Abstract

A high level of serum IgE is generally associated with human resistance to schistosomes, though the protective mechanisms of IgE remain undefined. We recently reported that whereas some individuals who are occupationally hyperexposed to Schistosoma mansoni display resistance to reinfection, others remain highly susceptible, in some cases due to HIV-1 co-infection. As IgE functions, in part, through FcεRI on mast cells, we characterized circulating CD117(+) FcεRI(+) mast cell precursors in this population. Surprisingly, a higher percentage of CD117(+) cells correlated with a susceptible phenotype in HIV-1 seronegative participants with schistosomiasis. There was no association between percentages of peripheral CD117(+) cells and susceptibility to reinfection in persons with HIV-1. Serum levels of polyclonal IgE were inversely correlated with percentages of CD117(+) cells regardless of HIV-1 status. Thus, immature mast cells may affect IgE availability, or IgE may affect immature mast cells, altering the balance of host susceptibility and resistance to schistosomes.