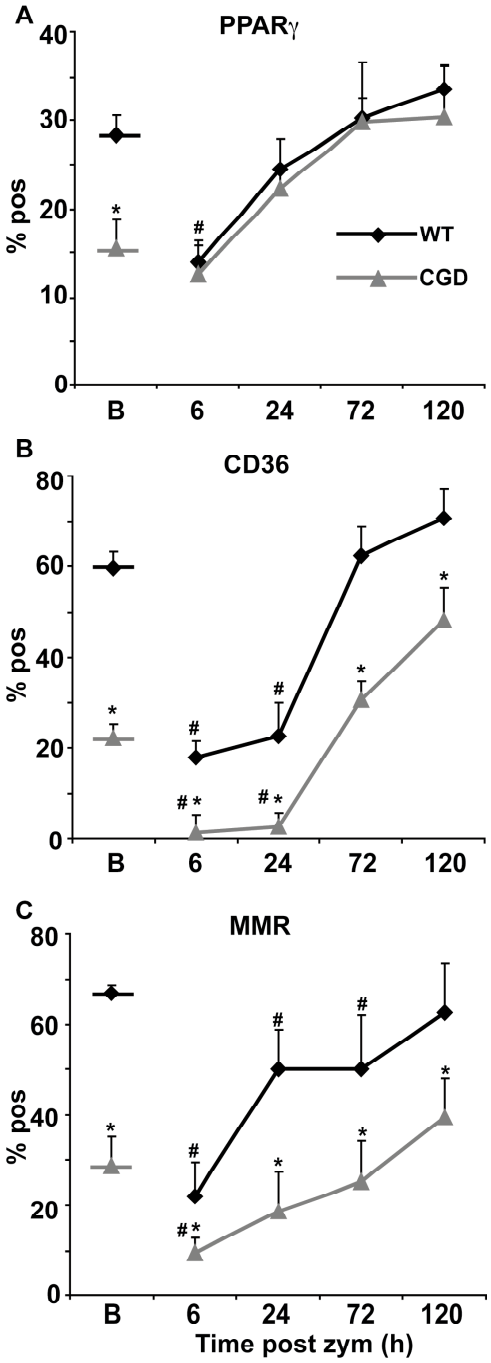

Figure S1. Percent of peritoneal macrophages staining positively for PPARγ and downstream M2 markers during peritonitis

Mice were treated as described in Fig. 1, lavaged and F4/80 positive macrophages analyzed for the percentage of cells also positive for intracellular PPARγ (A) and surface CD36 (B) and MMR (C) at indicated time points following zymosan injection. [B] indicates baseline without zymosan. Data represent mean ± S.E.; N=8 mice per time point. $^{*}p \leq 0.02$ compared to WT mice at the respective time points; $^{\#}p \leq 0.03$ compared to baseline values for each genotype, respectively.

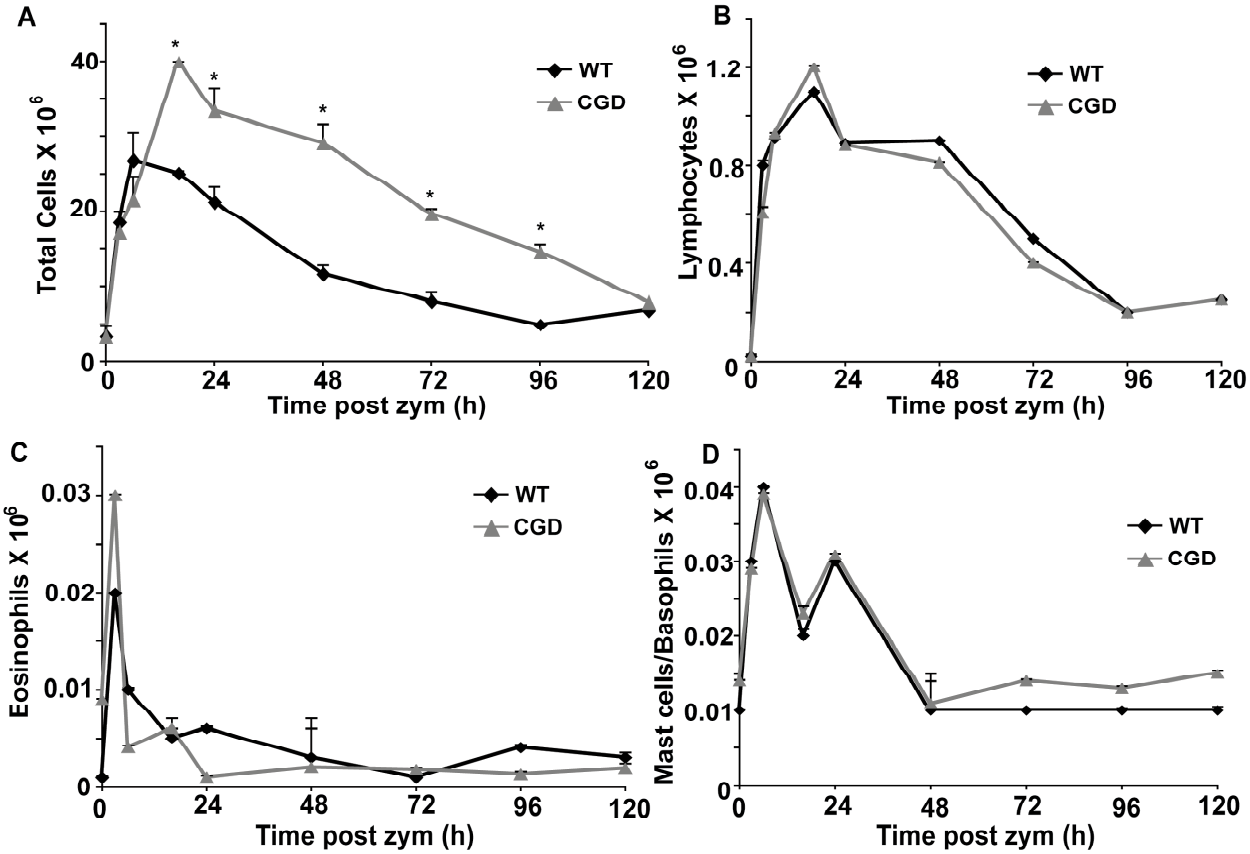

Figure S2. Total cell counts and minor leukocyte populations during zymosan induced peritonitis

Mice were treated as described in Fig. 1, lavaged, cells counted with a Coulter Counter and cytospins analyzed for cell type by visual inspection. Data represent mean ± S.E.; N=8 mice per time point. $^{*}p \leq 0.01$ compared to WT mice at the respective time points. Macrophage numbers are shown in Fig. 1A and neutrophil numbers are shown in Fig. 3A.

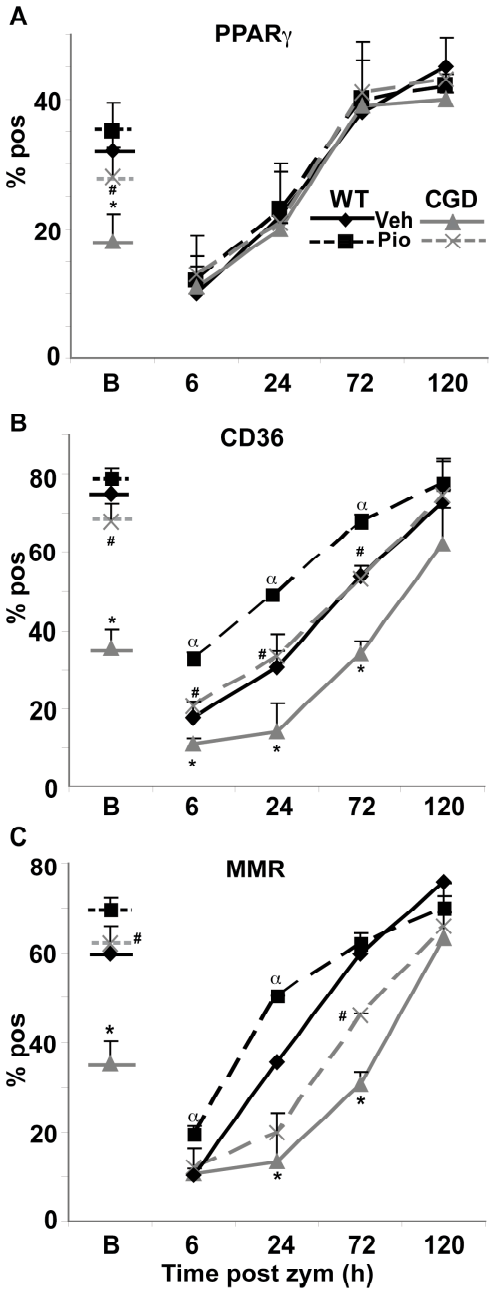

Figure S3. Pioglitazone enhances PPARγ expression and downstream markers in peritoneal macrophages

Mice were treated as described in Fig. 4, lavaged and F4/80 positive macrophages analyzed for the percentage of cells also positive for intracellular PPARγ (A) and surface CD36 (B) and MMR (C) at indicated time points following zymosan injection. [B] indicates baseline without zymosan. Data represent mean ± S.E.; N=8 mice per time point; # $p \leq 0.01$ compared to vehicle treated CGD mice at the respective time points; $^{\alpha,}$ *$p \leq 0.01$ compared to WT mice treated with vehicle, at the respective time points. Symbols for significant changes in values between baseline [B] and early time points following zymosan for WT and CGD mice, respectively, were as shown in Fig. S1, but omitted here for simplicity.

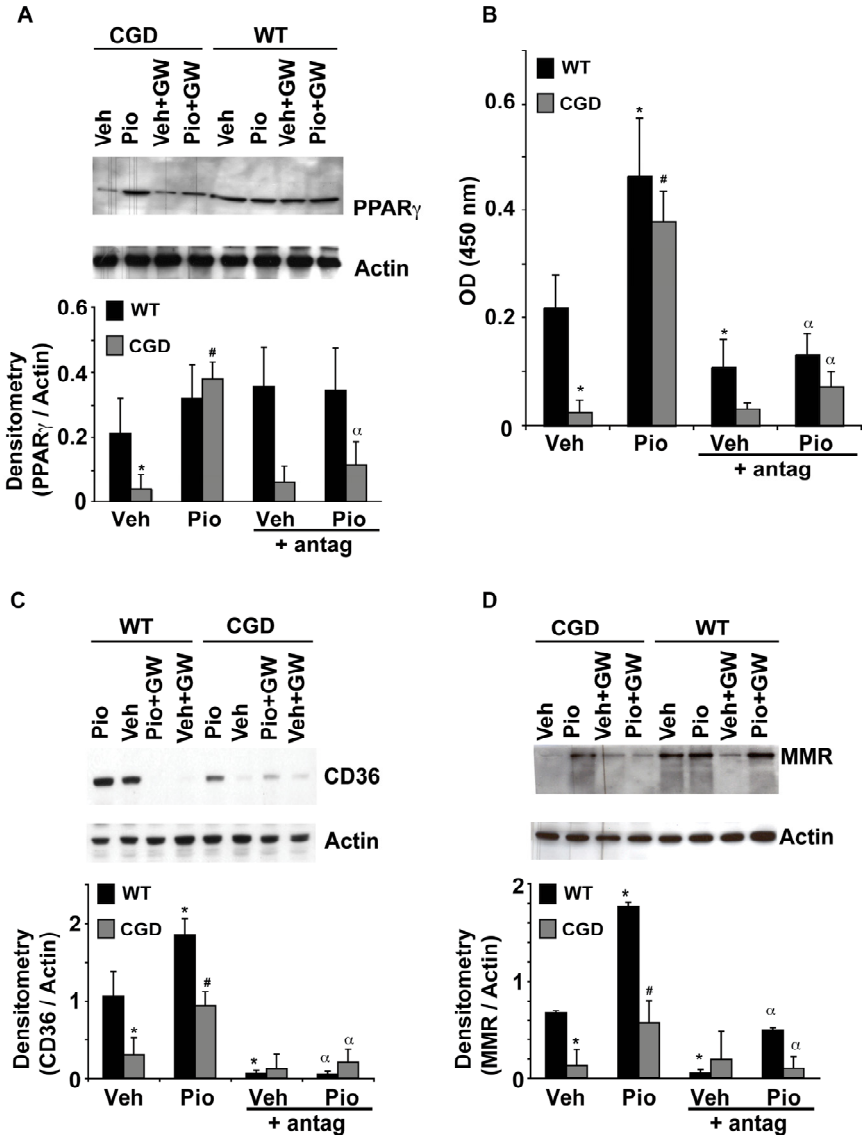

Figure S4. *Ex vivo* pioglitazone treatment enhances PPARγ activity in CGD and WT peritoneal macrophages

Mice were injected with zymosan and 48 h post injection, cells were lavaged and 10×10^6 macrophages plated. Cells were treated for 24 h with either vehicle or 1 μM pioglitazone in the presence or absence of 10 μM PPARγ antagonist (GW9662). (A) Cell lysates were analyzed for PPARγ protein by SDS-PAGE and densitometry analysis performed using ImageJ as described in the Material and Methods. (B) Nuclear extracts were prepared and PPARγ activity analyzed as described in Materials and Methods. (C, D) Cell lysates were analyzed for CD36 and MMR by SDS-PAGE PAGE and densitometry analysis performed using ImageJ as described in the Material and Methods. Data represent mean ± S.E.; N=3, *$p \leq 0.03$ compared to vehicle treated WT macrophages, $^{\#}$ $p \leq 0.004$ compared to vehicle treated CGD macrophages, $^{\alpha}$ $p \leq 0.004$ compared to the respective macrophage for each genotype treated with pioglitazone, respectively.

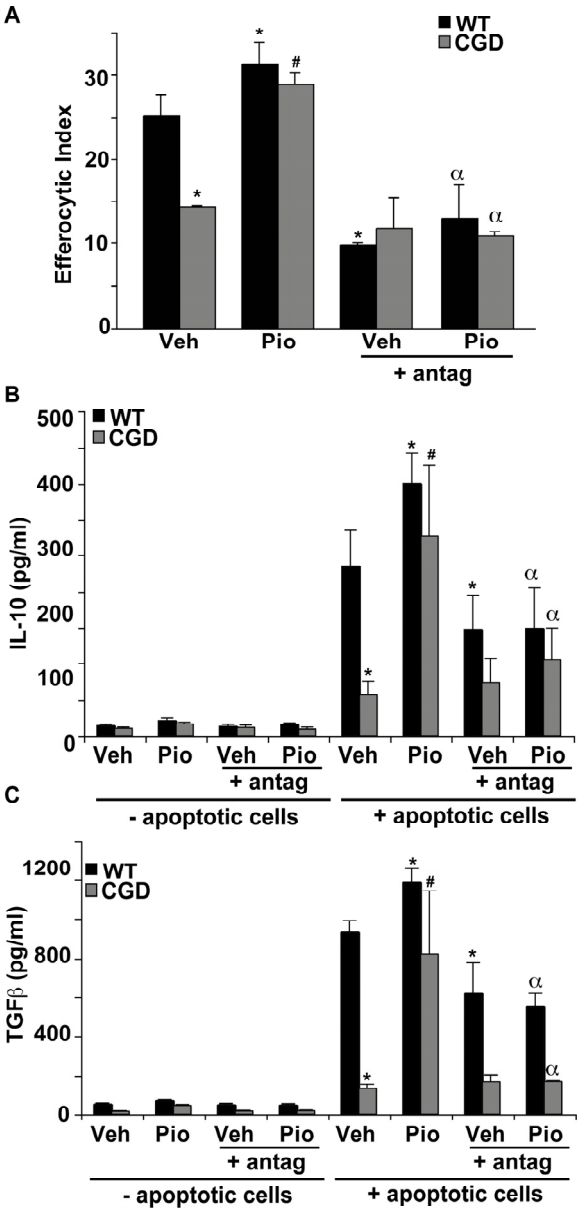

Figure S5. *Ex vivo* pioglitazone treatment enhances efferocytosis and production of anti-inflammatory cytokines by CGD and WT peritoneal macrophages

Mice were injected with zymosan and 48 h post injection, cells lavaged, macrophages plated (either 0.3×10^6 for A or 1×10^6 for B and C) in 24 well plates and 2h later cells were treated with either vehicle or pioglitazone as in Fig. S4. After 24 h incubation, macrophages were co-cultured with or without apoptotic Jurkat T cells (2:1, target to macrophage) for 1h. (A) Wells were washed, fixed, stained and efferocytosis determined. (B,C) Wells were washed, media replaced and 18h later supernatants were harvested and cytokines determined by ELISA. Data represent mean ± S.E.; N=3, *$p \leq 0.05$ compared to vehicle treated WT macrophages, $^{\#}$ $p \leq 0.01$ compared to vehicle treated CGD macrophages, $^{\alpha}$ $p \leq 0.03$ compared to the macrophages of each genotype treated with pioglitazone, respectively.