Supplemental Experimental Procedures

Method, reporter assay

3T3-L1 cells were plated at a concentration of 3.0x 104 cells/well in 24-well plates one day before transfection. Transfection with Lipofectamin 2000 (Invitrogen, Carlsbad, CA) according to the manufacturer's instruction. Briefly, per 1.9 cm2 well, 0.3μg pSG5-hPPARγ2, 0.15μg J3TH-luc, 0.0025 μg pRL renilla (Promega, Madison, WI) were mixed with 1 μl Lipofectamine 2000 in 100 μl OPTIMEM. Trasfection performed for 5h. Medium was changed to DMEM 10% FCS. 24 h post transfection cells were treated for 24 h with Wnt3a conditioned media 20% or Tnfα 5ng/ml (Sigma-Aldrich St Louis, MO) added 30 minutes before treatment with 100nM Rosiglitazon (Cayman Chemical, Ann Arbor, MI). Cell lysis and luciferase assay with Promega Dual Luciferase Assay (Promega, Madison, WI) according to the manufacturer's instruction. The pSG5-hPPARγ2 and the J3TH-luc plasmids were generous gifts from Dr.Johan Auwerx, Strasburgh, France.

Supplementary figure 1

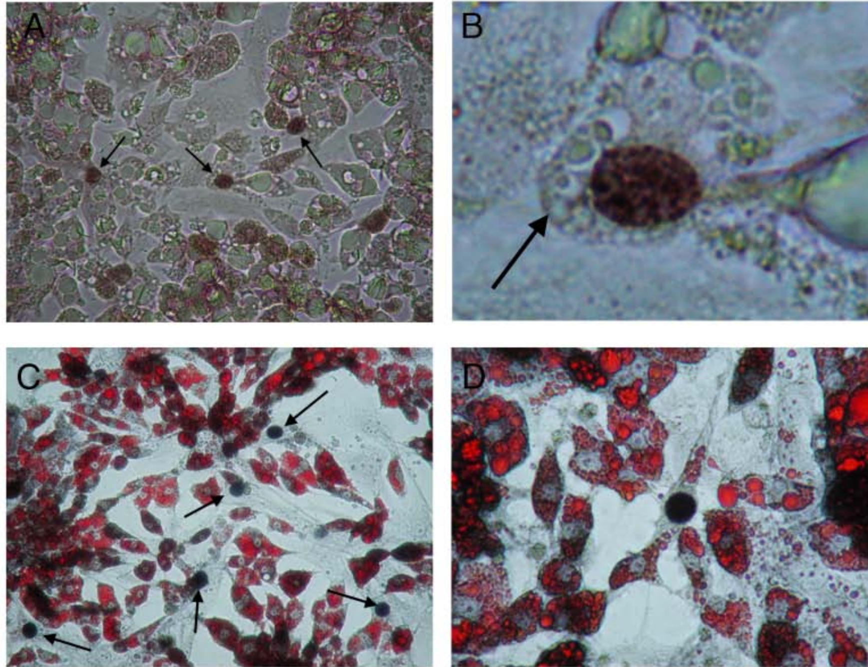

Fig. 1. Fully differentiated 3T3-L1 adipocytes were incubated with 10% Wnt3a-CM for 3 days. (*A*) Arrows showing BrdU uptake in proliferating dedifferentiated adipocytes.(*B*) BrdU uptake in an adipocyte, arrow showing lipid droplets around the nucleus. (*C and D*) Double staining with Oil Red O. No uptake of BrdU was seen in control cells without Wnt3a-CM. BrdU labelling was performed with Amersham Cell Proliferation Kit (RPN20, GE Healthcare, UK Ltd, UK).

Supplementary figure 2

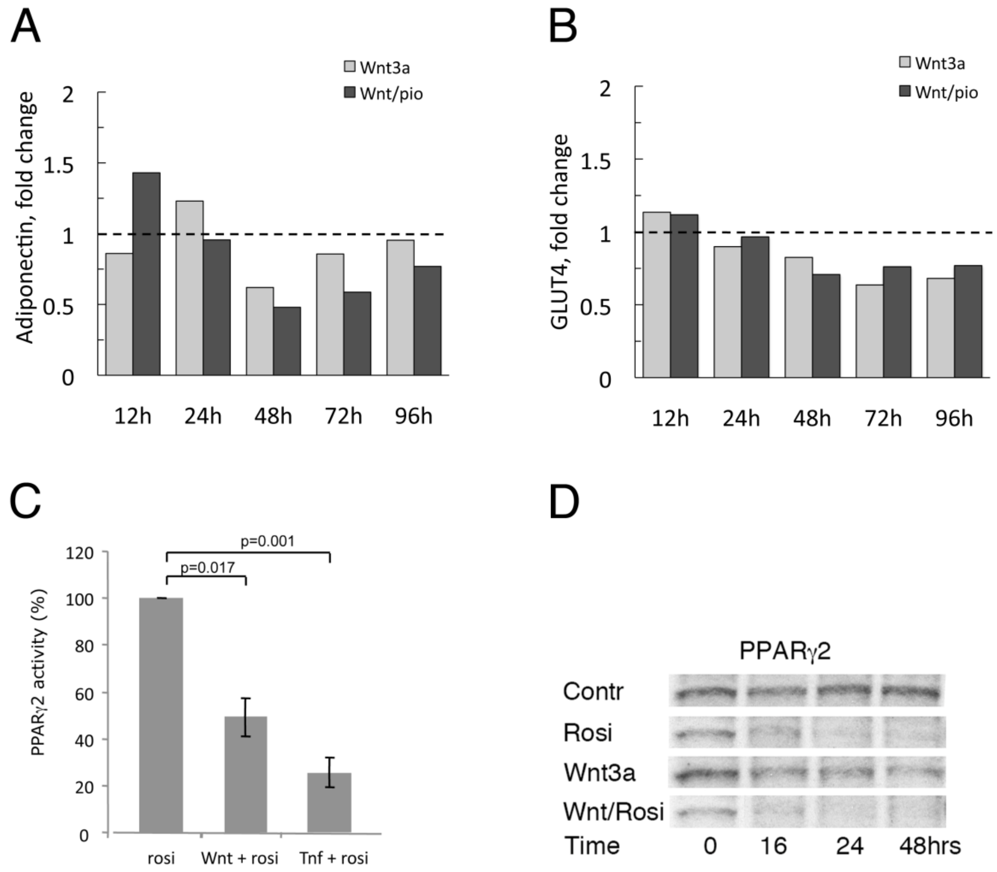

Fig. 2. Quantitation of adiponectin (*A*) and GLUT4 (*B*) mRNA in Wnt3a and Wnt3a+ pioglitazone stimulated 3T3-L1 adipocytes. RNA was extracted and mRNA levels determined with real-time PCR. The data were fist normalized to 18S rRNA then normalized to expression levels in the control sample (=1). (*C*) Comparison between the ligand-activated PPARg mediated by Wnt 3a and Tnfα. PPAR reporter assay. Wnt 3a and Tnfα inhibits the PPARγ activity induced by rosiglitazon. The results are means + SEM of three/four separate experiments. Paired t-test was used to evaluate the differences between treated and untreated samples. Method, see Supplementary Experimental Procedures. (*D*) Western blotting of PPARγ2. Cell lysates were prepared from Wnt3a and rosiglitazone treated 3T3-L1 adipocytes at indicated time points.

Supplementary figure 3.

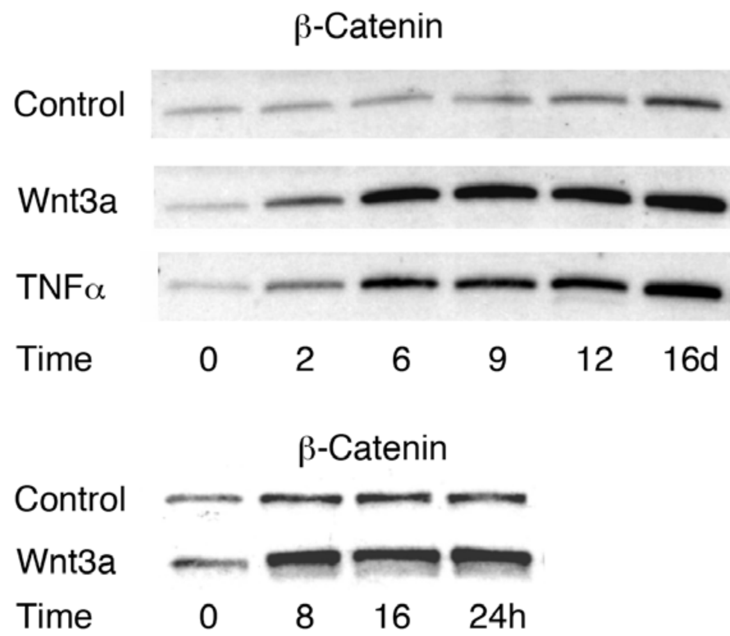

Fig. 3. Western blotting was performed on extracts from Wnt3a- and TNFα-incubated 3T3-L1 adipocytes to test for changes in β-catenin.

Supplementary figure 4

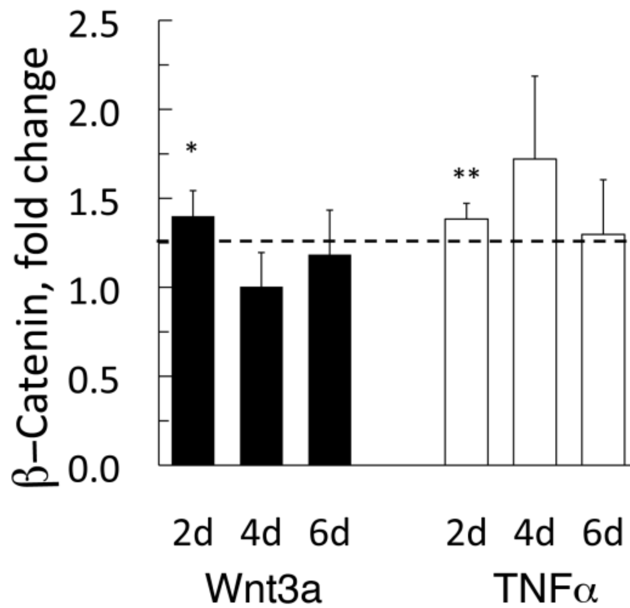

Fig. 4. Wnt3a and TNFα-induced mRNA expression of β-catenin. The data were first normalized to 18S rRNA then normalized to expression levels in the control sample (=1).
* $p<0.05$, ** $p<0.002$ compared with untreated.

Supplementary figure 5

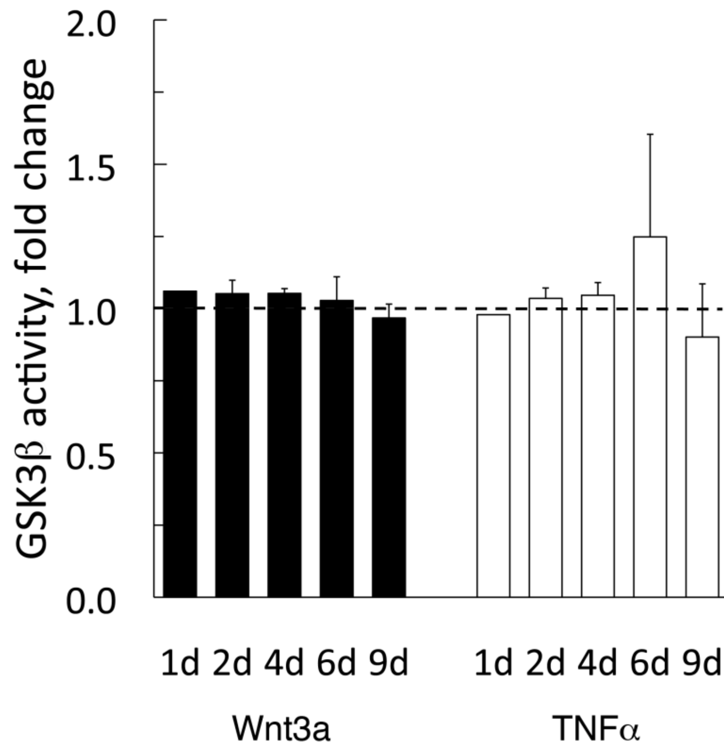

Fig. 5. GSK3β-activity was measured in lysates from 3T3-L1 adipocytes stimulated with Wnt3a and TNFα. Kinase activity was measured with Kinase-Glo Luminiscent Kinase Assay (Promega) and Calbiochem GSK3β Substrate (Cat. No 361530) was used as substrate. Results from four independent experiments are shown and the results are normalized against control values for each time point (mean +/- SEM).

Supplementary figure 6

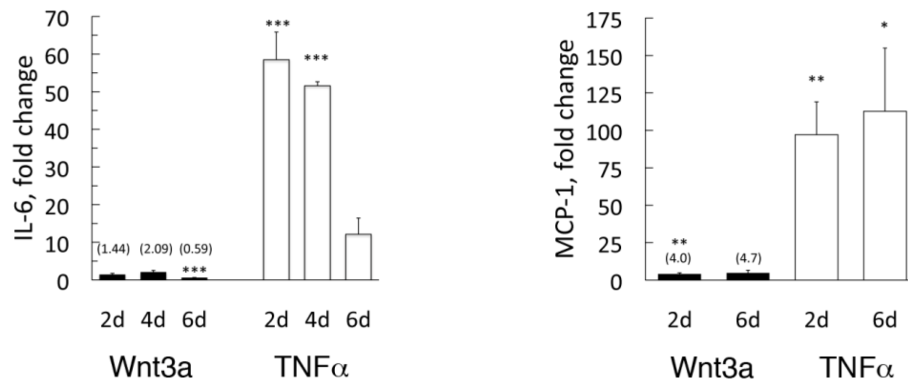

Fig. 6. Quantitation of IL-6 and MPC-1 mRNA in Wnt3a or TNFα stimulated adipocytes. RNA was extracted and mRNA levels determined with real-time PCR. The data were fist normalized to 18S rRNA then normalized to expression levels in the control sample (=1). Data are presented as the mean ± SEM (n = 4). *p < 0.05, **p < 0.02 and ***p<0.002 compared with untreated.