RNASEL cleaves viral ssRNA

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


Reactome database release: 83

This document contains 1 reaction (see Table of Contents)

https://reactome.org
Ribonuclease L (RNase L) is an ankyrin (ANK) repeat domain containing dual endoribonuclease-pseudokinase which is encoded by RNASEL gene (Wreschner DH et al. 1982; Zhou A et al. 1993; Hassel BA et al. 1993; Huang H et al. 2014; Han Y et al. 2014). Activated RNase L forms a homodimer (Dong B & Silverman RH 1995) which cleaves within single-stranded regions of different RNA substrates, predominantly after UpAp and UpUp dinucleotides, leaving 2',3'-cyclic phosphoryl and 5'-hydroxyl groups at the termini of the RNA cleavage products (Wreschner DH et al. 1981; Floyd-Smith G et al. 1981; Han Y et al. 2012; Cooper DA et al. 2014). The antiviral effect of RNase L occurs through a combination of effects and depends on the virus and cell type. This includes cleavage of viral genomic ssRNA that prevents viral replication (Cooper DA et al. 2015), cleavage of viral mRNA that inhibits viral protein synthesis, and cleavage of cellular RNA such as mRNA and rRNA that is required for viral replication (Wreschner DH et al. 1981; Silverman RH et al. 1983; Brennan-Laun SE et al. 2014; Cooper DA et al. 2014). RNase L induces apoptosis to eliminate virus-infected cells and autophagy to limit viral infections in some circumstances (Zhou A et al. 1997; Castelli JC et al. 1997; Chakrabarti A et al. 2012). Depending on the cell type and basal levels of RNase L, viral and cellular RNA cleavage products induce signaling to the IFN beta gene through RIG-I and/or MDA5 and MAVS (Malathi K et al. 2007; Banerjee S et al. 2014).

**Literature references**