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UW Office of News and Information

CONTACT: Joel Schwarz joels@u.washington.edu 206-543-2580

Dyslexic children use nearly five times the brain area as normal children while performing a simple language task, according to a new study by an interdisciplinary team of University of Washington researchers. The study shows for the first time that there are chemical differences in the brain function of dyslexic and non-dyslexic children.

The research, published in the current issue of the American Journal of Neuroradiology, also provides new evidence that dyslexia is a brain-based disorder. Dyslexia, a reading disorder, is the most common learning disability, affecting an estimated 5 percent to 15 percent of children.

The UW researchers, headed by developmental neuropsychologist Virginia Berninger and neurophysicist Todd Richards, used a novel noninvasive technique called proton echo-planar spectroscopic imaging (PEPSI) to explore the metabolic brain activity of six dyslexic and seven non-dyslexic boys during oral language tasks. The researchers used PEPSI to measure levels of brain lactate activation. Lactate is a by-product of energy metabolism produced by neurons when the brain is activated. Most, but not all, of this brain activity took place in the left anterior, or frontal, lobe of the brain, which is known to be one of the centers for expressive language function.

"The dyslexics were using 4.6 times as much area of the brain to do the same language task as the controls," said Richards, a professor of radiology. "This means their brains were working a lot harder and using more energy than the normal children."

"People often don't see how hard it is for dyslexic children to do a task that others do so effortlessly," added Berninger, a professor of educational psychology. "There are learning differences in children. We can't blame the schools or hold teachers accountable for teaching dyslexic children unless both teachers and the schools are given specialized training to deal with these children."

To capture the brain activity of the subjects, the UW research team used PEPSI, which is about 32 times faster than conventional magnetic resonance spectroscopy. Software developed at the UW enabled the researchers to detect specific brain chemicals.

The 13 boys in the study were between 8 and 13 years of age and the dyslexic and control groups were well-matched in age, IQ and head size, but not in reading skills. The controls were reading at a level above normal for their age and had a history of learning to read easily. The dyslexics had delayed reading skills and were reading well below average for their age. Their families also had a history of multi-generational dyslexia that was confirmed in a concurrent family genetics study.

The boys, fitted with earphones were asked to perform four tasks while their brains were being imaged. Three of the tests involved pairs of words and the fourth used pairs of musical tones.

In the language tests, the boys heard a series of word pairs that consisted of either two non-rhyming words such as "fly" and "church," two rhyming words such as "fly" and "eye," a non-rhyming real word and non-word such as "crow" and "treel," and a rhyming word and non-word such as "meal" and "treel." The boys were asked if the word pairs rhymed or didn't rhyme and if the pairs contained two real words or one real and one non-word. They responded by raising a hand to indicate yes or no. In the music test, the boys heard pairs of notes and raised one hand if they thought the notes were identical and the other if they believed them to be different.

While the dyslexic boys exhibited nearly five times more brain lactate activation during a language task that asked them to interpret the sounds of words, there was no difference in the two groups during the musical tone test. This means the difference between the dyslexics and the normal children relates to auditory language and not to nonlinguistic auditory function, according to Richards and Berninger.

They also said the findings are important because they shed new light on brain mechanisms involved with dyslexia at a developmental stage when it is still amenable to treatment. In addition, the functional differences between dyslexics and control subjects add evidence that dyslexia is a brain-based disorder.

"When a child has a brain-based disorder it is treatable, although it may not be curable, just as diabetes is," said Berninger. "Dyslexia is a lifelong condition, but dyslexics may learn to compensate for it later in life. We know dyslexia is a genetic and neurological disorder. It is not brain damage. Dyslexics often have enormous talents in other parts of their brain and shine in many fields. Einstein was a dyslexic, and so were inventor Thomas Edison and financier Charles Schwab.

"While it is useful to show there are brain differences between dyslexic and nondyslexic children, considerably more research is needed to precisely define the chemical and neurological markers of dyslexia. What we found is a metabolic marker, but there could be a more fundamental cause. We need to understand the molecular and neural mechanisms underlying dyslexia," she added.

Other members of the UW research team and co-authors of the study are Stephen Dager, professor of psychiatry and behavioral science; David Corina, assistant professor of psychology; Cecil Hayes, professor of radiology; Robert Abbott, professor of educational psychology; Susanne Craft, adjunct associate professor of psychiatry and behavior science; Dennis Shaw, assistant professor of radiology; and Stefan Posse, affiliate assistant professor of radiology. In addition, UW doctoral students Sandra Serafini, Aaron Heide, Keith Steury and Wayne Strauss participated in the research.

The study, part of a wider UW effort to understand the basis of dyslexia and develop treatments for it, was funded by the National Institute of Child Health and Human Development.

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For more information, contact Berninger at (206) 616-6372 or vwb@u.washington

## or Richards at (206) 598-6725 or toddr@u.washington.edu

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