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Overexpression of Ifnβ1 Gene in Mice Prevents Diet-Induced Obesity and its Complications in Mice

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The increased prevalence of obesity is raising global concerns, largely because of associated complications of the disease. In spite of significant research in the field, few therapies have been approved, and therapeutic outcomes remain insufficient. Therefore, alternative therapies are in need. Gene therapy has emerged as powerful strategy to tackle many of human diseases by modulation of gene expression levels. In this work, murine interferon beta 1 (IFNβ1)gene was overexpression using hydrodynamic gene delivery method in mice to prevent development of obesity. using diet induced obesity model in mice, we showed that IFNβ1 suppressed inflammation in adipose tissue adipose tissue hypertrophy. Also, it modulated cytokines expression toward anti-inflammatory interleukins. Importantly, IFNβ1 overexpression blocked weight gain without impacting food intake, and restored glucose homeostasis in treated mice. Together, these data suggest that IFNβ1 is a powerful anti-inflammatory cytokine with promising potential to treat inflammatory disorders. In addition, gene therapy is promising strategy to treat obesity and its related complications.

Biography:

Dr. Mohammad Alsaggar is an Assistant Professor of Pharmaceutical Biotechnology and Gene Therapy from Jordan. Mohammad received his BS in pharmacy in 2009 from the Jordan University of Science and Technology (Irbid, Jordan), and his PhD in Pharmaceutical and Biomedical Sciences from University of Georgia (Georgia, USA) in 2016. Mohammad's research focusses on the use of gene therapy strategies for management of cancer and obesity diseases. In addition, Mohammad is working on cancer metastasis modeling using the hydrodynamic cell delivery technique for the assessment of tumor microenvironment impacts on the behavior of tumor cells growing in different organs.