

P010 Control of Rab35 functions by a RabGAP during cytokinesis
**Laurent Chesneau, Ilektra Kouranti, Sylvain Lodeho,
Bruno Goud, Arnaud Echard**

Institut Curie, CNRS-UMR144, Paris, France

Phosphatidyl Inositol 4,5 bis Phosphate (PIP₂) is necessary for cytokinesis. But how PIP₂ domains are established during cytokinesis is not well understood.

In our lab, GTPase Rab35 has been implicated in this process (Kouranti et al, 2006). We showed that expression of Rab35 GDP-restricted mutant leads to a defect of enrichment of PIP₂ at the furrow. This primary defect induces destabilization of the intercellular bridge. Consistent with a role of Rab35 in a recycling pathway between endosomes to plasma membrane, Rab35 GDP-restricted mutant induces the formation of vacuoles during interphase as well as cytokinesis. These vacuoles are characterized by the presence of transferrin receptor, PIP₂ and actin filaments. We propose that the Rab35-regulated pathway plays an essential role during the terminal steps of cytokinesis by controlling PIP₂ subcellular distribution during cell division.

Since cytokinesis is perturbed when Rab35 GTP cycle is blocked, it appears that Rab35 regulation is important for proper cell division. We found that the overexpression of a RabGAP induced identical vacuole formation and cytokinesis defects as those induced by Rab35 GDP-restricted mutant, indicating they both regulate the same pathway. Moreover, these defects could be suppressed by co-expression of Rab35 GTP-locked mutant. Therefore, we have identified a GAP that controls Rab35 during cytokinesis.