

Neuronal stimulation regulates appetite and glucose levels in mice

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Drugs that target signaling by glucagon-like peptide (GLP-1) in the gut and pancreas are commonly used to regulate hypoglycemia in patients with type II diabetes. However, these drugs are associated with a number of unpleasant side effects that may be linked to other targets of GLP-1 signaling. A subset of neurons in the brain also produces GLP-1 and related peptides, but how brain-mediated GLP-1 signaling influences metabolism and appetite is not clear.

This week in the *JCI*, a study led by Michael Scott at the University of Virginia explores how stimulation of GLP-1-producing neurons can control appetite and glucose levels in mice.

The researchers selectively excited GLP-1 neurons in the hypothalamus of mice and found that the stimulation reduced animals' [food intake](#) and suppressed the generation of glucose from energy reserves.

However, the stimulation's effects on glucose handling varied between lean and obese mice—in obese mice, exciting GLP-1 neurons suppressed [appetite](#) without changing [glucose levels](#).

The finding suggests that distinct pathways with different sensitivities to individual metabolic state may mediate the behavioral and physiological effects of GLP-1 signaling.

More information: Ronald P. Gaykema et al, Activation of murine pre-proglucagon-producing neurons reduces food intake and body

weight, *Journal of Clinical Investigation* (2017). [DOI: 10.1172/JCI81335](https://doi.org/10.1172/JCI81335)

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