HEALTH

HEALTH BRIEFS

GENETIC FAMILY TREE HELPS PINPOINT GENE LINKED TO SUDDEN DEATH IN YOUNG PEOPLE

HOUSTON — By studying seven generations of one family, researchers identified a gene responsible for sudden death in young people. The gene, when defective, can cause a difficult-to-diagnose heart disorder called arrhythmogenic right ventricular dysplasi (ARVD). Dr. Robert Roberts, a cardiologist at Baylor College of Medicine in Houston, and colleagues worked with Canadian researchers to locate the gene. They studied genes from more than 200 members of a North American family, including 10 who were diagnosed with ARVD and still living. After getting blood samples from 149 family members of the 10 living individuals, they linked the mutant gene that causes ARVD in the family to chromosome 3. ARVD affects one in 5,000 people and accounts for about 15 percent of sudden deaths in young people. In people with the disorder, the heart-muscle cells in the right ventricle die and are replaced by fat cells and fibrous tissue. The first symptom of ARVD is often death from abnormal heart rhythms. Other patients with the disorder might develop symptoms of heart failure, such as swelling of the hands and feet and shortness of breath, during their 30s and 40s. Roberts predicts that within two years, researchers will clone the ARVD gene and identify the specific mutation that causes the heart disorder. "This should make it possible to develop a simple blood test that can tell family members of individuals with ARVD whether they have inherited the faulty gene," Roberts said. "People at risk for ARVD could have automated defibrillators implanted to help protect against sudden bursts of potentially fatal abnormal heart rhythms."

CHROMOSOME DIFFERENCES BETWEEN CHIMPS AND HUMANS STUDIED

HOUSTON—Flipped chromosomes might offer clues to how chimpanzees and humans evolved into different species. Dr. David Nelson, a geneticist at Baylor College of Medicine in Houston, and graduate student Elizabeth Nickerson are studying chimpanzee chromosomes on which central segments of DNA, the genetic blueprint, are inverted. About 98.5 percent of DNA is the same in chimps and humans. The inverted segments could be involved in the 1.5 percent of DNA that is different. "To rearrange the chromosomes into an inverted position, the chromosomes would had to have been broken and rejoined," Nelson said. Such changes could have altered the way genes are expressed, or turned on, along the inverted segments, resulting in the production of proteins at different times or in different tissues and possibly influencing whether the creature became more ape-like or human. "But this explanation is speculative," Nelson said.

A PILL FOR GENE THERAPY

HOUSTON—A pill that activates a specially designed gene has potential for use in controlling gene therapy. A research team headed by Dr. Bert O'Malley at Baylor College of Medicine in Houston developed a gene that is sensitive to the drug mifepristone. The gene can be programmed to help produce human growth hormone. A pill containing mifepristone can "flip the switch" on this gene, causing the gene to bind to and turn on a gene responsible for making human growth hormone. Genes instruct the body to produce different amounts of protein in specific places at specific times. During gene therapy, genes from a laboratory are placed in the body to carry out a function that might help treat a health problem. "The effectiveness of gene-delivery systems in gene therapy depends on the ability to regulate the protein production of genes inserted into the body," said O'Malley, a cell biologist. "We found a simple way to control protein production with a pill containing mifepristone." O'Malley performed his research in mice. He hopes to begin clinical trials in humans next year. "If this technique is successful in humans, people with recurring disease that can be treated with gene therapy could take a pill to activate selected genes on an as-needed basis," O'Malley said.

Drug tested to control Parkinson's progression

Special to Sentinel-Voice

HOUSTON — A drug for Lou Gehrig's disease/ALS is being studied as a possible treatment to slow or stop the progression of Parkinson's disease.

"Current Parkinson's drugs treat only the symptoms," said Dr. Joseph Jankovic, director of the Parkinson's Disease Center and Movement Disorders Clinic at Baylor College of Medicine in Houston. "This drug, riluzole, might offer a way to block a brain chemical contributing to nerve cell death."

Parkinson's disease, a progressive neurodegenerative disorder, affects movement and is characterized by tremor, slowness of movement, rigid muscles and balance problems. It is caused by deterioration of the brain's nerve cells that release dopamine, a chemical necessary for normal movements.

"Parkinson's patients have increased activity in a brain region called the subthalamic nucleus," said Jankovic, a staff neurologist at The Methodist Hospital. "This overactive area produces too much glutamate, a brain chemical that in excess can overstimulate nerve cells and cause them to die."

Researchers hope that blocking the release of glutamate will slow or stop Parkinson's-related nerve cell death. Riluzole is known to prevent the release of glutamate.

"In studies of animals with experimentally-induced Parkinson's disease, the blocking of glutamate has lessened the disease," Jankovic said. In a pilot study at Baylor, riluzole appeared to be well tolerated by Parkinson's patients. However, the study was too small to determine if the drug was able to slow progression.

The current multi-center study will involve approximately 1,050 patients in 11 countries. Forty-three U.S. centers are participating.

"Each center will enroll patients with early-stage Parkinson's disease who have not been treated with the standard Parkinson's medication, L-dopa, or with other dopamine-related drugs," Jankovic said. "Symptoms of resting tremor, rigidity or slowness must have been present less than three years."

Early-stage patients taking L-dopa or other Parkinson's drugs can be considered for the study if they stop their medication one month prior to entering the study.

Participants will receive either riluzole or an inactive medication for two years and will have several clinical evaluations. A subset of the patients will have brain images, called PET scans, taken at the beginning and end of the study. The imaging will allow researchers to see the condition of the nerve terminals in the brain that release dopamine.

"Since Parkinson's patients normally lose these nerve terminals, we hope to see that patients on riluzole have greater preservation of those areas in the brain," Jankovic said.

Patients wanting study information can call 1-800-220-8610.

Minimal oral feedings may help premature infants

Special to Sentinel-Voice

HOUSTON — Feeding premature infants a specific amount of nutrients orally may help their digestive systems develop properly, according to preliminary findings from new studies done on neonatal animals.

Researchers at the USDA/ARS Children's Nutrition Research Center and the department of pediatrics at Baylor College of Medicine in Houston assessed the intestinal nutrient needs and then compared intravenous feeding alone with intravenous and minimal oral feeding in neonatal animals.

"Our studies showed that the neonatal intestine has a much higher requirement for protein than for carbohydrate," said Dr. Douglas Burrin, a USDA/ARS research physiologist. "We found giving neonatal pups up to 30 percent of nutrients orally helped their digestive systems grow properly. We hope this finding will one day help neonatologists determine the optimum composition and volume of oral feedings to give premature infants."

A premature infant is fed intravenously because their digestive system cannot process food. But without minimal oral feedings, an infant's digestive system will not grow properly and be underdeveloped when it is time to nurse. However, increasing the volume of oral feedings too rapidly can cause serious complications, and in some cases, death.

"This finding may minimize the number of premature infants who receive too many nutrients orally and contract potentially fatal diseases like necrotizing enterocolitis, and short bowel syndrome," said Burrin.

The average premature infant spends 20 days on intravenous feeding. This finding could cut that time by five days, and cut health care costs by thousands of dollars.

"We believe premature infants will one day benefit from this finding," said Burrin. "Using minimal oral feeding to supplement intravenous feeding can improve weight gain, shorten hospital stays, and help premature infants learn how to suck and swallow much faster."

A portion of this work was published in a recent issue of the Journal of Nutrition.