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Non-coding RNAs in diabetes induced cardiac fibrosis

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Non coding RNAs play important roles in cellular process. Both long noncoding RNAs (lncRNAs) and miRNA (miR) control gene expression, thus regulating pathologic changes. miR-9 targets important extracellular matrix(ECM) proteins, such as fibronectin (FN) and collagen (Col), key molecules in cardiac fibrosis. In other systems, lncRNA ZNFX1-AS1 has been shown to regulate miR-9 through methylation. We investigated the role of miR-9 in the endothelial cells (ECs) and in the heart of diabetic animals and possible regulatory mechanism through ZNFX1-AS1. PCR array was used to examine miR expression in ECs exposed to various glucose levels. miR-9 expression was validated by qPCR in ECs and in the mice hearts. Expression of ZNFX1-AS1 and ECM proteins targeted by miR-9 were examined. Luciferase assay was used to determine the interaction between miR-9 and its target genes. Cardiac tissues from streptozotocin (STZ)-induced diabetic mice were similarly examined. PCR array showed glucose-induced alterations of multiple miRNAs in ECs. Fifty-one miRNAs were upregulated and 74 were downregulated. High glucose (HG) decreased miR-9 expression in association with increased ZNFX1-AS1 RNA. FN mRNA, Col1&4 mRNA and protein levels in ECs were also increased in HG. miR-9 mimic transfection prevented glucose-induced overexpression of these ECM proteins. Interestingly, methylation blockade also reduced ECM protein overexpression, in spite of high ZNFX1-AS1 RNA. Luciferase assay showed the binding of FN to 3'-UTR of miR-9. miR-9 expression was also decreased in the hearts of the diabetic animals. However expression of ZNFX1-AS1 and ECM proteins were increased. These studies indicate a novel glucose-induced mechanism of increased ECM protein production, which is regulated by miR-9 possibly via ZNFX1-AS1 in the ECs and hearts diabetes. Identifying such mechanisms may lead to potential RNA based therapeutics.

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