EXAMINATION OF REGULATROY MECHANISMS FOR CELL FUNCTIONS BY THE GOLGI APPARATUS

メタデータ 言語: jpn
出版者:
公開日: 2021-11-08
キーワード (Ja):
キーワード (En):
作成者: Nakamura, Nobuhiro
メールアドレス:
所属:

URL https://doi.org/10.24517/00063168

This work is licensed under a Creative Commons Attribution-NonCommercial-ShareAlike 3.0 International License.



Search Research Projects How to Use

2004 Fiscal Year Final Research Report Summary

EXAMINATION OF REGULATROY MECHANISMS FOR CELL FUNCTIONS BY THE GOLGI APPARATUS

Project/Area Number
15570156
Research Category
Grant-in-Aid for Scientific Research (C)
Allocation Type
Single-year Grants
Section
一般
Research Field
Cell biology
Research Institution
KANAZAWA UNIVERSITY
Principal Investigator
NAKAMURA Nobuhiro KANAZAWA UNIVERSITY, GRADUATE SCHOOL OF NATURAL SCIENCE AND TECHNOLOGY, ASSOCIATE PROFESSOR, 自然科学研究科, 助教授 (50294955)
Project Period (FY)
2003 – 2004
Keywords
COLGI ADDADATUS / PHOSPHODYLATION / KINASE / CDOWTH SIGNAL

Research Abstract

Research Project

We have shown that a 277th amino acid residue of GRASP65 (S277) is phosphorylated in interphase cells and the phosphorylation signal is markedly enhanced by the growth factor treatment including EGF. ERK is activated by the EGF induced growth factor signal and the activated ERK phosphorylates S277 directly. We further found that S277 is heavily phosphorylated during M phase and analyzed the molecular mechanism for this up regulation of the phosphorylation. The amino acid sequence around S277 (PGSPG) is well conserved among mammalian CTRASP65 homologues. This sequence is well fitted with the target sequence of cdk1/cyclinB. S277 was strongly phosphorylated by a cytoplasmic extract of M phase cells and this was completely inhibited by roscovitine, a cdk specific inhibitor. S277 was also phosphorylated by an ERK inactive cytoplasmic extract of M phase cells that was prepared in the presence of U0126, a MEK inhibitor. These results strongly suggested that cdk1/cyclinB, and not ERK, is responsible for the phosphorylation of S277 in M phase. Surprisingly, the mitotic entry was strongly inhibited by the microinjection of purified GRASP65 without N-terminal myristoylation (Δ m-GRASP65). This was not observed by the microinjection of Δ m-GRASP65 in which S277 was changed with alanine. These results suggested that Δ m-GRASP65 interact with some cytoplasmic factors and inhibits the mitotic entry. We have found that Plk1 specifically binds to phosphorylated S277 region of GRASP65 and there are some cytoplasmic factors that bind to unphosphorylated S277 region of GRASP65.

Research Products (8 results)

	All 2005 2004 2003
	All Journal Article
[Journal Article] Convergence of cell cycle regulation and growth factor signals on GRASP65	2005 ∨
[Journal Article] Convergence of cell cycle regulation and growth factor signals on GRASP65.	2005 ~
[Journal Article] Dynamics of Golgi matrix proteins after a block of ER to Golgi transport	2004 ×
[Journal Article] Dynamics of Golgi matrix proteins after a block of ER to Golgi transport.	2004 ×
[Journal Article] Structural Integrity of the Golgi is Temperature Sensitive in Conditional-Lethal Mutants with No Detectable GM130	2003 ×
[Journal Article] Identification of a five-pass transmembrane protein family localizing in the Golgi apparatus and the ER	2003 ×
[Journal Article] Structural Integrity of the Golgi is Temperature Sensitive in Conditional-Lethal Mutants with No Detectable GM130.	2003 ×
[Journal Article] Identification of a five-pass transmembrane protein family localizing in the Golgi apparatus and the ER.	2003 ×

Published: 2006-07-10

URL: https://kaken.nii.ac.jp/report/KAKENHI-PROJECT-15570156/155701562004kenkyu_seika_hokoku