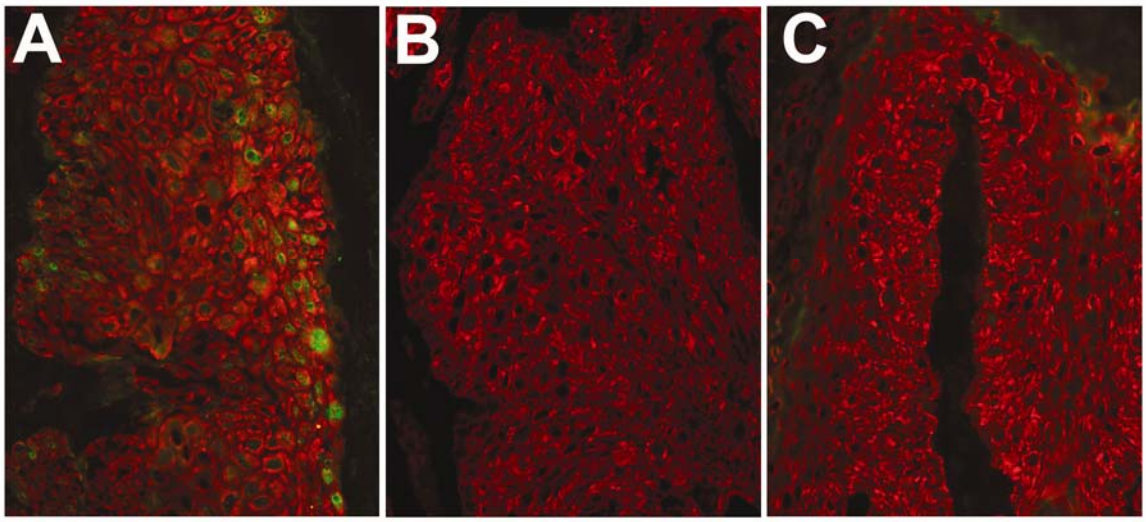
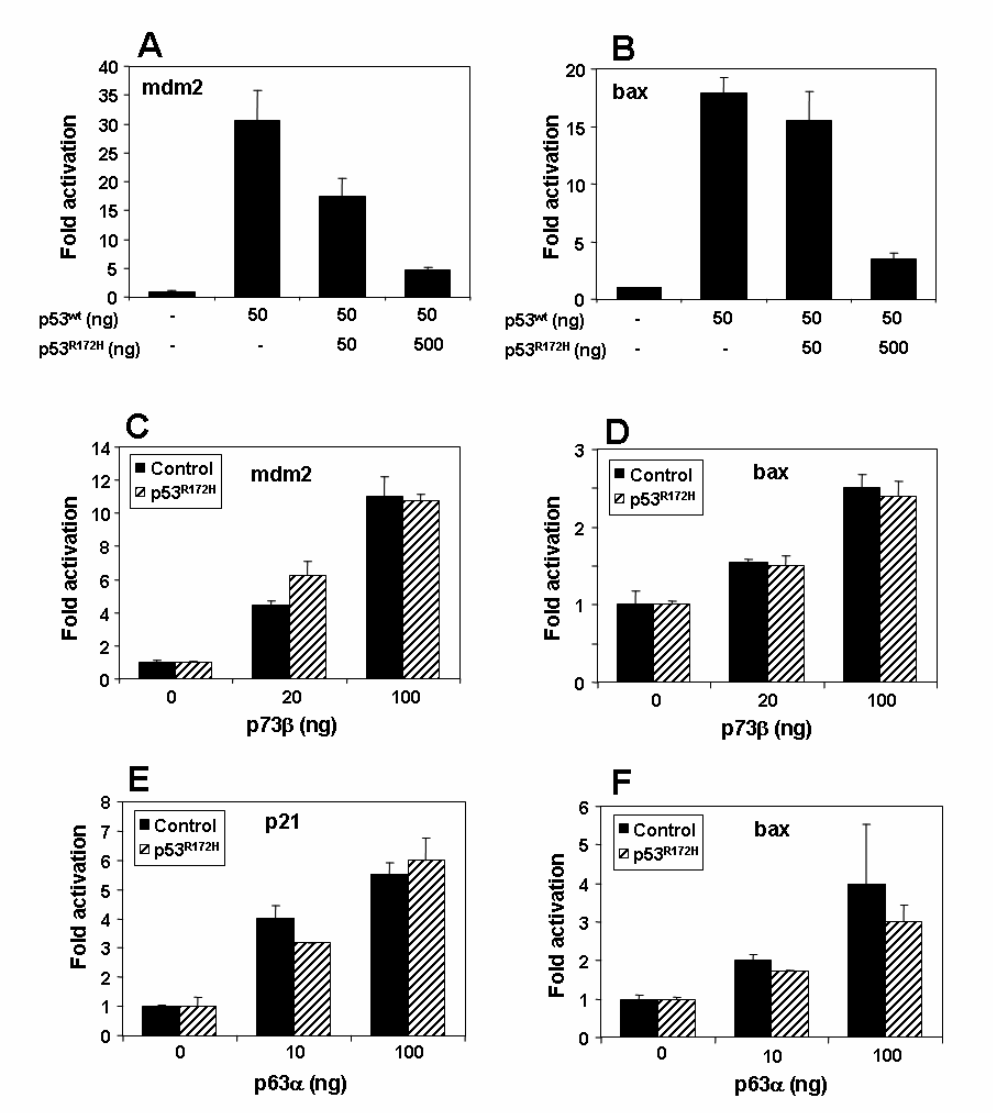
Supplemental Figure 1. Caulin et al



Supplemental Figure 1: Detection of $p53^{R172H}$ in tumors. Double immunofluorescence for p53 (green) and K14 (red) in tumors that developed in (A) Kras-$p53^{R172H/f}$, (B) Kras-$p53^{f/f}$ and (C) Kras-$p53^{wt/wt}$ mice. Note that p53 is only detected in the nuclei of tumors that express the mutant $p53^{R172H}$.

Supplemental Figure 2. Caulin et al



Supplemental Figure 2: p53^{R172H} does not modulate p63 and p73 transactivation in skin keratinocytes. (A-B) p53-null keratinocytes were co-transfected with the indicated amounts of expression vectors for p53^{wt} and/or p53^{R172H}, luciferase reporter constructs carrying the bax or mdm2 promoters as indicated and a β-galactosidase reporter gene. Luciferase activity was normalized with β-galactosidase activity and results are expressed as fold increase relative to basal activity of the reporter genes in the absence of p53^{wt} and p53^{R172H}. Note that p53^{R172H} inhibits the transactivation function of p53^{wt}, consistent with a dominant negative effect. (C-F) Luciferase reporter assays with the bax, mdm2 or p21 promoters using increasing concentrations of expression vectors for p73β (C-D) or p63α (E-F), in the presence of 1μg of the p53^{R172H} vector or in the absence of p53^{R172H} (Control). Note that the transactivation properties of p73β and p63α remain unchanged in the presence of p53^{R172H}.