

IKK α controls canonical TGF β -SMAD signaling to regulate genes expressing SNAIL and SLUG during EMT in Panc1 cells

Martina Brandl, Barbara Seidler, Ferdinand Haller, Jerzy Adamski, Roland M. Schmid, Dieter Saur and Günter Schneider

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There was an error published in *J. Cell Sci.* **123**, 4231-4239.

In Fig. 4A, the cRel siRNA western blot panel was inadvertently constructed using the wrong images and all three western blots showed incorrect loading controls. The correct images and loading controls are shown in the figure below. The mistake in the figure did not affect the conclusions of the paper.

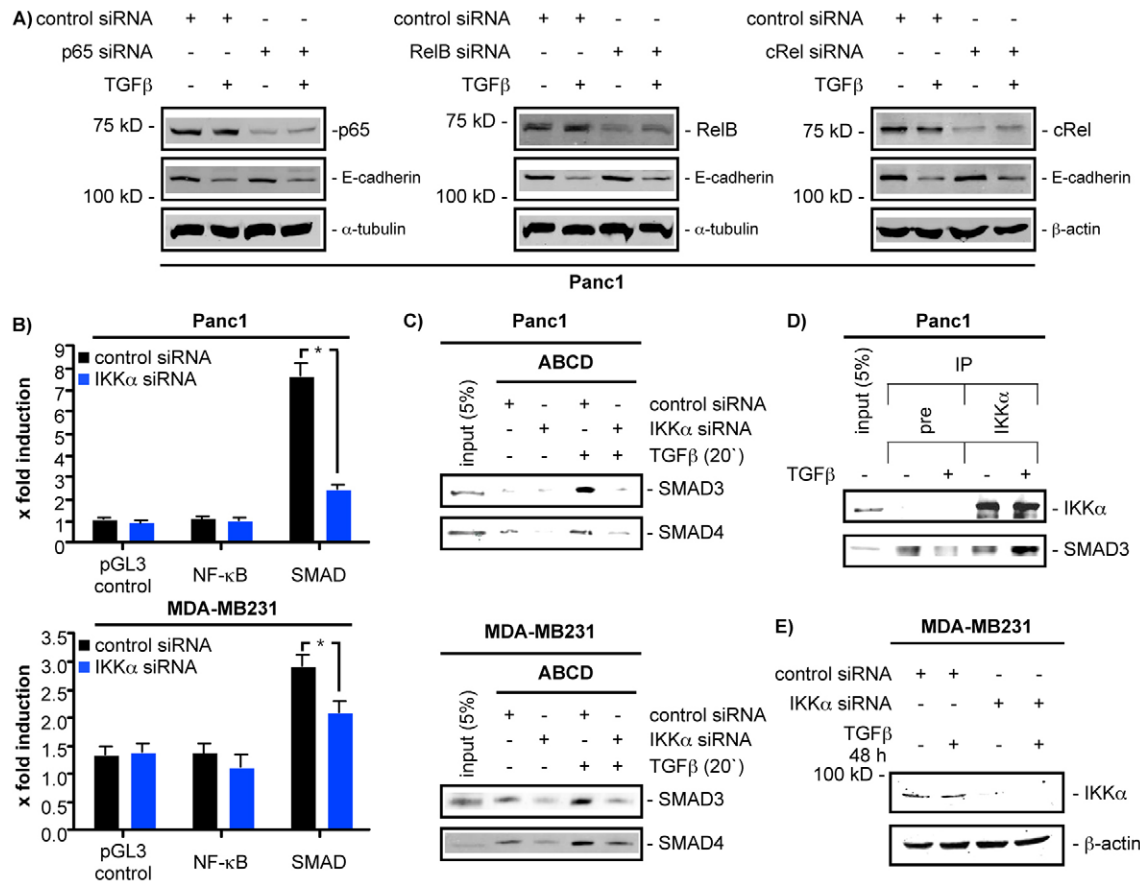


Fig. 4. TGF β -dependent downregulation of E-cadherin is NF κ B-independent. (A) Panc1 cells were transfected with a control or RelA/p65-, RelB- or c-Rel-specific siRNA. At 24 hours after the transfection, cells were treated with 10 ng/ml TGF β or were left as an untreated control in DMEM without FCS. After an additional 48 hours, western blots detected RelA/p65, RelB or c-Rel and E-cadherin expression. The membrane was stripped and probed for α -tubulin or β -actin to ensure equal protein loading. (B) Panc1 (upper graph) and MDA-MB231 cells (lower graph) were co-transfected with a control or IKK α -specific siRNA and 500 ng of the pGL3control-, NF κ B- or SMAD-luciferase reporter gene constructs as indicated. At 24 hours after the transfection, cells were treated with 10 ng/ml TGF β or were left as an untreated control. Luciferase activity was measured 6 hours after the TGF β treatment (Student's *t*-test: **P*<0.05 versus control). (C) Panc1 (upper graph) and MDA-MB231 (lower graph) cells were transfected with a control or IKK α -specific siRNA. At 48 hours after the transfection, cells were stimulated with TGF β (10 ng/ml) for 20 minutes and binding of SMAD3 and SMAD4 to a SMAD consensus oligonucleotide was detected using ABCD assays. Input represents 5% of whole-cell extract of control siRNA-transfected cells. (D) Panc1 cells were treated as in C. Immunoprecipitation was performed with an IKK α -specific antibody or pre-immune serum as a control. Western blots of immunoprecipitates were probed with antibodies against IKK α and SMAD3. Input represents 5% of whole-cell extract of control siRNA-transfected Panc1 cells. (E) MDA-MB231 cells were transfected with a control or IKK α -specific siRNA. At 24 hours after the transfection, cells were treated with 10 ng/ml TGF β or were left as an untreated control in DMEM without FCS. After an additional 48 hours, western blots detected IKK α expression. The membrane was stripped and probed for β -actin to ensure equal protein loading.

We apologise for this mistake.