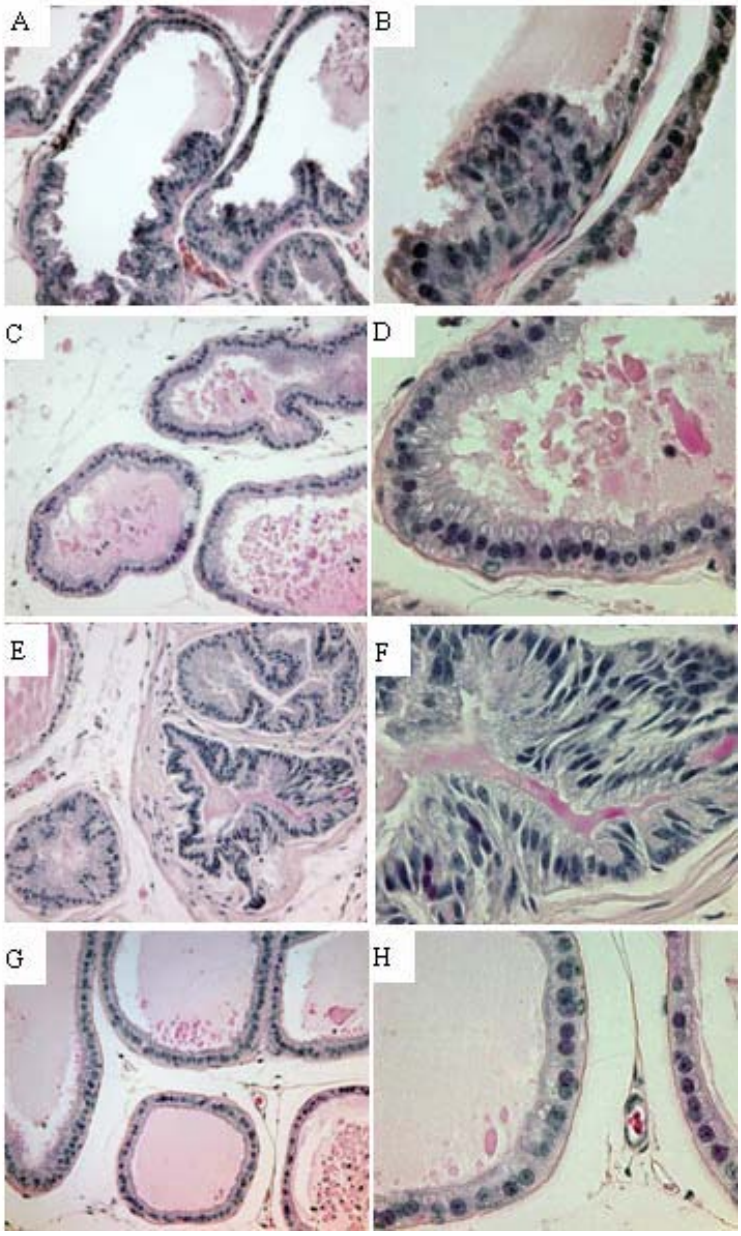
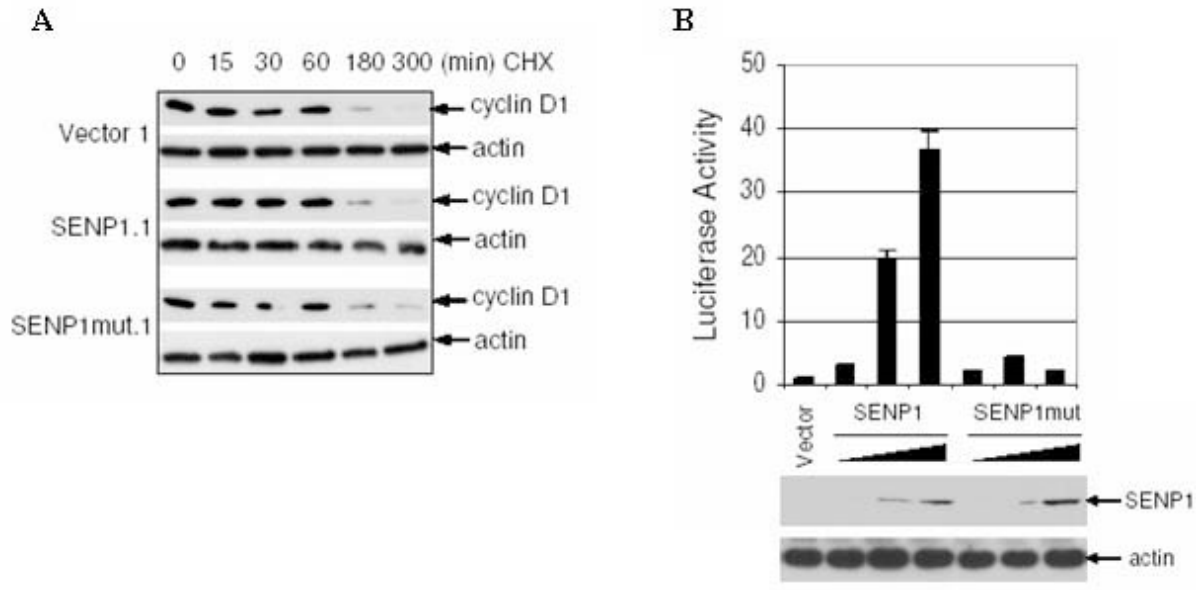
SUPPLEMENTAL FIGURE 1



SUPPLEMENTAL FIGURE 2



Supplemental Figure 1. PIN lesions in 6 and 10 month-old C-line transgenic mice. Tissue samples were isolated and evaluated in a manner analogous to that described in **Figure 2**. The prostate gland from 6 (**A-B**) and 10 (**E-F**) month-old C-line SENP1 transgenic mice exhibit clear signs of dysplasia when compared to their respective age-matched counterparts (**C-D** and **G-H**).

Supplemental Figure 2. SENP1 in prostate cancer cells induces transcription of cyclin D1. **A**. Vector-, SENP1-, or SENP1mut-LNCaP cells were treated with cycloheximide (20 μM). The cyclin D1 protein level was then determined at different times after treatment. The amount of loaded protein was normalized to the equal amount of cyclin D1 in each cell clone at time point of 0 min and the loaded protein at different time point of each cell clone was normalized to the equal amount of actin. **E**. PC-3 cells were co-transfected with the cyclin D1 promoter (-1745/+134) -luciferase reporter construct and different doses of SENP1 or SENP1 mutant plasmid. At 36 hours after transfection, luciferase activity and SENP1 expression were determined. All luciferase activity was normalized to individually determined β-gal activity and presented as the mean ± standard error of triplicate transfections.