Release

Marsh, G., Rush, MG., Squires, B.

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

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19/02/2020
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: 71

This document contains 1 pathway and 1 reaction (see Table of Contents)
Once the viral envelope has separated from the cell membrane Influenza virus particles are actively released to complete the budding process. HA (hemagglutinin) anchors the virus to the cell by binding to sialic acid-containing receptors on the cell surface. The enzymatic activity of the neuraminidase (NA) protein removes the sialic acid and releases the virus from the host cell. NA activity is also required to remove sialic acid from the carbohydrates present on the viral glycoproteins to prevent the viral particles from aggregating.

**Literature references**


**Editions**

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The release of influenza virus particles after separation of the virus and infected cell membrane is an active process. During the budding process, HA on the surface of the newly budding virion binds to cell surface molecules containing sialic acid residues as seen during attachment. The NA glycoproteins neuraminidase activity is essential to cleave the link between the HA and sialic acid on the surface of the host cell from which the budding virus is emerging from. Thus the NA mediated cleavage of sialic acid residues terminally linked to glycoproteins and glycolipids is the final step in releasing the virus particle from the host cell. This essential role of NA in release of virus particle has been demonstrated with the use of NA inhibitors (Palese, 1976; Luo, 1999; Garman, 2004), ts NA mutant viruses (Palese, 1974) and with viruses lacking NA activity (Liu, 1995). In all cases, viruses remain bound to the cell surface in clumps in the absence of NA enzymatic activity, resulting in loss of infectivity. Addition of exogenous sialidase results in virus release and recovery of infectivity. The sialidase activity of the NA is also important for removing sialic acid from the HA on virus particles, if this is not removed, virus particles aggregate.

Literature references


Editions

2007-05-01 Authorised Marsh, G.
2007-05-01 Reviewed Rush, MG., Squires, B.
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