Unexpected function of dyslexia gene

June 20 2011

(Medical Xpress) -- Scientists at Karolinska Institutet have discovered that a gene linked to dyslexia has a surprising biological function: it controls cilia, the antenna-like projections that cells use to communicate.

Dyslexia is largely hereditary and linked to a number of genes, the functions of which are, however, largely unknown. This present study from Karolinska Institutet and Helsinki University now shows that one of these genes, DCDC2, is involved in regulating the signalling of cilia in brain neurons.

"Our discovery presents us with a possible new neurobiological mechanism for dyslexia," says Professor Juha Kere, who co-led the study with Professor Eero Castr?n of Helsinki University.

Cilia are hair-like structures that project from the surface of most cells. Their purpose has long remained something of a mystery, but recent research has revealed that the cells use them to communicate and that they play a crucial part in the development of the body's organs.

These results tie into previous research in mice showing that DCDC2 and two other dyslexia genes are involved in cell migration, a process by which nerve cells are moved to their correct location in the brain during embryonic development.

"The cilia are important parts of the machinery that controls cell migration," says Professor Kere. "Just what they do and how it could result in dyslexia are interesting questions that will be given further

study."

The new findings, which are presented in the scientific journal *PLoS ONE* show that DCDC2 governs the length of the cilia and activates two different signal systems in the cell, depending on the degree of gene activity. When the human variant of the gene was transferred to nerve cells in the roundworm C. elegans, it gave rise to unusual neural projections exclusively in ciliated cells.

Ciliary dysfunction in different organs has been associated with a wide range of disorders from rare genetic diseases such as polycystic kidney disease and Kartagener's syndrome, to diabetes, obesity and schizophrenia.

More information: Satu Massinen, Marie-Estelle Hokkanen, Hans Matsson, Kristiina Tammimies, Isabel Tapia-Paez, Vanina Dahlström-Heuser, Juha Kuja-Panula, Jan Burghoorn, Kristian E. Jeppsson, Peter Swoboda, Myriam Peyrard-Janvid, Rune Toftgard, Eero Castrén, Juha Kere, Increased expression of the dyslexia candidate gene DCDC2 affects length and signaling of primary cilia in neurons, *PLoS ONE* 16 June 2011; 6(6): e20580.doi:10.1371/journal.pone.0020580

Provided by Karolinska Institutet