

## Supplementary figures and tables

### Supplementary Figure 1 Characterization of the mutants used in this study.

Primers were designed to flank the presumptive deletions in the genetic loci or the coding regions of *rad-54(ok615)* (a), *xpg-1(tm1670)* and *xpg-1(tm1682)* (c) mutants. Genomic DNA or total mRNA from wild-type (wt) animals and the corresponding mutants was used in a PCR or RT-PCR reaction. In each case, a picture of the genomic PCR amplification around the locus (left panel) and a picture of the transcript PCR detection (right panel) are shown. 18S rRNA was used as an internal control for the presence of transcripts. Below, the gene structures are illustrated and the location where the mutations map are depicted by bars and the corresponding allele name.

The *ok615* allele is a combined insertion/deletion that removes about 1 kb of *rad-54*, including the start codon and resulting in the absence of mRNA transcript. The *tm1268* allele deletes 811 bp in the middle of the gene and results in an out-of-frame fusion of exon 5 to exon 8. Thus, both mutations likely are null.

The *tm1670* deletion removes the second and part of the third exon of *xpg-1*, resulting in a truncated mRNA. The *tm1682* deletion removes the first two exons of *xpg-1*, the whole promoter region and part of the 3'UTR of the upstream gene (*tre-1*; F57B10.7). Because we could not detect any *xpg-1* mRNA in *tm1682* mutants, and in the *tm1670* mutant a region important for the endonuclease activity is missing, we suspect that both mutants are null.

(b) Rescue of the increased germ cell apoptosis in *rad-54(ok615)* mutants in transgenic animals carrying a *Prad-54::rad-54::yfp::3'UTR* construct (*opIs257*). Germ cell corpses were scored 24 h post the L4/adult molt. Data shown represent the average  $\pm$  SD of two independent experiments (n>15 animals for each experiment).

**Supplementary Figure 2** Increased apoptosis caused by loss of RAD-51 is mediated by ATL-1 but not ATM-1.

(a) *rad-51(lf)* animals induce apoptosis dependent on *atl-1*. Germ cell corpses were scored every 12 h until 48 h post L4/adult molt in staged control (RNAi), *rad-51(RNAi)*, *atm-1(gk186)*, *atl-1(tm853)*, *atm-1;rad-51(RNAi)* and *atl-1;rad-51(RNAi)* animals. Data shown represent the average  $\pm$  SD of two independent experiments (n>15 animals for each experiment).

**Supplementary Figure 3** UV-C induces formation of RAD-51 foci, independently of XPA-1 and RAD-54.

(a) Fluorescent microscopy of mid-late pachytene germ cell nuclei from staged wild-type (wt) young adult hermaphrodites stained with an anti-RAD-51 antibody, 3 h after exposure to 100 J/m<sup>2</sup> of UV-C. (b, c) Quantification of the RAD-51 foci shown in (a) in mid-late pachytene germ cell nuclei from wild-type, *xpa-1(ok698)* and *atm-1(gk186)* (b), or *rad-54(ok615)* (c) young adult animals, 3 h following treatment with 120 Gy of X-rays or 100 J/m<sup>2</sup> of UV-C. Data shown represent the average  $\pm$  SD of two independent experiments (n $\geq$ 15 animals for each experiment). (d) Increased levels of apoptosis in *rad-51(RNAi)* animals remain unaffected by *xpa-1* or *xpg-1*. Germ cell apoptosis was scored 12 and 24 h post L4/adult molt in staged: wild-type (wt), *xpa-1(ok698)*, *xpg-1(tm1670)*, *rad-51(RNAi)*, *xpa-1(ok698);rad-51(RNAi)* and *xpg-1(tm1670);rad-51(RNAi)* animals. Data shown represent the average  $\pm$  SD of two independent experiments (n>20 animals for each experiment).

**Supplementary Figure 4** RPA-1::YFP foci are formed shortly after treatment with UV-C or X-rays.

Staged wild-type young adults were treated with either 100 J/m<sup>2</sup> UV-C (a) or 120 Gy X-rays (b) and the mid-late pachytene germ cell nuclei were scored for the presence of foci 0.5 h, 1.5 h and 3.5 h later. Data shown represent the average of 15-20 gonads  $\pm$  SEM.

**Supplementary Figure 5** *xpa-1* and *xpg-1* mutants display normal potential for germ cell apoptosis.

(a) Germ cell apoptosis was scored 12 and 24 h post L4/adult molt in staged wild-type (wt), *xpa-1(ok698)*, *xpg-1(tm1670)*, *ced-9(RNAi)*, *xpa-1(ok698);ced-9(RNAi)* and *xpg-1(tm1670);ced-9(RNAi)* animals. Data shown represent the average  $\pm$  SD of two independent experiments (n>15 animals for each experiment). (b) For the egg laying rate, wild-type, *xpa-1(ok698)*, *xpg-1(tm1670)*, and *rad-54(ok615)* worms were irradiated at L4 stage (approx. 12 h prior to L4/adult molt) with 100 J/m<sup>2</sup> UV-C or left untreated, and 24 h later were left to lay eggs in groups of 5 for 6 h. *xpa-1* and *xpg-1* mutants showed somatic defects after UV, possibly a result of their inability to repair their damaged genomes. The reduction in the egg laying rate could thus be a secondary effect. Data represent average number of eggs laid per worm per hour  $\pm$  SD of 6 replicates.

**Supplementary Table 1** The loss of XPA-1 function results in apoptosis via activation of other signaling pathways.

(a) Increased levels of apoptosis in *xpa-1* mutants are suppressed by *hus-1* and *atl-1*. Germ cell apoptosis was scored 12, 24 and 36 h post L4/adult molt in staged: wild-type (wt), *xpa-1(ok698)*, *hus-1(op244)*, *atl-1(tm853)*, *hus-1(op244) xpa-1(ok698)* and *xpa-1(ok698);atl-1(tm853)* animals.

**Supplementary Table 2** Primers for the generation of the RNAi constructs and the translational reporters used in this study.