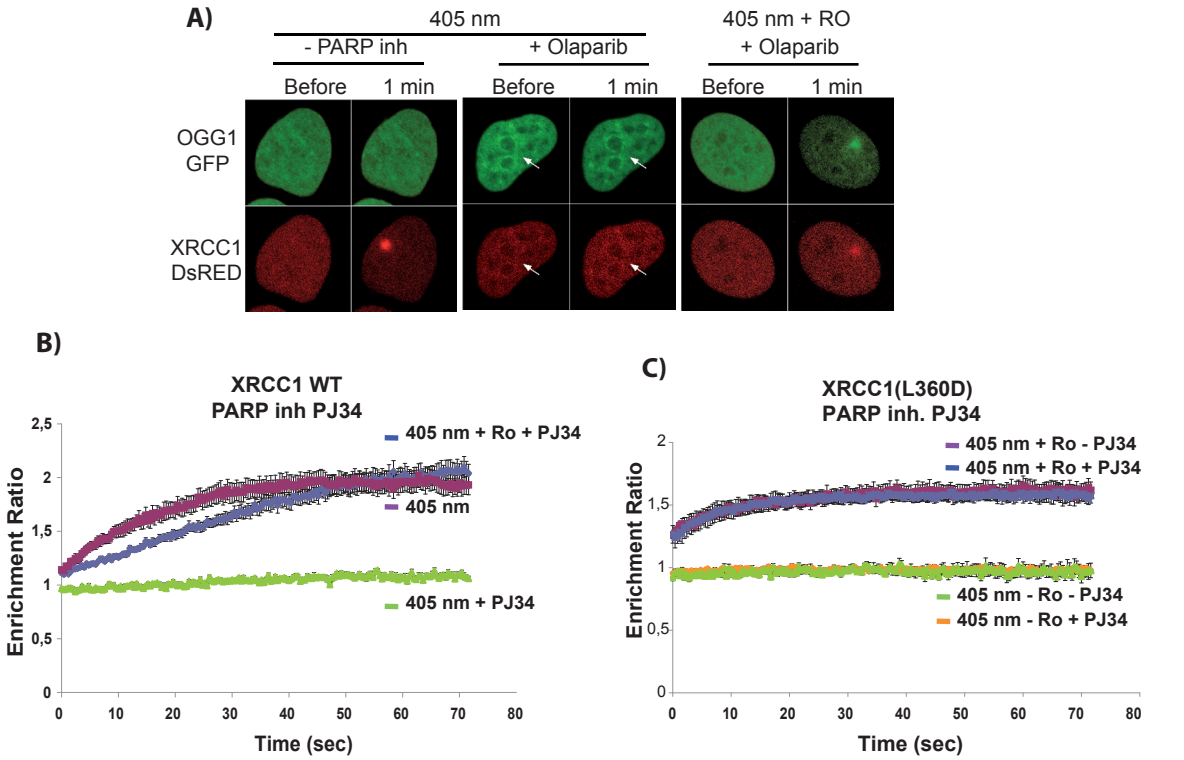
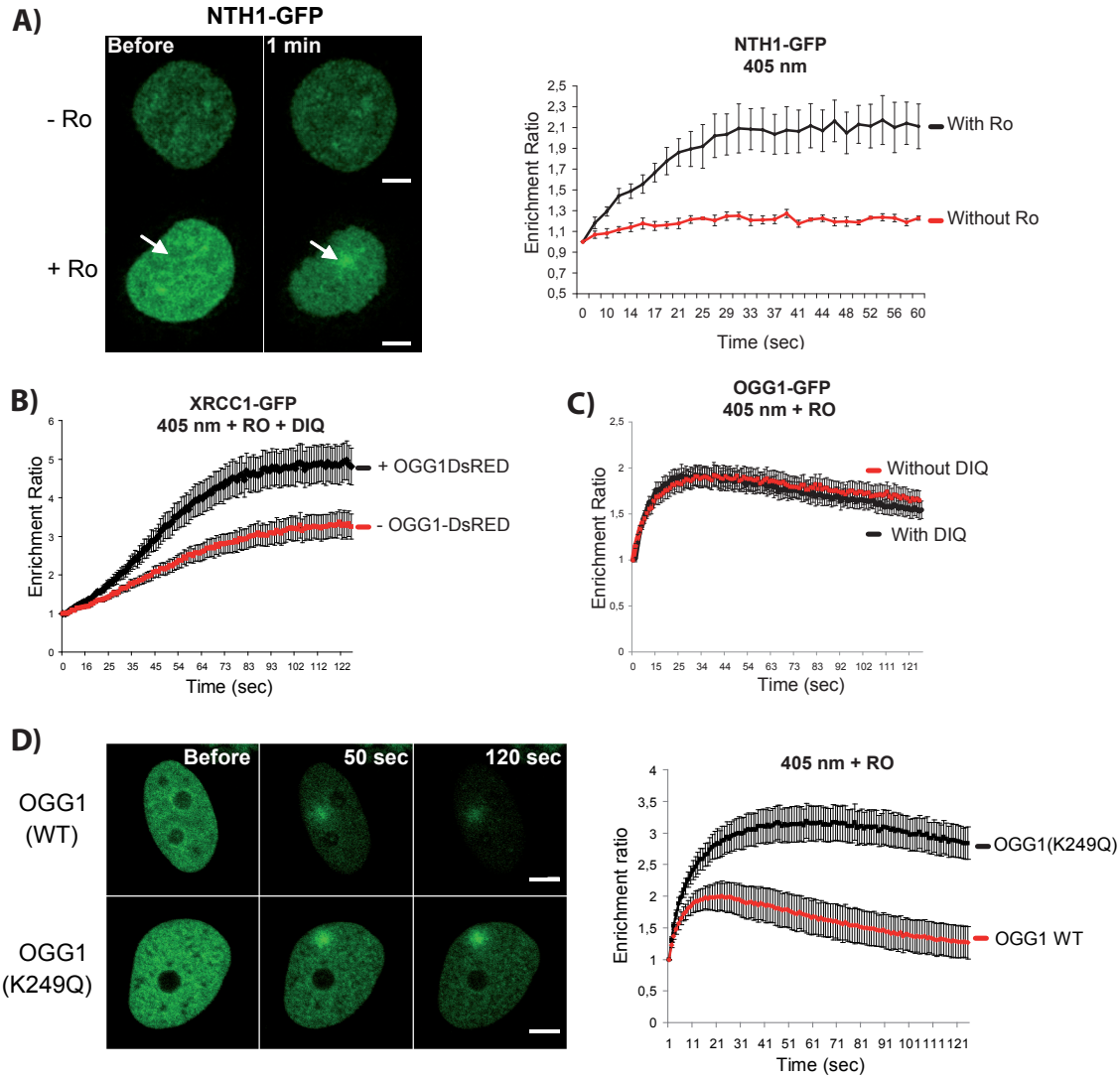
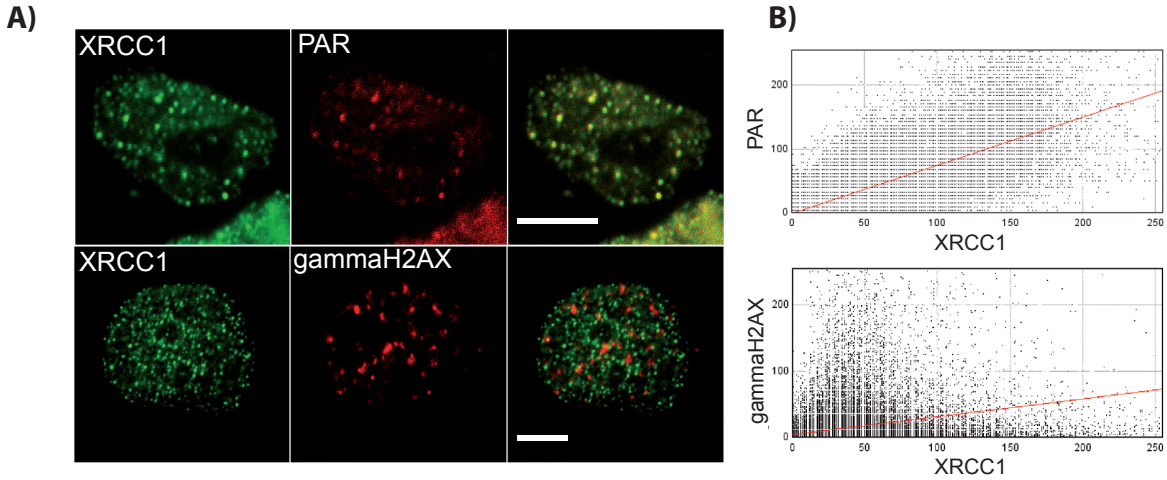


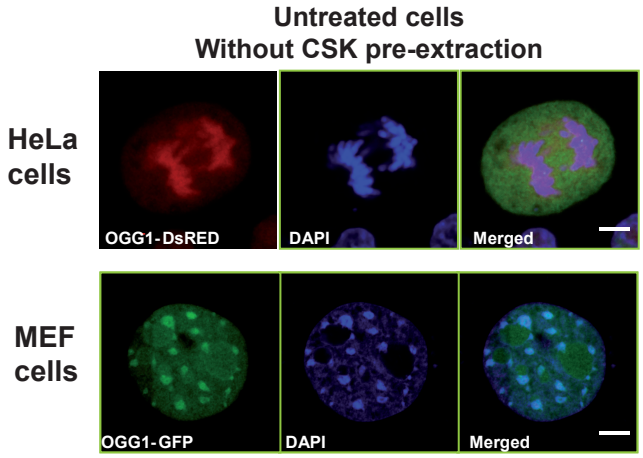
Supplementary Figure 1. PARP activity is not required for recruitment of XRCC1 to laser microirradiation-induced 8-oxoG. **(A)** The presence of the PARP inhibitor Olaparib in the medium abolishes recruitment of XRCC1-GFP to DNA damage sites induced by the 405 nm laser (mainly SSB). When Ro is added to the medium before microirradiation, resulting in the formation of oxidative damage, recruitment of XRCC1 is observed even in the presence of Olaparib. Scale bar, 5 μM. **(B)** Recruitment of XRCC1-GFP in living cells after microirradiation with the 405 nm laser (mainly inducing SSBs). XRCC1 accumulated very rapidly in the microirradiated region and its recruitment was abolished by the presence of the PARP-inhibitor PJ34. When microirradiation was done in the presence of the photosensitizer Ro 19-8022 (Ro, generating 8-oxoG), XRCC1 was recruited to the site of damage even in the presence of PJ34. (**C**) The BRCTI mutant XRCC1(L360D), unable to interact with PARP1, was not recruited after microirradiation with the 405 nm laser, while it was recruited after induction of oxidative damage (405 nm laser microirradiation in the presence of Ro). The presence of PARP inhibitor PJ34 in the medium had no impact in its recruitment. For B and C, microirradiation experiments were performed in a different set-up at the department of Genetics in Rotterdam. In this case, the system developed to induce a local oxidative DNA damage was built on a Zeiss LSM 510 using a continuous wave 405 nm laser light illumination. Damage was induced by applying several hundred iterations with the 405 nm laser diode to a narrow strip (80/120 x 10 pixels) and intensity of fluorescence was monitored every 100 ms at 1.5% laser intensity (argon laser 488 nm).



Supplementary Figure 2. Recruitment of OGG1 and XRCC1 to microirradiation-induced oxidative damage **(A)** The DNA glycosylase NTH1-GFP accumulates in the microirradiated region only in the presence of the photosentitizer. **(B)** The overexpression of OGG1-DsRED increases the recruitment of XRCC1-GFP to the site of oxidative DNA damage. Microirradiation with the 405 nm laser was performed in the presence of the photosentitizer Ro and the PARP inhibitor DIQ. **(C)** Recruitment of OGG1 to oxidative damage is not affected by PARP inhibition. After addition of the photosensitizer Ro19-8022 (Ro) to the medium, microirradiation with the 405 nm laser was done in the presence or absence of the PARP inhibitor DIQ. **(D)** The active site OGG1 mutant K249Q accumulated and remained longer at the microirradiated regions compared to the wild type protein. Graphs represent the mean of 10 cells, error bars represent the SEM. Scale bar, 5 μm.



Supplementary Figure 3. Co-localisation of XRCC1 foci with SSB but not DSB markers **(A)** Fast assembled XRCC1 foci partially co-localise with the PAR polymer (Pearson coefficient r=0.798) and but not with γH2AX (Pearson's coefficient r=0.365). Scale bar, 5 μm. **(B)** Correlation between green and red signals are presented in the cytofluorograms.



Supplementary Figure 4. Localisation of OGG1 in highly compacted chromatin in the absence of CSK pre-extraction. During HeLa cells' mitosis, OGG1-DsRED can be detected in chromosomes. In MEF cells, OGG1 is clearly enriched in chromocenters. No OGG1-GFP signal remained after the removal of soluble proteins. Scale bar, 5 μm.