

Analysis of the Effects of Ferulic Acid on the Proliferation and Migration of Renal Cell Carcinoma: FGFR1-mediated PI3K/AKT Pathway Mechanism

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SUMMARY. This study investigated the potential of ferulic acid (FA) as a therapeutic drug for clear cell renal cell carcinoma (ccRCC) by exploring its function and mechanism. This study conducted *in-vitro* utilizing Human ccRCC cell lines. Results showed that FA inhibited cell viability, colony formation, and migration. It was also noted that FA promoted cell apoptosis in a dose-dependent manner in ccRCC cells by down-regulating FGFR1 expression via blocking the PI3K/AKT pathway. Lentivirus-FGFR1 (lenti-FGFR1) eliminated the effects of FA on ccRCC cells, while LY294002, a PI3K/AKT pathway inhibitor, strengthened the inhibitory effects of FA. These findings suggest that FGFR1-mediated PI3K/AKT signaling is a promising mechanical target for ccRCC, and that FA may act as a novel therapeutic drug for ccRCC treatment.

RESUMEN. Este estudio investigó el potencial del ácido ferúlico (FA) como fármaco terapéutico para el carcinoma de células renales de células claras (ccRCC) mediante la exploración de su función y mecanismo. Este estudio se realizó *in vitro* utilizando líneas celulares ccRCC humanas. Los resultados mostraron que la FA inhibía la viabilidad celular, la formación de colonias y la migración. También se observó que FA promovió la apoptosis celular de una manera dependiente de la dosis en células ccRCC mediante la regulación negativa de la expresión de FGFR1 mediante el bloqueo de la vía PI3K/AKT. Lentivirus-FGFR1 (lenti-FGFR1) eliminó los efectos de la FA en las células ccRCC, mientras que LY294002, un inhibidor de la vía PI3K/AKT, reforzó los efectos inhibidores de la FA. Estos hallazgos sugieren que la señalización de PI3K/AKT mediada con FGFR1 es un objetivo mecánico prometedor para ccRCC, y que FA puede actuar como un nuevo fármaco terapéutico para el tratamiento de ccRCC.

KEY WORDS: clear cell renal cell carcinoma, ferulic acid, FGFR1, PI3K/AKT pathway

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