Supplemental Online Material

Supplemental Table 1

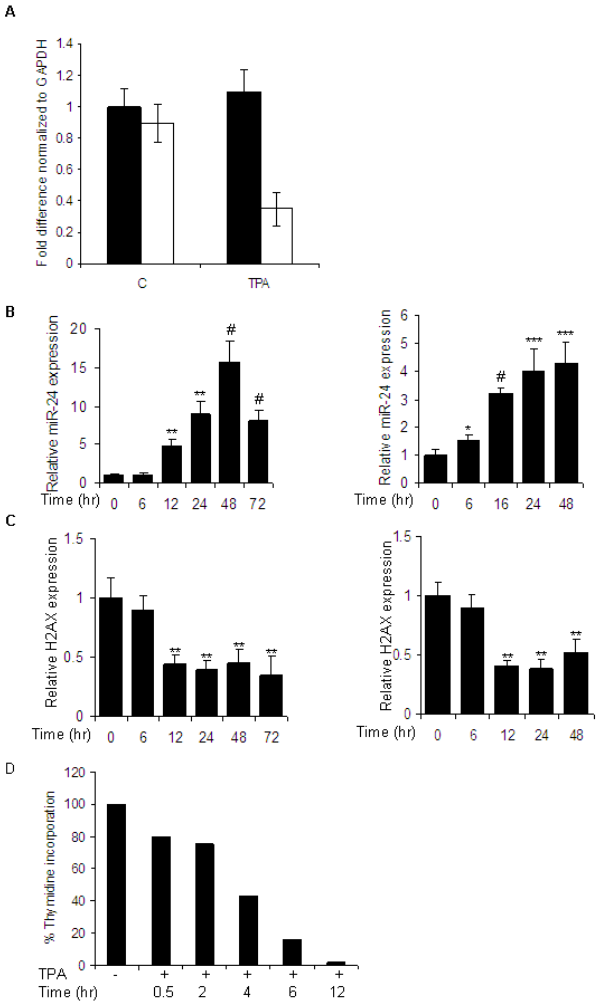
Gene	Forward Primer	Reverse Primer
GAPDH	TGCACCACCAACTGCTTAGC	GGCATGGACTGTGGTCATGAG
UBC	ATTTGGGTCGCGGTTCTTG	TGCCTTGACATTCTCGATGGT
H2AX (3'UTR)	AGCAAACTCAACTCGGCAAT	ACTCCCCAATGCCTAAGGTT
H2AX (coding)	GGCCTCCAGTTCCCAGTG	TCAGCGGTGAGGTACTCCAG

Supplemental Table 2

GENE	MRE	Location	
H2AX	MRE(1)	76 to 94 of H2AX 3'UTR sequence	5'UCAUGGAAAGAGCUGAGCC3' :: ::::::: 3'GACAAGGACGACUU--GACUCGGU5' 5'UCAUGGAAAGAGGAUUAGG3'
H2AX	MRE(2)	961 to 977 of H2AX 3'UTR sequence	5'CCUGUCUGGACUGAGCC3' :::: ::::::: 3'GACAAGGACGACUU-GACUCGGU5' 5'CCUGUCUGGAGAUUAGG3'

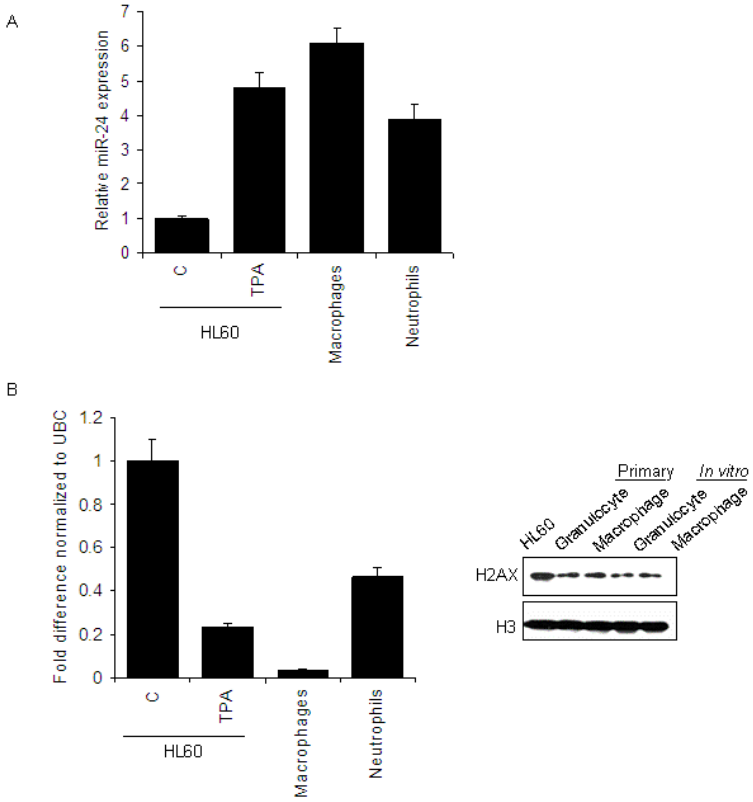
Supplemental Figures

Supplemental Figure 1



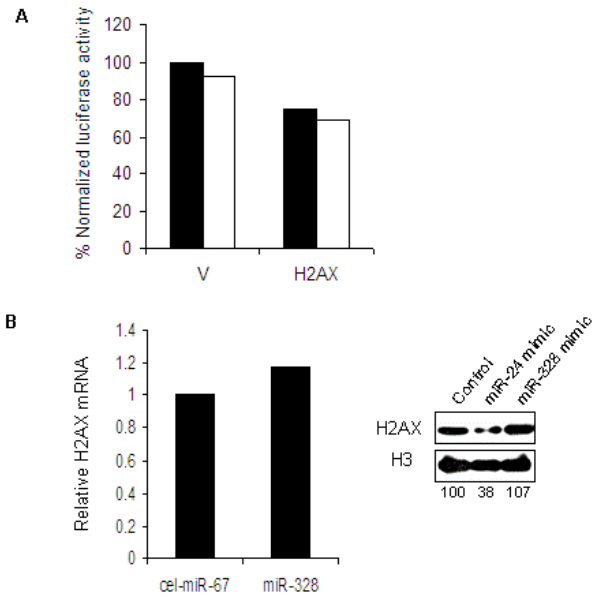
Supplemental Figure 1. A) H2AX mRNA analyzed by qRT-PCR using coding region primers decreases ~4-fold during TPA induced differentiation of K562 cells. These primers amplify both H2AX transcripts. GAPDH mRNA was used for normalization. B, C) Kinetics of miR-24 and H2AX mRNA expression in TPA-treated K562 and HL60 cells. TPA treatment of K562 cells (left panels) and HL60 cells (right panels) increases miR-24 expression (B) and concurrently decreases H2AX mRNA (C). D) Kinetics of thymidine incorporation in TPA-treated K562 cells. By 12 h there is no thymidine incorporation, indicating that cells have stopped dividing.

Supplemental Figure 2



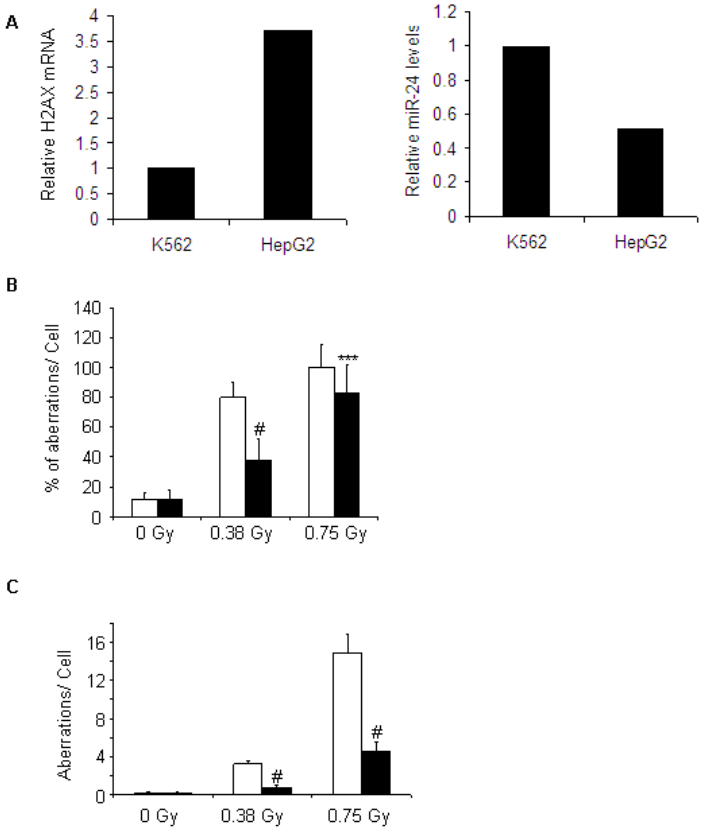
Supplementary Figure 2. miR-24 levels and H2AX levels in primary human peripheral blood monocytes and granulocytes are comparable to cells generated by *in vitro* differentiation. A) miR-24, analyzed by qRT-PCR relative to U6, increases during TPA-induced differentiation of HL60 cells to macrophages, and this increased miR-24 expression is also observed in primary human peripheral blood monocytes and granulocytes. B) H2AX mRNA (normalized to UBC mRNA), and protein (normalized to histone H3) is down-regulated during differentiation of HL60 cells and is comparable to levels in primary human peripheral blood monocytes and granulocytes.

Supplemental Figure 3



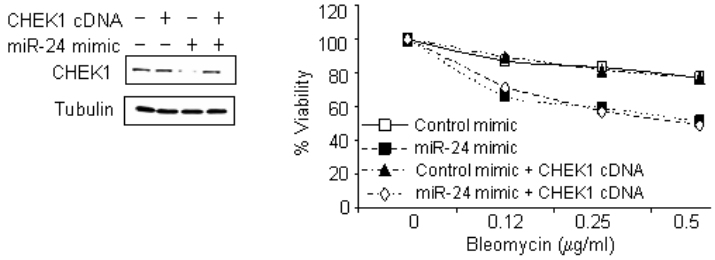
Supplemental Figure 3. A) miR-328 does not target the 3'UTR of H2AX mRNA in a luciferase reporter assay. HepG2 cells were transfected with control miRNA (black) or synthetic miR-328 (white) for 48 hr and then with H2AX 3'UTR-luciferase reporter (H2AX) or vector (V) for 24 hr. B) miR-328 over-expression in K562 cells has no effect on H2AX mRNA (left), analyzed by qRT-PCR normalized to GAPDH, and protein (right) 48 hr later.

Supplemental Figure 4



Supplemental Figure 4. Chromosomal aberrations after γ-irradiation are greater in K562 than HepG2 cells. HepG2 cells express less miR-24 (A, right panel) and more H2AX (A, left panel) than K562 cells. Cells were either untreated or irradiated and incubated for 24 h before metaphase spreads were prepared. For each condition, at least 50 metaphase spreads were examined. The average number of cells with chromosomal aberrations (B) and chromosomal aberrations per cell (C) were analyzed. In (B) and (C) white bars represent K562 cells; black bars, HepG2 cells.

Supplemental Figure 5



Supplemental Figure 5. miR-24-mediated hypersensitivity of K562 cells to bleomycin is not rescued by expression of miR-24-insensitive CHEK1. Cells were mock transfected or transfected with miR-24 mimic and/or CHEK1 cDNA lacking the 3'UTR. Cell viability was assayed 2 d after exposure to DNA damage and depicted relative to that of undamaged cells. Immunoblot demonstrates miR-24-mediated decrease in CHEK1 protein and its restoration by transfecting CHEK1 cDNA lacking the 3'UTR.