Figure S1. Anti-proliferative effect of HS-1793 on human lung cancer cells. A549 (grey bars), H460 (black bars), and H1299 (white bars) cells were treated with HS-1793 (0-10  $\mu$ M) for 24 h, and cell viability was measured using a CCK-8 assay.

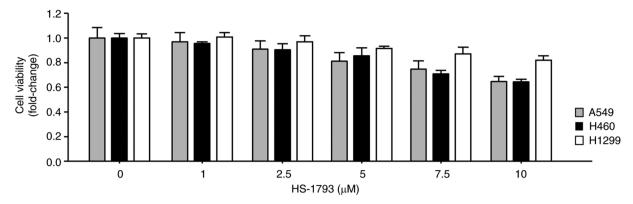
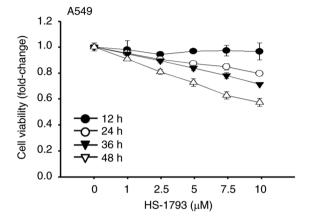


Figure S2. HS-1793 diminishes the cell viability of A549 and H460 cells. A549 and H460 cells were treated with HS-1793 at different concentrations (0, 1, 2.5, 5, 7.5, and 10  $\mu$ M) for different times (12, 24, 36, and 48 h), and absorbance was measured at 450 nm after 1 h incubation, using a CCK-8 reagent to confirm cell viability.



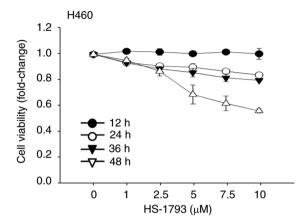
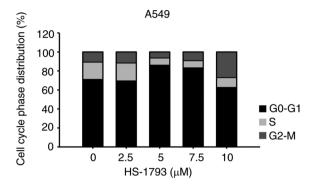


Figure S3. Effect of HS-1793 on A549 and H460 cell cycle. A549 and H460 cells were treated with 0 10  $\mu$ M HS-1793 for 24 h, and the cell cycle profile was determined using flow cytometry after staining with PI/RNase. Percentages of each cell cycle phase with various treatments or with control are shown.



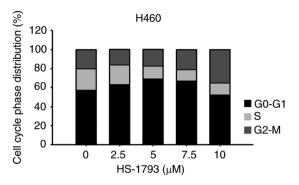


Figure S4. Endogenous p53 protein expression levels in both A549 and H460 cells by western blot analysis.

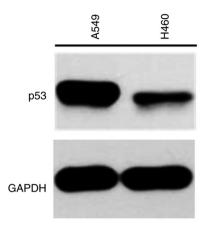


Figure S5. Increased p53 expression by HS-1793 upregulates its target genes p21. A549 and H460 cells were treated with HS-1793 at different concentrations (0, 2.5, 5, 7.5, and 10  $\mu$ M) for 24 h. mRNA expression of the cells was measured using a quantitative polymerase chain reaction. B2M was used as a control and normalized to obtain a relative quantification graph. Results are expressed as fold change. Data indicate the mean  $\pm$  standard deviation of independent experiments performed twice.

