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Scientists Tie Two Additional Genes to Dyslexia

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One year after scientists discovered a gene whose flaw contributes to [dyslexia](#), two more such genes have now been identified.

The findings, described yesterday in Salt Lake City at a meeting of the American Society of Human [Genetics](#), support the idea that many people deemed simply lazy or stupid because of their severe reading problems may instead have a genetic disorder that interfered with the wiring of their brains before birth.

"I am ecstatic about this research," said Dr. Albert M. Galaburda of Harvard Medical School, a leading authority on developmental disorders who was not involved in the latest discoveries.

The findings, added to last year's, mean that for the first time, "we have a link between genes, brain development and a complex behavioral syndrome," Dr. Galaburda said.

As many as a dozen genes are probably involved in the disorder, he said, with each playing a role in the necessary migration of neurons as the brain's circuitry develops.

Researchers said a genetic test for dyslexia should be available within a year or less. Children in families that have a history of the disorder could then be tested, with a cheek swab, before they are exposed to reading instruction. If children carry a genetic risk, they could be placed in early intervention programs.

"Reading ability is a proxy for intelligence in American culture," said Dr. Sally E. Shaywitz of Yale University School of Medicine, a pediatrician who is an expert on dyslexia. The findings should help overcome stereotypes and get children the assistance they need, she said.

One of the genes newly linked to dyslexia is called DCDC2. It is active in reading centers in the human brain, said Dr. Jeffrey R. Gruen, a Yale geneticist who described the discovery at a news conference yesterday. Large deletions in a regulatory region of the gene were found in one of every five dyslexics tested, making it less active.

Fluent readers and dyslexics alike have the protein made by this gene, Dr. Gruen said, but it is less abundant in dyslexic brains. The function of the protein is not known, he said.

Rats also have the DCDC2 gene, so it should not be misconstrued as a spelling or reading gene, Dr. Gruen said. Rather, the gene supports the circuitry that underlies reading. When it was perturbed in unborn rats, he said, neurons migrated shorter distances, undercutting early brain development.

The second gene, called Robo1, was discovered by Dr. Juha Kere, a professor of molecular genetics at the Karolinska Institute in Stockholm. It is a developmental gene that guides connections, called axons, between the brain's two hemispheres, Dr. Kere said in an interview.

When the gene's activity is reduced, the number of finer connections, called dendrites, is reduced in brain areas involved in reading.

"You get the right signals going, but they do less well in terms of rapid processing," Dr. Kere said.

Many dyslexia experts believe that reading problems stem from an inability to process the fast sounds of spoken words.

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