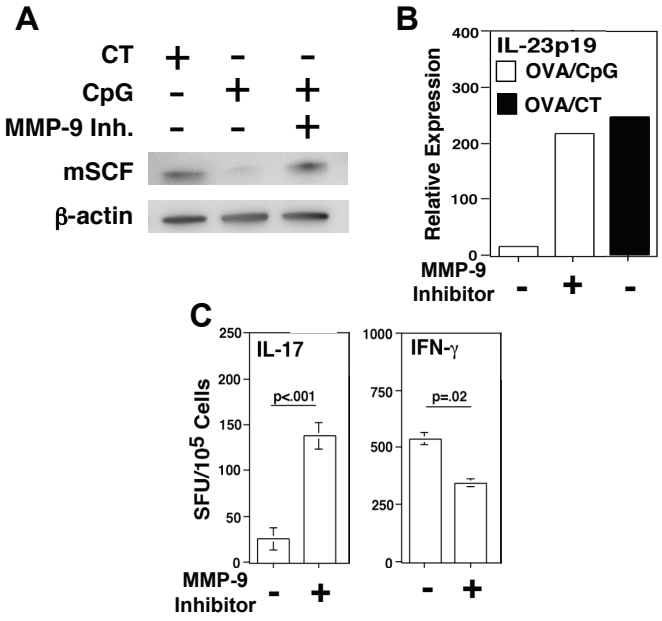
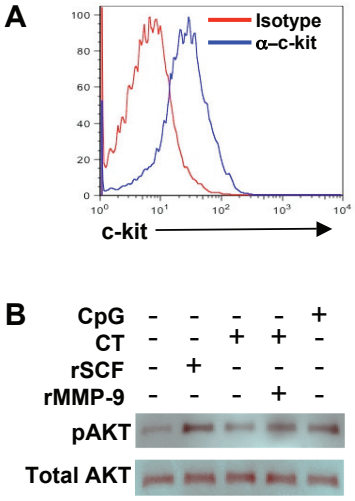


Supplemental Figure 1. ***In vivo*** **chemical inhibition of MMP-9 mimics the effects of MMP-9 deficiency.** Mice were treated with OVA/CT or OVA/CpG with or without specific MMP-9 inhibitor (MMP-9 Inhibitor II; Calbiochem) administered 1 hour prior to each treatment by i.p. injection at a dose of 20 mg/kg. (**A**) Immunoblotting for mSCF expression in $CD11c^+$ lung cells. (**B**) Sorted lung $CD11c^+$ DCs were analyzed for expression of IL-23p19 by qRT-PCR. (**C**) Lung $CD4^+$ T cell ELISPOT assay for IL-17 and IFN-γ.



Supplemental Figure 2. **Biological activity of sSCF.** (**A**) Bone marrow-derived mast cells were generated by treatment with IL-3 (5 ng/ml; Peprotech) for 4 weeks at which time they expressed a high level of c-kit. However, these cells do not express SCF but are responsive to SCF. (**B**) The c-kit^{+} mast cells were used to assay responsiveness to sSCF based upon AKT phosphorylation induced downstream of c-kit. BMDC culture supernatants containing sSCF generated in experiments described in Figs. 3A and 3B were incubated with mast cells for 5 min. AKT phosphorylation was barely detectable at later times of incubation (not shown) in keeping with transient AKT phosphorylation induced by sSCF. For BMDCs cultured with CT, supernatant of cells treated with 800 ng/ml of rMMP-9 was used. Total cell extracts were subjected to SDS-PAGE, blotted onto PVDF membrane and probed for AKT phosphorylation using anti-pAKT antibody (Cell Signaling). The blot was then stripped and re-probed for total AKT (Cell Signaling). Recombinant SCF (rSCF; 500 pg/ml; Peprotech) added to untreated BMDC culture supernatant (left-hand lane) served as a positive control. This dose was in the range of sSCF detected by ELISA in the culture supernatant of CpG- or CT-stimulated BMDCs in the presence of MMP-9 (800 ng/ml) (Fig. 3).