Response to Comment on "HIV-Specific IL-21 Producing CD4+ T Cells are Induced in Acute and Chronic Progressive HIV Infection and Are Associated with Relative Viral Control"

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Comment on “HIV-Specific IL-21 Producing CD4+ T Cells Are Induced in Acute and Chronic Progressive HIV Infection and Are Associated with Relative Viral Control”

The article by Yue et al. (1) provides novel insights into the role of IL-21 in HIV infection. Their results are in accord with ours (2) concerning the presence of HIV-specific CD4+ T cells in HIV-infected viremic individuals. However, there is an important point of divergence.

Yue et al. (1) conclude: “HIV-infected individuals had greater circulating IL-21 producing CD4+ T cells in blood as compared with uninfected volunteers.” On the contrary, we have shown decreased frequencies of the cytokine-producing CD4+ T cells in HIV-infected persons compared with those in control subjects. We determined these frequencies in PBMC by flow cytometry after stimulating them with ionomycin for 24 h. In contrast, the stimulus used by Yue et al. (1) is a bacterial superantigen staphylococcal enterotoxin B (SEB) that stimulates only CD4+ T cells bearing TCRs with certain Vβ domains (3). Our unpublished data show that SEB activates only a subset of the ionomycin-stimulated CD4+ T cells. Because humans differ from each other with respect to their repertoires of CD4+ T cells bearing TCRs with different families of Vβ domains, SEB is not an appropriate method to compare the cytokine-producing CD4+ T cell frequencies between control and virus-infected individuals. Should the authors have used a pan-T cell activator, they might have obtained different results. Furthermore, the data shown in Fig. 4B of Yue et al. (1) do not concur with their above-mentioned conclusion. Readers of the Yue et al. article (1) should be made aware of these caveats.

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We agree with Drs. Iannello, Samarani, and Ahmad that staphylococcal enterotoxin B stimulation will not activate all T cell subsets, but only the Vβs associated with staphylococcal enterotoxin B activation, thus possibly explaining the discordance in results between our group and that of Iannello et al. (1). It should also be noted, however, that ionomycin stimulation alone also may not be a physiological stimulus that provides two signals to T cells, as a phorbol ester such as PMA is often required in conjunction with Ca ionophores to accomplish this (2). The data in Fig. 4B (3) do not quantify the numbers of IL-21 producing cells between the two groups but show that, within the IL-21 producing populations observed, they are enriched more for CCR7+ CD45+ cells in HIV-infected individuals.

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