

READINGrockets®

Dissecting Dyslexia

Genetic differences in the brain make learning to read a struggle for children with dyslexia. Luckily, most of our brain development occurs after we're born, when we interact with our environment. This means that the right teaching techniques can actually re-train the brain, especially when they happen early.

Genetic causes and educational solutions

Children who do not learn to read fluently by age 10 or 11 are often thought to be lacking in intelligence or motivation. In most cases, however, they are neither stupid nor lazy. They have dyslexia, a learning disability that makes it very difficult for them to understand written language, despite having a normal—or higher-than-normal—IQ. Depending on the diagnostic criteria used, dyslexia affects 5 to 17 percent of people in the United States.

Recent studies suggest that the reading difficulties people with dyslexia experience are caused by "faulty wiring" in certain areas of the brain, and there are indications that this faulty wiring is due, at least in part, to identifiable genetic defects or variations. Early screening for such variations would make it possible to provide timely and appropriate remedial training, some experts suggest, allowing children with dyslexia to overcome their disability and learn to read at an acceptable level.

Dyslexia gene identified

Scientists have known for decades that genetics plays a very important role in dyslexia, with estimates of heritability ranging from about 40 to 70 percent. Over the years, several candidate genes have emerged as possible contributors to dyslexia, but only recently have researchers been able to establish a strong link between one specific gene and this common learning disability.

In a study published in the November 22, 2005, issue of the Proceedings of the National Academy of Sciences, a research team led by Jeffrey Gruen, an associate professor at Yale School of Medicine, found several lines of evidence indicating that reading ability is influenced by a gene called DCDC2, which is located on chromosome 6. By studying 153 families with children who are dyslexic, the investigators were able to identify unique genetic patterns, or variations, within the DCDC2 gene that were strongly associated with dyslexia. One of the most interesting findings was the discovery of a deletion (a missing stretch of DNA) in DCDC2, which strongly correlated with severe reading disability.

In order to further investigate the role of DCDC2 in dyslexia, the researchers analyzed postmortem brain tissue samples and found that the DCDC2 gene is highly active in areas of the temporal cortex that are thought to be involved in reading.

The scientists also performed an experiment using 14-day-old rat embryos, some of which were injected with a substance that inhibits the expression of DCDC2. The rat embryos were then allowed to grow inside their mothers for another four days, after which their brains were removed for analysis. The investigators found that, while the control animals' brains developed normally and showed typical "neuronal migration," this migration was arrested in the brains of animals with reduced DCDC2 expression.

These results indicate that the DCDC2 gene plays a role in the development of dyslexia, Gruen says. He adds, however, that other genes are probably involved as well: "It's very likely that dyslexia is caused by a mixture of some DCDC2 alleles, as well as mixtures of alleles from some other genes."

Different brain regions used

Neuroimaging studies of children and adults with dyslexia consistently show that the underlying genetic variations that appear to be present in many of these individuals are manifested in observable differences in brain structure and function.

"Most of the available evidence points to the fact that the way the brains of children with dyslexia are wired is different from the typical brain organization in children who never experience difficulties in learning to read," says Panagiotis Simos, an associate professor at the University of Crete in Greece. Simos, in collaboration with scientists at the University of Texas Health Sciences Center at Houston, recently has conducted a series of studies that looked at brain activation patterns of children with dyslexia during various reading tasks.

Using magnetic source imaging (MSI), a technique that records tiny magnetic impulses generated by the electrical activity of neurons inside the brain, the researchers found that the brain circuit that children with dyslexia used when they attempted to read did not include an area (located in the left temporal lobe) that is typically used by nondyslexic readers. Children with dyslexia instead used the corresponding region in the right hemisphere, as well as certain areas in the frontal lobes, which are not normally used during reading.

These findings were in line with several previous studies that employed positron emission tomography (PET) or functional magnetic resonance imaging (fMRI) to compare brain activity between dyslexic and nondyslexic readers. But because MSI (also known as magnetoencephalography, or MEG,) can record not only the spatial arrangement of brain activity but its timing pattern as well, Simos and colleagues were also able to detect—in real time—very fast changes taking place in neuronal activity during the performance of various reading tasks.

When the investigators analyzed these "spatiotemporal activation" profiles of children with dyslexia, they found that even when these subjects used the same brain regions that nonimpaired readers typically use, the time it took for different areas to become activated, as well as the order in which they became active, was markedly different between the two groups. However, the results of their latest study indicate that, with appropriate training, these differences can be minimized or, in some cases, completely eliminated.

Intensive intervention

In their most recent study, Simos and colleagues gave 15 children with dyslexia, ages 8 and 9, 16 weeks of intensive training aimed at improving reading skills. Phonological awareness, the awareness of speech sounds, was taught for two hours per day during the first eight weeks. The second half of the program emphasized recognition of words, comprehension, and fluency for one hour per day.

The researchers compared the pattern of brain activation during reading tasks before and after the intervention and found that the intensive training resulted in increased activity in a region that is normally used by nondyslexic people. They also saw that the timing of the activity in the temporal and frontal cortices shifted to a pattern similar to the one seen in nonimpaired readers.

Tests of reading performance before and after the 16-week program showed that this apparent

normalization of brain activity was accompanied by significant improvements in word recognition and decoding, as well as fluency and comprehension.

Simos says these results show that even if the brains of children with dyslexia are functionally and/or anatomically different from those of other children, these differences do not prohibit the retraining or "rewiring" of the brain circuit for reading. He admits, however, that some children with the disorder may not be able to become good (or even average) readers, despite extensive training.

"Our recent work has shown that children whose brain circuit for reading rewires in such a way as to become very similar to the brains of nonimpaired readers are those who show the greatest benefits from remedial instruction," he says. "Children who continue to use compensatory brain circuits do not generally respond well to intervention."

What about adults?

Although some people become dyslexic during their adult years (as a result of a stroke, for instance), in most cases dyslexia is a developmental (i.e., childhood) disorder. Yet the majority of people with dyslexia are adults who have had it since childhood, points out Guinevere Eden, a neuroscientist at Georgetown University in Washington.

"One of the assumptions early on in neuroscience research, based on animal work, was that plasticity occurred only in the young brain," she says. Although it may be easier to "rewire" the brains of children, Eden 's research shows that there is plasticity in the adult brain, too.

In a study published in October 2004 in *Neuron*, Eden 's research team looked at brain activation patterns (using fMRI) in a group of adults with dyslexia while they were performing reading-related tasks. The investigators also tested a matched group of nondyslexic adults and found that, compared to these control subjects, individuals with dyslexia exhibited less activity in certain areas of the left side of their brains.

Half of the subjects who had the disorder were then given an eightweek, intensive (three hours per day) training program aimed at reinforcing the relationship between sounds and printed letters and words.

A comparison of fMRI recordings done before and after the intervention showed that the training sessions resulted in increased activity in the left hemisphere (in the same region the control subjects used), and in the right hemisphere as well, indicating the use of compensatory mechanisms by subjects with dyslexia. Tests also showed that the intervention program resulted in significant improvements in phonological awareness and paragraph reading accuracy.

Eden says these data suggest that remedial training can be beneficial for adults with dyslexia, too, although improvements in phonological awareness and reading accuracy do not necessarily translate into improvements in reading speed and/or comprehension. "But once you have improved phonological awareness and reading accuracy, you can start working on fluency," she says. "And once you bring up fluency, you probably improve comprehension."

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> **Articles from A-Z** > **Dyslexia**

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