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Mapping of the binding sites between Apobec3G and the HIV-1 Gag and Vif proteins

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The HIV-1 virus affects the immune system leading to the development of AIDS. Because HIV-1 codes for only a handful of viral proteins, it makes extensive use of host proteins to complete its lifecycle. Host restriction factors are part of the defence mechanism against viral infections and interfere with infection and replication of the virus. Viruses on the other hand possess several proteins that counteract these antiviral responses. Due to its hypermutative character, the HIV-1 virus develops resistance to long-term administered drugs. Interference with selected host-viral protein interactions offers a new platform to the continuous need for new drug targets.

The host restriction factor Apobec3G is a cytidine deaminase that incorporates into HIV-1 virions and inhibits virus replication. The viral uptake of Apobec3G depends on the HIV-1 gag protein. The HIV-1 accessory protein Vif targets Apobec3G for proteasomal degradation and thus eliminates this antiviral activity. We modelled the Apobec3G dimer based on the Apobec2 crystal structure and used the MAPPIT mammalian two-hybrid technique to analyse the Apobec3G-Apobec3G and the Apobec3G-Vif and Apobec3G-Gag interactions in great detail. Results from both site-directed and random mutagenesis approaches will be presented unveiling the contact site residues involved in these protein interactions.