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

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