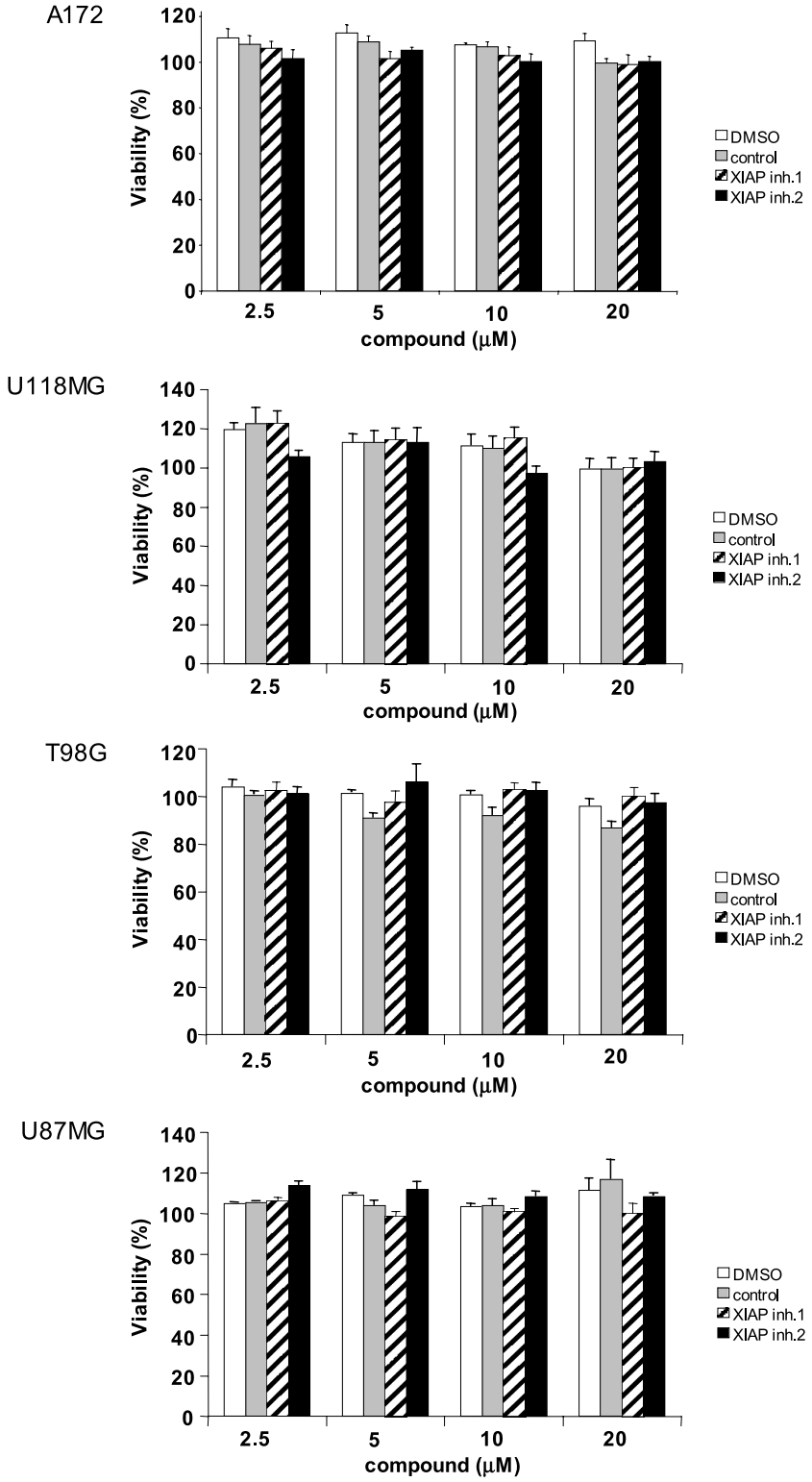

Figure W1. Effect of XIAP inhibitors on glioblastoma cells. Glioblastoma cells were treated for 144 hours with the indicated concentrations of XIAP inhibitors, control compound, or solvent. Cell viability was determined by MTT assay and expressed as a percentage of untreated controls. Means ± SEM of three independent experiments performed in triplicate are shown.

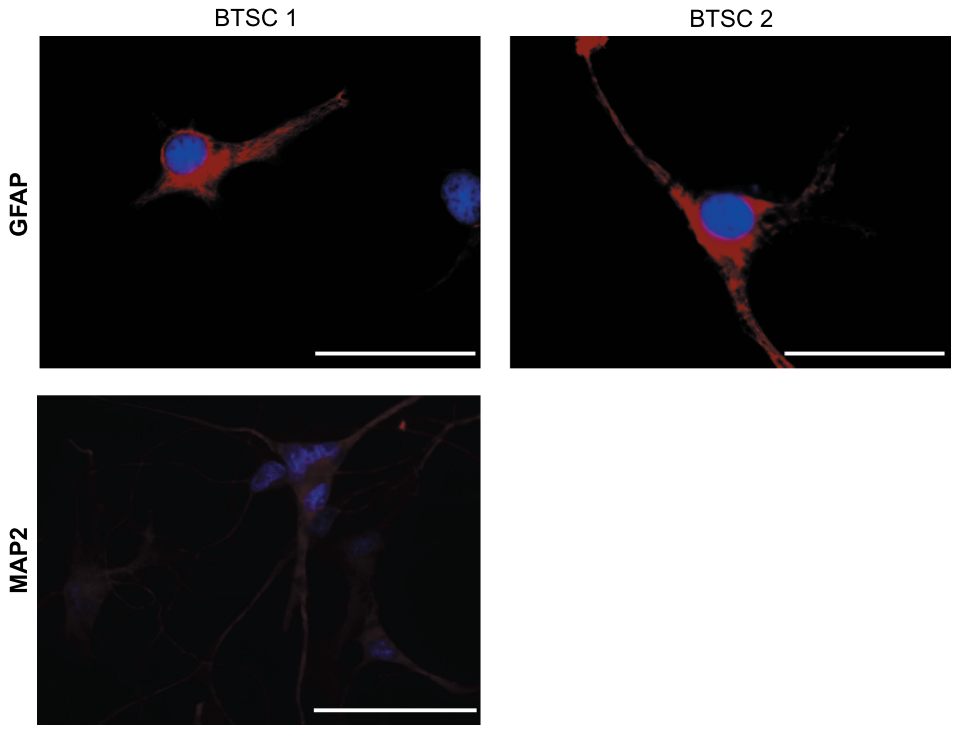

Figure W2. Differentiation of glioblastoma-initiating cells into neuronal and glial lineages. Glioblastoma-initiating cells (BTSC1 and BTSC2) were differentiated for 2 weeks. Neuronal and glial lineages were assessed by immunofluorescent staining of microtubule-associated protein 2 (MAP2) and glial fibrillary acidic protein (GFAP), respectively. For GFAP staining, cells were plated in chamber slides (BD Falcon; BD Biosciences) and were cultivated in DMEM supplemented with 10% heat-inactivated FCS, 10 mM HEPES, and 2 mM L-glutamine for 2 weeks. Cells were fixed with 4% PFA for 10 minutes, blocked with blocking buffer (2% BSA, 0.1% horse serum, and 0.1% Triton X-100) for 30 minutes, and stained with primary rabbit anti–GFAP antibody (1:200; Promega, Madison, WI) overnight at 4°C followed by staining with anti-rabbit IgG–Texas Red secondary antibody and staining with DAPI. For MAP2 staining, cells were plated on coverslips coated with poly-L-lysine and cultured in DMEM supplemented with 10% heat-inactivated FCS, 10 mM HEPES, and 2 mM L-glutamine overnight, and DMEM medium was exchanged with neurobasal medium (Invitrogen, Karlsruhe, Germany) supplemented with B-27 containing vitamin A (Gibco, Karlsruhe, Germany), 100 U/ml penicillin/streptomycin, 10 mM HEPES, and 2 mM L-glutamine for 2 weeks. Cells were fixed with 4% PFA for 10 minutes, blocked with blocking buffer (2% BSA, 0.1% horse serum, and 0.1% Triton X-100) for 30 minutes, and stained with primary rabbit anti-MAP2 antibody (1:50; Cell Signaling) overnight at 4°C followed by staining with goat anti-rabbit IgG–Texas Red secondary antibody (Vector Laboratories) and staining with DAPI. Slides were mounted using Vecta shield mounting medium (Vector Laboratories). Slides were analyzed using an immunofluorescence microscope (Olympus). MAP2 staining could not be performed in BTSC2 cells because these cells did not adhere to coverslips in neurobasal medium, possibly because they lack caspase-8 protein expression, which has been implicated in cell adhesion (Senft J, Helfer B, and Frisch SM (2007). Caspase-8 interacts with the p85 subunit of phosphatidylinositol 3-kinase to regulate cell adhesion and motility. *Cancer Res* **67**, 11505–9).

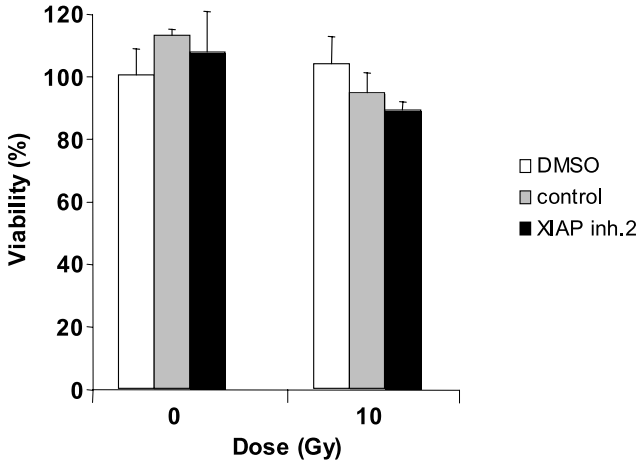

Figure W3. Effect of XIAP inhibitors in combination with γ-irradiation on glial cells. Rat neurons glial cells were treated with 10 Gy of γ-irradiation and/or 10 μM XIAP inhibitor 2, control compound, or DMSO for 144 hours. Cell viability was determined by MTT assay and expressed as a percentage of untreated controls. Means ± SEM of three independent experiments performed in triplicate are shown.