

Figure 4 - figure supplement 1

(A) Quantitation of SIF assay of RAD51 at HU stalled replication forks in p53 R248W and WT p53 HCT116 cells.

(B) Quantitation of SIF assay of RAD51 at HU stalled replication forks in p53 S47 and WT p53 H1299 cells.

(C) Representative images of SIF assay of RAD52 in HAP-1 cells at low (0.2mM) and high (4mM) HU concentrations show preferential binding of RAD52 at stalled forks with low HU concentrations compared to high HU concentrations, which are more favorable for break formation.

(D) Quantitation of SIF assay of RAD52 at HU stalled replication forks in p53 R175H, p53 R273H and p53 null Saos-2 sarcoma cells, and WT p53 U2OS sarcoma cells.

(E) Quantitation of SIF assay of RAD52 at HU stalled replication forks in p53 S47 and WT p53 H1299 cells. Bars represent the mean and the 95% confidence interval. Significance values are derived from student T-test analysis normalized to the respective EdU-PLA intensities (Table 1).

