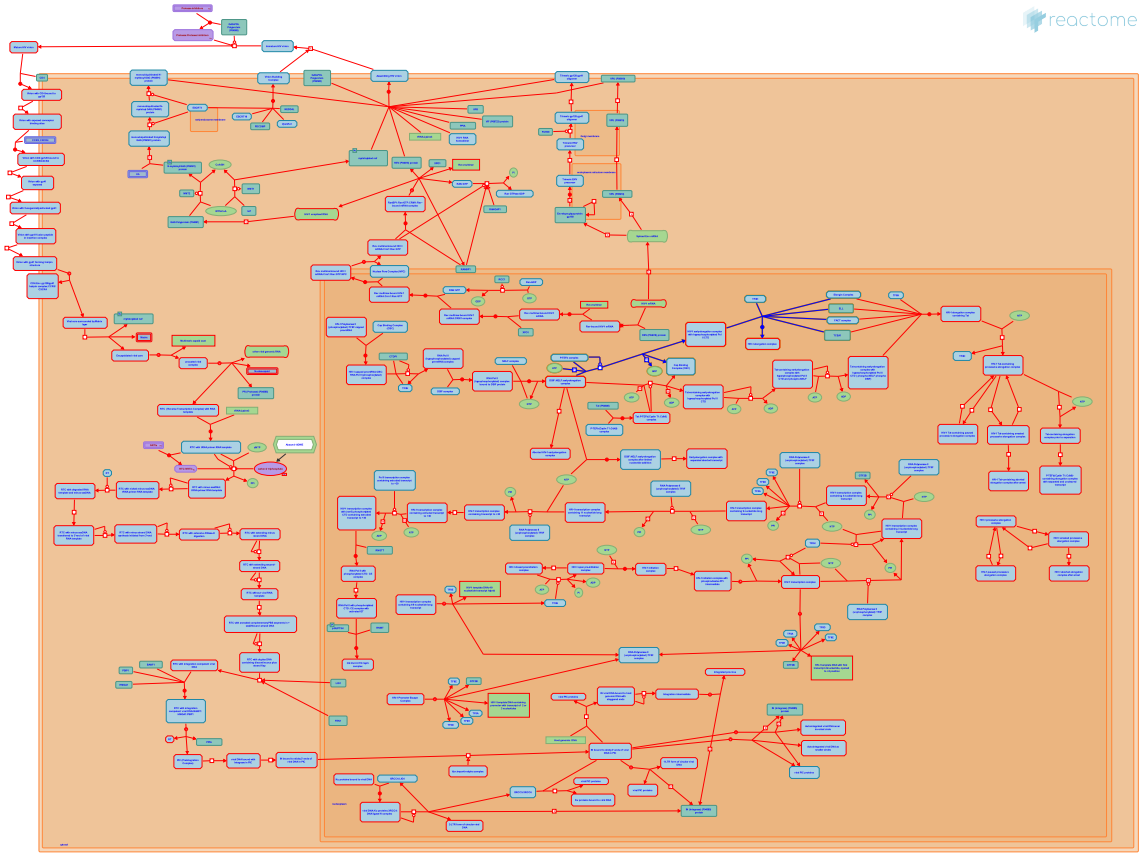
Formation of HIV elongation complex in the absence of HIV Tat



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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. ↗

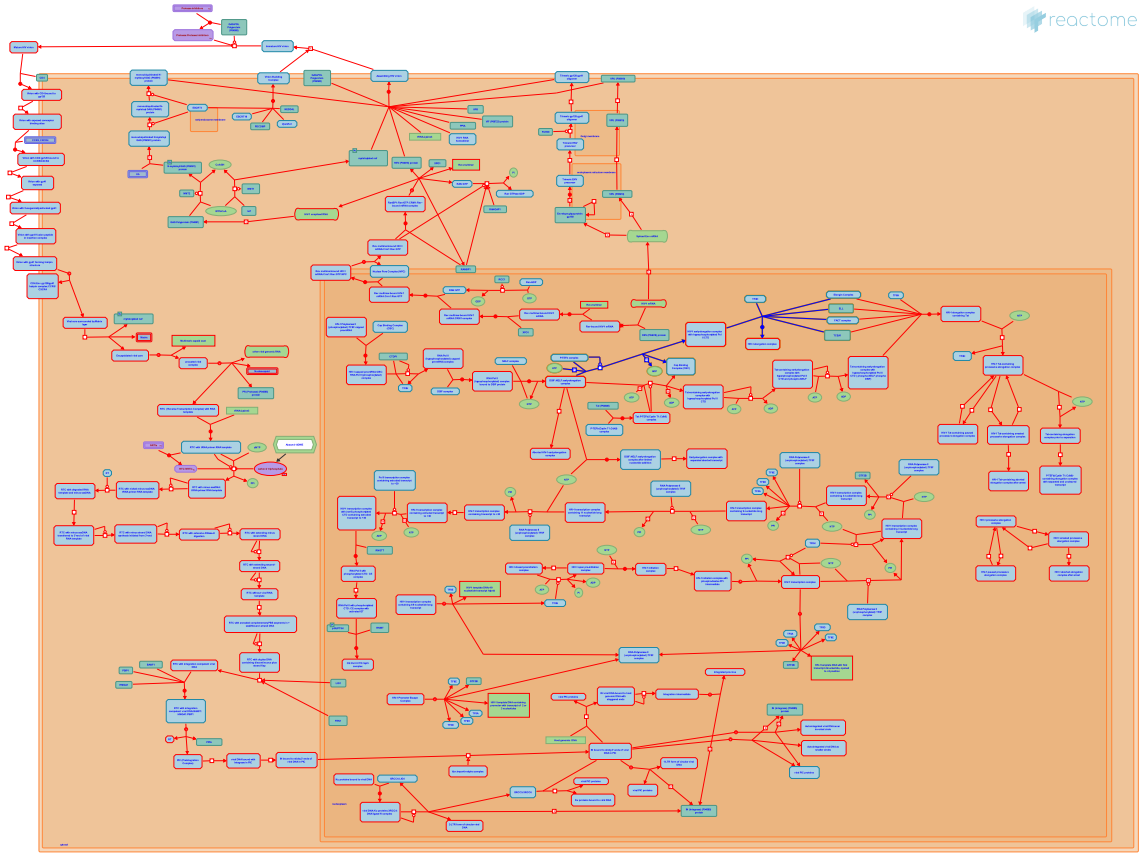
Formation of HIV elongation complex in the absence of HIV Tat ↗

Stable identifier: R-HSA-167152

Compartments: nucleoplasm

Diseases: Human immunodeficiency virus infectious disease

Inferred from: Formation of RNA Pol II elongation complex (Homo sapiens)



During the formation of the HIV elongation complex in the absence of HIV Tat, eongation factors are recruited to form the HIV-1 elongation complex (Hill and Sundquist 2013) and P-TEFb complex hyperphosphorylates RNA Pol II CTD (Hermann and Rice, 2005, Zhou et al., 2000).

Editions

2005-07-27	Authored	Matthews, L., Rice, AP.
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Hyperphosphorylation (Ser2) of RNA Pol II CTD by P-TEFb complex ↗

Location: Formation of HIV elongation complex in the absence of HIV Tat

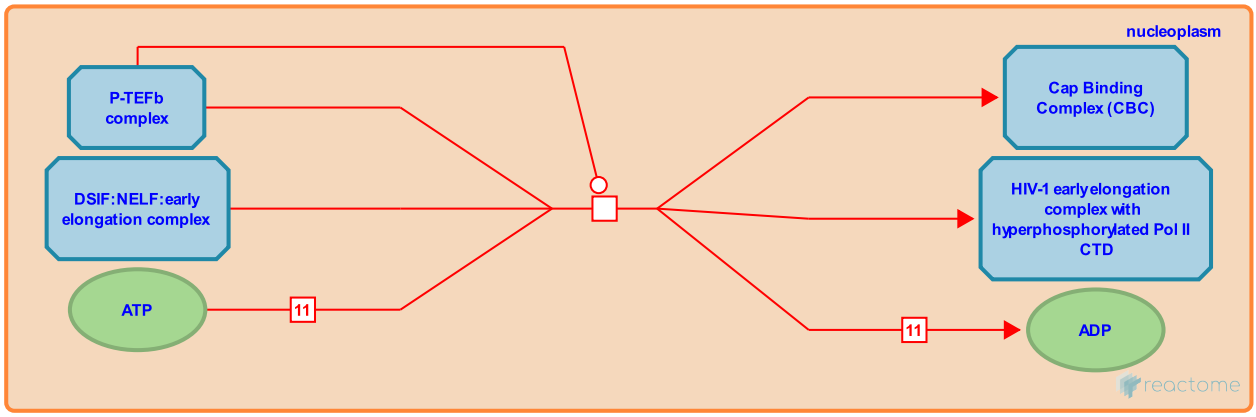
Stable identifier: R-HSA-167084

Type: transition

Compartments: nucleoplasm

Diseases: Human immunodeficiency virus infectious disease

Inferred from: Hyperphosphorylation (Ser2) of RNA Pol II CTD by P-TEFb complex (Homo sapiens)



The association between Tat, TAR and P-TEFb is believed to bring the catalytic subunit of P-TEFb(Cyclin T1:Cdk9) in close proximity to Pol II where it hyperphosphorylates the CTD of Pol II (Herrmann et al., 1995; Zhou et al. 2000). In the presence of Tat, P-TEFb(Cyclin T1:CDK9) has been shown to phosphorylate serine 5 in addition to serine 2 suggesting that modification of the substrate specificity of CDK9 may play a role in the ability of Tat to promote transcriptional elongation (Zhou et al. 2000).

Followed by: Recruitment of elongation factors to form HIV-1 elongation complex

Literature references

Kashanchi, F., Brady, JN., Halanski, MA., Radonovich, MF., Price, DH., Zhou, M. et al. (2000). Tat modifies the activity of CDK9 to phosphorylate serine 5 of the RNA polymerase II carboxyl-terminal domain during human immunodeficiency virus type 1 transcription. *Mol Cell Biol, 20*, 5077-86. ↗

Rice, AP., Herrmann, CH. (1995). Lentivirus Tat proteins specifically associate with a cellular protein kinase, TAK, that hyperphosphorylates the carboxyl-terminal domain of the large subunit of RNA polymerase II: candidate for a Tat cofactor. *J Virol, 69*, 1612-20. ↗

Editions

2005-07-27	Authored	Matthews, L., Rice, AP.
2005-07-27	Edited	Matthews, L.

Recruitment of elongation factors to form HIV-1 elongation complex ↗

Location: Formation of HIV elongation complex in the absence of HIV Tat

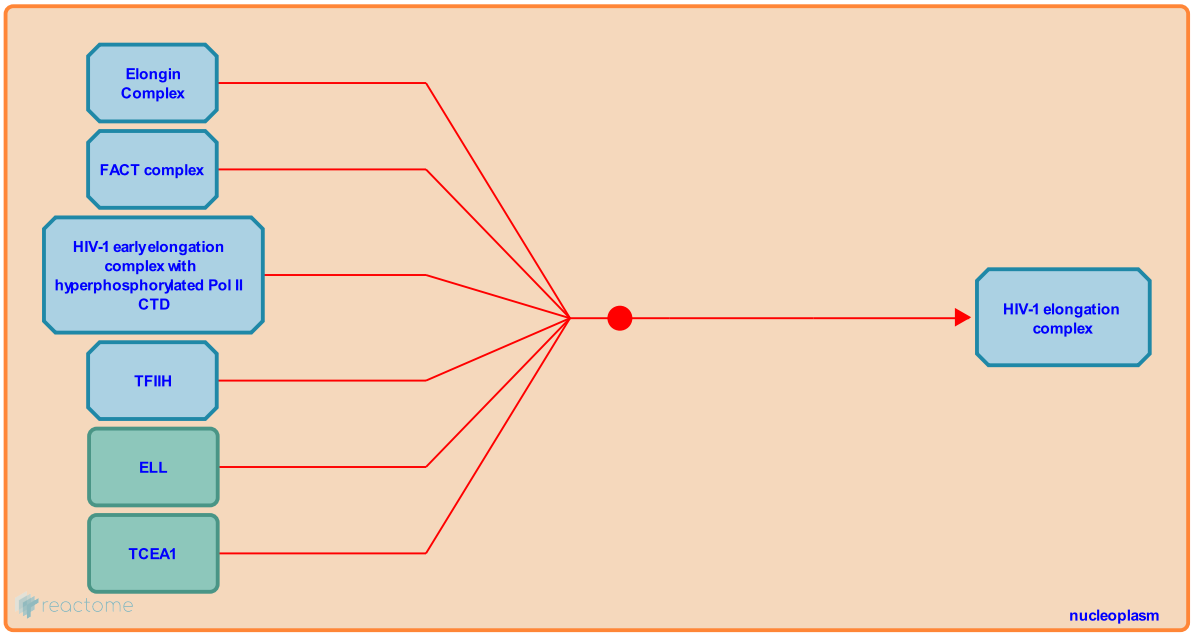
Stable identifier: R-HSA-167077

Type: binding

Compartments: nucleoplasm

Diseases: Human immunodeficiency virus infectious disease

Inferred from: Recruitment of elongation factors to form elongation complex (Homo sapiens)



Eongation factors are recruited to form the HIV-1 elongation complex (Hill and Sundquist 2013).

Preceded by: Hyperphosphorylation (Ser2) of RNA Pol II CTD by P-TEFb complex

Literature references

Sundquist, WI., Hill, CP. (2013). Building a super elongation complex for HIV. *Elife, 2*, e00577. ↗

Editions

2005-07-27	Authored	Matthews, L., Rice, AP.
2005-07-27	Edited	Matthews, L.

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