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Chemical in brain acts like a fuel gauge

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The concept that a drop in blood sugar triggers a craving for food is best understood just before lunchtime. But exactly how the process unfolds has proven difficult to explain, even on a full stomach.

Solving the puzzle would yield new insights in the fight against diabetes. Neuroscientists at the University of Southern California provide a partial answer in the July 4 issue of *The Journal of Neuroscience*.

Their study, highlighted by the journal on its news page, identifies a chemical that sends a “low blood sugar” message to a part of the brain that can do something about it.

The neurotransmitter norepinephrine travels from the hindbrain, which receives warnings of low glucose levels from the body, to the paraventricular hypothalamus, which authorizes the consumption of energy stores to replace the missing sugars.

The energy stores help for a while, but the end result is a feeling that the body is running on empty. Lunch, anyone"

While the study has few near-term clinical implications, except perhaps for diabetics with low blood sugar (hypoglycemia) from insulin overdoses, it is of fundamental interest in the field.

“There’s a huge interest in how the body senses glucose,” said Alan Watts, director of the Neuroscience Research Institute at USC and a co-author of the study.

“How that information is processed by the brain is really a hot current topic.”

Knowing how neurons relay hypoglycemia warnings is critical to understanding the overall glucose sensing mechanism in the brain, added corresponding author Arshad Khan, a research assistant professor at USC.

“That’s why I’m interested in this system, because it’s very poorly understood,” Khan said.

“If we don’t know how an automobile’s fuel system works to begin with, then how can we expect to fix one when it is not burning fuel appropriately""

In his study, Khan injected insulin in a group of animals to drop their blood sugar levels. In another group, he injected norepinephrine directly into the paraventricular nucleus.

Khan then compared brain tissue sections from both groups of animals and also examined blood samples for the presence of hormones released by paraventricular nucleus activity.

The same paraventricular neurons lit up in both sets of animals, and the animals displayed similar increases in hormone levels, suggesting that norepinephrine plays a role in transmitting the hypoglycemia warning.

“Norepinephrine is capable of activating these signals just like hypoglycemia does,” Khan said.

Khan then confirmed his findings with analogous experiments in vitro carried out in collaboration with neuroscientists at the University of California, Riverside.

Additional results from an ongoing study suggest that norepinephrine is not only sufficient but necessary for conveying hypoglycemia signals from the hindbrain, Khan added.

The current study, funded by the National Institute of Mental Health and the National Institute of Neurological Disorders and Stroke, was inspired by earlier work published in the journal Endocrinology by Sue Ritter of Washington State University.

In her studies, one of which was co-authored by Watts, Ritter showed that hypoglycemic animals lost their feeding and hormonal responses to hypoglycemia after damage to the norepinephrine pathways connecting the hindbrain to the hypothalamus.

Source: University of Southern California

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