The 83rd iCeMS SEMINAR

CeMI Seminar Series 25

Mon 27 June 2011
9:00-12:20

Venue: 2nd floor Seminar Room (#A207) Main Building
iCeMS Complex 1, Kyoto University

Refreshments will be served 15 minutes prior to the start of the seminar.
The seminar will be followed by an informal discussion with the lecturers over a light meal.

<Part 1: 9:00–9:45>

"How do membrane proteins become raftophilic?"

Prof. Kai Simons

Max Planck Institute of Cell Biology and Genetics (Dresden)

<Part 2: 9:45–10:30>

"Molecular mechanisms of myelination in the central nervous system"

Prof. Mikael Simons

Max Planck Institute for Experimental Medicine (Göttingen)
Department of Neurology, University of Göttingen

<Part 3: 10:50–11:35>

"Mechanisms of membrane bending and scission in clathrin-independent endocytosis"

Prof. Ludger Johannes

Institut Curie

<Part 4: 11:35-12:20>

"New insights into the formation and function of caveolae"

Prof. Robert G. Parton

Institute for Molecular Bioscience, University of Queensland

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Hosted by: iCeMS (Institute for Integrated Cell-Material Sciences), Kyoto University
Co-hosted by: Center for Frontier Medicine, Global COE Program, Kyoto University

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Abstracts for June 27 iCeMS Seminar

Prof. Kai Simons <*9:00–9:45*>

The study of lipid rafts as a membrane compartmentalization principle has been fraught with problems! Now the situation has changed by the discovery that plasma membranes can phase separate. These membrane systems are convenient models to study the basics of lipid-protein collectives. In this seminar, Prof. Kai Simons will discuss these newer developments of raft studies.

Prof. Mikael Simons <*9:45–10:30*>

The myelin sheath is one of the most abundant membrane structures in the vertebrate nervous system. It is produced by two types of specialized glial cells, oligodendrocytes in the central nervous system, and Schwanncells in the peripheral nervous system. The myelin sheath is formed by the spiral wrapping of glial plasma membrane extensions around the axons, followed by the extrusion of cytoplasm and the compaction of the stacked membrane bilayers. These tightly packed membrane stacks provide electrical insulation around the axons and maximize their conduction velocity. Axonal insulation by myelin not only facilitates rapid nerve conduction but also regulates axonal transport and protects against axonal degeneration. Damage to the myelin sheath, as it for example occurs in multiple sclerosis (MS) results therefore in severe neurological disability also as a result of neurodegeneration. The main goal of Prof. Mika Simons is to come up with new approaches of how to promote remyelination in demyelinating diseases such as MS. To realize this goal, he and his colleagues try to understand how myelin is formed during normal development. He will talk about his new progress in this front.

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Prof. Ludger Johannes <*10:50–11:35*>

Several endocytic processes exist that do not require the activity of clathrin, and it has been a conundrum how the plasma membrane would be bent in the apparent absence of membrane coats. Recent studies by Prof. Ludger Johannes and his colleagues have uncovered a novel mechanism through which nanodomain construction by glycosphingolipid-binding Shiga and cholera toxins or polyoma viruses (SV40) induces membrane curvature changes and drives the coat-independent formation of endocytic plasma membrane invaginations. They could show that actin polymerization on Shiga toxin-induced tubule membranes alone is sufficient to induce scission in a process that requires membrane reorganization and domain formation. Their data suggest that tubule membranes are poised to undergo actin induced demixing, thereby generating domain boundary forces that trigger line tension-driven squeezing of the tubules membranes leading to scission. Prof. Johannes will present these results as well as newer data, showing that cellular proteins that like the toxins use glycosphingolipids for membrane bending, thereby regulating the cell surface dynamics of various markers with critical roles in cellular processes such as cell migration.

Prof. Robert G. Parton <*11:35-12:20*>

One of the most abundant and characteristic surface microdomains of mammalian cells are surface pits termed caveolae. Despite their abundance the exact functions of caveolae remain elusive. Over the last ten years, the research by Prof. Rob Parton and his colleagues has focussed on the fine ultrastructure of caveolae, the major components of caveolae, and their specific cellular functions. Their recent studies, utilising a range of systems including mouse models, cultured cells, and the zebrafish embryo, have identified a new family of coat proteins which work with membrane proteins called caveolins to regulate caveola formation. PTRF-cavin family members regulate association of caveolin with caveolae and identify a cellular mechanism to regulate caveolar and non-caveolar functions of caveolins. In this seminar, Prof. Parton will speak about the results of these studies, which are providing insights into a novel role for caveolae as mechanosensory organelles.

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