Dear editor of PlosOne

We are pleased to submit the manuscript entitled, “Is the tryptophan codon of gene vif the Achilles’ heel of HIV-1?”, we present in this study results of the relationship between the hypermutation in the HIV gene vif and levels of CD4+ lymphocytes and levels of viral RNA.

We used 7072 proviral sequences HIV-1 gene vif from public databank to evaluate the impact of Apobec3 to the evolution of HIV in a population level.

First we found that tryptophan codons (TGG) of the gene vif targeted exclusively by RT have lower mutation rates than TGG codons targeted by Apobec3.

Then we used a subset of 857 sequences (sequences with information of CD+4 counts and viral loads) and found that patients harboring hypermutated viruses tend to have lower mRNA levels and higher counts of CD4+ cells.

Overall our findings indicate that at individual level Apobec3-induced hypermutation a subtle effect the HIV replication. However, at the population level Apobec3 activity is minimal to restrain HIV spread because it is rare (only 0.6% of all sequence in the genbank were hypermutated).

Sincerely yours
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Figure 1: Mutation rates for different codon positions.
Figure#2
Figure 3