CLIP3 and CYLD bind TNF signaling complex

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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. ↗

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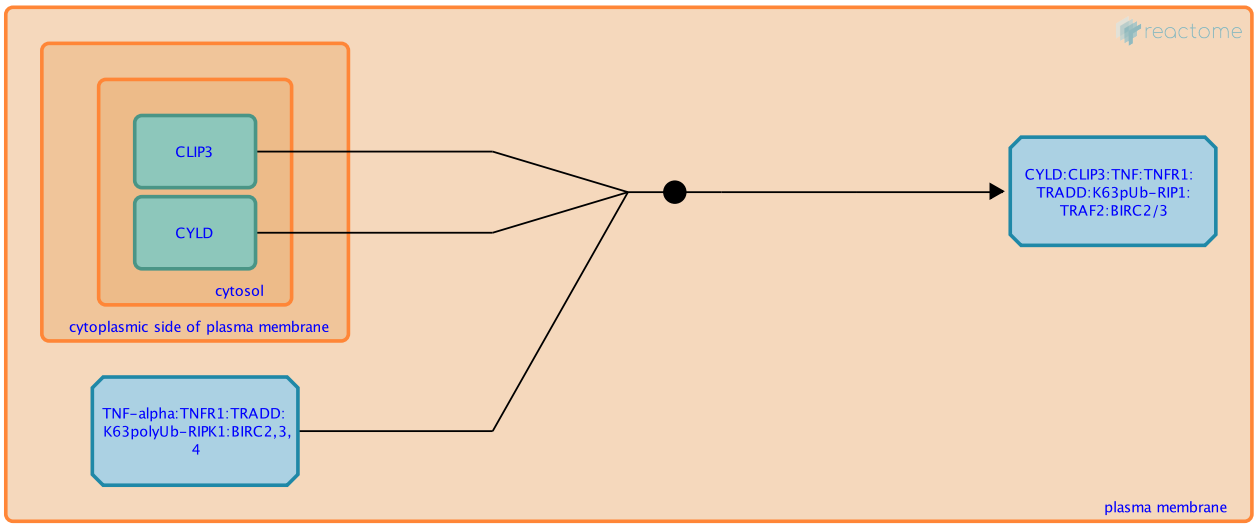
Stable identifier: R-HSA-5357928

Type: binding

Compartments: plasma membrane, cytosol



CAP-GLY domain containing linker protein 3 (CLIP3 or CLIPR-59) is thought to function as an adaptor protein recruiting CYLD into the TNFR1 signaling to facilitate CYLD-mediated deubiquitination of RIPK1 in TNFalpha signaling (Fujikura D et al. 2012). CLIP3-assisted CYLD-mediated K63-deubiquitination of RIPK1 may promote caspase-8 activation to induce apoptosis by TNFalpha. The effects of CLIPR-59 knockdown on apoptosis induction by TNFalpha were more effective in human cervical cancer HeLa cells than in human alveolar basal epithelial A549 cells or human fibrosarcoma HT1080 cells. These findings suggest that the role of CLIPR-59 on TNF-alpha-induced and RIP1-mediated pro-apoptotic signaling is dependent on cell type and context (Fujikura D et al. 2012).

Literature references

Harhaj, EW., Dixit, VM. (2012). Regulation of NF-?B by deubiquitinases. *Immunol. Rev., 246*, 107-24. ↗

Fujikura, D., Ito, M., Chiba, S., Harada, T., Perez, F., Reed, JC. et al. (2012). CLIPR-59 regulates TNF-?-induced apoptosis by controlling ubiquitination of RIP1. *Cell Death Dis, 3*, e264. ↗

Editions

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