理学部セミナー
（タンパク質のシグナル伝達機能研究）

日　時：　2013 年 1 月 10 日（木）午後 4:00～5:30

場　所：　研究棟 7 階談話室（739 室）

演　題：　**Interactomics for cell signaling: From discovery to functional study on signaling modulators**

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概　要：

Cells receive signals through receptors and transduce them into various responses. Signaling proteins mediate the processes through series of dynamic protein-protein interactions. We have been establishing platform technology for identification of binding proteins by using pull-down and mass spectrometry. After the identification of signaling proteins we have characterized the mechanisms and the roles of the proteins and interactions by using variety of approaches including molecular and cellular as well as single molecule techniques. In this seminar, I'd like to share some experiences during the experimental works for identification of trace and dynamic binders of some hub-proteins in cell growth and metabolism. Potential of new approaches using non-protein captures (aptamer) will be also introduced.

In addition to the technologies two of examples will be introduced with detail mechanistic and functional data:

(1) Interactome of phospholipase D (PLD) and the role in endothelial cell – By using pull-down technologies we established PLD interactome and characterized the relationships in detail. Among them we found GTPase-PLD-Kinase (GPK) signaling motifs are popular in the interactome. Rheb-PLD2-mTOR relationship was further studied in vitro and in vivo including PLD2-knock-out mice.

(2) To identify the signaling proteins involved in the insulin resistance and muscle atrophy we applied one of the interactomics approaches, domain-perturbation of signaling pathway by using ~100 SH2-domain library. We also analyzed the expression profile of the SH2-containing proteins in diabetic mouse muscle. Through these approaches we selected a SH2-containing protein of Tensin family, C1 domain-containing phosphatase and TENsin homologue (C1-Ten), and characterized the mechanism in the insulin resistance and atrophy. We suggest C1-Ten can induce muscle atrophy through IRS-1 degradation in catabolic diseases. Also, this proposes a novel potential mechanism of IRS-1 regulation through C1-Ten PTPase activity.

Ryu 教授は公益財団法人「ひょうご震災記念２１世紀研究機構(HUMAP)」の平成 24 年度研究者交流推進制度(HORN)事業の招聘研究者として県立大を訪問中です。学生・院生・スタッフの皆さんの参加を是非期待しております。

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