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ARTICLE

Recent Advances in Molecular Biomarkers of Triple-Negative Breast Cancer

Hongmei Dai Huizi Zhang Xinyu Fu Ran Li Xianguang Bai*

Pingdingshan University, Pingdingshan, Henan, 467000, China

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ABSTRACT

Triple-negative breast cancer (TNBC) is a highly heterogeneous and aggressive subtype of breast cancer characterized by the absence of estrogen receptor (ER), progesterone receptor (PR), and human epidermal growth factor receptor 2 (HER2) expression. Due to the lack of specific molecular targets, TNBC does not respond to conventional hormone or HER2-targeted therapies, posing a major challenge in breast cancer treatment. In recent years, molecular biomarkers have shown significant promise in the diagnosis, prognosis, and personalized treatment of TNBC. In-depth investigation of these biomarkers may lead to the development of more effective diagnostic and therapeutic strategies, ultimately improving patient outcomes. This review focuses on recent research progress concerning key molecular biomarkers in TNBC and explores their potential clinical applications, aiming to provide a theoretical basis for the advancement of precision therapy in TNBC.

Breast cancer is the most commonly diagnosed malignancy among women worldwide and remains the leading cause of cancer-related death in females [1]. It is estimated that over one million new cases of breast cancer are diagnosed globally each year, with at least 400,000 women dying annually from the disease, accounting for approximately 14% of all cancer-related deaths [2]. Approximately 11% of global breast cancer cases occur in China, and the incidence rate is rising rapidly [3]. By 2040, the global burden of breast cancer is projected to exceed 3 million new cases and 1 million deaths annually [4].

Triple-negative breast cancer (TNBC), characterized by the lack of estrogen receptor (ER), progesterone receptor (PR), and human epidermal growth factor receptor 2 (HER2) expression, represents the most aggressive subtype of breast cancer. It is associated with high recurrence rates, limited treatment options, and significant psychological and physical burdens on patients. Due to the absence of effective therapeutic targets, there is an urgent need for novel treatment strategies for TNBC[5].

1. Overview of Triple-Negative Breast Cancer

In clinical practice, breast cancer is typically classified into four molecular subtypes: Luminal A, Luminal B, HER2-enriched, and triple-negative. Among these, triple-negative breast cancer (TNBC) lacks the expression of estrogen receptor (ER), progesterone receptor (PR), and HER2, accounting for approximately 15%–20% of newly diagnosed breast cancer cases. Compared with hormone receptor-positive breast cancers, TNBC is more

**Corresponding Author:*
Xianguang Bai,
Associate Professor,
Email: 2644@pdsu.edu.cn

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frequently diagnosed in younger patients, shows a higher incidence of visceral metastases, an increased risk of early recurrence, and poorer prognosis. TNBC is widely recognized as the most aggressive and difficult-to-treat subtype of breast cancer[6].

1.1 Molecular Subtypes of TNBC

Based on the molecular characteristics and potential therapeutic targets in the Chinese population, TNBC can be further classified into four distinct molecular subtypes: immunomodulatory (IM), luminal androgen receptor (LAR), basal-like immunosuppressed (BLIS), and mesenchymal (MES) subtypes [7].

1.2 Prognosis and Therapeutic Challenges of TNBC

The clinical management of TNBC remains a significant challenge due to its unique biological features that contribute to poor prognosis and limited treatment efficacy. Compared with other breast cancer subtypes, TNBC exhibits more aggressive biological behavior, with approximately 60%–70% of recurrence events occurring within the first three years after surgery. Moreover, the 5-year overall survival rate is significantly lower than that of hormone receptor-positive breast cancers[8]. This disparity in outcomes is largely attributable to TNBC's triple-negative profile—the absence of ER, PR, and HER2 expression—which precludes the use of endocrine therapy and HER2-targeted treatments, thereby limiting therapeutic options.

Although standard chemotherapy regimens—such as anthracycline- and taxane-based neoadjuvant therapies—remain the cornerstone of TNBC treatment, only 15%–30% of patients achieve a pathological complete response (pCR)[8]. Moreover, the systemic adverse effects and the development of chemoresistance in some patients continue to pose significant challenges. With advances in modern medicine and a deeper understanding of TNBC, the identification and validation of additional molecular biomarkers have become a promising avenue for precision therapy, offering a new direction for future clinical applications.

2. Molecular Biomarkers Relevant to TNBC Diagnosis and Treatment

2.1 p53

p53 is a polypeptide-encoding protein that plays a crucial role in maintaining normal cell growth and development. When mutations occur in the *TP53* gene, oncogenic pathways within breast cancer cells can become hyperactivated, ultimately leading to tumorigenesis. As a key tumor suppressor gene, *TP53* is critically involved in the pathogenesis and progression of various malignancies. Mutations in p53 often result in the loss of its canonical functions, including cell cycle arrest, apoptosis induction, and DNA repair regulation. In turn, mutant p53 can exert dominant-negative effects or acquire gain-of-function properties that promote genomic instability and cooperate with oncogenes to drive malignant transformation.

Clinical studies have shown that p53 status is closely associated with both prognosis and treatment response in breast cancer, particularly in TNBC. Numerous studies have demonstrated the relevance of p53 in predicting tumor outcomes and chemotherapy resistance [9–13]. Further investigation into the prognostic significance and therapeutic potential of p53 in TNBC may help clarify its value as a clinical biomarker and guide more personalized treatment strategies.

2.2 Ki-67

Ki-67 is a nuclear antigen expressed in proliferating cells and is tightly correlated with the mitotic activity of malignant tumors. Elevated Ki-67 expression indicates rapid proliferation of breast cancer cells and serves as a valuable reference for molecular subtyping. Ki-67 has shown significant clinical relevance in the assessment of TNBC. In a study involving 586 breast cancer patients, the Ki-67 high expression rate in the TNBC group (67.26%) was markedly higher than in non-TNBC subtypes (42.48%), reflecting the higher proliferative potential of TNBC cells [14].

Survival analysis further revealed that patients with high Ki-67 expression (≥30%) had a 5-year disease-free survival (DFS) rate of 74.3%, approximately 11 percentage points lower than those with low Ki-67 expression (85%), indicating its utility in prognostic stratification. Ki-67 can be integrated with TNM staging and molecular subtyping to construct risk models that inform the intensity of adjuvant therapy. Additionally, a ≥50% reduction in Ki-67 expression following neoadjuvant chemotherapy suggests chemosensitivity and is positively associated with pathologic complete response (pCR). Dynamic changes in Ki-67 expression within circulating tumor cells (CTCs) may also reflect real-time disease progression[15].

However, the clinical utility of Ki-67 remains limited by the lack of standardized detection protocols, including the definition of cutoff values and the correction for intratumoral heterogeneity. Addressing these challenges will further enhance the role of Ki-67 as a decision-

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support biomarker in precision TNBC therapy.

2.3 BIRC5 Gene

The BIRC5 gene, also known as *Survivin*, is a key member of the inhibitor of apoptosis (IAP) protein family and plays a pivotal role in the development, progression, and therapeutic resistance of triple-negative breast cancer (TNBC). The protein encoded by BIRC5 has dual functions: it inhibits apoptosis by blocking caspase pathways and regulates mitotic progression by stabilizing microtubule structures. Abnormally high expression of BIRC5 is strongly associated with the aggressive phenotype of TNBC, as well as with chemoresistance and poor prognosis.

Studies have shown that BIRC5 expression is approximately 2.3 times higher in TNBC tissues compared to other breast cancer subtypes. Its expression is positively correlated with tumor grade and lymph node metastasis, making it a central hub gene with both prognostic and therapeutic relevance identified through differential gene expression analysis[16]. The underlying molecular mechanisms involve complex cross-regulation across multiple pathways. Upstream transcription factors such as STAT3 and NF-κB can activate the BIRC5 promoter, while cell cycle regulators like Aurora kinases and Cyclin B1 enhance BIRC5 stability through phosphorylation, collectively forming a pro-oncogenic survival network[17].

Currently, the development of targeted therapeutics against BIRC5 has become a research hotspot, with the potential to offer new avenues for overcoming treatment resistance in TNBC.

2.4 PD-1 and PD-L1

The PD-1/PD-L1 immune checkpoint pathway plays a central role in immune evasion mechanisms of triple-negative breast cancer (TNBC). Programmed death-ligand 1 (PD-L1), expressed on the surface of tumor cells, binds to its receptor PD-1 on T cells, thereby inhibiting T-cell proliferation, cytotoxic activity, and interferon-γ secretion, ultimately establishing an immunosuppressive tumor microenvironment. Studies have shown that PD-L1 is highly expressed in approximately 40–60% of TNBC cases, and its overexpression is significantly associated with enhanced tumor invasiveness, lymphovascular invasion, and shorter overall survival[18]. This immunosuppressive profile identifies PD-L1 not only as a biomarker for poor prognosis in TNBC, but also as a promising target for immunotherapy.

Atezolizumab, a PD-L1 inhibitor, functions by blocking the interaction between PD-L1 and PD-1/CD80, thereby restoring T-cell-mediated antitumor immunity. The pivotal phase III IMpassion130 clinical trial enrolled 902 patients with metastatic TNBC and demonstrated that in the PD-L1–positive subgroup (immune cells ≥1%), the combination of atezolizumab and nab-paclitaxel significantly improved median progression-free survival (PFS) by 2.5 months (7.5 vs. 5.0 months), increased overall survival (OS) by 7 months (25 vs. 18 months), and elevated the objective response rate by 14% compared to chemotherapy alone [19]. These findings support the use of atezolizumab in combination with chemotherapy as a means to significantly prolong survival in PD-L1–positive TNBC patients.

2.5 miRNA

MicroRNAs (miRNAs), a class of non-coding RNAs approximately 22 nucleotides in length, play vital roles in regulating cell proliferation, apoptosis, and metabolism, thus participating in cancer initiation and progression. Aberrant expression of specific miRNAs in TNBC has been closely linked to malignant phenotypes. Tumor-suppressive miRNAs such as miRNA-623 and miRNA-199a-5p are frequently downregulated, while oncogenic miRNAs including miRNA-221, miRNA-135b, and miRNA-93 are significantly upregulated.

Notably, miRNA-199a-5p inhibits tumor cell invasion, metastasis, and angiogenesis by targeting key epithelial–mesenchymal transition (EMT)-related genes such as CDH1, ZEB1, and TWIST [20]. Conversely, miRNA-221 enhances metastatic potential by promoting extracellular matrix degradation [21]. High expression of miRNA-135b is associated with increased invasiveness and poor prognosis in TNBC and contributes to tumor progression via modulation of the TGF-β and WNT signaling pathways [22]. Furthermore, miRNA-93 is significantly upregulated in TNBC and demonstrates high sensitivity as a diagnostic biomarker[23], whereas downregulation of miRNA-623 is associated with shorter survival, and its mimics have shown the ability to inhibit tumor cell proliferation and migration [24].

Despite the promising diagnostic and therapeutic potential of miRNA biomarkers in TNBC, several technical challenges remain, including tissue heterogeneity and the instability of circulating miRNAs. Future research should integrate multi-omics data and artificial intelligence approaches to establish refined molecular classification systems and accelerate the clinical translation of miRNA-based diagnostics and therapeutics.

2.6 EGFR

Epidermal growth factor receptor (EGFR), a critical member of the transmembrane receptor tyrosine

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kinase family, is encoded by a gene located on human chromosome 7p22–q21.1[25]. In malignancies, EGFR can be aberrantly activated through multiple mechanisms, including ligand overexpression, gene amplification, transcriptional upregulation, and somatic mutations, ultimately driving uncontrolled tumor cell proliferation[26]. In TNBC, EGFR overexpression is particularly prominent. Clinical-pathological data indicate that approximately 50–70% of TNBC cases exhibit EGFR protein overexpression, which is significantly associated with greater tumor aggressiveness, poor prognosis, and resistance to conventional chemotherapy[27].

Preclinical studies have confirmed that afatinib, a second-generation irreversible EGFR tyrosine kinase inhibitor, exerts potent antiproliferative effects across 14 immunogenic TNBC cell models, with the basal-like subtype demonstrating particularly enhanced sensitivity. Mechanistic investigations further revealed that the combination of afatinib with PI3K/AKT/mTOR pathway inhibitors can produce synergistic growth inhibition by blocking compensatory signaling activation. These findings suggest a promising therapeutic strategy for overcoming drug resistance and improving outcomes in EGFR-overexpressing TNBC.

2.7 CA15-3

Cancer antigen 15-3 (CA15-3) is frequently overexpressed in breast cancer and is widely used as a molecular marker in clinical diagnosis and treatment. CA15-3 is a variant glycoprotein found on the epithelial cells of the mammary gland. During malignant transformation of breast tissue, tumor cells progressively infiltrate the bloodstream, leading to elevated serum CA15-3 levels. The correlation between CA15-3 and breast cancer progression is well established, and the marker demonstrates relatively high specificity[27]. Disruption of normal cellular architecture compromises intercellular adhesion, which enhances the risk of tumor invasion and metastasis. Concurrently, serum levels of CA15-3 and CA125 often increase abnormally [28].

A cohort study revealed that patients with triple-negative breast cancer (TNBC) exhibited significantly higher serum CA15-3 levels (mean: 22.95 U/mL) compared to non-TNBC patients (mean: 15.32 U/mL), along with a higher positive rate. These findings suggest that CA15-3 may serve as a supportive marker in the differential diagnosis of TNBC [29]. Although the sensitivity and specificity of CA15-3 alone are limited, combined biomarker detection strategies can enhance diagnostic performance. Moreover, existing evidence indicates that dynamic changes in CA15-3 levels may reflect treatment response in TNBC. Nonetheless, its clinical utility warrants validation through large-scale prospective studies [30].

2.8 Cath-D and c-erbB-2

Cathepsin D (Cath-D) is a lysosomal aspartic protease involved in a variety of biological processes, including extracellular matrix degradation, cell migration, and apoptosis. While Cath-D is expressed across various subtypes of breast cancer, its overexpression in TNBC is strongly associated with enhanced tumor aggressiveness and increased metastatic potential.

A clinical study reported significantly higher expression levels of Cath-D and the proto-oncogene c-erbB-2 in TNBC tissues compared to fibroadenoma controls. In the TNBC group, the positive expression rates of Cath-D and c-erbB-2 were 60.47% and 65.12%, respectively. Notably, in patients with lymph node metastasis, the positive rates increased dramatically to 92.31% for Cath-D and 89.74% for c-erbB-2, significantly higher than in non-metastatic patients [31]. These findings suggest that the co-expression of Cath-D and c-erbB-2 could serve as important biological indicators for assessing the aggressiveness and metastatic potential of TNBC, with potential clinical relevance for prognosis evaluation.

Combined detection of Cath-D and c-erbB-2 may aid in identifying TNBC patients at high risk for metastasis and poor outcomes. Furthermore, Cath-D may represent a novel therapeutic target for anti-metastatic strategies, while reclassification efforts for c-erbB-2–low-expressing TNBC subtypes could open new avenues for targeted therapy development.

2.9 CXCL12 and CXCR4

CXCL12 (also known as stromal cell-derived factor-1, SDF-1) and its receptor CXCR4 form a key chemokine axis that plays a crucial role in tumor cell migration, angiogenesis, and immune evasion. In TNBC, overexpression of both CXCL12 and CXCR4 has been significantly associated with lymph node metastasis, suggesting their involvement in directing tumor cells toward organs with high CXCL12 expression [32].

Therapeutic strategies targeting the CXCL12/CXCR4 signaling axis—such as CXCR4 antagonists—have shown potential anti-metastatic effects in preclinical and early-phase clinical studies. This chemokine pair holds promise as a biomarker for predicting tumor metastasis and therapeutic responsiveness, and warrants further investigation for its clinical applicability.

Furthermore, ongoing studies are investigating the

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potential of GABRP, BRG1, and other candidates as molecular markers for triple-negative breast cancer[33].

3. Conclusion

Triple-negative breast cancer (TNBC) is a highly heterogeneous and aggressive subtype of breast cancer, characterized by the absence of estrogen receptor (ER), progesterone receptor (PR), and human epidermal growth factor receptor 2 (HER2) expression. This molecular profile results in limited treatment options and generally poor prognosis for affected patients. With the continuous advancement of molecular biology techniques, researchers have gained deeper insights into the molecular mechanisms underlying TNBC, leading to the identification and validation of numerous molecular biomarkers involved in tumorigenesis, disease progression, prognosis evaluation, and therapeutic responsiveness.

This review has summarized several representative molecular markers that have emerged in TNBC research in recent years, including P53, Ki67, BIRC5, PD-1/PD-L1, miRNAs, EGFR, CA15-3, cathepsin D (Cath-D), c-erbB-2, and the CXCL12/CXCR4 axis. These biomarkers are instrumental not only in revealing the molecular heterogeneity of TNBC but also in providing a theoretical foundation and potential targets for subtype classification, therapeutic efficacy prediction, treatment response monitoring, and the development of novel targeted agents. Notably, biomarkers related to immune checkpoint pathways, non-coding RNA networks, and tumor microenvironment regulation are steadily progressing from laboratory research to clinical application, serving as important pillars of precision medicine.

Nonetheless, the clinical application of molecular biomarkers in TNBC still faces multiple challenges, including a lack of standardized detection protocols, insufficient sensitivity and specificity, expression heterogeneity, and limitations in sample acquisition. Future research should prioritize the integration of multi-omics data, robust clinical validation, and personalized therapeutic strategies. In addition, leveraging artificial intelligence and big data analytics may enable the development of more precise and dynamic diagnostic and therapeutic evaluation systems.

By continuing to advance our understanding of the molecular landscape of TNBC and identifying clinically actionable biomarkers, it is expected that early diagnosis, stratified treatment, and improved long-term prognosis will become increasingly achievable, ultimately delivering tangible benefits to TNBC patients.

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