Supplemental Figure 1

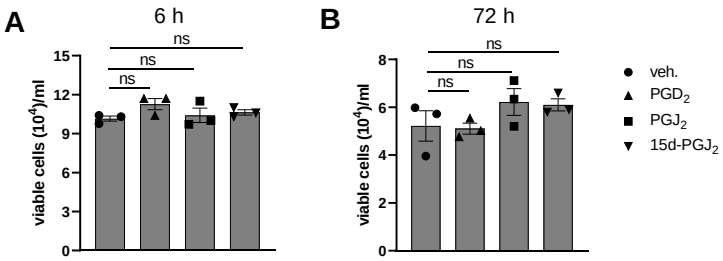

Fig S1. PGD_2, PGJ_2 and 15d-PGJ_2 have no significant cytotoxic effect on *M.tb*-infected macrophage. THP-1 macrophages were pretreated with 10 μM PGD_2, PGJ_2 and 15d-PGJ_2 for 1 h, then infected with H37Ra (MOI of 10:1). Number of viable cells was calculated 6 h (**A**) and 72 h (**B**) post infection. Data are shown as means ± SEM, unpaired *t*-test. ns, not significant.

Supplemental Figure 2

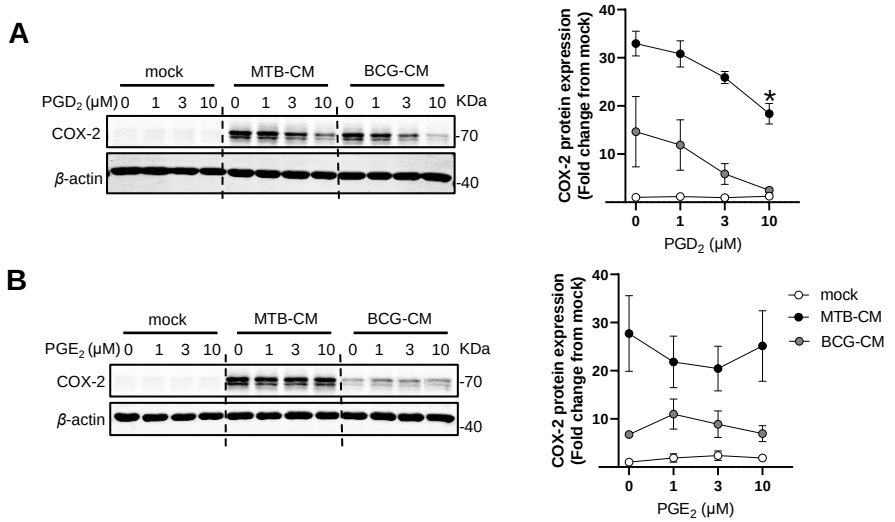

Fig S2. PGD_2 decreases COX-2 expression, but PGE_2 has no effect on COX-2 expression. (A-B) Polarized M1-MDM were pre-treated for 1 h with PGD_2 and PGE_2 at indicated concentration, methyl acetate as a vehicle (veh.) control (v/v), followed by 24 h incubation with 10% MTB-CM or 10% BCG-CM. Cells were lysed and released proteins were analyzed by WB. COX-2 and mPGES-1 expression was detected, *β*-actin was used as a loading control. For densitometric analysis, *β*-actin was used to normalize protein epression. Data are means ± SEM (n = 3 separate donors), **P*<0.05 vs. MTB-CM (veh.) treated cells, paired *t*-test.

Supplemental Figure 3

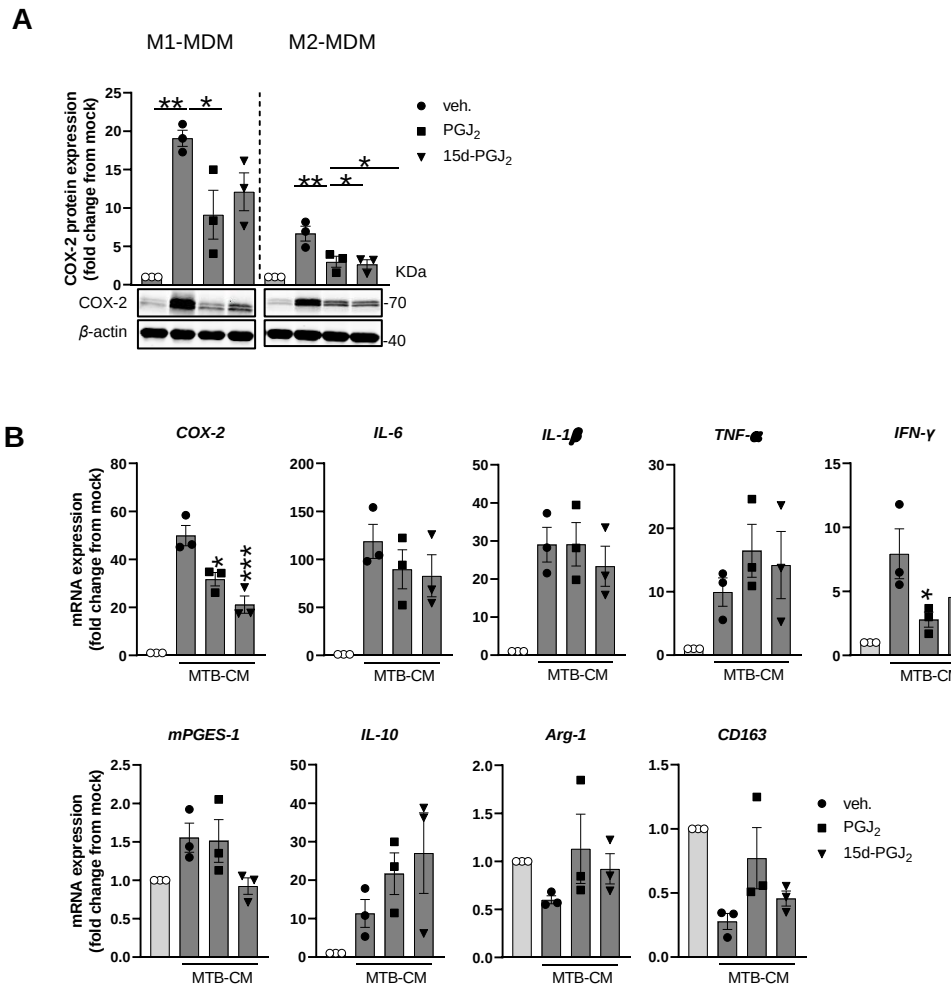

Fig S3. PGJ_2- and 15-PGJ_2-driven modulation of inflammatory responses in MTB-CM stimulated M1/M2-MDM after 12 h incubation. (A) Polarized M1/M2-MDM were pretreated with 10 μM PGJ_2 or 10 μM 15d-PGJ_2 for 1 h, methyl acetate was used as veh. control, followed by 12 h incubation with 10% MTB-CM. Cells were lysed and COX-2 protein and β-actin were analyzed by WB. Data are means ± SEM (n = 3 separate donors). *P<0.05; **P < 0.01, one-way ANOVA with Tukey's multiple comparisons test. (B) Polarized M1-MDM were pretreated with 10 μM PGJ_2 or 10 μM15d-PGJ_2 for 1 h, methyl acetate was used as veh. control, followed by 12 h incubation with 10% MTB-CM. mRNA levels of indicated genes were analyzed by real-time PCR. Data are means ± SEM (n = 3 separate donors). The data were analyzed by one-way ANOVA with Tukey's multiple comparisons test. *P<0.05; **P < 0.01 vs MTB-CM group.

Supplemental Figure 4

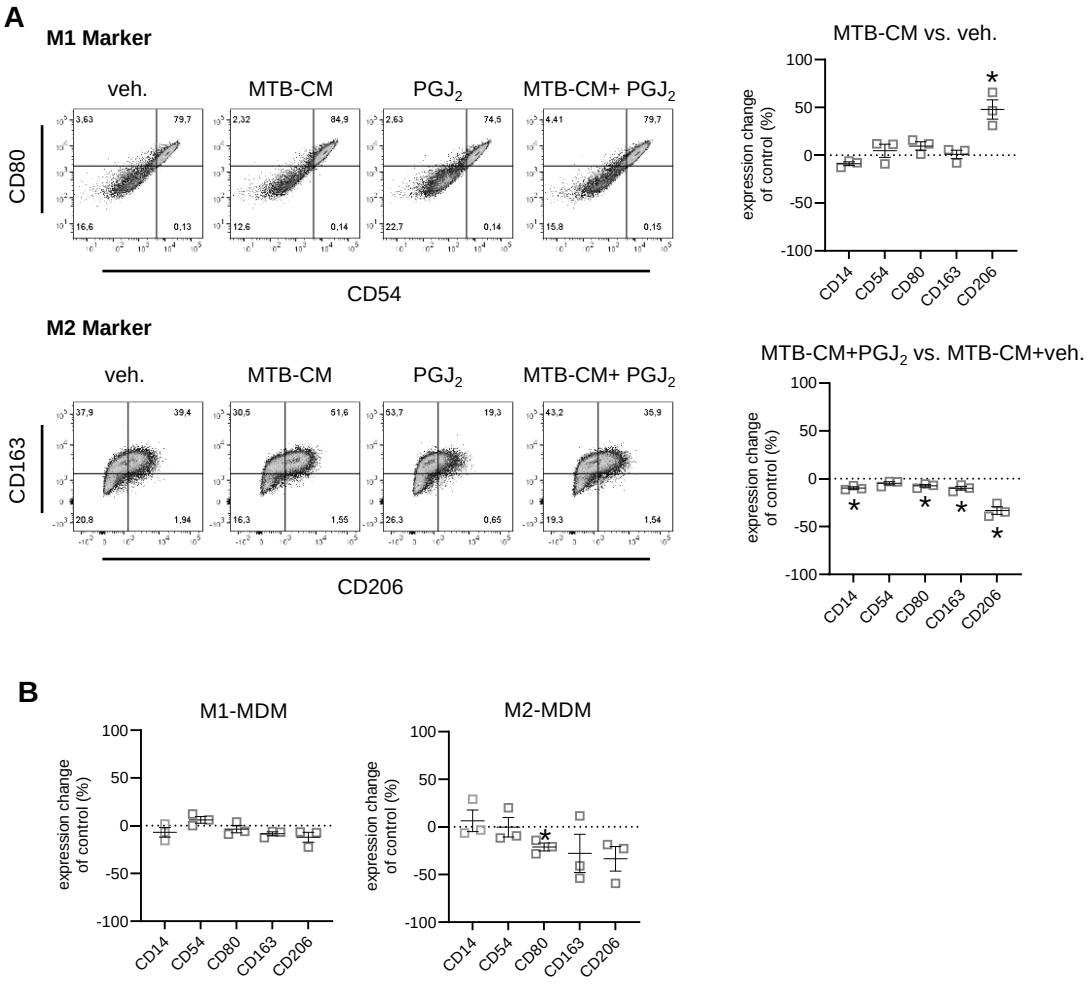

Fig S4. Impact of PGJ_2 on M1-/M2-like surface marker expression in MDM. (A) Polarized M1-MDM were pretreated with 10 µM PGJ_2 or methyl acetate as vehicle (veh.) control (v/v) for 1 h and incubated with MTB-CM for 24 h. Expression of M1-like surface markers CD54 and CD80 and of M2-like surface markers CD163 and CD206 were analyzed by flow cytometry among living $CD14^+$ cells.) Data are shown are representative pseudo color dot plots (left). Mean fluorescence intensity (MFI) of each marker was determined (right). The change of the MFI from MTB-CM-stimulated group against mock group and MTB-CM plus PGJ_2 group against MTB-CM group was calculated and is given in % in scatter dot plots as single values and means ± SEM, n=3. **P*<0.05, ratio paired *t*-test. (**B**) $M0_{GM\text{-}CSF}$ and $M0_{M\text{-}CSF}$ (2×10^6 cells/ml , each) were preincubated with 10 µM PGJ_2 or vehicle (v/v methyl acetate) for 30 min and then polarized at 37 °C for 24 h towards M1 and for 48 h towards M2 phenotypes. The surface markers CD54 and CD80 (M1-like) as well as CD163 and CD206 (M2-like) among living $CD14^+$ cells were analyzed, the mean fluorescence intensity (MFI) of each marker was determined and is given as mean ± SEM as % of vehicle control in scatter dot plots with individual values, n=3, ratio paired *t*-test vs. vehicle control,**P* < 0.05.