Supplementary Figure legends

Supplementary Figure 1. Effects of siRNA knockdown of 53BP1 in MCF7 cells. MCF7 cells were treated with a control or 53BP1-specific siRNA. After incubation for the indicated time periods, 53BP1 and RAD18 protein levels were examined by Western blotting with α-53BP1 and α-RAD18 antibody, respectively. The expression levels of α-tubulin were used as a loading control.

Supplementary Figure 2. **(A)** The FACS analysis of wild-type (wt, shown as WT) and 53BP1-null cells (53BP1KO). The cell cycle profile of asynchronous or confluent WT and 53BP1-null MEFs was determined by FACS. **(B)** HeLa cells were synchronized in S, G2/M and G1 phase by a double thymidine block. The synchronization efficiency of the cells was verified by FACS analysis.

Supplementary Figure 3. Cell cycle-dependent interaction between RAD18 and 53BP1. HeLa cells were synchronized in S, G2/M or G1 phase by a single thymidine block. Then, the cells were exposed to X-rays (10 Gy), and cultured for 2 h. The nuclear extracts were prepared from the cells and were used for immunoprecipitation with anti-RAD18 antibodies (Abs) or control IgG. The 53BP1 protein levels were then examined by Western blotting using anti-53BP1 Abs.

Supplementary Figure 4. (A) Synchronous cell-cycle progression of G1-synchronized cells. Asynchronous (AS) and synchronous cells were pulse-labeled with BrdU for 5 min before being harvested. The harvested cells were fixed and stained with fluorescein isothiocyanate-conjugated anti-BrdU antibodies (Abs) and propidium iodide. Cells synchronized in G1 phase were released into the cell cycle at time 0 then pulse-labeled with BrdU at the indicated time points after cell-cycle release. The upper, lower-left, and lower-right gates in each panel correspond to cells in S, G1, and G2/M phase, respectively. The percentage of cells in each gate is indicated. **(B)** Flow cytometric analysis of G1-synchronized cells. The indicated cells were synchronized in G1 phase with mimosine after release from nocodazole treatment. After release from mimosine-induced arrest, cells were pulse-labeled with BrdU for 5 min. The cells were then harvested, fixed and stained with fluorescein isothiocyanate-conjugated anti-BrdU Abs and propidium iodide. The upper, lower-left, and lower-right gates in each panel correspond to cells in S, G1, and G2/M phase, respectively. The percentage of cells in

each gate is indicated.

Supplementary Figure 5. The kinetics of IR-induced Rad18 foci formation during G1 phase. Wild-type MEFs were cultured until confluent for synchronization at G1-phase. Cells were X-irradiated (2 Gy) in the presence (+PKi) or absence (-PKi) of DNA-PKcs inhibitor and cultured for the indicated time. The cells or non-irradiated cells (0 h) were fixed, and then the Rad18 foci were visualized by immunofluorescence staining using anti-hRAD18 antibody. The number of the cells with RAD18 foci was counted.