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# Stress hormone impacts memory, learning in diabetic rodents

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Diabetes is known to impair the cognitive health of people, but now scientists have identified one potential mechanism underlying these learning and memory problems. A new National Institutes of Health (NIH) study in diabetic rodents finds that increased levels of a stress hormone produced by the adrenal gland disrupt the healthy functioning of the hippocampus, the region of the brain responsible for learning and short-term memory. Moreover, when levels of the adrenal glucocorticoid hormone corticosterone (also known as cortisol in humans) are returned to normal, the hippocampus recovers its ability to build new cells and regains the "plasticity" needed to compensate for injury and disease and adjust to change.

The study appears in the Feb. 17, 2008, issue of *Nature Neuroscience* and was conducted by the National Institute on Aging (NIA), part of the NIH. NIA's Mark Mattson, Ph.D., and colleagues in the Institute's Intramural Research Program performed the study with Alexis M. Stranahan, a graduate student at Princeton University in New Jersey.

"This research in animal models is intriguing, suggesting the possibility of novel approaches in preventing and treating cognitive impairment by maintaining normal levels of glucocorticoid," said Richard J. Hodes, M.D., NIA director. "Further study will provide a better understanding of the often complex interplay between the nervous system, hormones and cognitive health."

Cortisol production is controlled by the hypothalamic-pituitary axis

(HPA), a hormone-producing system involving the hypothalamus and pituitary gland in the brain and the adrenal gland located near the kidney. People with poorly controlled diabetes often have an overactive HPA axis and excessive cortisol produced by the adrenal gland. To study the interaction between elevated stress hormones and the hippocampal function, researchers tested the cognitive abilities and examined the brain tissue in animal models of rats with Type 1 diabetes (insulin deficient) and mice with Type 2 diabetes (insulin resistant).

Researchers found that diabetic animals in both models exhibited learning and memory deficits when cortisol levels were elevated due to impaired plasticity and declines in new cell growth. Returning the levels to normal, however, reversed the negative impact on the hippocampus and restored learning and memory.

“This advance in our understanding of the physiological changes caused by excessive production of cortisol may eventually play a role in preventing and treating cognitive decline in diabetes,” said Mattson, who heads the NIA’s Laboratory of Neurosciences. He and Stranahan explained these findings may also help explain the connection between stress-related mood disorders and diabetes found in human population studies.

Source: National Institute on Aging