DHX36 or DHX9 binds MyD88

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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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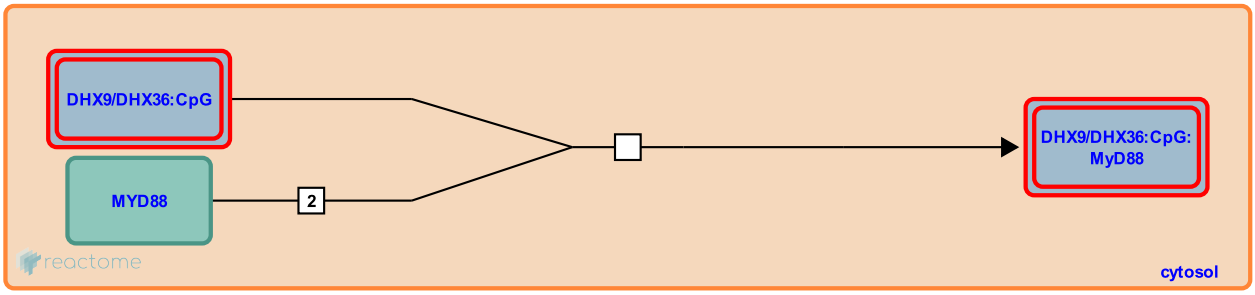
Stable identifier: R-HSA-3134953

Type: transition

Compartments: cytosol



Both DHX36 and DHX9 were found to interact with MyD88 when co-expressed in human embryonic kidney 293T cells. Moreover, the HA2 and DUF domains of DHX were critical for interaction with the TIR domain of MyD88 [Kim T et al 2010].

DHX9 or DHX36 knockdown by siRNA inhibited cytokine release in human GEN2.2 cell line (leukemic pDC cells) in response to CpG-ODN or to HSV but not to RNA viruses. Furthermore, knockdown of DHX36 diminished the nuclear localization of IRF7 in CpG-A-stimulated cells, while knockdown of DHX9 inhibited nuclear localization of NF-kappaB p50 in response to CpG-B. Thus, DHX36 and DHX9 are thought to trigger MyD88-dependent IRF7 and NF-kappaB activation respectively [Kim T et al 2010].

Literature references

Kim, T., Pazhoor, S., Bao, M., Zhang, Z., Hanabuchi, S., Facchinetti, V. et al. (2010). Aspartate-glutamate-alanine-histidine box motif (DEAH)/RNA helicase A helicases sense microbial DNA in human plasmacytoid dendritic cells. *Proc. Natl. Acad. Sci. U.S.A., 107*, 15181-6. ↗

Editions

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