Matrix Genomics, Elizabeth Corder, PhD - Gene Variation in the LRRK2 Gene and High Risk for Parkinson's Disease

Researchers at the National Institute on Aging, University College London, and Matrix Genomics, Inc. have identified a concise genetic signature found among a third of Parkinson's disease (PD) patients - but at very low frequency in the general population. The signature is found in the LRRK2 gene located on chromosome 12. The work was led by Elizabeth Corder, PhD, Scientific director at Matrix Genomics, Inc.

Santa Fe, NM (PRWEB) June 5, 2009 -- Researchers at the National Institute on Aging, University College London, and Matrix Genomics, Inc. have identified a concise genetic signature found among a third of Parkinson's disease (PD) patients - but at very low frequency in the general population. The signature is found in the LRRK2 gene located on chromosome 12. The work was led by Elizabeth H. Corder, PhD, Scientific Director at Matrix Genomics.

Parkinson's disease is the second most common neurodegenerative disorder in the Western world, and a major cause of disability and distress among older Americans. Family studies have long indicated that Parkinson's is a genetic disorder when certain mutations are inherited. However, specific genetic factors relevant to the general population have been elusive.

The PD gene is called leucine risk repeat kinase 2 (LRRK2), which encodes a protein called dardarin, derived from the Basque word dardara, meaning tremor. Mutations in LRRK2 are a common cause of familial Parkinson's disease.

This study published in the Annals of Human Genetics describes a combination of four gene variants found in a third of Parkinson's cases, but infrequent in the population. Thus the presence or absence of this signature can be used as a genetic test for Parkinson's disease (Patent pending).

Earlier work had identified each of the four variants as being associated with PD risk, but did not combine the information to make a specific risk signature.

Genetic testing using this approach is expected to identify a third of persons at very high risk. It will not identify other genetic factors or level of risk due to environmental exposures, such as pesticides, and lifestyle. Thus the absence of this risk signature does not guarantee low risk.

This finding helps us understand what causes PD and could lead to new and more effective avenues for prevention and treatment. The advance is expected to identify individuals at greatest risk for PD before symptoms arise, when therapies and lifestyle changes might be most effective in slowing disease progression.

In 1993, Dr. Corder was the lead author on the Science article that described how risk for Alzheimer's disease multiplied according to the number of copies of the apolipoprotein E allele 4 inherited from parents. This finding has been replicated in hundreds of studies and remains the one established genetic risk factor for Alzheimer's disease, and the prototype for investigating common gene variants for common disorders.

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