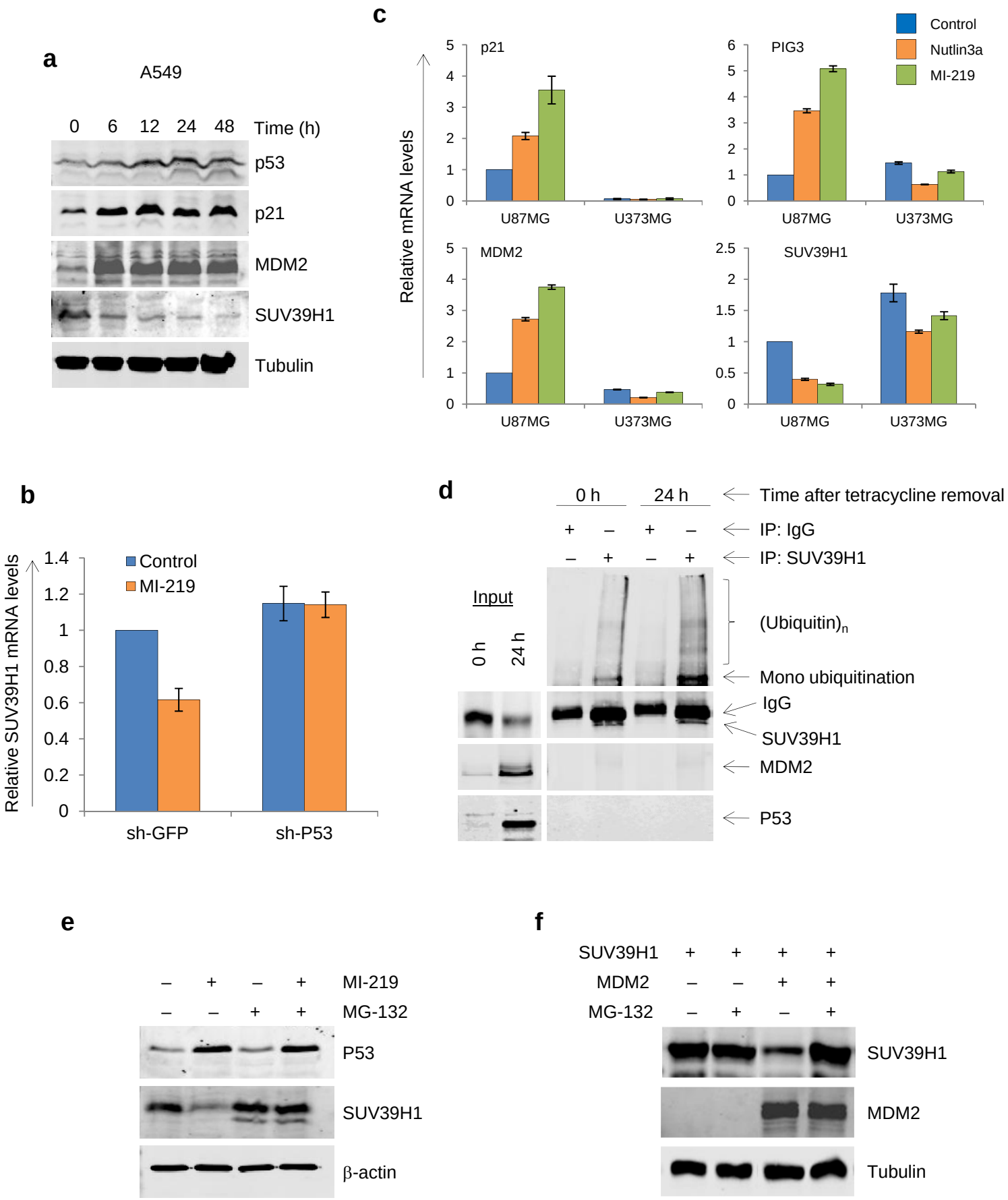
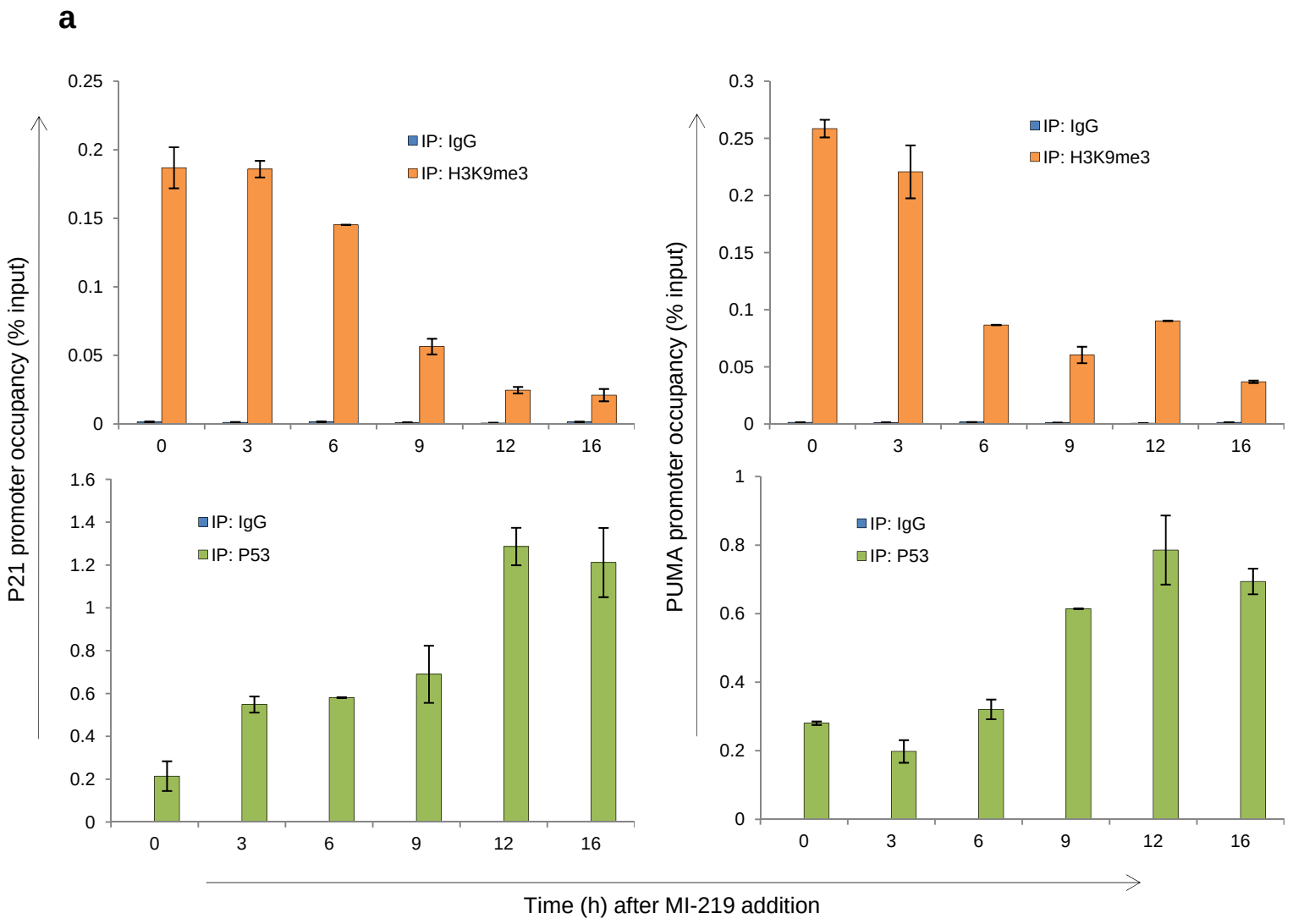
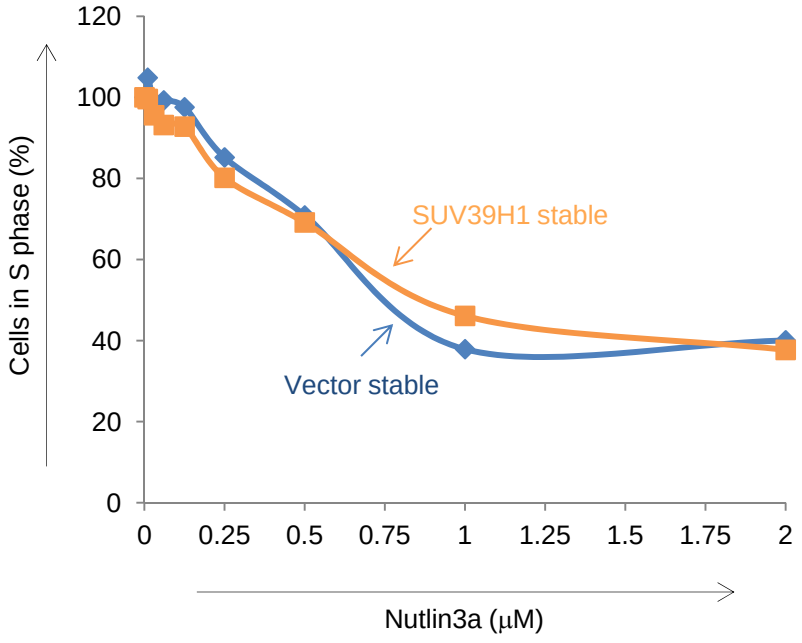
Supplementary Figure 1; Mungamuri et al., 2012

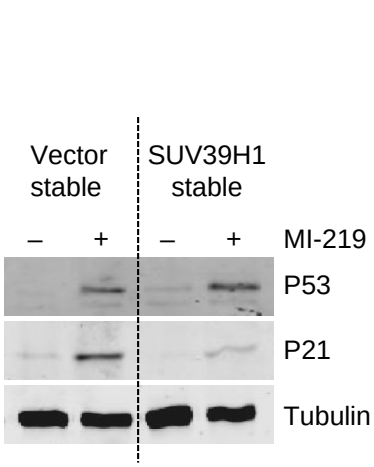


Supplementary Figure 1: p53 downregulates SUV39H1 expression. (**a**) Western blot analysis of A549 cells treated with hydrogen peroxide for the indicated time points. (**b**) Real time quantitative PCR of B5/589 cells expressing either sh-GFP or sh-p53 and treated with MI-219. (**c**) Real time quantitative PCR of U87MG (p53 WT) and U373MG (p53 mutant) glioblastoma cells treated with either Nutlin3a or MI-219. (**d**) Western blot analysis of SUV39H1 immunoprecipitated samples. EJ-p53 cells were induced for p53 expression and cell lysates were prepared after 24 h of induction. SUV39H1 protein was immunoprecipitated and loaded on a gradient SDS-PAGE gel, and probed with an anti-ubiquitin antibody. The samples were also probed with MDM2 and p53 antibodies. 10 % of the sample used for IP was also loaded and showed as the Input sample. (**e**) Western blot analysis of B5/589 cells treated with MI-219. The cells were also incubated with MG-132, for 4 h before cell lysis. (**f**) Western blot analysis of H1299 cells transfected with 2 μg of indicated plasmids and lysed after 24 h. The cells were treated with MG-132 for 4 h before cell lysis.

Supplementary Figure 2; Mungamuri et al., 2012







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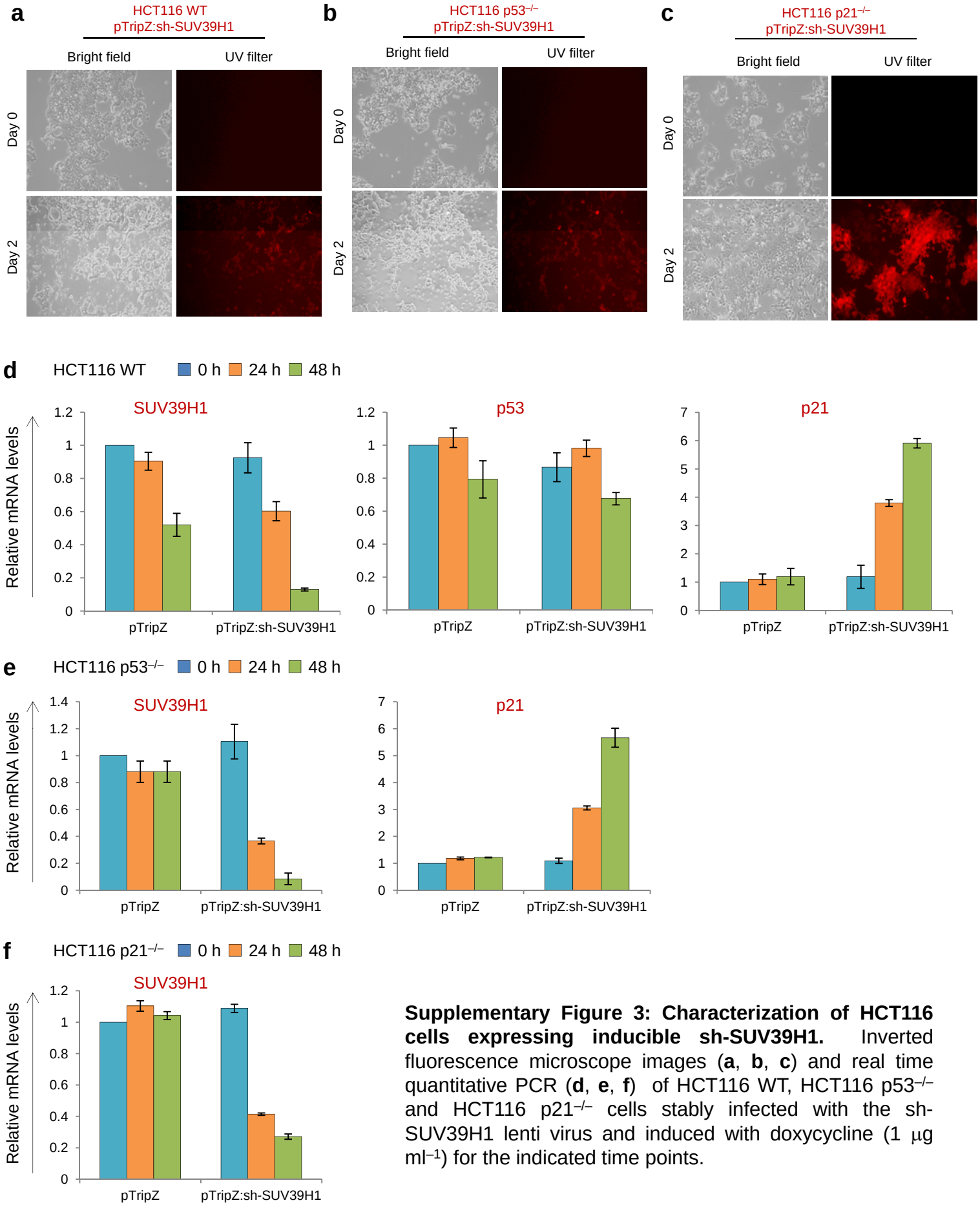
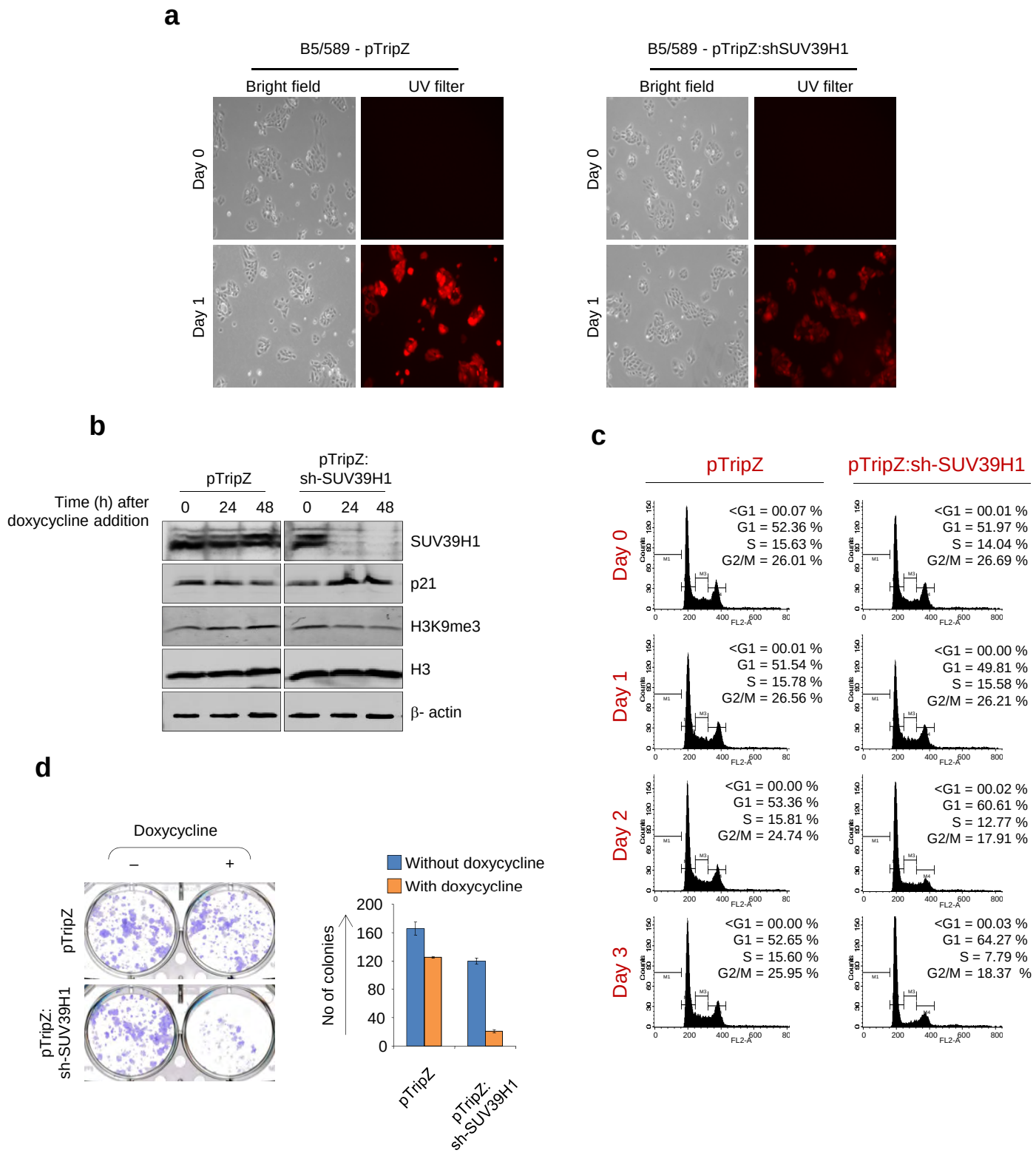
Supplementary Figure 2: Induction of p53 abrogates the H3K9me3 heterochromatin mark. (**a**) ChIP analysis in in B5/589 cells showing the occupancy of either H3K9me3 or p53 on p53 target promoters at the indicated time points after treatment with MI-219. The target sequences were detected by quantitative real-time PCR analysis of eluted DNA. The relative occupancy of either H3K9me3 or p53, over the % input is shown in the form of bar diagram. (**b**) Propidium iodide (PI) staining of HCT116 cells overexpressing SUV39H1 and treated with increasing doses of Nutlin3a as indicated. The % of cells which are in S phase of cell cycle is shown in the line diagram. (**c**) Western blot analysis of B5/589 cells overexpressing SUV39H1 treated with MI-219.

Supplementary Figure 3; Mungamuri et al., 2012



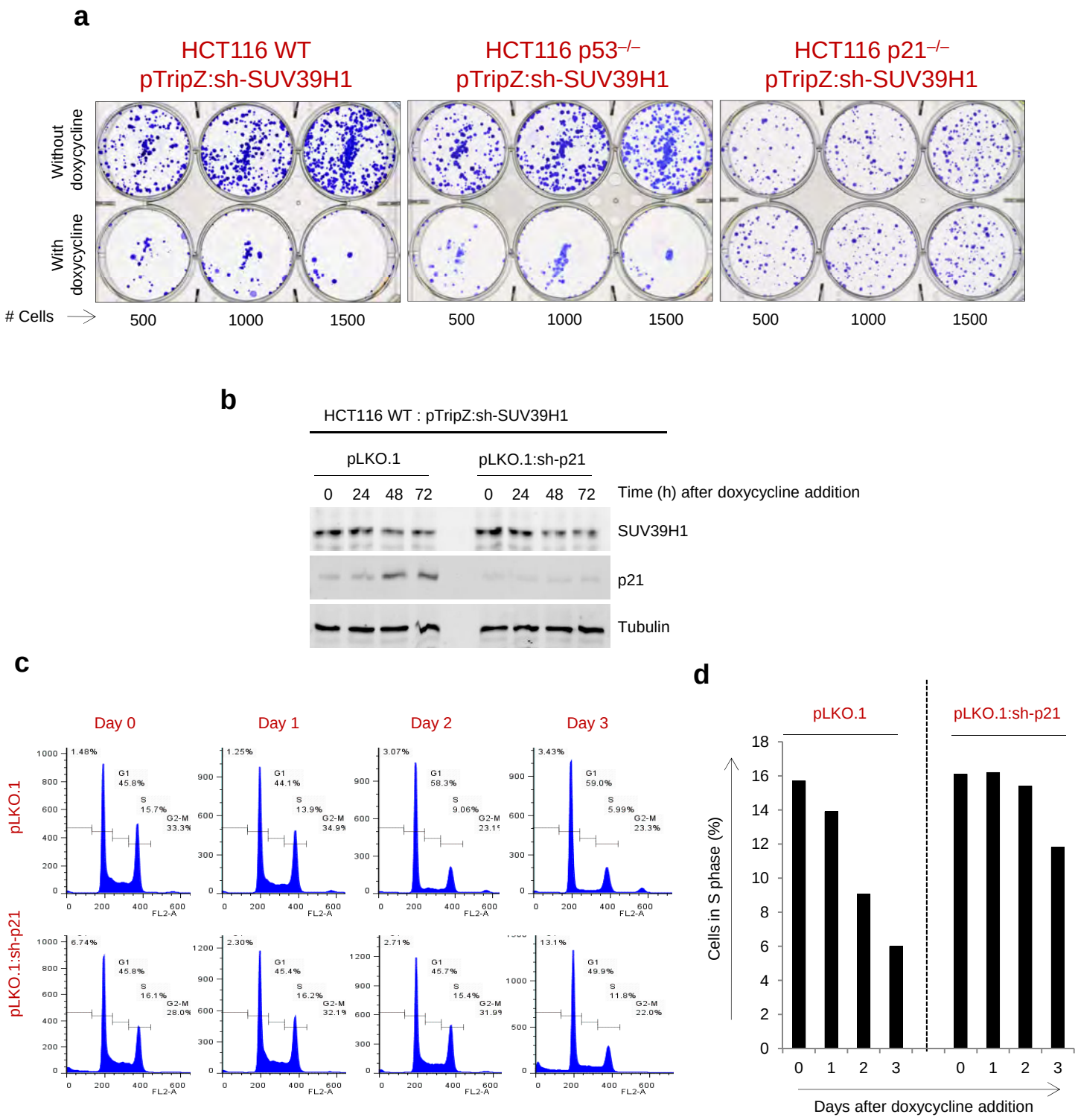
Supplementary Figure 3: Characterization of HCT116 cells expressing inducible sh-SUV39H1. Inverted fluorescence microscope images (**a**, **b**, **c**) and real time quantitative PCR (**d**, **e**, **f**) of HCT116 WT, HCT116 p53$^{-/-}$ and HCT116 p21$^{-/-}$ cells stably infected with the sh-SUV39H1 lenti virus and induced with doxycycline (1 μg ml^{-1}) for the indicated time points.

Supplementary Figure 4; Mungamuri et al., 2012



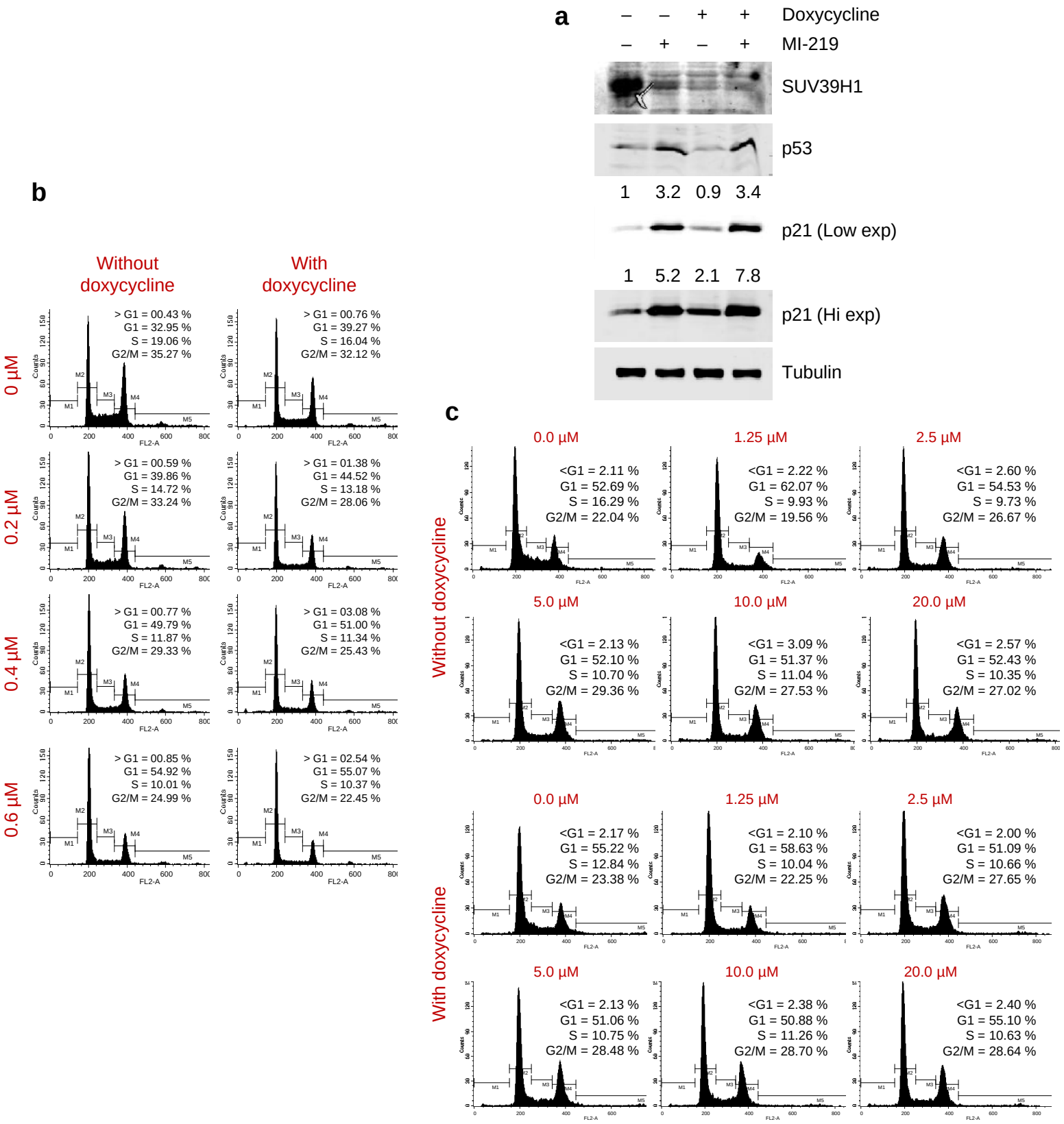
Supplementary Figure 4: Silencing of SUV39H1 induces p21 and cell cycle arrest. (**a**) Inverted fluorescence microscope images of B5/589 cells stably infected sh-SUV39H1 lenti virus and induced for 24 h to examine the expression of red fluorescent protein (RFP). (**b**) Western blot analysis of B5/589 cells stably expressing sh-SUV39H1 and induced for the indicated time points. (**c**) Cell cycle analysis by Propidium iodide (PI) staining in B5/589 cells stably transduced with inducible sh-SUV39H1. Day 0, 1, 2 and 3 represent time after addition of doxycycline into the medium. (**d**) Colony formation assay analyzing the effect of long term culturing of B5/589 cells stably transduced with sh-SUV39H1. The number of colonies formed after 9 days was counted and is shown in the form of a bar diagram.

Supplementary Figure 5; Mungamuri et al., 2012



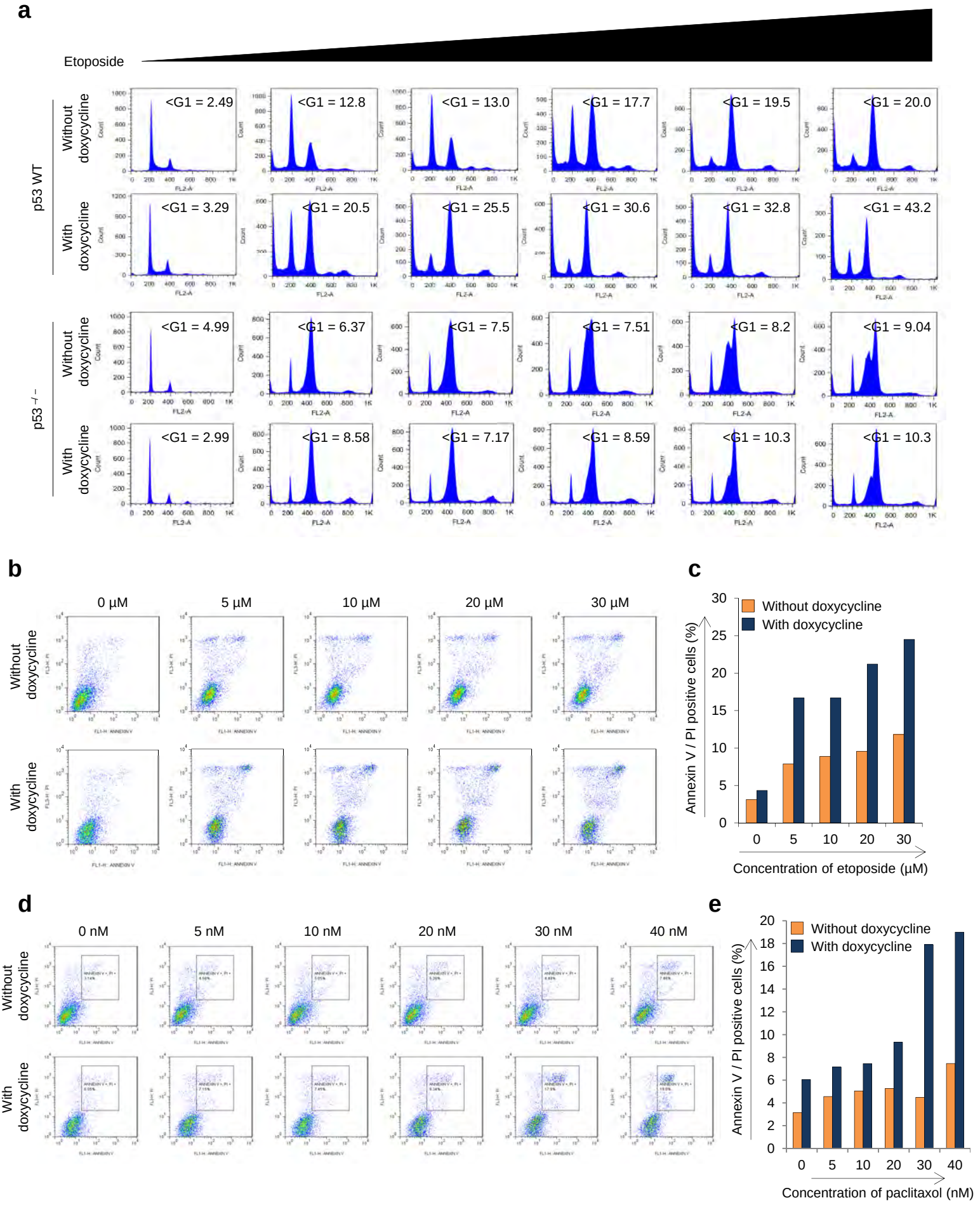
Supplementary Figure 5: p21 is required for sh-SUV39H1-induced colony suppression ability. (**a**) Colony formation assay analyzing the effect of long term culturing of HCT116 WT, HCT116 p53$^{-/-}$ and HCT116 p21$^{-/-}$ cells stably transduced with sh-SUV39H1. (**b**) HCT116 WT cells with pTripZ:sh-SUV39H1 alone or with sh-p21 were treated with doxycycline (1 µg ml^{-1}) for indicated time points and analyzed for the expression of the indicated proteins. (**c**) Propidium iodide (PI) staining of HCT116 WT cells with pTripZ:sh-SUV39H1 alone or with sh-p21 were treated with doxycycline (1 µg ml^{-1}) for indicated time points. The % of cells in S phase of the cell cycle is shown in (**d**).

Supplementary Figure 6; Mungamuri et al., 2012



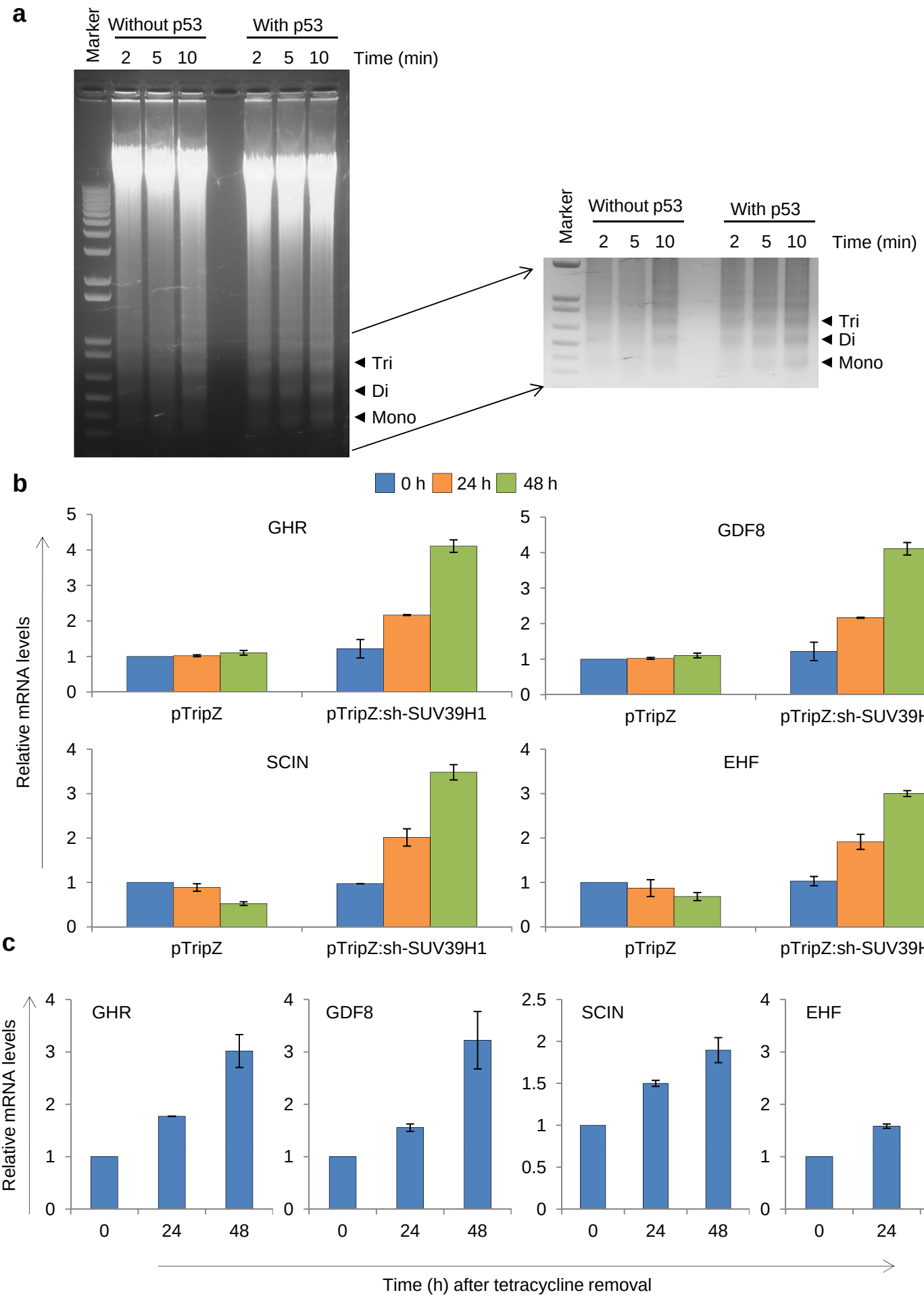
Supplementary Figure 6: SUV39H1 silencing does not cooperate with p53 in inducing cell cycle arrest. (**a**) Western blot analysis of indicated proteins in B5/589 pTripZ:sh-SUV39H1 cells. The cells were pre-silenced for SUV39H1 for 48 h before treating the cells with MI-219. (**b**) Propidium iodide (PI) staining of B5/589 pTripZ:sh-SUV39H1 cells. The cells were pre-silenced for SUV39H1 for 48 h before treating the cells with increasing doses of Nutlin3a. The % of cells in each phase of the cell cycle is also shown. (**c**) Propidium iodide (PI) staining of HCT116 WT pTripZ:sh-SUV39H1 cells. The cells were pre-silenced for SUV39H1 for 48 h before treating the cells with increasing doses of cisplatin. The % of cells in each phase of the cell cycle was also shown.

Supplementary Figure 7; Mungamuri et al., 2012



Supplementary Figure 7: Pre-silencing of SUV39H1 cooperates with chemotherapy induced apoptosis in a p53-dependent manner. (**a**) Cell cycle analysis by Propidium iodide (PI) staining in HCT116 WT and HCT116 $p53^{-/-}$ cells stably transduced with sh-SUV39H1. The cells were pre-silenced for SUV39H1 for 48 h before treating with increasing doses of etoposide. The percentage of cells showing less than a 2N content of DNA (apoptosis) in each condition is also shown. (**b, c**) Annexin V staining in HCT116 WT cells stably transduced with sh-SUV39H1. The cells were pre-silenced for SUV39H1 for 48 h and treated with increasing doses of etoposide (**b**) or paclitaxol (**d**). The % of cells that were double positive for Annexin V and PI in each condition is shown in the form of bar diagram in (**c**) and (**e**) respectively.

Supplementary Figure 8; Mungamuri et al., 2012



Supplementary Figure 8: p53 alters the global transcription through downregulating SUV39H1. (**a**) Micrococcal Nuclease (MNase) assay in EJ-p53 cells that were induced for p53 expression for 24 h. (**b**) Real-time quantitative PCR of B5/589 cells stably transduced with inducible sh-SUV39H1 and cultured in the presence of doxycycline for the indicated time points. (**c**) Real-time quantitative PCR of EJ-p53 cells induced for p53 for the indicated time points.

Supplementary Table 1; PCR primers used in this study

For Gene expression (Real time PCR)

Gene Name	Forward Primer	Reverse Primer
p53	CCCTTCCCAGAAAACCTACC	CTCCGTCATGTGCTGTGACT
p21	TGCCGAAGTCAGTTCCTTGT	CATGGGTTCTGACGGACATC
SUV39H1	ATATCCAGACTCAGAGAGCACC	CAGCTCCCTTTCTAAGTCCTTG
PUMA	GACCTCAACGCACAGTACGA	CACCTAATTGGGCTCCATCT
PIDD	GCTCTTGCTGTCTCACAACTG	AATCTCAGGAGGTAGCGTGTC
FAS	TCAGTACGGAGTTGGGGAAG	TCCTCAATTCCAATCCCTTG
MDM2	AAGCCTGGCTCTGTGTGTAA	CTGATCCAACCAATCACCTG
TP53INP1	TCTCATTGAACATCCCAGCA	TATGCTGCCCCATTTCATTT
APAF1	TGTCTGTCACCAGGGTACAGT	CGTTGTGGCCCCTCAATTCA
PIG3	CTCAGTACGTCACTGTCCCC	GATTAGCACATAGTCTCCAGCC
NOXA	CCGTGTGTAGTTGGCATCTC	GCACACTCGACTTCCAGCTC
GHR	GTCTGCAAAGTGTTAATCCAGGC	CTCTCGCTCAGGTGAACGG
GDF8	GACGATTATCACGCTACAACGG	TCCATAGTTGGGCCTTTACTACT
SCIN	ATGGCTTCGGGAAAGTTTATGT	CATCCACCATATTGTGCTGGG
EHF	CAGTGCAGTAGTGACCTGTTC	CTGTGCTACCATAGTTGGTGTC
18S	GTAACCCGTTGAACCCCATT	CCATCCAATCGGTAGTAGCG

For ChIP

Gene Name	Forward Primer	Reverse Primer
SUV39H1	GGCACAAATGGAAGCCAG	ACAGCGGCGGTAGCTAAA
PUMA	GCGAGACTGTGGCCTTGTGT	CGTTCCAGGGTCCACAAAGT
NOXA	CTCCTCTGCGGTGATTAAGG	GGGAAGGGTTTAACCAGGAG
p21	CTGGACTGGGCACTCTTGTC	CCCTTCCTCACCTGAAAACA
PIG3	TGCTCCGCGAGGATACAG	CCACCTTCAGGAGGACTTCA
TP53INP1	AAACCCTCGACCCTTCACTC	CGAGAGGTTGTCACCAACG
MDM2	TCGGGTCACTAGTGTGAACG	CACTGAACACAGCTGGGAAA
PIDD	CATCCAGAGTCCCTGTTTCC	CCGAATCCTCTGAAGCATCT
AChR	CCTTCATTGGGATCACCACG	GGAGATGAGTACCAGCAGGTTG