**Supplementary Figures Legends**

**Supplementary Figure S1: Down regulation of BRG1 does not significantly affects cell cycle progression. (A)** Western blot analysis shows the efficient depletion of the indicated proteins by siRNA. DR-U2OS cells were transfected with the indicated siRNAs and cells were harvested 96h post-transfection. Whole cell lysates were prepared and SDS-PAGE was performed using the indicated antibodies. GAPDH was used as a loading control. Representative blots are shown. **(B)** Flow cytometry profiles are shown of DR-U2OS cells transfected with the indicated siRNA. Cell cycle analysis was performed 72h post-transfection by flow cytometry. Cell cycle progression was measured by DNA content using PI as a DNA marker. Representative histograms are shown.

**Supplementary Figure S2: Inactivation of BRG1 results in a DSB repair defect but does not affect ATM activation. (A) (B)** Comet assay analysis shows that inactivation of BRG1 results in a defect in the repair of DSBs induced by bleomycin (Bleo). Control cells (Cas9) or cells lacking BRG1 (BRG1-KO) were treated with bleomycin (20 μM) for 1h, the drug was washed off, and cells were allowed to repair the damage for 2h or 24h. Then, cells were lysed, subjected to cell electrophoresis, and their DNA was stained and images were acquired and comet tail moment was calculated using ImageJ and the Open comet plug-in. **(C)** Representative images of the comets at 2h after Bleomycin treatment are shown. **(D)** Comet assay analysis of Cas9 and BRG1-KO#2 cells treated with CPT, as on **(A)** and **(B). (E)** Representative images of control cells (Cas9) or cells lacking BRG1 (BRG1-KO#1) stained with an antibody against the phosphorylated histone H2AX are shown after no treatment (NT) or treatment with CPT (1 μM) for 1h (+CPT) or treated for 1h and allowed to recover for 2h (+CPT+2h). Quantification shown on Fig. 2D. The ratio of the average number of foci per cell of BRG1-KO cells/Cas9 cells is shown. **(F)** Cas9 and BRG1-KO#2 cells were treated with CPT for the indicated time periods to assess the activation of the ATR kinase. Whole cells lysates were prepared and the amount of phosphorylated Chk1 (pChk1) were measured. Levels of total Chk1 and vinculin were used as loading controls. A representative image is shown. **(G)** U2OS cells transduced with lentivirus expressing shRNAs against BRG1 (shBRG1) or non-targeting (shCTRL) were exposed to no treatment (NT) or ionizing radiation (IR, 5Gy) and harvested at the indicated times. Whole cells lysates were prepared and the amount of phosphorylated Chk1 (pChk1), phosphorylated Chk2 (pChk2), phosphorylated H2AX (γH2AX) were measured by western blot. Levels of total Chk1 and GAPDH were used as loading controls. A representative image is shown. **(H)** Quantification of the number of phosphorylated ATP (pATM, S1981) nuclear foci after bleomycin treatment. Control cells (Cas9) or cells lacking BRG1 (BRG1-KO) stained with an antibody against the phosphorylated ATM (pATM) are shown after no treatment (NT) or treatment with Bleo (20 μM) for 1h (+Bleo) or treated for 1h and allowed to recover for 30 min (+Bleo+30min), 2h (+Bleo+2h) or 4h (+Bleo+4h). **(I)** Representative images of control cells (Cas9) or cells lacking BRG1 (BRG1-KO) stained with an antibody against the phosphorylated ATM (pATM) are shown after no treatment (NT) or treatment with Bleo (20 μM) for 1h (+Bleo) or treated for 1h and allowed to recover for 30 min (+Bleo+30min), 2h (+Bleo+2h) or 4h (+Bleo+4h). Dashed line represents the DAPI-stained nuclei. All experiments were done in triplicate and the graphs represent averages +/- SD (\* p < 0.05, \*\* p < 0.01, \*\*\* p < 0.001, \*\*\*\* p < 0.0001 by student *t* test).

**Supplementary Figure S3: Inactivation of BRG1 impairs DNA end resection. (A)** Representative flow cytometry plots show the increase in chromatin-bound RPA after CPT treatment (0.5μM, for 1h). Quantification shown in Figure 3E. **(B)** Representative cell cycle profiles show that cells were synchronized by double thymidine block at G2 phase of the cell cycle. Control cells (Cas9) or cells lacking BRG1 (BRG1-KO) were treated with 2mM thymidine for 18h, then released in fresh media for 9h, followed by another thymidine block for 18h, and a final release for 8h. At this time, a large proportion of the cells were in the G2 phase of the cell cycle and were treated with CPT for 1h (1 μM).

**Supplementary Figure S4: Inactivation of BRG1 does not affect MRE11 foci-formation after DNA damage.** Inactivation of BRG1 does not impair MRE11 foci-formation after DNA damage. Control U2OS cells (Cas9) and U2OS cells lacking BRG1 (BRG1-KO) were grown on coverslips and were not treated (NT), or treated with CPT (+CPT, 1 M for 1h), and allowed to perform repair for 4h [32]. Cells were then incubated with pre-extraction and extraction buffers and fixed with formaldehyde. **(A)** Quantification of MRE11 foci intensity. MRE11 foci intensity was quantified using ImageJ in cyclin A positive cells (late S/G2 cells). Approximately 50 cells were counted by time point, per experiment. **(B)** Cells were immuno-labeled with antibodies against cyclin A and MRE11 (green). The DNA was stained using DAPI (represented by the dotted lines). Images were acquired using a confocal microscope (60X oil). Representative images are shown. **(C)** Control cells (Cas9) and cells lacking BRG1 (BRG1-KO) were transduced with a retrovirus expressing ER\*-HA-I-PpoI. These cells were treated for 12h with tamoxifen (2 M, + I-PpoI) or not (- I-PpoI), crosslinked and nuclear extracts were prepared. Chromatin immunoprecipitation was performed for histone H3 and **(D)** gH2AX. Quantitative PCR was performed to determine the amount of histone H3 present at the specific locus (489 bp 3’ to the I-PpoI cut site in the rDNA region). The % of input refers to the amount of DNA obtained from the immunoprecipitation of the given factor divided by the total amount of DNA (input). All experiments were done in triplicate and graphs represent averages of three independent experiments +/- SD (\* p < 0.05, \*\* p < 0.01, \*\*\* p < 0.001 by student *t* test).

**Supplementary Figure S5: Inactivation of BRG1 does not sensitize cells to the PARPi olaparib.** (A) Cas9 and BRG1-KO cells were grown in 6-well plates for 10-14 days in the presence of the indicated amounts of olaparib. After the incubation time cells were crosslinked and colonies were stained and counted using ImageJ. (B) Cells were treated with CPT (100 nM) or Olaparib (2.5 µM) for 72h and the activation of the caspase 3/7 pathway was measured by flow cytometry. Fold increase in caspase 3/7 activation was calculated by dividing the % activation of a given sample by the control (Cas9, NT). All experiments were done in triplicate and graphs represent averages of three independent experiments +/- SD (\* p < 0.05, \*\* p < 0.01, \*\*\* p < 0.001 by student *t* test).

**Supplementary Table S1: List of antibodies used in this study.**