# Defective MyD88 does not bind MAL(TIRAP):TLR2/4

D'Eustachio, P., McDonald, DR., Shamovsky, V.

European Bioinformatics Institute, New York University Langone Medical Center, Ontario Institute for Cancer Research, Oregon Health and Science University.



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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

Fabregat, A., Sidiropoulos, K., Viteri, G., Forner, O., Marin-Garcia, P., Arnau, V. et al. (2017). Reactome pathway analysis: a high-performance in-memory approach. *BMC bioinformatics, 18*, 142. ↗

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. ↗

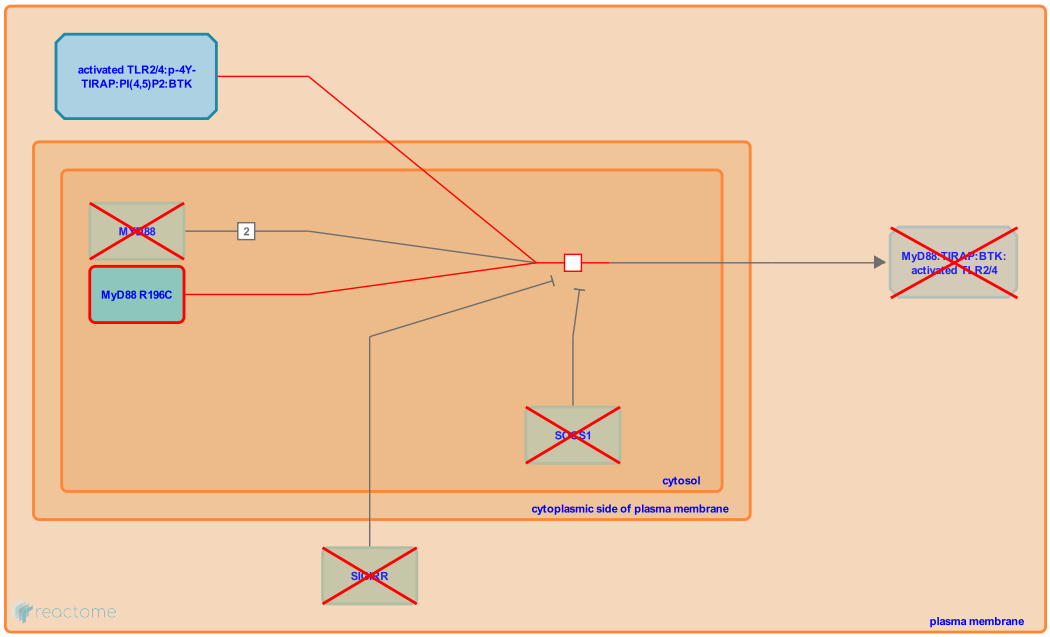
## Defective MyD88 does not bind MAL(TIRAP):TLR2/4

**Stable identifier:** R-HSA-5602606

**Type:** transition

**Compartments:** cytosol, plasma membrane

**Diseases:** primary immunodeficiency disease




The sorting MyD88 adaprtor-like (MAL or TIRAP) normally recruits MyD88 to activated TLR2 and TLR4 receptor complexes (Horng T et al. 2002; Verstak B et al. 2009). MyD88 interacts with MAL (TIRAP) via their TIR domains and activates a downstream signaling pathway mediated by TLR2 and TLR4 (Ohnishi H et al. 2009). A GST pull-down assay showed that defective MyD88 R196C variant loses its ability to bind to MAL (Yamamoto T et al. 2014).

## Literature references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Ohnishi, H., Tochio, H., Kato, Z., Orii, KE., Li, A., Kimura, T. et al. (2009). Structural basis for the multiple interactions of the MyD88 TIR domain in TLR4 signaling. *Proc. Natl. Acad. Sci. U.S.A., 106*, 10260-5.


## Editions

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