

**P052** p85/Cdc42-mediated cytokinesis control

**Silió V, Garcia Z, Checa AI, Serrano A, Carrera AC**

*Department of Immunology and Oncology, Centro Nacional de Biotecnología/CSIC, Universidad Autónoma de Madrid, Cantoblanco, Madrid E-28049, Spain*

Cytosolic division involves the function of a number of cytoskeletal proteins, whose coordination in the spatio-temporal control of cytokinesis is poorly defined. We studied the role of p85/p110 phosphoinositide kinase (PI3K) in mammalian cytokinesis. Deletion of the p85alpha regulatory subunit induced cell accumulation in telophase and appearance of binucleated cells, whereas inhibition of PI3K activity did not affect cytokinesis. We analyzed the mechanism by which p85alpha regulates cytokinesis; p85alpha deletion reduced Cdc42 activation in the cleavage furrow and septin 2 accumulation at this site. As Cdc42 deletion also triggered septin 2 and cytokinesis defects, a mechanism by which p85 controls cytokinesis is by regulating the local activation of Cdc42 in the cleavage furrow and in turn septin 2 localization. We show that p85 acts as a scaffold to bind Cdc42 and septin 2 simultaneously.

We are currently studying the mechanism that underlies Cdc42 translocation to the cleavage furrow using videomicroscopy and immunofluorescence techniques. Since p85 alpha binds to alpha/beta-tubulin, we have tested whether Cdc42 translocation relies on the microtubule cytoskeleton. In addition, considering that Cdc42 concentrates to the Golgi in interphase, we studied if Cdc42 localizes to vesicles during cytokinesis. We will present the results from these studies.