Form 3

Report for Joint/usage program for Endocrine/Metabolism

（ Research Program Fiscal Year (FY) 2016 ）

Date: 2017/4/28

To:

Director of Institute for Molecular and Cellular Regulation

1. Program No.
   Research title: Defining the targets of mTORC2-Akt pathway in stress response.

2. Objective of the research:
   We will define the molecular mechanism by which mTORC2-Akt signaling pathway regulates lipid remodeling during stress response using biochemistry and yeast genetics.

3. Period
   2016/4/1-2017/3/31

4. Project organization

Name of Applicant: Riko Hatakeyama
Position: Research Associate
Institution/department: University of Fribourg

Name of Co-applicant:
Position:
Institution/department:

Name of Researcher in IMCR: Satoshi Yoshida
Position: Associate Professor

5. Research plans:

Dr. Hatakeyama will visit IMCR and perform experiments and discuss with Dr. Yoshida to publish ongoing collaborative projects. Dr. Hatakeyama also plans to give a seminar at IMCR.

6. Research results:

Hatakeyama visited IMCR in the Nov. 2016 and gave a seminar. Hatakeyama and Yoshida worked together for publishing collaborative papers. One paper has recently been published in *J Cell Sci* ("Ypk1 and Ypk2 kinases maintain Rho1 at the plasma membrane by flippase-dependent lipid remodeling after membrane stresses."). And another paper is currently under review ("A role of Osh6/Osh7-dependent transport of phosphatidylserine in the cortical localization of Rho1 GTPase under stress").

7. Publications and/or Presentations made through this collaboration

-Original article

Hatakeyama R, Kono K, Yoshida S

Ypk1 and Ypk2 kinases maintain Rho1 at the plasma membrane by flippase-dependent lipid remodeling after membrane stresses.

J Cell Sci. 2017 Mar 15;130(6):1169-1178. doi: 10.1242/jcs.198382. Epub 2017 Feb 6.

-International Conference

Yoshida S

Mechanisms that specify Rho1 signaling outputs

14th International Congress on Yeast, Awaji 2016/ 9/ 11～15

(Please summarize the report in 2 pages)