GANP interacts with APOBEC3G and facilitates its encapsidation into the virions to reduce HIV-1 infectivity.

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The ssDNA-dependent deoxycytidine deaminase apolipoprotein B mRNA-editing, enzyme-catalytic, polypeptide-like 3G (A3G) is a potent restrictive factor against HIV-1 virus lacking viral-encoded infectivity factor (Vif) in CD4(+) T cells. A3G antiretroviral activity requires its encapsulation into HIV-1 virions. In this study, we show that germinal center-associated nuclear protein (GANP) is induced in activated CD4(+) T cells and physically interacts with A3G. Overexpression of GANP augments the A3G encapsidation into the virion-like particles and ΔVif HIV-1 virions. GANP is encapsidated in HIV-1 virion and modulates A3G packaging into the cores together with cellular RNAs, including 7SL RNA, and with unspliced HIV-1 genomic RNA. GANP upregulation leads to a significant increase in A3G-catalyzed G→A hypermutation in the viral genome and suppression of HIV-1 infectivity in a single-round viral infection assay. Conversely, GANP knockdown caused a marked increase in HIV-1 infectivity in a multiple-round infection assay. The data suggest that GANP is a cellular factor that facilitates A3G encapsidation into HIV-1 virions to inhibit viral infectivity.

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