Figure S1. Quantitative analysis of the immunoblotting data presented in Fig. 1. (A and B) The ratio of pp65/p65 presented in Fig. 1C and D, respectively. (C) The ratio of p65/Lamin B1 presented in Fig. 1E. (D) The ratio of pp65/p65 presented in Fig. 1F. *P<0.05. pp65, phosphorylated p65.



Figure S2. Ginsenoside Rg5 inhibits the phosphorylation of p65 in A549 cells. (A and B) Western blotting analysis of p65 expression and phosphorylation levels in A549 cells treated with various concentrations of ginsenoside Rg5 and stimulated with (A) TNF- α or (B) IL-1 β . pp65, phosphorylated p65.



Figure S3. Quantitative analysis of immunoblotting data presented in Fig. S2. (A) The ratio of pp65/p65 presented in Fig. S2A. (B) The ratio of pp65/p65 presented in Fig. S2B. *P<0.05. pp65, phosphorylated p65.



Figure S4. Quantitative analysis of immunoblotting data presented in Fig. 2. (A-C) The ratios of (A) pTAK1/TAK1, (B) pIKK/IKK α and (C) I κ B α / β -actin presented in Fig. 2A. (D-F) The expression levels of (D) TAK1, (E) TAB1 and (F) TAB2 presented in Fig. 2B. *P<0.05 vs. DMSO-treated cells. TAK1, transforming growth factor β -activated kinase 1; IKK, I κ B kinase; TAB1, TAK1-binding protein 1; TAB2, TAK1-binding protein 2; p, phosphorylated.



Figure S5. Ginsenoside Rg5 inhibits the phosphorylation of TAK1 by binding to the TAK1 complex. (A and B) Western blotting analysis of TAK1 expression and phosphorylation levels in the lysates of A549 cells were treated with DMSO or 50 μ M ginsenoside Rg5 and exposed to (A) TNF- α or (B) IL-1 β for the indicated duration. (C and D) Thermal shift assay analysis of (C) A549 or (D) TAK1, TAB1 or TAB2 plasmid-transfected 293T cells treated with DMSO or 50 μ M ginsenoside Rg5. (E) Western blotting analysis of TAK1, TAB1 and TAB2 expression levels in whole-cell lysates of 293T cells transfected with the overexpression plasmids or the empty pcDNA 3.1 vector. TAK1, transforming growth factor β -activated kinase 1; TAB1, TAK1-binding protein 1; TAB2, TAK1-binding protein 2; p, phosphorylated.



Figure S6. Quantitative analysis of immunoblotting data presented in Fig. S5. (A and B) The ratios of pTAK1/TAK1 presented in (A) Fig. S5A and (B) Fig. S5B. (C-H) The ratios of (C and F) TAK1, (D and G) TAB1 and (E and H) TAB2 presented in (C-E) Fig. S5C and (F-H) Fig. S5D. *P<0.05 vs. DMSO-treated cells. TAK1, transforming growth factor β -activated kinase 1; TAB1, TAK1-binding protein 1; TAB2, TAK1-binding protein 2; p, phosphorylated.



Figure S7. Quantitative analysis of immunoblotting data presented in Fig. 3. (A and B) The ratios of (A) pEphA2/EphA2 and (B) EphA2/ β -actin presented in Fig. 3A. *P<0.05 vs. TNF- α . (C-E) The ratios of (C) pEphA2/EphA2, (D) EphA2/ β -actin and (E) pRSK/RSK1 presented in Fig. 3B. (F and G) The ratios of EphA2/ β -actin presented in (F) Fig. 3C and (G) Fig. 3D. ns, non-significant; *P<0.05 vs. DMSO-treated cells. EphA2, erythropoietin-producing hepatocellular receptor A2; RSK, p90 ribosomal S6 kinase; CHX, cycloheximide; BA1, bafilomycin A1; p, phosphorylated.



Figure S8. Molecular mechanisms by which ginsenoside Rg5 inhibits EphA2 expression. (A and B) Western blotting analysis of EphA2 pathway protein expression and phosphorylation levels in A549 cells were pre-treated with various concentrations of ginsenoside Rg5 and stimulated with (A) TNF- α or (B) IL-1 β . (C and D) Reverse transcription-quantitative PCR analysis of *EPHA2* mRNA expression levels in (C) HeLa and (D) A549 cells treated with DMSO or 50 μ M ginsenoside Rg5. n=3. (E and F) Western blotting analysis of EphA2 pathway protein expression and phosphorylation levels in (E) HeLa cells and (F) A549 cells treated with DMSO or 50 μ M ginsenoside Rg5 for the indicated times. (G and H) Western blotting analysis of EphA2 expression levels in A549 cells treated with (G) 50 μ g/ml CHX and 10 nM MG-132 or (H) 10 nM BA1 for the indicated times, followed by treatment with DMSO or 50 μ M ginsenoside Rg5. EphA2, erythropoietin-producing hepatocellular receptor A2; RSK, p90 ribosomal S6 kinase; CHX, cycloheximide; BA1, bafilomycin A1; p, phosphorylated.



Figure S9. Quantitative analysis of immunoblotting data presented in Fig. S8. (A-C) The ratios of (A) pEphA2/EphA2, (B) EphA2/β-actin and (C) pRSK/RSK1 presented in Fig. S8A. (D-F) The ratios of (D) pEphA2/EphA2, (E) EphA2/β-actin and (F) pRSK/RSK1 presented in Fig. S8B. (G and H) The ratios of pEphA2/EphA2 presented in (G) Fig. S8E and (H) Fig. S8F. (I and J) The ratios of EphA2/β-actin presented in (I) Fig. S8G and (J) Fig. S8H. ns, non-significant; *P<0.05 vs. DMSO-treated cells. EphA2, erythropoietin-producing hepatocellular receptor A2; RSK, p90 ribosomal S6 kinase; CHX, cycloheximide; BA1, bafilomycin A1; p, phosphorylated.



Figure S10. Representative images of cell migration assays. (A) Transfection efficiency of the EphA2, RSK1 and p65 expression plasmids in 293 cells. (B-D) Representative images of the migration assays presented in (B) Fig. 4A, (C) Fig. 4C and (D) Fig. 4D.



