

Manuscript TUBI-D-15-01503 for review

"Editorial Office Tumor Biology" <em@editorialmanager.com>

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时 间: 2015-5-28 7:59:21

附 件:

Dear Prof. Guo,

In view of your expertise I would be very grateful if you could review the following manuscript which has been submitted to Tumor Biology.

Manuscript Number: TUBI-D-15-01503

Title: MicroRNA-138 negatively regulates non-small cell lung cancer cells through the interaction with Cyclin D3

Abstract: Background: Previous studies demonstrate that microRNA-138 (miR-138) is critical in non-small cell lung cancer (NSCLC) regulation. We further explored the molecular mechanism of miR-138 in NSCLC.

Methods: Lentivirus was used to upregulate miR-138 in NSCLC cell lines H460 and SPC-A1 cells. Previously known effects of miR-138 upregulation on NSCLC, proliferation, cell cycle division and cisplatin sensitivity, were assessed. Moreover, previously unknown effect of miR-138 upregulation on NSCLC migration was also examined in H460 and SPC-A1 cells. A new miR-138 downstream target, cyclin D3 (CCND3), was assessed by dual-luciferase reporter assay and qRT-PCR. CCND3 was then ectopically over-expressed in H460 and SPC-A1 cells. The effects of forced overexpression of CCND3 on miR-138 induced NSCLC regulations, were further examined by proliferation, cell cycle, cisplatin sensitivity and migration assays, respectively.

Results: Lentivirus-induced miR-138 upregulation inhibited NSCLC proliferation and cell cycle division, in line with previous findings. Moreover, we found miR-138 upregulation had other anti-tumor effects, such as increasing cisplatin sensitivity and reducing cancer migration, in H460 and SPC-A1 cells. Luciferase assay and qRT-PCR showed CCND3 was directly targeted by miR-138. Forced overexpression of CCND3 in H460 and SPC-A1 cells reversed the anti-tumor effects of miR-138 upregulation on cancer cell growth, cell cycle, cisplatin sensitivity and migration.

Conclusions: Our study revealed novel anti-cancer effects of miR-138 upregulation in NSCLC, as well as its new molecular target of CCND3.

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If you have any questions, please do not hesitate to contact us. We appreciate your assistance.

With kind regards,
Torgny Ingemar Stigbrand, Ph.D., MD., Prof
