Editorial
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miRNA as Potential Biomarkers for Cardiomyopathy

Pritun Pradhan*

Department of Biotechnology, Ravenshaw University, Odisha, India

Editorial

Heart failure implies that the heart works less effectively and not that the heart has quit working. Heart failure is caused by many conditions that damage the heart muscle. Coronary artery disease is where the arteries minimises the supply of oxygen and blood flow to the heart muscle. When the heart muscles are damaged from infections or alcohol or drug abuse rather than the minimal supply of oxygen or blood, is known as Cardiomyopathy. Conditions that exhaust the heart like hypertension, valve ailment, thyroid disease, kidney infection, diabetes, or hereditary heart defects may cause heart failure [1].

Cardiac diabetic diseases have become one of the most common causes of morbidity and mortality among diabetic patients. Specifically, diabetic cardiomyopathy (DCM) is described with a diastolic dysfunction and cardiac remodelling without indications of high blood pressure and coronary artery diseases [2]. It is difficult to identify DCM in its early stages because of its heterogeneity therefore, it is important to discover potential biomarkers in order to improve the prognosis of DCM since a strategy for prevention and treatment has not been found yet. As a possible alternative for treatment of this disease the use of miRNA as biomarkers has received a considerable attention in the biomedical field.

Altered level of micro-Ribonucleic acids (miRNAs) which modulates gene expression was found in the cardiomyocytes of experimental diabetes models. Among the group of noncoding RNA, miR-223 is associated with regulation of glucose transporter 4 (GLUT4) expressions in cardiomyocytes. Upregulation of miR-223 and downregulation of GLUT4 gets promoted by Insulin resistance [3]. miR-223 without affecting phosphoinositide 3-kinase signaling and AMP kinase activity might have a role in increasing nuclear factor IA expression. Considering that miRNAs act as stress response genes and specifically miR-223 has the ability to upregulate target genes such as GLUT4 in adult cardiomyocytes. So, this miR-223 might represent as valuable therapeutic target.

ERK1/2 derived-pathway which opposes oxidative stress-induced insulin resistance in cardiomyocytes are reported to be modulated by miRNAs as miRNA regulates the transcription extracellular signal-regulated kinases (ERK) in diabetic conditions. ERK MAPK activity requires further research as ROS are believed to activate ERKs through mechanisms which is might be helpful in preventing DCM [3].

The GLUT trafficking pathway and the exosome secretion pathway are inter connected during the absence of glucose. According to these interconnected molecular mechanisms it can be recommended that glucose uptake from the endothelium to cardiomyocytes could be regulated by short range exosome communication. This makes exosomes the potential biomarkers and they might also represent therapeutic targets or agents that could reverse the impaired insulin signaling observed in DCM [4].

To prevent the increased fatality rate due to DCM, it is necessary to find an early diagnosis of the disease for the asymptomatic patients. Biomarkers can be used to identify the early signs of DCM as the other advanced imaging techniques have been failed. The potential use of circulating miRNA as biomarker has been increased as the usage theory has been backed up by several studies done by research groups. The early detection of DCM by miRNA can help by treating the diabetes-derived diastolic dysfunctional patients with antidiabetic treatment and cardio prophylactic therapies [5].

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***Address for Correspondence:** Pradhan P, Department of Biotechnology, Ravenshaw University, Odisha, India, E-mail: pritun.leenu@gmail.com*

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