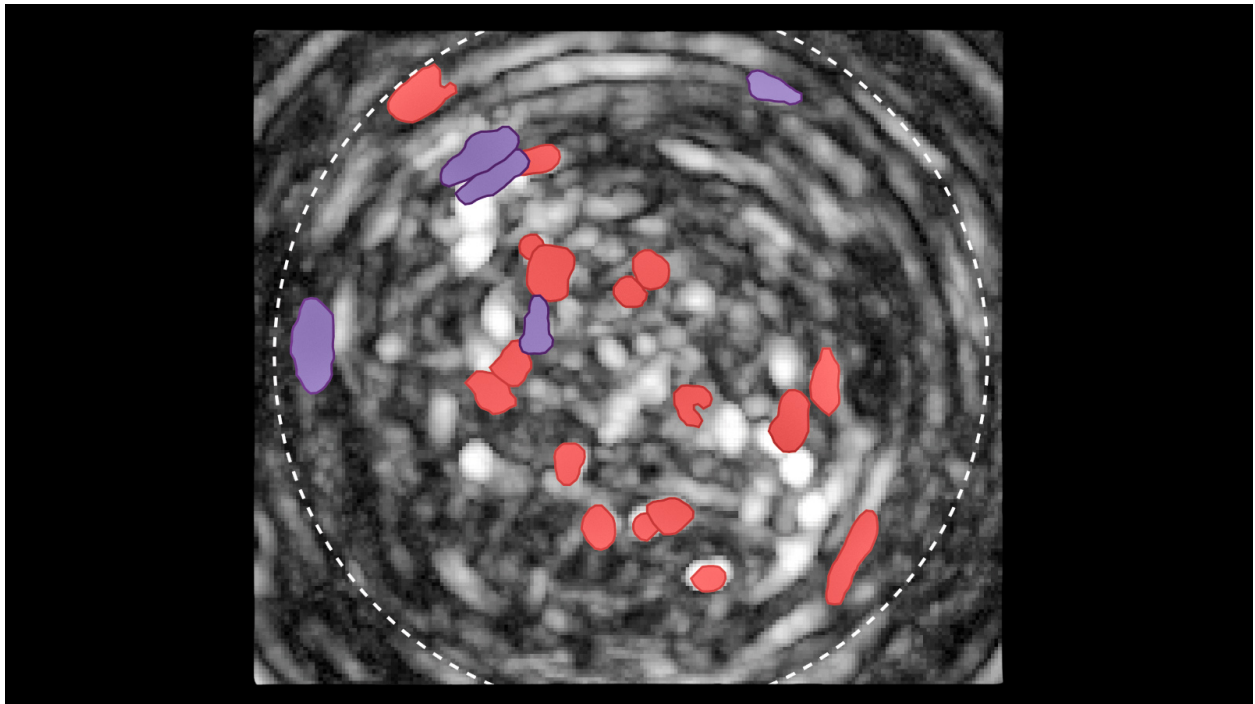


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“Brain Dial” for Consumption Found in Mice

Discovering ways to regulate this brain center may lead to new therapies for weight control and chemotherapy side effects



The image shows neurons in the bed nucleus of the stria terminalis that selectively respond to sweet (red) or to sodium (purple) to drive sweet or salt consumption, respectively (Li Wang and José Cánovas / Zuker lab / Columbia's Zuckerman Institute).

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CONTACT: News@zi.columbia.edu

NEW YORK — It's natural to crave sugar when you feel tired and want a boost of energy. Now scientists at Columbia University's Zuckerman Institute have linked a brain area in mice to the drive to consume not just sweets, but fats, salt and food. The findings show this area serves as a kind of dial that can amplify or repress consumption.

This discovery, detailed [today](#) in *Cell*, may inform novel treatments for both overeating and undereating. For instance, the results suggest that finding ways to modulate this brain circuit

may help treat people suffering from the severe loss of appetite and muscle wasting often seen in large numbers of chemotherapy patients.

"The relationship between something that stimulates the appetite, such as fat or sugar, and its capacity to drive us to consume it has been an open question in neuroscience," said [Charles S. Zuker](#), PhD, the study's corresponding author, a principal investigator at Columbia's Zuckerman Institute and a Howard Hughes Medical Institute investigator. "This work provides exciting new insights and identifies a brain center that orchestrates a unified control over consummatory behaviors."

Dr. Zuker and his colleagues started by analyzing the brain circuit in mice that responds to sweet tastes. Their goal was to explore how a sensation that can stimulate the appetite spurs the urge to consume.

The scientists first investigated the amygdala, the brain's emotion center, which also helps judge whether various sensations feel good or bad. They identified neurons, a type of brain cell, in the central amygdala that were activated by sweetness. Each of those neurons possessed branches that reached into a brain region called the bed nucleus of the stria terminalis (BNST), which prior work found was associated with feeding and responses to rewards.

When the researchers stimulated neurons connected to the BNST, they showed that mice that had recently fed until they were full could now be driven to continue to consume sweets. Conversely, suppressing the BNST neurons greatly suppressed sweet consumption even in animals that were very hungry.

The scientists went on to show that this brain region was associated with the urge to consume not just sweets, but also salt, fat, food and other substances.

"Our discovery far exceeded our expectations," said [Li Wang](#), PhD, the study's co-lead author and a postdoctoral fellow in the Zuker lab. "We did not expect this brain region to be so important and involved with such a broad range of consummatory behaviors in such a general way."

The scientists uncovered anatomical connections between the BNST and other parts of the brain that underscore its role in consumption. For example, they found links between the BNST and brain circuits sensing an animal's internal states, such as the need to eat when hungry or to consume salt when their body's salt levels become dangerously low.

"We now have a better understanding of how the brain integrates specific internal needs with sensory signals in order to elicit appropriate consummatory responses," said [José A. Cánovas](#), PhD, the study's co-lead author and a postdoctoral fellow in [the Zuker lab](#).

This discovery of a "brain dial" for consumption might one day help cancer patients undergoing chemotherapy, who often experience cachexia, a condition that can lead to a dangerous loss of appetite and weight. When mice were given a chemotherapy drug that can trigger a

cachexia-like state, the researchers found that stimulating the BNST could protect them from weight loss.

"Chemotherapy drugs are amazing in that they can kill cancer, but they also suppress the motivation to eat, which becomes a big problem," Dr. Wang said. "Maybe stimulating this brain area can help address this issue."

On the flip side, the researchers discovered that inhibiting the BNST resulted in substantial weight loss in mice. The scientists also found the weight-loss drug semaglutide (sold under brand names such as Ozempic and Wegovy) targets neurons in the BNST, shedding light on how it may work to help people reduce consumption.

"Taking semaglutide can lead to nausea and other negative side effects," Dr. Cánovas said. "Perhaps a better understanding of the BNST may lead to therapies that help suppress consumption without these effects."

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The [paper](#), "A Brain Center that Controls Consummatory Responses," was published in *Cell* on Sept. 10, 2025.

The full list of authors includes José A. Cánovas, Li Wang, Ahmed A.M. Mohamed, Larry F. Abbott and Charles S. Zuker.

José A. Cánovas, Li Wang and Charles S. Zuker are authors of a Columbia University patent application describing this work.