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Rediscovered CD8 Treg

Sudhir Gupta University of California, USA

R ecently CD8+ T cells have been demonstrated to regulate proliferation of CD8+ T cells. We have investigated a role of CD8+ T cells in regulating various functions of autologous CD4+ T cells. Activated CD8+ T cells, in a concentration-dependent manner, inhibited cell division (CFSE dilution) and DNA synthesis (3H thymidine incorporation) of autologous CD4+ T cells. CD8+ T cells also inhibit differentiation of Naïve CD4+ to effector memory CD4+ (TEM) and CD45RA+ terminally differentiated effector memory/exhausted CD4+ T cells (TEMRA). Activated CD8+ T cells mediated their regulatory effect, at least in part, by soluble mediator (s) and not due to direct cytotoxicity/apoptosis of CD4+ T cells. Activated CD8+ T cells is significantly blocked by neutralizing antibodies against IL-10 and MIP1β, but not by antibodies against TGFβ or PD-1. CD8+ Treg cells inhibit secretion of IFN-γ and IL-17A by autologous CD4+ T cells. CD8 Tcg were further characterized phenotypically for FoxP3+ expression and appears to be of CD8+CCR7+CD25hiICOS+CTLA-4+FoxP3+ phenotype. Furthermore, proportion of CD8 Treg (CD8+CD183+CCR7+CD45RA-FoxP3+) in aged humans is comparable to young subjects; however, they are impaired in their regulatory functions. In primary immunodeficiency, both quantitative and qualitative defects in CD8 Treg may explain increased susceptibility to autoimmune diseases.

sgupta@uci.edu

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