Supplementary Figure Legends

Supplementary Figure 1. Structures of the luciferase gene–fused *ARF, Hdm2, p53* and *p21* gene promoters of the p53 pathway tested in Fig. 1A. Binding sites for transcriptional factors are indicated. +1, Tsp, transcriptional start site; Hdm2, p53-dependent promoter 2 of Hdm2.

Supplementary Figure 2. FBI-1 represses transcription of *ARF, Hdm2, p21* and *Rb* in HCT116 colon cancer cells. FBI-1 represses transcription of *p21* by acting on the GC-rich proximal promoter. (A) Transcriptional analysis of the *ARF, Hdm2, Rb,* p53 and *p21* promoters fused with a luciferase gene in HCT116 $p53^{+/+}$ cells. **(B)** Transcriptional analysis of the four pGL2-p21-Luc plasmids in HCT116 cells for mapping the *p21* promoter region repressed by FBI-1. FBI-1 can repress transcription by acting on the GC-rich proximal promoter (-131 bp construct). (**C)** Induction of p53 by etoposide, and transcriptional repression of *p21* by FBI-1 in HCT116 $p53^{+/+}$ cells. **(D)** Transcriptional activation of *p21* by ectopic p53 in HCT116 $p53^{-/-}$ cells is repressed by FBI-1. **(E)** Western blot analysis of p53, FBI-1, p21 and GAPDH in control HCT116, and of stable HCT116 $p53^{+/+}$ or $^{-/-}$ cells overexpressing FBI-1, established by transfection with recombinant lentiviruses. FBI-1 represses *p21*gene expression in the presence or absence of p53.

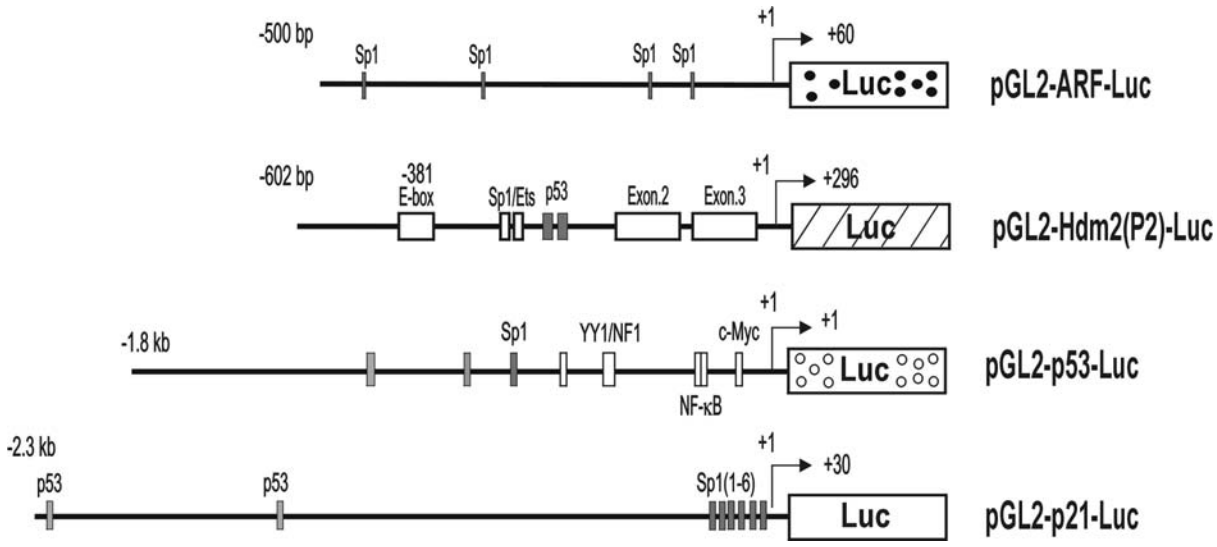
Supplementary Figure 3. FBI-1 can repress transcription activation of p53 responsive genes by p53 induced by etoposide treatment or by ectopic p53. (A, B) Transcription and Western blot assays. Induction of p53 by etoposide and transcriptional repression of *p21* by FBI-1 in HeLa cells. **(C)** Western blot analysis of stable HCT116 $p53^{+/+}$ or $p53^{-/-}$ cells prepared by infection with recombinant lentivirus control, or lentivirus FBI-1. FBI-1 repressed expression of Hdm2 and p21, in the presence or absence of p53. **(D)** ChIP assays and binding competition between FBI-1 and p53 for binding to the distal p53-binding element of endogenous *p21* in Saos-2 cells. p53 binding is decreased by increasing the amount of the FBI-1 expression vector transfected.

Supplementary Figure 4. FBI-1 promotes proliferation in stable HEK293Trex-Flag-FBI-1 cells upon induction by doxycyclin. (A) Western blot analysis of cell extracts prepared from stable HEK293Trex-Falg-FBI-1 cells induced by doxycyclin. FBI-1 induction repressed *p21* expression. **(B)** BrdUrd incorporation assay in HEK293Trex cells. HEK293Trex control and stable HEK293Trex-Flag-FBI-1-Flag cells were grown in DMEM containing BrdUrd, in the presence or absence of doxycyclin. The cells were analyzed by immunohistochemistry. **(C)** Histogram of BrdUrd incorporation assay in HEK293Trex cells. **(D-F).** BrdUrd incorporation assay. NIH/3T3 and stable NIH/3T3-FBI-1 cells were grown in media containing BrdUrd. BrdUrd incorporation was measured by immunohistochemistry, as described above.

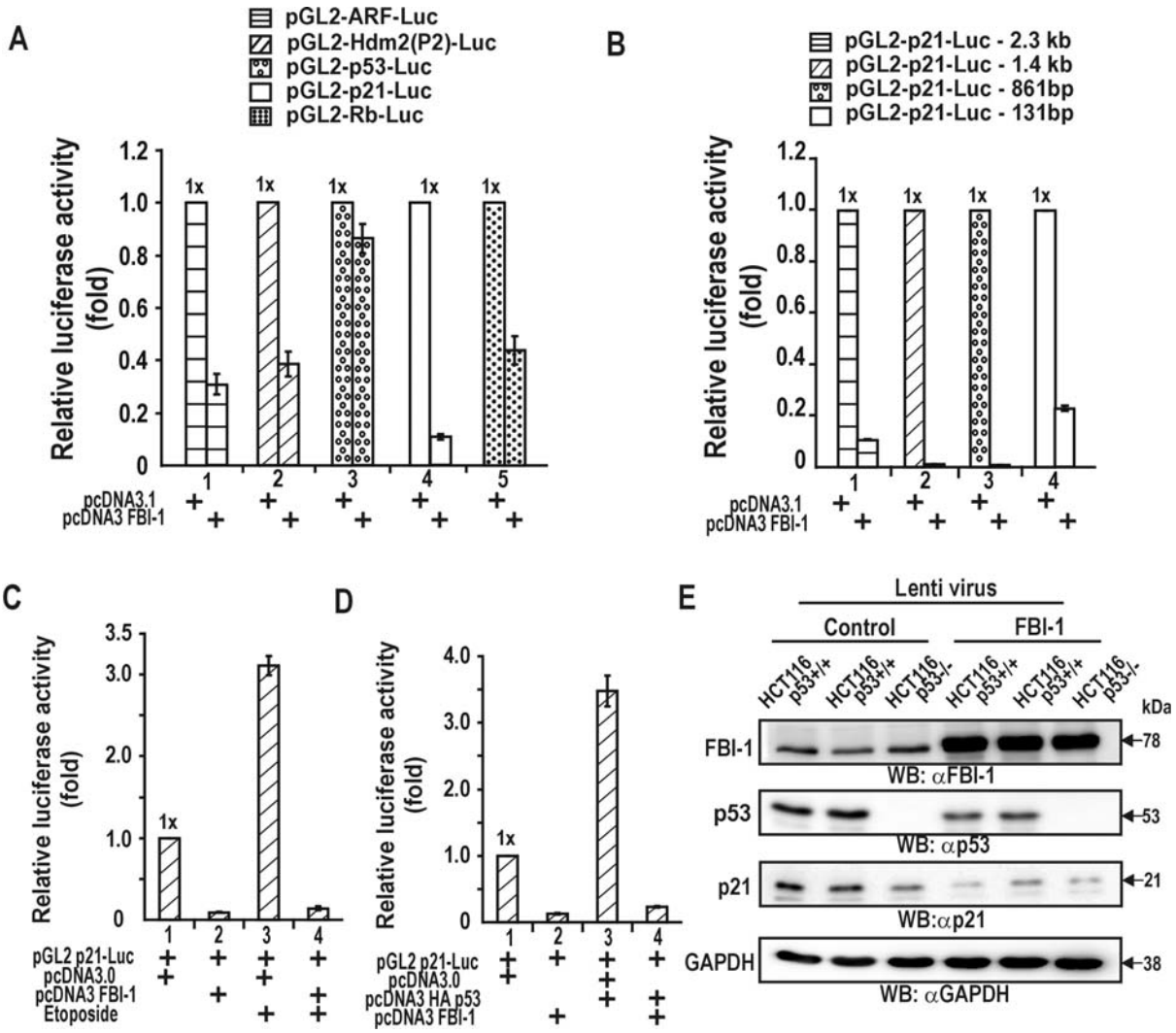
Supplementary Figure 5. FBI-1 represses *p21* expression in human colon HCT116 $p53^{+/+}$ and $p53^{-/-}$ cells, and causes cell cycle progression and proliferation. (A, B) Western blot analysis of p53, FBI-1, *p21* and GAPDH. Induction of p53 by etoposide and transcriptional repression of *p21* by FBI-1 in stable HCT116 $p53^{+/+}$ or $p53^{-/-}$ cells. HCT116 cells stably overexpressing FBI-1 were established by transfection with recombinant lentivirus overexpressing FBI-1. **(C, D)** MTT assay of HCT116 and stable HCT116-FBI-1 cells grown for 2, 4 or 6 days. All assays were performed in triplicate. The number of living cells was calculated from control cells cultured and treated with MTT under conditions the same as those of the experimental groups. Alternatively, MTT assays were performed in

HCT116 cells treated with either negative control siRNA or siRNAs specific to FBI-1. Error bars are included, but are too tight to see.

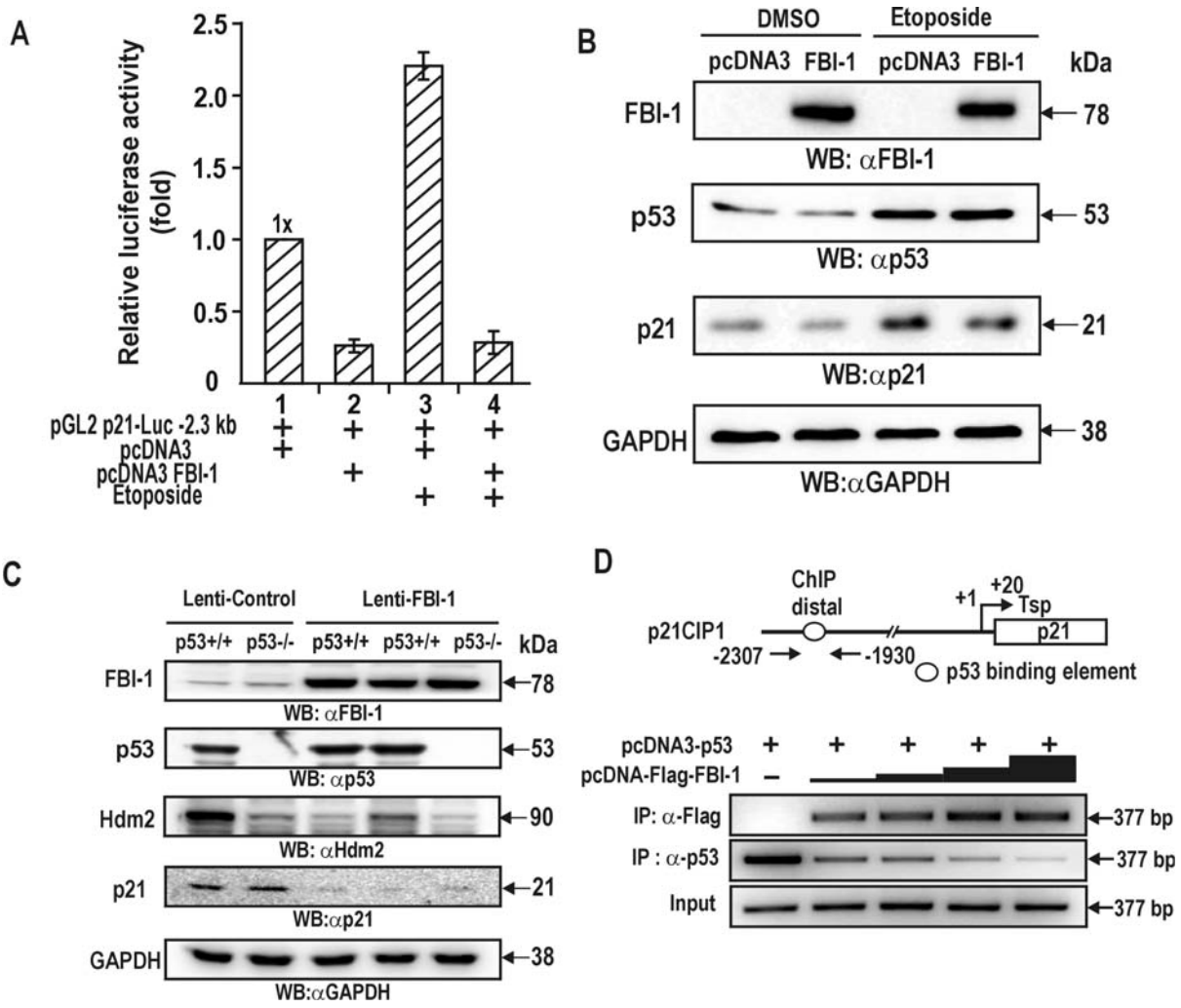
Supplementary Figure 6. FBI-1 stimulates cell cycle progression and increases the number of HCT116 $p53^{+/+}$ or $p53^{-/-}$ cells in S phase. (A, B) FACS analysis of cell cycle progression. Control HCT116 and stable HCT116-FBI-1 $p53^{+/+}$ or $p53^{-/-}$ cells were stained with propidium iodide, and cell proliferation was measured by FACS. Ectopic FBI-1 stimulates cell cycle progression and increases the number of HCT116 cells in S phase. **(C)** Knock-down of FBI-1 by siRNAs resulted in a significant decrease in the number of cells in S-phase and a concomitant increase in the number of cells in G0-G1 phase. **(D, E)** The number of cells in S-phase was significantly increased after etoposide treatment. Cells in Go-G1 phase were nearly eliminated by etoposide treatment.



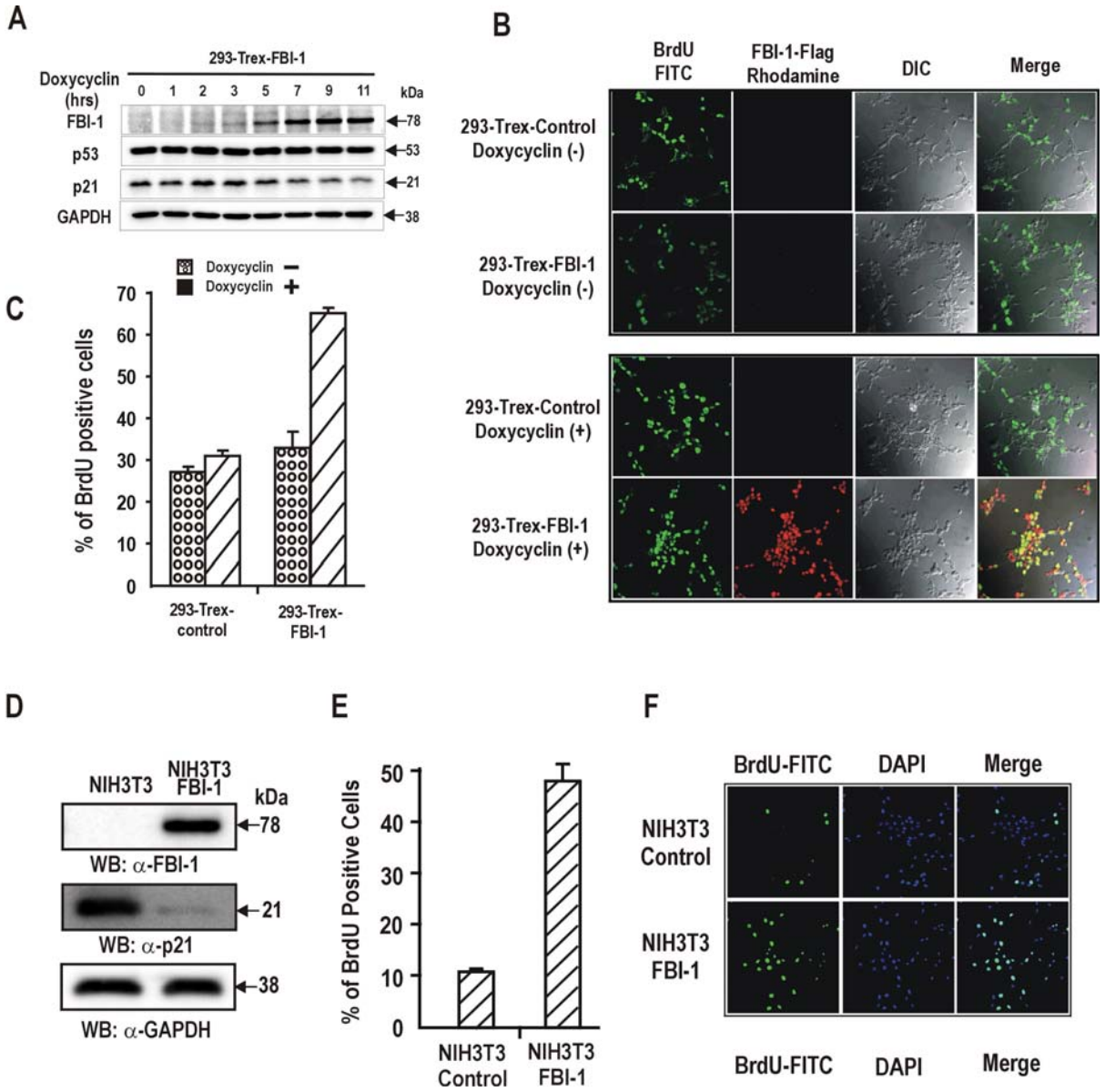
Supplementary Figure 1. Choi et al. 2008



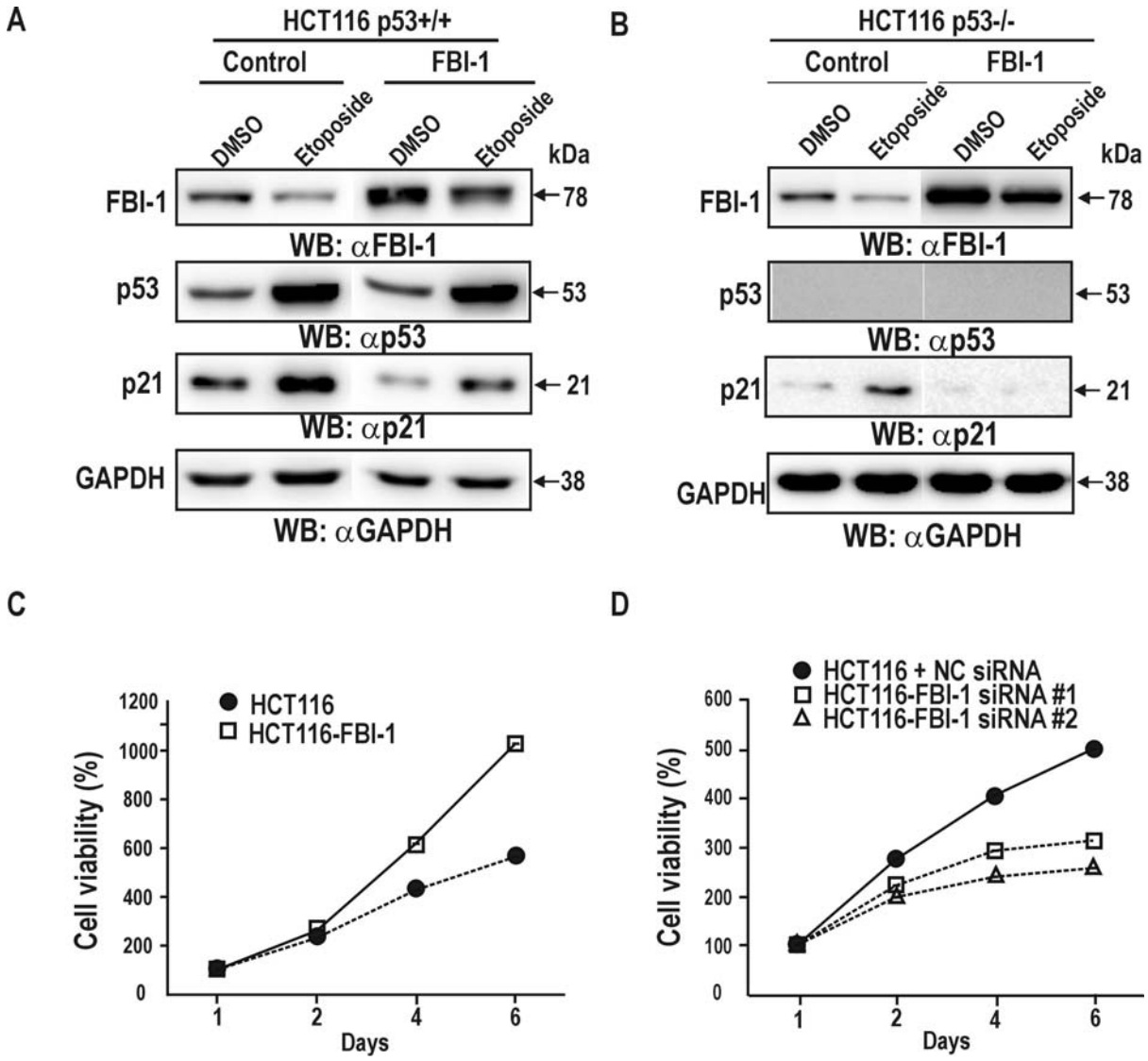
Supplementary Figure 2. Choi et al. 2008



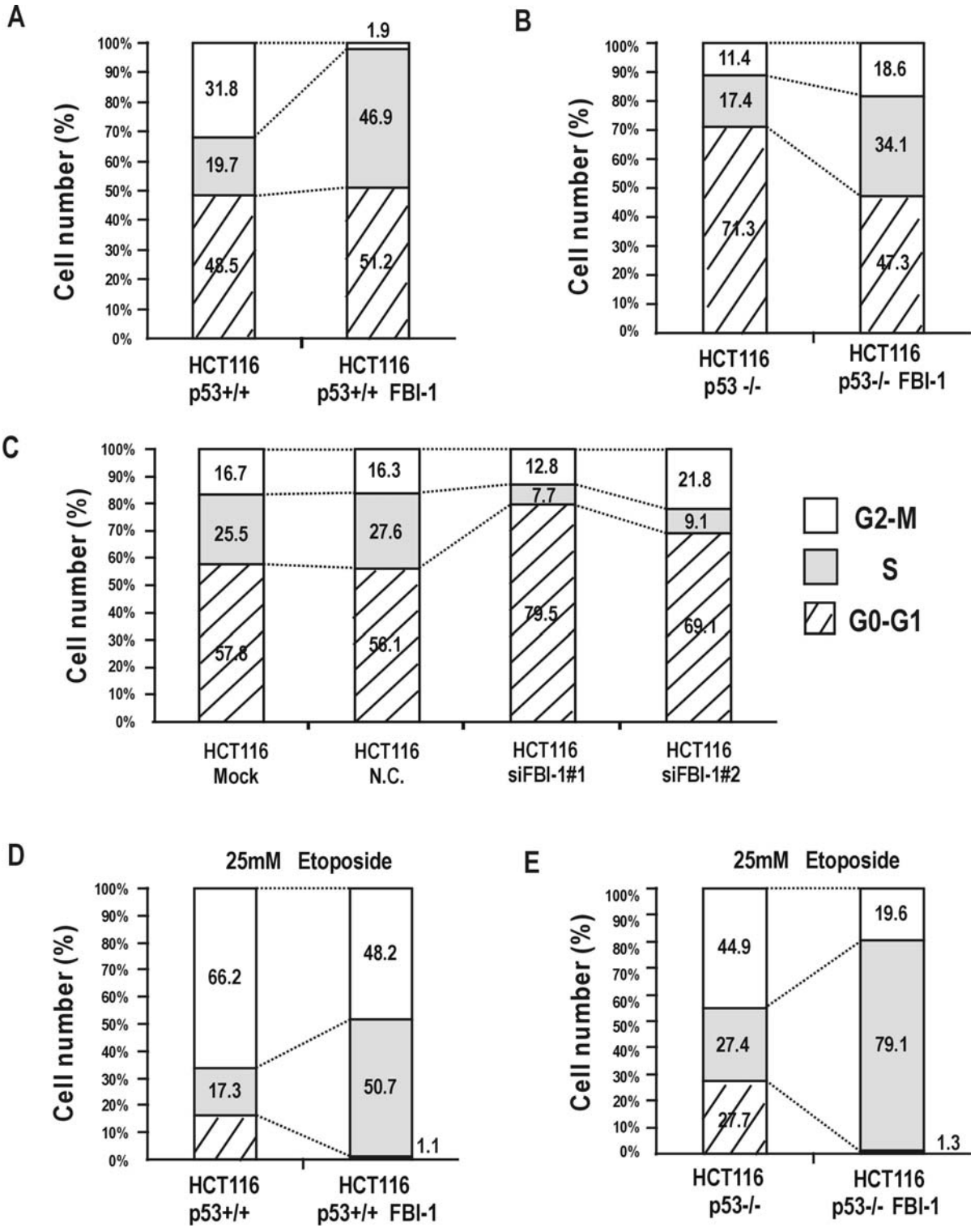
Supplementary Figure 3. Choi et al. 2008



Supplementary Figure 4. Choi et al. 2008



Supplementary Figure 5. Choi et al. 2008



Supplementary Figure 6. Choi et al. 2008