Targeting the PTER/N-acetyltaurine pathway to treat obesity

Stanford scientists have discovered that treatment with the metabolite N-acetyltaurine leads to weight loss. They found that the removal of PTER, a key enzyme that regulates N-acetyltaurine metabolism, leads to N-acetyltaurine accumulation and a reduction in food intake. Therefore, modulating N-acetyltaurine metabolism may be an effective way of treating obesity and other metabolic disorders.

N-acetyltaurine is an abundant endogenous metabolite whose levels are dynamically regulated by diverse physiologic perturbations that increase taurine and/or acetate flux, including endurance exercise, alcohol consumption, and nutritional taurine supplementation. Interestingly, taurine supplementation has been reported to reduce mitochondrial redox stress, enhance exercise performance, and suppress body weight. However, the enzyme responsible for the biochemical interconversion of taurine and N-acetyltaurine had remained to be elucidated.

PTER, an orphan body mass index-associated enzyme, was shown to have N-acetyltransferase/hydrolase activity and be capable of converting taurine to N-acetyltaurine. Genetic ablation of PTER and/or pharmacological administration in mice resulted in suppressed body weight and adiposity. Consequently, PTER inhibitors or taurine analogs can potentially be used as a method of treatment for obesity.

Stage of Development:
Research - in-vivo

Applications

- Treatment of obesity and other metabolic disorders
- Energy balance and body weight control
Advantages

- Novel pathway of energy balance linked to taurine metabolism
- N-acetyltaurine or its analogs can be administered as a therapeutic
- PTER activity can be targeted as a method of increasing N-acetyltaurine levels

Publications


Innovators

- Wei Wei
- Jonathan Long

Licensing Contact

Kimberly Griffin

Technology Licensing and Strategic Alliances Manager

Email