Supplemental Fig. 1. BBR suppresses the expression of adipogenic genes in the adipose tissues of db/db mice. Total RNA and cDNA were obtained as described in Fig. 1A. Expression of adipogenic/lipogenic marker genes including aP2, FAS, ACC1, PPARγ, and LPL in WAT was analyzed by qRT-PCR and normalized by GAPDH. Each bar represents mean ± S.D. of five individual animals. * P<0.05 vs. db/db + vehicle mice.

Supplemental Fig. 2. Dose response of BBR on inflammatory gene in macrophages. RAW 264.7 cells were pre-treated with various concentrations (1, 2.5, 5, and 10 μM relatively) of for 2 hours and then cells were incubated with LPS (10 ng/ml) for 6 hours. mRNA level of IL-1β, IL-6, iNOS, COX2, and MMP9 were measured by use of qRT-PCR and normalized by GAPDH. * P<0.05 vs. negative control, ## P<0.01 vs. negative control, ### P<0.001 vs. negative control, * P<0.05 vs. LPS, ** P<0.01 vs. LPS, *** P<0.001 vs. LPS.

Supplemental Fig. 3. BBR does not induce cytotoxicity in RAW 264.7 macrophages. Cells were treated with various concentrations (0.78, 1.56, 3.125, 6.25 and 12.5 μM) of BBR for 24 hours, and cell viability was determined by use of MTS assay kit. Precipitated formazan reduced from MTS by
living cells was measured at the absorbance of 492 nm. Numbers of living cells were compared to vehicle-treated control and shown in percentage. Data are representative of two independent experiments performed in triplicate.

Supplemental Fig. 4. Dominant negative AMPKα1 blunts AMPK phosphorylation induced by BBR. RAW 264.7 macrophages were transfected with Mock or DN-AMPKα1 expression vector and treated with BBR (5 μM) for 120 minutes. The levels of both phosphorylated AMPK and AMPKα1 in total cell lysates were determined by Western blot analysis. Ratio of p-AMPK/AMPK was represented in number.