HD also suffer from hyperglycemia and tissue wasting. A mutation in the huntingtin protein causes HD; the normal function of the huntingtin protein and the manner in which the mutant form damages nerve cells remain unknown. However, the mutant huntingtin is associated with abnormalities in cellular energy metabolism and deficits in the amounts of two proteins involved in cellular resistance to stress—brain-derived neurotrophic factor (BDNF) and heat-shock protein-70 (HSP-70)—suggesting that neurons in HD have a reduced ability to cope with stress.

Because of the alterations in energy metabolism associated with HD, Mark P. Mattson of the National Institute on Aging (Baltimore, MD) and co-workers tested the effect of an intermittent-fasting diet on mice carrying the defective huntingtin gene. HD mice deprived of food for 24 h every other day had delayed onset of motor dysfunction by about two weeks and lived 10–15% longer than HD mice on a normal diet. In addition, mice on the intermittent-fasting diet showed abatement of the diabetes-like hyperglycemia and weight loss associated with a hypermetabolic state (*Proc. Natl. Acad. Sci. USA*, 4 March).

The HD mice on the restrictive diet had higher levels of BDNF and HSP-70 in the striatum and cortex, which possibly protect the neuron cells from stress. Indeed, the HD mice fed the intermittent-fasting diet showed less atrophy of the striata. Increased BDNF concentrations were also involved in the improved glucose tolerance. The authors postulate that the ability of the intermittent-fasting diet to reduce brain pathology and increase the life span is mainly the result of beneficial effects on neurons, such as increasing their resistance to stress.

HD is a progressive disease for which there is no known cure. However, because early identification of affected individuals is possible with genetic testing, dietary restriction may be an effective means of delaying disease onset and increasing the life span of individuals who find they have inherited the disease-causing gene.

—Karen Zolnowski

Cold Confounds Behavior Test

The results of a commonly used test for learning and memory in mice may be inaccurate. A group of Finnish researchers report that the mice develop severe hypothermia, which impairs their performance on the test.

The Morris water maze (MWM) is the most commonly used test for measuring learning and memory in rodents. An investigator places the animal in a pool of water that contains a hidden escape platform. The animal's ability to find the platform depends on a number of variables, including memory, cognitive ability, motor coordination, and vision. Although the MWM was originally developed for use with rats, it is now in general use to assess learning and memory in mice, however, mice do poorly at this task in comparison with rats.

A group led by Jukka Puoliväli of the University of Kuopio (Kuopio, Finland) tested transgenic mice that carry Alzheimer's disease-associated mutations and their wild-type littermates. They found that five swims of 45 s each in 20 °C water with 30 s between trials caused a drop of as much as 9 °C in the animal's body temperature and an associated slowing of swimming speed (*Behav. Brain Res.*, published online, doi: 10.1016/S0166-4328(02)00369-8). Females were more susceptible to hypothermia than males, and the transgenic animals were more vulnerable than the controls. Using 24 °C water lessened the drop in body temperature but resulted in an increase in the number of mice that floated rather than actively searching for the platform. However, by increasing the inter-interval time from 30 s to 13 min, Puoliväli's group was able to prevent hypothermia and the slowing of swimming speed.

—Tanja Schub

Erratum

A number of features were inadvertently omitted from the chart in "Inhalation Anesthetics in Rodents", March 2003. Please note that Euthanex Corp. sells endotracheal tubes; Viking Medical supplies induction chambers and gas scavenger systems.