

microRNA-26a-5p regulates the cancer stemness and enhances the chemosensitivity of lung adenocarcinoma

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Background: Cancer stem cells (CSCs) identified in lung cancer exhibit resistance to chemotherapy, radiotherapy, and targeted therapy. Therefore, a technology to control of CSCs is needed to overcome such resistance to cancer therapy. Various evidences about the association between epithelial-mesenchymal transition related transcriptomic alteration and acquisition of CSC phenotype have been proposed recently. In our previous research, down-regulated miR-26a-5p is closely related to mesenchymal-like lung cancer cell lines. These findings suggest that miR-26a-5p might be involved in lung cancer stemness.

Methods: Five cell lines, A549, BEU, H292, H358, HCC827, were chosen. Each cell lines was treated with fine dust, methylated by MDB sequencing. Cell lines have two groups, treated with fine dust and not treated with fine dust. In this study, we found genes that showed similar changes in the gene expression of the fine dust treated group in the three cell lines compared to the group not treated with fine dust. Results miR-26a-5p regulated the expression of POLR3G directly. Overexpression of miR-26a-5p induced down regulation of POLR3G and a marked reduction of colony formation and sphere formation. Co-treatment of miR-26a-5p with paclitaxel decreased cell growth, suggesting that miR-26a-5p might play a role as a chemotherapy sensitizer. In the cancer genome atlas data, down-regulated miR-26a-5p and up-regulated POLR3G were shown compared to adjacent normal tissue. High miR-26a-5p and low POLR3G expression were also related to higher survival rate of patients with lung adenocarcinoma.

Conclusions: Overexpression of miR-26a-5p can suppress lung cancer stemness and make cancer cell become sensitive to chemotherapy. This finding provides a novel insight into a potential lung cancer treatment by regulating stemness.

