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TFAP2A downregulation mediates tumor-suppressive effect of miR-8072 in triple-negative breast cancer via inhibiting SNAI1 transcription

Abstract:

Triple-negative breast cancer (TNBC) is a highly aggressive subtype of breast cancer characterized by challenging clinical management and poor prognosis. While TFAP2A, a member of the AP-2 transcription factor family, is known to maintain the basal phenotype of breast cancer, its specific regulatory role in TNBC has remained undefined. In this study, in vitro assessments using MTS, colony formation, and EdU assays were conducted to evaluate TNBC cell growth and migratory potential. Quantitative PCR and Western blot analyses were employed to assess mRNA and protein expression levels, as well as the phosphorylation status of AKT and ERK. The post-transcriptional regulation of TFAP2A by miR-8072 and the transcriptional activation of SNAI1 by TFAP2A were investigated through luciferase reporter assays. A xenograft mouse model was used to evaluated the in vivo growth capacity of TNBC cells. The results demonstrated that selective silencing of TFAP2A significantly inhibited TNBC cell proliferation and migration, with elevated TFAP2A expression observed in breast cancer tissues. Notably, TNBC patients exhibiting heightened TFAP2A levels experienced shortened overall survival. Mechanistically, TFAP2A was identifed as a transcriotional activator of SNAI1, a crucial regulator of epithelial-mesenchymal transition (EMT) and cellular proliferation, thereby augmenting the oncogenic properties of TFAP2A in TNBC. Moreover, miR-8072 was unveiled as a negative regulator of TFAP2A, exerting potent inhibitory effects on TNBC cell growth and migration

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