The Journal of Experimental Medicine

Ilaria Guasparri, Shannon A. Keller, and Ethel Cesarman
Vol. 199, No. 7, April 5, 2004. Pages 993–1003.

The authors regret that an incorrect gel image was shown in Fig. 4 A. The correct data are shown below.

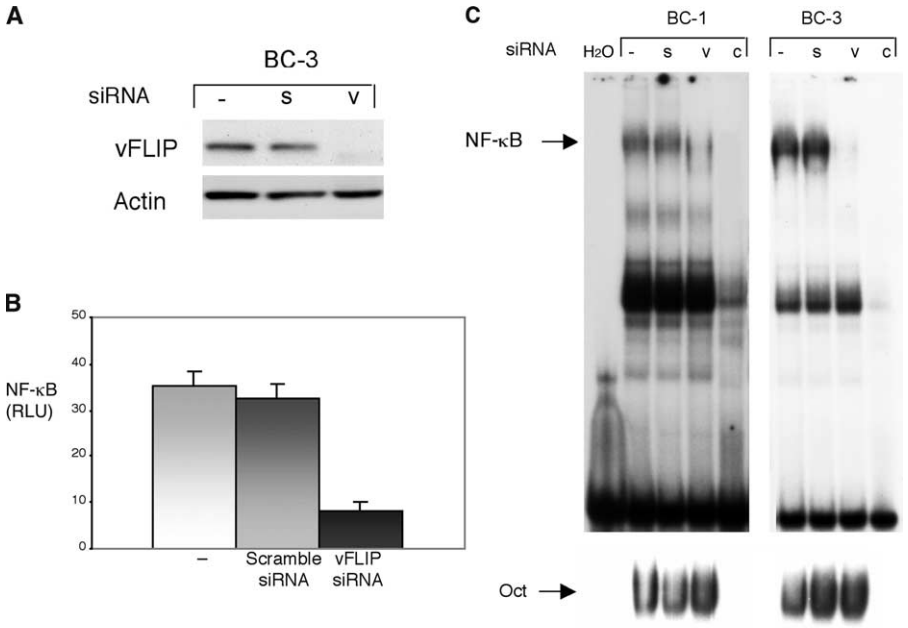

Figure 4. Inhibition of endogenous vFLIP by siRNA results in depletion of constitutive NF-κB activity in PEL cells. (A) BC-3 cells were transfected with a vFLIP siRNA (v) and scramble siRNA (s) and compared with mock-transfected cells (–). Protein extracts were prepared 48 h after transfection. Actin reprobing was performed to assure even protein loading. This experiment has been performed at least 10 times, and a representative immunoblot is shown. (B) BC-3 cells were transfected with an NF-κB luciferase reporter plasmid and either scramble siRNA as a control or siRNA for vFLIP. Luciferase assays were performed 48 h after transfection. Values shown are averages (± SEM) of one representative experiment out of three in which each transfection was performed in triplicate. (C) Electrophoretic mobility shift assays using a radiolabeled probe containing an NF-κB–binding site. BC-1 and BC-3 cells were transfected with vFLIP siRNA (v) or scramble siRNA (s) and compared with mock-transfected cells (–). Cold competition using 50-fold molar excess of an unlabeled NF-κB oligonucleotide (c) demonstrated the specificity of the protein–DNA-binding complexes. Nuclear extracts were prepared 48 h after transfection. In the lane labeled H_2O, water was used instead of nuclear extract. Binding to a radiolabeled oligonucleotide containing an octamer (Oct) motif was similarly examined as a control for specificity and for nuclear protein amount (bottom), where cold competition (c) was performed with nuclear extracts from mock-transfected cells and excess unlabeled oligonucleotide containing an octamer motif. This experiment was performed three times with similar results.