
Yuji Chatani, Eiko Tanaka, Kazuyuki Tobe, Akira Hattori,
Masahiro Sato, Hiroyuki Tamemoto, Naomi Nishizawa, Hiroshi Nomoto,
Tatsuo Takeya, Takashi Kadowaki, Masato Kasuga, Michiaki Kohno*

We have raised two antisera, one against the peptide 307–327 and the other against the C92 peptide of the deduced amino acid sequence of ERK1. Using these antibodies, we have clearly identified that the 41- and 43-kDa proteins, the increased tyrosine phosphorylation of which we and others had originally described in various mitogen-stimulated cells, are family members of ERKs/MAP2 kinases which are activated by phosphorylation both on tyrosine and threonine residues.

Hepatocyte Growth Factor Rapidly Induces the Tyrosine Phosphorylation of 41-kDa and 43-kDa Proteins in Mouse Keratinocytes.

Yuji Chatani, Akio Itoh, Eiko Tanaka, Akira Hattori,
Toshikazu Nakamura, Michiaki Kohno*

We have examined the hepatocyte growth factor (HGF)-mediated changes in protein-tyrosine phosphorylation in mouse keratinocytes (PAM-212) and canine kidney epithelial cells (MDCK). In PAM-212 cells HGF and epidermal growth factor, both of which stimulated the DNA synthesis, rapidly induced the tyrosine phosphorylation of two 41-kDa and two 43-kDa proteins: increased tyrosine phosphorylation of those proteins has been commonly observed when quiescent fibroblasts are stimulated with a variety of mitogenic agents. In contrast, HGF did not stimulate the DNA synthesis but induced cell dissociation in MDCK cells.

Biphasic Activation of Two Mitogen-activated Protein Kinases during the Cell Cycle in Mammalian Cells.

Hiroyuki Tamemoto, Takashi Kadowaki, Kazuyuki Tobe, Kojiro Ueki, Tetsuro Izumi,
Yuji Chatani, Michiaki Kohno*, Masato Kasuga, Yoshio Yazaki, Yasuo Akanuma

We studied mitogen-activated protein kinase (MAPK) activities during the cell cycle of Chinese hamster ovary (CHO) cells using site-specific antibodies against extracellular signal-regulated kinase-1, a 44-kDa MAPK. These antibodies detected two distinct MAPKs (44- and 42-kDa MAPKs) in CHO cells. CHO cells were arrested at metaphase in the M phase by treatment with nocodazole, and activities of MAPKs were analysed at specific time points after release from arrest.