SUPPLEMENTAL FIGURE LEGENDS

Figure S1. Effect of PARP1 and ATM inhibition on Brca1 recruitment

ChIP of Brca1 in MCF7 cells expressing ddl-Ppol as described in Figure 1. Cells were treated with (A) DMSO or 5 μ M PARP1 inhibitor olaparib (AZD2281), (B) DMSO or 5 μ M ATM inhibitor CP466722 for 1 h prior to ddl-Ppol induction.

Figure S2. Knock-down of Nbs1, Brca1 and Rap80

Immunoblot showing a knock-down of Nbs1, Brca1 and Rap80 in MCF7 cells. Cells were treated with either non-targeting control, Nbs1, Brca1 or Rap80 targeting siRNA.

Figure S3. Accumulation of MCF7 cells in G1 following serum starvation

Cell cycle analysis of MCF7 cells by propidium iodide staining after cultivation in medium containing either 10% FBS or 0.1% FBS for 24 h.

Figure S4. DSB repair in cycling cells depends on Brca1 and NBS1 but not RAP80

DNA repair measured by quantitative real-time PCR spanning the unique I-Ppol cleavage site at chromosome 1 in cycling MCF7 cells expressing ddI-Ppol that were cultivated in medium containing 10% fetal bovine serum for 24 h prior to DSB induction. Time indicated is hours following addition of 4-OHT. Data of three independent experiments are shown as mean +/- SEM.

Figure S5. RAP80 suppresses excessive DSB processing

ChIP of RPA32 in MCF7 cells expressing ddI-PpoI that were cultivated in medium containing 10% fetal bovine serum prior to DSB induction. Cells were transfected with either non-targeting control siRNA or RAP80 targeting siRNA. Data of two independent experiments are shown as mean +/- SEM.