Supporting Information

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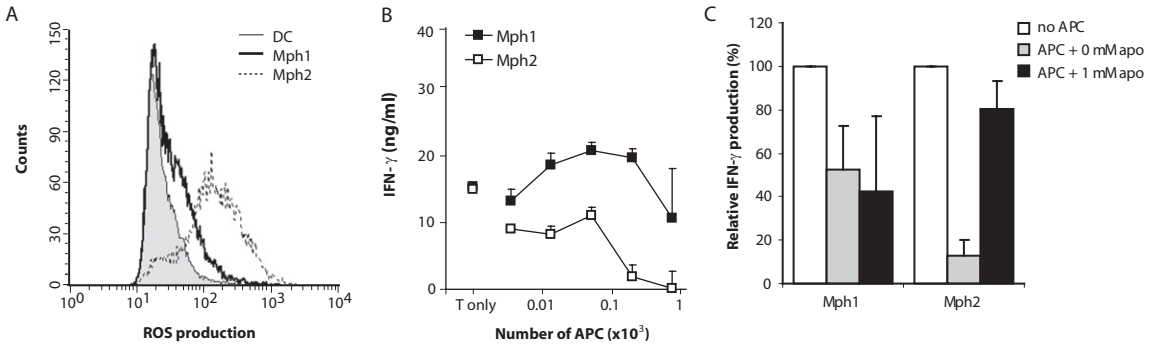

Fig. S1. Antiinflammatory macrophages (Mph2) but not proinflammatory macrophages (Mph1) suppress T-cell activation in a reactive oxygen species (ROS)-dependent fashion. (*A*) Mph1 produce lower levels of ROS than antiinflammatory Mph2 as measured by dihydrorhodamine123 fluorescence by flow cytometry after stimulation with phorbol 12-myristate 13-acetate. DC, dendritic cells. (*B*) Suppression of anti-CD3/28 Ab induced T-cell IFN-γ production by proinflammatory Mph1 is less efficient than suppression by antiinflammatory Mph2. APC, antigen-presenting cells. (*C*) Apocynin (apo) prevents inhibition of T-cell activation by antiinflammatory Mph2 but not by proinflammatory Mph1.

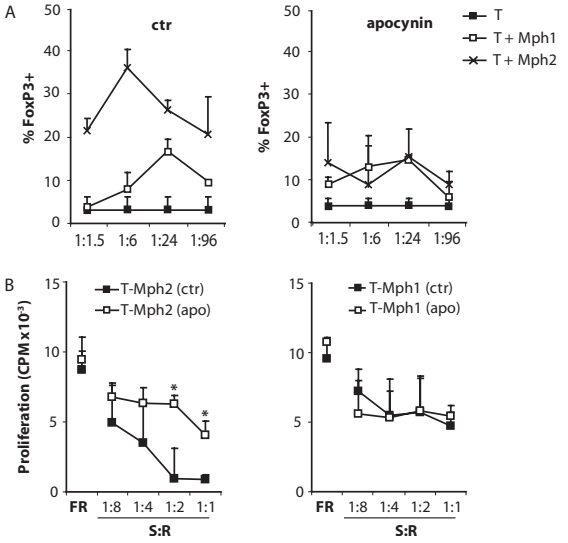

Fig. S2. Mph2 but not Mph1 induce regulatory T cells in a ROS-dependent fashion. (*A*) Priming of $CD4^+CD25^-$ T cells with antiinflammatory Mph2 but not with proinflammatory Mph1 induces FoxP3 expression by these T cells. (*B*) T cells primed with Mph2 but not those primed with Mph1 suppress proliferation of responder T cells in a ROS-dependent fashion. Apocynin (apo) was present during priming to inhibit the Nox2 complex. F, feeder cell; R, responding cell; S, suppressor cell.

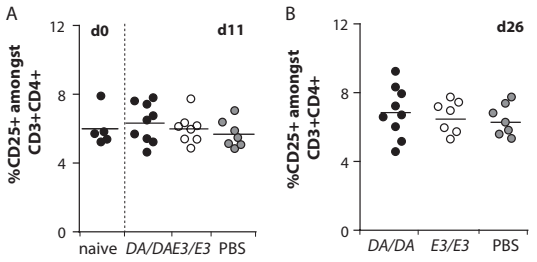

Fig. S3. The number of activated T cells during delayed-type hypersensitivity is similar among groups. The percentage of $CD25^+$ cells among $CD3^+CD4^+$ cells 11 d (*A*) or 26 d (*B*) after rats were primed with Dark Agouti neutrophil cytosolic factor 1 (DA.*Ncf1*$^{DA/DA}$) Mph, DA.*Ncf1*$^{E3/E3}$ Mph, or PBS is comparable among groups.