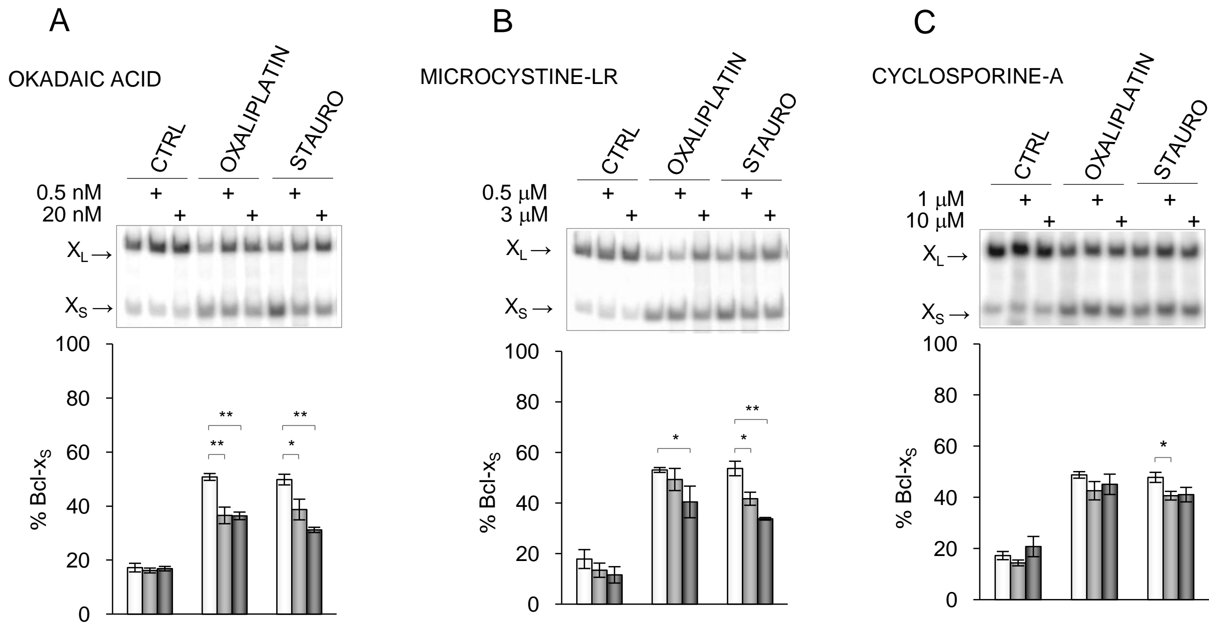
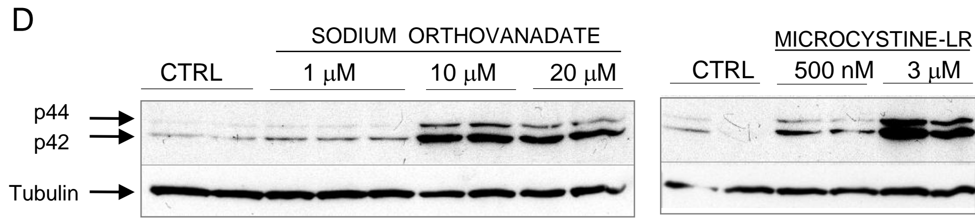
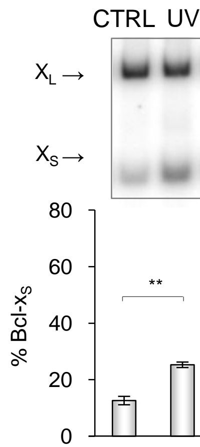
SUPPLEMENTARY FIGURE LEGENDS

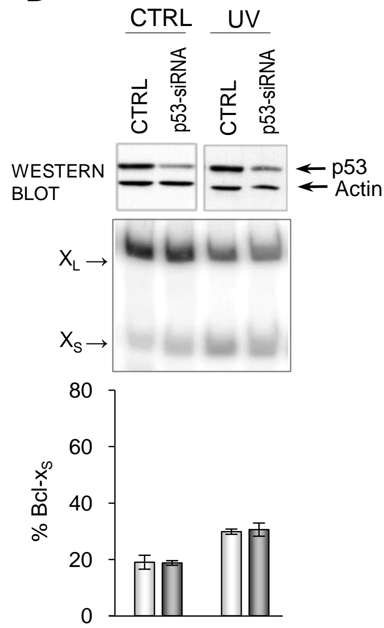
Fig. S1. Phosphatase inhibitors and *Bcl-x* splicing. The phosphatase inhibitors okadaic acid *A.* microcystine-LR *B.* and cyclosporine-A *C.* were used to pre-treat 293 cells for 1 h prior to the application of oxaliplatin and staurosporine. Microcystine-LR and okadaic acid are inhibitors of serine/threonine phosphatases; PP1 and PP2A are equal targets of microcystine-LR, while okadaic acid inhibits PP2A with higher selectivity than PP1 (50-52). Higher concentrations of either inhibitors can also affect the activity of PP2B. Cyclosporine-A specifically inhibits PP2B (53). Orthovanadate is a broad-spectrum inhibitor of protein tyrosine phosphatases (54,55). Total RNA was extracted 24 h later and RT-PCR analysis was carried out. *D.* Confirmation of the activity of orthovanadate and microcystine-LR. 293 cells were treated with inhibitors of protein phosphatases at different concentrations for 24 h. Proteins were extracted, fractionated on SDS-acrylamide gels and transferred onto nitrocellulose. Western analysis was performed with a monoclonal antibody (M8159, Sigma Aldrich) that specifically recognizes the diphosphorylated form of MAP kinases (ERK-1/p42 and ERK-2/p44) but not the non-phosphorylated or monophosphorylated forms. Because the MAP-kinase epitope includes phosphorylated threonine and tyrosine residues, the antibody can also detect the activity of threonine-specific phosphatases. The diphosphorylated forms of MAP-kinase are hardly detectable in untreated cells. The phosphorylated versions are significantly augmented by increasing the concentrations of vanadate and microcystine-LR due to the inhibition of phosphatases that target the dephosphorylation of tyrosine and threonine residues.

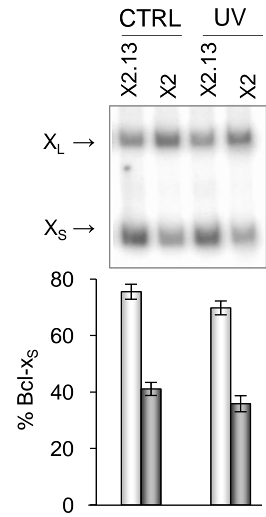
Fig. S2. UV shifts the alternative splicing of *Bcl-x* in 293 cells independently of p53 and of the SB1 element. *A*. Twenty-four hours after treatment of 293 cells with 20 J/m^2 of UV at 254 nm, total RNA was extracted and analyzed to determine the relative abundance of the endogenous Bcl-x_S splice form. *B.* The same analysis was conducted following the siRNA-mediated depletion of p53 in 293 cells. *C.* Minigenes HIV-X2 and HIV-X2.13 were transfected in 293 cells. Four hours post-transfection, cells were irradiated with UV and RNA was extracted 20 h later. The relative abundance of the exogenously-derived *Bcl-x* splice variants was determined by RT-PCR.


A
OKADAIC ACID
CTRL
OXALIPLATIN
STAURO
0.5 nM
20 nM
X_L
X_S
% Bcl-x_S
B
MICROCYSTINE-LR
0.5 μM
3 μM
C
CYCLOSPORINE-A
1 μM
10 μM


D
SODIUM ORTHOVANADATE
CTRL
1 μM
10 μM
20 μM
MICROCYSTINE-LR
500 nM
3 μM
p44
p42
Tubulin







SUPPLEMENTARY FIGURE 2