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Crosstalk mediated by Adipo-BRD7 gene therapy improves insulin resistance and improved liver and heart functions

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The most critical factor in the emergence of metabolic diseases is obesity. Malfunctioning of Adipose tissue instigates several pathophysiological conditions in the liver, kidney, and heart. BRDs are conserved protein modules that recognize acetyl-lysine motifs. BRD7 is a ubiquitously expressed 75kDa molecular weight protein, which is a member of the BRD family. BRD7 revealed in the multiple organs, including the liver, brain, heart, colon, lung, and skin, but the exact function of BRD7 is still not fully understood.

The study aimed to demonstrate the adipocyte-specific BRD7 gene therapy approach for preventing the development of obesity-induced pathophysiology of metabolic diseases. Adipocytes' specific expression of BRD7 was achieved using a lentiviral vector expressing BRD7 under the adiponectin vector (Ln-adipo- BRD7). The Mice fed a high-fat diet (HFD) developed adipocyte hypertrophy, fibrosis, increased inflammatory adipokines, decreased mitochondrial respiration, insulin resistance, vascular dysfunction, and impaired heart mitochondrial signaling. Furthermore, mice developed macrostetosis and lipid droplet hypertrophy in the liver hepatocytes. The dangerous effects prevented by the selective expression of BRD7 in adipocytes. Ln-adipose- BRD7-transfected mice on an HFD display increased cellular respiration, increased oxygen consumption, improved mitochondrial function, and decreased adipocyte size.

Moreover, PCR arrays confirmed that targeting adipocytes with BRD7overrides the genetic susceptibility of adiposopathy and correlated with restoration of anti-inflammatory, thermogenic and mitochondrial genes. Furthermore, crosstalk of adipocytes improves liver macrostetosis. Our data demonstrate that BRD-7gene therapy improved adipose tissue function and positively impacted distal organs like the liver and heart suggesting that specific targeting of BRD7 gene therapy is an attractive therapeutic approach for improving insulin sensitivity and metabolic activity and vascular function in metabolic syndrome.

Keywords: Metabolic Syndrome, BRD7, metabolism, mitochondria, type II diabetes.

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