Figure S1. PARP1 cleavage is induced by reversine in glioma cells. Bar graphs represent the mean ± SD of the ratio of cleaved/total PARP1 band intensities (arbitrary units, A.U.) of 3 independent experiments from HOG, T98G and U251MG cells treated with (A) vehicle (DMSO) or graded concentrations of reversine (vehicle, 1.6, 3.2 or 6.4 μM) for 24 h or (B) graded time of exposure (24, 48 and 72 h) to reversine at 1.6 μM. *P<0.05, **P<0.01 and ***P<0.001. PARP1, poly(ADP-ribose) polymerase 1.

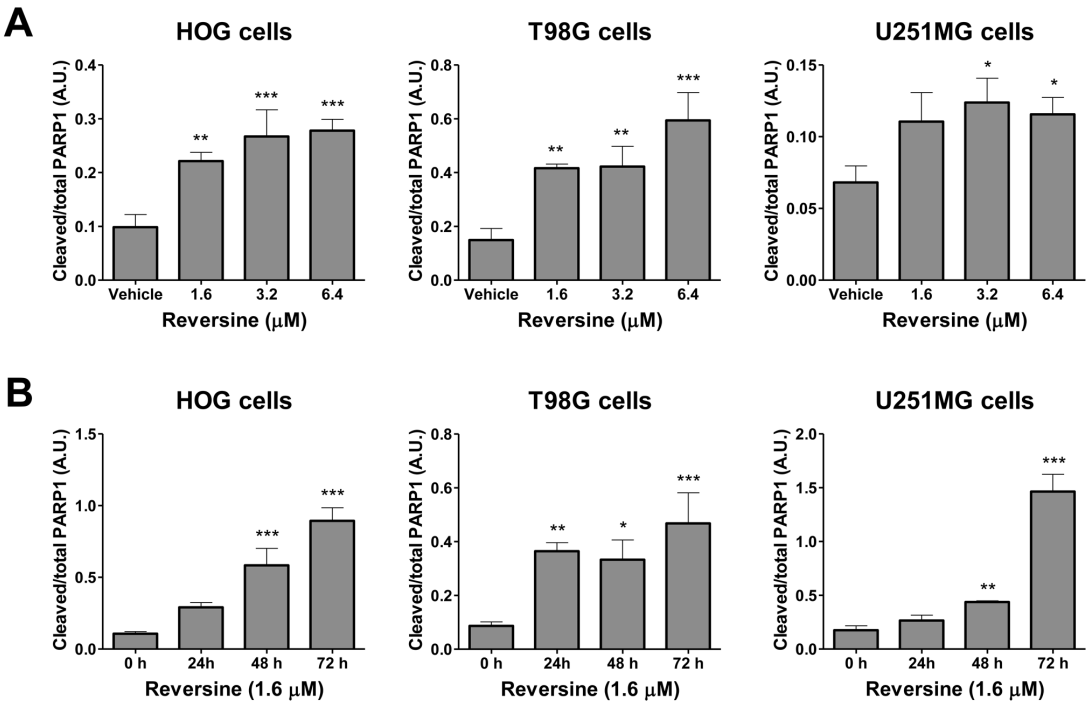

Figure S2. Pan-caspase inhibitor, Z-VAD-FMK, partially attenuated the reversine-induced reduction of cell viability in glioma cells. Cytotoxicity was analyzed using a sulforhodamine B (SRB) assay for HOG, T98G and U251MG cells treated with vehicle (DMSO) or graded concentrations of reversine (0.8, 1.6, 3.2 and 6.4 μM) in combination or not with Z-VAD-FMK (20 μM) for 48 h. Values are expressed as the percentage of viable cells for each condition relative to vehicle-treated cells. Results are shown as mean ± SD of at least 3 independent experiments. *P<0.05, reversine-, and/or -Z-VAD-FMK vs. vehicle-treated cells; $^{\#}$P<0.05 for reversine- or Z-VAD-FMK-treated cells vs. combination treatment at the corresponding doses.

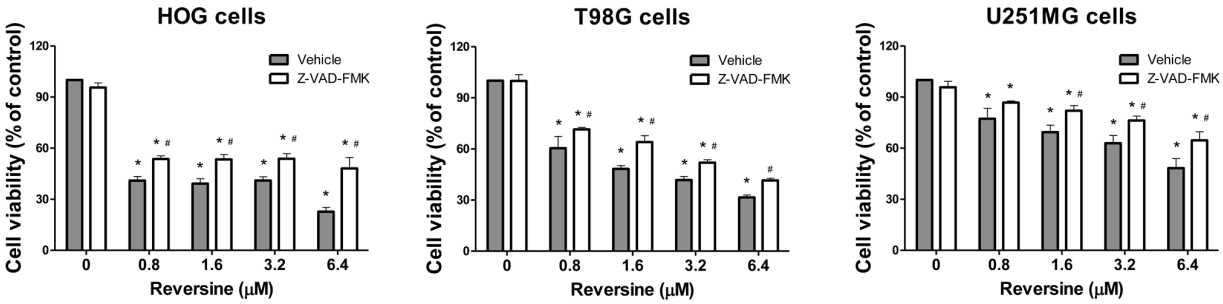

Figure S3. Venetoclax (selective BCL2 inhibitor) or obatoclax (BH3 mimetic) potentiated reversine-induced reduction of cell viability in glioma cells. Cell viability was analyzed using a sulforhodamine B (SRB) assay. (A) Dose-response cytotoxicity for HOG, T98G, and U251MG cells treated with vehicle (DMSO) or graded concentrations of venetoclax (0.1, 0.5, 1, 5, 10 and 50 μM) or obatoclax (0.01, 0.03, 0.1, 0.3, 1 and 3 μM) for 48 h. Values are expressed as the percentage of viable cells for each condition relative to vehicle-treated cells. (B) HOG, T98G, and U251MG cells treated with vehicle or graded concentrations of reversine (0.8, 1.6, 3.2 and 6.4 μM) in combination or not with venetoclax (5 μM) or obatoclax (HOG, 10 nM; T98G, 30 nM; and U251MG, 100 nM) for 48 h. Doses between IC_{30} and IC_{40} for venetoclax and obatoclax were selected for combination studies in each cell line. Values are expressed as the percentage of viable cells for each condition relative to vehicle-treated cells. Results are shown as mean ± SD of at least 3 independent experiments. $^{*}P<0.05$ for reversine-, venetoclax- and/or obatoclax-treated cells vs. vehicle-treated cells; $^{\#}P<0.05$ for reversine-, venetoclax-, or obatoclax-treated cells vs. combination treatment at the correspondingdoses. IC, inhibitory concentration.

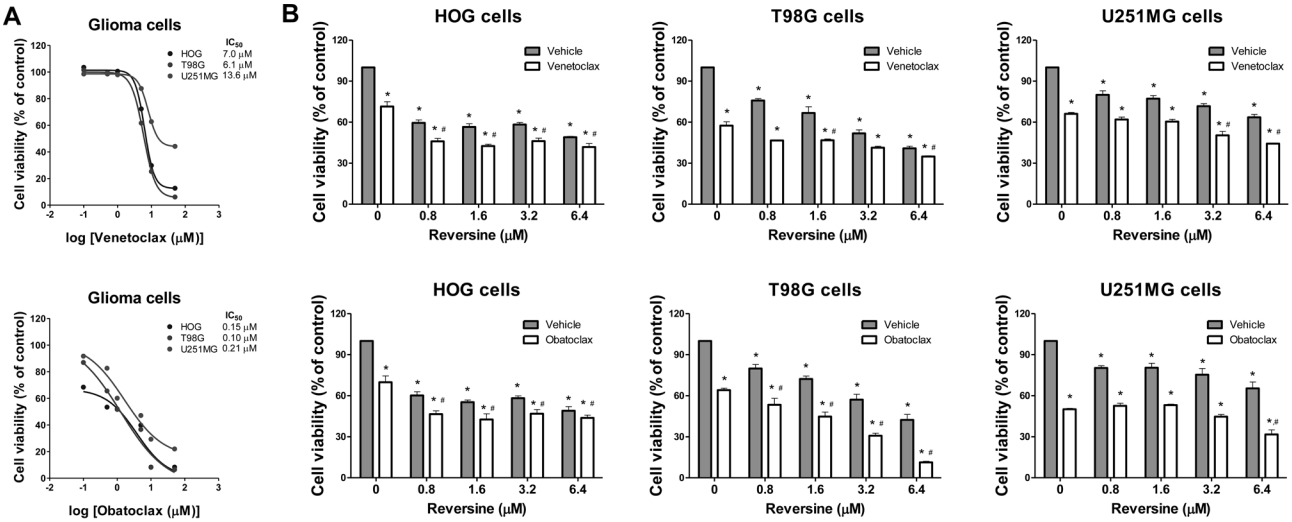

Table SI. RT-qPCR primer sequences and concentrations.

Gene	Sequence (5'-3')	Concentration
AURKA	FW: CCACCTTCGGCATCCTAATA	
	RV: TCCAAGTGGTGCATATTCCA	300 nM
AURKB	FW: CCCTGAGGAGGAAGACAATG	
	RV: GCACCACAGATCCACCTTCT	300 nM
BCL2	FW: ATGTGTGTGGAGAGCGTCAA	
	RV: ACAGTTCCACAAAGGCATCC	300 nM
BCL2L1	FW: CTTGGATGGCCACTTACCTGAA	
	RV: GCTGCTGCATTGTTCCCATA	300 nM
BIRC5	FW: GCCCAGTGTTTCTTCTGCTTCA	
	RV: GCACTTTCTCCGCAGTTTCCTC	300 nM
BNIP3	FW: ATATGGGATTGGTCAAGTCGG	
	RV: CGCTCGTGTTCCTCATGCT	300 nM
BNIP3L	FW: ACACCAGCAGGGACCATAGC	
	RV: TTTCTTCAAAGCCTCGACTTCC	300 nM
BAD	FW: CACCAGCAGGAGCAGCCAAC	
	RV: CGACTCCGGATCTCCACAGC	300 nM
BAX	FW: GAGCTGCAGAGGATGATTGC	
	RV: CAGCTGCCACTCGGAAAA	300 nM
BBC3	FW: GACCTCAACGCACAGTACGAG	
	RV: AGGAGTCCCATGATGAGATTGT	300 nM
PMAIP1	FW: CGCGCAAGAACGCTCAACC	
	RV: CACACTCGACTTCCAGCTCTGCT	300 nM
CDKN1A	FW: TGTCACTGTCTTGTACCCTTGT	
	RV: GCCGGCGTTTGGAGTGGTAG	300 nM
CDKN1B	FW: ACTCTGAGGACACGCATTTGGT	
	RV: TCTGTTCTGTTGGCTCTTTTGTT	300 nM
GADD45A	FW: AAGGATGGATAAGGTGGGG	
	RV: CTGGATCAGGGTGAAGTGG	300 nM
HPRT1	FW: GAACGTCTTGCTCGAGATGTGA	
	RV: TCCAGCAGGTCAGCAAAGAAT	150 nM
ACTB	FW: AGGCCAACCGCGAGAAG	
	RV: ACAGCCTGGATAGCAACGTACA	150 nM

FW, forward; RV, reverse; RT-q, reverse transcription-quantitative; AURKA, aurora kinase A; AURKB, aurora kinase B; BCL2, BCL2 apoptosis regulator; BCL2L1, BCL2 like 1; BIRC5, baculoviral IAP repeat containing 5; BNIP3, BCL2 interacting protein 3; BNIP3L, BCL2 interacting protein 3 like; BAD, BCL2 associated agonist of cell death; BAX, BCL2 associated X, apoptosis regulator; BBC3, BCL2 binding component 3; PMAIP1, phorbol-12-myristate-13-acetate-induced protein 1; CDKN1A, cyclin dependent kinase inhibitor 1A; CDKN1B, cyclin dependent kinase inhibitor 1B; GADD45A, growth arrest and DNA damage inducible gene 45 alpha; HPRT1, hypoxanthine phosphoribosyltransferase 1; ACTB, actin beta.