Supporting Information

Hauber et al. 10.1073/pnas.0811827106

SI Materials and Methods

Viruses. HIV-1 stocks were generated by transient transfection of 3×10^6 293T cells with 10 μg of plasmid encoding either the T cell-tropic (X4) strain NL4/3 (1), the multidrug-resistant isolate BE4 (2), the X4-tropic reporter virus NL4/3Luc (in which the *nef* gene was substituted with a cDNA encoding firefly luciferase by using PCR technology), or the R5-tropic reporter virus NL4/3–92TH014Luc (3) in OptiMEM I (Invitrogen) without antibiotics and 30 μL of polyethylenimine (1 mg/mL) according to the manufacturer's protocol (Polysciences). At day 2 after transfection, viral supernatants were collected and passed through a 0.2-μm pore size filter, and $p24^{Gag}$ antigen levels were determined by ELISA (Innotest HIV p24 Antigen mAb; Innogenetics). Vesicular stomatitis virus glycoprotein-pseudotyped NL4/3 virus stocks were generated as previously described in detail (4). The primary HIV-1 isolates 92TH026 (R5) and 93BR020 (R5X4) were obtained as cell-free virus stocks from the National Institutes of Health AIDS Research and Reference Reagent Program.

1. Adachi A, et al. (1986) Production of acquired immunodeficiency syndrome-associated retrovirus in human and nonhuman cells transfected with an infectious molecular clone. *J Virol* 59:284–291.
2. Hauber I, et al. (2005) Identification of cellular deoxyhypusine synthase as a novel target for antiretroviral therapy. *J Clin Invest* 115:76–85.
3. Papkalla A, Munch J, Otto C, Kirchhoff F (2002) Nef enhances human immunodeficiency virus type 1 infectivity and replication independently of viral coreceptor tropism. *J Virol* 76:8455–8459.
4. Sarkar I, Hauber I, Hauber J, Buchholz F (2007) HIV-1 proviral DNA excision using an evolved recombinase. *Science* 316:1912–1915.

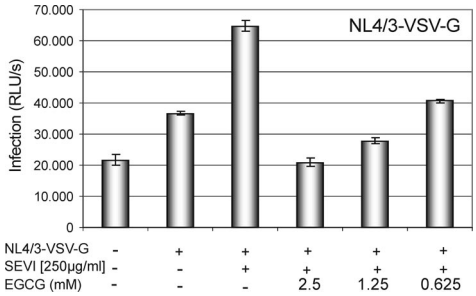

Fig. S1. The SEVI effect on the infectivity of HIV pseudotypes is abrogated by EGCG. Infection of Jurkat 1G5 cells using vesicular stomatitis virus glycoprotein (VSV-G)-pseudotyed NL4/3 virions was performed. Before infection, virions were incubated for 20 min in the presence and absence of the indicated concentrations of SEVI and EGCG. Cell cultures were assayed for luciferase activity at 24 h after infection as described before.

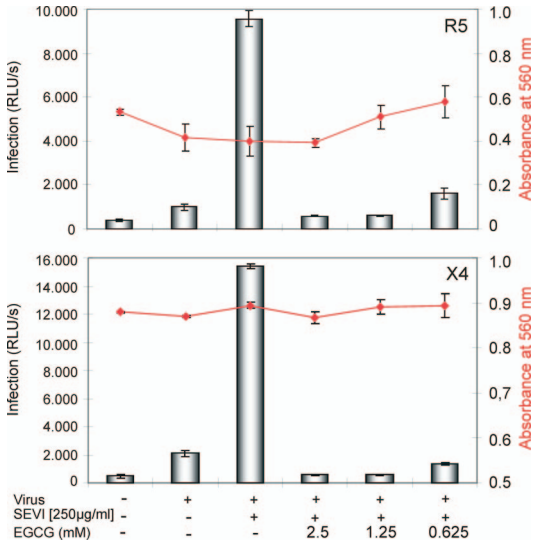

Fig. S2. EGCG inhibits SEVI-mediated HIV infection of primary human cells. PBMCs were infected with luciferase-expressing R5- or X4-tropic HIV-1. Before infection, virions were incubated for 20 min in the presence and absence of the indicated concentrations of SEVI and EGCG. At 24 h after infection, cell cultures were assayed for luciferase activity (bars) and cell viabilities (red plots) as described before. Infections were performed in triplicate.

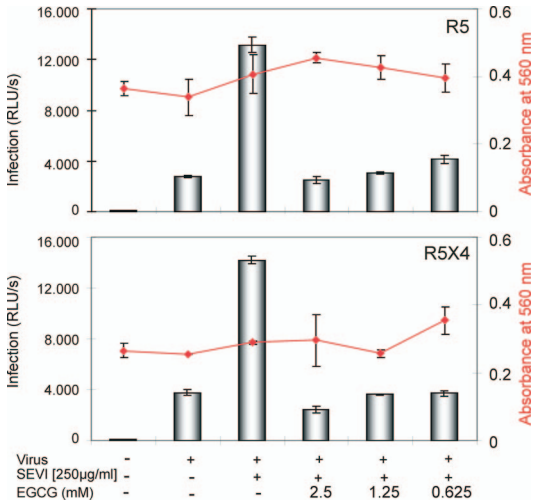

Fig. S3. Analysis of a primary R5 and R5X4 HIV-1 isolate with or without SEVI and EGCG in TZM-bl cells, which contain a stably integrated HIV-LTR-firefly luciferase construct. Before infection, virions were incubated for 20 min in the presence and absence of the indicated concentrations of SEVI and EGCG. At 24 h after infection, cell cultures were assayed for luciferase activity (bars) and cell viabilities (red plot) as described before. Infections were performed in triplicate.

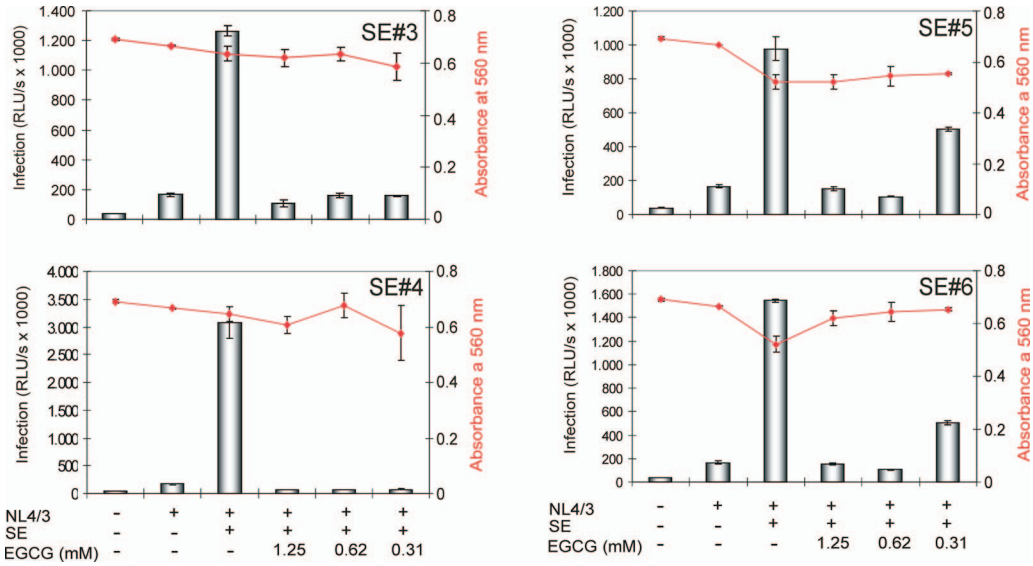

Fig. S4. EGCG inhibits SE-mediated enhancement of HIV-1 de novo infection. HIV-1 NL4/3 was exposed for 20 min to individual SE samples 3–6 (1:4 dilution) in the presence or absence of the indicated EGCG concentrations. Subsequently, these mixtures were used for infection of TZM-bl cell cultures. Luciferase activity (bars) and cell viability (red plot) were determined at 24 h after infection as described before. Error bars represent 3 independent experiments.