MDC1 cleavage by caspase-3:
a novel mechanism for inactivating the DNA damage response during apoptosis

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Supplementary Material
**Supplemental Figure 1. MDC1 cleavage following treatment with various anticancer agents in cancer and normal cells.**

**A**, Jurkat cells were treated with Fas antibody (0.1 μM) for the indicated times. MDC1 and γ-H2AX were analyzed by Western blotting. Tubulin was used as loading control. **B**, HCT116 cells were treated with camptothecin (CPT, 25 μM, 28 h) or cisplatin (CDDP, 50 μM, 28 h). MDC1 and γ-H2AX were analyzed by Western blotting. Tubulin was used as loading control. **C**, Prostate Epithelial cells (PrEC) were treated with camptothecin (CPT, 25 μM, 72 h). MDC1 was analyzed by Western blotting. GAPDH was used as loading control.

The percentage of cells in sub-G1 is indicated at the bottom of each panel. The different bands observed for MDC1 represent alternatively spliced forms. Electrophoretic migration of molecular mass markers (kDa) is indicated to the right side of each panel.
Supplemental Figure 2. List of the potential caspase-3 cleavage sites in the MDC1 sequence that were tested as caspase-3 sites in in vitro caspase cleavage assay.

The amino acid sequence (P4-P1) and the position of the aspartate (D) in P1 that was replaced by an alanine (A) are indicated. Except for the D173A mutation (in bold), all the other mutations had no impact on caspase-3-mediated cleavage.