oxPL binds LBP

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03/05/2024

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

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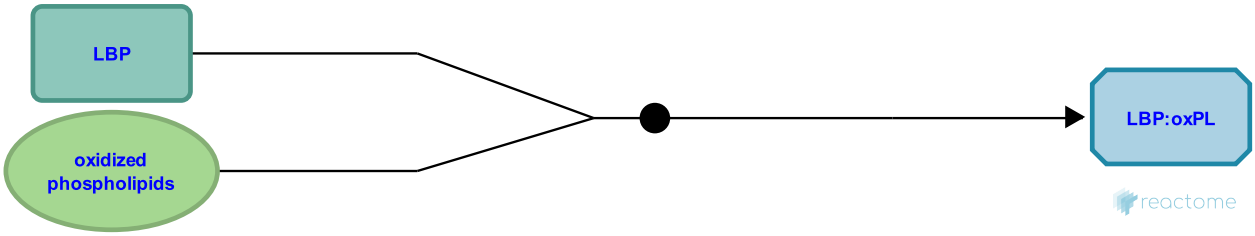
oxPL binds LBP ↗

Stable identifier: R-HSA-8869683

Type: binding

Compartments: extracellular region



Antibacterial defence involves activation of neutrophils that generate reactive oxygen species (ROS) capable of killing bacteria. The ROS production results in the oxidation of host phospholipids. Endogenously formed oxidized phospholipids, such as 1-palmitoyl-2-arachidonyl-sn-glycero-3-phosphorylcholine (OxPAPC), have been shown to inhibit TLR4- & TLR2-mediated signaling induced by bacterial lipopeptide or lipopolysaccharide (LPS) in various human cells (Bochkov VN et al., 2002; von Schlieffen E et al., 2009). Oxidized phospholipids were found to bind LPS binding protein (LBP) and soluble CD14 suggesting that the binding prevented recognition of LPS by these proteins thus preventing recognition of LPS and activation of TLR4 (Erridge C et al., 2008; von Schlieffen E et al., 2009). In addition, oxPAPC protected mice treated with a lethal dose of LPS (Bochkov VN et al., 2002). Thus, oxidized phospholipids may function as a negative feedback to blunt innate immune responses.

Literature references

Erridge, C., Spickett, CM., Webb, DJ., Kennedy, S. (2008). Oxidized phospholipid inhibition of toll-like receptor (TLR) signaling is restricted to TLR2 and TLR4: roles for CD14, LPS-binding protein, and MD2 as targets for specificity of inhibition. *J. Biol. Chem., 283*, 24748-59. ↗

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Editions

2016-05-06	Authored	Shamovsky, V.
2016-05-10	Edited	Shamovsky, V.
2016-05-12	Reviewed	Zanoni, I., Granucci, F.