LncRNAH19 improves insulin resistance in skeletal muscle

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Abstract

Type 2 diabetes is one of the most common health problems, affecting over 450 million people worldwide. One major contributing factor is insulin resistance – a metabolic disorder causing elevated insulin, glucose, uric acid, and lipid levels in the blood. Skeletal muscle regulates glucose and lipid metabolism, making it an ideal target for new treatments for type 2 diabetes. A new study examined a potential regulator of lipid metabolism in skeletal muscle. LncRNA H19 belongs to a class of molecules called long non-coding RNAs (LncRNAs), master regulators that affect cell functions without encoding proteins. The researchers found that H19 was typically downregulated in a mouse model of diabetes, db/db. Overexpressing H19 in these mice inhibited lipid deposits in skeletal muscle and inhibited insulin resistance. In skeletal muscle cells, H19 overexpression increased cellular respiration and blocked lipid accumulation, while silencing H19 had opposite effects. A closer look revealed that H19 interacted with the protein hnRNPA1, resulting in increased translation of genes related to fatty acid oxidation. Although clinical studies are needed, the data suggest that H19 may be an ideal target to reverse insulin resistance and treat type 2 diabetes.