

Supplementary Data 1. Suppressive effect of Wnt-C59 on lipopolysaccharide (LPS)-induced proinflammatory cytokine expression in human umbilical vein endothelial cells. (A–F) Cells were treated with 0 or 50 μ M of Wnt-C59, followed by stimulation with 0.1 μ g/ml of LPS for various time periods of 0.5 to 4 h. (A–E) Messenger RNA levels of proinflammatory cytokines were measured by RT-qPCR. (F) Cell viability was measured. Cells treated with 0 or 50 μ M of Wnt-C59, followed by stimulation with 0.1 μ g/ml of LPS for various to 50 μ M of Wnt-C59 with the same duration of LPS stimulation were compared. **p < 0.01, ***p < 0.001. (G–L) Cells were treated with 0 to 50 μ M of Wnt-C59, followed by stimulation with 0.1 μ g/ml of LPS for 4 h. (G–K) Messenger RNA levels of proinflammatory cytokines were measured by RT-qPCR. (L) Cell viability was measured. *p < 0.01, ***p < 0.001 compared with cells stimulated with LPS with 0 μ M of Wnt-C59. ***p < 0.001 compared with cells stimulated cells. Experiments were conducted in triplicate. Data are shown as mean ± standard deviation, and statistical significance was measured by unpaired t-test.







A BEAS-2B cells B RAW 264.7 cells



Supplementary Data 3. Input samples of co-immunoprecipitation experiments. Input samples of the co-immunoprecipitation experiments of Fig. 7A–D and Fig. 8A–D were analyzed by Western blotting with anti-NF- κ B p65 and anti- β -catenin antibody. β -Actin was used as an equal loading control. The total amount of NF- κ B showed no variation and the amount of β -catenin showed same patterns with Figs. 3F and 4F.