

Results BALB/c mice that received the IL-13Ra2 and IL-4R antagonist vaccines showed significantly reduced IL-13 expression by ILC2 at the lung mucosae compared to the BALB/c that received the unadjuvanted vaccine ($p < 0.001$). Interestingly, the IL-13Ra2 adjuvanted vaccinated group showed significantly elevated ILC1-like cells expressing IFN- γ compared to the IL-4R antagonist vaccine ($p < 0.0001$) or BALB/c mice given the FPV-HIV unadjuvanted vaccination ($p < 0.001$). Furthermore, IL-4 and IL-13 milieu also influenced the dendritic and macrophage cell subsets (i.e. CD11b $^{+}$ CD103 $^{-}$ DC, plasmacytoid DC, alveolar macrophages) recruited to the lung mucosae 24 h post vaccination.

Conclusion Our findings suggest that i) the outcome of a vaccine is determined within the first 24 h of vaccination, ii) ILC1-like cells most likely play a role in B cell immunity and iii) ILC2 are the major source of IL-13 that dampens CD8 T cell avidity by altering DC/macrophage recruitment to the vaccination site.

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017.4 DEVELOPMENT AND VALIDATION OF A HUMAN T-CELL LYMPHOTROPIC VIRUS TYPE-1 PROVIRAL LOAD ASSAY

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Introduction Human T-cell lymphotropic virus (HTLV-I) infects approximately 20 million people world-wide. Transmission requires cell to cell contact and infection can be acquired through breast milk, exposure to HTLV-1 contaminated blood products or sexual contact with an infected person. The HTLV-I proviral load (PVL) is a strong predictor of the risk of transmission and may also serve as an indicator of those most at risk of acquiring significant complications following infection.

The Australo-Melanesian variant (HTLV-I subtype c) is endemic amongst indigenous communities in Central Australia and demonstrates a highly divergent sequence from other known HTLV-I subtypes. Currently, there are no commercially available HTLV PVL assays and published methods fail to reliably detect HTLV-1c.

Methods We developed and validated a quantitative, real time PCR (qPCR) assay, specific for the current circulating strains of HTLV-I. Primers and probes were designed by sequencing the *gag* gene from HTLV-1c samples. A highly conserved region of the *gag* gene which did not cross-react with HTLV-II was chosen.

A dilution series of SP cells which contain 1 copy of the HTLV-I genome, was used for quantification. The standards and specimens were run in parallel throughout the entire extraction and qPCR process, allowing us to eliminate variations due to extraction efficiency, PCR amplification and detection. The albumin gene, was used to determine the number of cells/sample and the PVL expressed as HTLV-I copies/cell.

Results We have now fully validated this assay using both clinical specimens and cultured cell lines. Clinical specimens consisting

of buffy coats, whole blood specimens and dry blood spots have been tested on the HTLV-I PVL assay and demonstrated good concordance with results obtained using gold standard serology assays.

Conclusion We have developed and validated an assay that can reliably quantify the HTLV PVL which may serve as a predictor of the risk of transmission and disease progression.

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017.5 INCREASED HERPES SIMPLEX VIRUS-2 SHEDDING IN HIV-1 INFECTED PERSONS IS DUE TO POOR IMMUNOLOGIC CONTROL IN BOTH GANGLIA AND GENITAL MUCOSA

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Introduction A signature feature of HIV infection is poor control of herpesvirus infections, which reactivate from latency and lead to opportunistic infections. While the general mechanism underlying this observation is deficient CD4 $^{+}$ T-cell function, it is unknown whether increased severity of herpes virus infections is due primarily to poor immune control in latent or lytic sites of infection, or whether CD4 $^{+}$ immunodeficiency leads to more critical downstream deficits in humoral or cell-mediated immunologic responses.

Methods Here we compare genital shedding patterns of herpes simplex virus-2 (HSV-2) in 98 HIV infected and 98 HIV uninfected men matched on length of infection, HSV-1 serostatus and nationality.

Results We demonstrate that high copy HSV-2 shedding is more frequent in HIV positive men, particularly in participants with CD4 $^{+}$ T-cell count $<200/\mu\text{L}$. Genital shedding is more frequent due to higher rate of shedding episodes, as well as a higher proportion of prolonged shedding episodes. Peak episode viral load was not found to differ between HIV infected and uninfected participants regardless of CD4 $^{+}$ T-cell count. We simulated a mathematical model which recapitulated these findings and identified that rate of HSV-2 release from neural tissue increases, duration of mucosal cytolytic immune protection decreases, and cell-free viral lifespan increases in HIV infected participants.

Conclusion These results suggest that increased HSV-2 shedding is due to impaired immune function in both latent and lytic tissue compartments, with deficits in both humoral and cell-mediated HSV-2 clearance.

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