

Phosphorylation of L1 by ERK

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Introduction

Reactome is open-source, open access, manually curated and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.

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Literature references

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- Fabregat, A., Jupe, S., Matthews, L., Sidiropoulos, K., Gillespie, M., Garapati, P. et al. (2018). The Reactome Pathway Knowledgebase. *Nucleic Acids Res*, 46, D649-D655. [↗](#)
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Reactome database release: 70

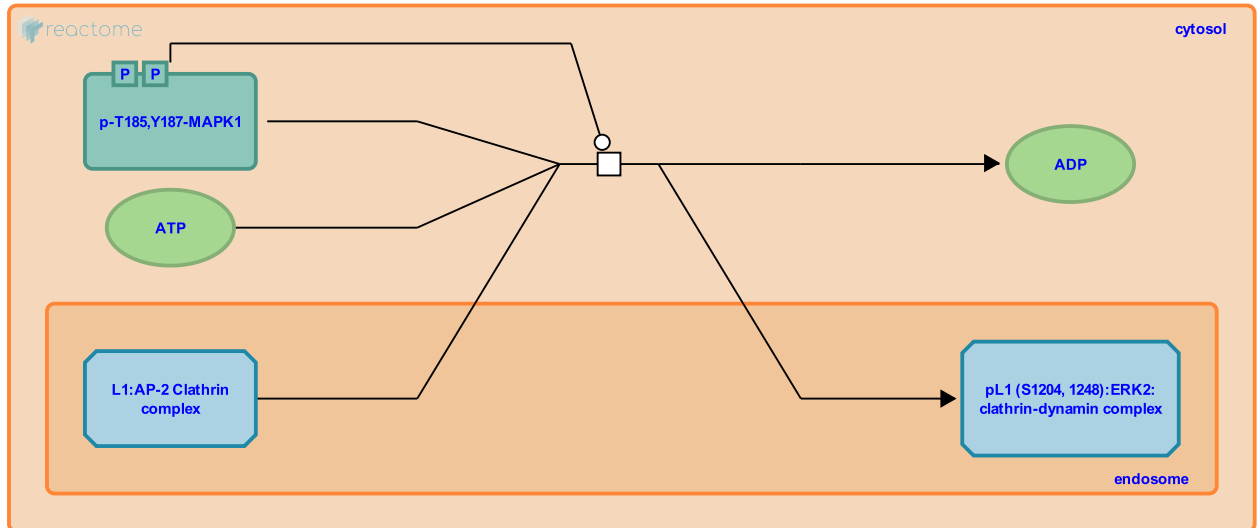
This document contains 1 reaction ([see Table of Contents](#))

Phosphorylation of L1 by ERK ↗

Stable identifier: R-HSA-445079

Type: transition

Compartments: cytosol



L1 cross linking can activate MAPK cascade components MEK1/2, ERK1/2, as well as Src, Raf-1, and p90rsk. MAP kinase signaling requires endocytosis mediated by Src. ERK2 can phosphorylate internalized L1 at serine residues 1204 and 1248. This phosphorylation may increase the neurite growth.

Literature references

Schmid, RS., Pruitt, WM., Maness, PF. (2000). A MAP kinase-signaling pathway mediates neurite outgrowth on L1 and requires Src-dependent endocytosis. *J Neurosci*, 20, 4177-88. ↗

Schaefer, AW., Kamiguchi, H., Wong, EV., Beach, CM., Landreth, G., Lemmon, V. (1999). Activation of the MAPK signal cascade by the neural cell adhesion molecule L1 requires L1 internalization. *J Biol Chem*, 274, 37965-73. ↗

Editions

2008-07-30	Authored, Edited	Garapati, P V.
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