

Brain's role in eating

Glucose-sensitive cells in the **hindbrain (1)** signal the body's neural networks to control appetite and metabolism. She studies how a shortage of glucose activates these networks and how their signaling mechanisms are altered in diabetics.

The **hypothalamus (2)** integrates the many internal and external signals influencing food intake and metabolism. One of Sue's goals is to find the neural pathways between the hindbrain's glucose-sensitive cells and how the hypothalamus processes their signals.

The **pituitary gland (3)** joins the hypothalamus in the timely secretion of hormones that affect growth and metabolism and help counteract a shortage of glucose.

The outer shell of the **adrenal gland (4)** secretes glucocorticoids, which promote the metabolism of fat, conserving glucose. The gland's core secretes adrenalin, which mobilizes glucose and fat from storage sites, boosting the availability of glucose for the brain.

The **vagus nerve (....)** is a superhighway of information between the brain and the digestive tract. Work in Bob Ritter's lab has shown that rats with damaged vagus nerves are not as satiated by fats and sugars.

In the brain, the vagus nerve uses glutamic acid as a neurotransmitter. Bob Ritter's group finds that blocking certain glutamic acid receptors in the hindbrain reduces satiety.

The gastrointestinal, or GI, tract has as many nerve cells as the spinal cord and secretes more hormones than any other organ system in the body.

We may think we stop eating because our stomach is full, but that feeling is only part of the process. More important, sensors along the GI tract tell the brain not only how much we've eaten but what—and when to stop eating.

Cells in the **duodenum (5)** secrete cholecystokinin to signal satiety. High amounts of dietary fat cause nerves along the intestine to become less sensitive to the hormone, possibly causing people who eat a lot of fat to not feel full as quickly.

The **pancreas's (6)** islets of Langerhans secrete insulin, which helps cells use glucose.

When blood sugar levels fall, the liver converts glycogen to glucose, which is needed to fuel the brain. Fat cells secrete leptin, which lowers appetite in people with low or average body weights. Some research shows on how reduced sensitivity to gut signals may contribute to obesity by reducing sensitivity to leptin.



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:: Anatomical, behavioral and physiological experimentation to identify metabolic events that control the onset of feeding behavior and the neural pathways responsible for monitoring these events.



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:: Neural and endocrine controls of appetite and body weight—how signals from the gastrointestinal tract and body fat are used by the brain to control food intake and body weight.