MYC in Breast Cancer: A Genomic and Transcriptomic Perspective

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Breast cancer is one of the most prevalent cancers among women globally and is characterized by distinct molecular subtypes that influence treatment response and prognosis. Among oncogenes, Myelocytomatosis (MYC) has been recognized as a key regulator of cell cycle progression, growth, metabolism, and apoptosis, yet its clinical relevance in breast cancer remains incompletely defined. In this study, a comprehensive bioinformatics analysis of The Cancer Genome Atlas (TCGA) breast cancer cohort was conducted using cBioPortal and the University of Alabama CANcer (UALCAN) resource to evaluate MYC amplification, mRNA expression, and associated clinical outcomes. MYC was frequently amplified, particularly in aggressive subtypes such as triple-negative breast cancer (TNBC), but transcript-level expression was paradoxically reduced in tumors compared to normal tissues. Amplification was associated with higher expression relative to non-amplified tumors, though both remained below normal levels. Importantly, MYC alterations correlated with reduced progression-free survival. Coexpression analysis identified MYC-associated genes including TAF4B and TWNK, implicating transcriptional regulation and mitochondrial function as part of MYC-driven networks. These findings demonstrate that MYC is frequently altered in breast cancer and suggest that its regulation is more complex than gene amplification alone. The results highlight MYC's prognostic relevance, particularly in TNBC, and support the need for further research to clarify its biological mechanisms and evaluate its potential clinical applications.

Keywords: Breast cancer, Myelocytomatosis (MYC) oncogene, gene amplification, Cancer Genome Atlas (TCGA), bioinformatics, cancer subtypes, progression-free survival, TAF4B, cBioPortal, University of ALabama CANcer (UALCAN)

Introduction

Cancer is a genetic disease in which cells grow and spread uncontrollably throughout the body. As a deadly genetic disease, cancer has been one of the leading causes of death. About 10 million people die from cancer every year. This means that about one in six deaths worldwide is caused by cancer[1]. One of the most common cancers is breast cancer, which occurs primarily among women, though it can also occur in men. Breast cancer is known to originate from different parts of the breast, including ductal carcinoma, which carries milk to the nipple, or lobular carcinoma, the glands that produce milk. Even with today's knowledge about cancer and its treatments, the number of breast cancer cases continues to rise in women today. Poor eating habits, lack of exercise, drugs, alcohol, smoking, and various types of infections caused by poor hygiene are all potential contributors to breast cancer[2,3].

Breast cancer is a heterogeneous disease that has been classified into distinct molecular subtypes based on gene expression profiling. These subtypes—Luminal A, Luminal B, Human epidermal growth factor receptor 2 (HER2)-positive, triple-negative, and basal-like—exhibit unique biological behaviors and guide treatment strategies and prognosis[4] Luminal A tumors are typically ER+/PR+, low in HER2 expression and Ki-67, and associated with favorable outcomes and responsiveness to hormone therapy[5]. Luminal B tumors, also ER+, show higher proliferation (Ki-67) and/or HER2 expression, making them more aggressive; treatment often includes both hormone therapy and chemotherapy. HER2-positive cancers overexpress the HER2 gene and are usually ER-, historically linked to poor outcomes but now effectively managed with HER2-targeted therapies such as trastuzumab[6]. Triple-negative breast cancer (TNBC) lacks ER, PR, and HER2 expression. It is aggressive, with limited targeted therapy options, and is primarily treated with chemotherapy, though immunotherapy and BRCA-targeted agents are emerging[7]. Basal-like tumors, which often overlap with TNBC, resemble the gene expression profile of basal breast cells and are similarly aggressive and chemotherapy-dependent. Research continues into more targeted treatment options for this subtype[8]. A less common group, normal-like breast cancers, share gene profiles with healthy breast tissue and generally have a better prognosis, treated similarly to Luminal subtypes depending on individual tumor features[9]. This diversity in tumor biology makes breast cancer a challenging disease to diagnose, treat, and manage effectively. Advances in understanding the underlying molecular mechanisms are essential for improving both prognosis and therapeutic strategies[10].

Genetic elements known as genes are Deoxyribonucleic Acid (DNA) sequences that provide the instructions to produce proteins. Alterations or mutations in these genetic components can contribute to the emergence and progression of cancer[11]. Cancer genes can be classified into two broad categories. The first category comprises oncogenes, which are normal genes that have the potential to become cancer-promoting genes when mutated. Under normal conditions, these proto-oncogenes regulate cell growth and DNA repair processes. However, when they become mutated, they can transform into oncogenes, leading to uncontrolled cell growth and division, which is characteristic of cancer. The second category consists of tumor suppressor genes, which play a crucial role in preventing cancer development. These genes typically inhibit cell division, repair DNA errors, or induce cell death, thereby maintaining cellular homeostasis. When these genes are mutated or inactivated, their loss of function contributes to cancer development[12].

Among the various oncogenes implicated in breast cancer, the MYC gene, also known as Myelocytomatosis oncogene, has emerged as a pivotal player, exerting its influence on numerous cellular processes, including cell cycle progression, cell growth, apoptosis, and DNA replication[13]. Located on chromosome 8q24 in humans, MYC is highly conserved across species and has been shown to have fundamental roles in both normal cellular functions and the development of disease[14]. Under normal physiological conditions, the MYC protein plays a critical role in promoting the transition from the G1 phase to the S phase of the cell cycle, thus driving cell division[15]. Additionally, it regulates various aspects of cellular metabolism, such as protein synthesis and glucose metabolism, facilitating cell growth and proliferation[16]. MYC also serves as a gatekeeper for cellular health by inducing apoptosis (programmed cell death) in response to stress or DNA damage, helping maintain cellular homeostasis[17]. However, when MYC becomes overexpressed, mutated, or amplified, it can acquire oncogenic properties, contributing to the initiation and progression of a variety of cancers[18]. In such cases, MYC promotes unchecked cell proliferation and survival, often by disrupting normal regulatory mechanisms[19,20]. MYC amplification is commonly observed in cancers such as Burkitt lymphoma, small cell lung cancer, and breast cancer, where its overexpression correlates with tumor aggressiveness and poor prognosis[21]. Additionally, chromosomal translocations involving MYC, such as those observed in Burkitt lymphoma, lead to aberrant expression and contribute to cancer pathogenesis[22].

Despite its well-established importance, the precise mechanisms by which MYC contributes to the development and behavior of different breast cancer subtypes remain incompletely understood. While MYC-driven alterations in metabolism and apoptosis have been widely documented[23,24], their broader implications, including genomic instability, immune evasion, and treatment response, are less clear. Amplification occurs frequently, but the relationships between MYC status and clinicopathological features such as age, tumor stage, and molecular subtype are not fully resolved. Moreover, the prognostic value of MYC alterations and expression for survival outcomes remains uncertain in large patient cohorts.

This study tests the hypothesis that MYC amplification and expression patterns are not only common but also hold clinical significance in breast cancer. Specifically, the hypothesis proposes that MYC status may influence progression-free survival, vary by demographic and molecular subgroups, and form part of distinct coexpression networks that reveal functional dependencies in tumor biology. To address this hypothesis, a multi-step bioinformatics analysis of The Cancer Genome Atlas (TCGA) breast cancer data was performed using cBioPortal and UALCAN. The objectives of this study are to:

- Characterize the frequency and type of MYC alterations in breast cancer.
- Compare MYC mRNA expression in tumor versus normal tissues and across clinical subgroups.
- Assess whether MYC amplification and expression are associated with patient survival outcomes.
- Identify coexpressed genes and enriched biological pathways linked to MYC activity.

Through these objectives, this study aims to clarify the prognostic relevance of MYC in breast cancer and provide a foundation for future research into its functional and therapeutic implications.

Results

First, data from the Cancer Genome Atlas (TCGA) PanCancer Atlas studies[25] encompassing various cancer types, were analyzed using cBioPortal. A total of 10,967 samples from 32 studies were included, and these data were filtered to identify samples with mutations in the MYC gene. Figures 1a and 1b present a list of cancer studies by cancer type, showing the presence of MYC variants in terms of absolute count and alteration frequency, respectively. The results indicate that ovarian cancer has the highest proportion of MYC variants, followed by breast and uterine cancers, placing these tumor types among those most associated with MYC-driven biology (Figure 1a). Specifically, 15.0% of 1,084 breast cancer cases (163 patients) contained MYC alterations, with amplification representing 98.2% of these events and only a small fraction classified as mutations or structural changes. In Figure 1b, variant

types are color-coded: green represents mutations, while red indicates amplifications. These results highlight amplification as the predominant alteration and emphasize MYC's role as a recurrent genomic event in breast cancer.

Figure 2 shows the demographic and clinical characteristics of breast cancer patients with MYC alterations in the TCGA PanCancer Atlas cohort (163 cases). Among these patients, 98.1% were female and 1.9% were male. Invasive ductal carcinoma was the predominant histological type (88.2%), followed by invasive lobular carcinoma (5.6%). The median age at diagnosis was 59 years. With respect to race, most patients were White (68.7%), followed by Black or African American patients (20.5%). According to the American Joint Committee on Cancer (AJCC) staging system, T2 tumors—measuring more than 2 cm but not more than 5 cm—were the most common, seen in 62.3% of patients. This was followed by T1C tumors—measuring more than 1 cm but not more than 2 cm—in 19.3% of cases. The overall survival rate in this group was 83.2%. These demographic and clinical features closely parallel the broader TCGA breast cancer cohort, indicating that MYC alterations are broadly distributed across patient subgroups rather than confined to a specific demographic or clinical profile.

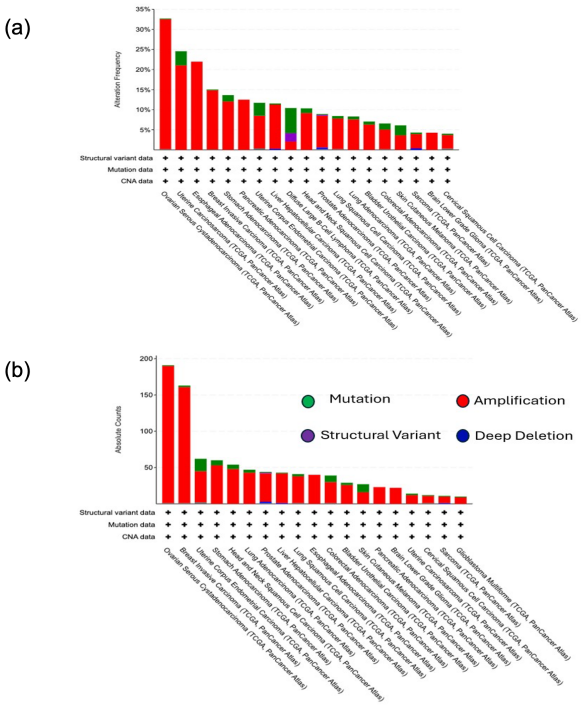

Fig. 1 MYC alterations across cancer types in the TCGA PanCancer Atlas.

Next, the association between MYC expression and clinicopathological characteristics was examined. Using the University of Alabama at Birmingham Cancer Data Analysis Portal (UALCAN)[26,27], TCGA Breast Invasive Carcinoma (BRCA) data were analyzed to plot MYC expression levels based on sample type, gender, menopause status, and patient age, as shown in Figure 3. Figure 3a shows MYC expression according to sample type, indicating that breast cancer patients exhibit significantly lower MYC expression levels compared to normal samples, consistent with the amplification-expression paradox. The median expression values for normal and BRCA samples were 138.7 and 60.3 transcripts per million (TPM), respectively. In Figure 3b, MYC expression levels between genders are compared to those in normal tissue. In male patients, MYC expression was reduced relative to adjacent normal male breast tissue (67.0 vs. normal), although overall levels were similar between male and female patients (male = 67.0, female = 60.4 TPM). A significant difference was observed between normal tissue and male breast cancer samples, whereas MYC expression levels between male and female patients showed only marginal differences. In Figure 3c, MYC expression levels between menopausal groups are compared. Premenopausal (69.3 TPM) and perimenopausal (68.1 TPM) patients displayed higher MYC expression than postmenopausal patients (55.8 TPM). While this could reflect hormonal or endocrine-related influences on MYC regulation, menopausal status is strongly correlated with age and may not represent an independent factor. Figure 3d shows the relative expression of MYC in normal samples and in patients across different age groups. The results indicate that MYC expression tends to decrease with increasing patient age. The median MYC expression values for BRCA samples from patients aged 21–40 years, 41–60 years, 61–80 years, and 81–100 years were 67.9, 65.0, 56.1, and 37.9 transcripts per million, respectively, suggesting attenuation of MYC activity with increasing age. Additionally, MYC expression levels were compared between normal samples and BRCA samples from different racial groups. The results suggested no statistically significant differences in MYC expression levels among normal, Caucasian, African American, and Asian groups (data not shown). Together, these findings indicate that MYC expression is influenced by both hormonal and age-related factors, highlighting potential interactions between MYC activity, endocrine signaling, and tumor progression.

Figure 4 examined MYC expression across tumor stage, molecular subtype, and TP53 mutation status, and evaluated their impact on survival outcomes. Expression stratified by stage showed consistently lower MYC transcript levels in primary tumors compared to adjacent normal breast tissue, with stage 2 cancers displaying the lowest expression and stage 4 cancers showing relatively higher levels (Figure 4a). This pattern suggests that MYC expression may be dynamically reg-

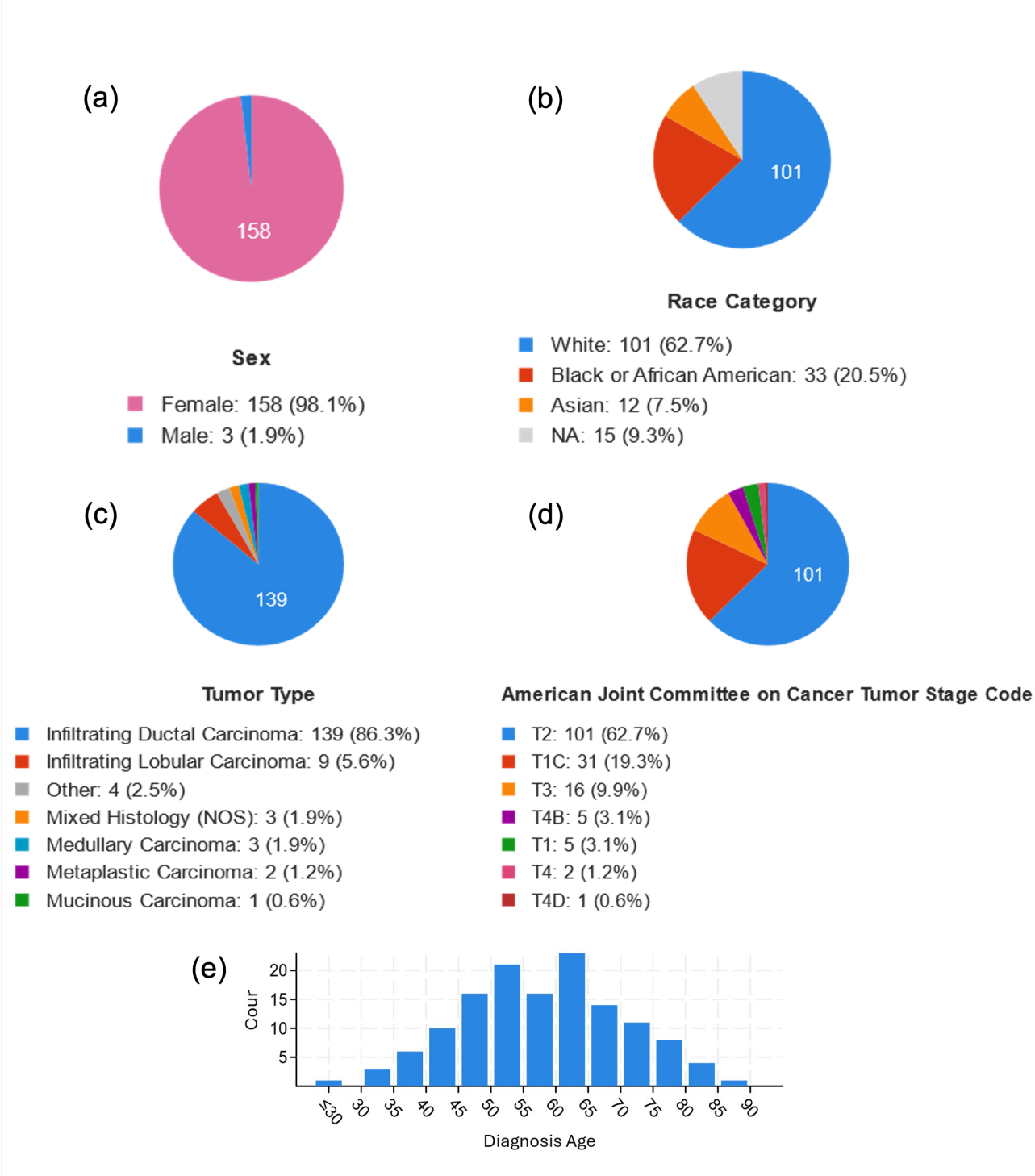

Fig. 2 Demographic and clinical characteristics of breast cancer patients with MYC gene alterations in the TCGA PanCancer Atlas cohort. Alterations were observed across (a) sex, (b) race, (c) tumor histology, (d) tumor stage, and (e) age at diagnosis, indicating that MYC alterations are broadly distributed across diverse patient groups without strong demographic or clinical restriction.

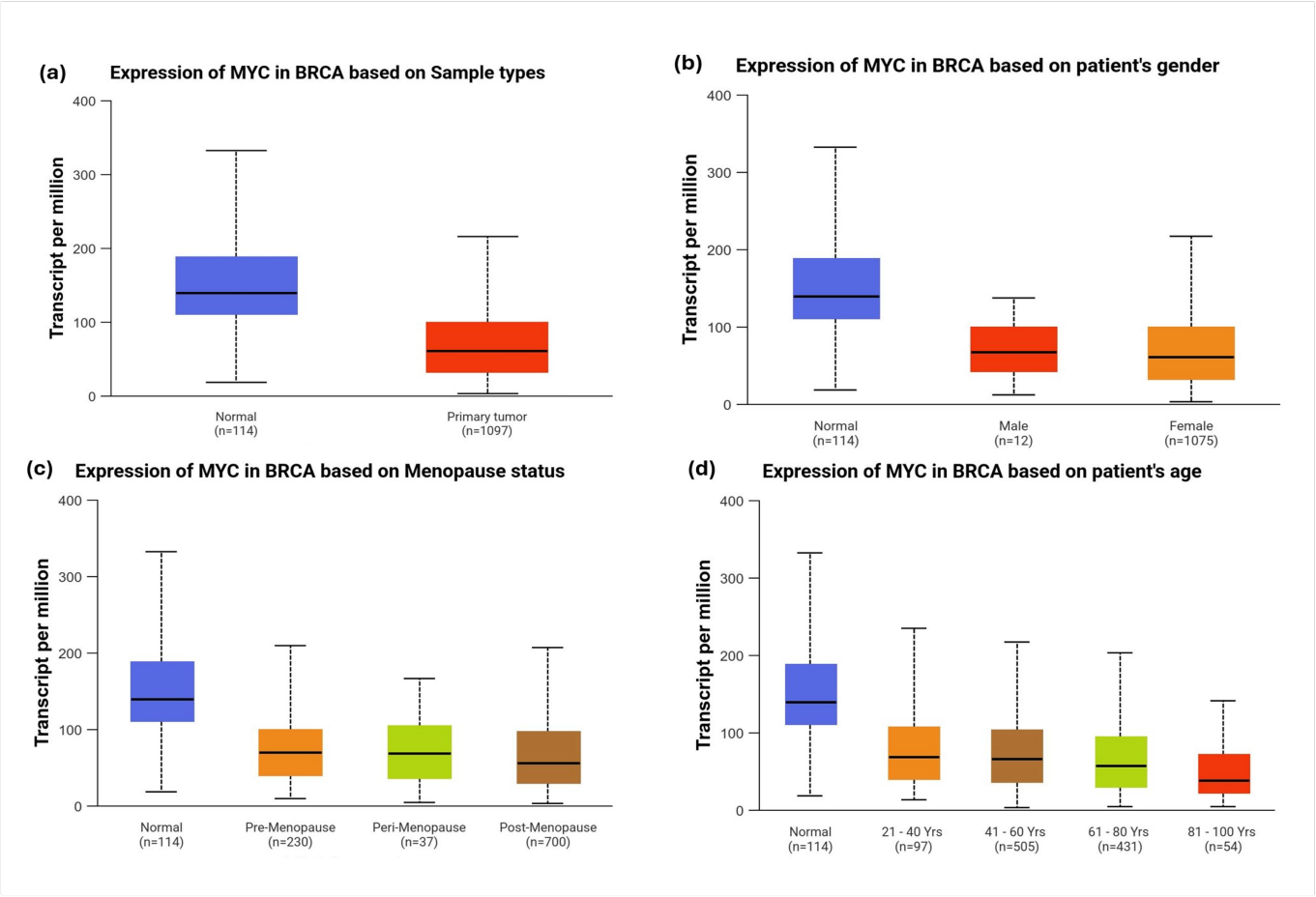

Fig. 3 Box-whisker plots showing MYC expression in breast invasive carcinoma (BRCA) samples from the TCGA cohort using UALCAN. Expression levels are grouped by (a) adjacent normal tissue vs. primary tumors, (b) patient gender, (c) menopausal status, and (d) age group.

ulated during disease progression, potentially suppressed in intermediate stages before reactivation in advanced disease. Subtype analysis confirmed reduced MYC expression in Luminal, HER2-positive, and triple-negative breast cancers compared to adjacent normal tissue (Figure 4b). Among these, TNBC showed the lowest expression despite frequent amplification, reinforcing the amplification-expression paradox and underscoring the complexity of MYC regulation in this aggressive subtype. Analysis by TP53 status revealed that TP53-nonmutant tumors had the lowest MYC expression, although both mutant and nonmutant groups exhibited significantly reduced levels compared to adjacent normal tissue (Figure 4c). Survival analyses supported the prognostic significance of MYC. Kaplan-Meier curves[28] from UALCAN demonstrated that patients in the MYC-high group (upper quartile of expression) had significantly shorter progression-free survival than those in the low/medium groups (log-rank $p < 0.05$; Figure 4d). Complementary analysis using cBioPortal indicated that MYC alterations were associated with worse outcomes, with progression-free survival approximately 14% higher in the unaltered group compared to the altered group (figure not shown). Together, these results demonstrate that MYC expression varies by stage, subtype, and TP53 status, and that both transcript-level expression and genomic alterations have prognostic relevance. The paradox of reduced transcript levels despite amplification, particularly in TNBC, points toward additional regulatory mechanisms that warrant further study.

Table 1 shows the top 10 genes coexpressed with MYC in the TCGA breast invasive carcinoma (BRCA) cohort, as identified using cBioPortal with Spearman's rank correlation. The strongest associations were observed with TAF4B ($r = 0.58$, $q < 0.001$) and TWNK ($r = 0.56$, $q < 0.001$). TAF4B encodes a transcription factor involved in RNA polymerase II initiation, while TWNK encodes a mitochondrial DNA helicase. These correlations suggest that MYC activity may be coordinated with both transcriptional control and mitochondrial regulatory

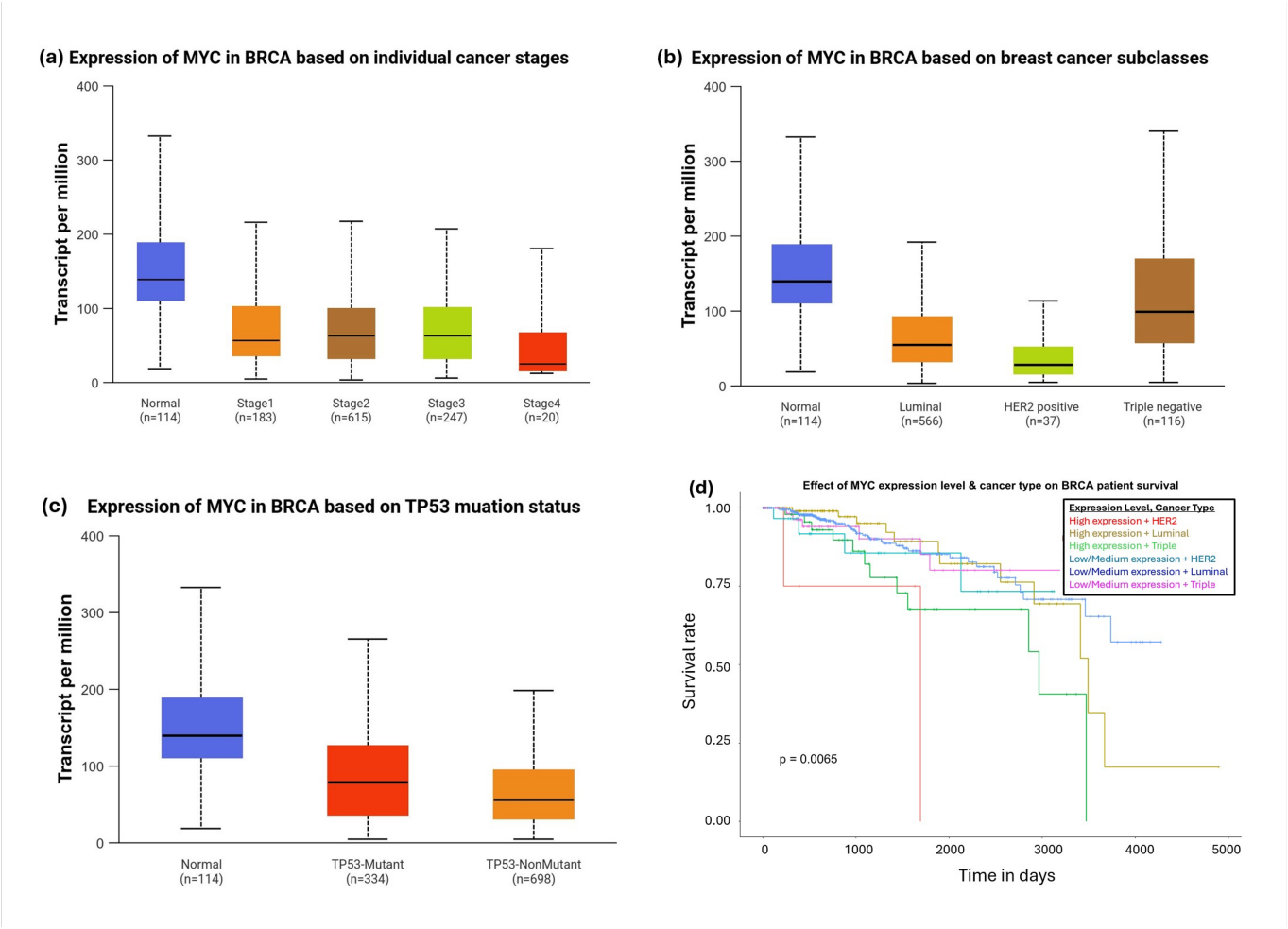

Fig. 4 Box-whisker plots showing MYC expression in breast invasive carcinoma (BRCA) subgroups from the TCGA cohort using UALCAN. Expression levels are stratified by (a) cancer stage, (b) molecular subtype, and (c) TP53 mutation status. (d) Kaplan-Meier survival curves illustrate the effect of MYC expression level on progression-free survival in BRCA patients.

pathways. Other highly correlated genes included FAM216A ($r = 0.53$), POLR1E ($r = 0.49$), and LDHB ($r = 0.49$), pointing to potential links between MYC activity, transcriptional regulation, and cellular metabolism. These associations are consistent with prior studies that identify MYC as a central regulator of transcriptional control and metabolic function[29,30]. Together with the survival analyses, these results support the view that MYC contributes to breast cancer progression not only through genomic alterations but also through coordinated regulation of broader transcriptional and metabolic networks.

Discussions

This study provides a multi-dimensional analysis of MYC in breast cancer, integrating gene alterations, transcript expression, clinical correlations, and coexpression networks using TCGA data. The results show that MYC is frequently amplified, particularly in aggressive subtypes; transcript-level expression is paradoxically reduced in tumors compared to adjacent normal tissue; amplification is associated with higher expression relative to non-amplified tumors but remains lower than normal controls; and MYC alterations correlate with reduced progression-free survival. Coexpression analyses further implicate MYC in transcriptional and mitochondrial regulatory pathways.

A key paradox observed was the frequent amplification of MYC contrasted with reduced transcript expression in tumors. While amplification modestly increased expression compared with non-amplified tumors, both groups still exhibited lower levels than adjacent normal tissue. Several mechanisms may account for this discrepancy, including enhanced mRNA degradation, restricted chromatin accessibility,

Table 1 Top 10 genes coexpressed with MYC in the TCGA breast invasive carcinoma (BRCA) cohort. Correlations were calculated using Spearman's rank method in cBioPortal.

Correlated Gene	Spearman's correlation	p-Value	q-Value
TAF4B	0.58	2.01E-98	4.03E-94
TWNK	0.561	1.29E-90	1.29E-86
FAM216A	0.529	4.93E-79	3.29E-75
POLR1E	0.486	2.90E-65	1.46E-61
LDHB	0.486	4.33E-65	1.74E-61
NOB1	0.473	2.42E-61	8.09E-58
ADCY3	0.465	4.40E-59	1.26E-55
SIK1	0.461	5.19E-58	1.30E-54
ADAT2	0.456	9.18E-57	2.05E-53
YBX3	0.454	4.72E-56	9.47E-53

and microRNA-mediated repression. This pattern is consistent with prior reports indicating that amplification does not necessarily translate into overexpression[31,32]. In breast cancer, for example, MYC amplification has been shown not to consistently correlate with elevated transcript levels, and only high MYC mRNA expression - not DNA amplification - was associated with poor survival in triple-negative breast cancer. More broadly, the longstanding assumption that gene amplification directly causes overexpression has been challenged as a reasonable but unproven notion[31]. Supporting this, Reverse Phase Protein Array (RPPA) data from TCGA, as reported by Katsuta et al. (2020), further demonstrated that MYC protein abundance is not reliably linked to either copy number or transcript levels, underscoring the importance of post-transcriptional and post-translational regulation. Together, these findings highlight that amplification alone is not sufficient to predict MYC activity and emphasize the need to consider multiple layers of regulation when interpreting its role in tumor biology.

Stratified analyses revealed subtype- and stage-specific patterns. Stage 2 tumors exhibited the lowest MYC expression, suggesting dynamic regulation during intermediate disease progression. Subtype analysis confirmed reduced expression across Luminal, HER2-positive, and TNBC tumors compared to adjacent normal tissue, with TNBC displaying the lowest transcript levels despite frequent amplification. Survival analyses further showed shorter progression-free survival in MYC-high and MYC-altered groups, consistent with MYC's potential role as a prognostic factor, particularly in TNBC where therapeutic options remain limited.

The identification of coexpressed genes such as TAF4B and TWNK further underscores MYC's involvement in regulatory networks beyond cell cycle control. TAF4B, a transcription factor involved in RNA polymerase II initiation, has emerging roles in breast cancer biology, while TWNK, a mitochondrial DNA helicase, is associated with mitochondrial function. These findings suggest that MYC may integrate into transcriptional and metabolic regulatory networks. Other strongly correlated genes, such as FAM216A and POLR1E, are less well studied in breast cancer and may help refine understanding of MYC-associated pathways. Prior pathway analyses reinforce the plausibility of these associations, given MYC's established roles in transcriptional control and metabolic adaptation[29,30].

Nevertheless, this study has several limitations. Analyses relied on bulk RNA-seq data, which measure average transcript levels across heterogeneous tumor and stromal compartments. TCGA clinical annotations also lack detailed treatment and receptor-status information, which may confound associations between MYC status and outcomes. Finally, while associations were consistent, they remain correlative. Single-cell RNA sequencing data could provide a more refined understanding of MYC's role in the tumor microenvironment. Future work using single-cell sequencing, spatial transcriptomics, and functional assays will be important to clarify MYC's regulatory mechanisms. Expanding analyses to include immune features and treatment response data could also help determine MYC's predictive value.

In conclusion, this study emphasizes MYC's complex and multifaceted role in breast cancer. Although MYC amplification is common, its mRNA expression patterns, and prognostic significance reveal context-specific behaviors that must be carefully considered in the development of MYC-targeted therapies. A deeper understanding of MYC's interaction with tumor biology may ultimately contribute to more effective, personalized treatment strategies for breast cancer patients

Methods

Data for this study were obtained from two publicly available resources: The Cancer Genome Atlas (TCGA) PanCancer Atlas via cBioPortal and the TCGA Breast Invasive Carcinoma (BRCA) cohort via UALCAN. These portals provide curated

and standardized genomic and transcriptomic datasets and are widely used in cancer bioinformatics research. cBioPortal was used to assess MYC gene alterations, including amplification, mutation, and frequency, as well as to perform survival and coexpression analyses.

UALCAN was used to evaluate MYC mRNA expression across clinical subgroups, including age, tumor stage, molecular subtype, gender, and race. Both cBioPortal and UALCAN apply internal pipelines for normalization and batch correction, which standardize expression values across samples. Expression data were log2-transformed within the portals to stabilize variance, and analyses were conducted directly on these processed datasets to preserve consistency with the source pipelines. Demographic and clinicopathological annotations were cross-checked against TCGA metadata for accuracy.

Group comparisons of MYC expression between tumor and normal tissues, as well as across clinical subgroups, were conducted using the built-in statistical functions provided by UALCAN. These rely on Student's t-test for differential expression analysis. Coexpression was assessed with Spearman's rank correlation using cBioPortal's correlation analysis tool. P- and q-values reported in Table 1 are directly obtained from cBioPortal, which applies FDR adjustment internally. An adjusted q-value < 0.05 was considered statistically significant.

Survival outcomes were evaluated using UALCAN's Kaplan-Meier survival analysis tool[28] for the TCGA BRCA cohort. Patients were stratified into high and low/medium MYC expression groups using UALCAN's default cutoff: the top 25% of tumors by expression were classified as "high," while the remaining 75% were grouped as "low/medium". Statistical significance between groups was tested using the log-rank method[33].

Coexpression analysis identified genes most strongly correlated with MYC expression in the TCGA BRCA cohort. Biological interpretation of these associations was guided by prior studies linking MYC activity to transcriptional regulation and mitochondrial metabolism. All analyses were carried out using cBioPortal (`https://www.cbioportal.org`) and UALCAN (`http://ualcan.path.uab.edu`). Visualizations were generated with built-in portal functions, and statistical calculations were cross-validated in R to ensure reproducibility.

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