OAS antiviral response

D'Eustachio, P., Shamovsky, V., Silverman, RH.

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


Reactome database release: 79

This document contains 1 pathway and 15 reactions (see Table of Contents)
The human oligoadenylate synthetase (OAS) family consists of four proteins whose production is stimulated by interferon, OAS1, OAS2, OAS3, and OASL. The first three members have the 2'-5'-oligoadenylate synthetase activity for which the family is named (Sadler AJ & Williams BR 2008), whereas OASL is devoid of this activity despite sharing significant sequence similarity with the other OAS proteins (Zhu J et al. 2015). OAS1, 2, and 3 are activated by double-stranded RNA to synthesize 5'-triphosphorylated 2'-5'-oligoadenylates (2-5A) from ATP (Kerr IM & Brown RE 1978). The 2-5A serve as chemically unique second messengers that induce regulated RNA decay by activating ribonuclease L (RNase L), thus mediating antiviral innate immunity (Zhou A et al. 1993; Lin RJ et al. 2009; Huang H et al. 2014; Han Y et al. 2014). RNase L has also been implicated in antibacterial innate immunity (Li XL et al. 2008). RNase L cleaves single-stranded RNA (ssRNA) in U-rich sequences, typically after UU or UA dinucleotides leaving a 5'-OH and 2',3'-cyclic phosphate (Floyd-Smith G et al. 1981; Wreschner DH et al.1981; Cooper DA et al. 2014).

Some OAS proteins have additional or alternative antiviral functions that are independent of RNase L activity (Perelygin AA et al., 2002; Kristiansen H et al. 2011). The precise mechanisms of RNase L-independent OAS antiviral activities remain to be fully elucidated.

**Literature references**


## Editions

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OAS1 binds viral dsRNA

Location: OAS antiviral response

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