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# Exploring the Role of miRNA in Regulating CD36 Fat Activity

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# Exploring the Role of miRNA in Regulating CD36 Fat Activity

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Obesity-driven metabolic complications such as diabetes and heart disease are major public health issues in the United States. To treat these issues, it is important to understand the regulation of proteins involved in fat metabolism. One such protein, CD36, has been extensively linked to fat metabolism in laboratory mice. However, its regulation after being transcribed is not well understood. MicroRNA molecules, miRNAs, can regulate protein activity by the process of mRNA silencing: binding to and destabilizing the mRNA, decreasing the level of protein transcribed. The mRNA transcript for CD36 has two untranslated regions (UTRs), one long and one short. Previous studies have linked lower levels of transcripts containing the long UTR to metabolic disease or dysfunction. In addition, the long UTR has been found to contain binding sites for the miRNAs studied, suggesting that mRNA silencing is possible. However, the results do not support this hypothesis. pmirGLO plasmids containing the long or short UTR were introduced into HEK293 cells via transfection, as were the appropriate miRNAs. The UTRs were first cloned into pmirGLO as a short string of nucleotides, then as a longer string which contained more of the surrounding wild-type sequence. CD36 activity was then tested via luciferase assay, and there was no consistent change in activity in any case. This result indicates the binding sites in the long UTR are competed for by another molecule in the cell, possibly the RNA-binding protein MBNL1. This method of regulation thus remains under investigation.