Impact of MiR-93 on Non-Small Cell Lung Carcinoma

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Lung cancer is the most common non-cutaneous diagnosed cancer, and the leading cause of cancer death in various parts of the world. It is classified into small-cell lung carcinoma (SCLC) & non-small cell lung carcinoma (NSCLC) and corresponds in the ratio of 1:4 of all lung carcinomas, respectively [1]. Several microRNAs [miR-21, miR-93, miR-17-92 cluster (miR-17, miR-18a, miR-19a, miR-19b-1, miR-20a, and miR-92a-1 etc.] involve as diagnostic, prognostic, and targeted therapeutic tools in various cancers including NSCLC [2–4]. The aberrant expression and dysfunction of miR-93 (an oncomiR) involved in tumor progression, metastasis, and poor prognosis in hepatocellular carcinoma (HCC), lung cancer [5], breast cancer, gastric carcinoma, and nasopharyngeal carcinoma [6]. It is due to downregulation of various tumor suppressor genes (TSG) [PTEN, CDKN1A, SMAD7, PDCD4, NEDD4L and TGFβR2]. Although miR-93 expression increased in tissues-plasma of NSCLC patients yet the precise causal mechanism of miR-93 involvement in NSCLC is unknown.

The miR-93 overexpression cause proliferation, colony formation, migration, and invasion in NSCLC and it is directed by phosphotidyl inositol-3 kinase (PI3)/Akt pathway [7]. The overexpression of miR-93 blocks the activity of network and TSG like PTEN, CDKN1A and LKB1. The consequence is that it inhibits the mammalian target of rapamycin (mTOR) and related network, therefore, it can’t regulate proliferation, migration and invasion of NSCLC. In contrast, PI3K and AKT pathway activate invasion, migration, proliferation of NSCLC cells. Therefore, application of suitable protease inhibitors (like Mk-2206) & miR-93 down regulation inactivate Akt pathway and thus inhibit NSCLC progression and invasion. In effect, due to multiple target pathophysiology of NSCLC, the interaction of several other miRNA, gene-gene, gene-environment interaction (epigenetic) should be decipher, and thus providing a promising and valuable therapeutics tools.

References

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