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Erratum

# Erratum to "The death associated protein (DAP) kinase homologue Dlk/ZIP kinase induces p19ARF- and p53-independent apoptosis" [European Journal of Cancer, 39 (2003) 249–256]☆

D. Kögel[a,*], C. Reimertz[a], H. Düßmann[a], P. Mech[a], K.H. Scheidtmann[b], J.H.M. Prehn[a,c]

[a] *Interdisciplinary Center for Clinical Research (IZKF), Research Group "Apoptosis and Cell Death", Westphalian Wilhelms-University, D-48149 Münster, Germany*

[b] *Institute of Genetics, University of Bonn, D-53117 Bonn, Germany*

[c] *Department of Pharmacology and Toxicology, Faculty of Medicine, Westphalian Wilhelms-University, D-48149 Münster, Germany*

The Publisher regrets that errors occurred in Figs. 2 and 3 of the published article mentioned above. These figures are correctly printed overleaf.

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* Corresponding author. Tel.: +49-251-83-52941; fax: +49-251-85-52250.

*E-mail address:* koegel@uni-muenster.de (D. Kögel).

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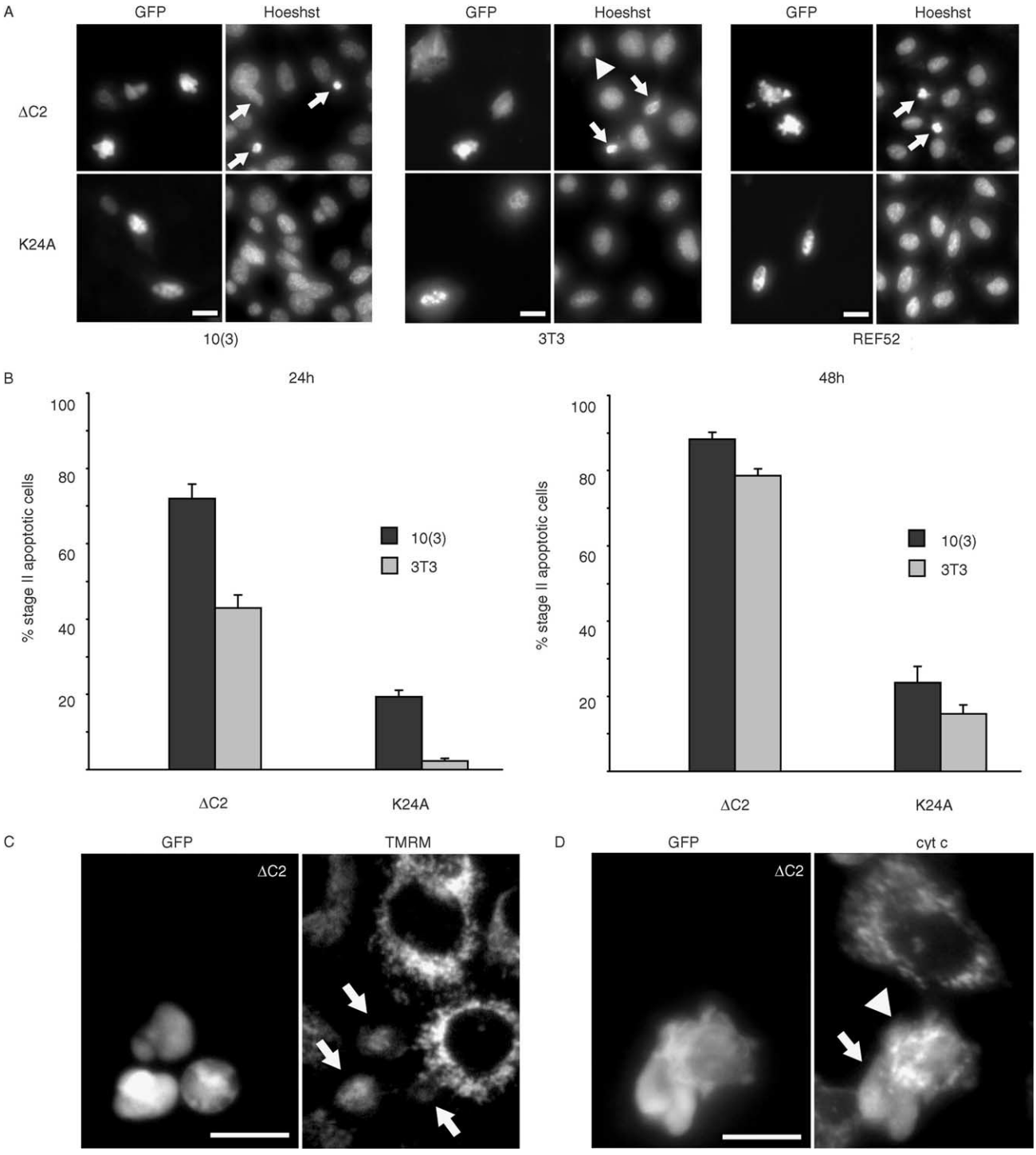


Fig. 2. Apoptosis induced by Dlk/ZIP kinase does not require the p19ARF/p53 signalling pathway. Mouse 10(3) cells, mouse NIH 3T3 cells and rat REF52 cells were transiently transfected with plasmids encoding GFP fusion proteins of Dlk/ZIP kinase deletion mutant ΔC2 and kinase-negative mutant K42A. Cells were stained with Hoechst 24 hours after transfection. ΔC2-induced cell death is accompanied by membrane blebbing and chromatin condensation (indicated by arrows) in all three cell lines (a). Arrow head denotes a ΔC2-expressing NIH 3T3 cell without the apoptotic morphology. Scale bar = 25 μm. Quantitative analysis of apoptosis (b). 24 and 48 h after transfection of 10(3) cells and NIH 3T3 with the indicated plasmids, nuclei were stained with Hoechst and the percentage of GFP-positive cells revealing apoptotic morphology was counted as described in Materials and methods. Data are given as means ± Standard Error of the Mean (S.E.M.) Dlk/ZIP kinase induces the mitochondrial apoptosis pathway in p53-deficient cells (c and d). 10(3) cells were transiently transfected with ΔC2. 24 h after transfection, live cells were stained with TMRM (**C**). After equilibration, images of live cells were acquired. Arrows point to cells with depolarised mitochondria. For analysis of cytochrome c release, 10(3) cells were fixed 24 hours after transfection with ΔC2. Subcellular distribution of cytochrome c was investigated by immunofluorescence analysis with a monoclonal anti-cytochrome c antibody (d). The arrow points to an apoptotic cell having completely released its cytochrome c. The arrowhead indicates a cell undergoing apoptotic membrane blebbing preceding cytochrome c release. Scale bar = 25 μm.

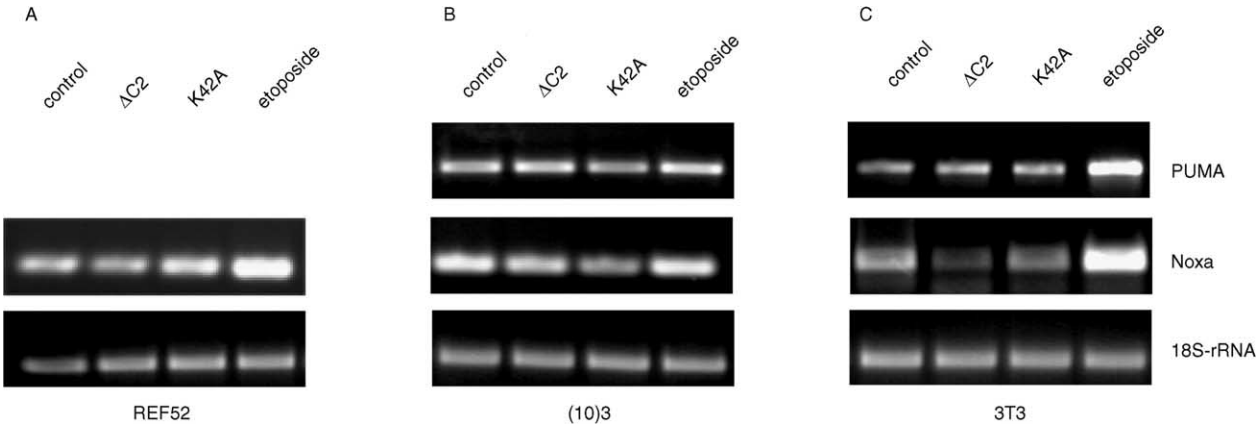


Fig. 3. Analysis of p53 target gene expression by reverse transcriptase-polymerase chain reaction (RT-PCR) analysis. REF52 cells (a), (10)3 cells (b) or NIH 3T3 cells (c) were either left untreated (control) or transfected with the indicated GFP-fusion plasmids. 24 h after transfection, the cells were microscopically analysed for transfection efficiency which was in the range between 20 and 25%. As a positive control for p53 acvtivation, cells were treated with 100 μM etoposide for 24 h. Total RNA was prepared from all cultures and subjected to RT-PCR analysis as described in Materials and methods. Expression analysis of *Noxa* was performed in all three cell lines. Since the rat *PUMA* cDNA sequence is not currently available, expression analysis of *PUMA* was restricted to mouse cell lines (10)3 and NIH3T3. *18S*-rRNA served as an internal control.