Supplemental Figure Legends

Figure S1 Time course of p53 and Chk2 phosphorylation in response to BrdU photolysis. (A) U87 cells were exposed to BrdU photolysis (UV-A dose of 1500 J/m^2) and cells collected for western blot analysis at the indicated times. β-actin denotes equal loading. **(B)** Densitometric analysis of (A) presented in graph form. *Data points*, fold changes in phosphorylation levels over time compared to control (no UV-A) after normalization to β-actin expression.

Figure S2 H2AX phosphorylation levels correlate with increasing DSBs. **(A)** U87 cells were exposed to BrdU photolysis with increasing doses of UV-A (no UV-A, lane 1; 125 J/m^2, lane 2; 250 J/m^2, lane 3, 500 J/m^2, lane 4; 750 J/m^2, lane 5; 1000 J/m^2, lane 6; 1250 J/m^2, lane 7; and 1500 J/m^2, lane 8), and collected after 1 h for western blotting. β-actin denotes equal loading. **(B)** Densitometric analysis of (A) presented in graph form. *Data points*, fold changes in phosphorylation levels over time compared to control (no UV-A) after normalization to β-actin expression.

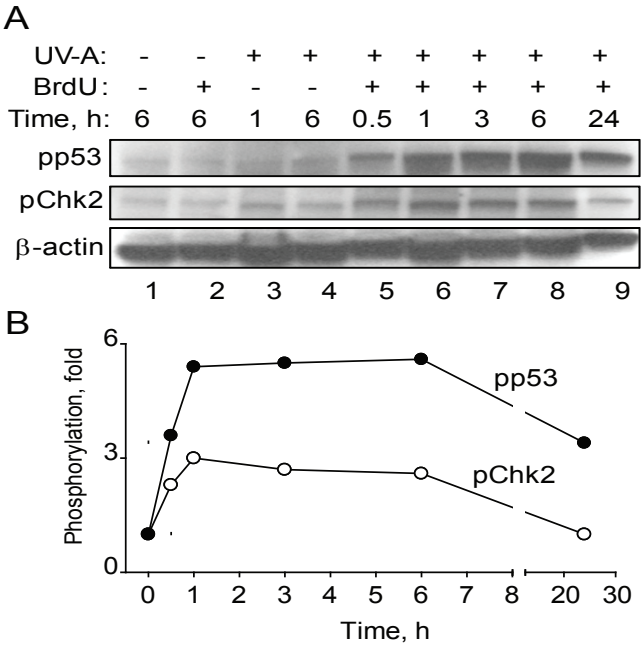
Figure S3 BrdU photolysis activates ATM. U87 cells were exposed to BrdU photolysis with increases doses of UV-A (0, 50, or 150 J/m^2). Cells were collected 3 h after irradiation and processed for western blotting with anti-(pS1981) ATM antibody. Parallel samples were immunoprecipitated with anti-ATM antibody prior to western blotting to confirm equal ATM expression levels.

Figure S4 BrdU-induced p53 and H2AX phosphorylation in MCF-7 cells. MCF-7 cells were exposed to BrdU photolysis with various doses of UV-A ([150 J/m^2 (lanes 1, 4, 7); 500 J/m^2 (lanes 2, 5, and 8); 1500 J/m^2 (3, 6, and 9); no UV-A (lane 10)]. Cells were collected at the indicated times and processed for western blotting. β-actin was used for normalization.

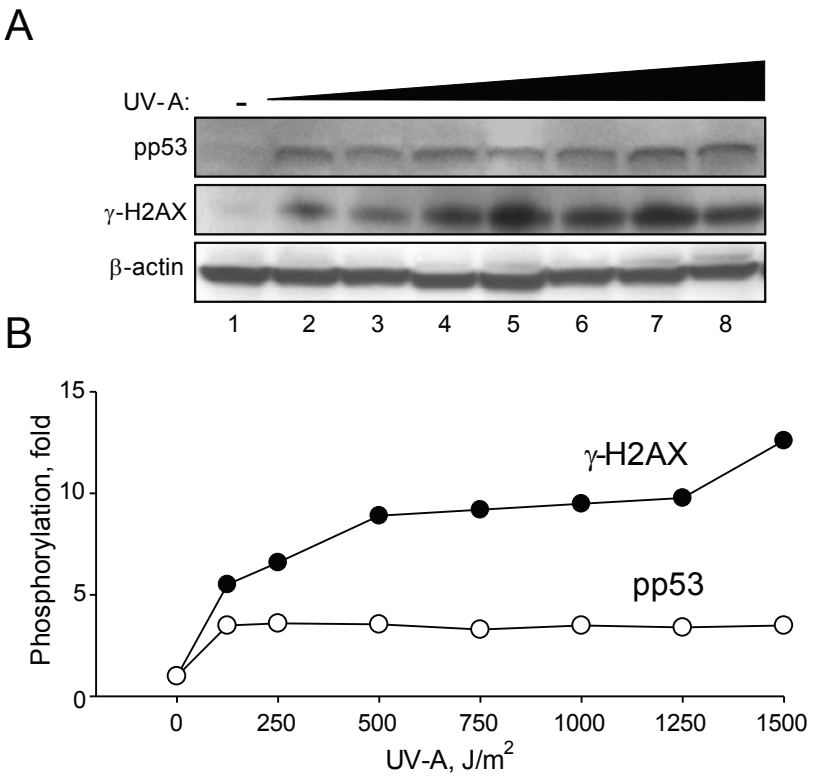
Figure S5 ERK phosphorylation is bi-phasic in response to DSBs and independent of p53. (A) U87 cells were exposed to BrdU photolysis with increasing doses of UV-A [0 J/m^2 (lane 1); 15 J/m^2 (lane 2); 50 J/m^2 (lane 3); 150 J/m^2 (lane 4); 500 J/m^2 (lane 5); and 1500 J/m^2 (lane 6)]. Cells were collected after 3 h for western blotting. β-actin denotes equal loading. **(B)** Densitometric analysis of (A) presented in graph form. *Data points*, fold changes in phosphorylation compared to control (no UV-A) after normalization to β-actin expression plotted against UV-A dose.

Figure S6 BrdU photolysis activates ERK via MEK. (A) U87 cells were treated with U0126 at 5 μM (lanes 4-6), or not (lanes 1-3) 30 min prior to BrdU photolysis with increasing doses of UV-A (Lanes 1 and 4, 0 J/m^2; 2 and 5, 50 J/m^2; 3 and 6, 150 J/m^2). Cells were collected 3 h after UV-A radiation for western blot analysis. ERK denotes equal loading. Figure shows representative images of triplicate repeats. **(B)** Densitometic analysis of (A) presented in graph form. *Data points*, ERK phosphorylation levels. *Error bars*, SEM; n = 3. Fold (x) denotes changes in p-ERK levels compared to control (no UV-A) normalization to ERK. *, $p < 0.05$.

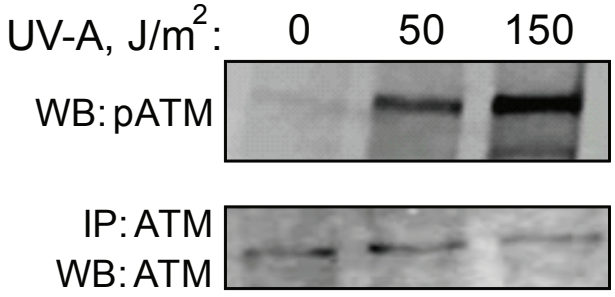
Figure S7 EcoRI-induced DSBs activate γ-H2AX in a time and dose-dependent manner. (A) HEK293 were electroporated with either buffer alone (lane 1- 4) or buffer with increasing units of EcoRI enzyme (10 units, lane 5-8; 50 units, lane 9-12; or 200 units, lane 13-16). Cells were collected at 2, 10, 30, and 60 min for western blotting analysis. ERK denotes equal loading. **(B)** Densitometric analysis of (A) presented in graph form. *Data points*, fold changes in γ-H2AX over time compared to control (no EcoRI) after normalization to ERK.



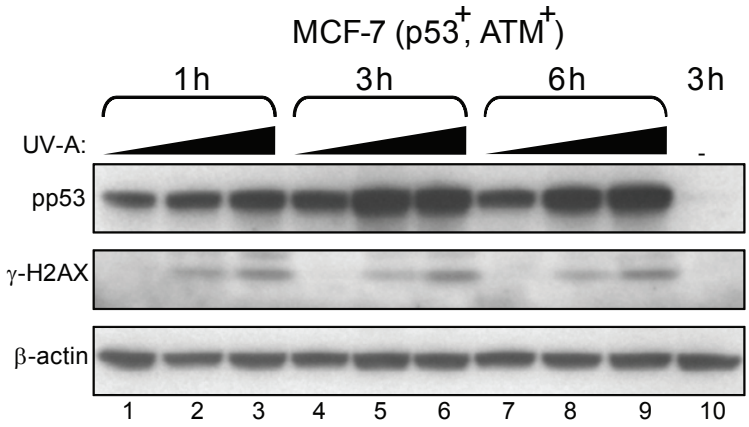
Khalil et al ., Supplemental S1



Khalil et al., Supplemental S2

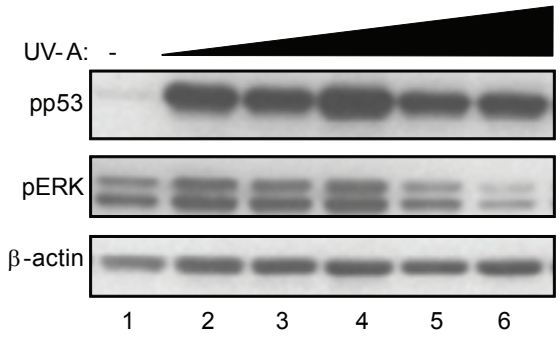


Khalil et al., Supplemental S3



Khalil et al., Supplemental 4

A



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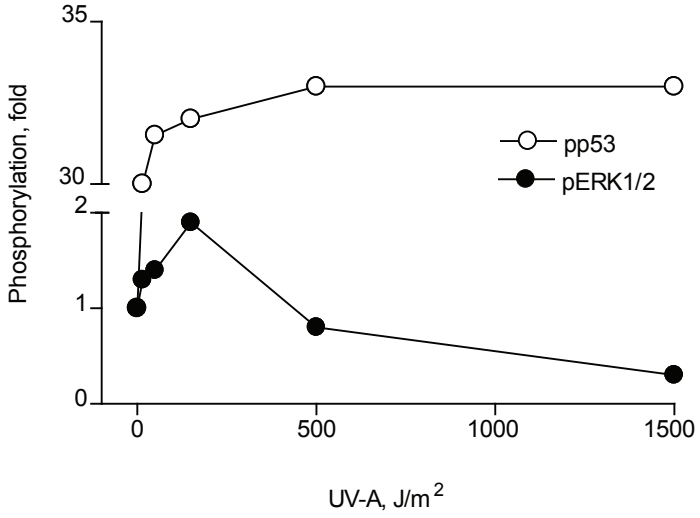
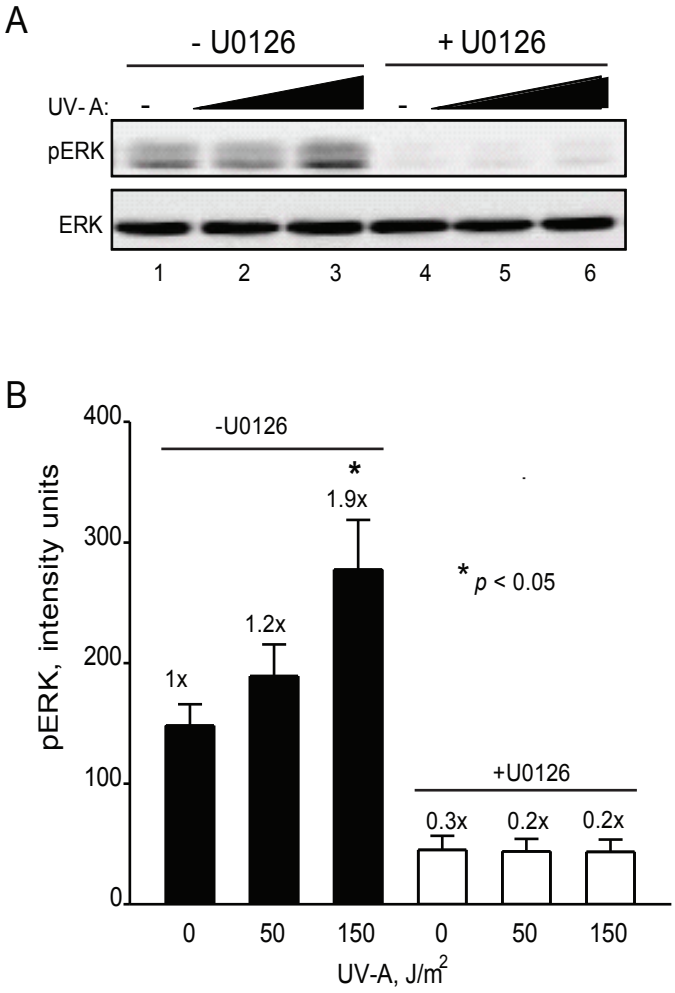
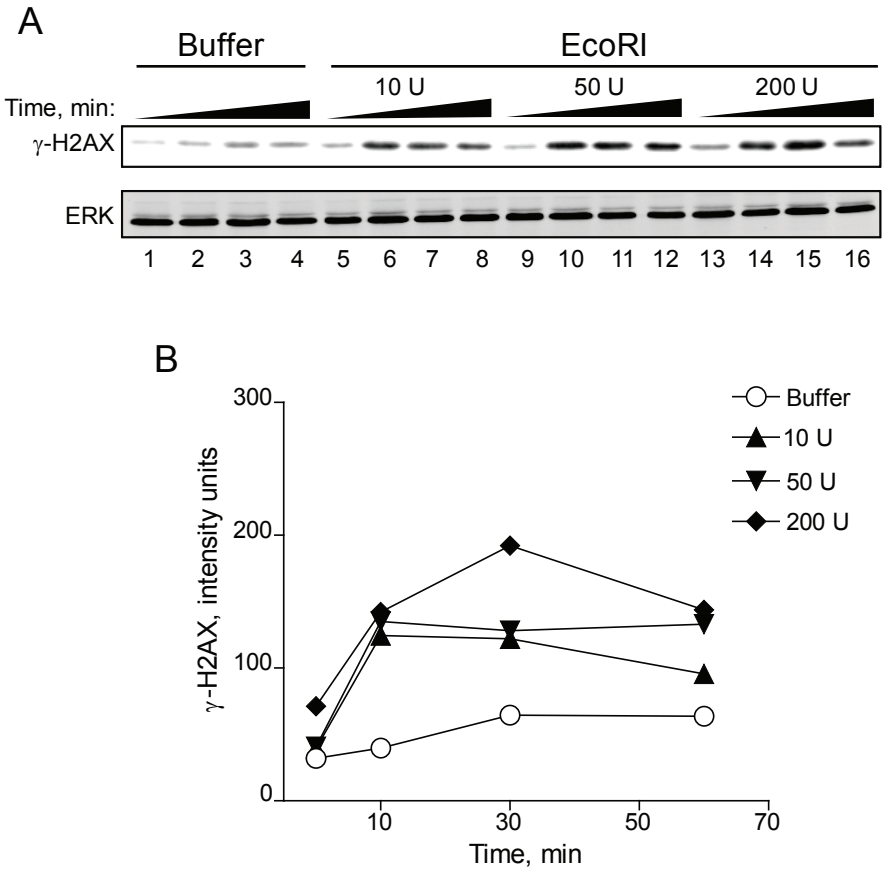


Khalil at al., Supplemental Fig. S5.



Khalil et al ., Supplemental Fig. S6.



Khalil et al., Supplemental Figure 7