

Clusterin binds C5b-C7, C8, C9

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https://reactome.org

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: 88

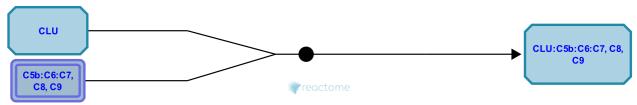
This document contains 1 reaction (see Table of Contents)

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Stable identifier: R-HSA-8852580

Type: binding

Compartments: extracellular region



Clusterin is a dimer of two fragments of the same translation product, which are disulfide bonded by five cysteines on each peptide (Tobe et al. 1991). It is able to modulate the terminal complement cascade in vitro and prevent cellular lysis by the membrane attack complex (MAC), C5b-9. Clusterin forms complexes with C5b:C6:C7, or C5b:C6:C7:C8 or C5b:C6:C7:C8:C9, as the proteins assemble into the amphiphilic MAC. Clusterin binding renders the complexes soluble and lytically inactive (Jenne & Tschopp 1989, Choi et al. 1989, Murphy et al. 1989, Tschopp et al. 1993).

Literature references

Chonn, A., Hertig, S., French, LE., Tschopp, J. (1993). Clusterin, the human apolipoprotein and complement inhibitor, binds to complement C7, C8 beta, and the b domain of C9. *J. Immunol.*, 151, 2159-65. *▶*

Editions

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