Supplemental Figure 1. A. Promoter analysis. HeLa cells were transfected with a pLuc-1430 survivin promoter-luciferase construct, synchronized at the indicated cell cycle phases or exposed to IR, and analyzed for β-galactosidase-normalized luciferase activity. Data are representative of one experiment out of at least two independent determinations. B. Mitotic targeting. MCF-7 cells were transfected with vector or p34^{cdk2} DN mutant, treated with or without IR, and analyzed by Western blotting.

Supplemental Figure 2. A. p53-independent release of mitochondrial survivin after IR. p53^{+/+} or p53^{-/-} HCT116 cells were treated with or without IR, and isolated mitochondrial fractions were analyzed by Western blotting. B. Chk2 requirement for mitochondrial release of survivin in p53^{+/-} cells. p53^{-/-} HCT116 cells were transfected with control or Chk2 siRNA, treated with or without IR, and analyzed by Western blotting. C. Bax-independent modulation of survivin after IR. Bax^{+/-} HCT116 cells were transduced with control GFP or Chk2-DN adenovirus, treated with or without IR, and analyzed by Western blotting.

Supplemental Figure 3. A. Mitochondrial membrane potential. WT HCT116 cells transfected with control or Chk2 siRNA were treated with or without IR, labeled with the mitochondrial membrane potential-sensitive dye JC-1, and analyzed for changes in red/green fluorescence ratio by flow cytometry. The percentage of cells in each quadrant is indicated. B. Cytochrome c release. WT HCT116 transfected with control or Chk2 siRNA were treated with or without IR and analyzed by Western blotting.

Supplemental Figure 4. p53-independent apoptosis. p53^{-/-} or p53^{+/-} HCT116 cells were transduced with Chk2-DN, treated with or without IR, and analyzed by multiparametric flow cytometry. The percentage of cells in each quadrant is indicated.

Supplemental Figure 5. Conditional expression of Chk2-DN. WT HCT116 cells were stably transfected with Chk2-DN under the control of a tet-regulated promoter (tet-on system). Two independent clones (#101, #102) were cultured with or without dox, and analyzed for expression of Chk2-reactive material by Western blotting.

Supplemental Figure 6. Histology. Tumor samples from the indicated treatment groups were excised, formalin-fixed and analyzed by H&E. Magnification, x100.

Supplemental Figure 7. Conditional expression of recombinant Chk2-DN, in vivo. Frozen tumor sections from mice treated with or without dox were stained with IgG or FITC-conjugated antibody to HA. DNA was stained with DAPI. Magnification, x200.
Supplemental Figure 2
Supplemental Figure 3
Supplemental Figure 4