of Vδ2 T cells tends to be lower among HIV-positive groups. However, they studied terminally differentiated T cells [4] and did not focus on the effector memory subset, as defined previously [5].

Hartjen et al also raised a concern about our conclusion that CD56+ Vδ2 T cells are depleted in HIV disease except among natural viral suppressors [6] (designated “EC” for “elite controllers” in their letter). Specifically, they reported elevated CD56+ Vδ2 T cells in patients with high viremia with a mean level of 20% CD56+ Vδ2 T cells. We consider this value to be low compared with that for healthy, matched controls [6]. The control donors in Figure 1E in the report by Hartjen et al also have very low levels of CD56 expression on Vδ2 T cells, especially compared with our previous studies. Among HIV-negative control donors who we studied (matched for age and sex), only 3 of 28 whites and 10 of 29 African Americans had <20% CD56+ Vδ2 T cells in blood. This contrasts sharply with the mean value shown by Hartjen et al, which is approximately 7% CD56+ Vδ2 T cells among control donors.

Our conclusions were based on extensive databases for healthy controls and HIV-positive donors from studies conducted in North America, Europe, Western Africa, and China [2]. On the basis of multiple phenotyping and T-cell receptor sequencing studies [2, 7], we are confident that HIV infection drives specific depletion of the Vδ2 subset and lowers circulating levels of CD56+ cytotoxic effectors. We are unable to account for differences between our studies and the report from Hartjen et al, especially concerning the low expression of CD56 and low abundance of Vδ2 T cells among their control donors.

Notes

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