



Wed., **20 November**, 4:00pm



Jukhyun Bio Auditorium(RM.121)

English

Role of cell cycle dependent kinase inhibitors to stimulate neural and glial differentiations in the developing brain



Speaker | Yoichi Kosodo



Affiliation | Korea Brain Research Institute



Host | Prof. Mi-Ryoung Song

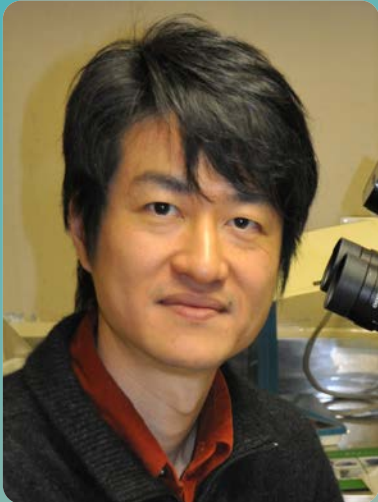


광주과학기술원 생명과학부

Gwangju Institute of Science and Technology School of Life Sciences

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 Yoichi Kosodo, Ph.D.

Education/Experience

1991.04-1996.03

B.Sc. in Engineering, Dept. of Chemistry and Biotechnology, University of Tokyo

1996.04-1998.03

M.Sc. in Life Sciences, Dept. of Biotechnology, University of Tokyo

1998.04-2001.03

Ph.D. in Life Sciences, Laboratory of Koji Yoda, Dept. of Biotechnology, University of Tokyo

2001.07-2010.08

Postdoctoral Fellow, Lab. of Wieland B. Huttner, Max-Planck-Institute of Molecular Cell Biology and Genetics (MPI-CBG), Dresden, Germany

2005.09-2010.08

Research Scientist, Lab. for Cell Asymmetry, RIKEN Center for Developmental Biology, Kobe, Japan

2010.09-2015.03

Associate Professor, Department of Anatomy, Kawasaki Medical School, Kurashiki, Japan

2015.04-present

Lab Head, Korea Brain Research Institute, Daegu, South Korea

Abstract

Astrocyte is a type of glia that plays a wide role in maintaining CNS. In spite of several efforts, the mechanism of astrocyte differentiation is largely unclear. Here, I present our recent findings that Cyclin Dependent Kinase Inhibitor (CDKI), which controls CDK4/6 during G1 phase, can trigger the differentiation of astrocyte in the developing cortex. In general, cell cycle exit precedes differentiation, and the length of G1 is one of the crucial factors to regulate the balance between proliferation and differentiation. By observing the expression pattern of astrocyte-specific marker, Aldh1l1, in various embryonic stages of Aldh1l1-EGFP mice, we found that EGFP in the ventricular zone was gradually increased from E15. Notably, expression level of CDKIs was accompanied in the same manner. We further examined whether CDKIs can regulate the generation of astrocyte in both in vitro and in vivo. When the CDKI genes were overexpressed in embryonic brain of Aldh1l1-EGFP mice as well as cultured cell with various conditions, EGFP+ cells were increased. We further confirmed that, with markers of astrocyte and cortical neurons, CDKI-overexpressed cells tend to differentiate into astrocyte rather than upper layer neuron depending on the stages of CDKI introduction. Furthermore, CDKI-overexpressed cells showed morphological characteristics of astrocyte. Taken together, CDKI can potentiate astrocyte production in a stage dependent manner, which can be considered as the novel effect of cell-cycle inhibition towards the astrocyte differentiation.