Applications

- Obesity
- Diabetes
- Cancer
- Obstructive respiratory disorders (asthma, emphysema)
- Metabolic syndrome

Advantages

- Novel therapeutic
- Unique mechanism and target specificity

Inventors

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Market Need

It is estimated that approximately 25% of the USA adult population have insulin resistance, which further leads to obesity, type 2 diabetes and other metabolic disorders. Insulin resistance is a common metabolic derangement that contributes to the development of obesity-related comorbidities. Also, there is evidence that individuals diagnosed with diabetes/insulin resistant may increase their risk of developing certain types of cancers (i.e. kidney, pancreatic and colorectal)

Technology Summary

This technology describes the development of several novel IGFBP-3R specific monoclonal agonist antibodies. IGFBP-3 receptors are reported to have anti-inflammatory and anti-tumor properties in several human diseases including asthma, inflammatory disease and cancer. Dr. Oh has shown that his novel IGFBP-3 receptor agonist antibodies inhibit cell growth in NNKA cells (see figure). In addition, Dr. Oh found that IGFBP-3 agonists antibodies abrogate TNF-α-induced insulin-resistance. Taken together, this invention describes a new antitumor, anti-inflammatory signaling cascade and the therapeutic potential of IGFBP-3R agonist antibodies in cancer, metabolic syndrome, and obstructive respiratory disorders.
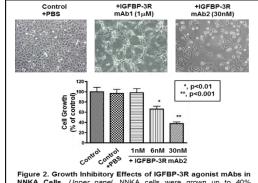

Figure 2. Growth Inhibitory Effects of IGFBP-3R agonist mAbs in NNKA Cells. *Upper panel*, NNKA cells were grown up to 40% confluency and treated with 1μM mAb1 or 30 nM mAb2 in 1% FBS congaing media for 3days. *Bottom panel*, Similarly NNKA cells were treated with different concentrations of IGFBP-3R agonist mAb for 3 daysand live cells were counted using the TC20 automated cell counter. n=3 in duplicate.
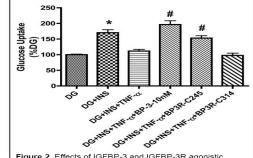

Figure 2. Effects of IGFBP-3 and IGFBP-3R agonistic monoclonal antibody (#C245) on TNF-a-induced inhibition of glucose uptake in C57BL/J mouse adipocytes. Treatment of 100nM insulin results in 180% increase of glucose uptake in adipocytes. while TNF-α (40ng/ml) treatment completely inhibits insulin-induced glucose uptake, co-treatment of IGFBP-3 (10nM) or IGFBP-3R agonistic antibody (#C245, 0.5 μM) abrogates TNF-α–induced insulin-resistance. Non-agonistic IGFBP-3R antibody (#C314) shows no insulin-sensitizing effect. *, <0.05, vs DG; #, < 0.05, vs DG+INS+TNF-α, n=3 in duplicate.

Technology Status

Patent pending: U.S. and foreign rights are available.

In vitro and *in vivo* data available.

This technology is available for licensing to industry for further development and commercialization.