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OVEREXPRESSION OF MIR-155-5P INHIBITS THE PROLIFERATION AND MIGRATION OF IL-13-INDUCED HUMAN BRONCHIAL SMOOTH MUSCLE CELLS BY SUPPRESSING TGF-BETA ACTIVATED KINASE 1/MAP3K7 BINDING PROTEIN 2

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Aims: The molecular mechanisms leading to asthma is still ill-defined. Though the function of microRNAs (miRNAs) in asthma was reported previously, the involvement of miR-155 in important features of this disease remains unknown. The present study was designed to uncover the probable involvement of miR-155-5p in the proliferation and migration of IL-13-induced human bronchial smooth muscle cells (BSCMs) and the intrinsic regulatory mechanism.

Methods: The effects of different concentration of IL-13 on the proliferation and migration of BSMCs as well as the expression of miR-155-5p and its predicted target TGF-beta activated kinase 1/MAP3K7 binding protein 2 (TAB2) were investigated. The effects of miR-155-5p on the proliferation and migration of IL-13-induced BSMCs was determined in vitro using BSMCs transfected by miR-155 mimics and inhibitors and induced by a high concentration of IL-13. The quantitative real time PCR (qRT-PCR) was employed for determining the expression of miR-155-5p and TAB2. Western blotting was applied to analyze the expression of TAB2 at protein level. Cell proliferation and migration were assessed using the MTT and transwell assays, respectively.

Results: The proliferation and migration of BSMCs were dose-dependently increased with IL-13 treatment. Contrariwise, IL-13 dose-dependently inhibited the expression miR-155-5p in BSMCs. Mechanistic studies showed that inhibition of miR-155-5p further promoted the stimulatory effects of IL-13 whereas overexpression of miR-155 significantly inhibited these effects. *In silico* studies and luciferase reporter assays indicated that TAB2 was a negatively regulated miR-155-5p target.

Conclusion: These data suggested that miR-155-5p inhibits the IL-13-induced proliferation and migration of BSMCs by targeting TAB2. Our data also suggested that the IL-13/miR-155/ TAB2 pathway could serve as a therapeutic target for pulmonary diseases, especially asthma.

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