Supplemental Fig. 1. PPARδ displays Pro-Inflammatory Activity in Human Monocytes

A, MIP-1 and Eotaxin RNA levels in THP-1 cells were measured by real-time PCR. THP-1 cells were treated with vehicle (Veh), TNFa (50 ng/ml), 10⁻⁵ M PPARδ agonist carbaprostacyclin (Carb) or Carb+TNFα for 24 h. Total RNA was harvested, and cDNA was prepared and used as a template for gene expression analysis. All values were normalized to a 36B4 control. Graphical data is represented as fold induction over vehicle (set at 1). Data points represent the average of triplicate amplification reactions for each condition in a representative experiment. Very similar results were observed in THP-1s that were differentiated into macrophages. B, IL-6 receptor RNA expression in THP-1 cells was analyzed by real-time PCR as described in A.

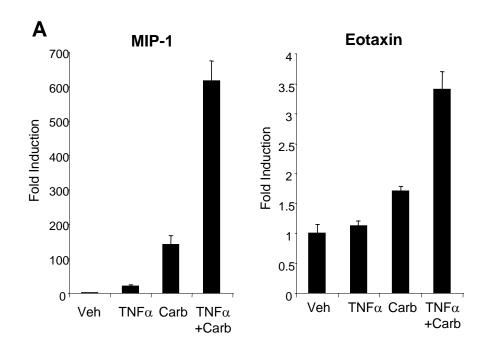
Supplemental Fig. 2. Cell Lacking PPARδ Are Resistant to Anti-Inflammatory Action of PPARδ Antagonist GSK660

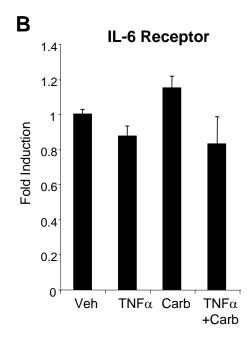
IL-1 β and IL-6 RNA levels in HeLa cells (which lack detectable PPAR δ activity) were measured by real-time PCR. HeLa cells were treated with vehicle (Veh), TNF α (50 ng/ml), 10^{-6} M PPAR δ antagonist GSK660 or TNF α +GSK660 for 24 h. Total RNA was harvested, and cDNA was prepared and used as a template for gene expression analysis. All values were normalized to a 36B4 control. Graphical data is represented as fold induction over vehicle (set at 1). Data points represent the average of triplicate amplification reactions for each condition in a representative experiment.

Supplemental Fig. 3. PPARδ Lacks Pro-Inflammatory Activity in Mouse Monocytes and Macrophages

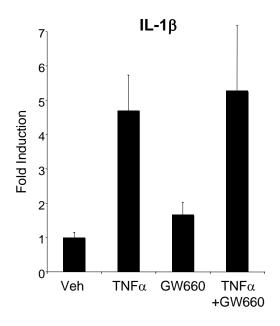
RAW cells contain functional PPARδ and PPARγ (26). MCP-1 RNA levels in RAW cells were measured by real-time PCR. Cells were treated with vehicle (Veh), TNFa (50 ng/ml), 10⁻⁵ M PPARδ agonist carbaprostacyclin (Carb), TNFα+Carb, LPS (20 ng/ml) or LPS+Carb for 24 h. Total RNA was harvested, and cDNA was prepared and used as a template for gene expression analysis. All values were normalized to a cyclophilin control. Graphical data is represented as fold induction over vehicle (set at 1). Data points represent the average of triplicate amplification reactions for each condition in a representative experiment. Very similar results were observed in RAW cells that were differentiated into macrophages. Note that TNFα does not elicit an inflammatory response in mouse monocytes. However, the proinflammatory activity of LPS is not potentiated by Carb administration as it is in human monocytes. Furthermore, Carb alone is marginally anti-inflammatory in these cells.

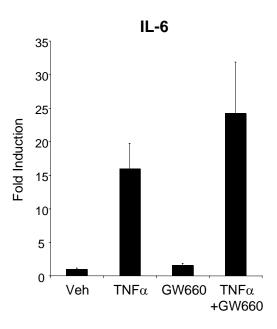
Supplemental Figure 1





Supplemental Figure 2





Supplemental Figure 3

