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Diagnosis and genetics of Parkinson's disease: State of the art in Asia



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Breakthroughs in genetics over the last decade have radically advanced our understanding of the etiological basis of Parkinson's disease (PD). Although much research remains to be done, the main genetic causes of this neurodegenerative disorder are now partially unraveled, allowing us to feel more confident that our knowledge about the genetic architecture of PD will continue to increase exponentially. How and when these discoveries will be introduced into general clinical practice, however, remains uncertain. Moreover, a number of genetic parkinsonian conditions have been recognized that share some features with the clinical syndromes of atypical parkinsonism. This has made differential diagnosis difficult for practitioners. Therefore, the information makes clinicians aware of these genetic disorders as well as investigative features that may help in diagnosing these disorders. In contrast, environmental factors like neurotoxins such as MPTP and life styles are also risks for developing PD. Indeed, increased westernization with Japanese migration to the U. S. in the early 20th century is thought to have altered the risk of cardiovascular disease. Like cardiovascular diseases, whether similar effects include changes in the risk of PD is not clear. Recently, the Honolulu-Asia Aging Study reported the relations between environmental, life-style, and physical attributes and the incidence of PD. The study indicated that overall incidence (7.1/10,000 person-years) was generally higher than in Asia and similar to rates observed in Europe and the U.S. Thus, not only genetic factors but also several environmental, life-styles, and physical attributes contribute the onset of this disease. In the other word, genetic-environmental interaction is a key event for PD. In this presentation, we review the differential diagnosis for PD and atypical parkinsonism and genetics of PD.

《略歴》

Dr. Hattori received MD degree from Juntendo University in 1985. He became a resident in Neurology of Juntendo Hospital. He was admitted to a graduate school of Juntendo University in 1990. In that period he was trained for molecular biology at Department of Biomedical Chemistry of Nagoya University from 1990 to 1993. He was appointed as an associate professor in 2003. Finally, Dr. Hattori became the professor and chairman of Neurology of Juntendo University School of Medicine in 2006.

He has been interested in the etiology and pathogenesis of Parkinson's disease (PD) and he found decrease in the amount of complex I in the substantia nigra of Parkinson's disease patients. Then he and his collaborators identified the disease gene for an autosomal recessive form of young onset familial PD, and named the gene as "parkin". This is the second form of familial PD in which the disease gene was identified. And they found that the gene product, parkin is direct linked to ubiquitin-proteasome pathway as an ubiquitin ligase. This discovery suggested that protein degradation system is involved in the pathogenesis of not only monogenic PD but also sporadic form.