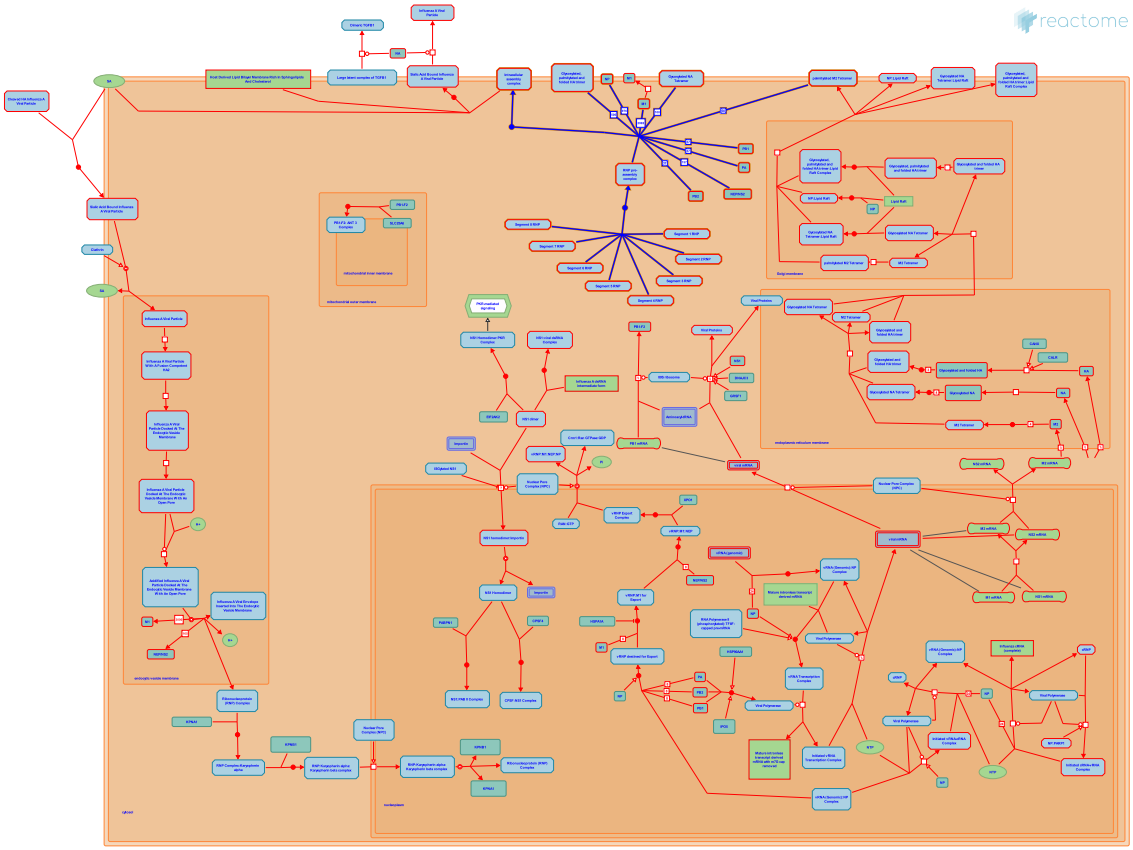
Packaging of Eight RNA Segments



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This is just an excerpt of a full-length report for this pathway. To access the complete report, please download it at the Reactome Textbook.

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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.
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

Fabregat, A., Sidiropoulos, K., Viteri, G., Forner, O., Marin-Garcia, P., Arnau, V. et al. (2017). Reactome pathway analysis: a high-performance in-memory approach. *BMC bioinformatics, 18*, 142. ↗

Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

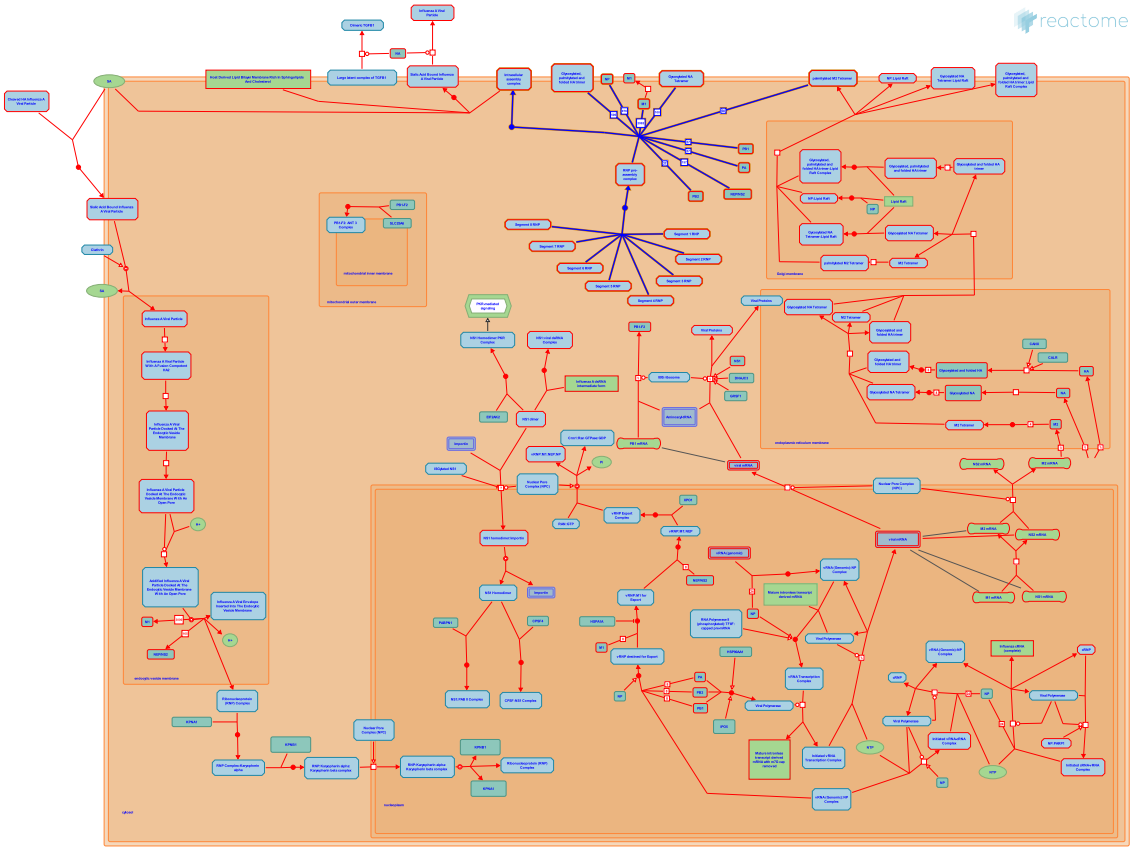
Fabregat, A., Jupe, S., Matthews, L., Sidiropoulos, K., Gillespie, M., Garapati, P. et al. (2018). The Reactome Pathway Knowledgebase. *Nucleic Acids Res, 46*, D649-D655. ↗

Fabregat, A., Korninger, F., Viteri, G., Sidiropoulos, K., Marin-Garcia, P., Ping, P. et al. (2018). Reactome graph database: Efficient access to complex pathway data. *PLoS computational biology, 14*, e1005968. ↗

Packaging of Eight RNA Segments ↗

Stable identifier: R-HSA-168303

Diseases: influenza



For a budding influenza virus to be fully infectious is it essential that it contains a full complement of the eight vRNA segments. Two different models have been proposed for packaging of the vRNPs into newly assembling virus particles; the random incorporation model and the selective incorporation model.
The random incorporation model as its name suggests proposes that there is no selection at all on which vRNPs are packaged. It is assumed that each vRNP has equal probability of being packaged, and that if enough vRNPS are packaged a particular percentage of budding virions will receive at least one copy of each genome segment. This model is supported by evidence that infectious virions may possess more than eight vRNPs assuring the presence of a full complement of eight vRNPs in a significant percentage of virus particles. Mathematical analysis of packaging suggested that twelve RNA segments would need to be packaged in order to obtain approximately 10% of virus particles that are fully infectious (Enami, 1991), a number that is compatible with experimental data (Donald, 1954). Due to the low amount of RNA per virion (estimated at 1-2% w/w), enumeration of the precise number of RNAs packaged in a virion is difficult.
The selective incorporation model, suggests that each vRNA segment contains a unique "packaging signal" allowing it to act independently, with each vRNA segment being packaged selectively. There is increasing evidence to support the theory of a packaging signal within the coding regions at both the 5' and 3' end of the genomic RNA, with signals being reported for all segments except segment 7 (Ozawa 2007, Muramoto 2006, Fujii 2005, Fujii 2003, Watanabe 2003, Liang 2005). The exact method by which individual vRNP segments are packaged is not known but it has been hypothesized to occur via specific RNA-RNA or protein-RNA interactions. This model is also supported by thin section electron microscopy images of influenza particles that show eight distinct "dots", presumably vRNPs within virus particles (Noda 2006).

Literature references

Shaw, ML., Palese, P. (2001). Orthomyxoviridae: The Viruses and Their Replication. *Fields Virology, 5th edition D.M. Knipe and P.M. Howley, Editors. 2006, Lippencott Williams and Wilkins: Philadelphia ISBN-10: 0-7817-6060-7*, 1647-1689. ↗

Editions

2007-05-01	Authored	Marsh, G.
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RNP association ↗

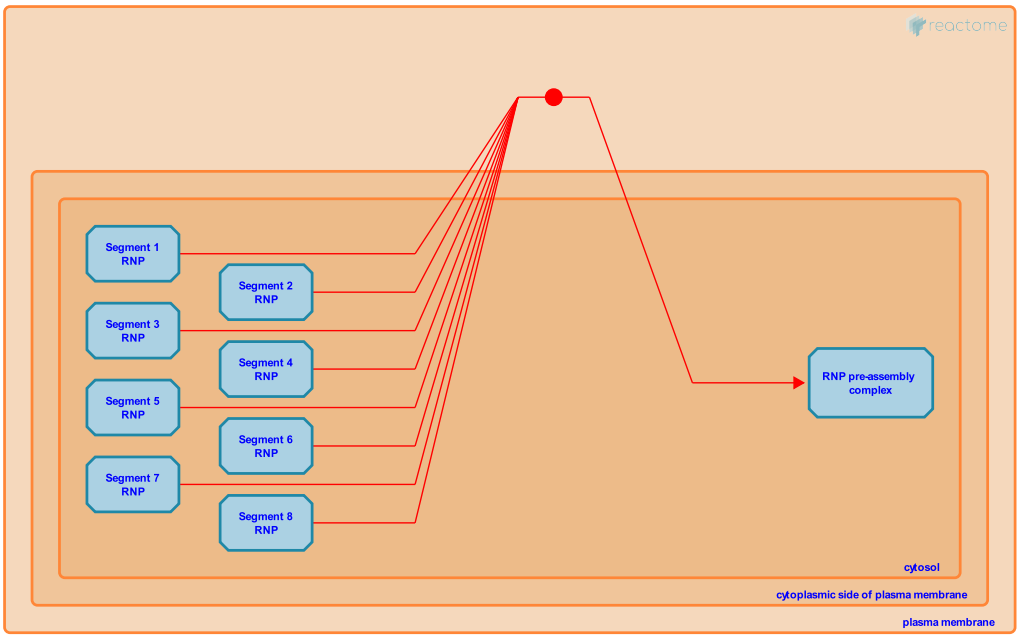
Location: Packaging of Eight RNA Segments

Stable identifier: R-HSA-168895

Type: binding

Compartments: plasma membrane

Diseases: influenza



The random incorporation model as its name suggests proposes that there is no selection at all on which vRNPs are packaged. It is assumed that each vRNP has equal probability of being packaged, and that if enough vRNPS are packaged a particular percentage of budding virions will receive at least one copy of each genome segment. This model is supported by evidence that infectious virions may possess more than eight vRNPs assuring the presence of a full complement of eight vRNPs in a significant percentage of virus particles. Mathematical analysis of packaging suggested that twelve RNA segments would need to be packaged in order to obtain approximately 10% of virus particles that are fully infectious (Enami, 1991), a number that is compatible with experimental data (Donald, 1954). Due to the low amount of RNA per virion (estimated at 1-2% w/w), enumeration of the precise number of RNAs packaged in a virion is difficult.

Literature references

ISAACS, A., DONALD, HB. (1954). Counts of influenza virus particles. *J Gen Microbiol, 10*, 457-64. ↗

Benham, C., Sharma, G., Palese, P., Enami, M. (1991). An influenza virus containing nine different RNA segments. *Virology, 185*, 291-8. ↗

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Association with M1 at cell membrane ↗

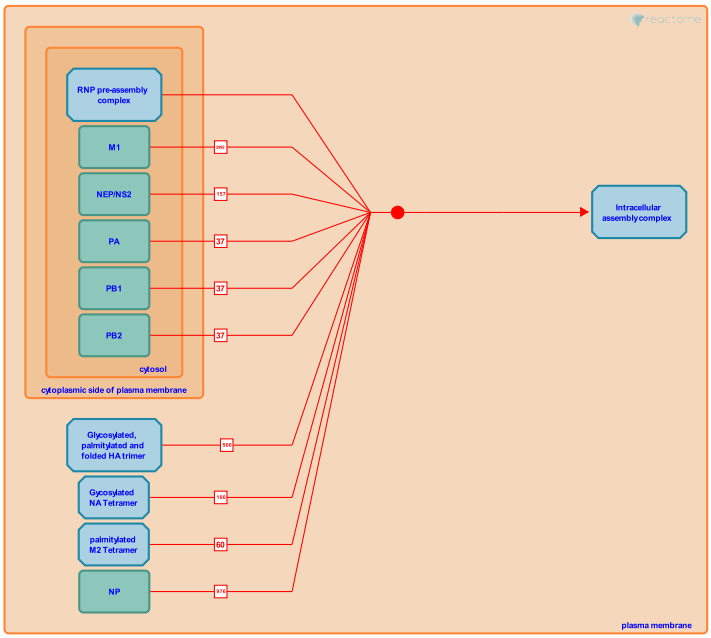
Location: Packaging of Eight RNA Segments

Stable identifier: R-HSA-195926

Type: binding

Compartments: plasma membrane, cytosol

Diseases: influenza



As influenza viruses bud from the plasma membrane of infected cells, complete virions are not seen inside cells. In polarized epithelial cells, assembly and budding of influenza occurs from the apical plasma membrane (Schmitt, 2004). For efficient assembly, all virion components must accumulate at the budding site, and it is believed that the viral glycoprotein accumulation determines the site of virus assembly and budding (Nayak, 2004). M1 is thought to be the bridge between the envelope glycoproteins and the RNPs for assembly (Schmitt, 2004). M2 is also required, because if it is not present RNPs are not packaged into budding virions (McCown, 2005), however it role is not known.

Literature references

McCown, MF., Pekosz, A. (2005). The influenza A virus M2 cytoplasmic tail is required for infectious virus production and efficient genome packaging. *J Virol, 79*, 3595-605. ↗

Schmitt, AP., Lamb, RA. (2004). Escaping from the cell: assembly and budding of negative-strand RNA viruses. *Curr Top Microbiol Immunol, 283*, 145-96. ↗

Barman, S., Nayak, DP., Hui, EK. (2004). Assembly and budding of influenza virus. *Virus Res, 106*, 147-65. ↗

Editions

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