FAM50A PROMOTES ESOPHAGEAL SQUAMOUS CELL CARCINOMA PROGRESSION VIA SPLICING-DEPENDENT ABERRANT CIRCSDK1 AND DUSP22-MEDIATED MAPK SIGNALING ACTIVATION

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Research identifies splicing factor FAM50A as an oncogenic driver in esophageal squamous cell carcinoma through a novel TFAP2C/FAM50A/circSDK1/DUSP22 axis that activates MAPK signaling.

KEY POINTS

- FAM50A was upregulated in ESCC tissues, and its high expression correlated with aggressive clinicopathological features and poor prognosis.
- In vitro and in vivo experiments demonstrated that FAM50A promoted proliferation, migration, and invasion of ESCC cells.
- FAM50A drives aberrant formation of circSDK1 through back-splicing of exon 2, 3 and 4 of SDK1; circSDK1 binds to DUSP22 and prevents it from dephosphorylating MAPKs, leading to MAPK activation.
- Transcription factor TFAP2C enhances the transcriptional level of FAM50A.
- Rifabutin was identified as a small molecule inhibitor of FAM50A with cancer-inhibiting properties demonstrated in patient-derived organoids and xenograft models.

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