Study: Stuttering may be genetic

For many years, stuttering has been identified primarily as an emotionally based disorder — an audible sign of extreme anxiety or nervousness. Evidence compiled recently, however, indicates that stuttering is not only a physical abnormality but one that may be passed from one generation to the next.

A study of 555 stutterers and more than 2,000 of their close relatives "suggests that susceptibility to stuttering is genetically transmitted," reports Kenneth L. Kidd, professor of human genetics at Yale University School of Medicine. Results of the study also show that males appear to be more susceptible to the disorder than are females. Kidd presented his findings recently at the annual meeting of the American Association for the Advancement of Science.

The research confirms that stuttering "runs in families" — the frequency of stuttering among relatives of subjects is "much greater than the frequency in the population at large," Kidd says. Although this does not in itself constitute conclusive proof that stuttering is transmitted genetically, Kidd says he has ruled out two major psychological/cultural possibilities as modes of transmission.

First, the explanation that the child stutters as a way of *imitating* a parent could account for a maximum of only 10 percent of the cases, according to Kidd. "Ninety percent [of stutterers] had two fluent parents" at the time of birth, Kidd told SCIENCE NEWS. In many such cases, either the father or mother had at one time stuttered and recovered prior to the baby's birth and/or other relatives had been stutterers.

Second, the suggestion that familial anxiety can be internalized by the child to cause stuttering also may be ruled out, he says. Since anxiety is known to exacerbate the severity (measured, in this case, by frequency) of stuttering, any hypothesis pinpointing anxiety as a cause would mean that more severe stutterers would have a higher proportion of stuttering relatives, Kidd says. "But we found no such correlation," he says.

Further indication that the affliction is genetically transmitted, he says, is confirmed by the apparent "sex-specific" nature of stuttering. Statistics indicate that up to 5 percent of males, compared with just 2 percent of females, stutter for at least six months sometime during childhood. But beyond that, Kidd has found that although female stutterers are less common, they "have significantly more relatives who have ever stuttered." This suggests, he says, that "whatever contributes to susceptibility to stuttering, more of those factors would need to be present for a female to surpass the stuttering threshold than for a male to cross the threshold...if more factors of promoting stuttering are required to make a female stutter, families of female stutterers would have more of those factors, and hence, more stutterers" — which, apparently, they do.

"Definite proof" of a genetically transmitted disability, Kidd concedes, "is elusive. Yet all available evidence suggests that susceptibility to stuttering is genetically transmitted." He hypothesizes that the transmission may occur through either a single gene or "through many different genes [each] with a very tiny effect." In the latter case, the accumulation of a certain number of such genes above a critical point causes the individual to surpass the threshold and develop a stutter, according to the researcher.

Physiologically, stuttering involves a combination of factors that cause the person to "breathe abnormally or move his vocal cords inappropriately," says Martin Adams of the University of Houston. About 80 percent of the children who ever stutter recover before adulthood, Kidd estimates. Little research has been undertaken to determine what, if any, differences exist between those children and the other 20 percent who continue to stutter as adults. But, says Kidd, "once it persists, it is very hard to overcome."

Just how the Yale findings could help stuttering victims is unclear. "It probably won't mean anything immediately," Kidd says. "But if we can find out what the problem is [genetically and physiologically], it could have major implications for therapy and... possibly prevention." □

Lab-grown virus for a diarrhea vaccine

A major cause of serious diarrhea in infants and young children is a virus known as rotavirus. The virus is thought to be responsible for a large share of the millions of deaths annually from diarrhea, among children in developing countries. The first step toward an urgently needed rotavirus vaccine was reported in the Jan. 11 SCIENCE. A team headed by Richard G. Wyatt of the National Institute of Allergy and Infectious Diseases succeeded in growing a strain of human rotavirus under laboratory conditions. The keys to the success were patience and piglets. Virus taken directly from human stool specimens does not grow in cultured cells. So the scientists instead orally infected a newborn, germfree piglet with "type 2" rotavirus from feces of a young patient. They then transferred the virus from piglet to piglet for 11 passages, until the accumulated mutations allowed the virus to propagate in laboratory-grown monkey kidney cells. With the new procedure, the investigators plan to analyze the rotavirus genetic structure and hope to create a weakened virus suitable for use as a vaccine. □

A protein linked to photosynthesis

Joseph Priestly first twisted the key that unlocks the secrets of photosynthesis when he demonstrated in 1772 that a mouse in a closed jar could survive with a plant but not alone. Although Priestly did not realize what was happening, his experiments were the first to show that plants produce oxygen. Ironically, what was first observed about photosynthesis remains one of the last mechanisms of the energy-converting process to be fully understood by scientists. Now, however, two researchers may be hot on the trail of the protein responsible for oxygen evolution in photosynthesis.

Douglas Winget of the University of Cincinnati and Mark Spector of Cornell University have isolated from the thylakoids or inner chloroplast membranes of spinach a protein necessary for photosynthetic oxygen evolution. The protein has an apparent molecular weight of 65,000 and contains two manganese atoms per molecule. Whether this particular protein is the enzyme that catalyzes the photosynthetic splitting of water to generate oxygen remains to be seen. What has been observed, though, is that removal of the specific protein from thylakoid membranes stops oxygen evolution; addition of the same protein restores oxygen evolution.

The trail to the elusive protein involved in oxygen evolution has been blazed for some time. "So far, for many people who have tried to find such a protein, there have been some tantalizing suggestions of findings, but never before has anyone been able to pull out the protein and purify it," Winget says.

The success story of the two protein pinpointers, which will appear in the February PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES, is just one chapter in the continuing saga of enzyme isolation. The guidepost for enzyme purification is some measurement of what the enzyme should be doing — if the enzyme catalyzes the splitting of water, for example, oxygen evolution is measured. For soluble enzymes, or enzymes that float inside cells and organelles, the cell is simply broken, the enzyme extracted and the enzyme-specific activity measured. As the enzyme becomes increasingly purified, more specific activity can be measured.

But a membrane protein, such as the protein involved in photosynthetic oxygen evolution, poses more of a problem for enzyme isolators. Extracting the protein from its membrane often renders it inactive. "In order to see the activity of the membrane protein, one has to put it back in the membrane where it feels more at home," Spector says, explaining the rationale behind a procedure pioneered by other researchers — Yasuo Kagawa and