

Figure S1. A, Placenta for positive control of CPM immune-staining using VIP peroxidase substrate. Arrows indicate trophoblasts that are labeled (purple staining). Panel **B**, In normal lung, few CD68 positive alveolar macrophages are seen (brown cells), which are negative for CPM. Panel **C**, Fibrinous pleural inflammatory exudate contains many CD68-reactive macrophages (brown cells), which are negative for CPM. Panel **D**, Nearly normal (mildly reactive) lymph node exhibits moderate number of CD163 positive sinus histiocytes, but only very few express CPM (arrows). B-D images are immune-peroxidase stainings using DAB (brown) chromogenic substrate and hematoxylin nuclear counterstaining.



Figure S2. High magnification of a middle-sized artery obtained from a case of Wegener granulomatosis vasculitis, demonstrated in Figure 2. **A-B**, images from identical microscopic field show that CPM antibody labels more prominently the lesional (epithelioid) macrophages (B, purple cells), confirming the granulomatous nature of the necrotizing mural vascular inflammation, as opposed to the reference marker CD163 (A) decorating scattered macrophages more randomly, and without any characteristic pattern. Both images are from immune-peroxidase reactions with methyl-green (A) and hematoxylin (B) nuclear-counterstaining. *Original magnifications: 40x.*



Figure S3. Flow cytometry (FACS) analysis of untreated macrophages (-) and macrophages exposed to mycobacteria (-) for 24 hours. Isotype control (...). As expected from previous publications⁴⁰, TB-infection results in increased expression of CD14, CD11c but MHC-II does not show up-regulation. One representative experiment of three performed is shown.



Figure S4. A, CPM-mRNA levels of 12-hour macrophages that were differentiated into dendritic cells (DCs) for 5 days in the presence of IL-4+GM-CSF plus various doses of TGF- β . Note that TGF- β did not influence the CPM-transcript expression levels. **B**, The corresponding flow cytometry (FACS) data show basically no changes in HLA-DR surface expression pattern of differentiating macrophages into DCs up to 5 days in response to any doses of TGF- β applied.

Supplementary Figure S5. (for online publication, only)



FL1-H:CD11c, FL2-H:CD14

Figure S5. Flow cytometry (FACS) results for CD11c and CD14 of macrophages that were differentiated in the presence of 10% FBS (control), 1% FBS, charcoal-stripped serum (CCSS) and CCSS supplemented with 20 ug/ml low density lipoprotein (LDL) for 24 hours. As shown, except for 1%FBS which moderately decreased CD11c-CD14 expressions, neither CCSS nor CCSS+LDL altered substantially the expression pattern of these differentiation markers as compared to control macrophages (10%FBS). The same pattern was observed in macrophages differentiated for 48 hours (not shown).

Supplementary Figure S6. (for online publication, only)



Figure S6. When human monocytes (upper panel, typically with no detectable CPM) are treated with the cytokine cocktail IL4+GM-CSF to allow cells to differentiate into early dendritic cell (DC) phenotype, they exhibit intracellular lipid accumulation, indicated by the vacuolated cytoplasm on HE-stained section (middle panel, left) while expressing moderate amounts of nuclear protein peroxisome proliferator-activated receptor-gamma (PPAR γ , dark nuclei at middle), but up-regulating the CPM protein (purple), mainly along the cell membrane (i.e., control DCs, middle panel). However, when the cytokine cocktail was supplemented with rosiglitazone to up-regulate PPAR γ expression to facilitate lipid efflux (lower panel, middle), the lipid content of cells becomes decreased, indicated by the diminished cytoplasmic vacuoles (lower left), parallel with a reduced CPM-protein expression level (lower right). Immune-peroxidase labeling with methyl-green nuclear counterstaining. *Magnifications of all images: 40x.*