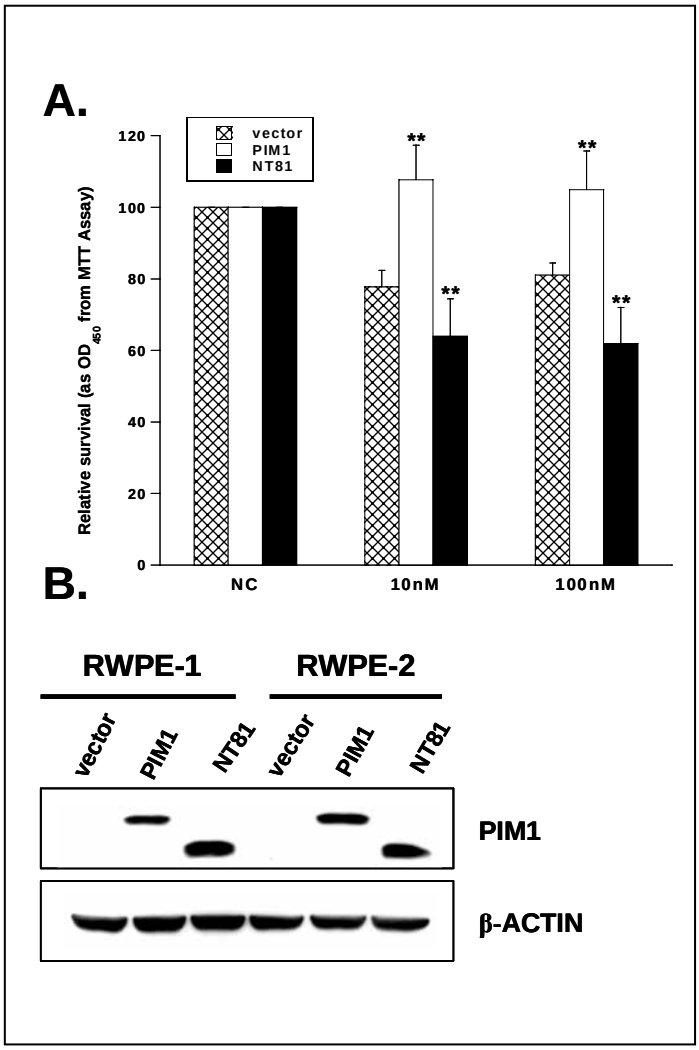
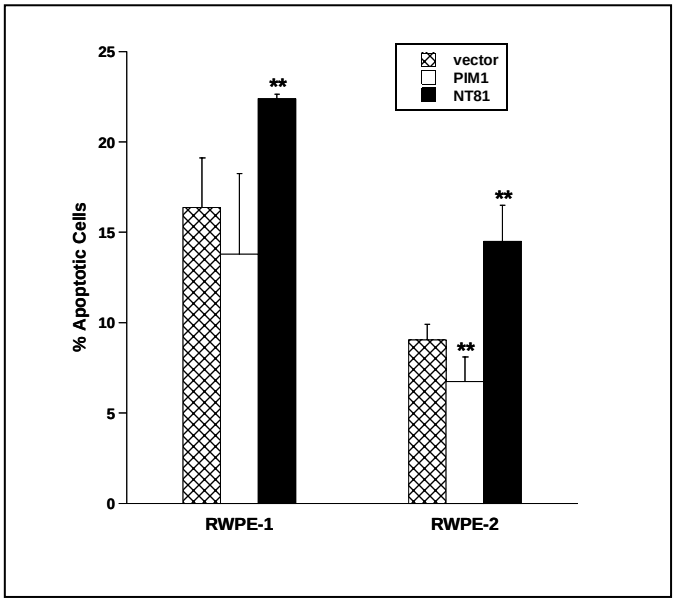
Fig. 1S



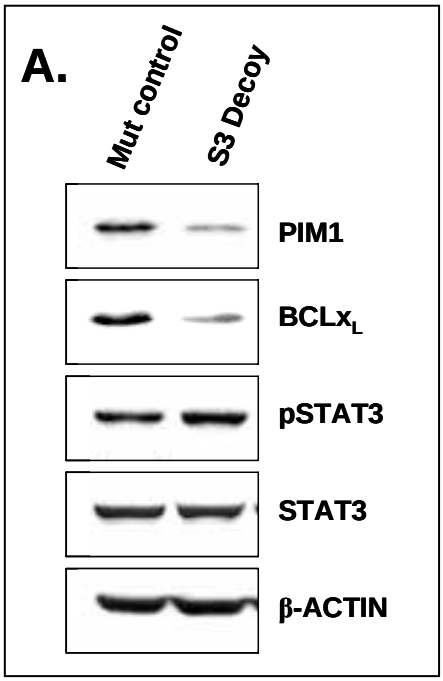
Expression of a dominant-negative (NT81) PIM1 protein sensitizes prostate cells to docetaxel-induced cell death. (*A*), RWPE-2 stable pools transfected with vector pLNCX, or pLNCX/ PIM1 or pLNCX/NT81 were treated with the indicated concentrations of docetaxel for 24 hours. Cell viability was measured by MTT assay, and showed the proportion of viable cells in treated cultures compared with those in untreated cultures (NC). Each bar shows the mean (± SD) of nine measurements pooled from three independent experiments. P values were calculated by T-tests. (**) = $p<0.01$ and indicates that the chance of no difference between treated and untreated cultures is less than one percent (*B*), Identification of PIM1 and NT81 transgene expression in RWPE-1 and RWPE-2 stable pools was determined by Western blot with indicated antibodies.

Fig. 2S



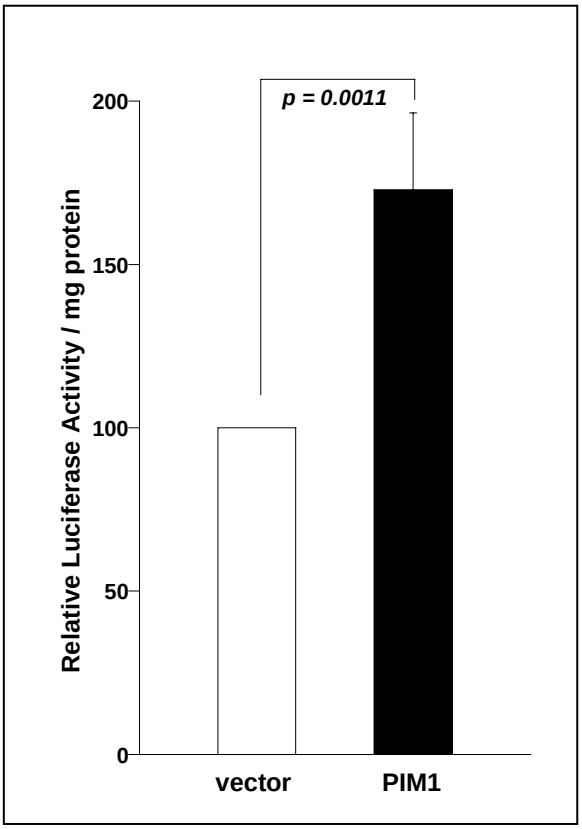
Detection of apoptosis induced by treatment with 100nM docetaxel for 24 hours in RWPE-1 and RWPE-2 cells trancfected with vector (pLNCX) ; pLNCX/PIM1 or pLNCX/NT81. “Percent Apoptotic Cells” represents the percent cells positive for carboxyfluorescein caspase, determined by flow cytometery. The basal percent-positive-cells (typically <5%) from untreated cultures has been subtracted from each bar value. Each bar shows the mean (± SD) of nine measurements pooled from three independent experiments. P values were calculated by T-tests. (**) = $p<0.01$ and indicates that the chance of no difference between different cell lines is less than one percent.

Fig. 3S



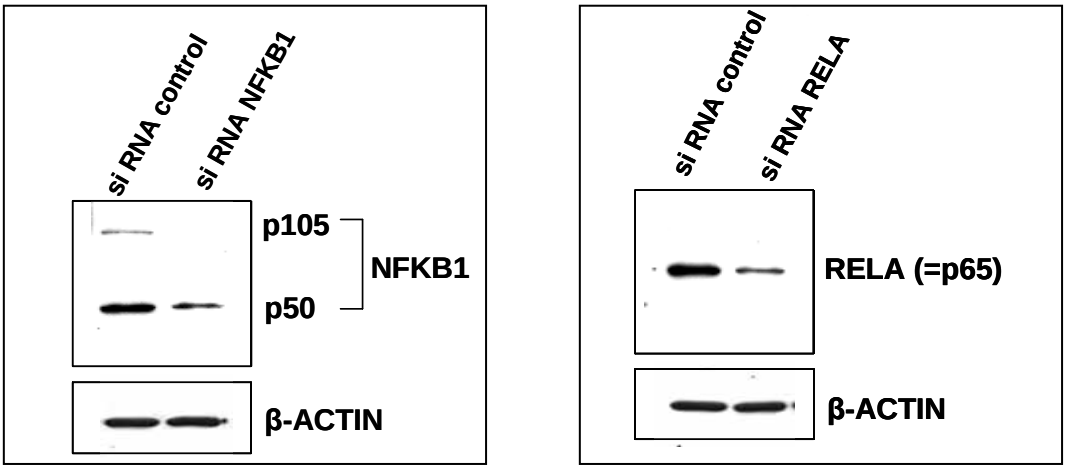
STAT3 decoy inhibits basal PIM1 expression in prostate RWPE-2 cells. RWPE-2 cells were transfected with STAT3 mutant control oligonucleotides or STAT3 decoy oligonucleotides and incubated for 48 hours. Expression of PIM1 and the known STAT3 target gene $BCLX_L$ were analyzed by immunoblotting. The STAT3 decoy, but not the mutant oligonucleotide, inhibit basal expression of PIM1 and BCLxL, but not that of pSTAT3, STAT3, or β-ACTIN.

Fig. 4S



Elevated level of PIM1 protein enhances NFκB activity in RWPE-2 prostate cancer cells. A pool of RWPE-2 cells stably expressing a NFκB-luciferase reporter plasmid was infected with retroviruses encoding a PIM1 cDNA. After selection with G418, stable pools were analyzed for expression of PIM1 by immunoblotiing (data not shown). RWPE-2/NFκB-luciferase/pLNCX cells and RWPE-2/NFκB-luciferase/PIM1 cells were plated and allowed to grow overnight. On the next day the expression of luciferase was measured and normalized to the protein concentration for each sample. The normalized activity in PIM1 expressing cells was compared to that of vector-transfected cells. The values present the mean (± SD) of the means from five independent experiments. The paired T-test was used to determine statistical significance for the values of luciferase activity measured in the PIM1 expressing cell line as compared to those of the vector transfected cells.

Fig. 5S



siRNA oligonucleotides inhibit expression of p65/RELA and p50/NFKB1 proteins. Immunoblot analysis of NFKB1 (left) and RELA (right) protein expression in RWPE-2/PIM1 cells treated with siRNAs. β-ACTIN expression documents equal loading of the lanes.