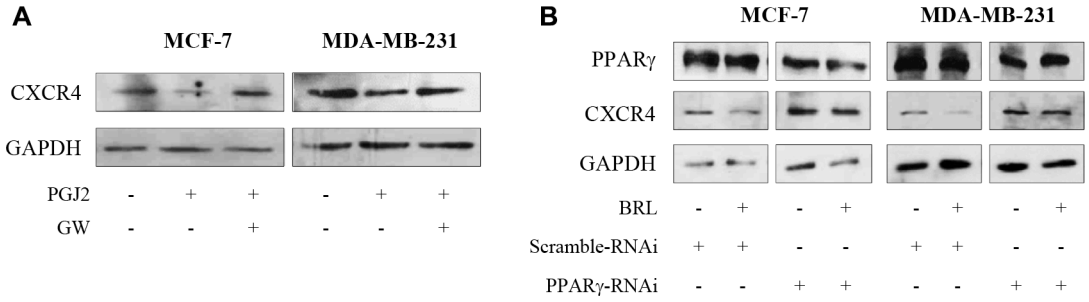
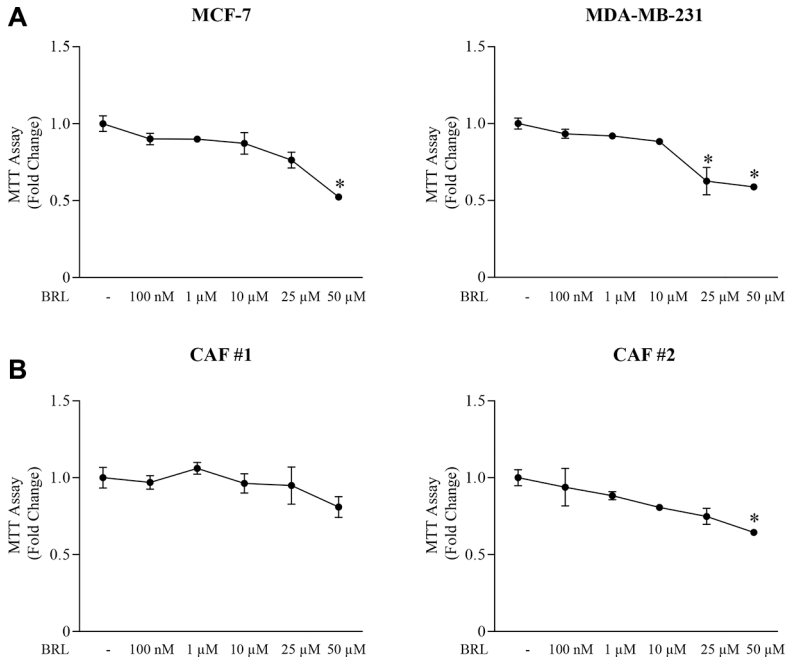
Ligand-activated PPARγ downregulates CXCR4 gene expression through a novel identified PPAR response element and inhibits breast cancer progression

Supplementary Materials



Supplementary Figure S1: (**A**) Immunoblots of CXCR4 protein expression in MCF-7 and MDA-MB-231 cells treated with vehicle (−), PGJ2 at 10 μM with or without GW 10 μM for 24 h. GAPDH was used as loading control. (**B**) Immunoblots of CXCR4 protein expression in MCF-7 and MDA-MB-231 cells transfected with scramble RNA interference (RNAi) or with PPARγ RNAi as reported in Materials and Methods Section and treated with vehicle (−) or with BRL 10 μM for 24 h. GAPDH was used as loading control.



Supplementary Figure S2: Cell viability was determined by 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium (MTT) assays in MCF-7 and MDA-MB-231 breast cancer cells (A) and in Cancer-Associated Fibroblasts (CAF) (B) treated with vehicle (−) or with increasing concentrations (100 nM, 1, 10, 25, 50 μM) of BRL for 24 h. The results are expressed as fold change respect to vehicle-treated cells. The values represent the mean ± SD of three different experiments, each performed with triplicate samples. *$P < 0.05$ vs vehicle-treated cells.