

S100A1 binds TLR4:LY96

D'Eustachio, P., Granucci, F., Shamovsky, V., Zanoni, I.

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

The contents of this document may be freely copied and distributed in any media, provided the authors, plus the institutions, are credited, as stated under the terms of <u>Creative Commons Attribution 4.0 International (CC BY 4.0)</u> <u>License</u>. For more information see our <u>license</u>.

01/04/2024

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.

The development of Reactome is supported by grants from the US National Institutes of Health (P41 HG003751), University of Toronto (CFREF Medicine by Design), European Union (EU STRP, EMI-CD), and the European Molecular Biology Laboratory (EBI Industry program).

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. 7
- Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics*, 33, 3461-3467. A
- 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. *オ*

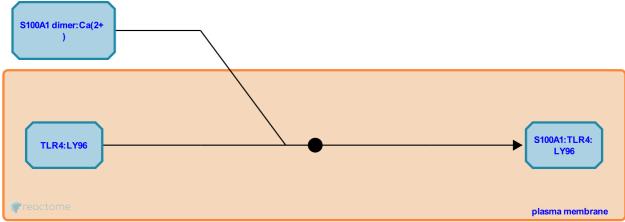
This document contains 1 reaction (see Table of Contents)

S100A1 binds TLR4:LY96 7

Stable identifier: R-HSA-6805943

Type: binding

Compartments: extracellular region, plasma membrane



The human event of S100A1 is inferred from the mouse data.

S100A1 is a Ca(2+)-sensing protein of the EF-hand family. S100A1 is expressed predominantly in cardiomyocytes, where it regulates Ca(2+)-dependent signaling events (Wright NT et al. 2005; Cannon BR et al. 2011; Brinks H et al. 2011; Yu J et al. 2015; Rohde D et al. 2014; Ritterhoff J & Most P 2012). In response to ischemic/hypoxic damage of cardiomyocytes, S100A1 is released or transferred to the extracellular region through open channels on membrane (Rohde D et al. 2014). The extracellular S100A1 activates signal and promotes cell survival pathways, including inflammation response via Toll-like receptor 4 (TLR4) (Brinks H et al. 2011; Yu J et al. 2015; Rohde D et al. 2014). In rodent H9C2 cells S100A1 was found to regulate the inflammatory response and oxidative stress via TLR4/ROS/NFkappaB pathway (Yu J et al. 2015).

Literature references

- Li, Y., Wu, L., Xiao, L., Yu, J., Lu, Y., Xing, Y. et al. (2015). Role of S100A1 in hypoxia-induced inflammatory response in cardiomyocytes via TLR4/ROS/NF-ΰB pathway. J. Pharm. Pharmacol., 67, 1240-50. ↗
- Most, P., Tsoporis, JN., Parker, TG., Kubatzky, KF., Gao, E., Ritterhoff, J. et al. (2014). S100A1 is released from ischemic cardiomyocytes and signals myocardial damage via Toll-like receptor 4. *EMBO Mol Med*, *6*, 778-94.

Editions

2015-09-12	Reviewed	D'Eustachio, P.
2015-09-12	Authored	Shamovsky, V.
2016-05-10	Edited	Shamovsky, V.
2016-05-12	Reviewed	Zanoni, I., Granucci, F.