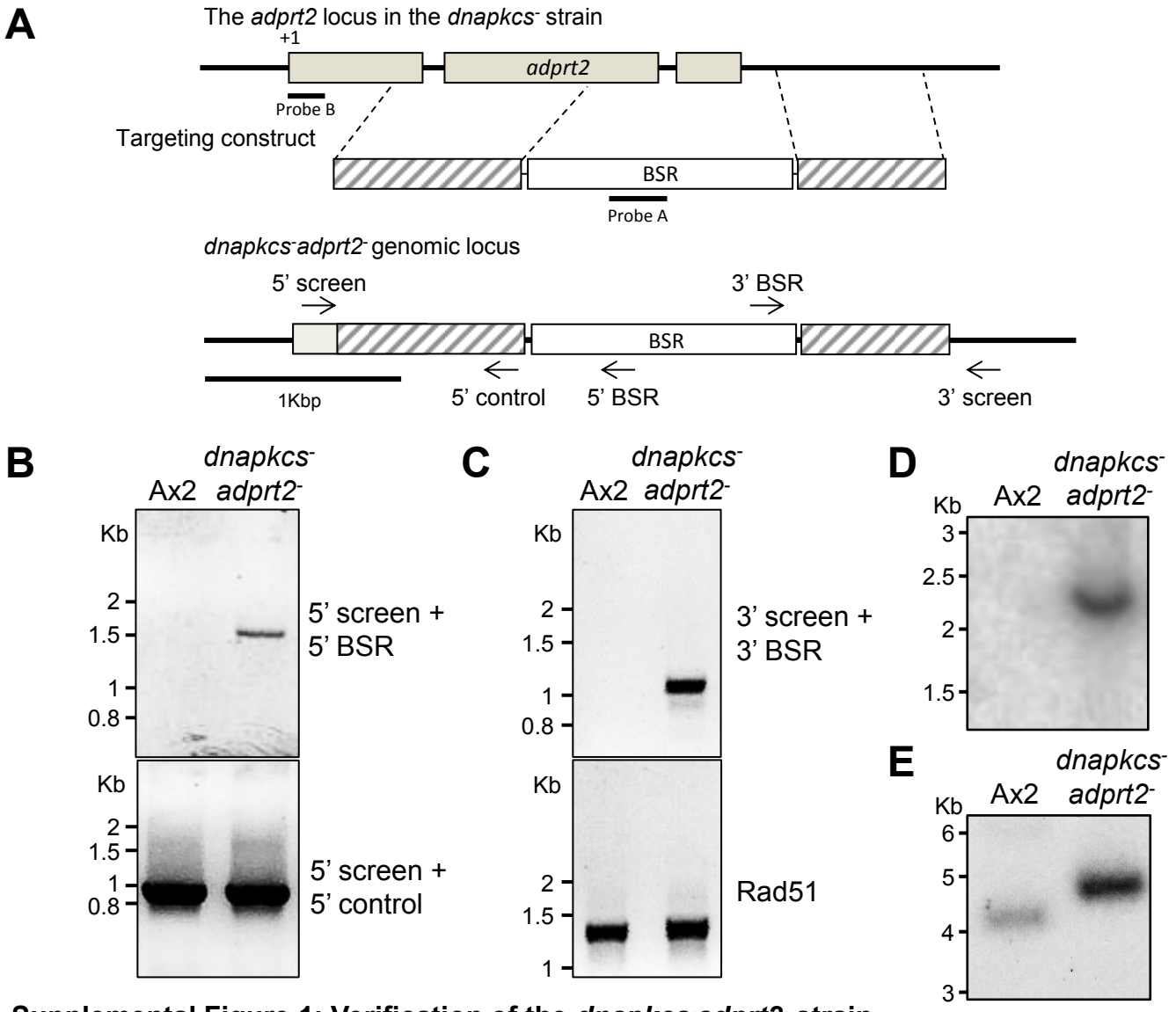
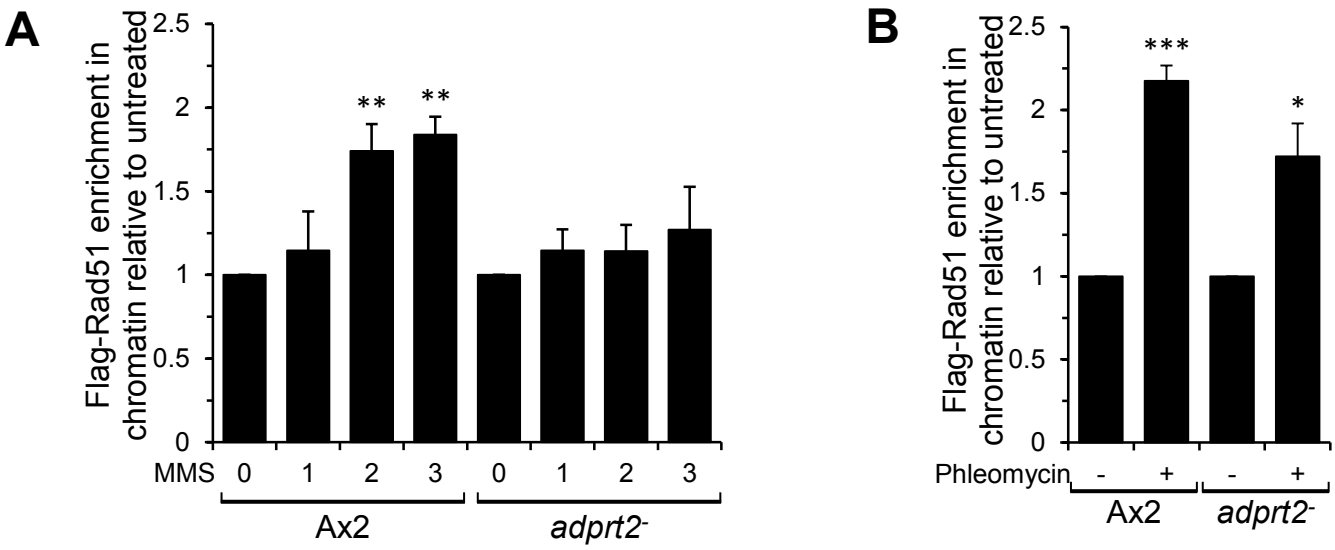


Supplemental Figure 1: Verification of the *dnapkcs*$^-$*adprt2*$^-$ strain

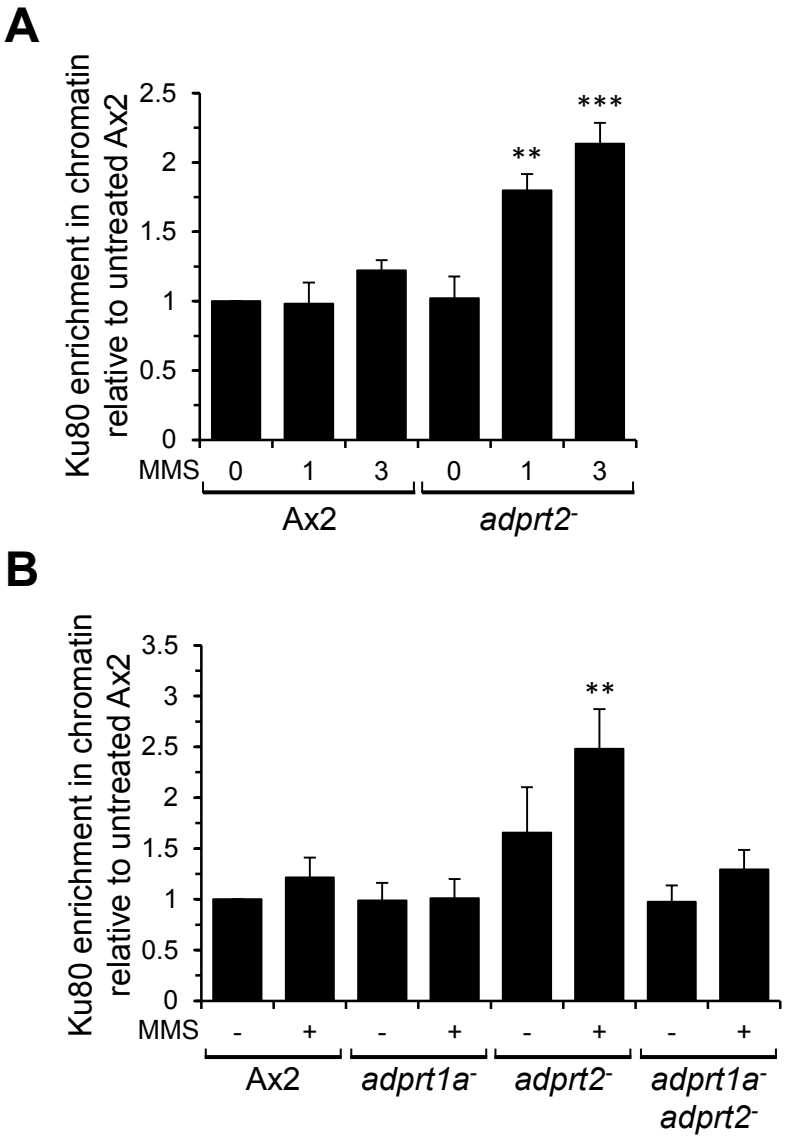
A. Map of the *adprt2* locus in the parental *dnapkcs*$^-$ strain (upper panel) and *dnapkcs*$^-$*adprt2*$^-$ disruption strain is illustrated. Hatched boxes represent regions of homology used in the disruption construct. The disruption strategy leads to replacement of the *adprt2* locus region that encodes the catalytic domain with the BSR cassette. Primers used in PCR screening for targeted integration of the BSR cassette are indicated, as are the locations of probes used in Southern blotting. **B.** The *dnapkcs*$^-$ strain was transfected with the *adprt2* disruption construct to generate the *dnapkcs*$^-$*adprt2*$^-$ strain. Verification by PCR used the indicated primers. The 5' screen/5' BSR primers give rise to a 1631 base pair product only when targeted integration of the BSR cassette has occurred. The 5' screen/5' control primers are able to amplify a 958 base pair product in both Ax2 and the disruption strain. **C.** The *dnapkcs*$^-$*adprt2*$^-$ strain was verified for targeted integration at the 3' end of the *adprt2* gene. The 3' screen/3' BSR primers give rise to a 1366 base pair product only when targeted integration of the BSR cassette has occurred. The Rad51 primers are able to amplify a 1234 base pairs product in both Ax2 and the disruption strain. **D.** Southern hybridisation using a probe against the BSR cassette (Probe A) on *Pvu*II digested genomic DNA from Ax2 or the *dnapkcs*$^-$*adprt2*$^-$ strain. Targeted integration of the BSR cassette gives rise to a predicted 2.3kb band only in the disruptant strain. **E.** Southern hybridisation using a probe against the first 189 base pairs of *adprt2* coding sequence (Probe B) on *Eco*RI digested genomic DNA from Ax2 or the *dnapkcs*$^-$*adprt2*$^-$ strain. A predicted 4.1kb band is evident in Ax2 cells. Disruption of the *adprt2* gene gives rise to a predicted band of 4.5kb.



Supplemental Figure 2:

A. *Quantification of Flag-Rad51 enrichment in chromatin following exposure of Ax2 and* adprt2^{-} *cells to MMS***.** Ax2 and *adprt2*$^{-}$ cells expressing Flag-Rad51 were left untreated or exposed to 5mM MMS for indicated times (hours), chromatin fractions prepared, and western blotting performed using Flag or histone H3 antibodies. The membrane was developed with ECL and the signal quantified using the Odyssey Fc system (LI-COR). The Flag signal is normalised to the H3 signal. Data is expressed as the fold-induction of Flag-Rad51 relative to the untreated control for each strain. Error bars represent the standard error of the mean (SEM) from 3 independent experiments. **, $p<0.01$ compared with the untreated control for each strain

B. *Quantification of Flag-Rad51 enrichment in chromatin following exposure of Ax2 and* adprt2^{-} *cells to phleomycin.* Ax2 and *adprt2*$^{-}$ cells expressing Flag-Rad51 were treated with 200μg/ml phleomycin and after 90 minutes chromatin fractions prepared and analysed by Western blotting using Flag or histone H3 antibodies. The signal was detected, quantified and analysed as described in (A). Error bars represent the SEM from 3 independent experiments. *, $p<0.05$; ***, $p<0.001$ compared with the untreated control for each strain



Supplemental Figure 3:

A. *Quantification of Ku80 enrichment in chromatin following exposure of Ax2 and* adprt2$^-$ *cells to MMS.* Quantification of Ku80 enrichment in chromatin after administration of MMS to Ax2 and *adprt2*$^-$ cells. Ax2 and *adprt2*$^-$ cells were left untreated or exposed to 5mM MMS for the indicated times (hours). Western blotting was performed using Ku80 or histone H3 antibodies. The membrane was developed with ECL and the signal quantified using the Odyssey Fc system (LI-COR). The Ku80 signal is normalised to the H3 signal. Data is expressed as the fold-induction of Ku80 relative to the untreated Ax2 strain. Error bars represent the standard error of the mean (SEM) from 4 independent experiments. **, $p<0.01$; ***, $p<0.001$ relative to untreated Ax2.

B. *Quantification of Ku80 enrichment in chromatin following exposure of the Ax2,* adprt1a$^-$*,* adprt2$^-$ *and* adprt1a$^-$adprt2$^-$ *strains to MMS.* Ax2, *adprt1a*$^-$*, adprt2*$^-$ and *adprt1a*$^-$*adprt2*$^-$ cells were left untreated or exposed to 5mM MMS and after 90 minutes chromatin fractions prepared and analysed by Western blotting using Ku80 or histone H3 antibodies. The signal was detected, quantified and analysed as described in (A). Error bars represent the SEM from 5 independent experiments. **, $p<0.01$ relative to the Ax2 untreated control.