Symposium report

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ISN-JSN Joint symposium entitled "Transdisciplinary strategies to neurodegenerative disease" was held on September 29th, 2014 in the annual meeting of the Japanese Society of Neurochemistry.

Three distinguished researchers of different research fields gave talks on their recent research findings.

The first speaker, **Dr. Takeshi Morihara** from Department of Psychiatry, Osaka University School of Medicine, Japan, talked about identification of KLC1E as an A beta accumulation modifier in Altzheimer's disease: a novel approach to complex disease by transcriptome analysis of distinct mouse strains. Based on the findings that DBA mouse strain has lower expression level of A beta 40 and 42, which are the main causal proteins of Alzheimer's disease pathology, Dr. Morihara fished out kinesin light chain 1 (KLC1) gene using microarray analyses. KLC1 gene expression was indeed lower in the Alzheimer's disease patients. Dr. Morihara concluded that rather than tedious genome-wide association study (GWAS), trascriptomic analyses are powerful strategies.

The second speaker, **Dr. Sylvie Bellville** from Psychology Department of University Montreal, presented her recent data about human functional MRI. She found that the patients with mild cognitive impairments showed paradoxical hyper-activities in several brain regions, which will be silenced in the later stages with severe cognitive dysfunction. She also showed that relatively simple cognitive tasks named MEMO could enhance the brain regional activities in mild cognitive impaired patients. She concluded that there are neuronal plastic events in the Alzheimer's disease progression and in the memory-task training. These plasticities can be targets of therapeutic interventions.

The third speaker, **Dr. Zu-Lin Chen** from Laboratory of Neurobiology and Genetics, The Rockefeller University, talked about laminin gamma-knockout mice, in which neuronal apoptosis induced by kainic acid (KA) was inhibited. The mechanisms underlying this phenomenon were that the fragments of laminin gamma upregulated KA receptor. KA receptor activation led to excitotoxic cell death of neurons. Taken together, laminin gamma subunit has neuro-toxic effects. Laminin gamma-conditional knockout in glial cells showed severe hemorrhage in the striatum. Dr. Chen showed that this conditional KO affects development and integrity of smooth muscle cells of brain arteries. Hemorrhage was due to the functional impairments of brain arteries.

For each talk, the audience actively participated in discussion by asking questions. Through these active discussions, we think these three speakers promoted the audience's understandings on the mechanisms and therapeutic strategies of neurodegenerative disease, especially of Alzheimer's disease.