Legends of Supplementary Figures

**Figure S1. Cytotoxic effect of the treatment with AA861 on glioma cell death.** Cytotoxic effect on different glioma cell lines (U-87MG, U-138MG, and U-251MG) or primary glioma cells (GBM) treated with increasing doses of AA861 (0–100 μmol/L) at 48 h by CCK-8 cytotoxicity assay.

**Figure S2. Upregulation of DR5 after treatment with MK886 in glioma cells.** A, flow cytometry analysis of TRAIL receptors (DR5) on glioma cell lines (U-87MG, U-138MG, U-251MG, and U-373MG) and primary glioma cells (GBM) in response to 24-hour treatment with increasing doses of MK886 (0–40 μmol/L) in comparison with untreated control. Normal human astrocytes (NHA) were also examined to confirm whether the treatment of MK886 induced DR5 expression in normal cells. Mouse isotype IgG1 antibody served as a negative control.

**Figure S3. DR5-mediated cytotoxic effect of combined treatment with AA861 and TRAIL.** A, Western blotting analysis of DR5 on U-87MG and GBM cells exposed to AA861 dose dependently (0–100 μmol/L) for 24 h. B, cytotoxic effect on glioma cells cotreated with AA861 and TRAIL at 48 h by CCK-8 cytotoxicity assay. Cells were treated with AA861 (100 μmol/L), rTRAIL (10 ng/mL), or a combination with or without DR5/Fc chimeric protein (100 ng/mL).