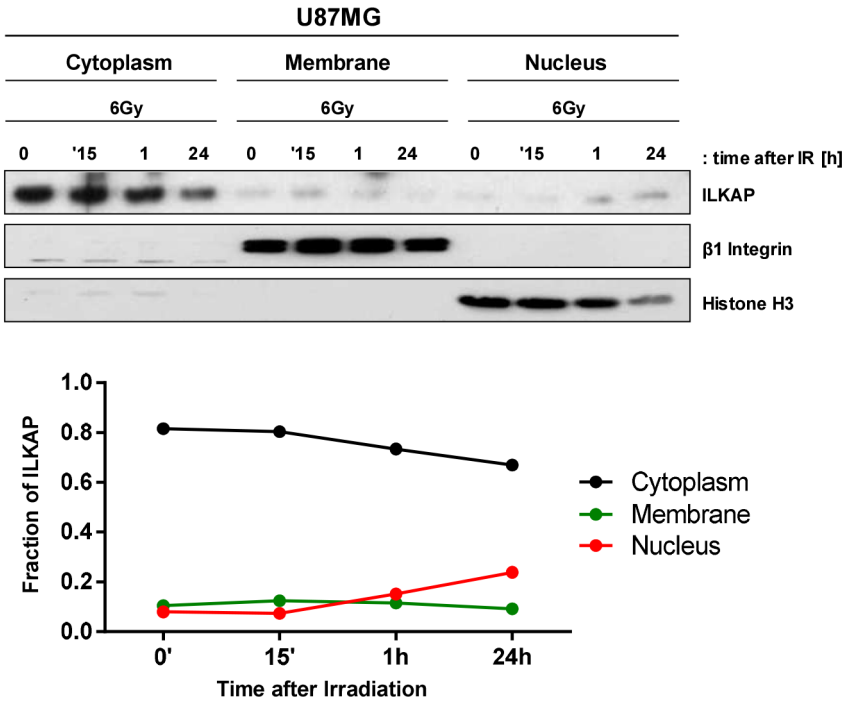
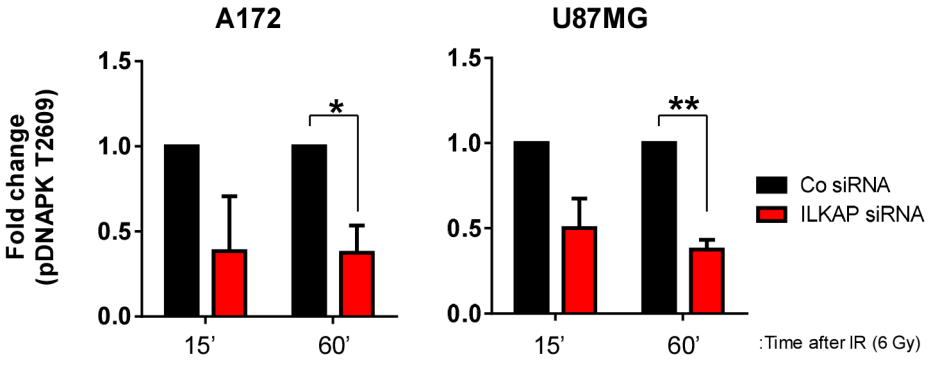
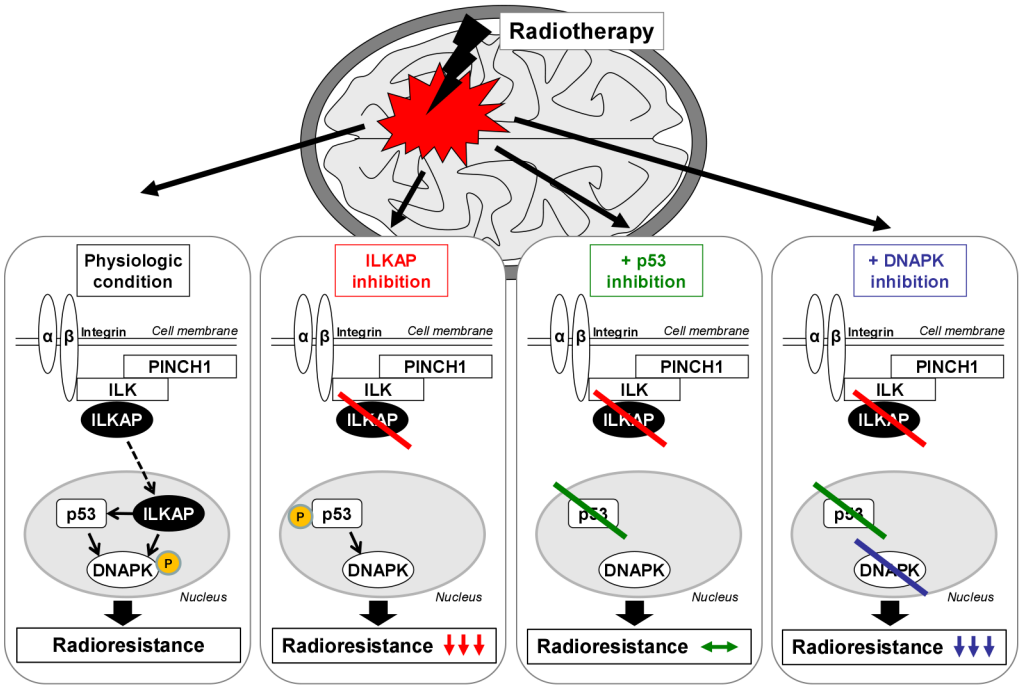
## SUPPLEMENTARY FIGURES AND TABLE




**Supplementary Figure S1: ILKAP translocated to the nucleus after irradiation.** For protein fractionation experiments, cells were plated 24 h prior to irradiation. At the indicated time points after irradiation, cells were lysed as described. Non-irradiated cells were used as controls. Detection of D1 integrin and Histone H3 were performed to confirm efficacy of fractionation. Western blots were evaluated using densitometric analysis.




**Supplementary Figure S2: ILKAP deletion reduced radiation-induced DNAPK phosphorylation.** Densitometric analysis of Western blots in A172 and U87MG glioblastoma cells at indicated time points after irradiation with 6 Gy. ILKAP knockdown was performed with specific siRNA. Non-specific siRNA was used as control. Data are mean ± SD ($n = 2$; t-test; $^{*}P < 0.05$, $^{**}P < 0.01$).




**Supplementary Figure S3: Schematic of ILKAP, p53 and DNAPK regulation of radioresistance in p53-wildtype glioblastoma cells.** Under physiologic conditions integrin-associated signaling confers radioresistance by regulating the DNA repair protein DNAPK. ILKAP knockdown results in radiosensitization, whereas a combined inhibition of ILKAP and p53 has no effect on cellular radiosensitivity. Additional targeting of DNAPK restores the radiosensitizing effect, which can be observed after single ILKAP inhibition.

## Supplementary Table S1: List of Data sets included in Microarray analysis of *PINCH1, ILK* and *ILKAP* mRNA expression using the oncomine database (www.oncomine.org)

| | Data sets/Publications |
|---|---|
| **1.** | Sun L, et al. (2006) Neuronal and glioma-derived stem cell factor induces angiogenesis within the brain. Cancer Cell. 9(4):287–300. |
| **2.** | Bredel M, et al. (2005) Functional network analysis reveals extended gliomagenesis pathway maps and three novel MYC-interacting genes in human gliomas. Cancer Res. 65(19):8679–89. |
| **3.** | Shai R, et al. (2003) Gene expression profiling identifies molecular subtypes of gliomas. Oncogene. 22(31):4918–23. |
| **4.** | Liang Y, et al. (2005) Gene expression profiling reveals molecularly and clinically distinct subtypes of glioblastoma multiforme. Proc Natl Acad Sci U S A. 102(16):5814–9. |
| **5.** | Murat A, et al. (2008) Stem cell-related "self-renewal" signature and high epidermal growth factor receptor expression associated with resistance to concomitant chemoradiotherapy in glioblastoma. J Clin Oncol. 26(18):3015–24. |
| **6.** | Lee J, et al. (2006) Tumor stem cells derived from glioblastomas cultured in bFGF and EGF more closely mirror the phenotype and genotype of primary tumors than do serum-cultured cell lines. Cancer Cell. 9(5):391–403. |
| **7.** | National Cancer Institute, National Institutes of Health, Bethesda, MD 20892 (2013) The Cancer Genome Atlas - Glioblastoma Multiforme Gene Expression Data. *http://tcga-data.nci.nih.gov/tcga/* |