## Legends to Supplementary Figures:

**FIGURE 1S: Transgenic expression of dnFADD or CrmA prevents FAS-induced apoptosis in human neuroblastoma cells.** SH-EP cells infected with vectors coding for dnFADD, CrmA or with empty control vectors were treated for up to 48 hours with 0.1µg/ml anti-CD95 antibody, clone CH11 (Beckman Coulter, Fullerton, USA). Apoptotic cell death was measured by flow cytometric analyses of PI-stained nuclei.

**FIGURE 2S: Bortezomib represses Bcl-xL mRNA steady state expression.** Total RNA was prepared from SH-EP cells after treatment with 50nM bortezomib for 0, 3, 6, 9 and 16 hours and the level of bcl-xl mRNA was assessed by quantitative RT-PCR. Shown are the mean values of three independent experiments, each performed in triplicate.

**FIGURE 3S: Transgenic BCL2 inhibits bortezomib-induced cell death.** SH-EP cells were infected with a retrovirus coding for human BCL2 (1). Bulk-selected SH-EP-BCL2 cells and mock-infected controls (SH-EP-ctr) were treated with 50nM bortezomib for the times indicated and subjected to PI-FACS analyses. Bars represent mean of three independent experiments. Similar to Bcl-xL, transgenic BCL2 inhibits bortezomib-induced apoptosis in neuroblastoma cells.

**FIGURE 4S: Transgenic expression of MCL1L protects against FAS, TRAIL and doxorubicin-induced apoptosis.** SH-EP-ctr and SH-EP-Mcl1L cells were treated with (A) 0.1µg/ml CH11 antibody, (B) 5ng/ml recombinant human TRAIL (Alexis, CA, USA) or (C) the chemotherapeutic agent doxorubicin (0.25µg/ml) for the times indicated and subjected to PI-FACS analyses, respectively. Mean values represent three independent experiments.

**FIGURE 5S: Stabilisation of p53 is not essential for induction of Noxa and Puma by bortezomib.** Since Noxa and Puma are both transcriptional targets of p53 which was shown to be stabilized by bortezomib we assessed the effect of proteasome-inhibition by bortezomib on steady state protein levels of p53 and its target BAX. For this purpose SH-EP and STA-NB15 cells were cultured in the presence of 50nM bortezomib for 0, 4 and 8 hours and subjected to immunoblot analyses using antibodies directed against p53, BAX, GAPDH and α-Tubulin. p53 was stabilized in SH-EP cells whereas in STA-NB15 cells high levels of p53 were already detected in untreated cells suggesting that p53 is mutated in this cell line. However, bortezomib-treatment also let to some further accumulation of, presumably non-functional p53 in STA-NB15 cells. The pro-apoptotic p53 target BAX showed transitory elevation at four hours of bortezomib-treatment in SH-EP cells but remained unaffected in STA-NB15 cells, consistent with the assumption that p53 is mutated in STA-NB15 cells. The

combined data suggest that induction of Puma and Noxa by bortezomib does not depend on functional p53 in neuronal cells.

**Supplementary References:**

1. Obexer, P., Geiger, K., Ambros, P. F., Meister, B., and Ausserlechner, M. J. (2007) *Cell Death Differ* 14, 534-547

## *Supplemental Figures:*

**Fig. 1S:**

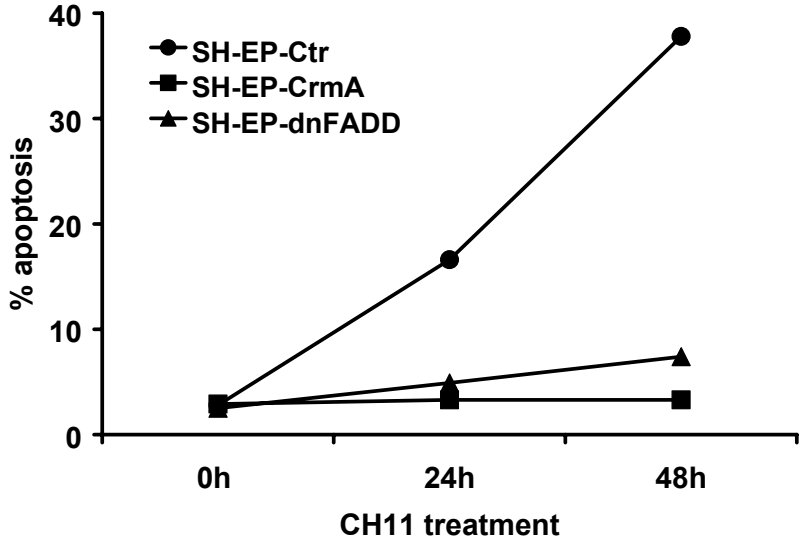



**Fig. 2S:**

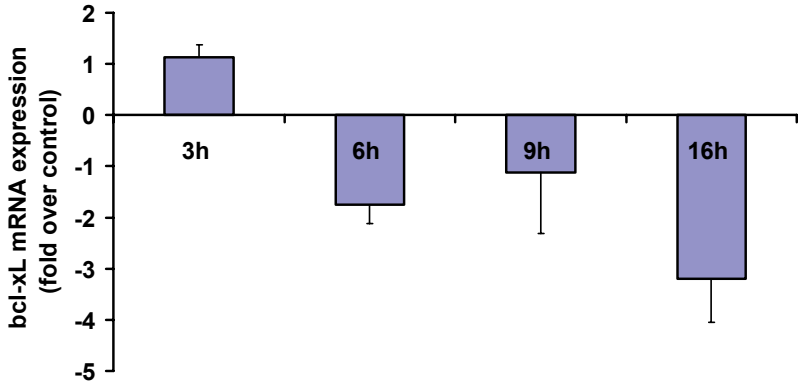
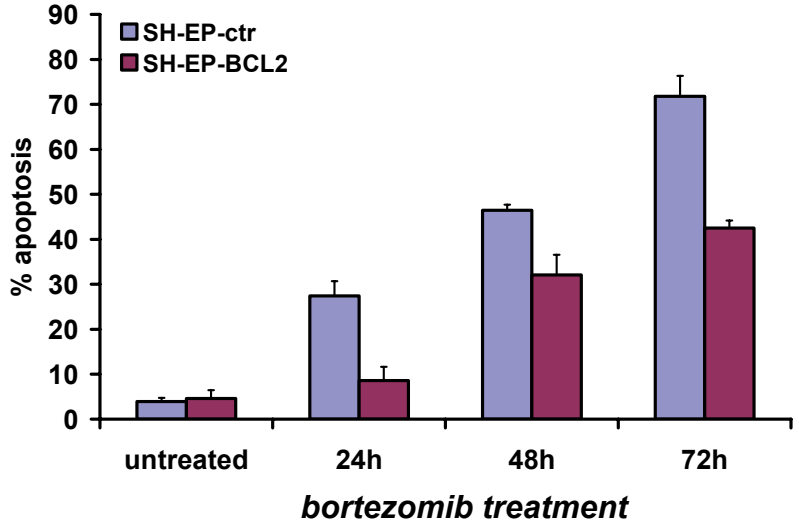
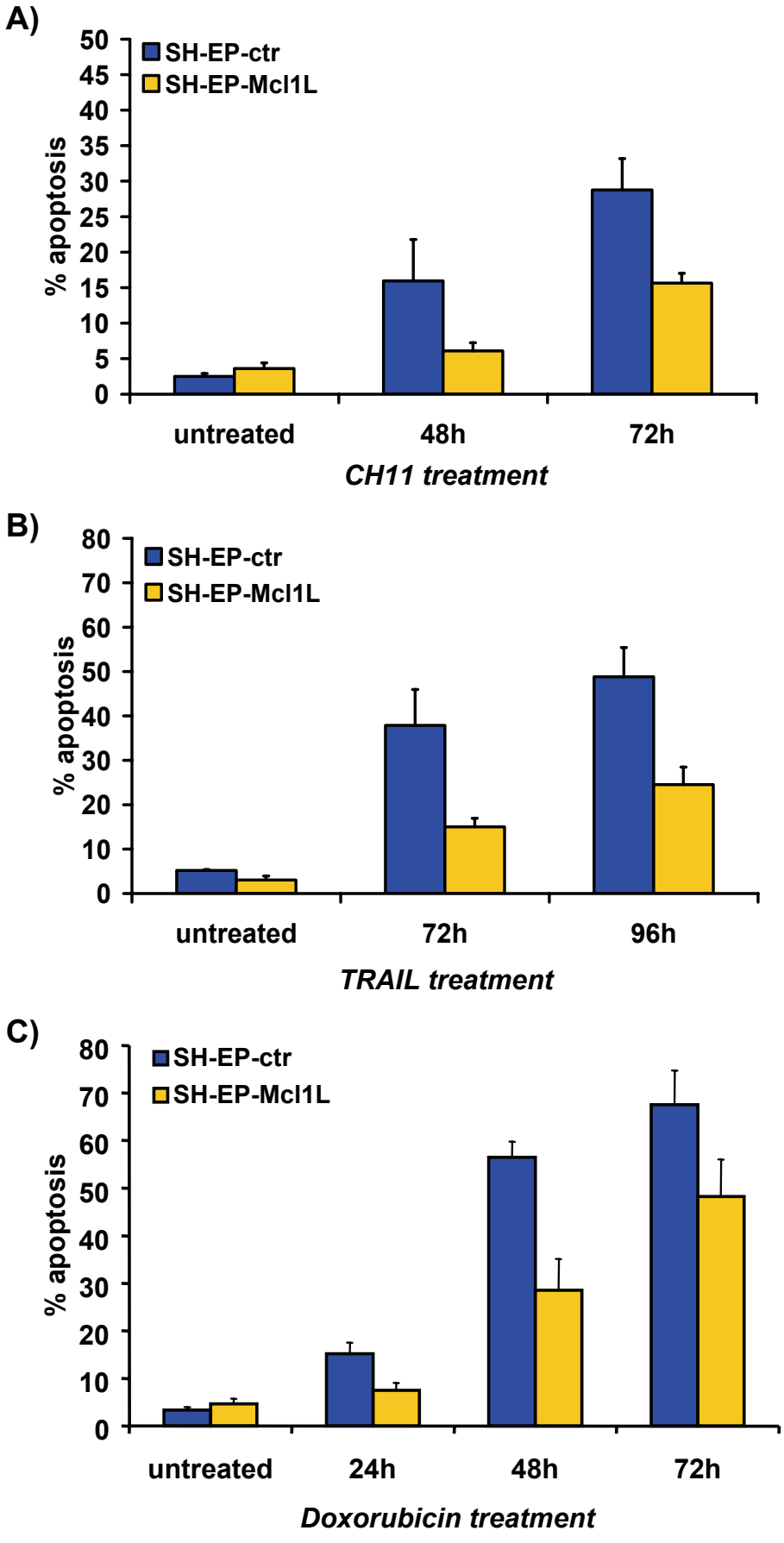



**Fig. 3S:**



## *Supplemental Figures:*

### Fig. 4S:



## *Supplemental Figures:*

### Fig. 5S: