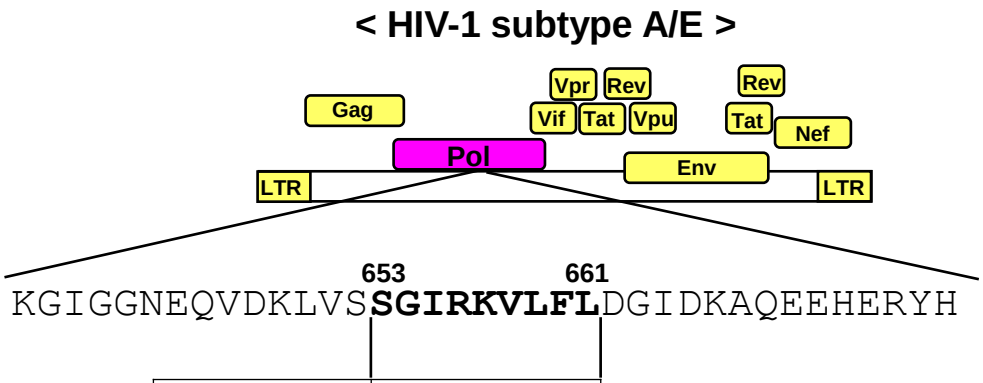

Pol SL9 (WT)	SGIRKVLFL
Pol SL9-1A	AGIRKVLFL
Pol SL9-1T	TGIRKVLFL
Pol SL9-1L	LGIRKVLFL
Pol SL9-1K	KGIRKVLFL
Pol SL9-1Q	QGIRKVLFL

Figure S1. Sequences and positions of SL9 and its mutant epitope peptides.

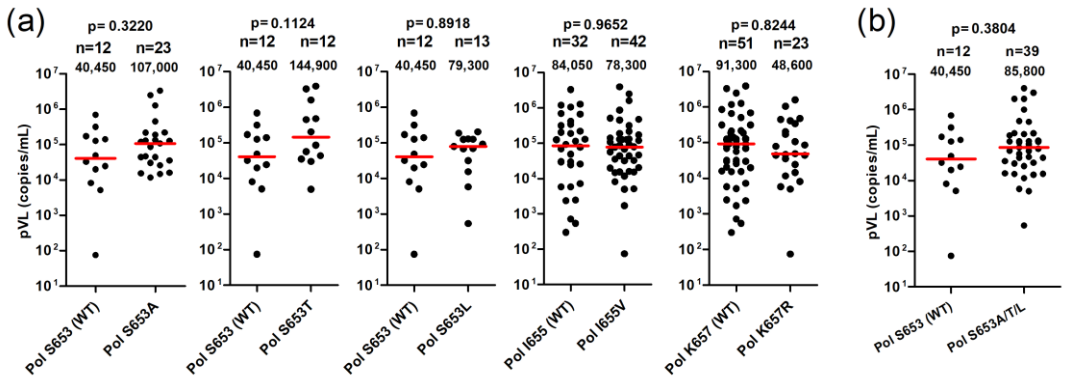

Figure S2. Effects of Pol S653A, Pol S653T, Pol S653L, Pol I655V, and Pol K657R mutations on pVL.

Comparison of pVL between HLA-C*15:05-positive individuals infected with Pol S653 (WT) virus and those with each of Pol S653A, Pol S653T, Pol S653L, Pol I655V, and Pol K657R (a) or with at least one of Pol S653A/T/L mutant viruses (b). The red lines and the values in each graph represent medians of pVL. Statistical analysis was performed by using the Mann-Whitney test.

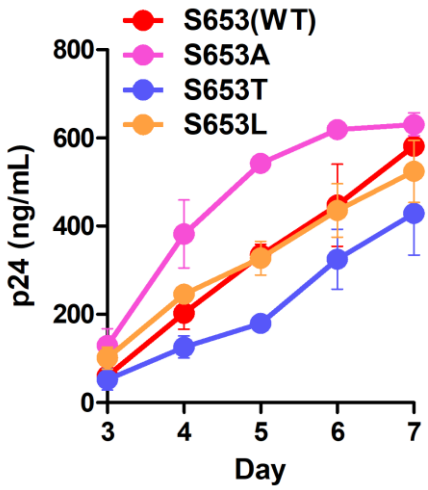

Figure S3. *In vitro* replication capacity of Pol S653A/T/L mutants. 721.221 cells expressing HLA-C*15:05 were infected with 5×10^7 relative luminescence units (RLU) of wild-type virus (93JP-NH1) or each of the 3 mutants (93JP-$NH1_{Pol\ S653A}$, 93JP-$NH1_{Pol\ S653T}$, and 93JP-$NH1_{Pol\ S653L}$) virus. The culture supernatant was then collected from day 3 to day 7 post-infection. The concentration of supernatant p24 antigen was measured by using ELISA. The results are shown as the means and SDs of triplicate assays. The appropriate RLU was determined by infecting the NP2/CD4 cell line carrying CXCR4 and HIV-1-LTR firefly luciferase reporter at various titers of virus and then RLU was measured by using a luciferase assay system (Promega). The NP2/CD4 cell line was provided by Dr. Y. Maeda, Department of Microbiology, Kumamoto University, Japan.