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Therapeutic Advancement in Lung Cancer Treatment by MicroRNA-582-5p: a Comparative Study

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SUMMARY. **MicroRNAs play important regulatory roles in the pathogenesis of non-small cell lung cancer (NSCLC). This study is aimed to investigate the function of miR-582-5p in NSCLC and reveal relevant downstream mechanisms. The expression of miR-582-5p and EIF4G2 were measured in NSCLC cell lines (H460, H647, and H1299) and a normal lung epithelial cell line (BEAS-2B) by qRT-PCR. MiR-582-5p mimic and inhibitor were used to overexpress and silence miR-582-5p in H1299 cells, respectively. Cell viability and invasion were measured by CCK-8 and trans-well assays, respectively. The targeting relationship between miR-582-5p and EIF4G2 was determined by DLR assay. In addition, the protein expression of EIF4G2, ERK, and p-ERK were detected by Western blot. The expression of miR-582-5p was lower in NSCLC cells than that in normal lung epithelial cells. MiR-582-5p mimic decreased cell viability and invasion, up-regulated E-cadherin, and down-regulated Vimentin in H1299 cells. EIF4G2 was determined as a target of miR-582-5p, which was up-regulated in NSCLC cell lines. In addition, miR-582-5p mimic significantly decreased the protein expression of EIF4G2 and p-ERK in H1299 cells. MiR-582-5p inhibitor showed opposite results with miR-582-5p mimic in H1299 cells. MiR-582-5p inhibits the viability and invasion of LUAD cells through down-regulating EIF4G2 and ERK phosphorylation.**

RESUMEN. Los microARN desempeñan funciones reguladoras importantes en la patogenia del cáncer de pulmón de células no pequeñas (NSCLC). Este estudio tiene como objetivo investigar la función de miR-582-5p en NSCLC y revelar mecanismos posteriores relevantes. La expresión de miR-582-5p y EIF4G2 se midió en líneas celulares de NSCLC (H460, H647 y H1299) y una línea celular epitelial de pulmón normal (BEAS-2B) mediante qRT-PCR. Se utilizaron miméticos e inhibidores de MiR-582-5p para sobreexpresar y silenciar miR-582-5p en células H1299, respectivamente. La viabilidad celular y la invasión se midieron mediante ensayos CCK-8 y trans-well, respectivamente. La relación de orientación entre miR-582-5p y EIF4G2 se determinó mediante el ensayo DLR. Además, la expresión de proteínas de EIF4G2, ERK y p-ERK se detectó mediante Western blot. La expresión de miR-582-5p fue menor en las células de NSCLC que en las células epiteliales de pulmón normales. MiR-582-5p imita la disminución de la viabilidad e invasión celular, la E-cadherina regulada al alza y la vimentina regulada a la baja en las células H1299. EIF4G2 se determinó como un objetivo de miR-582-5p, que estaba regulado al alza en las líneas celulares de NSCLC. Además, miR-582-5p mimic disminuyó significativamente la expresión de proteínas de EIF4G2 y p-ERK en células H1299. El inhibidor de MiR-582-5p mostró resultados opuestos con el mimético de miR-582-5p en células H1299. MiR-582-5p inhibe la viabilidad y la invasión de células LUAD mediante la regulación negativa de la fosforilación de EIF4G2 y ERK.

KEY WORDS: EIF4G2, ERK, microRNA-582-5p, non-small cell lung cancer.

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