Fig. 1 A3G/B chimera is located in the cytoplasm and active in wt HIV and HIVΔvif restriction. (A) Antiviral activity of A3 proteins, chimeras and A3B SYR. Single cycle replication assays were performed with VLPs produced in 293T cells co-transfected with varied amounts of A3B, A3G, A3G/B and A3B/G and A3BSYYR plasmids, and pCMVHIV-1 Δvif, pUCHR-GFPLuc and pCMV-VSV-G. 293T cells were infected with ELISA-standardized amounts of VLPs and productive infection was measured as luciferase activity. Values are presented as percent infectivity relative to virus produced in absence of A3. Data are the means of 3 independent experiments and error bars represent the standard deviation. (B) Localization of A3B mutants in 293T cells. 293T cells were transiently transfected with plasmids encoding A3B-HA, A3G/B-HA, A3G-HA, A3B/G-HA and A3B-HA mutants SY, YR and SYYR. Cells were fixed, permeabilized and stained with mouse anti-HA antibodies and Alexa Fluor 488 goat anti-mouse IgG. (C) Comparison of antiviral activity of A3 proteins against HIV-1 wild type (wt) and HIV-1 Δvif. Single cycle replication assays were performed with VLPs produced in 293T cells co-transfected with 0.25 μg of A3B, A3G, A3G/B and A3B/G and A3B SYR plasmids, and pCMVHIV-1 Δvif or pCMVHIV-1 wt, pUCHR-GFPLuc and pCMV-VSV-G. Experiments were performed as detailed in part A.