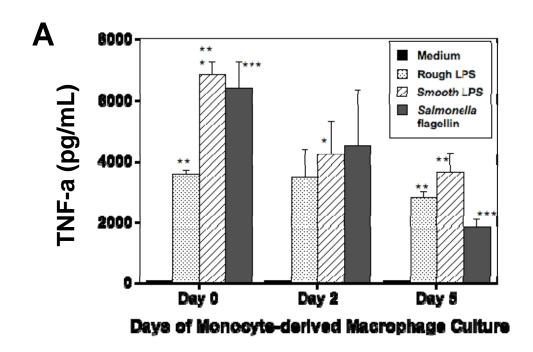
Supplementary Figure Legends

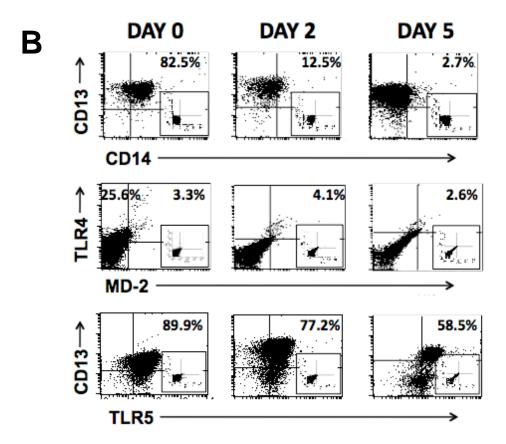
Supplementary Figure 1. Monocyte-derived macrophages display reduced pro-inflammatory function, but not inflammation anergy, over time. (A) Monocyte-derived macrophages exposed to rough LPS (*S. arbortus*), smooth LPS (*E. coli*) and flagellin (*S.* Typhimurium) released significantly lower levels of TNF- α over time, reaching minimal levels on day 5. Values represent mean \pm S.E. of triplicate wells from a representative experiment (n=4) (p<0.05*; p<0.01**; p<0.001*** for inducible TNF- α within the stimuli at different timepoints). (B) Monocyte-derived macrophage expression of CD14, TLR4 and TLR5 decreased over the same time-course, but surface levels were still detectable on day 5. Data is from a representative experiment (n=3).

Supplementary Figure 2. TLR4 and MD-2 gene transfection of intestinal macrophages. Low numbers of intestinal macrophages expressed TLR4 and MD-2 following transfection with TLR4 and MD-2 (left panel) but not after transfection with vector alone (middle panel). Quadrant limits were set at the 1% cursor, using appropriate isotype controls (right panel). Data are from a representative experiment (n=3).

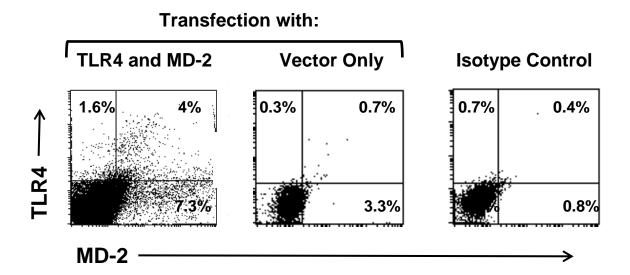
Supplementary Figure 3. Stromal factors down-regulate NF- κ B translocation in blood monocytes. (A) Blood monocytes from the same experiment in Figure 6B were treated with S-CM (500 μ g/mL; bottom right panel) and analyzed for NF- κ B nuclear translocation by immunofluorescence and confocal microscopy. NF- κ B in the S-CM-treated monocytes localized predominantly to the cytoplasm. (B) NF- κ B in monocytes treated with (a) medium alone or (b) M-CSF (10 ng/mL) plus either rhTGF- β (50 pg/mL) or S-CM plus irrelevant mouse antibody also localized predominantly in the cytoplasm. NF- κ B in monocytes stimulated with M-CSF alone localized predominantly in the nucleus.

Supplementary Figure 1





Supplementary Figure 2



Supplementary Figure 3

