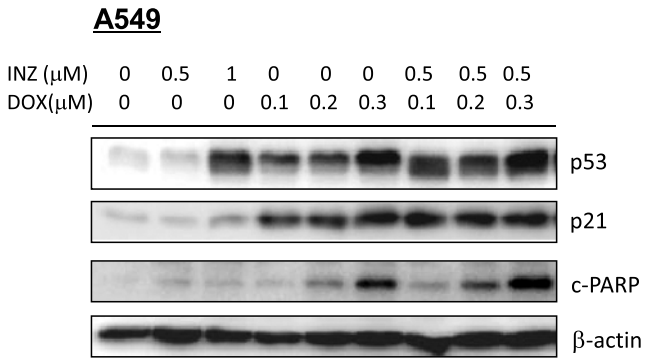

Figure W1. INZ significantly enhances the expression level and activity of p53 as well as the apoptotic response induced by DOX in A549 cells in a dose-dependent manner. Cells were treated with INZ or DOX at the indicated concentrations for 18 hours and harvested for WB analysis. Fifty micrograms of proteins was loaded in each lane, and an anti–β-actin antibody was used to confirm equal loading. Similar results were obtained from three separate experiments.

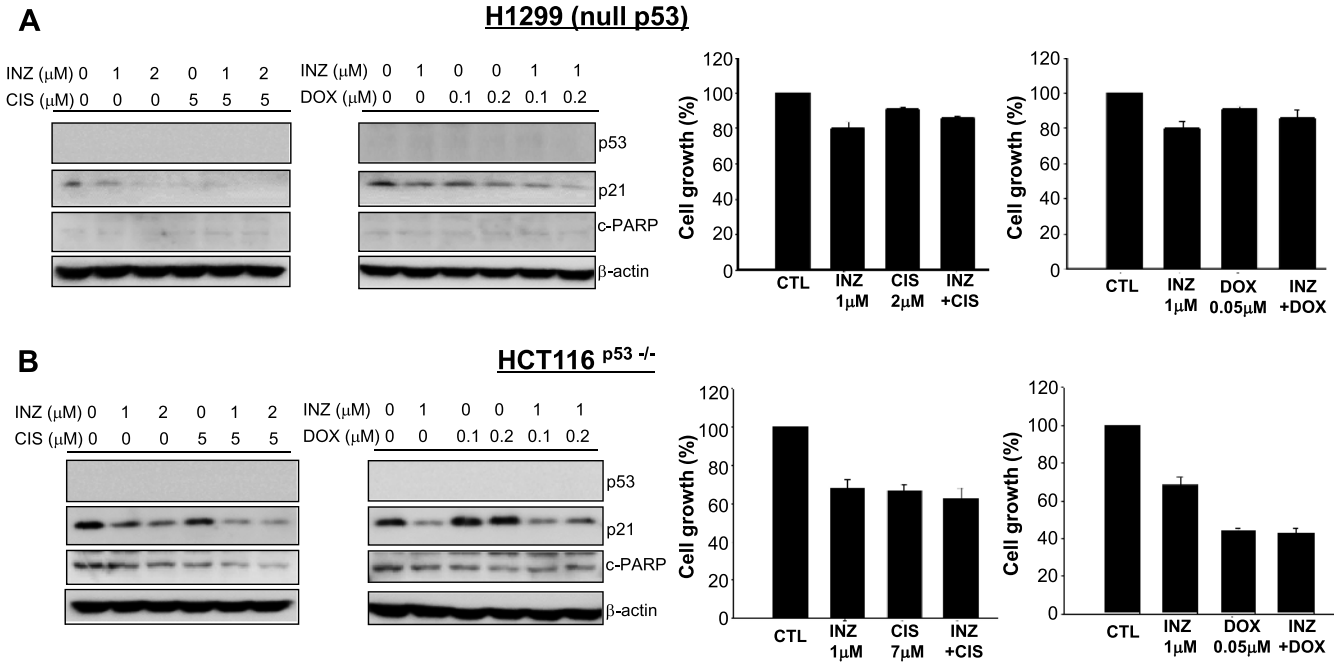

Figure W2. INZ does not enhance the cell apoptotic response and cytotoxicity in the presence of CIS or DOX in p53-null cells. (Left) H1299 (A) and HCT116$^{p53-/-}$ (B) were treated with INZ or/and CIS/DOX at the indicated concentrations for 18 hours and harvested for WB analysis. Fifty micrograms of protein was loaded in each lane, and an anti–β-actin antibody was used to confirm equal loading. Similar results were obtained from three separate experiments. (Right) H1299 (A) and HCT116$^{p53-/-}$ (B) were plated in 96-well plates and treated with INZ or CIS/DOX individually or both simultaneously at the indicated concentrations for 72 hours. Cell viability was determined using the WST cell growth assay. The data shown represent the mean of three independent experiments ± SEM.

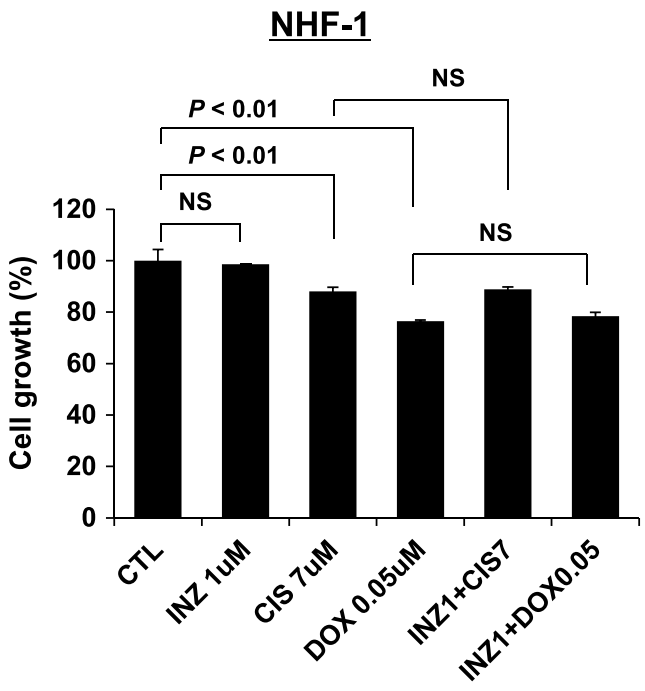

Figure W3. INZ does not enhance the cytotoxic effect of CIS or DOX on NHF-1 cells. NHF-1 cells were plated in 96-well plates and treated with INZ or CIS/DOX individually or both simultaneously at the indicated concentrations for 72 hours. Cell viability was determined using the WST cell growth assay. The data shown represent the mean of three independent experiments ± SEM.