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CORRIGENDUM

Adenoviral expression of XIAP antisense RNA induces apoptosis in glioma cells and suppresses the growth of xenografts in nude mice

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Correction to: *Gene Therapy* (2007) **14**, 147–161. doi:10.1038/sj.gt.3302845

Since the above publication, the authors have noticed that there are some errors in the western blots of Figures 1,4,6 and 7, which were unfortunately introduced when assembling the figures. The revised figures with the correct blots circled are shown below. The authors apologize for these errors, which did not affect the scientific content of the article.

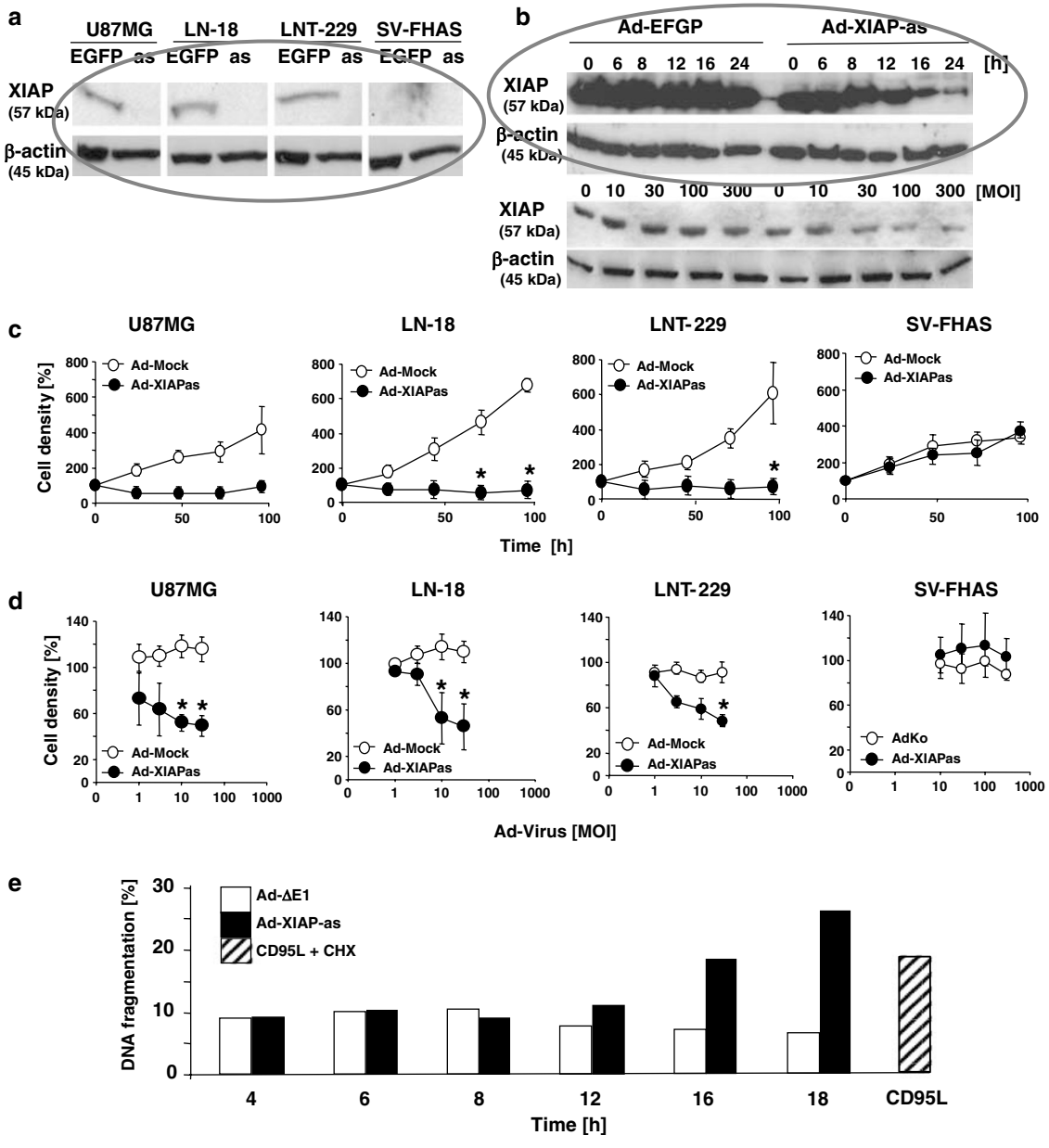

Figure 1 For caption see next page

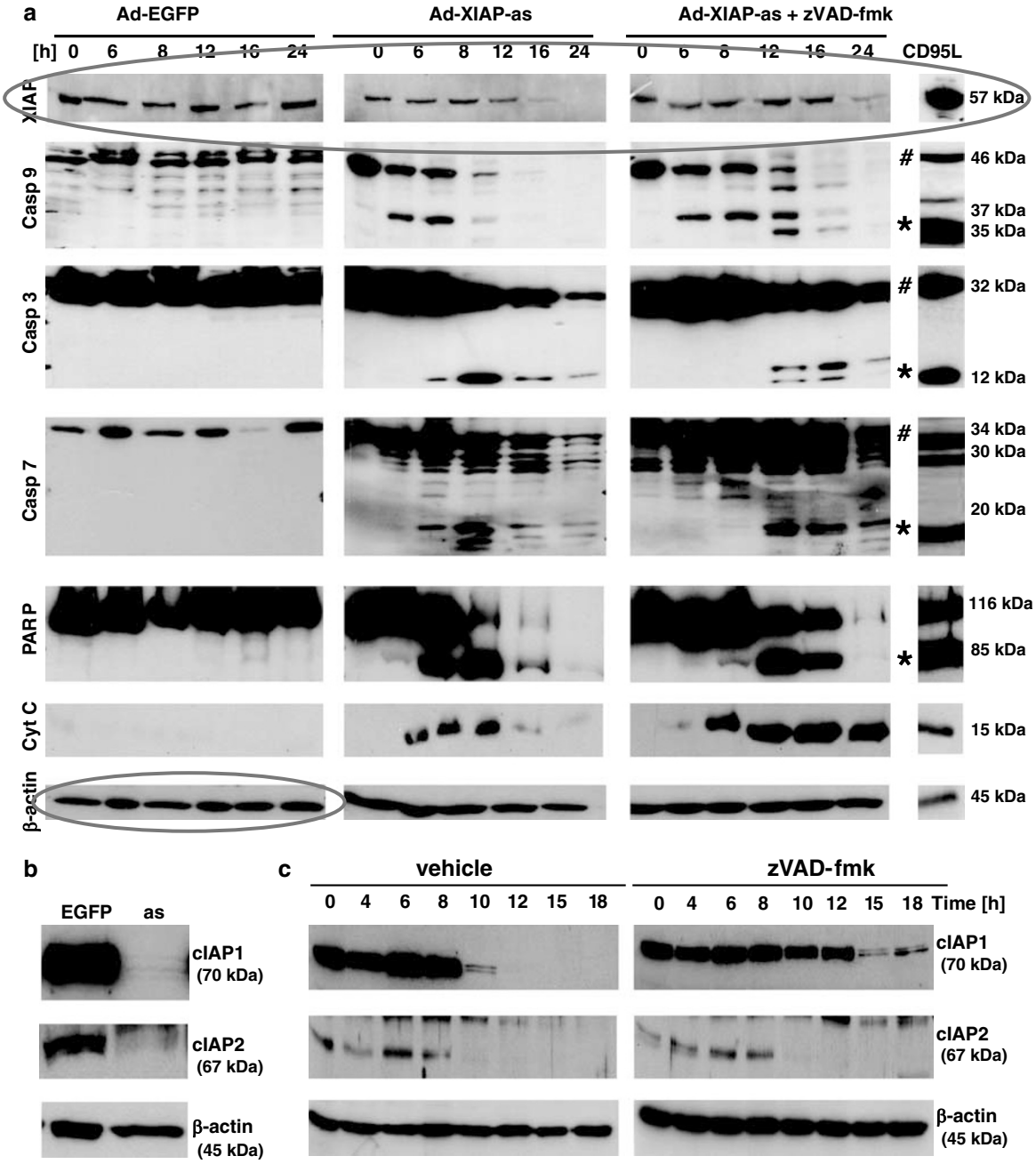

Figure 4 Caspase processing during Ad-XIAP-as-induced apoptosis. (**a**) LNT-229 cells were infected with 100 MOI Ad-EGFP or Ad-XIAP-as in the absence or presence of zVAD-fmk (100 μM) for different time periods. As a control for caspase cleavage, the cells were treated with 100 U/ml CD95L+10 μg/ml CHX for 4 h. Caspase processing and activation were visualized by the detection of p37 and p35/caspase-9, p12/caspase-3, p20/caspase-7 (*) and p85/PARP; #: procaspase. Mitochondrial Cyt *c* release into the cytoplasm was assessed in parallel. β-Actin served as a reference. (**b**) The cells were infected with 100 MOI Ad-EGFP or Ad-XIAP-as for 16 h and assessed for the levels of cIAP1, cIAP2 or β-actin. (**c**) The cells were infected with 100 MOI Ad-XIAP-as in the absence or presence of zVAD-fmk (100 μM). cIAP-1, cIAP-2 and β-actin levels were assessed by immunoblot.

Figure 1 Ad-XIAP-as depletes endogenous XIAP levels and induces cell death in human glioma cells, but not in untransformed astrocytes. (**a**) The cells were infected with Ad-EGFP (EGFP) or Ad-XIAP-as (as) (100 MOI) for 24 h. (**b**) LNT-229 cells were infected at 100 MOI for different periods of time (upper panel) or for 12 h at increasing MOI (lower panel). XIAP or β-actin protein levels were assessed by immunoblot. (**c**, **d**) The cells were infected in triplicates at 100 MOI of control virus (Ad-Mock) or Ad-XIAP-as for up to 20 h (**c**) or at increasing MOI for 24 h (glioma cells) or 48 h (SV-FHAS) (**d**). Cell density was assessed by crystal violet staining (mean and s.e.m., $n = 3$). (**e**) LNT-229 cells were infected in triplicates at 100 MOI for 16 h or treated with CD95L (100 U/ml) plus CHX (10 μg/ml) for 6 h as a control. DNA fragmentation was quantified by differential fluorimetry of fragmented and intact DNA (one representative experiment).

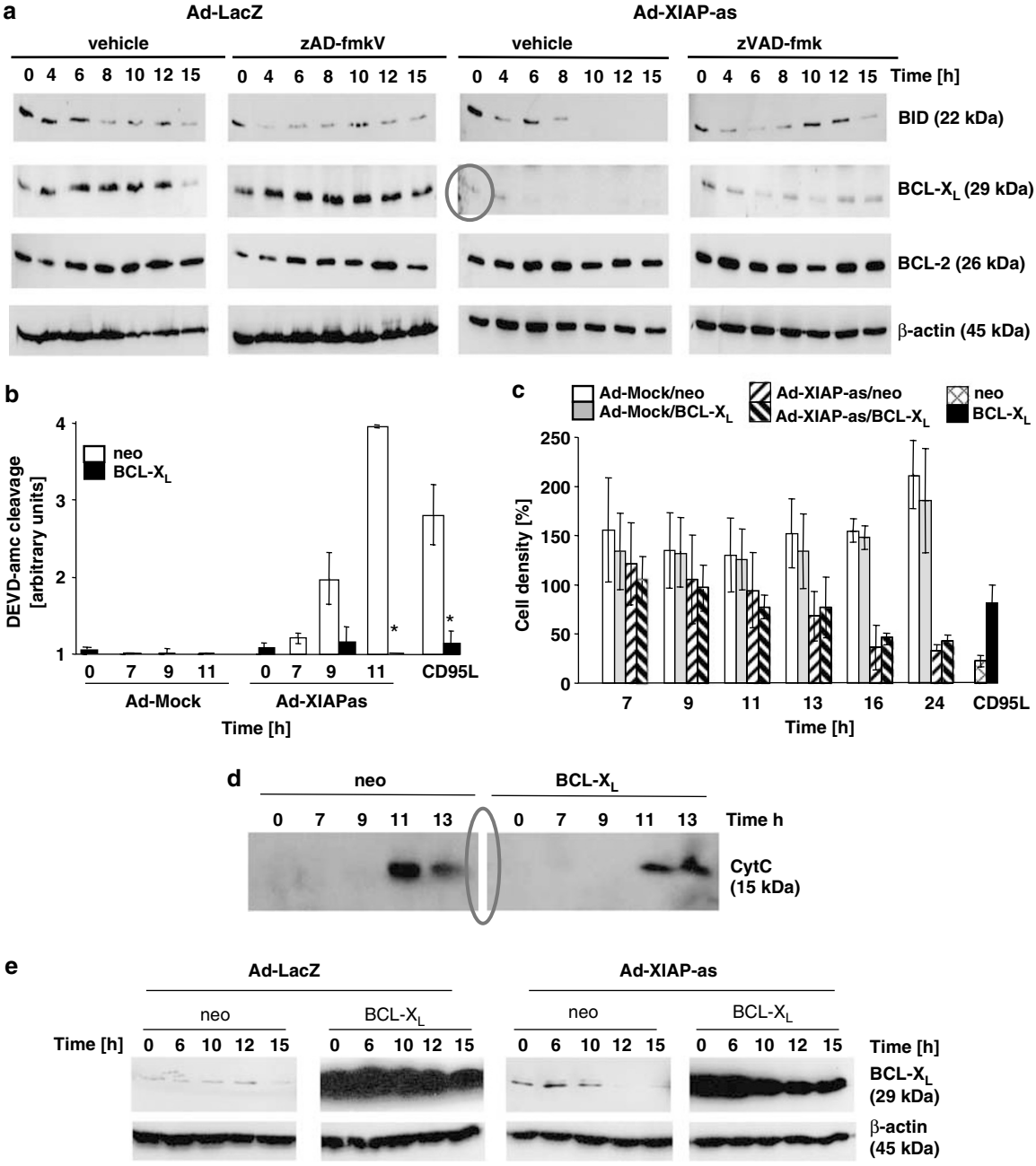

Figure 6 Ad-XIAP-as expression overcomes Bcl-X_L-mediated protection from apoptosis. (**a**) LNT-229 cells were infected with 100 MOI Ad-LacZ or Ad-XIAP-as for increasing time periods in the absence (vehicle) or presence of zVAD-fmk (100 μM). Protein levels were assessed by immunoblot. (**b**) Neo control or BCL-X_L-transfected LNT-229-cells were infected with 100 MOI control virus (Ad-Mock) or Ad-XIAP-as and monitored for DEVD-amc cleaving caspase activity at different time points after infection (mean and s.e.m., $n=2$). (**c**) LNT-229 neo or BCL-X_L cells were treated as in (**b**). Cell density was assessed by crystal violet staining (mean and s.e.m., $n=3$). (**d**) LNT-229 neo or BCL-X_L cells were infected with 100 MOI Ad-XIAP-as and assessed for cytoplasmic Cyt *c* levels. (**e**) The cells were infected with 100 MOI Ad-LacZ or Ad-XIAP-as. BCL-X_L or β-actin protein levels were assessed by immunoblot.

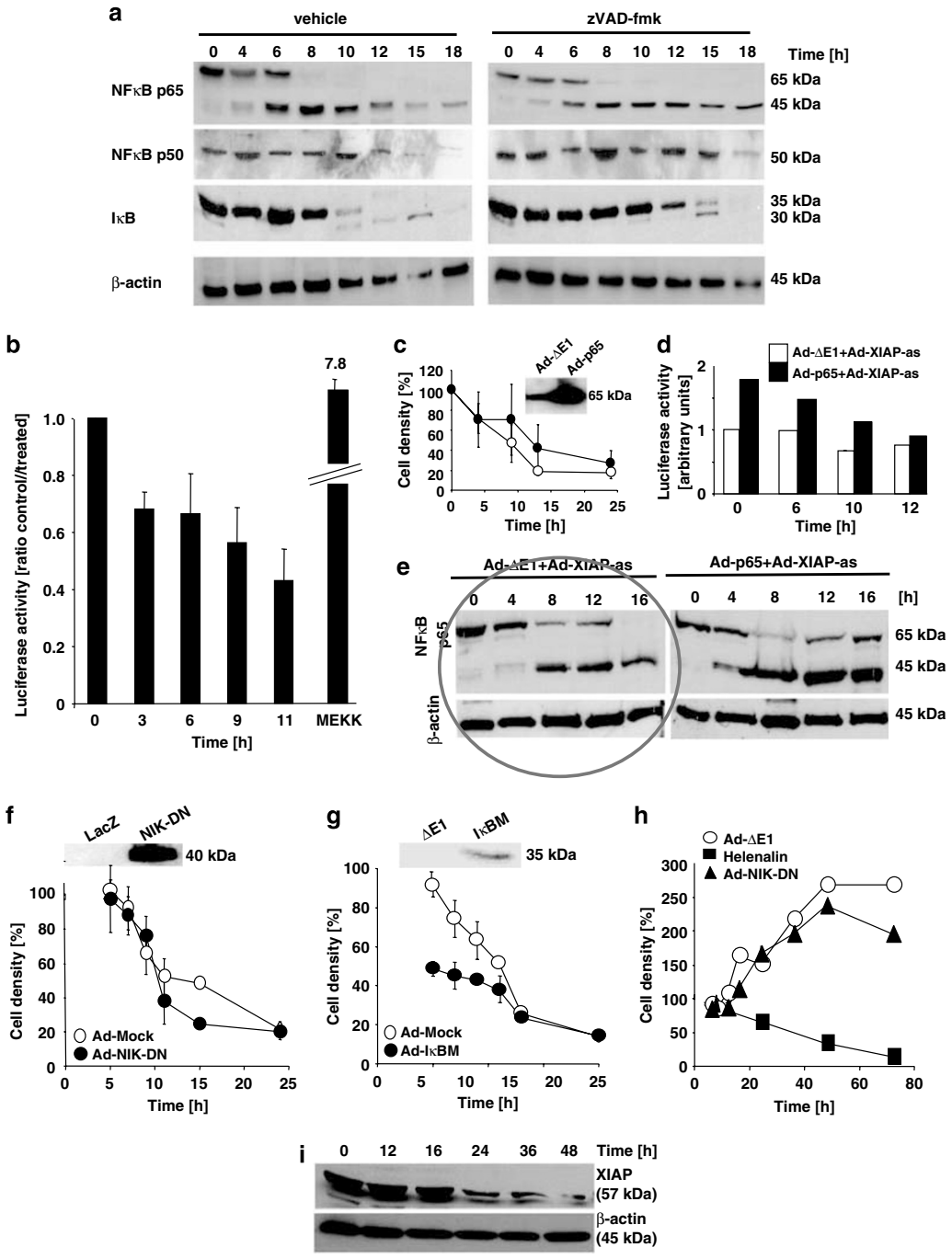

Figure 7 Ad-XIAP-as promotes the loss of NF-κB activity. (**a**) LNT-229 cells were infected with 100 MOI Ad-XIAP-as in the absence or presence of zVAD-fmk (100 μM). The levels of NF-κB p65, NF-κB p50 and IκB were assessed by immunoblot. (**b**) LNT-229 cells were transiently transfected with pNF-κB-Luc and pRL-CMV for 48 h and then infected with 100 MOI Ad-XIAP-as. NF-κB-induced luciferase activity was normalized to constitutively active renilla luciferase activity and surviving cells. As a positive control, the cells were co-transfected with pNF-κB-Luc and either empty vector (pcDNA3, set to 1) or pMEKK (mean and s.e.m., $n=3$). (**c**) LNT-229 cells were infected with 100 MOI Ad-ΔE1 (open circles) or Ad-p65 (closed circles) for 24 h and reinfected with 100 MOI Ad-XIAP-as for increasing time periods. Cell density was assessed using crystal violet staining (mean and s.e.m., $n=2$). The inset shows NF-κB p65 expression 24 h after infection. (**d**) LNT-229 cells were transiently transfected with pNF-κB-Luc and pRL-CMV for 24 h, infected with 100 MOI Ad-ΔE1 or Ad-p65 for 16 h and then reinfected with 100 MOI Ad-XIAP-as for increasing time periods. NF-κB-induced luciferase activity was measured as in (**b**) (representative experiment). (**e**) LNT-229 cells were infected with 100 MOI Ad-ΔE1 or Ad-p65.After 24 h, the cells are reinfected with 100 MOI Ad-XIAPas for increasing time periods. NF-κB p65 expression (upper panel) was assessed by immunoblot. β-Actin expression served as a reference (lower panel). (**f**) The cells were infected with 100 MOI control virus (Ad-Mock) or Ad-NIK-DN, reinfected with100 MOI Ad-XIAP-as 16 h later and monitored for cell density (mean and s.e.m., $n=3$). The inset shows HA-NIK-DN expression 24 h after infection as detected by anti-HA antibody. (**g**) The cells were infected with 100 MOI of control virus (Ad-Mock) or Ad-IκBM, reinfected with 100 MOI Ad-XIAP-as 16 h later and monitored for cell density (mean and s.e.m., $n=3$). The inset shows IκBM expression 24 h after infection. (**h**) The cells were infected with 100 MOI of Ad-ΔE1 or Ad-NIK-DN or were treated with helenalin (5 μM) and monitored for survival (representative experiment). (**i**) LNT-229 cells were treated with helenalin (5 μM) for increasing time periods. XIAP levels were assessed by immunoblot.