Co-stimulation of ILC2s by GITR Ameliorates Type 2 Diabetes

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Technology description

Market Opportunity

Because an estimated two-thirds of the U.S. population is overweight, and more than one-third of the population is pre-diabetic, and more than 25 million people have Type 2 Diabetes Mellitus (T2DM), effective mechanism-based therapeutic strategies are urgently needed to meet this market demand. Obesity is associated with inflammation and recruitment of inflammatory immune cells to adipose tissue to expand and remodel immune cells in visceral adipose tissue (VAT). This pro-inflammatory environment promotes insulin resistance and elevation of blood glucose levels, predisposing patients to the development of T2DM. ILC2s are immune cells that are present in VAT and express GITR. GITR acts as a costimulatory molecule that promotes the effector function and survival of ILC2s. Agonists that target GITR are a novel class of effective compounds to prevent and treat T2DM.

USC Solution

USC researchers have shown for the first time that ILC2s express GITR and can function to improve glucose tolerance and insulin sensitivity. They further characterized the signaling pathway of GITR as a biological target for treatment and prevention of T2DM. The findings provide new insight regarding GITR's role in ILC2s and introduce GITR engagement as a potentially novel therapeutic target for the treatment of T2DM.

Application area

A novel biological target to prevent and treat T2DM
GITR is an immune checkpoint molecule and member of the TNFR superfamily that is a well-characterized immune signaling molecule
In vivo results showed improved metabolic disturbances associated with T2DM
A method of preventing and treating T2DM with a GITR agonist to improve glucose tolerance and insulin sensitivity

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