CORRIGENDUM

Efficient delivery of lentiviral vectors into resting human CD4 T cells

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Figure 1c and Figure 2b of our previously published paper are incorrect as presented. The experiments were independently repeated with multiple donors and the new data are now presented as Figure 1c, Figure 1d and Figure 2b. The original conclusions drawn from these figures and the techniques described in the full manuscript remain the same. The VLP-Vpx

technology provides an efficient and reliable method for introducing lentiviral shRNAs into resting CD4 T cells, especially into primary blood-derived lymphocytes that are in a deep resting state.

The authors apologize for not detecting the error in these two figures prior to publication.

Figure 1. Genetic manipulation of resting CD4 T cells by lentiviral vectors using sequential spinoculation of Vpx-VLP and shRNA-encoding HIV LV particles. (a) The spinoculation procedure: fresh target cells were spinoculated with Vpx-VLPs, followed by shRNA-encoding HIV LV particles. Particles were pseudotyped with the CXCR4-tropic Env of HIV-1, which supports efficient fusion of virions to resting CD4 T cells. A detailed description is available in the Methods section. (b) Spinoculation of Vpx-VLPs degrades SAMHD1 in resting CD4 T cells isolated from peripheral blood, tonsil, or spleen after 24 h. (c) Efficient infection of resting CD4 T cells by three independent HIV LV particles indicated by mCherry transgene expression. Cells expressing mCherry remained viable (Supplementary Figure 2) and negative for CD25/CD69 activation markers, as indicated. (d) Fold increase of HIV LV infection after spinoculation with Vpx-VLPs as observed in CD4 T cells from peripheral blood, tonsil and spleen obtained from three independent donors. (e) A marked decrease in surface CD4 expression in blood CD4 T cells productively infected with shCD4 HIV LV particles. (f) Reduced intracellular NLRP3, mRNA and protein expression in mCherry-positive CD4 T cells after infection with shNLRP3 HIV LV particles. Note that CD4 and NLRP3 expression were not reduced in mCherry-negative CD4 T cell populations. These data are representative of four independent experiments performed using CD4 T cells from peripheral blood, tonsil and spleen from four different donors.

Figure 2. Modulating specific CD4 T cells responses by HIV LV (**a**, **b**) Infection of shCD4 HIV LV impedes HIV-1 fusion to resting CD4 T cells isolated from blood. To test the ability of HIV-1 to fuse with infected cells, we used an HIV virion-based fusion assay that measures BlaM activity delivered to target cells upon the fusion of X4-tropic NL4-3 strain of HIV-1 containing BlaM fused to the Vpr protein (BlaM-Vpr-NL4-3).⁴⁵ Inhibition of fusion was specifically observed in the mCherry-positive (**a**), but not in the mCherry-negative (**b**) cells residing in the same cultures. (**c**) Infection of shNLRP3 HIV LV blocks caspase-1 activation in tonsillar CD4 T cells treated with nigericin. (**d**) Infections with shNLRP3 HIV LV prevent caspase-1-mediated death of mCherry-positive CD4 T cells treated with nigericin. Inhibition of cell death was not inhibited by mCherry-positive Scramble HIV LV.