SUPPLEMENTAL INFORMATION

Supplemental Figure Legends

Figure S1. Modulation of p53-RPA binding is dependent in ATM and ATR. HCT-116 $^{ATR-/-}$ cells were synchronized in S phase and treated with 10 μM ATM inhibitor (Ku55933) for 1 hour prior to CPT treatment (10 μM for 2.5 hrs.) Whole cell lysates were subjected to DNase I digestion and 5% of the sample was loaded as input. IP was subsequently performed using anti-p53 antibody and co-immunoprecipitated proteins were analyzed by Western blotting with the indicated antibodies.

Figure S2. Hyperphosphorylation of RPA promotes RPA-Rad51 interaction. Stable U2-OS WT or PD cells were synchronized in S phase with APH, followed by treatment with 10 uM CPT for 3 hrs. Chromatin was isolated and incubated with DNase I. Subsequently, samples were incubated with anti-p53 antibody and co-immunoprecipitated proteins were analyzed by Western blotting (lanes 3-4). The supernatant after IP was immunoprecipitated again using Rad51 antibodies and analyzed (lanes 5-6).

Figure S3. Effects of ATR, ATM, Chk2 and Chk1 on p53-RPA interaction. A549 cells were transfected with siRNAs targeting ATM, ATR, Chk1, Chk2 or combinations. Then, cells were treated with 10 uM CPT for 3 hrs to induce RPA phosphorylation. Whole cell lysates were prepared and analyzed by Western blotting to confirm silencing (bottom). Nuclear lysates were isolated and treated with DNase I. Samples were incubated with anti-p53 antibodies and co-immunoprecipitated proteins were analyzed by Western blotting.