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MiRNA Biomarkers and Regulatory Networks Associated with Insulin Therapy Response and Beta Cell Function in Diabetes

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Introdution

Diabetes is a chronic metabolic disorder characterized by hyperglycemia resulting from either an absolute deficiency of insulin secretion or a combination of insulin resistance and insufficient insulin secretion. Insulin therapy is an essential treatment for patients with diabetes who have inadequate glycemic control. However, the response to insulin therapy varies among individuals, and predicting individual response to insulin therapy remains challenging. Recently, microRNAs (miRNAs) have emerged as potential biomarkers for insulin therapy response and diabetes management. MiRNAs are small, non-coding RNA molecules that play important roles in post-transcriptional gene regulation. They can modulate gene expression by binding to the 3' Untranslated Region (UTR) of target mRNAs and inhibiting their translation or promoting their degradation. Several studies have reported that miRNAs are dysregulated in diabetes and can be potential biomarkers for predicting insulin therapy response. One study investigated the association between baseline levels of miRNAs and short-term Insulin Therapy (IIT) response in patients with Type 2 Diabetes (T2D). The researchers measured the expression levels of miR-145 and miR-29c in Peripheral Blood Mononuclear cells (PBMCs) from 62 patients with T2D who received IIT for eight weeks. The results showed that higher baseline levels of miR-145 and miR-29c were associated with better short-term IIT response. The researchers also found that the expression levels of miR-145 and miR-29c were negatively correlated with Fasting Plasma Glucose (FPG) and HbA1c levels, indicating their potential as biomarkers for diabetes management [1].

Description

Another study investigated the changes in miRNA expression in response to insulin therapy in patients with T2D. The researchers measured the expression levels of miR-138, miR-192, miR-195, miR-320b, and let-7a in PBMCs from 15 patients with T2D who received IIT for 12 weeks. The results showed that the expression levels of miR-138, miR-192, miR-195, and miR-320b were significantly upregulated after insulin therapy, while the expression level of let-7a was downregulated. The researchers also found that the changes in miRNA expression correlated with changes in beta cell function and insulin sensitivity, indicating their potential as biomarkers for insulin therapy response [2].

Several studies have identified a network of miRNA-overtargeted genes that regulate cell death and proliferation in diabetes. For example, miR-29 family members, including miR-29a, miR-29b, and miR-29c, are involved in regulating extracellular matrix remodeling and fibrosis in diabetic nephropathy. MiR-192 and miR-215 are involved in regulating oxidative stress and inflammation in diabetic nephropathy. MiR-146a and miR-146b are involved in regulating inflammation and insulin resistance in adipose tissue. miRNAs have emerged as potential biomarkers for insulin therapy response and diabetes management. Baseline levels of miR-145 and miR-29c can predict short-term IIT response, while miR-138, miR-192, miR-195, miR-320b, and let-7a change in response to insulin therapy and correlate with changes in beta cell function and insulin sensitivity. A network of miRNA-overtargeted genes regulates cell death and proliferation in diabetes. Further studies are needed to validate these findings and explore the potential clinical applications of miRNAs in diabetes management [3].

MicroRNAs (miRNAs) are small, non-coding RNA molecules that play important roles in the regulation of gene expression. They have been implicated in various biological processes, including cell differentiation, proliferation, and apoptosis. In recent years, there has been growing interest in the role of miRNAs in the pathogenesis of diabetes and their potential as biomarkers for diabetes management. Beta cells in the pancreas play a critical role in glucose homeostasis by secreting insulin in response to elevated blood glucose levels. Dysfunction of beta cells leads to impaired insulin secretion and contributes to the development of diabetes. Several studies have reported that changes in miRNA expression correlate with changes in beta cell function and insulin sensitivity in diabetes [4].

One study investigated the changes in miRNA expression in response to hyperglycemia in vitro and in vivo. The researchers found that the expression levels of miR-7, miR-375, and miR-96 were decreased in beta cells exposed to high glucose levels. They also observed that the downregulation of miR-7 and miR-375 correlated with impaired insulin secretion and decreased beta cell proliferation. In vivo, they found that the expression levels of miR-7 and miR-375 were decreased in beta cells from diabetic mice and that the downregulation of these miRNAs correlated with impaired insulin secretion and decreased beta cell mass.

Another study investigated the role of miRNAs in regulating beta cell function and insulin sensitivity in patients with Type 2 Diabetes (T2D). The researchers measured the expression levels of miR-143, miR-146a, and miR-375 in pancreatic islets from 36 patients with T2D and 12 non-diabetic controls. They found that the expression levels of miR-143 and miR-146a were decreased in islets from patients with T2D, while the expression level of miR-375 was increased. They also observed that the downregulation of miR-143 and miR-146a correlated with decreased insulin secretion and impaired beta cell function, while the upregulation of miR-375 correlated with increased insulin resistance [5].

Conclusion

In addition to their role in regulating beta cell function and insulin sensitivity, miRNAs also play a role in regulating cell death and proliferation in diabetes. Several studies have identified a network of "miRNA-overtargeted" genes that are involved in regulating these processes. For example, miR-29 family members, including miR-29a, miR-29b, and miR-29c, are involved in regulating extracellular matrix remodeling and fibrosis in diabetic nephropathy. MiR-192 and miR-215 are involved in regulating oxidative stress and inflammation in diabetic nephropathy. MiR-146a and miR-146b are involved in regulating inflammation and insulin resistance in adipose tissue. Changes in miRNA expression correlate with changes in beta cell function and insulin sensitivity in diabetes. Dysregulation of miRNA expression can contribute to impaired insulin secretion and beta cell dysfunction in diabetes. A network of "miRNA-overtargeted" genes regulates cell death and proliferation in diabetes, providing potential targets for therapeutic intervention. Further studies are needed to validate these findings and explore the potential clinical applications of miRNAs in diabetes management.

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