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Hsa-miR-326 Functions as a Tumor Suppressor in Non-Small Cell Lung Cancer through Targeting CCND1

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Abstract : Hsa-miRNA-326 (miR-326) has recently been discovered having anticancer efficacy in different organs. However, the role of miR-326 on non-small cell lung cancer (NSCLC) is still ambiguous. In this study, we investigated the role of miR-326 on the development of NSCLC. The results indicated that miR-326 was significantly down-regulated in primary tumor tissues and very low levels were found in NSCLC cell lines. Ectopic expression of miR-326 in NSCLC cell lines significantly suppressed cell growth as evidenced by cell viability assay, colony formation assay and BrdU staining, through inhibition of cyclin D1, cyclin D2, CDK4, and up-regulation of p57(Kip2) and p21(Waf1/Cip1). In addition, miR-326 induced apoptosis, as indicated by concomitantly with up-regulation of key apoptosis protein cleaved caspase-3, and down-regulation of anti-apoptosis protein Bcl2. Moreover, miR-326 inhibited cellular migration and invasiveness through inhibition of matrix metalloproteinases (MMP)-7 and MMP-9. Further, oncogene CCND1 was revealed to be a putative target of miR-326, which was inversely correlated with miR-326 expression in NSCLC. Taken together, our results demonstrated that miR-326 played a pivotal role on NSCLC through inhibiting cell proliferation, migration, invasion, and promoting apoptosis by targeting oncogenic CCND1.

Keywords : hsa-miRNA-326 (miR-326),cyclin D1, non-small cell lung cancer (NSCLC), proliferation, apoptosis
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