Treating Type 2 Diabetes by Targeting CAP Protein in the Macrophage

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Technology description

UCSD researchers have found thata tissue specific gene deletion of CAP in the macrophage results in a similar protection from insulin resistance without limiting the exercise capacity in the host. By transplanting CAP deleted bone marrow to normal mice, macrophage-specific deletion of CAP was achieved. CAP deletion in the macrophage appeared to protect target tissues such as the muscle and liver from impaired insulin sensitivity without incapacitating the mouse. Thus, targeting CAP in the macrophage may be effective in treating patients in impaired glucose tolerance and/or type 2 diabetes. CAP (Cbl associated protein) is an adapter protein that is ubiquitously expressed. CAP acts in concert with Cbl to stimulate glucose uptake in skeletal muscle and adipose tissue as well as to induce the proliferation and migration of macrophages. Whole body CAP gene deletion in mice results in a protection from insulin resistance induced by high fat diet. However, exercise capacity is severely blunted in these mice.

Application area

This technology can potentially enable the development of therapeutics for insulin resistance and type 2 diabetes.

Institution

University of California, San Diego

Inventors

Lisa Lesniewski
Mohammad Pashmforoush
Jerrold Olefsky
Kenneth Chien

联系我们

叶先生

电 话：021-65679356

手 机：13414935137

邮 箱：yeyingsheng@zf-ym.com