Monoubiquitination of N-myristoyl GAG (P12493) protein

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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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Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

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. ↗

Fabregat, A., Korninger, F., Viteri, G., Sidiropoulos, K., Marin-Garcia, P., Ping, P. et al. (2018). Reactome graph database: Efficient access to complex pathway data. *PLoS computational biology, 14*, e1005968. ↗

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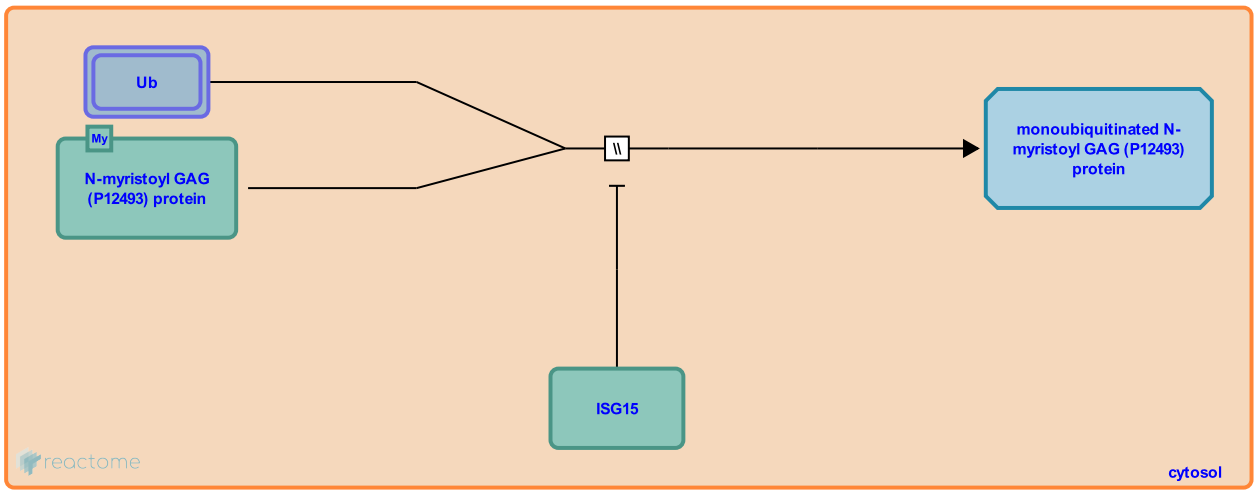
Stable identifier: R-HSA-1169307

Type: omitted

Compartments: cytosol



Cytosolic N-myristoyl Gag polyprotein is conjugated with a single molecule of ubiquitin. Conjugation is typically to one of two lysine residues in the p6 domain of Gag but can be to lysine residues in the MA, CA, NC, and SP2 domains of the protein. The specific host cell E2 and E3 proteins that mediate Gag ubiquitination have not been identified. The same studies that first identified the p6 ubiquitination sites in Gag also called the biological significance of Gag ubiquitination into question by demonstrating that Gag proteins in which the p6 ubiquitination sites had been removed by mutagenesis could still assemble efficiently into infectious viral particles (Ott et al. 1998, 2000). More recent work, however, has identified additional ubiquitination sites throughout the C-terminal region of the Gag polyprotein, and when all of these sites are removed by mutagenesis, both viral assembly involving the mutant Gag polyprotein and infectivity of the resulting viral particles are sharply reduced (Gottwein et al. 2006).

Note: Reactions directly involving interactions of human host proteins with foreign ones are highlighted in red.

Literature references

Gottwein, E., Jager, S., Habermann, A., Krausslich, HG. (2006). Cumulative mutations of ubiquitin acceptor sites in human immunodeficiency virus type 1 gag cause a late budding defect. *J Virol, 80*, 6267-75. ↗

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Editions

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