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REGULATORY T CELLS IN INFANT MACAQUES SUPPRESS ANTI-SIV RESPONSES AMONG CD4+ T CELLS

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OBJECTIVES: The impact of regulatory T cells (T-regs) on the course of HIV and SIV disease remains unclear. We found that infant macaque blood and tissue samples contain more regulatory T cells than adult samples and that these infant T-regs exhibit greater *in vitro* suppressive activity. We tested the impact of regulatory T cells on antiviral T cell responses by using infant and adult macaques as models of infected patients with high and low regulatory T cell numbers, respectively.

METHODS: We followed immune responses and the course of SIV disease in infant macaques, containing an average of 10% T-regs among CD4+ cells, and adult macaques, containing on average only 4% T-regs. SIV-specific T cell responses were tested in the presence and absence of CD25+ T-regs. By comparing anti-SIV immune responses among infants, harboring many T-regs, and adults, harboring few T-regs, we sought to understand the importance of regulatory T cell numbers for anti-SIV responses.

RESULTS: Infant macaques mounted only transient CD8+ T cell responses to SIV during the first two weeks of infection, while adult macaques maintained CD8+ T cell responses throughout infection. Depletion assays showed, however, that infant SIV-specific CD8+ T cells were not directly inhibited by T-regs. Infant SIV-specific CD4+ T cells expressing IL-2 and IFN- γ , however, were directly inhibited by T-regs throughout infection and were detectable only after depletion of CD25+ cells. SIV-specific CD4+ T cells were consistently detected in adults, whether in the presence or absence of T-regs.

CONCLUSIONS: The presence of larger numbers of regulatory T cells among infant macaques is

associated with active suppression of CD4+ T cells throughout infection and early failure of SIV-specific CD8+ T cell responses. Our findings implicate T-regs as potentially important direct antagonists of multifunctional CD4+ T cell responses and indirect antagonists of antiviral CD8+ T cell responses.

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Immune Activation in HIV Pathogenesis

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