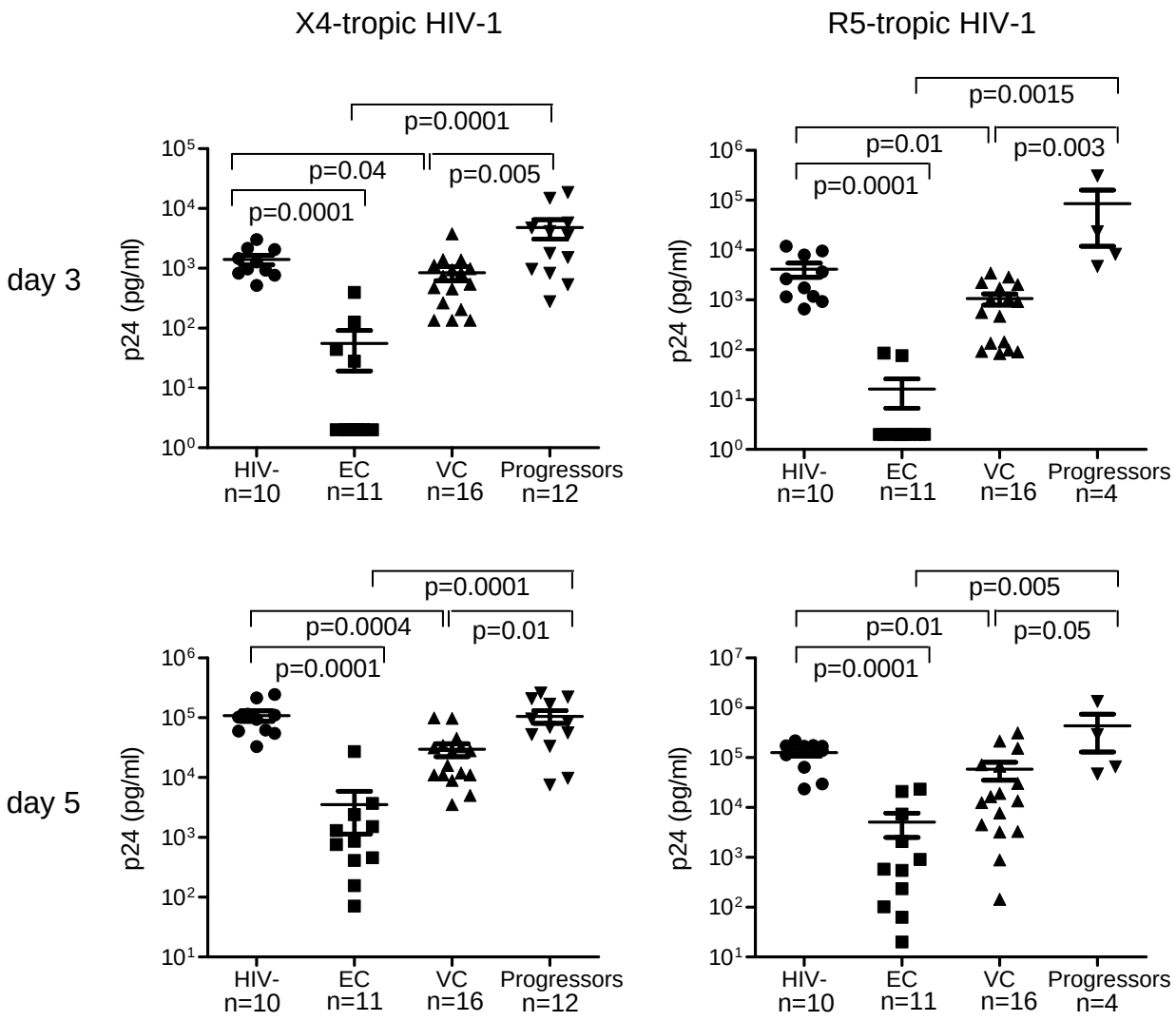
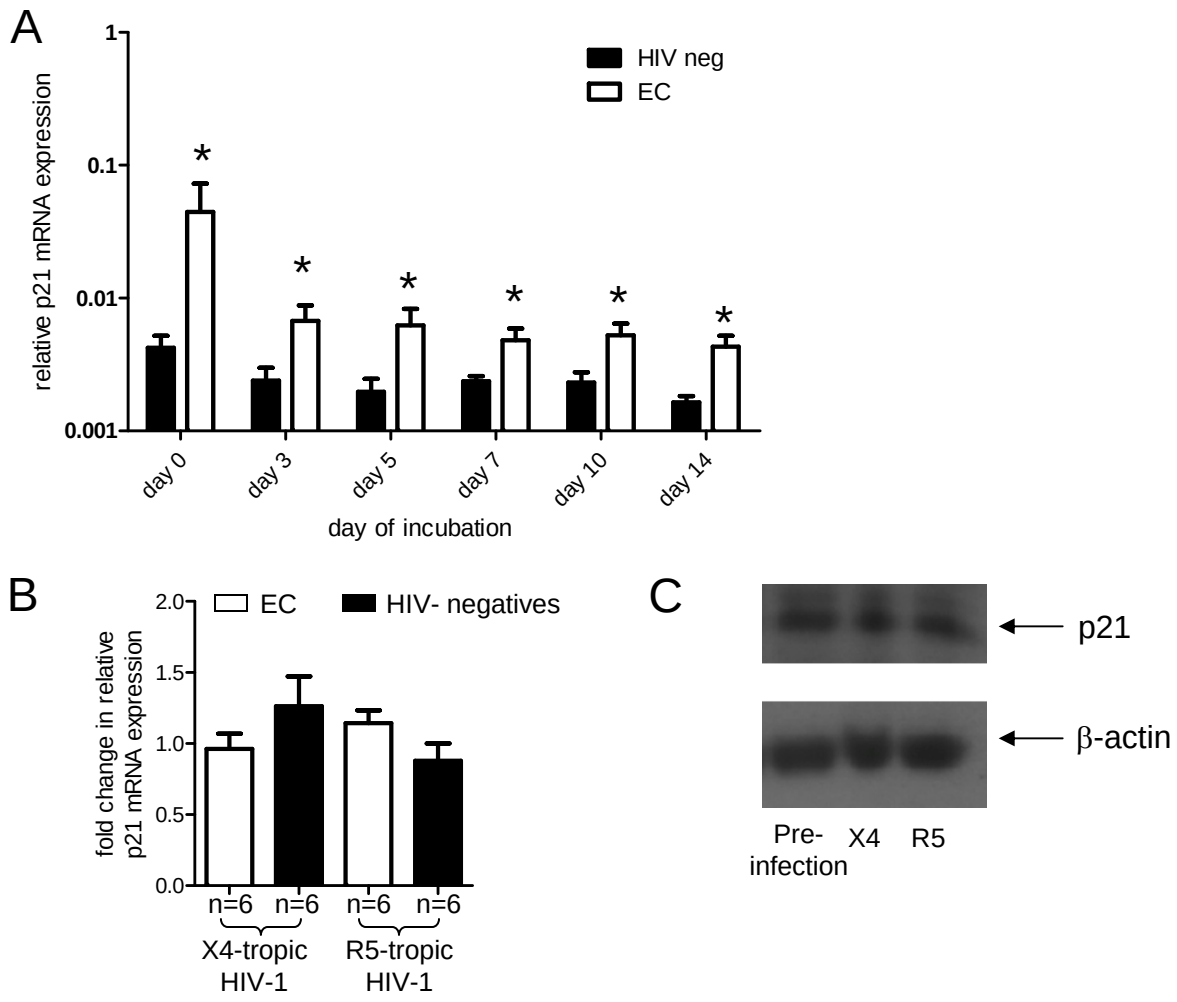
Supplemental Figure 1



Supplemental figure legends

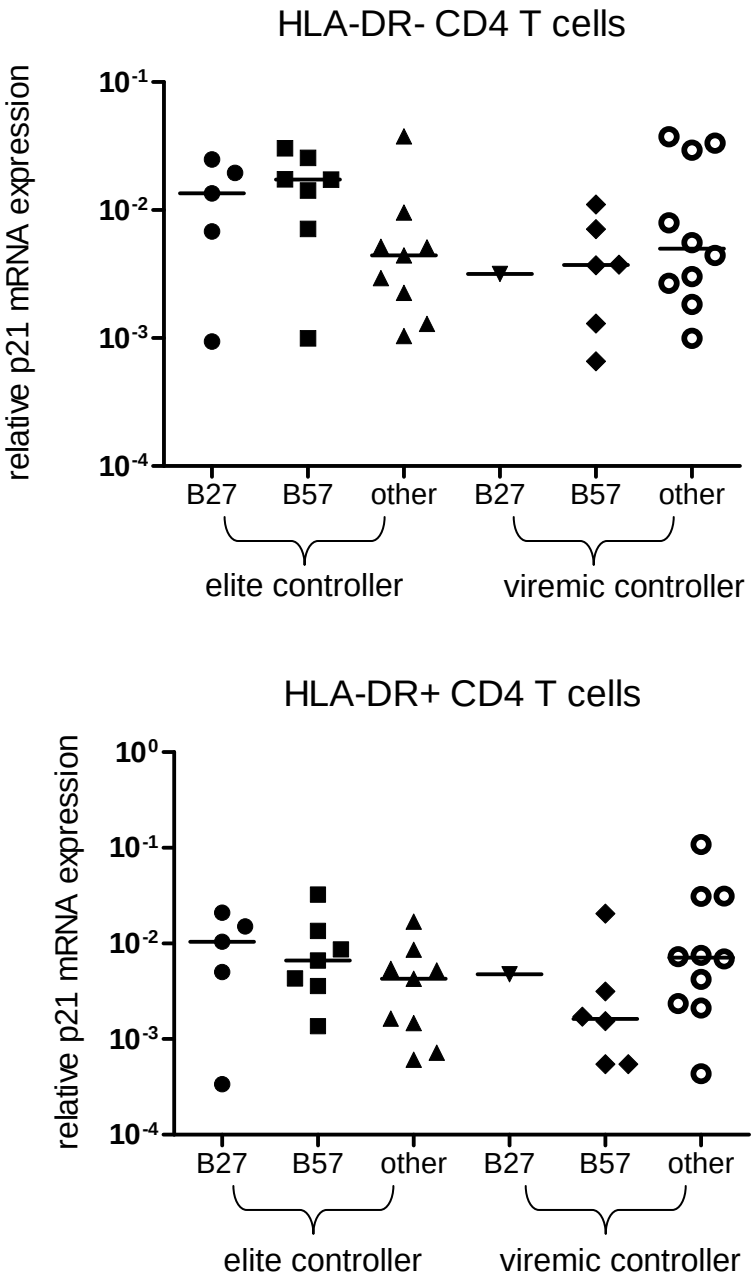
Supplemental Figure 1: Reduced p24 antigen production in activated CD4 T cells from elite controllers after *ex vivo* infection. Activated CD4 T cells were infected with the X4-tropic primary isolate 92HT599 and the primary R5-utilizing strain 91US056; p24 levels in the supernatant were measured on day 3 and day 5. Results demonstrate data from indicated patients from which culture supernatant from day 3 and day 5 was available for analysis. Baseline p24 levels in autologous cells without exogenous HIV-1 infection were subtracted as background. Significance was tested using Mann-Whitney U test.

Supplemental Figure 2



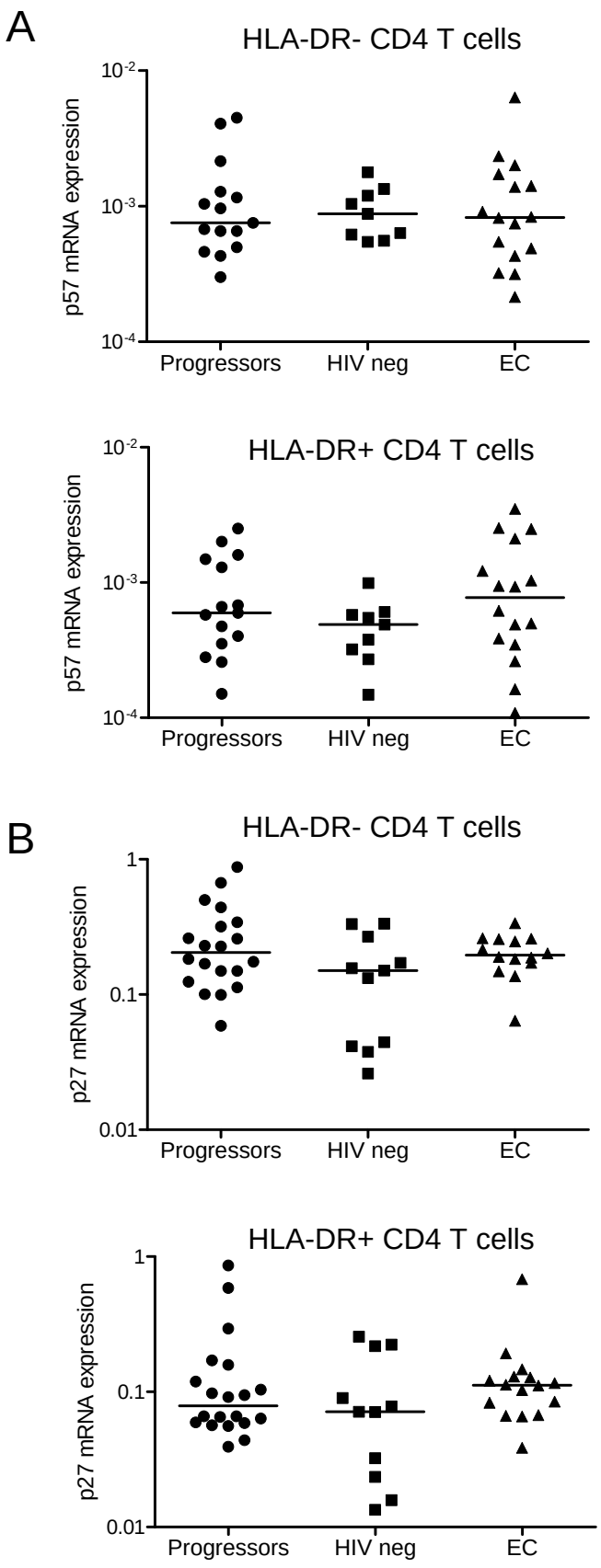
Supplemental Figure 2: Effects of *in vitro* activation and HIV-1 infection on p21 expression in CD4 T cells. (A) Relative p21 mRNA expression in CD4 T cells following *ex vivo* activation with CD3/CD8 bi-specific antibodies and IL-2 for indicated time period. Data reflect mean and standard deviation from n=10 elite controllers and n=7 HIV-1 negative persons (*$p<0.05$). (B-C) p21 mRNA and protein expression in CD4 T cells before and 24 hours after HIV-1 infection of activated CD4 T cells with X4- or R5-tropic HIV-1. (B): Data show mean and standard deviation for the fold-change of relative p21 mRNA expression in indicated study subjects. (C) demonstrates p21 western blots from CD4 T cells before and after infection with X4 – and R5-tropic HIV-1. One representative experiment out of three is shown.

Supplemental Figure 3



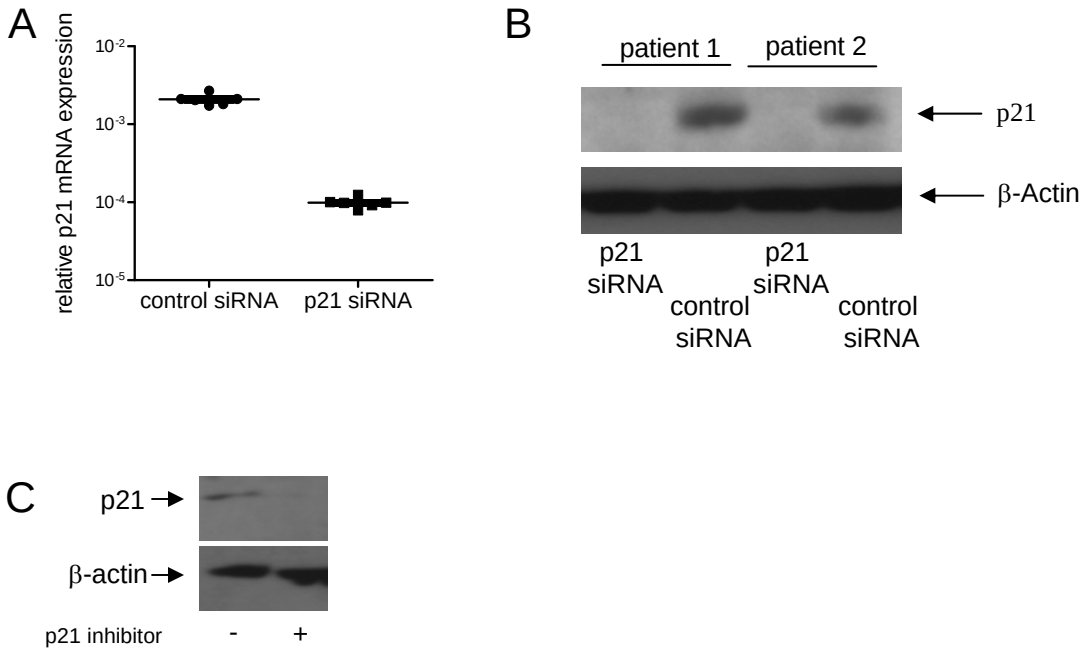
Supplemental Figure 3: p21 expression in carriers of HLA-B57 and –B27. Data indicate relative p21 mRNA expression in HLA-DR- and HLA-DR+ CD4 T cells from carriers of HLA-B57, HLA-B27 or other HLA class I alleles (other) in elite controllers and viremic controllers. No significant differences were observed between the different groups using Mann Whitney U tests.

Supplemental Figure 4



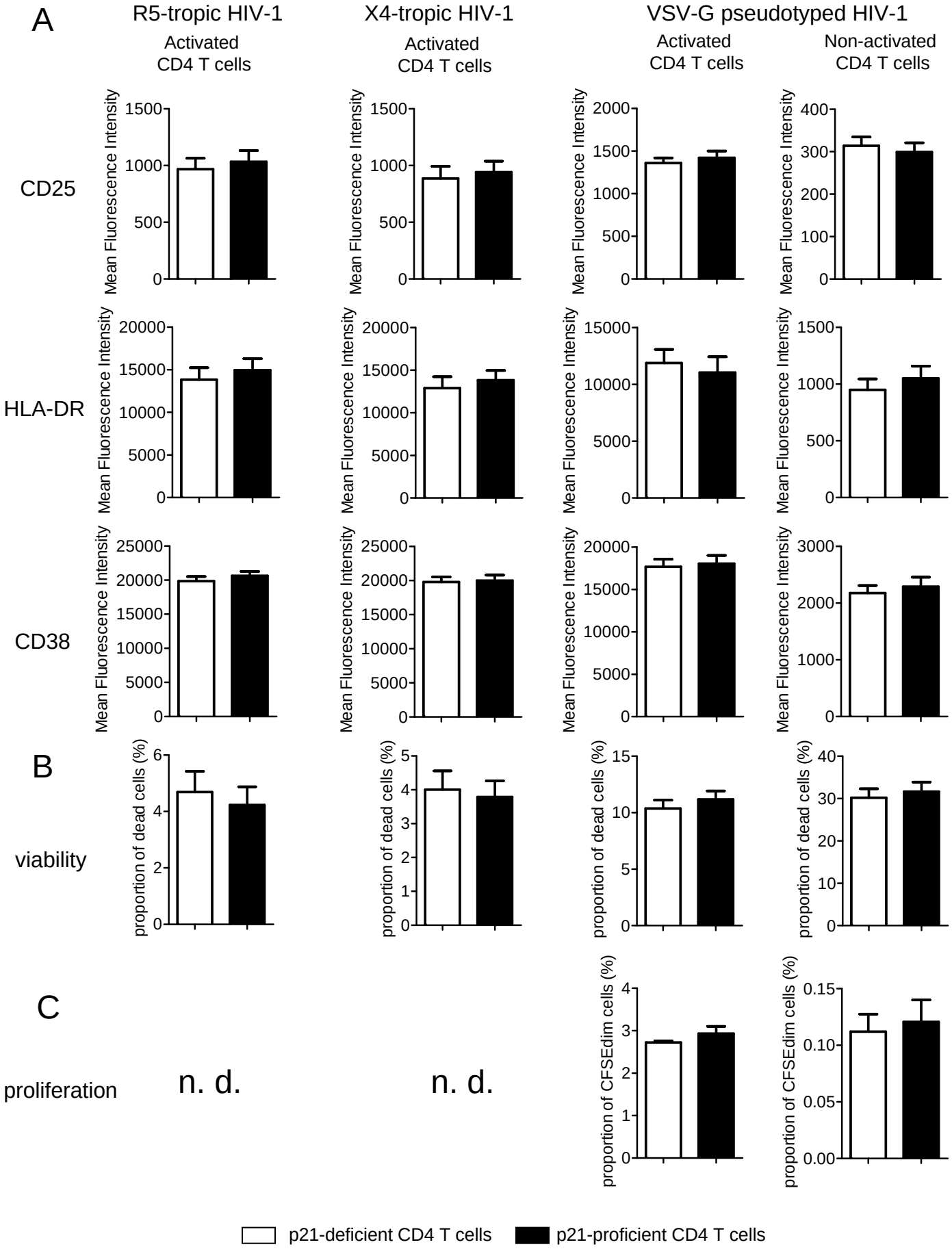
Supplemental Figure 4: Relative mRNA expression of the CDKI p27 and p57 in HLA-DR- and HLA-DR+ CD4 T cells from elite controllers (EC), HIV-1 negative persons, and HIV-1 progressors.

Supplemental Figure 5



Supplemental Figure 5: Inhibition of p21 by siRNA silencing or by a pharmaceutical small molecule inhibitor. (A-B): Downregulation of p21 by siRNA. Results show relative p21 mRNA (A) and protein (B) expression in CD4 T cells 24 hours after electroporation with p21-specific or control siRNA. (C): Inhibition of p21 by a pharmaceutical inhibitor inducing selective proteasomal degradation of p21 protein. CD4 T cells were incubated with or without p21 inhibitor for 36 hours. Subsequently, cells were immunoblotted with antibodies against β-actin or p21.

Supplemental Figure 6



Supplemental Figure 6: p21 inhibition does not affect activation of *in vitro* stimulated CD4 T cells. Mean fluorescence intensity of CD25, CD38, HLA-DR on CD4 T cells (A), proportion of dead CD4 T cells (B) and proportion of proliferating CD4 T cells (C) infected with the indicated HIV-1 viruses in the presence of p21-siRNA or control siRNA (cells infected with R5/X4 tropic virus) or of a pharmacological p21 inhibitor or a DMSO control (cells infected with VSV-G pseudotyped virus). Results were obtained 4 days after infection with X4/R5 tropic viruses; cells infected with VSV-G pseudotyped virus were analyzed 48 hours (activated cells) or 96 hours (non-activated cells) after infection.