Perspective
Volume 8:1, 2023

Journal of Diabetic Complications & Medicine

ISSN: 2475-3211 Open Access

Controlling of Protein Binding by NcRNA in Diabetic Foot

Lobmann Thole*

Department of Diabetes, University of Bridgeport, Park Avenue, Bridgeport, USA

Abstract

Almost 90% of the human genome is made up of non-coding RNA, a specific kind of RNA transcript. A variety of biological processes, such as cellular metabolism, development, proliferation, transcription and post-transcriptional modification, are indirectly regulated by ncRNA, despite the fact that it normally does not encode proteins. NcRNAs include small interfering RNAs, PIWI-interacting RNAs and small RNAs produced from tRNA, among others. The three that have received the most attention are the miRNA, lncRNA and circRNA, which play a key role in the development of diabetes and its side effects. The ncRNAs mentioned above are associated with a variety of complications of diabetes, such as diabetic foot, diabetic nephropathy, diabetic cardiomyopathy and diabetic peripheral neuropathy, by binding proteins.

Keywords: Protein binding • Diabetic foot • Metabolic disorder

Introduction

Almost 90% of the human genome is made up of non-coding RNA, a specific kind of RNA transcript. A variety of biological processes, such as cellular metabolism, development, proliferation, transcription and post-transcriptional modification, are indirectly regulated by ncRNA, despite the fact that it normally does not encode proteins. NcRNAs include small interfering RNAs, PIWI-interacting RNAs and small RNAs produced from tRNA, among others. The three that have received the most attention are the miRNA, lncRNA and circRNA, which play a key role in the development of diabetes and its side effects. The ncRNAs mentioned above are associated with a variety of complications of diabetes, such as diabetic foot, diabetic nephropathy, diabetic cardiomyopathy and diabetic peripheral neuropathy, by binding proteins [1].

Description

According to current research, lncRNA GAS5 can activate the HIF1A/VEGF pathway by binding to TAF15 to promote DFU wound healing, whereas Mir-146a can influence the AKAP12 axis to promote the proliferation and migration of diabetic foot ulcer cells. However there are still a lot of unsolved concerns regarding how ncRNAs work. We investigated the mechanism and recent developments of ncRNA-protein binding in DF in this study, which can support and direct the use of ncRNA in the early diagnosis and potential tailored treatment of DFU [2].

The metabolic disorder known as diabetes, which is characterised by high blood glucose levels, is mostly brought on by the body's pancreas failing to produce enough insulin or the cells becoming resistant to it. The two most common types of the condition are type 1 and type 2 and both are characterised by patients' chronically increased blood glucose levels, tiredness and obesity. People all across the world are impacted by the chronic disease known as diabetes. Uncontrolled diabetes has the potential to cause serious short- and long-term issues, such as diabetic foot ulcers, retinopathy and diabetic nephropathy [3].

DFUs, which are ischemic, neurologic and neurobiochemical lesions of the foot, are developed in diabetics with poor long-term glucose management. Reduced insulin growth factor-1 IGF-1, which promotes skin development, sensory, motor and autonomic neuropathy, vasculopathy, circulatory issues, immunological abnormalities and immune disorders are additional contributing factors to its pathogenesis. Patients frequently experience peripheral neuropathy and peripheral arterial disease, which can cause foot deformities, the breakdown of deep tissues, the development of infected ulcers and ultimately wounds that force amputation of the lower limb owing to malfunction. DFU cause severe economic and social hardship for the entire country as well as physical and mental suffering for the patients [4].

RNAs that don't code 98% of the RNA in humans is made up of the heterogeneous class of ncRNAs. NcRNAs can be broadly categorised as miRNAs, long non-coding RNAs and circular RNAs. Instead of encoding proteins, these sequences function as post-transcriptional and transcriptional regulators of gene expression. By attaching to proteins participating in protein transport, RNA processing and modification, chromosomal structure and mRNA stabilisation and regulation at the translational level [5].

Recent research has shown that ncRNAs can have a substantial impact on conditions like cancers, cardiovascular disorders and diabetes. As an illustration, mitochondrial localization-related miRNAs can be produced from the mitochondrial genome as well as imported into the organelles. There are also some exosomal ncRNAs that can be employed as biomarkers for diabetes complications, which may offer fresh perspectives on how to avoid, identify and treat diabetic problems.

The aberrant expression of ncRNAs can be exploited as markers for the diagnosis, prevention and therapy of DFU, according to a growing body of clinical studies as well as basic research. For instance, therapy with miRNA-497 in vivo and in vitro can decrease the expression of pro-inflammatory cytokines such IL-1, IL-6 and TNF- and hasten DFU wound healing. Via miR-155/HIF-1, CASC2 overexpression in DFU enhances wound healing.

CircRNA with the sequence circ 0084443 was increased in DFU patients, which promoted keratinocyte chemotaxis and proliferation. In DFU, ncRNAs and the proteins that bind to them have not yet been completely understood. As a result, the pathophysiology of DFU is reviewed in this article from the standpoint of ncRNA-protein binding.

MicroRNAs are a class of non-coding RNAs that range in length from 21 to 25 nucleotides. They are essential for RNA silencing and post-transcriptional regulation of gene expression and are consequently involved in a wide range of cellular processes, including migration, differentiation, proliferation and apoptosis [6].

The inner surface of blood arteries all over the body is coated with endothelial cells, which arrange themselves in a monolayer. In the majority of mammalian systems, ECs have a constrained capacity to split. MiRNA has been well examined and investigated for its possible contribution to endothelial dysfunction and it is essential for controlling EC activity such as nitric oxide generation, vascular inflammation and antithrombotic creation. Among them, miRNA is crucial in controlling the EC senescence mechanism. Vascular performance is hampered by endothelial cells, which also causes tissues and organs to age.

***Address for Correspondence:** Lobmann Thole, Department of Diabetes, University of Bridgeport, Park Avenue, Bridgeport, USA, E-mail: lobmannthole@gmail.com*

Received: 02 January, 2023, Manuscript No. jdcm-23-90492; **Editor assigned:** 04 January, 2023, PreQC No. P-90492; **Reviewed:** 17 January, 2023, QC No. Q-90492; **Revised:** 23 January, 2023, Manuscript No. R-90492; **Published:** 31 January, 2023, DOI: 10.37421/2475-3211.2023.08.198

A miRNA may occasionally control several target genes to control the growth, death and chemotaxis of VSMCS. The best study to look into how miRNAs affect VSMC proliferation is the one that is currently underway. Our study's main objective is to learn more about the regulatory function of miRNA in VSMC multiplication. For instance, the production of cytokines, growth factor receptors and other membrane receptors can be controlled by miRNAs that regulate cell cycle progression regulators, miRNA-9, miRNA-34a, miRNA-141 and miRNA-22, or viral ecological integration site protein homolog genes can be blocked in VSMCs. cell cycle regulators that influence the process [7].

Conclusion

In particular diabetic nephropathy, lncRNA is crucial for the tissue regeneration caused by diabetes and its consequences. In diabetic nephropathy, CREB is highly expressed and strongly associated with the mouse lncRNA DLX6-AS1. The levels of DLX6-AS1 or DLX6os1 are markedly lowered by CREB silencing, which also lessens kidney damage. Hence, the selective inhibitor 666-15's inhibition of CREB reduces albuminuria and improves podocyte inflammatory infiltration.

The investigation of circRNA-miRNA patterns and interactions is the starting point for diabetic foot ulcers. To start, it was discovered that 279 mRNAs were up-regulated and 353 mRNAs were down-regulated in serum samples taken from patients with Diabetic Foot compared to diabetic patients in a study of circular RNA circRNA and messenger RNA mRNA expression profiling in patients with Diabetic Foot and diabetes mellitus.

Acknowledgement

None.

Conflict of interest

None.

References

1. Zhang, Yujia, Jing Zhang, Zhou Xu and Deju Zhang, et al. "Regulation of NcRNA-protein binding in diabetic foot." *Biomed Pharmacother* 160 (2023): 114361.
2. Li, Bo, Song Luan, Jing Chen, Yue Zhou and Tingting Wang, et al. "The MSC-derived exosomal lncRNA H19 promotes wound healing in diabetic foot ulcers by upregulating PTEN via MicroRNA-152-3p." *Mol Ther Nucleic Acids* 19 (2020): 814-826.
3. Peng, Wei-Xia, Pei-Xiang He, Li-Jun Liu and Ting Zhu, et al. "LncRNA GAS5 activates the HIF1A/VEGF pathway by binding to TAF15 to promote wound healing in diabetic foot ulcers." *Lab Invest* 101 (2021): 1071-1083.
4. Asadi, Gelayol, Fatemeh Rezaei Varmaziar, Mojgan Karimi and Misagh Rajabinejad, et al. "Determination of the transcriptional level of long non-coding RNA NEAT-1, downstream target microRNAs and genes targeted by microRNAs in diabetic neuropathy patients." *Immunol Lett* 232 (2021): 20-26.
5. Li, Bo, Yue Zhou, Jing Chen and Tingting Wang, et al. "Long noncoding RNA H19 acts as a miR-29b sponge to promote wound healing in diabetic foot ulcer." *FASEB J* 35 (2021): e20526.
6. Wan, Jiangbo and Bo Liu. "Construction of lncRNA-related ceRNA regulatory network in diabetic subdermal endothelial cells." *Bioengineered* 12 (2021): 2592-2602.
7. Xu, Yu-Xin, Sheng-Dan Pu, Xin Li and Zi-Wei Yu, et al. "Exosomal ncRNAs: Novel therapeutic target and biomarker for diabetic complications." *Pharmacol Res* (2022): 106135.

How to cite this article: Thole, Lobmann. "Controlling of Protein Binding by NcRNA in Diabetic Foot." *J Diabetic Complications Med* 8 (2023): 198.