Commentary

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NEAT1/miR-140-3p/MAPK1 Mediates the Viability and Survival of Coronary Endothelial Cells and Affects Coronary Atherosclerotic Heart Disease?

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Description

The Nuclear enriched abundant transcript 1 (NEAT1) is a newly discovered lncRNA, which was identified to be involved in biological behaviors of endothelial cells. However, the role and molecular mechanism of NEAT1 in coronary atherosclerotic heart disease (CAD) are still unknown. Reports that NEAT1 was up-regulated in CAD and lncRNA NEAT1 increased cell viability and inhibited CAD cell apoptosis possibly by activating the miR-140-3p/MAPK1 pathway. NEAT1 is a nuclear-restricted lncRNA, which is a crucial architectural part of nuclear paraspeckles and regulates multiple gene expressions by nuclear retention. Existing researches report that lncRNA NEAT1 participates in various cellular and biological processes in cardiac and cerebrovascular diseases, such as atherosclerosis, myocardial infarction, and stroke. For example, one functional experiment in cardiomyocyte injury suggests that lncRNA NEAT1 is upregulated in the ischemia/reperfusion myocardium, and its knockdown decreases the trend of hypoxia/reoxygenation-induced cardiomyocyte apoptosis found that NEAT1 could predict increased major adverse cardiac and cerebrovascular even trisk independently in patients with unprotected left main coronary artery disease underwent coronary artery bypass grafting. In NEAT1, a miR-140 binding site was identified and they also found that miR-140 could physically bind to Neat1 in the nucleus. MicroRNAs (miRNAs) are small, endogenous, non-coding RNAs, which are essential regulators of cellular function and gene expression.

MiRNA precursors are exported to the cytoplasm after nuclear processing, where the mature miRNA is formed. MiRNAs, which regulate various physiological processes such as cell proliferation, metabolism, apoptosis, and organ development at the post-transcriptional level, have been extensively studied in a variety of cancer diseases.

Several miRNAs have been found to play important regulatory roles in either promoting (miR-140 and miR-455) or suppressing (miR-27 and miR-133) adipogenesis6. Recent work has identified a role of miR-140 in adipogenesis, miR-140 is activated by bone morphogenic protein 4 (BMP4) and induces commitment to the adipogenic lineage by targeting the adipogenesis inhibitor osteopetrosis-associated transmembrane protein 1 (OSTM1) for degradation. miR-140 plays a critical role in preadipocyte differentiation and regulates adipocyte differentiation through transforming growth factor-β signaling pathway. Besides, through targeting YES proto-oncogene 1 (YES1), miR-140 attenuates myocardial ischemia-reperfusion. Expression of miR-140 is upregulated in afterload-enhanced engineered heart tissue, while miR-140-3p inhibitors have been demonstrated in protective function. Recent studies show that Neat1/miR-140 axis was involved in cardiomyocyte apoptosis11 in diabetic cardiomyopathy and exacerbated nonalcoholic fatty liver through interrupting AMPK/SREBP-1 signaling pathway. In the present article, show NEAT1 was up-regulated and miR-140-3p was down-regulated in CAD patients. To illustrate the molecular mechanism, this study measured the expression levels of miR-140-3p in human coronary endothelial cells (HCAECs) and human umbilical vein endothelial cells (HUVECs). Colony formation experiments, cell proliferation experiments, apoptosis experiments and dual luciferase reporter gene experiments were used to study cell colony migration, cell proliferation, apoptosis and targeting capabilities. The results showed lncRNANEAT1 increased the viability of CAD cells and inhibited apoptosis by activating the miR-140-3p/MAPK1 pathway. To reproduce these results, more people need to be included. Despite this limitation have provided a strong argument for using lncRNA NEAT1 as a potential therapeutic target for CAD.