Researchers at the University of South Florida have shown that certain inhibitors of Clk 1 and/or 4 induce the expression of UCP1 in differentiating pre-adipocytes. We predict that this marker of “beige” or “brite” adipocytes would enhance weight loss since beige adipocytes burn energy rather than store it as triglycerides.

Obesity contributes to increased heart disease, diabetes, and the likelihood of various other diseases over the age of 65. However, the battle of the bulge is an age old problem, that many have failed through diet and exercise alone. Although there are treatments such as gastric bypass surgery, appetite suppressants and blockers of lipid intake, there are no treatments that target white adipose tissue.

White adipose tissue is capable of massive expansion or contraction in response to altered energy balance. Adipogenesis of resident stem cells is important for this expansion and is related to obesity since it controls differentiation, lipogenesis, secretion of adipokines and glucose uptake in adipocytes. Thus, modulation of differentiation may be a promising new method for treating obesity.

Our study investigates how inhibition of Clk kinase activity in developing fat cells can alter the phenotype of cells toward beige or “brite” adipocytes. These adipocytes burn more energy and store less triglyceride. This may provide a promising therapeutic strategy for regulating white adipose tissue mass.

**ADVANTAGES:**
- Burn more energy
- Store less fat

**New Weight Loss Therapy**

**Different origins for distinct types of adipocyte**

*white and beige adipocytes derive from Pax7−/Myf5− cells via*

*Tech ID # 14B155*