepia.cancer-pku.cn/, https://bcgenex.ico.unicancer.fr/) were used to evaluate how ARID3A mRNA expression affects survival in breast cancer patients. The prognostic values of ARID3A in different cohorts were also investigated using Biomarker Exploration of Solid Tumors (BEST) database (https://rookieutopia.com/app_direct/BEST/)

2.3. Association between ARID3A expression and clinicopathological features

Using the bc-GenExMiner, ARID3A expression levels were analyzed in relation to breast cancer clinicopathological characteristics, including age, estrogen receptor (ER), progesterone receptor (PR), human epidermal growth factor receptor 2 (HER2), p53, Scarff-Bloom-Richardson (SBR) grades, Nottingham prognostic index (NPI), and molecular subtype.

2.4. Molecular docking

The three-dimensional models of ARID3A (PDB ID 4LJX) and TP53 (PDB ID 2OCJ) were retrieved from RCSB Protein Data Bank (https://www.rcsb.org/). Docking was done in Cluspro 2.0 (https://cluspro.org/) [10]. The results of the molecular docking were visualized using PyMOL.

2.5. Immunohistochemistry (IHC)

Formalin-fixed, paraffin-embedded (FFPE) breast cancer and paired normal tissue sections used a rabbit monoclonal ARID3A antibody (Abcam, ab169787). Sections (4-μm) were deparaffinized, rehydrated, and underwent antigen retrieval in citrate buffer. After overnight incubation at 4 °C with primary antibody (1:50), sections were treated with an HRP-conjugated secondary antibody (1:500) and visualized using DAB. Images were captured with a KEYENCE BZ-X800 microscope. IHC scoring [11] was based on stained

cell percentage (0: 0 %, 1: 1–25 %, 2: 26–50 %, 3: 51–75 %, 4: >75 %) and staining intensity (0: negative, 1: weak, 2: moderate, 3: strong), yielding scores (0–12) summarized in scatter plots.

2.6. Statistical analysis

All statistical analyses were conducted using GraphPad Prism version 10.3.1. Data are presented as the mean ± standard deviation (SD), unless specified otherwise. Spearman correlation analysis was used to detect the correlation among the observed indicators. A Student's t-test was applied for comparisons between two groups, while one-way ANOVA with Bonferroni post-hoc analysis was used for multiple group comparisons. A two-tailed p-value <0.05 was considered statistically significant.

3. Results

3.1. ARID3A expression in pan-cancer and breast cancer

To investigate the expression profile of ARID3A across different cancers, we first utilized TIMER and UALCAN databases. TIMER analysis showed that ARID3A mRNA expression is significantly higher in most cancer types, including breast cancer and 15 other types, compared to normal tissues (Figure S1A). This observation was further supported by UALCAN, which revealed that ARID3A is over-expressed in most tumor types (Fig. S1B). To focus specifically on breast cancer, we examined ARID3A mRNA expression levels in tumor versus adjacent normal tissues using GEPIA and UALCAN databases. GEPIA analysis showed higher ARID3A expression in breast cancer (1085 cases) relative to normal breast tissues (112 cases) ($p < 0.05$, Fig. 1A), consistent with UALCAN results (Fig. 1B). Additionally, CPTAC analysis confirmed significant upregulation of ARID3A at the protein level in most tumor tissues (Fig. 1C). This finding aligns with UALCAN data, which also demonstrated higher ARID3A protein expression in breast cancer tissue compared to normal breast tissue (Fig. 1D). Together, these results suggest that ARID3A is upregulated at both mRNA and protein levels in breast cancer, indicating its potential role as an oncogene.

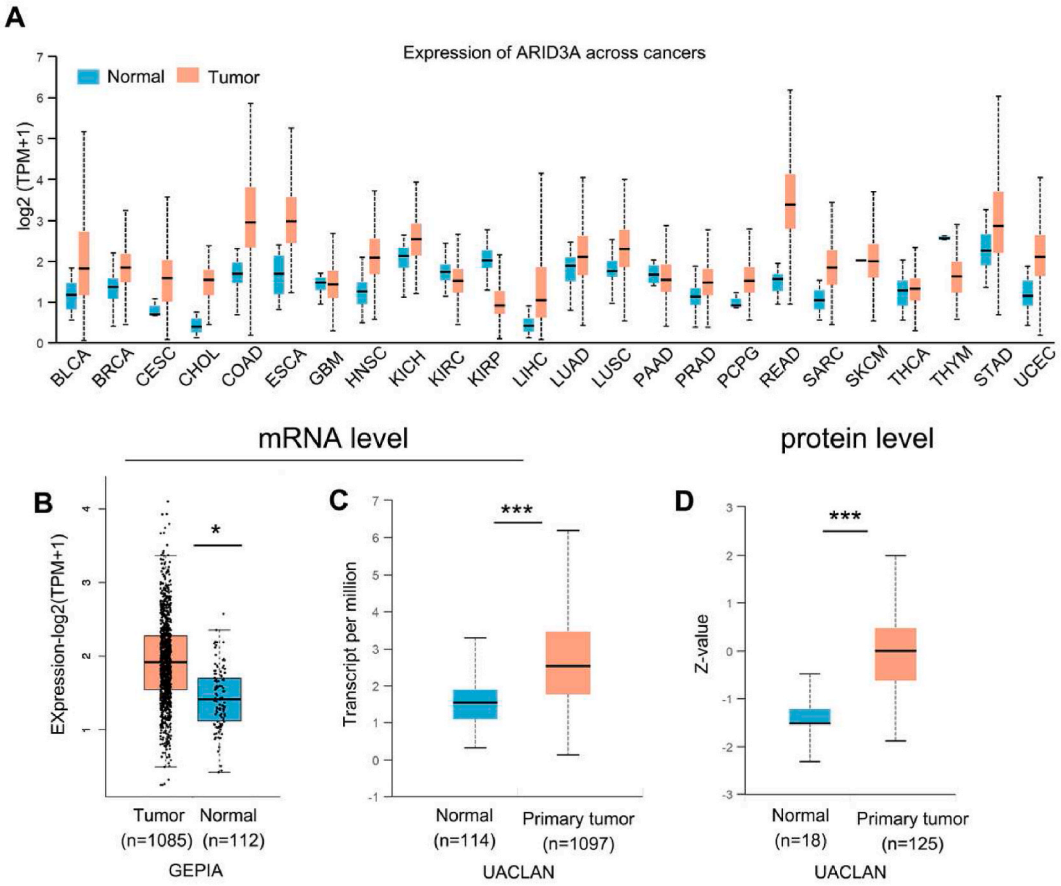

Fig. 1. ARID3A Expression Levels in Human Cancers. (A) Pan-cancer analysis displaying differential ARID3A expression across various cancers using the UALCAN database. (B & C) ARID3A mRNA expression in breast cancer compared to normal tissues, as analyzed by the GEPIA (B) and UALCAN (C) databases. (D)Detection of ARID3A protein in both breast cancer and normal tissues. (*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$).

3.2. Correlation between ARID3A expression and breast cancer prognosis

To explore the prognostic relevance of ARID3A expression in breast cancer, we examined its correlation with clinical outcomes using data from GEPIA, bc-GenExMiner, and BEST databases. Initially, GEPIA data showed that higher ARID3A mRNA levels are significantly associated with worse overall survival (OS) (Fig. 2A, $p < 0.05$, HR = 1.5). No significant correlation, however, was found between ARID3A expression and disease-free survival (DFS) (Fig. 2B, $p = 0.05$, HR = 1.2). We then validated these findings using the bc-GenExMiner dataset, which included a larger sample size. This analysis confirmed that elevated ARID3A expression predicts a poorer prognosis, including worse OS (Fig. 2C, HR = 1.24, 95 % CI = 1.01 to 1.53, $p = 0.0410$) and DFS (Fig. 2D, HR = 1.25, 95 % CI = 1.02 to 1.53, $p = 0.0292$). Furthermore, BEST database analysis showed a consistent trend: higher ARID3A expression was negatively associated with OS, and lower ARID3A expression corresponded to better RFS (recurrence-free survival), PFS (progression-free survival), and DFS (Fig. 2E–J). The sample sizes used in each survival analysis across the different datasets are summarized in Table S2.

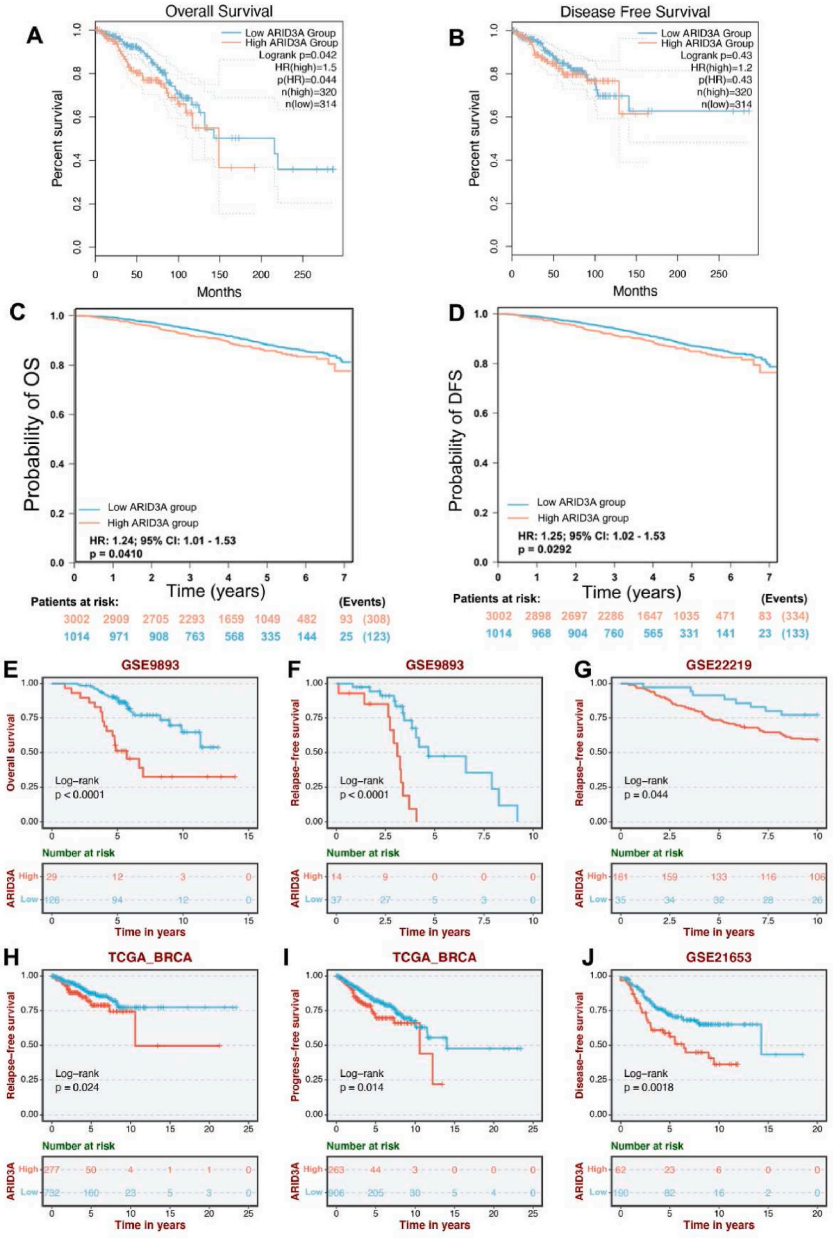

Fig. 2. Correlation Between Breast Cancer Survival Prognosis and ARID3A Expression Across Different Datasets. (A & B) Analysis of overall survival (OS)(A) and disease-free survival (DFS) (B) using the GEPIA dataset. (C & D) Assessment of OS (C) and DFS (D) with the bc-GenExMiner dataset. (E–J) Evaluation of OS (E), relapse-free survival (RFS) (F–H), progression-free survival (PFS), and DFS across various cohorts using the BEST dataset.

These findings collectively support the prognostic importance of ARID3A in breast cancer, suggesting its role in predicting patient outcomes.

3.3. ARID3A expression and breast cancer clinicopathological characteristics

To understand the relationship between ARID3A expression and specific clinicopathological features in breast cancer, we conducted a further analysis using the bc-GenExMiner database. Higher ARID3A expression was found in patients with positive lymph node status (LN+) compared to those with negative nodal status (LN-) ($p < 0.001$, Fig. 3A). Additionally, the Scarff-Bloom-Richardson (SBR) grading showed a positive correlation between higher SBR grade and increased ARID3A transcription levels (SBR3 > SBR2 > SBR1, $p < 0.001$, Fig. 3B). Notably, higher Nottingham Prognostic Index (NPI) scores were also associated with elevated ARID3A expression (Fig. 3C). Further, high ARID3A expression was linked to certain breast cancer features: lymph node metastasis-positive, p53 wildtype, HER2-positive, non-basal-like, and non-TNBC (triple-negative breast cancer) (Fig. 3D–F, Table 1). No correlation, however, was found with age, ER, or PR status. These associations emphasize the potential clinical utility of ARID3A in characterizing breast cancer.

To validate the database findings, we performed immunohistochemistry (IHC) to assess ARID3A expression in breast cancer tissues and paired normal samples from patients at our hospital (Fig. 4). A total of 50 paired samples from breast cancer patients were included in the IHC analysis. The results demonstrated a significantly higher expression of ARID3A in breast cancer tissues compared to normal tissues. Consistent with the database data, elevated ARID3A expression was associated with a higher histopathologic grade. Furthermore, ARID3A levels were higher in Luminal and HER2-positive subtypes compared to the TNBC subtype.

3.4. Co-expression analysis of ARID3A

Using LinkedOmics, we identified genes co-expressed with ARID3A in a cohort of 121 breast cancer patients (CPTAC dataset) to understand its biological and molecular interactions. We identified 1676 genes that significantly co-expressed with ARID3A (FDR <0.05), indicating ARID3A's critical role in breast cancer pathogenesis. In the LinkFinder module, genes positively correlated with ARID3A are shown as red dots, while negatively correlated genes are shown in green (Fig. 5A). Heatmaps of the top 50 positively and

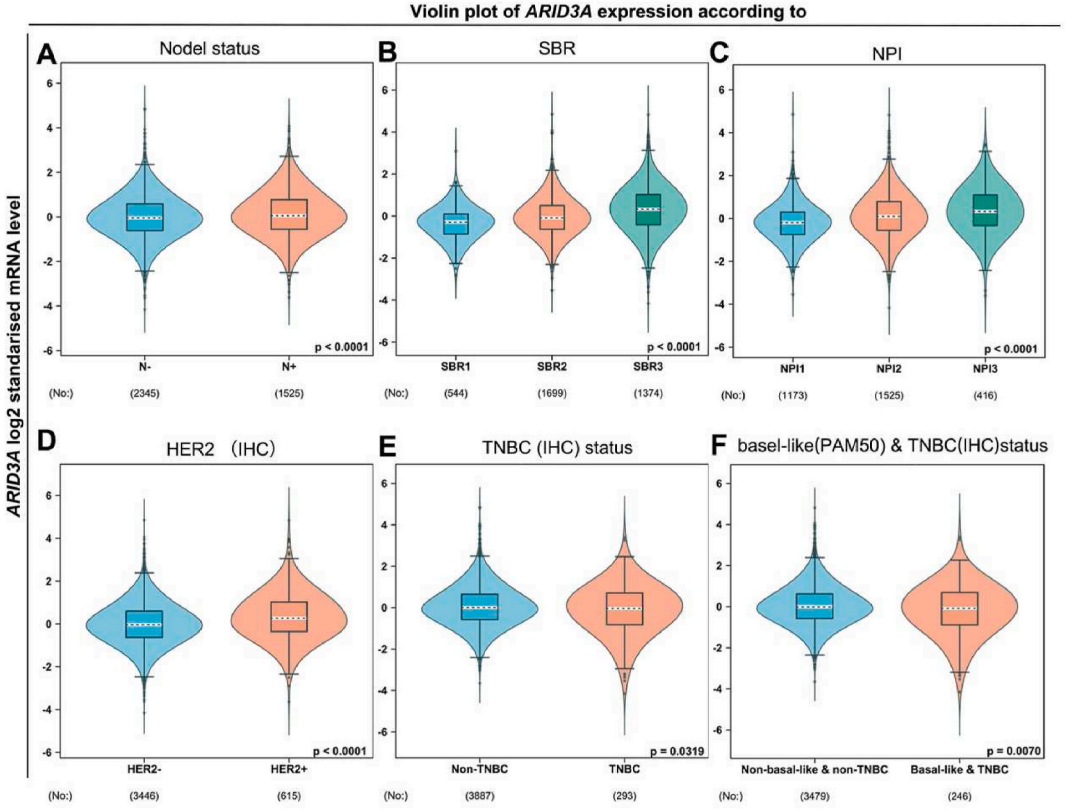

Fig. 3. ARID3A Gene Expression in Breast Cancer Patients by Clinical Parameters, as Evaluated by bc-GenExMiner. (A) Node status, (B) SBR grade, (C) Nottingham Prognostic Index (NPI), (D) HER2 status (IHC), (E) Triple-Negative Breast Cancer (TNBC) status (IHC), and (F) Basal-like status and TNBC. Abbreviations: ER, estrogen receptor; PR, progesterone receptor; HER2, human epidermal growth factor receptor 2; SBR, Scarff-Bloom-Richardson; TNBC, triple-negative breast cancer.

Table 1
ARID3A expression in different clinical parameters of breast cancer with Bc-GenExMiner v5.0.

Variables	No. of patients	ARID3A mRNA	*p*-value
Age			0.1388
≤51	1021		
>51	2995	-	
SBR			<0.0001
SBR1	544		
SBR2	1699		
SBR3	1374	Increased	
NPI			<0.0001
NPI1	1173		
NPI2	1525		
NPI3	416	Increased	
p53status (sequence-based)			<0.0001
Wild type	477		
Mutated	252	Decreased	
ER			0.7701
Negative	3685		
Positive	510	-	
PR			0.161
Negative	3312		
Positive	746	-	
HER2			<0.0001
Negative	3446		
Positive	615	Increased	
Nodal status			<0.0001
Negative	2345		
Positive	1525	Increased	
Basal-like status (PAM50)			0.7442
Non-basal-like	3604		
Basal-like	783	-	
Triple-negative status (IHC)			0.0319
Non-triple-negative	3887		
Triple-negative	293	Decreased	
Basal-like (PAM50) & TNBC (IHC) status			0.007
Non-basal-like & non-TNBC	3479		
Basal-like & TNBC	246	Decreased	

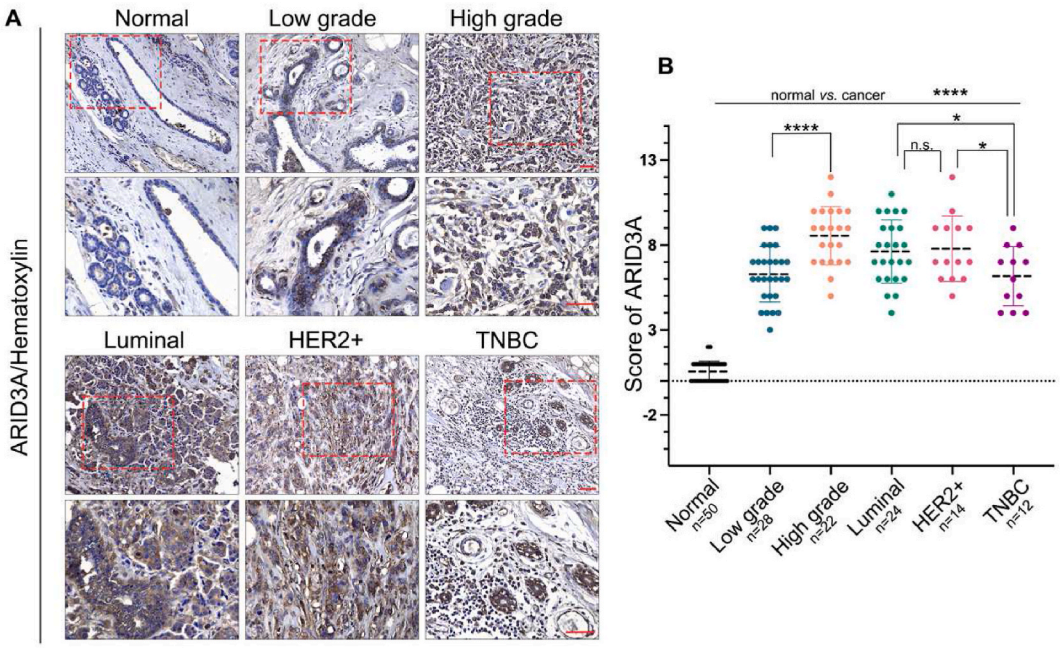

Fig. 4. The expression of ARID3A in breast cancer (A) Representative images of ARID3A IHC staining in breast cancer tissues with paired normal samples. Scale bars, 50 μm. (B) IHC analysis was performed on 50 samples, with the resulting IHC scores presented as scatter plots. (Data are shown as mean ± SD; ****$p < 0.0001$, *$p < 0.05$, n.s. indicates no significance.)

negatively correlated genes highlighted REXO1 as the most significant positive correlate (r = 0.543, p = 1.24e-10, Fig. 5B–D). Analyzing TIMER data showed that the ARID3A-REXO1 correlation varies among breast cancer molecular subtypes, being higher in luminal A and luminal B subtypes (Fig. 5E), which was further supported by bc-GenExMiner analysis (Fig. 5F). The results indicate that ARID3A may have specific regulatory roles in breast cancer, particularly in the Luminal A and Luminal B subtypes, through its co-expression with REXO1.

3.5. *Enrichment analyses of genes co-expressed with ARID3A*

Using LinkedOmics' LinkInterpreter module, we performed gene set enrichment analysis (GSEA) to examine biological pathways linked with ARID3A co-expression. Gene ontology (GO) biological process analysis revealed enrichment in immune-related functions, including respiratory burst, interferon-gamma production, and cell activation regulation (Fig. 6A). GO cellular component analysis showed that ARID3A is primarily associated with structures like the phagocytic cup, heterochromatin, and collagen trimer (Fig. 6B). GO molecular function analysis indicated significant correlations with chromatin binding, Rho GTPase binding, and MHC protein binding (Fig. 6C). Kyoto encyclopedia of genes and genomes (KEGG) pathway analysis also identified primary immunodeficiency, the intestinal immune network for IgA production, and the NF-kappa B signaling pathway as key pathways associated with ARID3A co-expression (Fig. 6D). These findings collectively highlight ARID3A's role in immune and molecular signaling pathways.

3.6. *The protein-protein interaction network of ARID3A*

Protein-protein interactions underpin many biological processes. To identify potential ARID3A binding partners, we used the GeneMANIA and STRING databases to construct a protein-protein interaction network (Fig. 7A and B), revealing BTK, TP53, ARID3B, and ARID3C as possible interacting proteins (Fig. 7C). Previous studies have documented ARID3A's interactions with BTK [12], ARID3B [13], and ARID3C [14], but its interaction with TP53 (tumor protein p53) remains unstudied. Given TP53's oncogenic role and its co-localization with ARID3A, we performed molecular docking experiments to explore this potential binding. The results indicate that ARID3A can form stable hydrogen bonds with TP53, with donor-acceptor distances below 3 Å, suggesting strong interaction stability (Fig. 7D). Notably, ARID3A binds to several key residues within TP53's DNA-binding domain (DBD) [15], specifically ARG-181, LYS-120, SER-261, and ASP-207. Based on these findings, we hypothesize that in breast cancer, high ARID3A expression may competitively bind to TP53's DBD, blocking TP53's DNA-binding region from engaging with target gene promoter regions. This interaction could inhibit TP53's transcriptional activation of tumor suppressor genes, potentially impacting its transcriptional activity and promoting tumor progression.

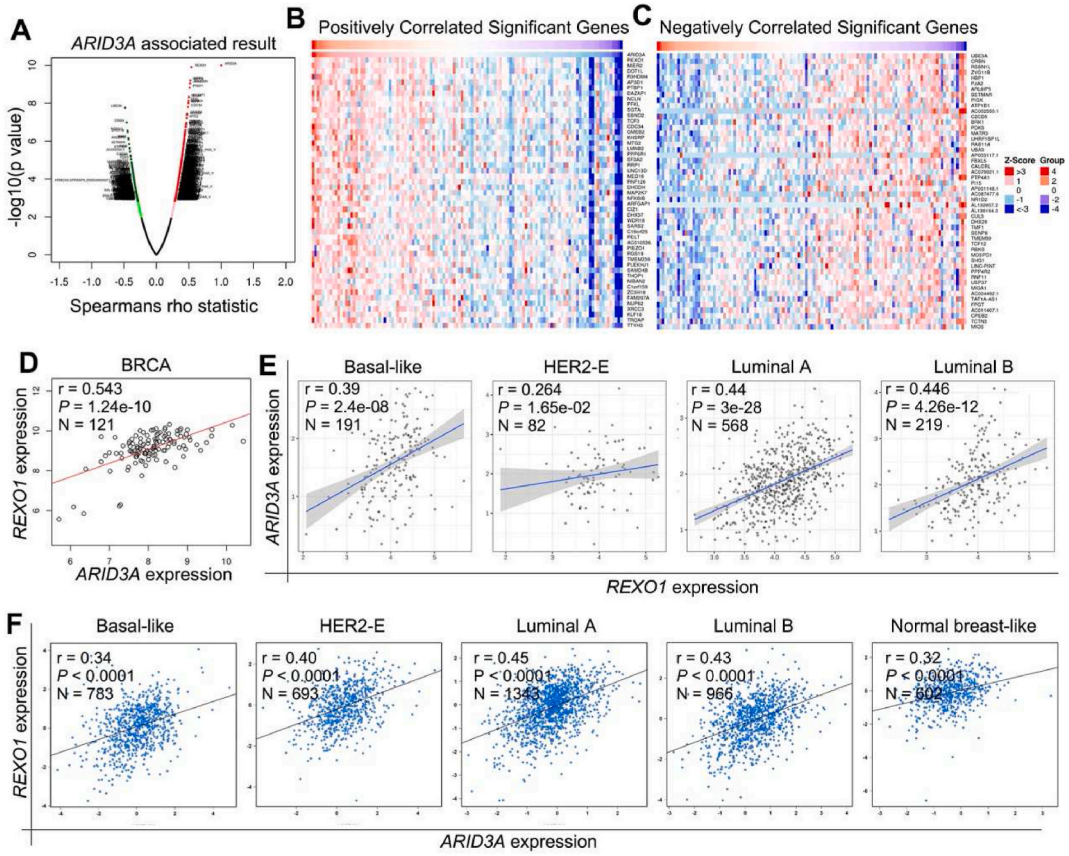

Fig. 5. ARID3A Co-expression Genes in Breast Cancer and Its Molecular Subtypes. (A) Genes highly correlated with ARID3A, determined by Spearman's test (LinkedOmics). (B & C) Heat maps displaying the top 50 positively (B) and negatively (C) co-expressed genes with ARID3A in breast cancer (LinkedOmics). (D).Spearman correlation between ARID3A and REXO1 in breast cancer (LinkedOmics). (***E***).Spearman correlation between ARID3A and REXO1 across different molecular subtypes of breast cancer (TIMER). (F). Pearson correlation of ARID3A with REXO1 in molecular subtypes and normal breast tissue (Bc-GenExMiner).

4. Discussion

Accumulating evidence has shown that the ARID family plays a crucial role in cancer development and progression. As a key member of this family, ARID3A exhibits differential expression across various diseases. Although ARID3A has been associated with tumor progression in several cancers, its prognostic significance in breast cancer remains insufficiently understood.

In this study, both bioinformatic and IHC analyses consistently reveal elevated ARID3A expression in breast cancer tissues compared to normal counterparts. This overexpression correlates with more aggressive clinical features, including high SBR grades, HER2-positive, and lymph node metastasis—indicators of more invasive and rapidly proliferating tumors.

Elevated ARID3A levels are especially notable in Luminal and HER2-positive breast cancer subtypes, with lower expression observed in TNBC. This suggests that ARID3A may activate distinct molecular pathways within specific subtypes, fostering proliferation and survival in Luminal and HER2-positive cancers. Co-expression analysis further supports this, showing a positive correlation between ARID3A and REXO1, particularly in Luminal breast cancer. REXO1 [16,17], RNA exonuclease homolog 1, is involved in cervical cancer progression [17], suggesting that ARID3A may influence genes related to cell proliferation, thereby driving tumor growth in breast cancer.

Functional enrichment analysis reveals that ARID3A is linked to immune-related pathways, such as "MHC protein binding [18]," "primary immunodeficiency [19]," and "interferon-gamma production [20]." These results suggest that ARID3A may modulate immune responses within the tumor microenvironment, potentially aiding cancer cells in evading immune surveillance.

PPI and molecular docking experiments highlight ARID3A's interaction with TP53, a key tumor suppressor. ARID3A binds to crucial residues within TP53's DBD [15]—including ARG-181, LYS-120, SER-261, and ASP-207—hinting at a possible mechanism for its oncogenic behavior. This interaction may inhibit TP53's ability to engage with target gene promoters, weakening its tumor-suppressive functions. Such competitive inhibition could impair TP53-regulated pathways involved in cell cycle arrest and apoptosis, thereby accelerating cancer progression.

In conclusion, ARID3A's elevated expression in breast cancer, particularly in aggressive subtypes, and its potential interference

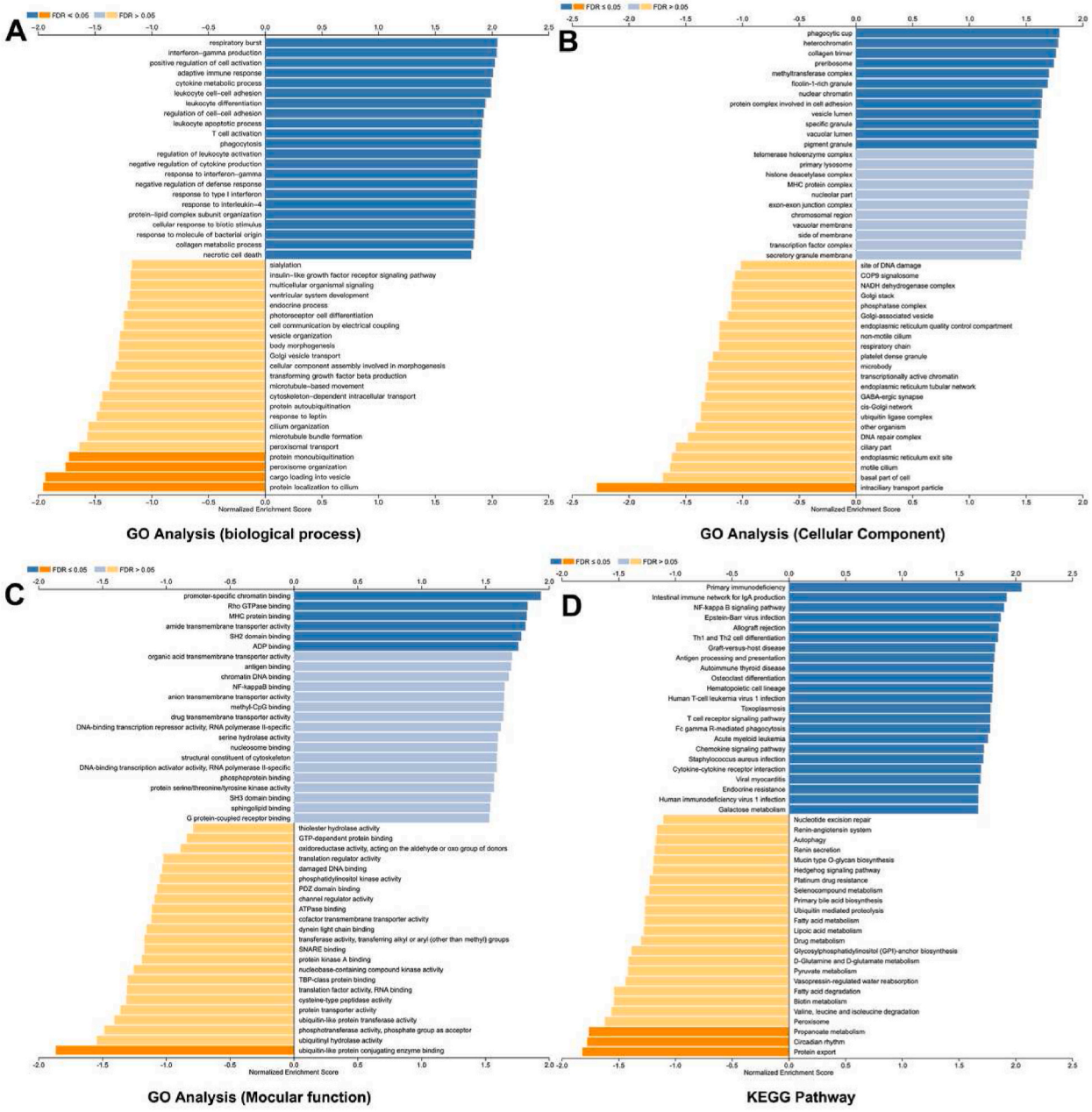

Fig. 6. GO and KEGG Enrichment Analysis of ARID3A Co-Expressed Genes. (A–C) Enrichment analysis of ARID3A co-expressed genes by LinkedOmics using GSEA, highlighting: (A) Biological processes, (B) Cellular components, and (C) Molecular functions. (D) KEGG pathway analysis of ARID3A co-expressed genes using GSEA, presented as a bar chart.

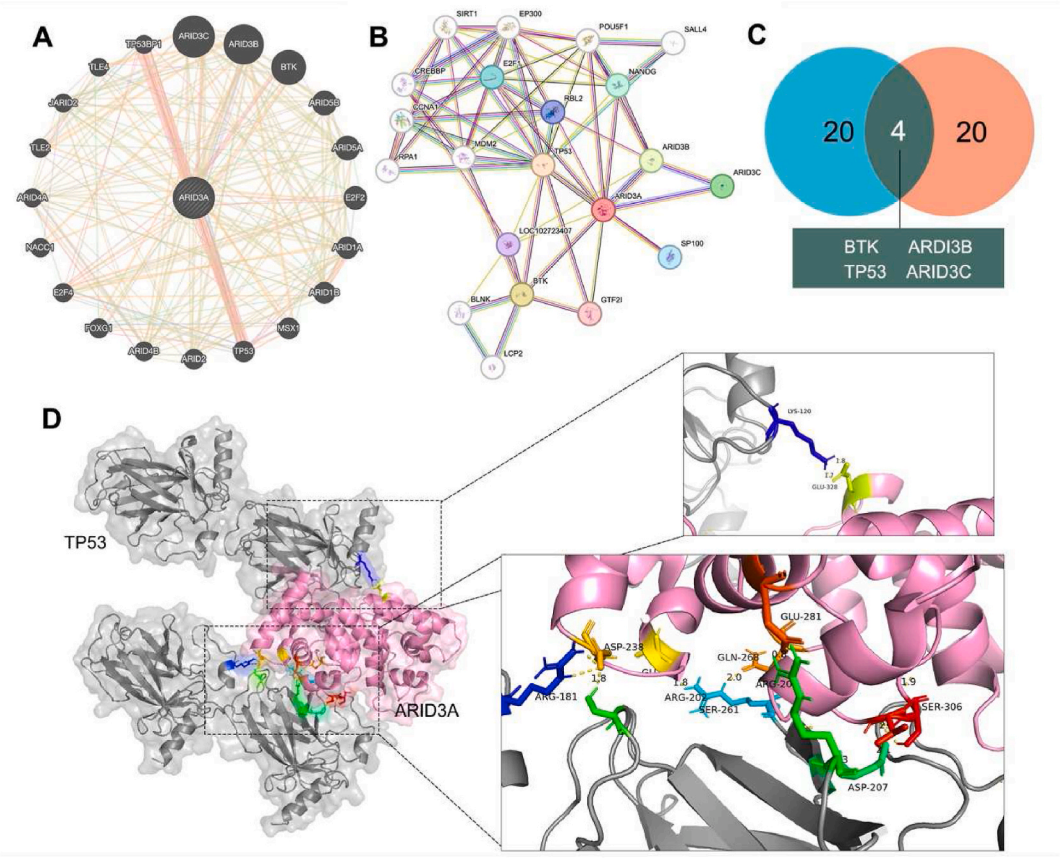

Fig. 7. Protein-Protein Interaction Network of ARID3A and Predicted Binding Mode of TP53. (A) Interaction network of ARID3A generated using GeneMANIA. (B) Interaction network of ARID3A constructed *via* the STRING database. (C) Venn diagram highlighting shared interacting proteins with ARID3A identified from both GeneMANIA and STRING databases. (D) Cartoon representation of the overall structure showing TP53 bound to ARID3A, with ARID3A and TP53 colored in pink and grey, respectively. (E) Detailed interaction between ARID3A and the TP53 hexamer. Key residues from both proteins are shown as sticks. Hydrogen bonds are depicted as yellow dashed lines, with distances (acceptor to donor heavy atom) labeled for clarity.

with TP53's tumor-suppressive activities underscore its role as a critical oncogenic factor. These findings identify ARID3A as a promising prognostic biomarker and potential therapeutic target, particularly in Luminal and HER2-positive breast cancers. Further research is necessary to clarify ARID3A's molecular mechanisms and confirm its impact on TP53 function in breast cancer.

CRediT authorship contribution statement

Wen-Run Cai: Writing – original draft, Resources, Funding acquisition, Conceptualization. **Xu-Gang Sun:** Investigation, Formal analysis. **Yue Yu:** Validation, Supervision. **Xin Wang:** Supervision. **Xu-Chen Cao:** Supervision. **Xiao-Feng Liu:** Writing – review & editing, Data curation.

Availability of data and materials

The data supporting the results of this study are accessible and obtained from publicly available resources.

Ethics approval and consent to participate

This study was conducted in accordance with the ethical guidelines of the Declaration of Helsinki. The use of human tissue samples was approved by the Ethics Committee or Institutional Review Board of Tianjin Medical University Cancer Institute & Hospital, under approval number [bc20240402]. Written informed consent was obtained from all patients prior to sample collection, ensuring their voluntary participation and understanding of the study's purpose. Patient confidentiality and data privacy were strictly maintained throughout the study.

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Declaration of competing interest

The authors declare no competing interests related to this manuscript.

Appendix A. Supplementary data

Supplementary data to this article can be found online at doi:mmcdoino

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