Expanded View Figures

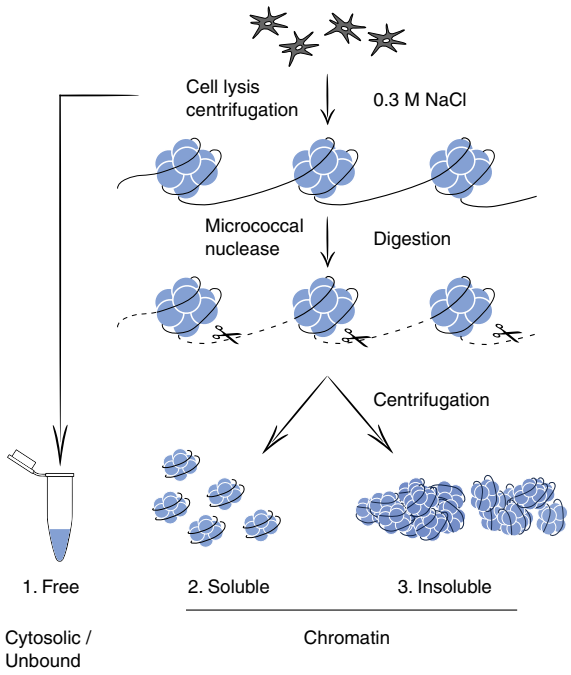

Figure EV1.

Figure EV1. Chromatin fractionation.

Flow diagram illustrating the chromatin dissection after removal of unbound or loosely bound (free) proteins by salt extraction (0.3 M NaCl). MNase digestion removes linker DNA, thus generating a supernatant of solubilized chromatin proteins and a remaining insoluble pellet strongly enriched in nucleosome cores.

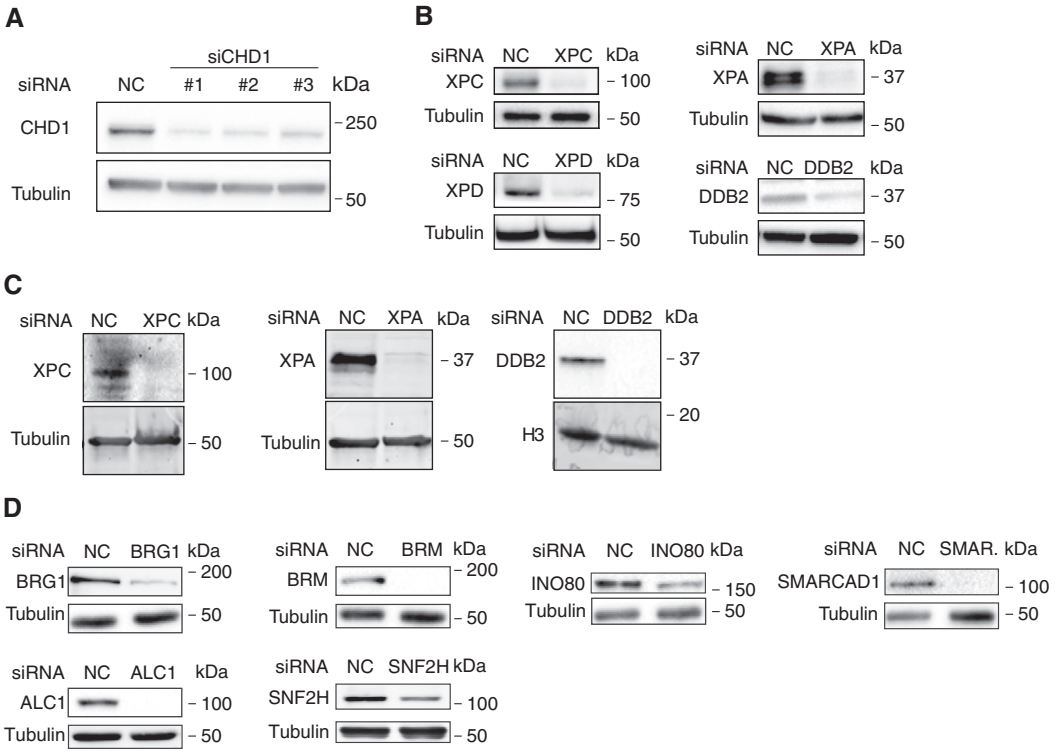

Figure EV2. Efficiency of siRNA-mediated protein depletions.

A Analysis of CHD1 protein levels after depletion with three different siRNA sequences (16 nM) directed against the CHD1 transcript. Immunoblots of HeLa cell lysates were carried out 2 days after transfection. Tubulin served as the loading control.

B Protein depletion achieved in U2OS cells 2 days after transfection with the indicated siRNA sequences (16 nm).

C Protein depletion achieved in HeLa cells 2 days after transfection with the indicated siRNA sequences (16 nm). Tubulin and histone H3 served as loading controls.

D Protein depletion achieved in HeLa cells 2 days after transfection with siRNA sequences (16 nM) against the indicated chromatin remodelers. Tubulin served as the loading control.

Source data are available online for this figure.

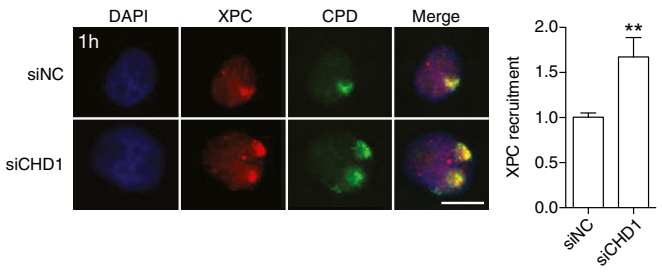

Figure EV3. XPC accumulation in the chromatin of UV-irradiated HeLa cells.

Representative immunofluorescence images of HeLa cells that were UV-irradiated (dose applied to filters: 100 J/m^2) through micropore filters to generate local spots of DNA damage. Immunostaining was carried out after 1 h with antibodies against CPDs and XPC protein. Cells were pretreated 2 days earlier with siRNA targeting the CHD1 transcript (siCHD1) or with non-coding control RNA (siNC). DAPI was used to stain nuclear DNA. Scale bar: 10 μm. The recruitment of XPC protein was quantified by measuring spot intensities followed by normalization to the nuclear background. Control values were set to 1. Data are presented as mean ± SEM (n = 3, 100 cells for each experiment). **$P \leq 0.01$ (unpaired, two-tailed t-test).

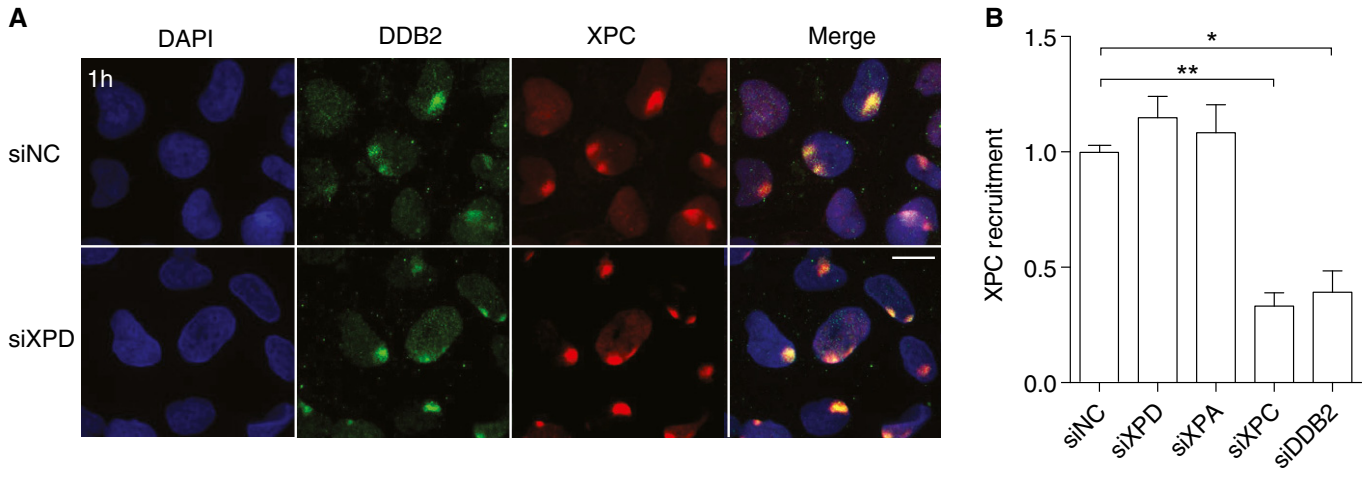

Figure EV4. XPC recruitment to local spots of UV damage.

A Representative immunofluorescence images of U2OS cells that were UV-irradiated (dose applied to filters: 100 J/m^2) through micropore filters to generate local spots of DNA damage. Immunostaining was carried out after 1 h with antibodies against DDB2 (as a marker of UV lesions) and XPC proteins. Cells were pretreated 2 days earlier with siRNA targeting the XPD transcript (siXPD) or with non-coding control RNA (siNC). DAPI was used to stain nuclear DNA. Scale bar: 10 μm.

B The recruitment of XPC protein was quantified by measuring spot intensities followed by normalization to the nuclear background. Control values were set to 1. Cells were pretreated 2 days earlier with siRNA targeting the XPD, XPA, XPC, or DDB2 transcripts, as indicated, or with non-coding control RNA (siNC). Data are presented as mean ± SEM (n = 3, 100 cells for each experiment). *$P \leq 0.05$, **$P \leq 0.01$ (unpaired, two-tailed t-test).

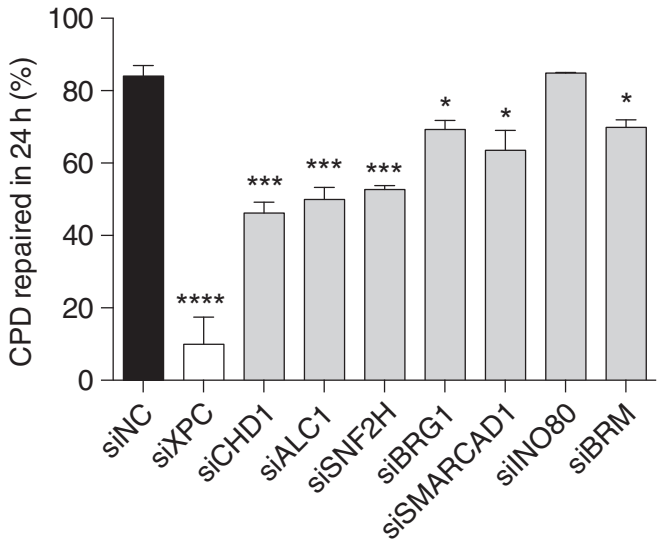

Figure EV5. Comparison between chromatin remodelers.

Excision of CPDs in HeLa cells treated with siRNA (16 nM) targeting the indicated chromatin-remodeling factors, compared to transfection with siNC. The cells were UV-irradiated (10 J/m^2) 2 days after siRNA transfections, and the proportion of excised CPDs was determined after a repair incubation of 24 h. The efficiency of protein downregulation is shown in the immunoblots of Fig EV2A and D. Data are presented as mean ± SEM (n = 5–7 independent experiments with four replicates). ****$P \leq 0.0001$, ***$P \leq 0.001$, *$P \leq 0.05$ (unpaired, two-tailed t-test).