Supplemental Figure 1. A, EGCG treatment had no effect on the SIRT1 activity. SIRT1 activity was assayed with an SIRT1/sir2 Deacetylase Fluorometric Assay Kit. Deacetylase enzyme activity was assessed by measuring the fluorescence intensity. Nicotinamide was used as a control inhibitor for SIRT1 activity. The results represent the average values ± SD of three independent experiments. B. EGCG uncompetitively inhibits the p300/CBP HAT activity. Lineweaver-Burk plot showing the effect of EGCG on p300-mediated acetylation of core histones. HAT assays were carried out with a fixed concentration of histones (8 PM) and increasing concentration of [^{3}H]acetyl CoA in the presence (50 and 100 μM) or absence of EGCG (left panel), the same LB plot of EGCG effect on p300 HAT activity at a fixed concentration of [^{3}H]acetyl CoA (354 nM) and increasing concentrations of histones (0.033-0.165 μM) in the presence (50 and 100 μM) or absence of EGCG (right panel). The results were plotted using Sigma Plot Ver 10.0 (SYSTAT Software). C, Histone H4 tail peptide was acetylated efficiently by GST-p300 (HAT) efficiently acetylated, but not by GST-p300 (ΔHAT). Synthetic H4 tail peptide (1 μg) was used for in vitro acetylation assay with either GST-p300 (HAT) or GST-p300 (ΔHAT). Reaction products were analyzed by SDS-PAGE and autoradiography.

Supplemental Figure 2. A, The effect of EGCG treatment on the protein expression of target genes was analyzed by Western blot analysis with antibodies as indicated. B, Primary peritoneal cells from EGCG treated or untreated mice were washed in PBS and were cultured for 2 days. The level of p65 acetylation was assessed by Western blot analysis with antibody against acetyl-p65. C, EGCG generally inhibits inflammatory responses. The effect of EGCG treatment on the expression of IL-6 gene in HEK293 was examined by RT-PCR analysis. The level of p65 acetylation in the HEK293 cells was assessed by Western blot analysis.