Figure S1. B16BL6 cells expressed higher levels of p-c-Kit and c-Kit compared with B16F1 cells. (A) Expression levels of p-c-Kit and c-Kit in B16F1 and B16BL6 cells were detected via western blotting. (B) Semi-quantification of the ratio of p-c-Kit/c-Kit. β-actin was used as internal control. Data are presented as the mean ± SD of three independent experiments. *P<0.05 vs. B16F1. p-, phosphorylated.

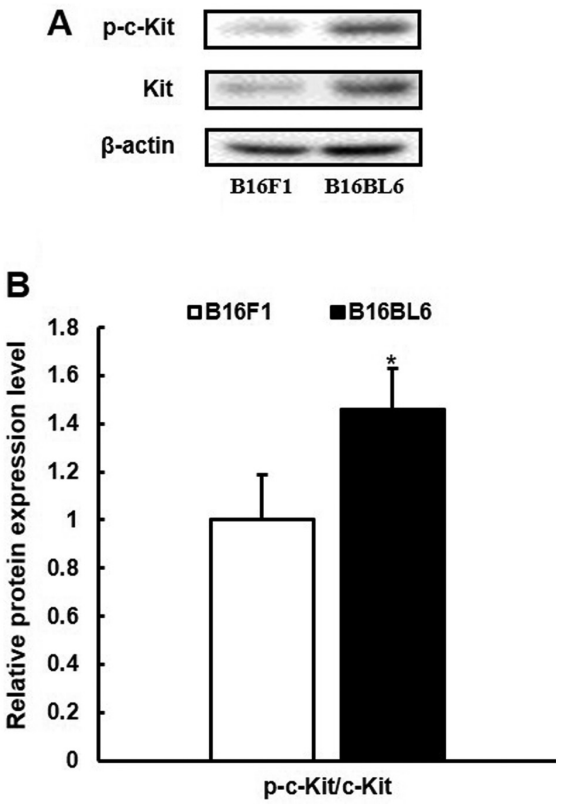

Figure S2. Sorafenib inhibits the viability of B16F1 cells. B16F1 cells were treated with sorafenib (0.1, 0.5, 1, 5, 10, 25 and 50 μM). After incubation, the cells were stained with trypan blue and the number of stained cells was counted at 1, 3 and 5 days. The results are representative of three independent experiments. *P<0.05 vs. control.

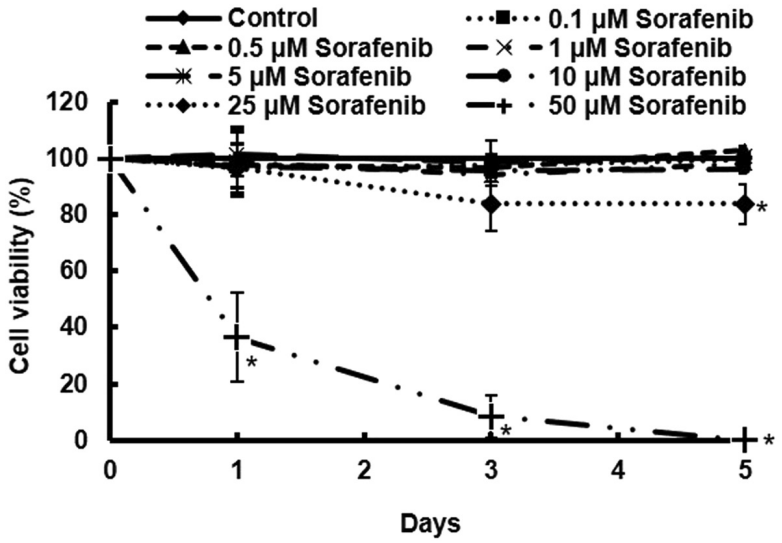

Figure S3. Sorafenib inhibits the migration and invasion of melanoma cells. (A) B16BL6 and MeWo cells were treated with sorafenib (0.5, 1, 5 and 10 μM), and the migrated cells were measured in Falcon cell culture inserts. Representative images of migration assay of B16BL6 and MEWO cells. Magnification, x20. Scale bar, 50 μm. (B) B16BL6 and MeWo cells were treated with sorafenib (0.5, 1, 5 and 10 μM), and the migrated cells were measured in Falcon cell culture inserts with Matrigel. Representative images of invasion assay of B16BL6 and MEWO cells. Magnification, x20. Scale bar, 50 μm.

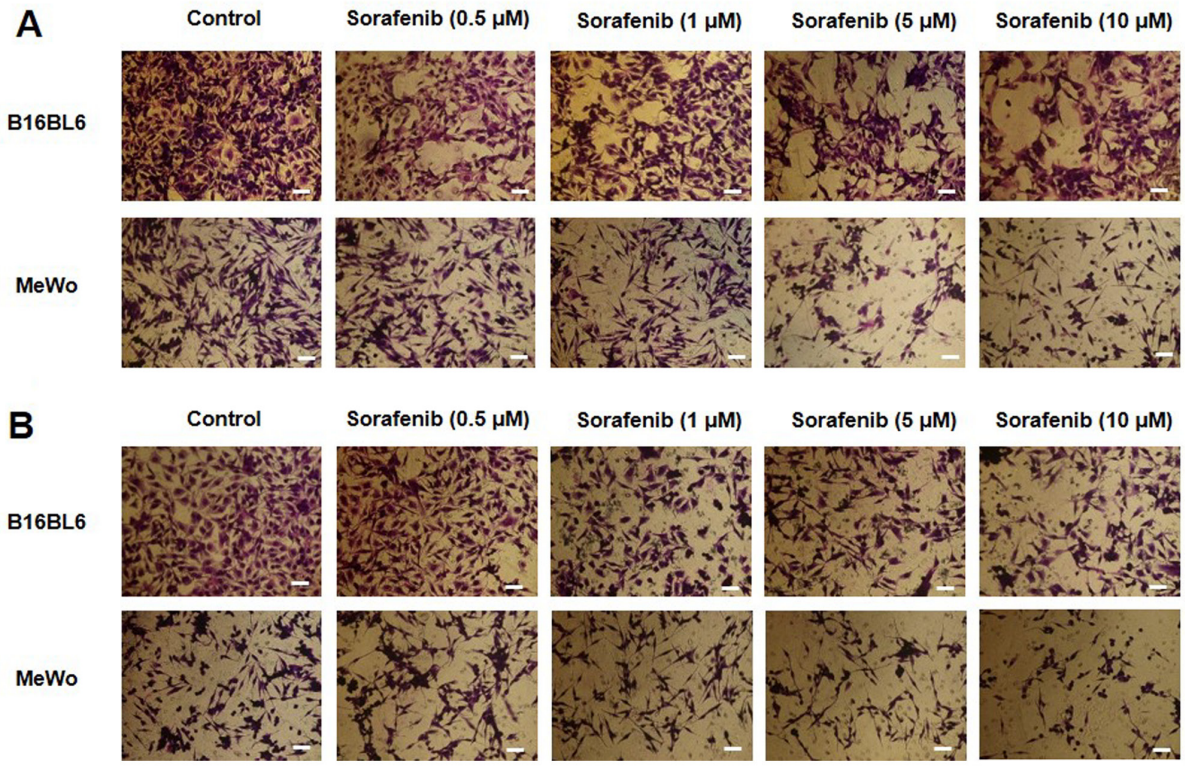

Figure S4. Sorafenib inhibits c-Kit, PDGFR, VEGFR, B-Raf and c-Raf signaling pathways in melanoma cells with c-Kit aberration. B16BL6 cells were treated with sorafenib (0.5, 1, 5 and 10 μM), and the protein expression levels of p- and total c-Kit, PDGFR, VEGFR, B-Raf, c-Raf, ERK, Akt, STAT3, NF-κB and p38 were analyzed via western blotting. β-actin was used as internal control. Semi-quantification of the amount of p-c-Kit, p-PDGFR, p-VEGFR, p-B-Raf, p-c-Raf, p-ERK, p-Akt, p-STAT3, p-NF-κB and p-p38, normalized to the amount of c-Kit, PDGFR, VEGFR, B-Raf, c-Raf, ERK, Akt, STAT3, NF-κB and p38. Data are presented as the mean ± SD of three independent experiments. *P<0.05 vs. control. p-, phosphorylated; PDGFR, platelet derived growth factor receptor.

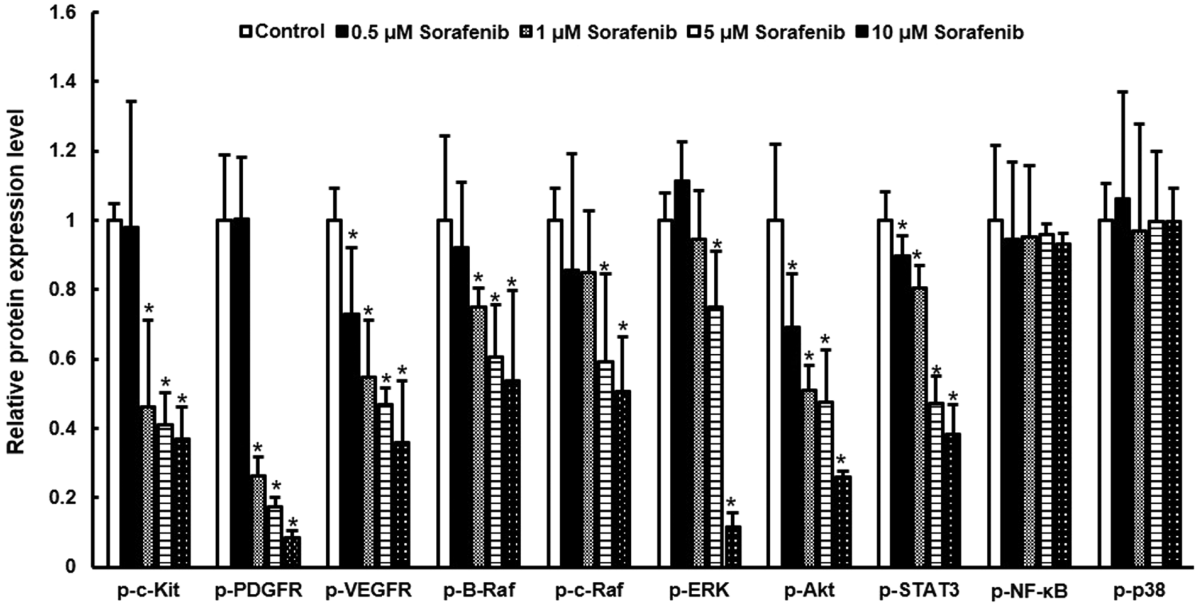

Figure S5. Sorafenib decreases the protein expression levels of MMP-14, VLA-1, VLA-2, VLA-4, VLA-5 and VLA-6 *in vitro*. B16BL6 cells were treated with sorafenib (0.5, 1, 5 and 10 μM), and the expression levels of MMP-14, VLA-1, VLA-2, VLA-3, VLA-4, VLA-5 and VLA-6 were analyzed via western blotting. β-actin was used as internal control. Semi-quantification of the amount of (A) MMP-14, (B) VLA-1, VLA-2, VLA-3, VLA-4, VLA-5 and VLA-6, normalized to the amount of β-actin. Data are presented as the mean ± SD of three independent experiments. *P<0.05 vs. control. VLA, very late antigen.

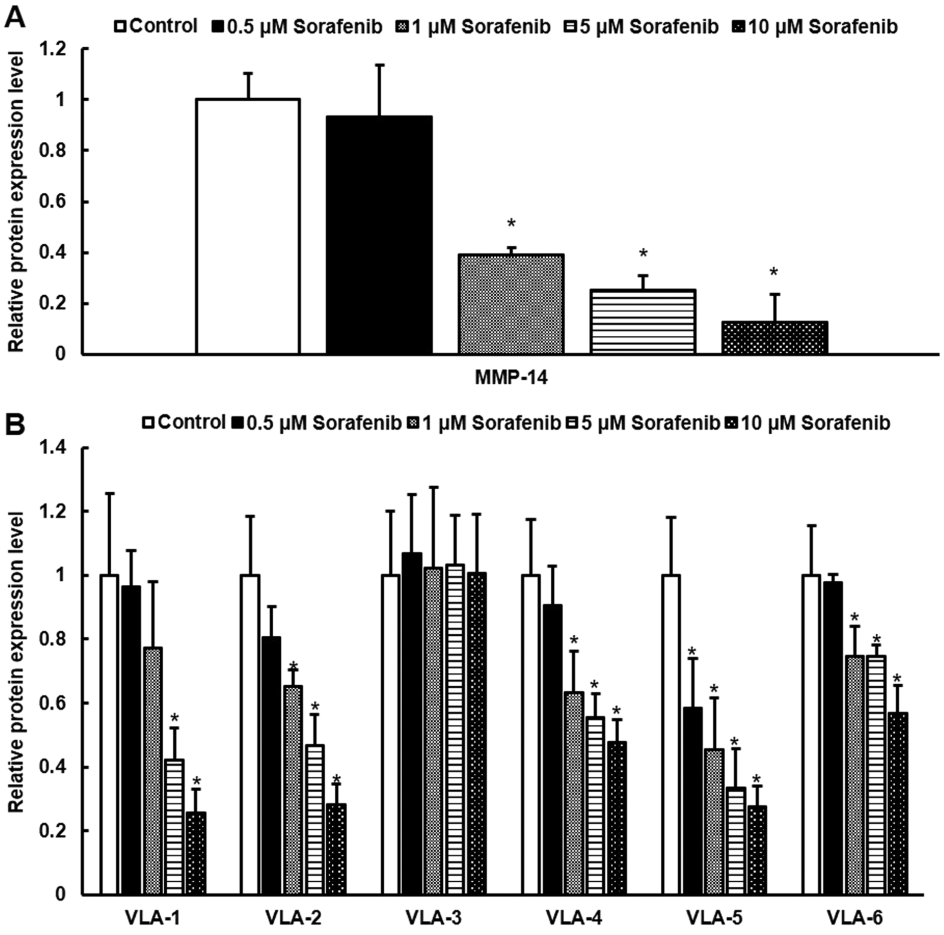

Figure S6. Sorafenib inhibits c-Kit, PDGFR, VEGFR, B-Raf and c-Raf signaling pathways *in vivo*. B16BL6 cells were inoculated into the right hind footpad of mice and treated with sorafenib (10, 30 and 50 mg/kg). After 21 days, the mice were sacrificed, and the right footpads were harvested. The expression levels of p- and total c-Kit, PDGFR, VEGFR, B-Raf and c-Raf were analyzed via western blotting. β-actin were used as internal control. Semi-quantification of the amount of p-c-Kit, p-PDGFR, p-VEGFR, p-B-Raf and p-c-Raf, normalized to the amount of c-Kit, PDGFR, VEGFR, B-Raf and c-Raf. Data are presented as the mean ± SD of three independent experiments. *P<0.05 vs. control. p-, phosphorylated; PDGFR, platelet derived growth factor receptor.