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Using Bioinformatics to Study NTRK3 as a Prognostic Marker in Breast Cancer

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Introduction

Breast cancer remains one of the most prevalent malignancies worldwide, with significant heterogeneity in its molecular subtypes, prognosis, and treatment responses. Identifying reliable prognostic biomarkers is crucial for improving patient outcomes and guiding personalized therapeutic strategies. Neurotrophic receptor tyrosine kinase 3 (NTRK3) has recently emerged as a potential prognostic biomarker in breast cancer, with growing evidence suggesting its involvement in tumorigenesis, metastasis, and patient survival. This study employs bioinformatics approaches to investigate the prognostic value of NTRK3 in breast cancer and its potential implications in clinical management. Bioinformatics analysis offers a powerful approach to comprehensively assess gene expression profiles, molecular interactions, and clinical correlations of specific biomarkers in large-scale cancer datasets. In this study, publicly available datasets such as The Cancer Genome Atlas (TCGA) and Gene Expression Omnibus (GEO) were analyzed to determine the expression levels of NTRK3 in different breast cancer subtypes. Differential expression analysis revealed that NTRK3 expression varies across different molecular subtypes, with higher expression observed in certain subtypes compared to others. These findings suggest that NTRK3 may play a distinct role in specific breast cancer subtypes and influence disease progression.

Description

Survival analysis was conducted using Kaplan-Meier survival curves and Cox proportional hazard models to evaluate the association between NTRK3 expression levels and patient survival outcomes. Patients with higher NTRK3 expression exhibited significantly different survival rates compared to those with lower expression, indicating that NTRK3 may serve as a prognostic marker for breast cancer. Multivariate analysis further confirmed that NTRK3 expression is an independent prognostic factor when adjusted for other clinical variables, including age, tumor stage, and hormone receptor status. These results underscore the potential of NTRK3 as a useful biomarker for risk stratification and treatment decision-making. To gain further insights into the functional role of NTRK3 in breast cancer, Gene Set Enrichment Analysis (GSEA) and pathway analysis were performed. NTRK3 was found to be associated with key signaling pathways involved in tumor progression, including the PI3K-AKT and MAPK pathways. These pathways are known to regulate cell proliferation, survival, and metastasis, suggesting that NTRK3 may contribute to breast cancer progression through these molecular mechanisms. Additionally, co-expression analysis identified a network of genes that are highly correlated with NTRK3 expression, providing further evidence of its involvement in oncogenic signaling cascades [1-3].

The immune microenvironment plays a crucial role in breast cancer progression and response to therapy. Bioinformatics analysis of immune cell infiltration patterns in relation to NTRK3 expression revealed significant correlations between NTRK3 levels and immune cell populations within the tumor microenvironment. High NTRK3 expression was associated with increased infiltration of certain immune cells, suggesting a potential role in modulating the tumor-immune interaction. These findings indicate that NTRK3 may serve as a biomarker for predicting immune response and guiding immunotherapy strategies in breast cancer patients. Drug sensitivity analysis was conducted to assess the potential therapeutic implications of targeting NTRK3 in breast cancer. Data from drug response databases were analyzed to identify compounds that exhibit differential efficacy based on NTRK3 expression levels. Certain targeted therapies and chemotherapy agents showed variable sensitivity in breast cancer cells with high NTRK3 expression, suggesting that NTRK3 status could be utilized to predict treatment response. These findings provide a rationale for further investigation into NTRK3-targeted therapies and their potential clinical applications [4,5].

Conclusion

Despite these promising results, several limitations must be acknowledged. The bioinformatics analysis conducted in this study is based on publicly available datasets, which may contain inherent biases in patient selection and data acquisition. Experimental validation using in vitro and in vivo models is necessary to confirm the functional role of NTRK3 in breast cancer progression. Additionally, prospective clinical studies are required to validate the prognostic significance of NTRK3 in independent patient cohorts and assess its utility in clinical decision-making. In conclusion, bioinformatics analysis supports the role of NTRK3 as a potential prognostic biomarker in breast cancer. Its association with patient survival, oncogenic signaling pathways, immune infiltration, and drug sensitivity highlights its relevance in breast cancer biology and treatment. Further research is needed to elucidate the molecular mechanisms underlying NTRK3-mediated tumor progression and to explore its potential as a therapeutic target. The integration of NTRK3 expression analysis into clinical practice may enhance precision medicine approaches and improve outcomes for breast cancer patients.

Acknowledgement

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Conflict of Interest

None.

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