Table 1: Sequences of primers used

AMAC1	forward	*5'-AGC TCT GCT GCC TCG TCT AT-3'*
	reverse	*5'-CCC ACT TCT TAT TGG GGT CA-3'*
MR	forward	*5'-CGA GGA AGA GGT TCG GTT CAC C-3'*
	reverse	*5'-GCA ATC CCG GTT CTC ATG GC-3'*
IL-1Ra	forward	*5'-TTG AGC CTC ATG CTC TGT TC-3'*
	reverse	*5'-CAG TGA TGT TAA CTG CCT CCA G-3'*
IL-10	forward	*5'-GAT CCA GTT TTA CCT GGA GGA G-3'*
	reverse	*5'-CCT GAG GGT CTT CAG GTT CTC -3'*
TGFβ	forward	*5'-CTC CGA GAA GCG GTA CCT GAA C-3'*
	reverse	*5'-CAC TTG CAG TGT GTT ATC CCT-3'*
Human 11β- HSD1	forward	*5'-CAT GTG GTG GTG ACA GCG AGG TC-3'*
	reverse	*5'-GGT TGA GAA TGA GCA TGT CTA GTC-3'*
Mouse 11β- HSD1	forward	*5'-AAC CAC ATC ACT CAG ACC-3'*
	reverse	*5'-GAG TTC TGT TCT AAT GGT G-3'*
Mouse CD36	forward	*5'-GCA CCA CTG TGT ACA GAC AG-3'*
	reverse	*5'-GTG CAG CTG CTA CAG CCA G-3'*
IL-1β	forward	*5'-AGC TCG CCA GTG AAA TGA TGG-3'*
	reverse	*5'-CAG GTC CTG GAA GGA GCA CTT C-3'*
GILZ	forward	*5'-GCA CAA TTT CTC CAT CTC CTT CTT-3'*
	reverse	*5'-TCA GAT GAT TCT TCA CCA GAT CCA-3'*
ANGPTL4	forward	*5'-GAT GGC TCA GTG GAC TTC AAC C-3'*
	reverse	*5'-TGA TGC TAT GCA CCT TCT CCA G-3'*
PDK4	forward	*5'-GGT TAC GGC TTG CCA ATT TCT CGT C-3'*
	reverse	*5'-TTG GGA TAC ACC AGT CAT CAG CCT C-3'*
cyclophilin	forward	*5'-GCA TAC GGG TCC TGG CAT CTT GTC C-3'*
	reverse	*5'-ATG GTG ATC TTC TTG CTG GTC TTG C-3'*

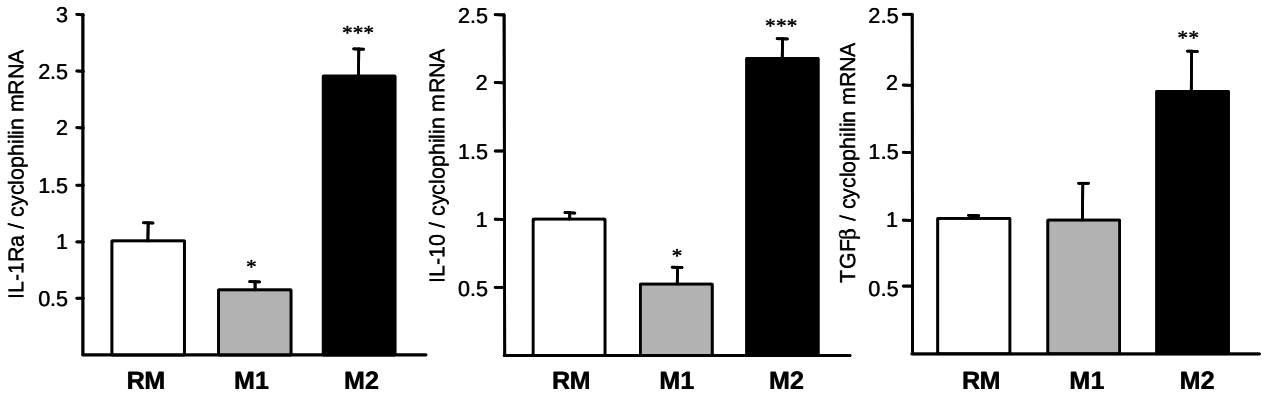
Supplemental figure legends

Figure 1. Primary human macrophages were cultured for 7 days in the absence (RM) or in the presence of IL-4 (15 ng/ml) (M2). Pro-inflammatory M1 macrophages were obtained by activating RM macrophages with LPS (100ng/ml) during 4h. Total RNA was extracted and IL-1Ra, IL-10 and TGFβ mRNA levels measured by Q-PCR and normalized to those of cyclophilin. Results are expressed as the mean value ± SD of triplicate determinations, representative of three independent experiments. Statistically significant differences are indicated (**p<0.01, ***p<0.001).

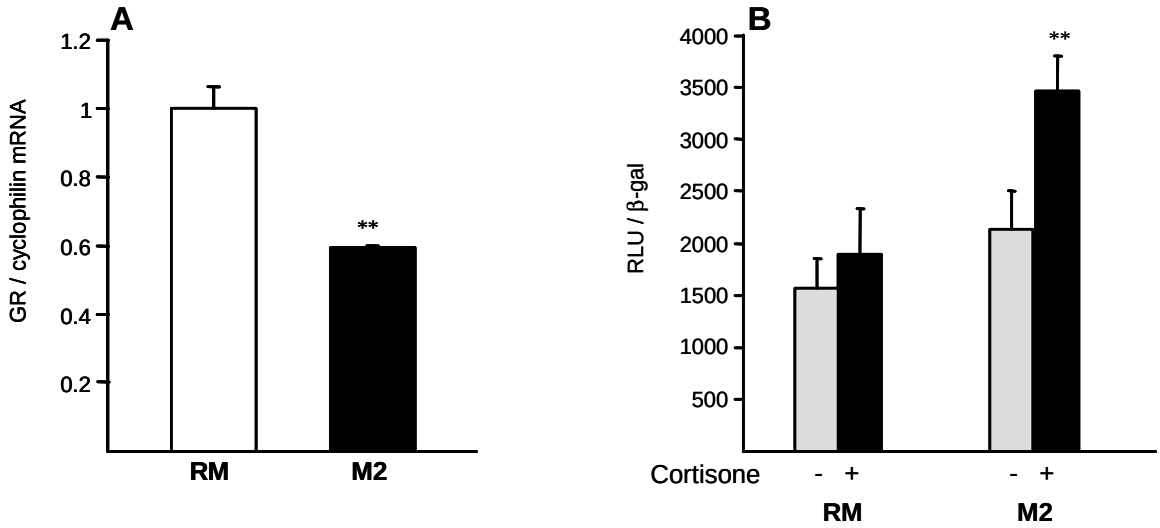
Figure 2. (A) Total RNA was extracted from RM and M2 macrophages and GR mRNA levels measured by Q-PCR and normalized to those of cyclophilin. **(B)** Primary human RM and M2 macrophages were transfected with a reporter construct containing 3 GRE (GR responsive element) sites, treated or not with cortisone (200nM) and luciferase activity was measured. Statistically significant differences are indicated (**p<0.01).

Figure 3. (A) RM macrophages were treated or not with increasing concentrations of rosiglitazone (50, 100nM and 1μM) or **(B)** with GW1929 (600nM) for 6, 12, 24, 36 or 48h. **(C)** RM macrophages were treated for 24h with PPARγ (GW1929; 600nM), PPARα (GW7647; 600nM) or PPARβ/δ (GW501516; 100nM) ligands. **(D, E)** Bone marrow-derived macrophages were treated with increasing concentrations of GW1929 (0.5, 1, 5μM) and rosiglitazone (RSG, 1, 5, 10μM) for 24h. Total RNA was extracted and 11β-HSD1 **(A-D)** and CD36 **(E)** mRNA levels measured by Q-PCR and normalized to those of cyclophilin. Results are expressed as the mean value ± SD of triplicate determinations, representative of three independent experiments. Statistically significant differences are indicated (*p<0.05, **p<0.01, ***p<0.001).

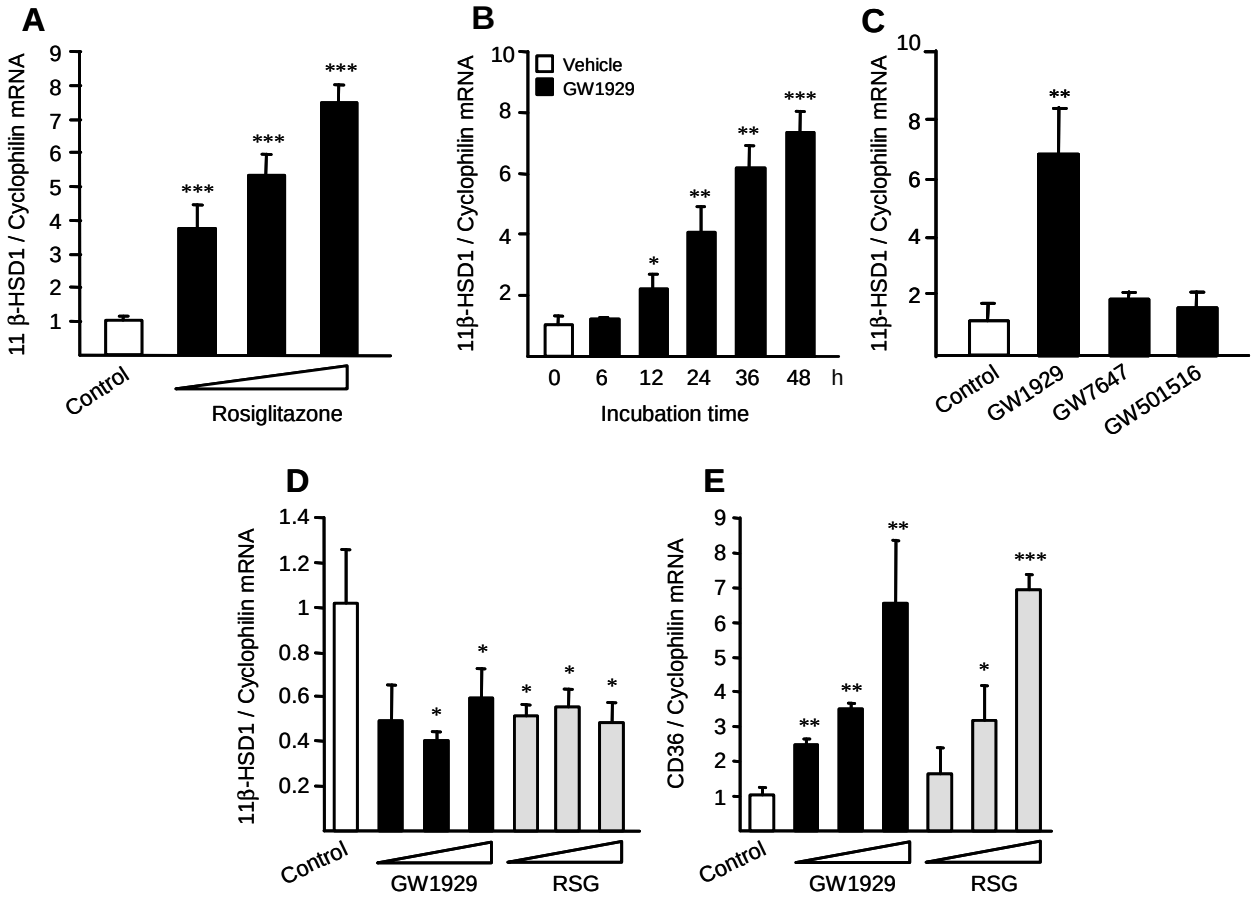
Figure 4. RM **(A)** and M2 **(B)** macrophages were activated or not with GW1929 (600nM) for 24h and subsequently treated for another 24h with cortisone (1µM), RU486 (1µM) or their combination. Total RNA was extracted and ANGPTL4 mRNA levels measured by Q-PCR. Statistically significant differences between control and treated cells are indicated (**p<0.01; ***p<0.001).



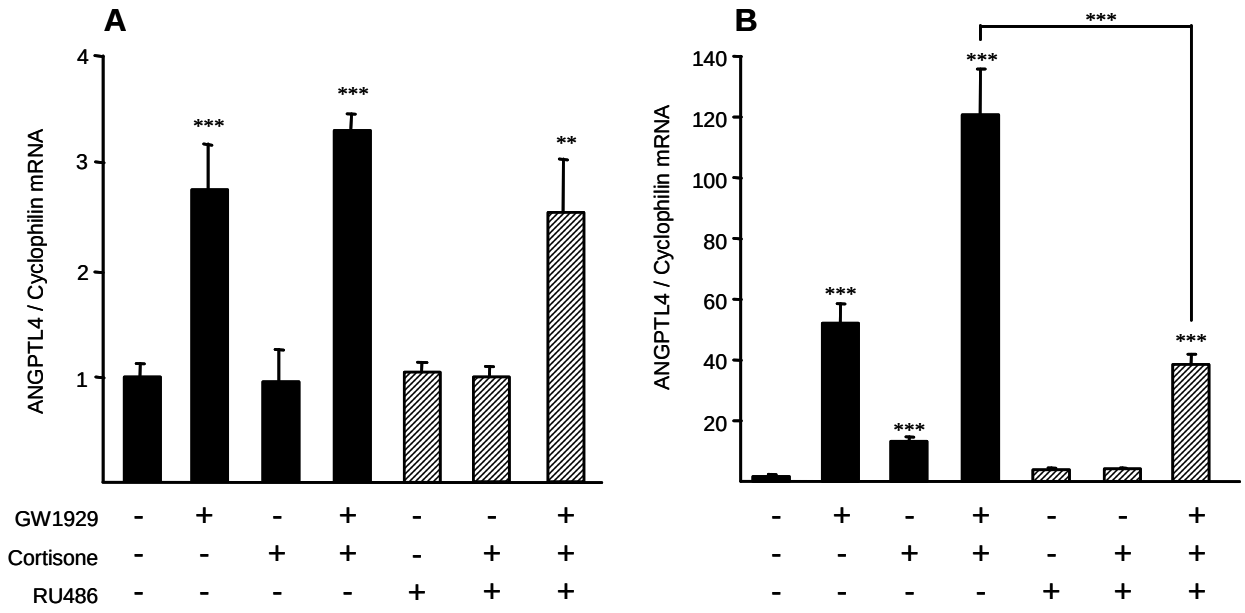
Supplements; figure 1



Supplements; figure 2



Supplements; figure 3



Supplements; figure 4