BEACON IN VIVO A Eurofins Discovery Partner Lab

DIET-INDUCED OBESE (DIO) MICE

Male C57BL6/J mice administered a 60% kcal fat diet for 6-8 wks[1]

Both a high fat diet and a predisposed genetic background contribute to obesity in humans and mice. Male C57BL6/J mice administered a high fat diet demonstrate a propensity for weight gain, specifically adipose tissue expansion, leading to insulin resistance and dyslipidemia, and are often used to demonstrate efficacy of anti-obesogenic compounds. Other physiological changes include hepatic steatosis and increased levels of inflammatory markers in plasma and target tissues.

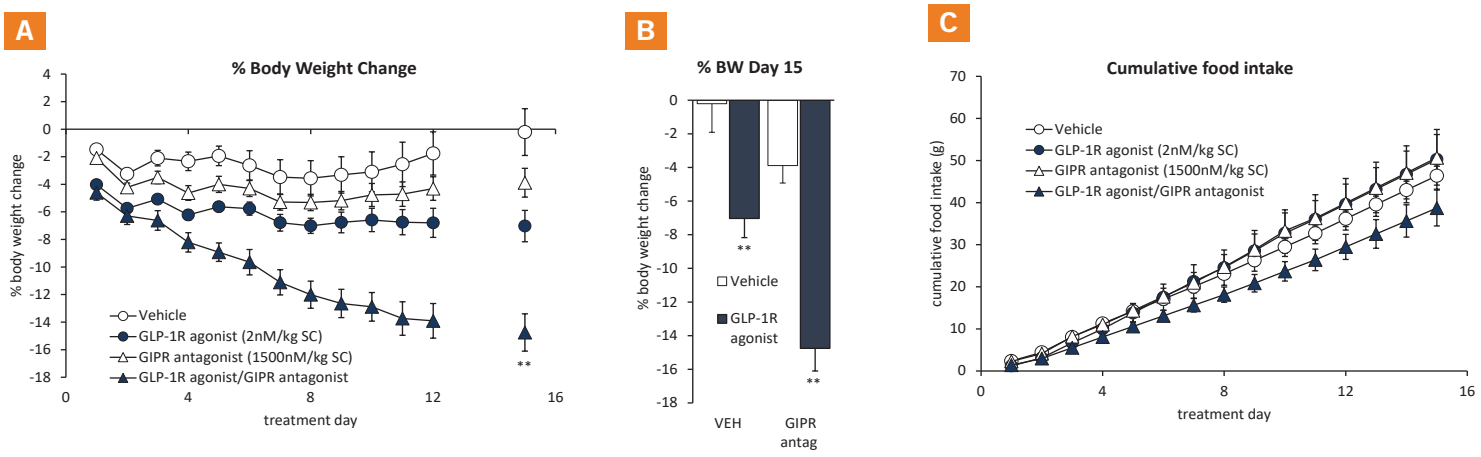
DOSING PARADIGM

- Daily dosing once the obese phenotype is established
- Possible routes of administration: PO, IV, IP, SC

CLINICAL ASSESSMENT

- Daily body weight
- Daily/cumulative food and water intake

SAMPLE DATA



Increased body weight and food intake in DIO mice is reversed by GLP-1R agonists and GIPR antagonists. **A)** Body weight is suppressed by a GLP-1R agonist/GIPR antagonist combination administered to DIO mice, as compared to vehicle controls **B)** Body weight at the terminus of the study. **C)** Cumulative food intake over the course of the study.

OPTIONAL ENDPOINTS

- Body weight/Body weight percentage
- Food intake
- Oral glucose tolerance test
- Serum or plasma hormone concentrations
- Serum or plasma lipid profiling
- Inflammatory markers
- H & E staining
- Hepatic Oil Red O staining
- c-Fos activation of brain nuclei
- In situ hybridization
- Immunohistochemistry

REFERENCE

Yang B, Gelfanov VM, El K, Chen A, Rohlfs R, DuBois B, Kruse Hansen AM, Perez-Tilve D, Knerr PJ, D'Alessio D, Campbell JE, Douros JD, Finan B. (2022). Discovery of a potent GIPR peptide antagonist that is effective in rodent and human systems. Mol Metab. 2022 Dec; 66:101638.

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