esearchers find link Alzheimer's disease

ASHINGTON (AP) — A defect genes that drive energy me- ism in cells may play a role in eimer's disease, new labora- studies have found.

esearchers say they have dis- red a mutation in the mito- dria DNA of cells in eimer's patients that may to a rise of a destructive ical, called oxygen-free rad- in the brain.

r. W. Davis Parker of the Uni- ity of Virginia and senior au- of a study to be published day in the Proceedings of the onal Academy of Sciences the findings suggest a link be- n the way brain cells process gy and Alzheimer's disease.

aven Khachaturian, a scientist the Alzheimer's Association, the new finding "is a poten- very important new piece of damental information" that ld lead to diagnostic tests for disease and a new under- ding of how it develops.

lzheimer's is a disorder that brain cells and causes a grad- decline in memory, a change rsonality and behavior, and, ntually, death. About 4 million Americans, mostly elderly, have been diagnosed with the disease. There is no known cure.

In the new study, researchers found that a mutation in cellular DNA leads to a failure of glucose processing in brain cells. This breakdown causes a rise in oxygen free radicals which could kill brain cells, a characteristic of Alzheimer's disease.

Mitochondria are structures that produce energy to make cells function. The mitochondria have their own DNA which is different from the DNA in the genes of each cell. The mitochondria DNA is only inherited from the mother.

To find the gene defect, the researchers removed mitochondria DNA from normal neuron cells and inserted DNA from Alzheimer's disease patients. The altered cells then functioned in cultures using the transplanted DNA.

The researchers found that the altered cells developed energy processing failures, leading to the excessive oxygen-free radicals. They found the mutation was in a mitochondria gene called cytochromic oxidase.

Gene offers insight to cause of shortness

► *Researchers are trying to determine how a gene called SHOX affects a person's height.*

NEWYORK (AP) — Why are really short people so short? It is largely a mystery. But now scientists have discovered a gene that might cause some cases.

Followup work might help scientists understand the biology of what determines a person's height and lead to new drugs that could make short children taller.

The study defined "short" as belonging to the shortest 2.5 percent of the population. That's a standard cutoff. For American men, that translates to about 5-foot-4 and under; for American women it's just under 5 feet.

Nobody knows exactly why most people in this group are so short. Genes clearly play a role, but few genes that stunt growth have been identified. Other factors like nutrition and environment also clearly enter in.

Most of these people are healthy, so medical conditions can explain only a small percentage.

The newly found gene is called SHOX. Researchers found it was damaged in one person out of a group of 91 healthy short people, or about 1 percent.

The mutation did not show up in 300 DNA samples from people of normal height.

The short person who showed the mutation was a girl who stood 35 inches tall at age 4.

On average, girls in the United States are about 40 inches tall on their fourth birthdays.

The researchers looked at DNA from several generations of the girl's family, which lived in Germany. They found the SHOX gene flaw in all five family members who met the criteria for shortness, but not in three family members who did not.

The SHOX gene is a "strong candidate" for causing some cases of shortness, researchers from Germany, the Netherlands and Japan write in the May issue of the journal Nature Genetics.

Dr. Judith Ross, a pediatric endocrinologist at Thomas Jefferson University in Philadelphia, called the results provocative. But it would take more work to prove that a mutated SHOX gene really makes people short, she said.

cientists say hormone burns fat inside cells

ASHINGTON (AP) — Leptin, a hormone acts in the brain to suppress appetite, also s obesity by burning up fat within cells, re- chers report.

r. Roger H. Unger of the University of Texas thwestern Medical Center at Dallas said ratory rats that are genetically altered to high levels of leptin lose all the fat in their es within days.

he researchers say a study to be published day in the Proceedings of the National Acad- of Sciences shows for the first time that leptin tive inside body cells and not just in the brain. There is a huge effect in cells on the metabo- of fatty acids," Unger said. "The fat never es the cells. It is burned up inside."

Fat was depleted in cells that normally store fat and in muscle, liver and pancreas cells, he said.

"Within seven days, there is no fat detectable in the body at all," Unger said.

Earlier studies of leptin had shown that the hormone acts in the brain to repress appetite. Rats with high levels of leptin were found to eat about half the food of normal rats.

Unger and his associates transferred genes that boosted the leptin levels in laboratory rats to about 20 times normal.

The animals' appetite and feeding habits were then carefully monitored.

For comparison, normal rats were fed exactly the amount of food as the high-leptin rats, Unger said.

Later, tissue of all of the rats was examined for fat content.

Unger said the normal rats lost fat, but only about one-half the amount shed by the leptin rodents. Also, the normal rats retained fat in their muscle cells.

The study suggested that leptin may be useful in the control of Type II diabetes, the form of the disease that often develops in older, overweight people. He said the rat study showed that by burning up fat in the cells, the excess leptin also lowered blood sugar and made the cells more sensitive to insulin, the hormone that controls sugar levels. Diabetes develops when the body stops making or becomes insensitive to insulin.