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## DRP1

Dynamin-related protein-1 (DRP1) is a regulator of mitochondrial fission.

Breast cancer (BC) is the leading cause of cancer-related mortality in women worldwide. The identification of effective markers for early diagnosis and the prognosis is important for reducing mortality and ensuring that therapy for BC is effective.

The TCGA, Oncomine, UALCAN and HPA databases were used to examine DRP1 expression in BC. Kaplan-Meier plotter and PrognoScan were used to evaluate the association of DRP1 with the prognosis of patients with BC. The mechanism was investigated with Gene Ontology (GO) and Kyoto Encyclopedia of Genes and Genomes (KEGG) analyses, and the relationship between DRP1 expression and immune infiltration in BC was investigated using the TIMER database and CIBERSORT algorithm.

DRP1 expression was significantly upregulated in BC compared to healthy breast tissues. In addition, elevated DRP1 expression was associated with various clinicopathological parameters. High DRP1 expression was significantly correlated with poor survival of BC patients. GO and KEGG analyses indicated that DRP1 was closely correlated with various signaling pathways and immune response. Functional analyses revealed that DRP1 was positively correlated with infiltration levels of B cells, CD8+ T cells, CD4+ T cells, macrophages, neutrophils, and dendritic cells. Moreover, DRP1 affected the prognosis of BC patients partially via immune infiltration.

The results suggest that DRP1 is a marker of poor prognosis in patients with BC and plays an important role in tumor-related immune infiltration <sup>1)</sup>.

DRP1 expression was measured by immunohistochemistry and Western blotting. Correlations between DRP1 expression and clinicopathological parameters were determined by statistical analysis. Differences in survival were compared using the log-rank test. DRP1 expression was detected in 87.2% (41/47) of the investigated patients with Glioblastoma.

Results: The patients with higher DRP1 levels had worse survival (p = 0.0398). In vitro, the silencing of DRP1 reduced cell proliferation, invasive potential, and radiation resistance. The addition of shikonin inhibited DRP1 expression and increased drug uptake. Moreover, shikonin reduced the nuclear entry of DNA repair-associated enzymes and increased radiation sensitivity, suggesting that reducing DRP1 expression could inhibit DNA repair and increase the radiation sensitivity of Glioblastoma cells.

TheOur results indicate that DRP1 overexpression is a prospective radio-resistant phenotype in Glioblastoma. Therefore, DRP1 could be a potential target for improving the effectiveness of radiation therapy <sup>2)</sup>.

1)

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