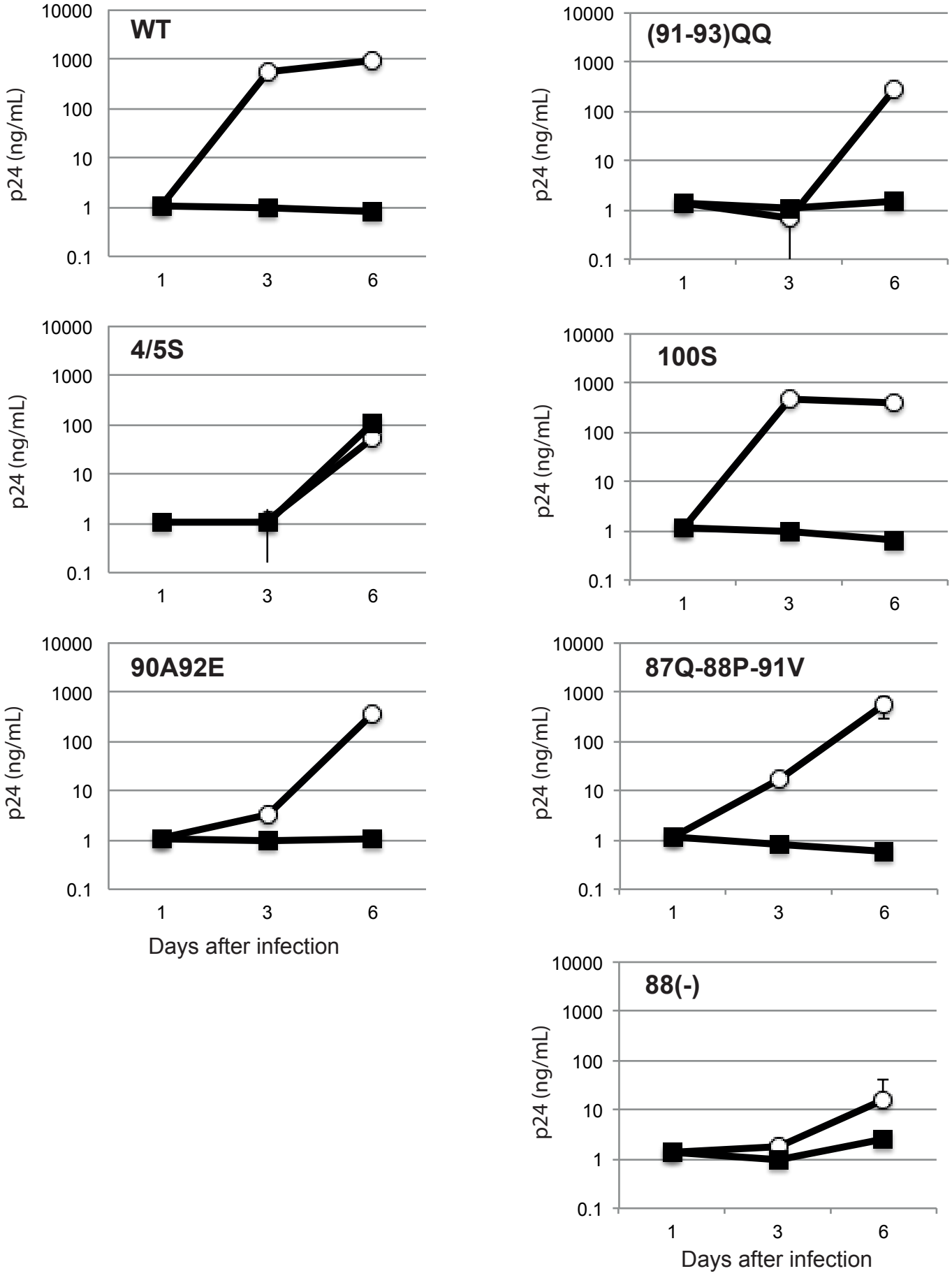

Fig. S1. Growth kinetics of HIV-1 with point mutations in capsid L4/5 in the presence of CM TRIMCyp
HIV-1 variants containing a few amino acid mutations in capsid were inoculated into MT4 cells expressing CM TRIMCyp (black squares) or CM TRIM5α SPRY (-) (white circles) and culture supernatants were periodically assayed for the levels of p24. The results shown are representative of at least two independent experiments. Error bar shows actual fluctuations between measurements of p24 in duplicate samples.

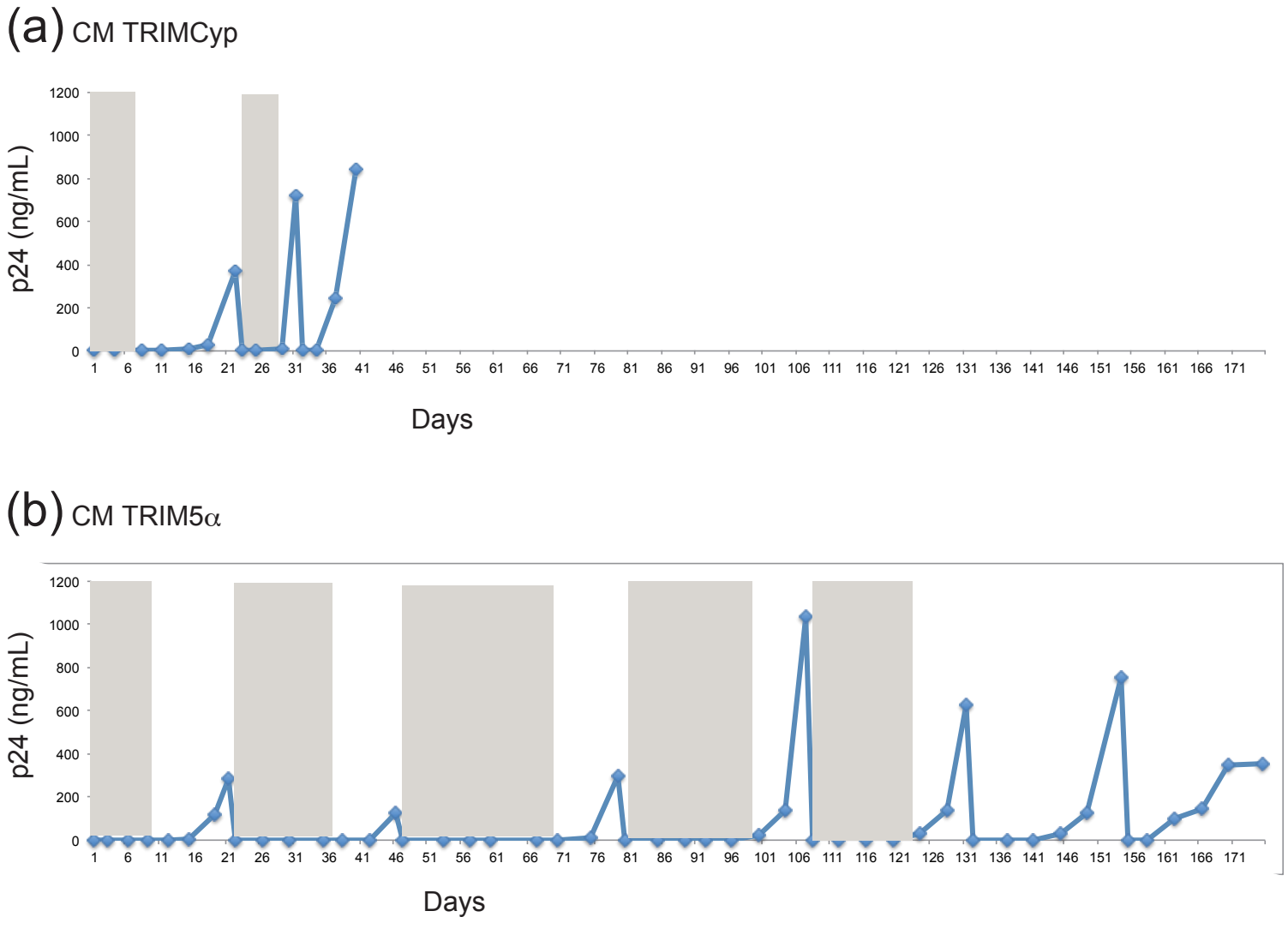

Fig. S2. Adaptation of HIV-1 derivatives to MT4 cells expressing CM TRIMCyp or CM TRIM5α

1 x 104 HIV-1 variants containing mutations in L4/5 (amino acid position 85 to 93 of capsid) were inoculated into MT4 cells infected with Sendai virus expressing CM TRIMCyp (a) and CM TRIM5α (b), and culture supernatants were periodically assayed for the levels of p24. Non-shaded areas denote co-culture with fresh MT4 cells without Sendai virus infection.

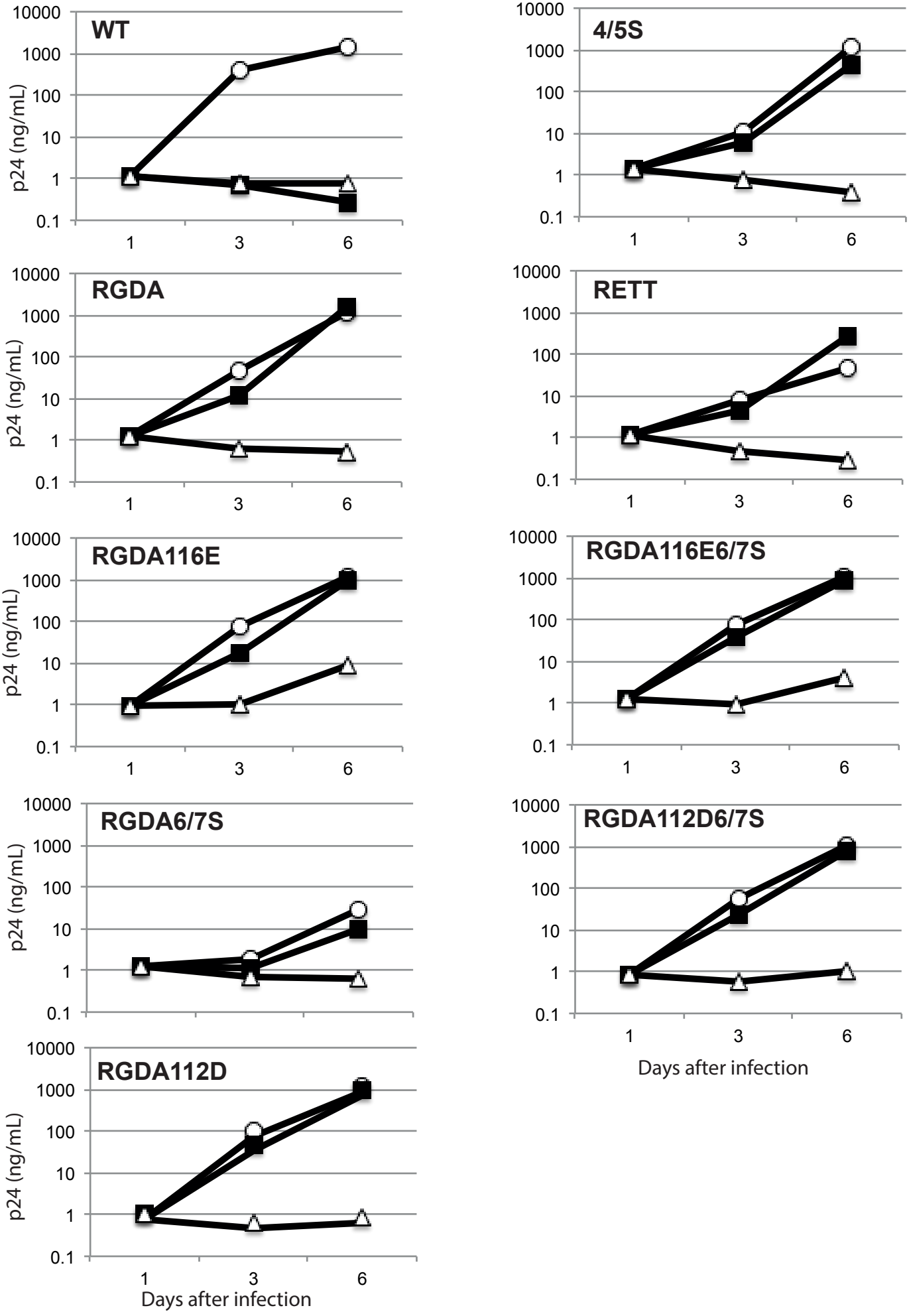

Fig. S3. H87R/A88G/P90D/P93A and H87R/G89E/A92T/P93T mutations in capsid confer a high degree of resistance to CM TRIMCyp

MT4 cells expressing CM TRIMCyp (black squares), CM TRIM5α (white triangles), or CM-SPRY (-) TRIM5α (white circles) were superinfected with the indicated HIV-1 derivatives. The results shown are representative of at least three independent experiments. Error bar shows actual fluctuations between measurements of p24 in duplicate samples

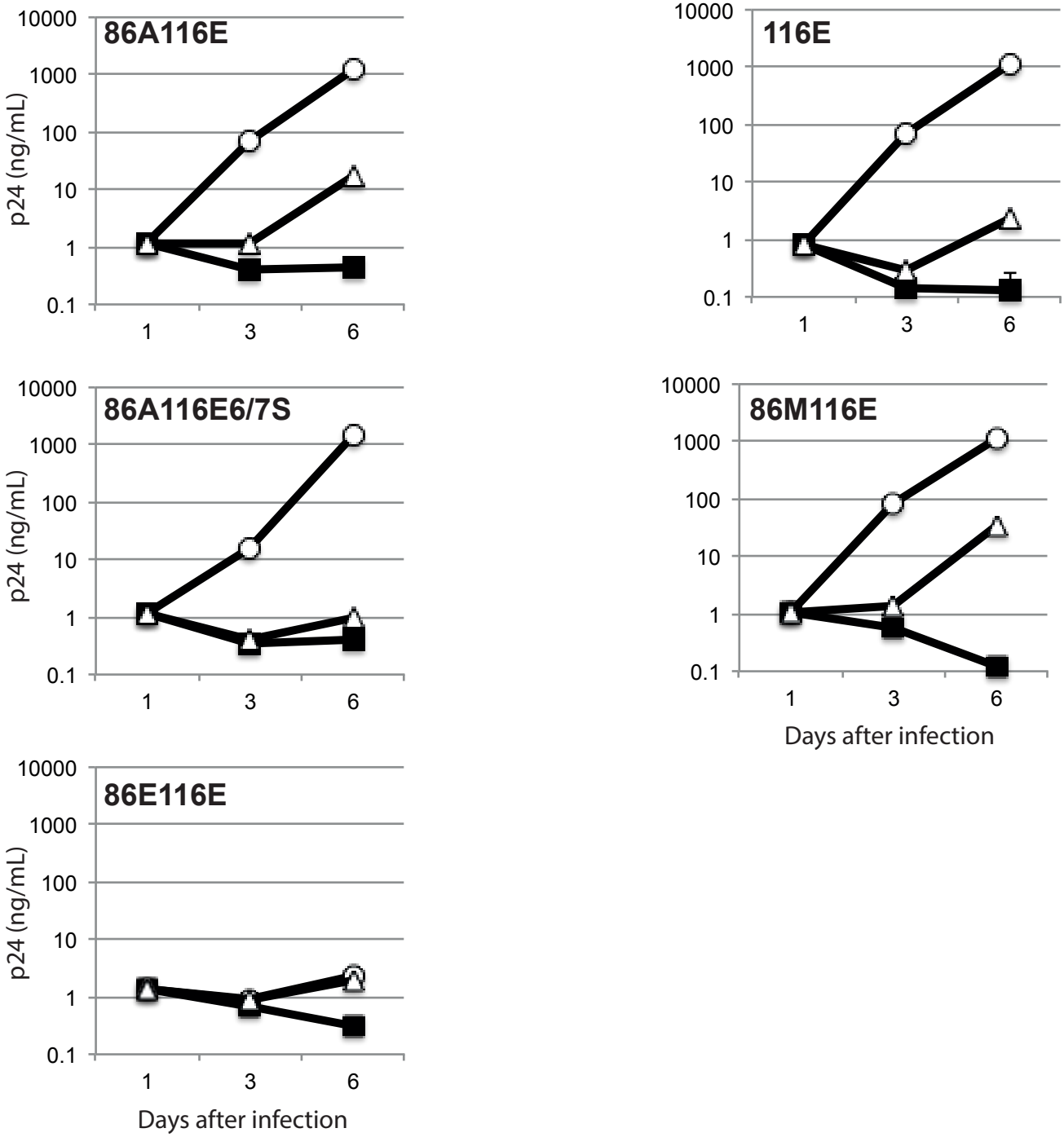

Fig. S4. Novel V86A/G116E mutant shows partial resistance to CM TRIM5α
MT4 cells expressing CM TRIMCyp (black squares), CM TRIM5α (white triangles), or CM-SPRY (-) TRIM5α (white circles) were superinfected with the indicated HIV-1 derivatives. The results shown are representative of at least three independent experiments. Error bar shows actual fluctuations between measurements of p24 in duplicate samples.

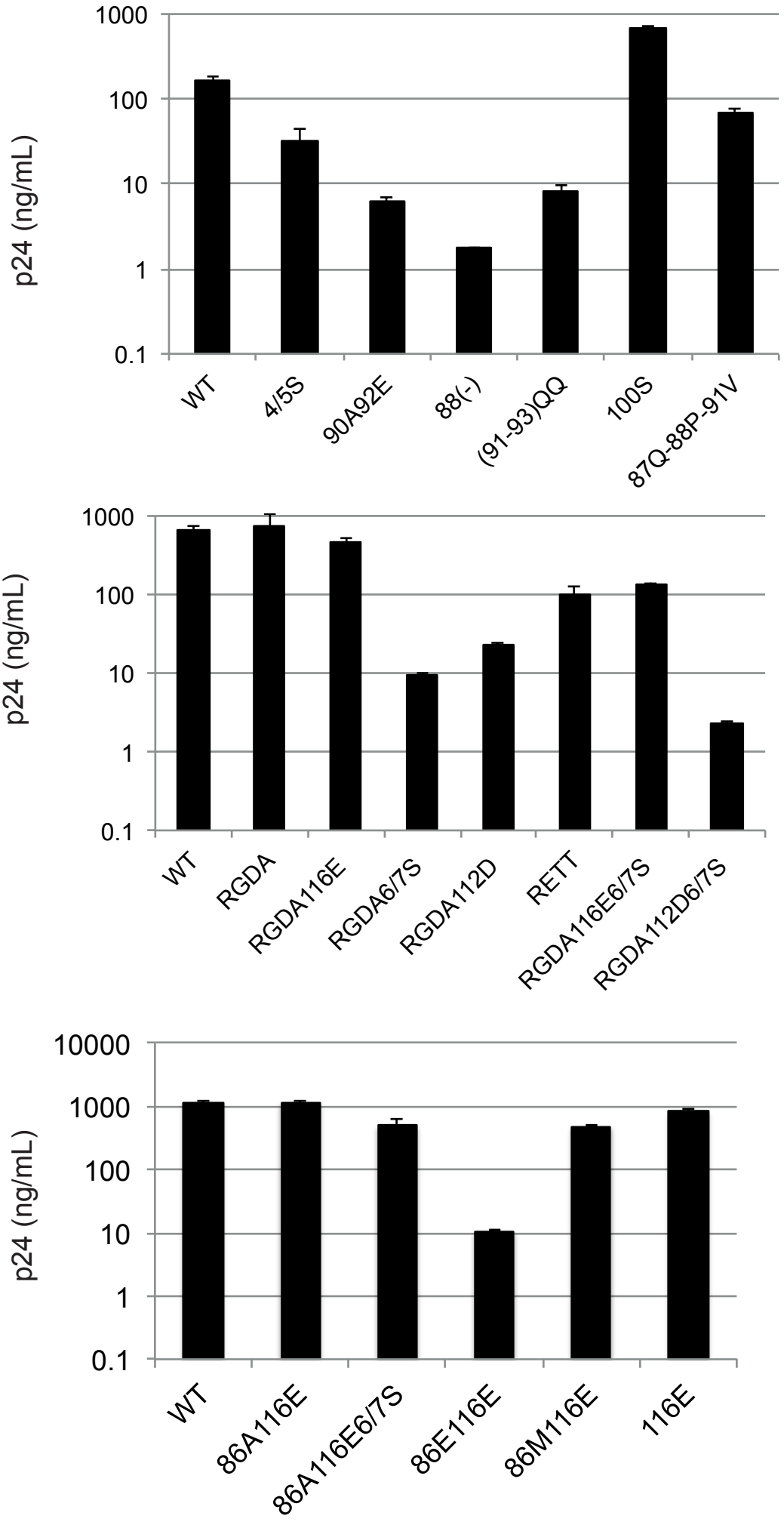

Fig. S5. Growth kinetics of HIV-1 with point mutations in capsid L4/5 in unmodified MT4 cells
HIV-1 variants containing a few amino acid mutations in capsid were inoculated into unmodified MT4 cells and culture supernatants were assayed for the levels of p24 at 9 days after infection.
Error bar shows actual fluctuations between measurements of p24 in duplicate samples.

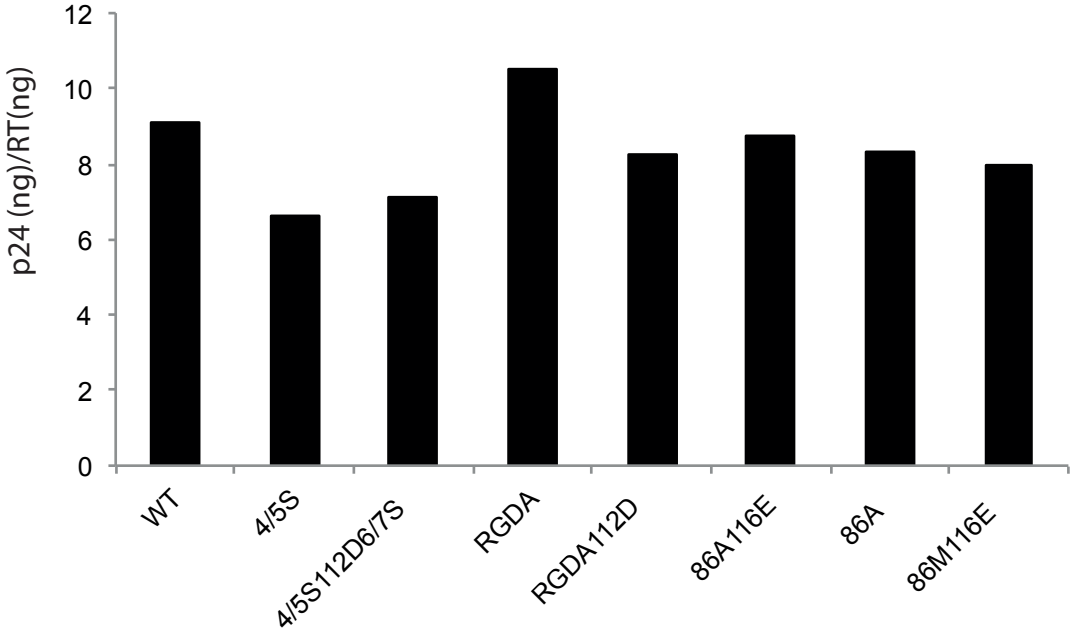

Fig. S6. Comparison between p24 antigen levels and reverse transcriptase activity
Reverse transcriptase activity of each mutant was measured according to the manufacturer's instruction on Reverse Transcriptase Assay, colorimetric (Roche Diagnostics GmbH, Mannheim, Germany). The same culture supernatants containing mutants were subjected to p24 ELISA. Comparison was done by dividing the amount of p24 (ng/mL) by the amount of RT (ng/mL) of that clone. This result is a representative for at least two independent experiments.

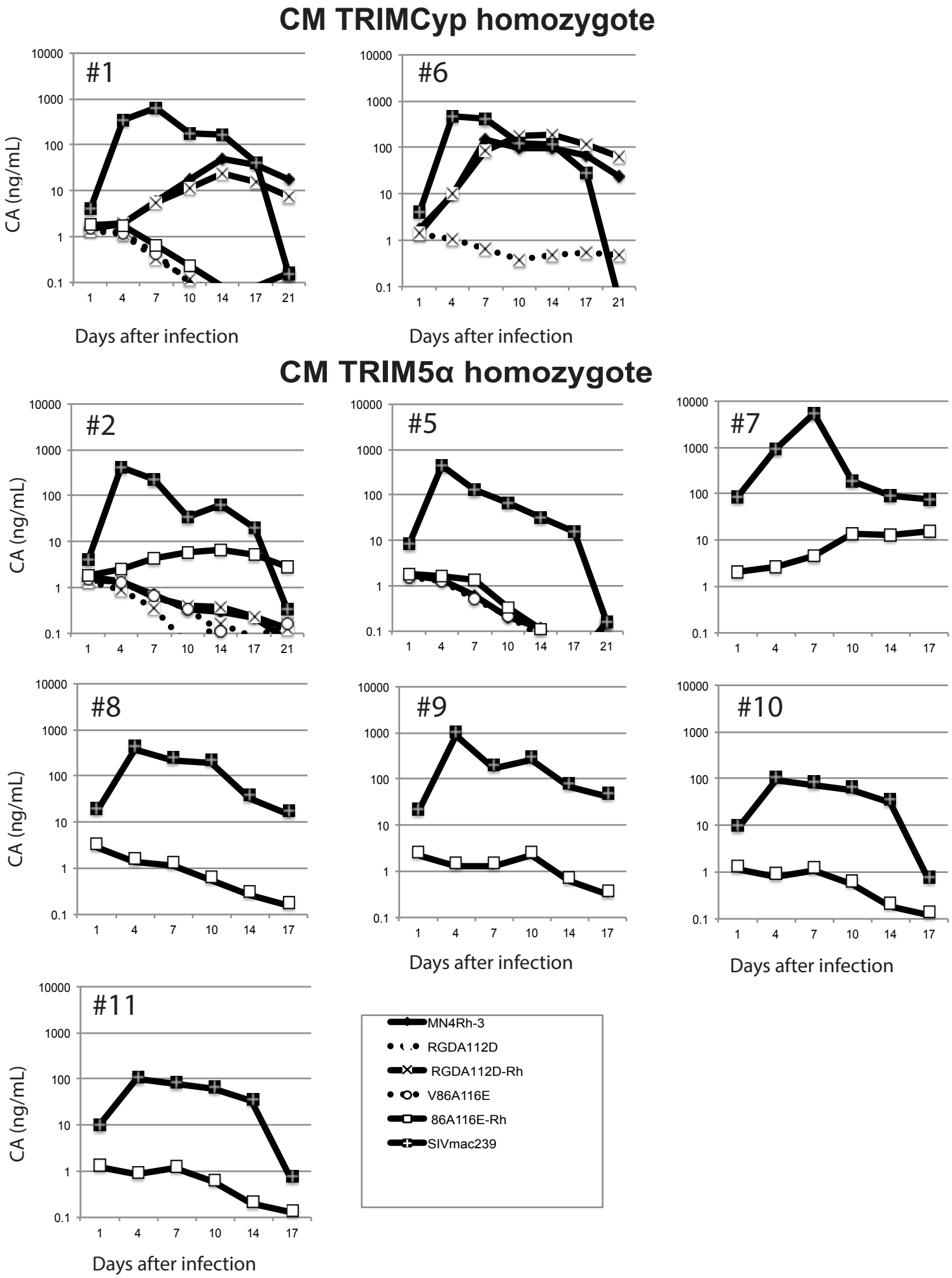

Fig. S7. Replication capabilities of HIV-1 derivatives in different types of CD8-depleted CM PBMC
Virus replication capabilities in two additional CM TRIMCyp homozygotes (#1 and #6) and in seven additional CM TRIM5α homozygotes (#2, #5 and #7-11) were examined periodically by p24 or p27 ELISA assay. The results on #3 CM TRIMCyp homozygote and #4 CM TRIM5α homozygote are shown in Fig. 6. MN4Rh-3 (black line with diamond) and SIVmac239 (black line with plus square) served as controls.