Viral reprogramming of cells increases risk of cancers in HIV patients, finds study

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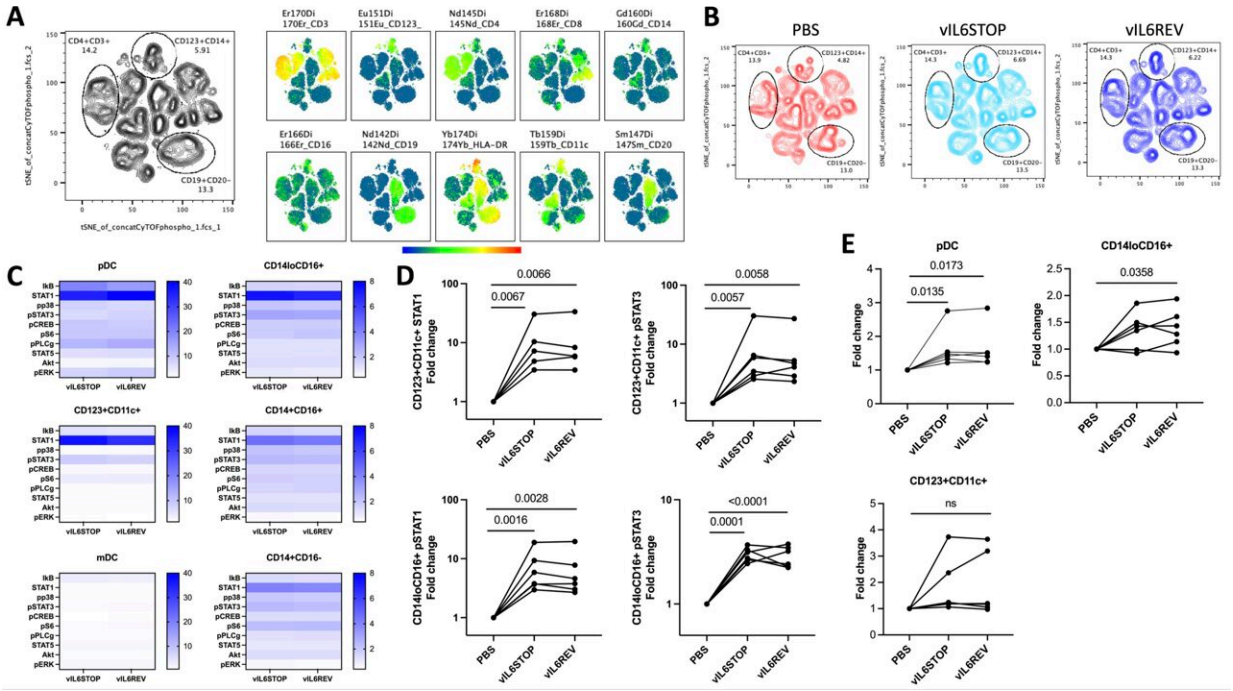

KSHV infection preferentially triggers STAT activation in monocytes and dendritic cells. PBMC from healthy donors (n = 6) were infected with vIL-6REV (KSHV with vIL-6 expression) or vIL-6STOP (KSHV without vIL-6 expression) (MOI = 1), or mock-infected (PBS). At 1 dpi, cells were fixed and subjected to CyTOF analysis. The expression levels of 10 signaling molecules were evaluated. Data combine two experiments (n = 3 each). (A) tSNE for dimension reduction analysis was applied for each infection group after 1000 events from 6 samples in each group were concatenated. Immune cell subsets were clustered based on each lineage's marker expression (Right panel). (B) The phenotypic changes induced in three immune cell populations corresponding to CD4+CD3+, CD19+CD20-, and CD123+CD14+ clusters by infection were identified by

comparison between vIL-6STOP, vIL-6REV, and the mock-infected (PBS) control group. (C) The activation status of 10 signaling molecules among plasmacytoid dendritic cells (pDC) and monocytic cell subsets, including CD123+CD11c+ cells, myeloid (m)DC, CD14loCD16+ non-classical monocytes, CD14+CD16+ intermediate monocytes, and CD14+CD16- classical monocytes are shown in heat maps. Each subset was identified based on the lineage cell surface marker expression with gating strategies shown in S6 Fig. (D) The fold change in the CyTOF signal intensity of STAT1 and pSTAT3 for CD123+CD11c+ cells (TOP panel) and CD14+CD16+ cells (Bottom panel) in infected groups compared to mock-infected (PBS) control group are shown. p values shown are by ratio paired t-test. p