CDB SEMINAR

Speaker: Kenji Matsuno

Science University of Tokyo

Department of Biological Science and Technology

http://www.tus.ac.jp/edu/gakubu/kyouin/tb/matsuno.html

Title: "Function of *O*-fucosylation in Notch signaling"

Date: Tuesday, February 24

Time: 15:00 P.M.～16:00 P.M.

Place: 7th floor Conference Room of Building A, CDB

Summary

Notch signaling, which is highly conserved from nematodes to mammals, plays critical roles in many developmental processes. In the *Drosophila* embryo, deficiency in Notch signaling results in neurohyperplasia, commonly referred to as the neurogenic phenotype. We have identified a novel maternal neurogenic gene, *neurotic*, and have shown that it is essential for Notch signaling. *neurotic* encodes a protein highly homologous to mammalian GDP-fucose protein *O*-fucosyltransferase, which adds fucose sugar to epidermal growth factor-like repeats of Notch. Neurotic was indispensable for the binding between Notch and Delta, which could account for observed neurogenic phenotype in neurotic mutation. This idea was consistent with our observation that shortage of GDP-fucose in GDP-mannose-4, 6-dehydratase mutations resulted in the perturbation of Notch signaling. We also found that Neurotic has other functions, which is independent of its *O*-fucosyltransferase activity. Neurotic promoted proteolytic degradation of Notch and inhibited the transportation of Notch to the plasmamembrane.

Contact: Shigeo Hayashi /Morphogenetic Signaling, CDB e-mail: shayashi@cdb.riken.jp
Tel: 078-306-3185; RIKEN Center for Developmental Biology, http://www.cdb.riken.go.jp/