

Supporting Information

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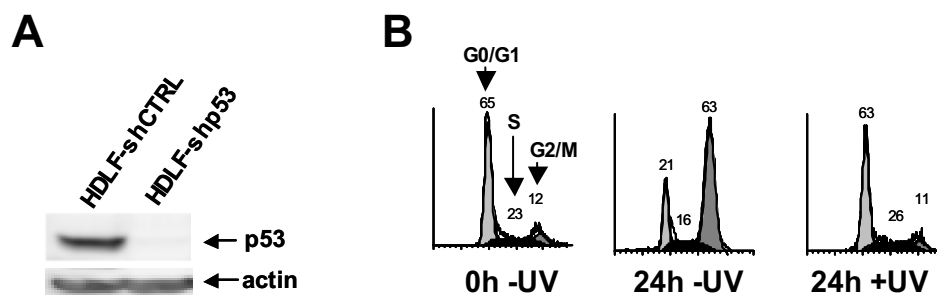


Fig. S1. (A) Knockdown of p53 in HDLFs as shown by Western analysis of p53 protein expression at 6 hours postirradiation with 25J/m^2 UV. (B) (Left panel) Cell cycle profile of normally cycling HDLF-shCTRL cells stained with PI; (middle and right panels) HDLF-shCTRL were either mock irradiated or irradiated with 25J/m^2 UV as indicated, and immediately re-fed with medium containing the mitotic inhibitor nocodazole (200 ng/ml) to block reentry of cells from G2/M into G0/G1. PI-stained cells were processed for cell cycle analysis and analyzed at 24 hours posttreatment. The numerals above each peak represent the percentage of the population in each phase at the time of analysis.

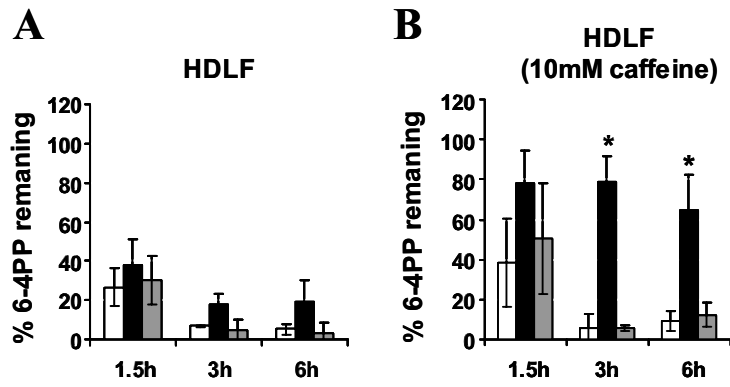


Fig. S3. Caffeine treatment abolishes GG-NER of 6-4PPs during S, but not during G0/G1 or G2/M, in HDLFs irradiated with 10J/m² UV. (A,B) Graphical depiction of 6-4PP repair in wild-type HDLFs treated or not treated with 10 mmol/l caffeine followed by irradiation with 10 J/m² UV. Mean ± SEM from four independent experiments is shown. *, *P* < 0.05; two-tailed paired *t* test (S phase relative to G1).

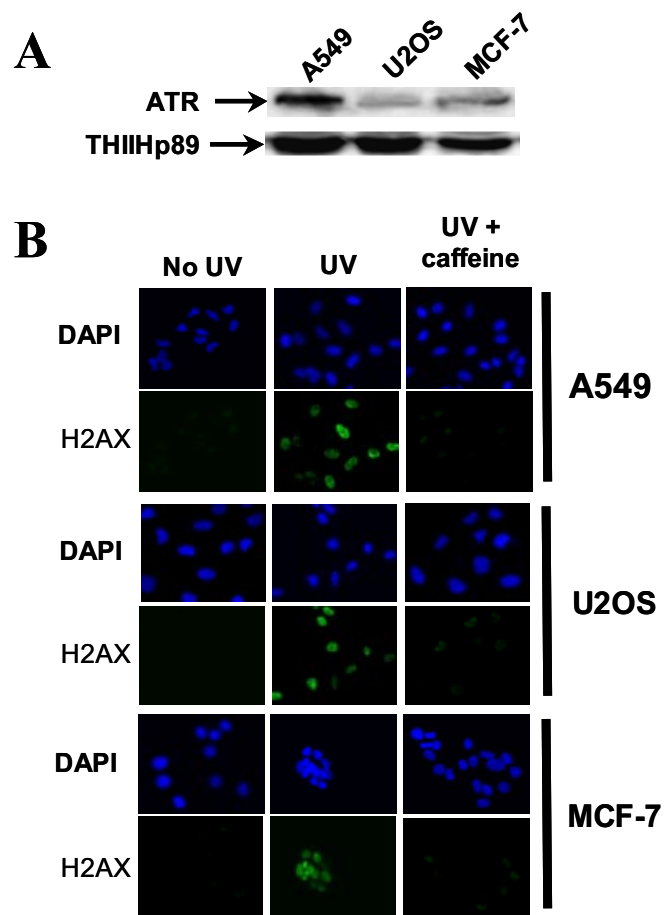


Fig. S4. (A) Expression of ATR protein in three model human tumor cell lines deficient in GG-NER during S phase, as determined by Western blotting. (B) Phosphorylation of H2AX in these model human tumor cell lines. As indicated, cells were mock irradiated, irradiated with 25 J/m² UV, or irradiated with 25 J/m² UV in the presence of 10 mmol/l caffeine. γ H2AX was detected at 2 hours post-UV, as described in Materials and Methods.