



**Supplementary Figure 5. A, B. Effects of axitinib on VEGFR and PDGFR signaling pathways in endothelial cells.**

**A.** Human umbilical vein endothelial cells (HUVEC) were starved (no growth factors) overnight, followed by stimulation with or without recombinant human VEGF (100 ng/ml) or PDGF (100 ng/ml) for 15 min. Simultaneously, cells were also treated with or without axitinib (10  $\mu$ M) for 15 min. Afterwards, cells were collected and processed for western blotting with antibodies to Vinculin, VEGFR2, p-VEGFR2, p-PDGFR $\beta$ , p-ERK1/2, and p-AKT. **B.** Mouse brain microvascular endothelial cells (MBMEC) were starved (no growth factors) overnight, followed by stimulation with or without recombinant mouse VEGF (100 ng/ml) for 15 min. Simultaneously, cells were also treated with or without axitinib (10  $\mu$ M) for 15 min. Cells were then collected, lysed in RIPA buffer and processed for western blotting with antibodies to VEGFR2, p-ERK1/2, and p-AKT. **C. Relative expression (to vinculin) of p-PDGFR $\beta$ , p-ERK1/2, and p-AKT in MGG123 GBM cells after axitinib and/or G47 $\Delta$ -mIL12 treatment.** Same experiment as Fig. 4D. Data are presented as Mean  $\pm$  SEM from two independent experiments (one is illustrated in Fig. 4D). Statistical comparisons were performed between untreated (no virus or axitinib) vs. treated groups (virus, axitinib, or combination) by applying unpaired two-tailed Student *t* test, \*  $P < 0.05$ , \*\*  $P < 0.01$ , \*\*\*  $P < 0.001$ , \*\*\*\*  $P < 0.0001$ .