Lecture title: Schizophrenia and brain development

Lecture 1: Schizophrenia - a general overview
Schizophrenia is a debilitating mental condition (syndrome, a group of related disorders) that affects 1% of the population. Despite intensive study, its molecular etiology remains enigmatic. Like many common diseases, schizophrenia is multifactorial in origin, with both genetic and environmental contributions likely playing an important role in the manifestation of symptoms. Recent advances based on pharmacological studies, brain imaging analyses, and genetic research are now converging on tantalizing leads that point to a central role for several neurotransmitters, including dopamine and glutamate, that are impacted by neurodevelopmental defects reflecting disease-related genetic aberrations.

Lecture 2: Frontline of basic/translational neuroscience for schizophrenia
In this lecture, the frontline of basic/translational neuroscience for schizophrenia will be overviewed from five key aspects: 1) pathogenesis and pathophysiology; 2) disease pathways involving major risk factors; 3) Modeling-from causes to phenotypes; 4) stress sensing and susceptibility; and 5) Biomarkers.

Lecture 3: Gene-environmental interaction in the pathology of congenital cytomegalovirus brain anomaly
Schizophrenia is caused by a combination of genetic and environmental factors. Among environmental factors, birth complications and congenital infection of viruses, such as cytomegalovirus (CMV), are the most promising. Some cases of congenital CMV infection are known to result in aberrant corticogenesis and brain anomaly. Our recent unpublished study indicates that risk factors for schizophrenia play a role in this pathology by interacting with a specific protein from CMV. This study will be introduced as a prototype of molecular study for schizophrenia and related disorders from neurodevelopmental viewpoint.

References (relevant to this lecture)
PCM1 is recruited to the centrosome by the cooperative action of DISC1 and BBS4 and is a candidate for psychiatric illness. Arch. Gen. Psychiatry in press (2008)
A schizophrenia-associated mutation of DISC1 perturbs cerebral cortex development.

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Schizophrenia: diverse approaches to a complex disease.
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Increased apoptosis of Huntingtonís disease lymphoblasts associated with repeat length-dependent mitochondrial depolarization.

**Brief Bio (Akira Sawa, M.D.)**

Akira Sawa received MD from University of Tokyo in 1990. He stayed the University and University hospital, major in psychiatry (1990-1996). Then, he switched his gear from clinical to more basic neuroscience, having postdoctoral research training under Solomon Snyder in Department of Neuroscience at Johns Hopkins University (1996-2001). In 2001, he started his own laboratory in Departments of Psychiatry and Neuroscience at Johns Hopkins. In 2006, he appointed to the Director, Program in Molecular Psychiatry, a translational research program for major mental illnesses, with several junior faculty members from both clinical and basic science backgrounds, in Departments of Psychiatry and Neuroscience at Johns Hopkins. From 2008, he serves as the director by a center grant from the NIMH Silvio O. Conte Center for the Translational Neuroscience of Mental Disorders.