Erratum


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Abstract: Metastasis remains the leading cause of the majority of cancer-related mortality. MicroRNAs (miRNAs) have frequently emerged as tumor metastatic regulator by acting on multiple signaling pathways. In the present study, we demonstrated that miR-338-3p was significantly downregulated in highly metastatic NSCLC cell lines and clinical metastatic tissues. Then, we found that introduction of miR-338-3p significantly suppressed the migration and invasion of lung cancer cells both in vitro and in vivo, suggesting that miR-338-3p may be a novel tumor suppressor. Further studies indicated that the EMT-related transcription factor Sox4 was one direct target gene of miR-338-3p, evidenced by the direct binding of miR-338-3p with the 3'untranslated region (3'UTR) of Sox4. Furthermore, miR-338-3p could decrease the expression of Sox4 both at mRNA and protein levels. Notably, the EMT marker E-cadherin or vimentin, a downstream regulator of Sox4, was also down-regulated or up-regulated upon miR-338-3p treatment. Additionally, over-expressing or silencing Sox4 could elevate or inhibit the migration and invasion of lung cancer cells, parallel to the effect of miR-338-3p on the lung cancer cells. Meanwhile, knockdown of Sox4 reversed the enhanced migration and invasion mediated by miR-338-3p. These results indicated that miR-338-3p suppressed the migration and invasion of NSCLC cells through targeting Sox4 involving in the EMT process. Thus, our finding provides new insight into the mechanism of NSCLC progression. Therapeutically, miR-338-3p may serve as a potential target in the treatment of human lung cancer.

Keywords: MiR-338-3p, NSCLC, metastasis, sox4

For this article, it’s type has been published as “review article”, it should be “original article”, we publish this revised notes to correct this article type as “original article”. The authors express regrets for this error.

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