

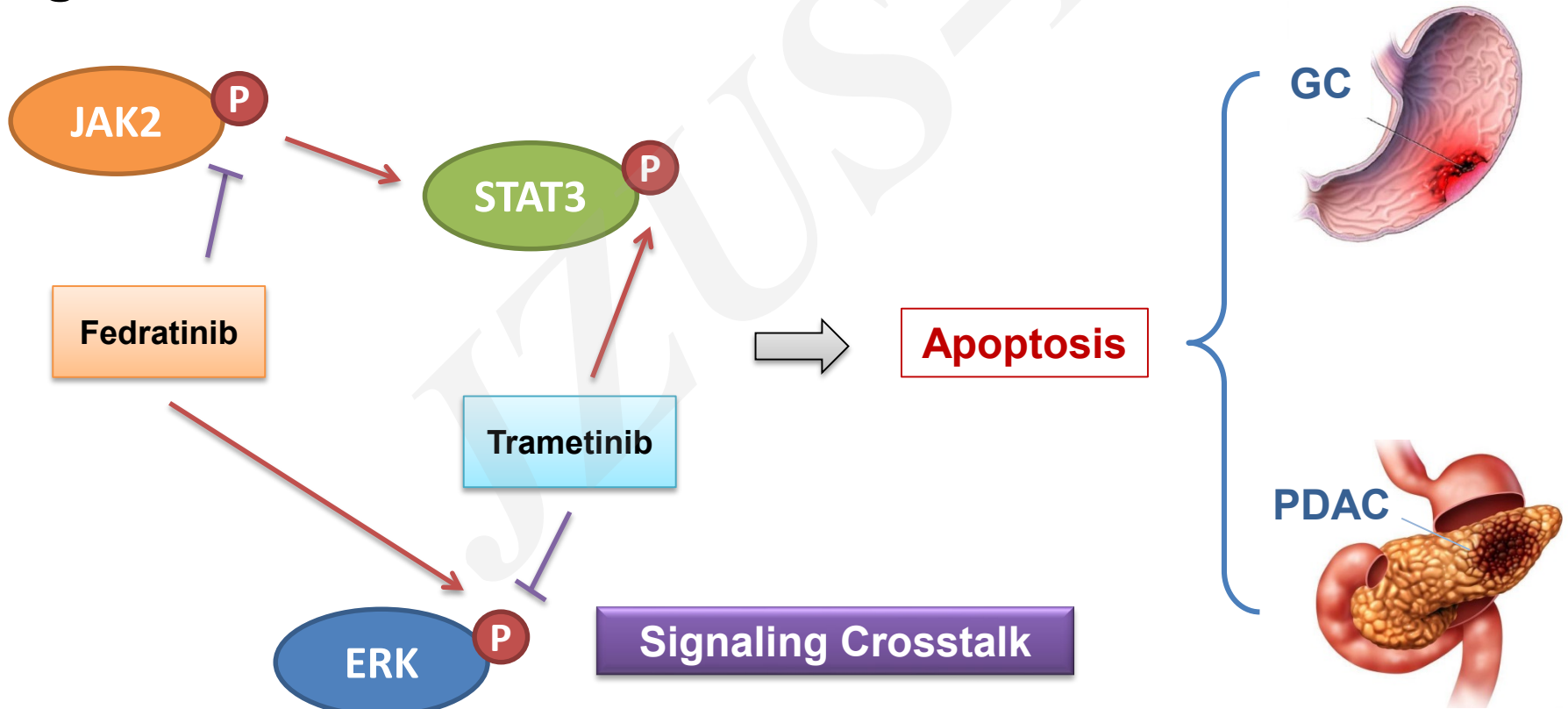
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Blocking the JAK2-STAT3 and ERK pathways suppresses the proliferation of gastrointestinal cancers by inducing apoptosis

Key words: Gastrointestinal cancers; JAK2-STAT3 pathway; ERK pathway; Crosstalk; Apoptosis

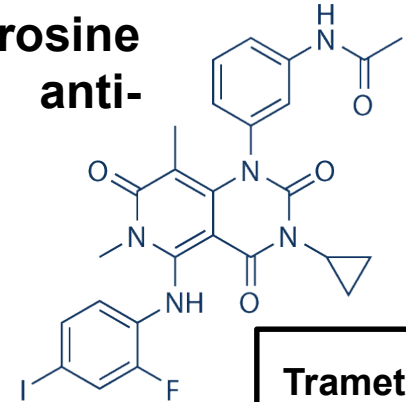
Research Summary

A novel crosstalk between JAK2/STAT3 and ERK signaling in gastric cancer (GC) and pancreatic ductal adenocarcinoma (PDAC) cells and provides a therapeutic strategy to overcome potential resistance in gastrointestinal cancer.



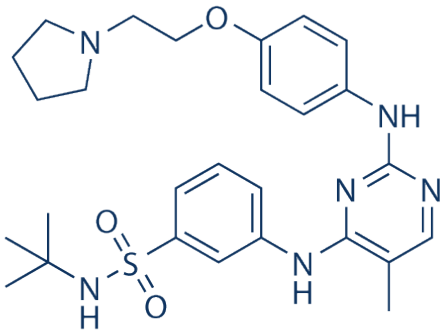
Innovation points

- Abrogation of MEK/ERK signaling upregulates tyrosine phosphorylation of JAK2 and STAT3, blocking anti-proliferative effects in gastric and PDAC cancer cells



Trametinib

- Inhibition of JAK2/STAT3 signaling upregulates the tyrosine phosphorylation level of EGFR and ERK in GC and PDAC cells, respectively

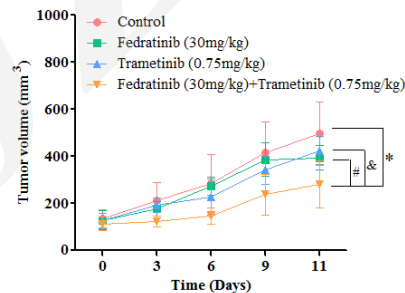
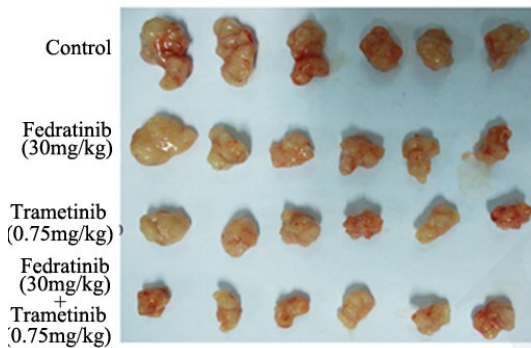
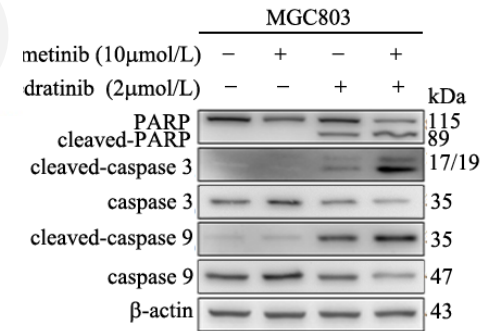
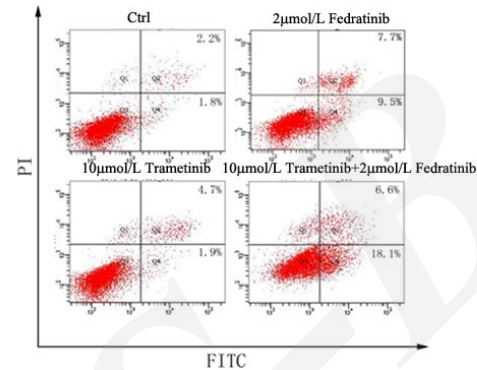


Fedratinib

- Proliferation of GC and PDAC cells is effectively retarded by inhibition of JAK2/STAT3 and ERK signaling

Innovation points

- Interference with JAK2/STAT3 and ERK signaling triggers apoptosis in MGC803 gastric cancer cells



- Disturbance of JAK2/STAT3 and ERK signaling profoundly impairs tumor growth in mice by inducing apoptosis