Dyslexia-linked genetic variant decreases midline crossing of auditory pathways

February 1 2012

Finnish scientists have found that a rare dyslexia-linked genetic variant of the ROBO1 gene decreases normal crossing of auditory pathways in the human brain. The weaker the expression of the gene is, the more abnormal is the midline crossing. The results link, for the first time, a dyslexia-susceptibility gene to a specific sensory function of the human brain. Credit: Satu Lamminmaki

Finnish scientists have found that a rare dyslexia-linked genetic variant of the ROBO1 gene decreases normal crossing of auditory pathways in the human brain. The weaker the expression of the gene is, the more abnormal is the midline crossing. The results link, for the first time, a dyslexia-susceptibility gene to a specific sensory function of the human brain. This collaborative study between Aalto University and University

of Helsinki in Finland and the Karolinska Insitutet in Sweden was published in the *Journal of Neuroscience*.

According to previous animal studies, dysfunction of the Robo1 gene prevents normal midline crossing of neurons during fetal development. Humans with totally dysfunctional ROBO1 gene have not been found. However, dyslexic individuals in a large Finnish family have inherited one poorly functioning copy of the ROBO1 gene. This association between ROBO1 and dyslexia was found already in 2005.

In the present study, the scientists quantified the functional crossing of auditory pathways in ten members of this family, applying a sensitive method based on the recording of weak magnetic fields of the brain (magnetoencephalo¬graphy, MEG). The functional crossing of auditory pathways was significantly weakened in individuals who carried the dyslexia-linked version of the ROBO1 gene.

Dyslexia is the most common learning disability, affecting roughly 10 per cent of the population in most countries.

Provided by Aalto University