



POSTER PRESENTATION

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# Definition of the interacting interfaces of Apobec3G and HIV-1 Vif using MAPPIT mutagenesis analysis

Delphine Lavens<sup>1,2\*</sup>, Frank Peelman<sup>1,2</sup>, José Van der Heyden<sup>1,2</sup>, Isabel Uyttendaele<sup>1,2</sup>, Dominiek Catteeuw<sup>1,2</sup>, Bertrand Van Schouwbroeck<sup>3</sup>, Julia Kurth<sup>3</sup>, Sabine Hallenberger<sup>3</sup>, Reginald Clayton<sup>3</sup>, Jan Tavernier<sup>1,2</sup>

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## Background

The host restriction factor Apobec3G is a cytidine deaminase that incorporates into HIV-1 virions and interferes with viral replication. The HIV-1 accessory protein Vif subverts Apobec3G by targeting it for proteasomal degradation. We studied the Apobec3G homomerisation and the interaction of Apobec3G with Vif in detail.

## Methods

We used the MAPPIT two-hybrid technique to analyse the Apobec3G-Apobec3G and the Apobec3G-Vif interactions in intact human cells. MAPPIT is based on the functional complementation of a cytokine receptor signalling pathway.

## Results

We propose a model in which Apobec3G N-terminal domains symmetrically interact via a head-to-head interface containing residues 122 RLYYFW 127. Mutations in the head-to-head interface abrogate the Apobec3G-Apobec3G interaction. All mutations that inhibit Apobec3G-Apobec3G binding also inhibit the Apobec3G-Vif interaction, indicating that the head-to-head interface plays an important role in the interaction with Vif. Only the D128K, P129A and T32Q mutations specifically affect the Apobec3G-Vif association. In our model, D128, P129 and T32 cluster at the edge of the head-to-head interface, possibly forming a Vif binding site composed of two Apobec3G molecules.

## Discussion

We propose that Vif either binds directly at the Apobec3G head-to-head interface or associates with an RNA-stabilized Apobec3G oligomer.

## Author details

<sup>1</sup>University of Ghent, Ghent, Belgium. <sup>2</sup>VIB, Ghent, Belgium. <sup>3</sup>TIBOTEC, Mechelen, Belgium.

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\* Correspondence: [Delphine.Lavens@UGent.be](mailto:Delphine.Lavens@UGent.be)

<sup>1</sup>University of Ghent, Ghent, Belgium