

NOD1 induced apoptosis is mediated by RIP2 and CARD8

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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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Reactome database release: 77

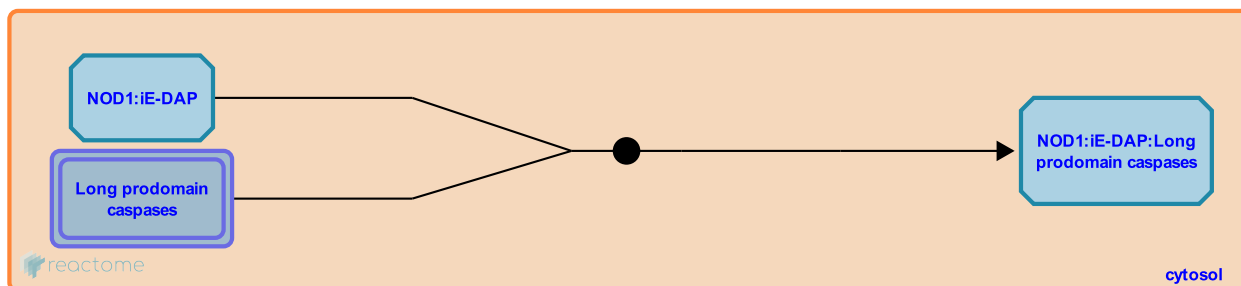
This document contains 1 reaction ([see Table of Contents](#))

NOD1 induced apoptosis is mediated by RIP2 and CARD8 ↗

Stable identifier: R-HSA-622420

Type: binding

Compartments: cytosol



NOD1 was found to coimmunoprecipitate with several procaspases containing long prodomains with CARDs or DEDs, including caspase-1, caspase-2, caspase-4, caspase-8, and caspase-9, but not those with short prodomains like caspase-3 or caspase-7. Deletions of caspase-9 determined that the CARD domain was required for this interaction (Inohara et al. 1999). More recently, NOD1 activation of apoptosis was shown to require the RIP2-dependent activation of caspase-8, this effect being inhibited by CASP8 and FADD-like apoptosis regulator, also called FLICE-inhibitory protein, FLIP or CLARP (da Silva Correia et al. 2007), which is a specific inhibitor of caspase-8 (Irmeler et al. 1997).

Literature references

Inohara, N., Koseki, T., del Peso, L., Hu, Y., Yee, C., Chen, S. et al. (1999). Nod1, an Apaf-1-like activator of caspase-9 and nuclear factor-kappaB. *J Biol Chem*, 274, 14560-7. ↗

Editions

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