Supporting information

Tworkowski *et al.* 10.1073/pnas.0802095105

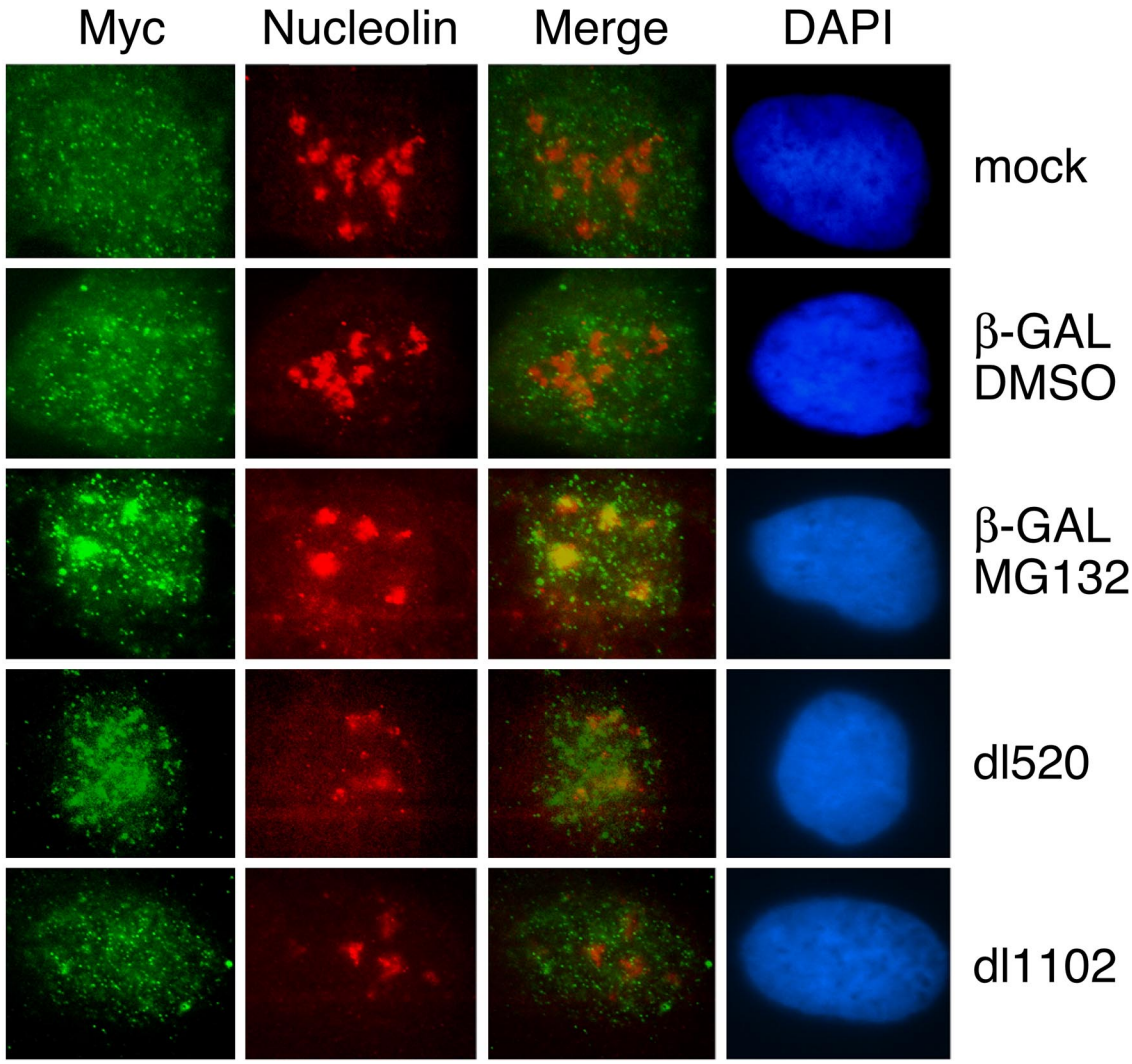

Fig. S1. Effects of proteasome inhibition versus E1A expression on the subcellular localization of Myc. U2OS cells were grown on coverslips and infected with control β-gal virus (+/− MG132) and *dl520* adenovirus. Six hours after infection, cells were fixed and immunostained for Myc (N262), Nucleolin (C23), and DNA (DAPI). Myc accumulates in the nucleolus in response to MG132 treatment, but not in response to adenoviral infection. Nearly 70–80% cells showed Myc accumulation in the nucleolus upon MG132 treatment in two independent experiments. Representative cells are shown.

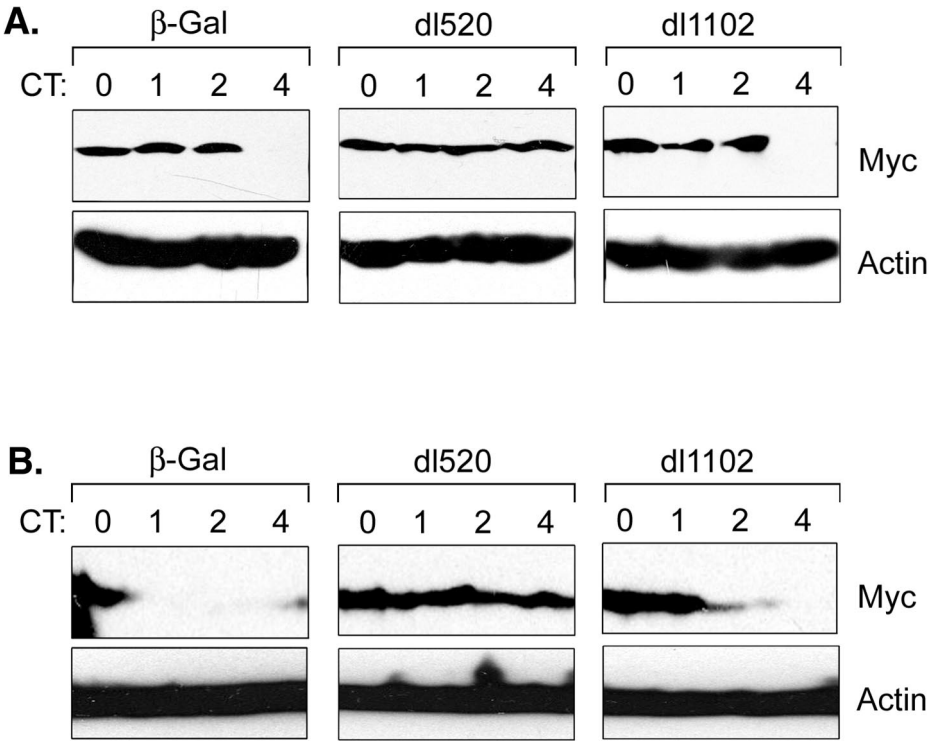

Fig. S2. E1A stabilizes Myc in Rat1 and IMR90 cells. Rat1 (*A*) or IMR90 (*B*) cells were either infected with adenovirus *dl520*, *dl1102*, or a control virus expressing β-gal, and incubated for 10 h. CHX was added to inhibit protein synthesis, and protein samples were taken at the indicated times (CT; hours). WB shows levels of Myc and actin.

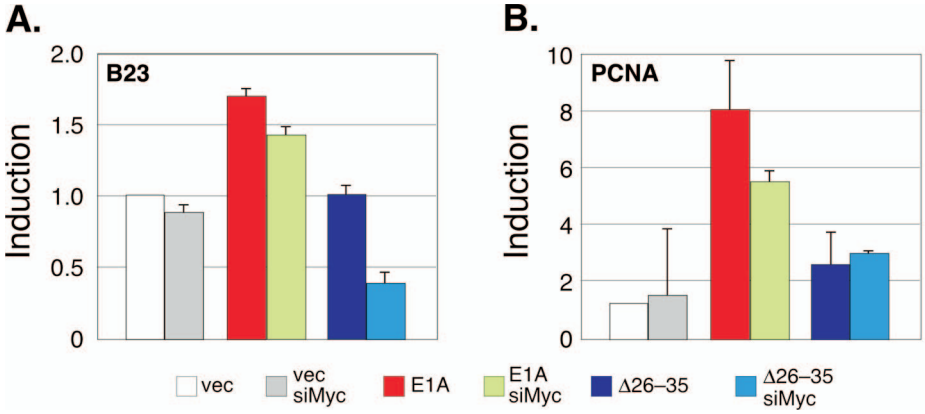

Fig. S3. E1A activates B23 and PCNA expression. IMR90 cells were transduced with the indicated retroviruses, and then transfected with either nontargeting siControl, or siMyc RNA, duplexes for 48 h. RNA was then harvested and levels of B23 (*A*) or PCNA (*B*) cDNA analyzed by quantitative PCR. Fold induction is normalized to an actin control for each sample.

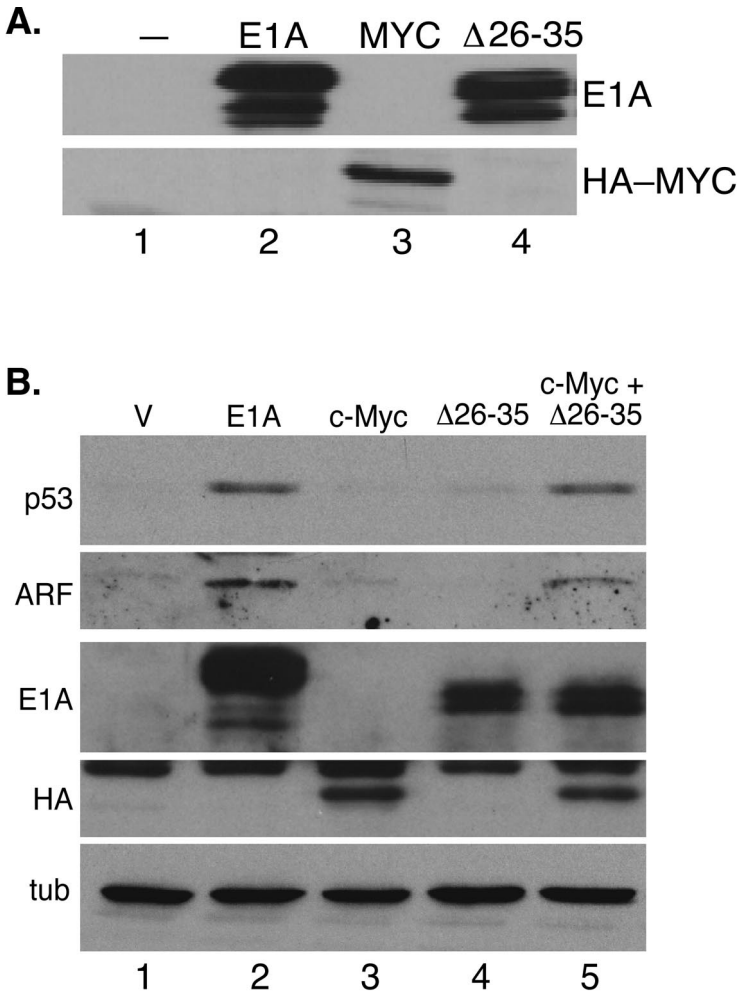

Fig. S4. Overexpression of Myc rescues the ability of the Δ26–35 E1A mutant to induce p53/ARF. (*A*) Protein was harvested from cells depicted in Fig. 4*D*, and levels of E1A and Myc (HA) were determined by WB. (*B*) Protein was harvested from cells depicted in Fig. 4*F*, and levels of p53, ARF, E1A, Myc (HA), and tubulin were determined by WB.