Gene That Causes Lou Gehrig's Disease Discovered

By Nicole Sherry

Researchers reported Thursday the discovery of the gene, which causes one form of Lou Gehrig's disease, a disorder which results in muscle deterioration, paralysis and death when the gene is defective.

H. Robert Horvitz, professor of biology, Robert H. Brown of the Massachusetts General Hospital, and Teepu Siddique of the Northwestern University Medical School led 20 scientists working at 13 research institutions across North America in the search for the gene.

The scientists examined genetic material from 18 patients affected by the familial version of Lou Gehrig's disease, also known as amyoprophic lateral sclerosis. In 13 patients, researchers found a mutation in the superoxide gene, which produces the enzyme superoxide dismutase. When this enzyme is healthy, it detoxifies free-radicals, byproducts of metabolism which can destroy cells.

Since there is already much known about how this gene operates, therapies for the disease may be possible sooner than researchers previously expected. However, there are still many questions researchers must answer before they can propose possible treatments.

"More questions remain unanswered than have been answered so far. The first questions we have is, what do the mutations do? Do they increase or decrease the activity of the enzyme?" Horvitz said.

Researchers' other immediate goals are to determine whether the other individuals with the familial version of ALS have defects in the superoxide gene and to look for other genes which may be involved in ALS, Horvitz said.

For those who have the familial version, the new discovery will have immediate effects. These individuals will now be able to have a genetic test which will tell them if they have a mutation in the superoxide gene. This will enable doctors to counsel patients on the likelihood that they will develop ALS and to do prenatal diagnosis.

Although only about 10 percent of the 20,000 to 30,000 people with Lou Gehrig's disease have the familial version, the symptoms are identical in both versions of ALS said, therefore, therapies developed through studying the superoxide gene will likely apply to many of the sporadic cases. In addition, researchers will investigate whether people who do not have the familial version have mutations in the superoxide gene, Horvitz said.

Scientists also hope that this new finding will aid the research of other diseases. Free radicals have been linked to tissue damage caused by aging, Parkinson's disease, Alzheimer's disease and others. Discovering the role of the free-radicals in Lou Gehrig's disease may lead to new treatments for these other conditions.

---

Check Out Our Super Selection of Lotus® Software.

Lotus® 1-2-3 R 2.4. Easy-to-use WYSIWYG graphical environment. Includes a library of 63 Smarticons for one-click shortcuts to the commands you use most. Includes the backsolver. File Viewer lets you preview files before retrieving them. Analyze numbers using a variety of graph types and test styles.

$99

Also Available: Lotus 1-2-3 R 1.1 for Windows®. $129.99

What a selection - Why I could flip my lid!

Work More Accurately with Hewlett Packard.


$299.99

Hewlett Packard HP 42S Scientific Calculator. With RPN system, 2 line by 22 dot display, statistics, real/imaginary functions. Solve for unknown. Integer, Complex numbers, base conversions, simultaneous equations.

$109.99

Also Available: Hewlett Packard HP 41CV with BASIC. $399.99

More questions remain unresolved.

By Nicole Sherry

Researchers reported Thursday the discovery of the gene, which causes one form of Lou Gehrig's disease, a disorder which results in muscle deterioration, paralysis and death when the gene is defective.

H. Robert Horvitz, professor of biology, Robert H. Brown of the Massachusetts General Hospital, and Teepu Siddique of the Northwestern University Medical School led 20 scientists working at 13 research institutions across North America in the search for the gene.

The scientists examined genetic material from 18 patients affected by the familial version of Lou Gehrig's disease, also known as amyoprophic lateral sclerosis. In 13 patients, researchers found a mutation in the superoxide gene, which produces the enzyme superoxide dismutase. When this enzyme is healthy, it detoxifies free-radicals, byproducts of metabolism which can destroy cells.

Since there is already much known about how this gene operates, therapies for the disease may be possible sooner than researchers previously expected. However, there are still many questions researchers must answer before they can propose possible treatments.

"More questions remain unanswered than have been answered so far. The first questions we have is, what do the mutations do? Do they increase or decrease the activity of the enzyme?" Horvitz said.

Researchers' other immediate goals are to determine whether the other individuals with the familial version of ALS have defects in the superoxide gene and to look for other genes which may be involved in ALS, Horvitz said.

For those who have the familial version, the new discovery will have immediate effects. These individuals will now be able to have a genetic test which will tell them if they have a mutation in the superoxide gene. This will enable doctors to counsel patients on the likelihood that they will develop ALS and to do prenatal diagnosis.

Although only about 10 percent of the 20,000 to 30,000 people with Lou Gehrig's disease have the familial version, the symptoms are identical in both versions of ALS said, therefore, therapies developed through studying the superoxide gene will likely apply to many of the sporadic cases. In addition, researchers will investigate whether people who do not have the familial version have mutations in the superoxide gene, Horvitz said.

Scientists also hope that this new finding will aid the research of other diseases. Free radicals have been linked to tissue damage caused by aging, Parkinson's disease, Alzheimer's disease and others. Discovering the role of the free-radicals in Lou Gehrig's disease may lead to new treatments for these other conditions.