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/+}, (B) Kras-p53^{+/+} 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: p53R172H 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 p53wt and/or p53R172H, 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 p53wt and p53R172H. Note that p53R172H inhibits the transactivation function of p53wt, 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 p53R172H vector or in the absence of p53R172H (Control). Note that the transactivation properties of p73β and p63α remain unchanged in the presence of p53R172H.