Supplement Data.

Figure S1 DT-13 exhibited no obvious effect on unactivated ECs *in vitro* and *in vivo*.

(A) The cytotoxicity of DT-13 detected by MTT assay. After 48 h of incubation with various concentrations of DT-13, the viability of the treated cells was determined by MTT assays. No significant cytotoxic effects of DT-13 on HUVECs were observed with concentrations up to 1 μM. (B) HUVECs were pretreated with 1 μM DT-13 without TNF-α stimulation. No significant difference between the control and the DT-13-treated group on HUVECs adhesion was noted. Bar=20 μm. (C) No differences were noted between the control group and DT-13-treated group regarding the number of mouse monocyte WEHI3 cells bound to the endothelium of mouse aortas. Bar=100 μm. (D) DT-13 exhibited no significant effect on the inhibition of leukocyte migration in normal mice. The data represent the mean ± SD of three experiments.
Figure S2 DT-13 inhibited TNF-α-induced the activation and degradation of IκB-α in HUVECs.

HUVECs were pretreated with DT-13 (0.01, 0.1 or 1 μM) for 1 h followed by TNF-α (10 ng/ml) exposure. The expression of phospho-IκB-α (B) and IκB-α (C) were detected by western blotting. The data represent the mean ± SD of three experiments. # $P<0.05$, ## $P<0.01$ vs. the control group; * $P<0.05$, ** $P<0.01$ vs. the TNF-α group.
Figure S3 The over-expression of NF-κB-p65.

HUVECs were transfected with 1 μg of pNF-κB–M98 or pcDNA for 43 h, cells were pretreated with or without DT-13 at 1 μM for 1 h before TNF-α (10 ng/ml) induction for 4 h. The expression of p65 was detected by western blotting. The data represent the mean ± SD of three experiments. ** P<0.01 vs. corresponding groups.