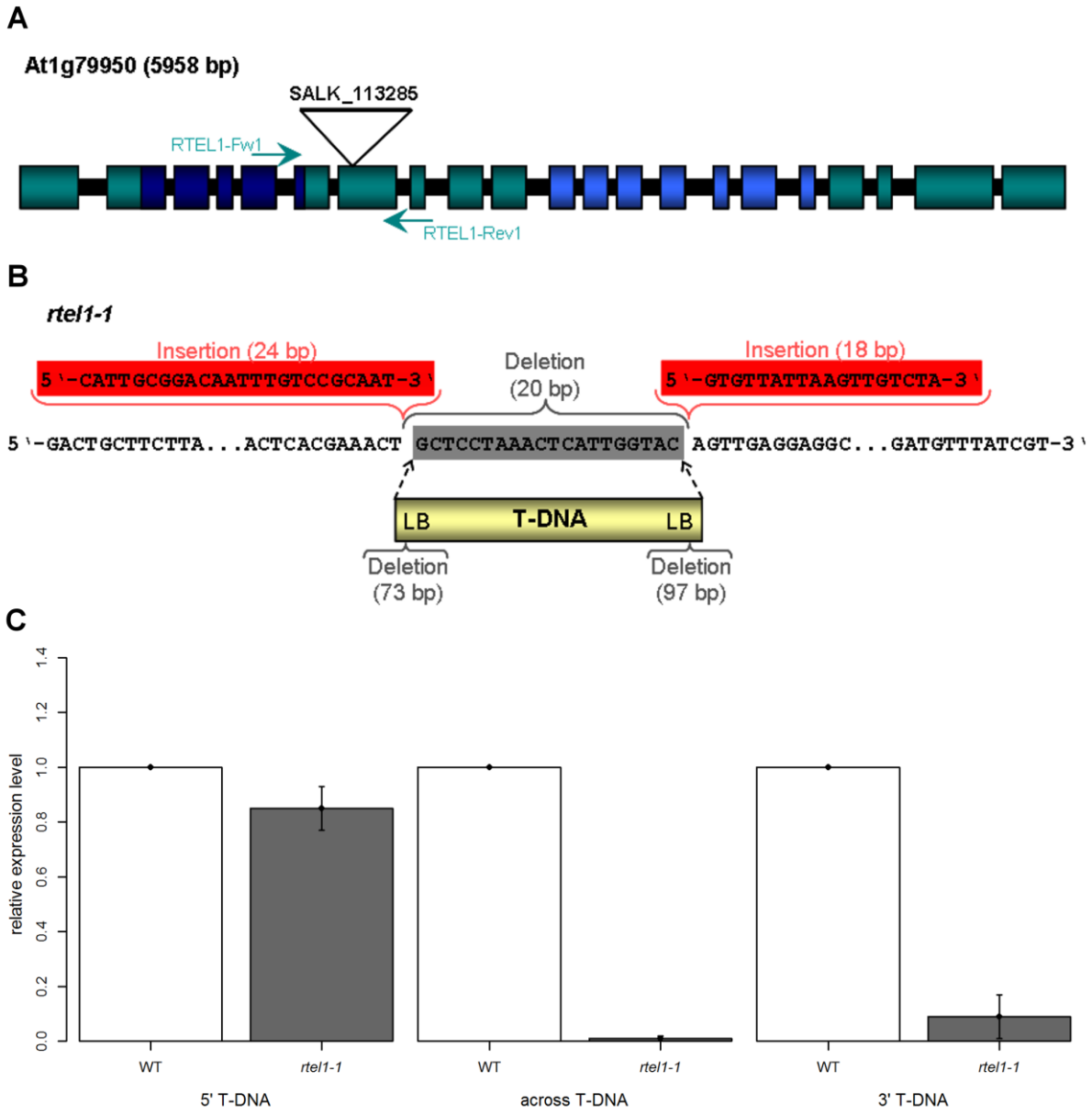


Supplemental Figure 1. Characterization of the T-DNA insertion site of *rtel1-1*.

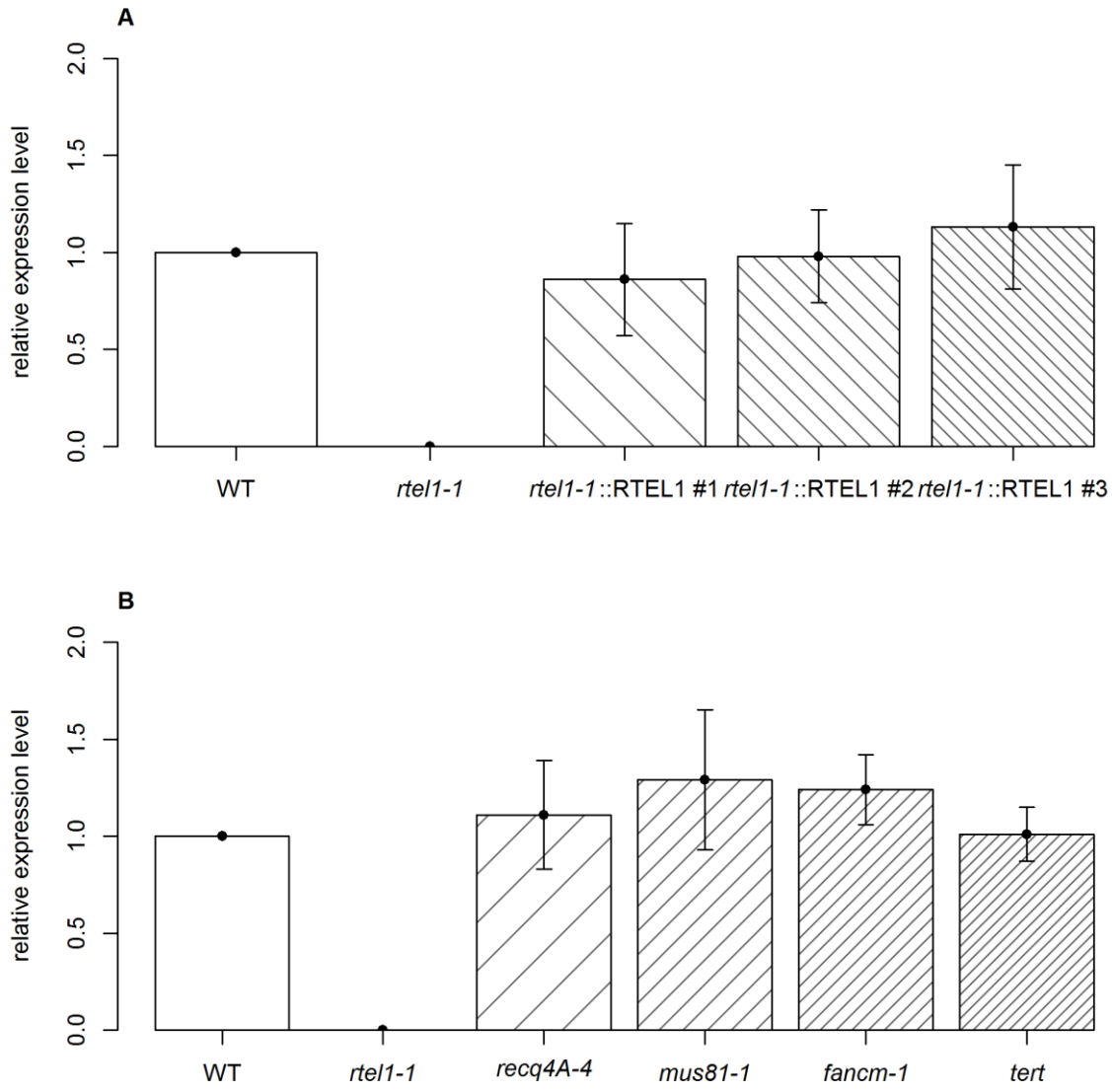
(A) Gene structure of *A. thaliana RTEL1*. The gene At1g79950 (RTEL1) has a length of 5958 bp from the start codon to the stop codon, and it contains 21 exons and 20 introns. The locations of the DEAD_2 (dark blue) and Helicase_C_2 (light blue) domain coding regions are shown. The location of the inserted T-DNA in line SALK_113285 in exon 7 is shown by a triangle flanked by the genotyping primers used in this study.

(figure legend continued on the next page)

Supplemental Figure 1. (continued from previous page)

(B) Sequencing of both sides of the T-DNA insertion site in exon 7 of RTEL1 revealed the presence of a multicopy insertion with deletions of 73 bp and 97 bp of the flanking left borders, respectively. A total of 20 bp of the exon 7 coding sequence was deleted, and 24 bp and 18 bp of foreign sequence were inserted on both sides of the T-DNA during the insertion event.

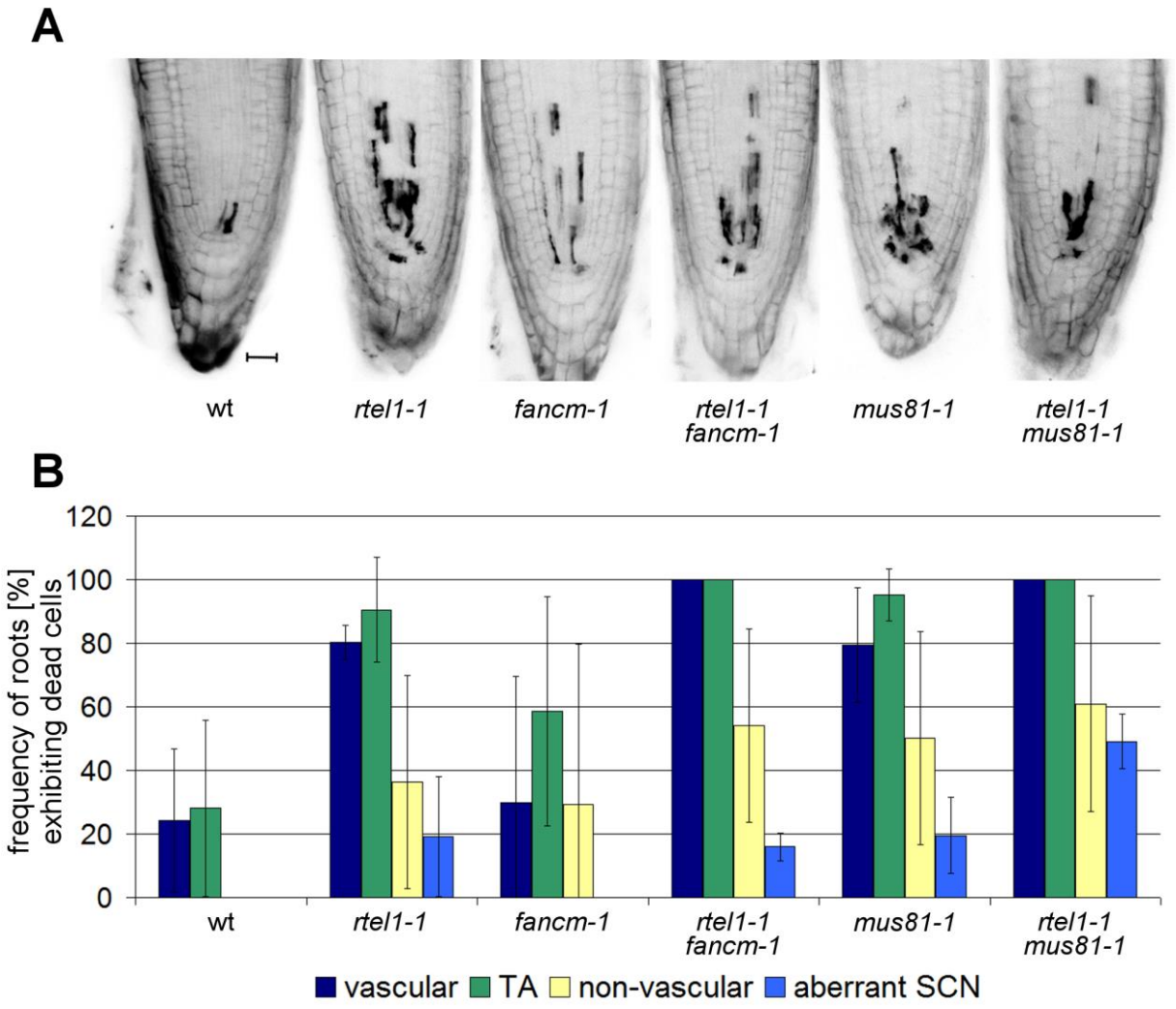
(C) Using primer pairs located 5', 3' and across the T-DNA insertion site, expression was tested in wild type and *rtel1-1* cDNA. Relative to wild type, expression across the T-DNA in *rtel1-1* was less than 0.01. 5' of the insertion, about 80% of wild type expression could be detected in *rtel1-1*, while it was only about 10% 3' of the insertion site. Columns correspond to the mean values (n=3), error bars represent the standard deviations.



Supplemental Figure 2. Gene expression analysis of *RTEL1*.

(A) Expression of *RTEL1* was analyzed in cDNA of wild type, *rtel1-1* and the three complementation lines *rtel1-1*::RTEL1 #1, #2 and #3. Relative to wild type, almost no expression could be detected in *rtel1-1*. All three complementation lines showed a *RTEL1* expression that was not significantly different from wild type.

(B) Relative expression of *RTEL1* was analyzed in cDNA of wild type, *rtel1-1* and the four other single mutants used in this study: *recq4A-4*, *mus81-1*, *fancm-1* and *tert*. Again, while there was almost no detectable signal in *rtel1-1*, all other single mutants displayed a *RTEL1* expression at the same level as the wild type. Columns correspond to the mean values (n=4), error bars represent the standard deviations.

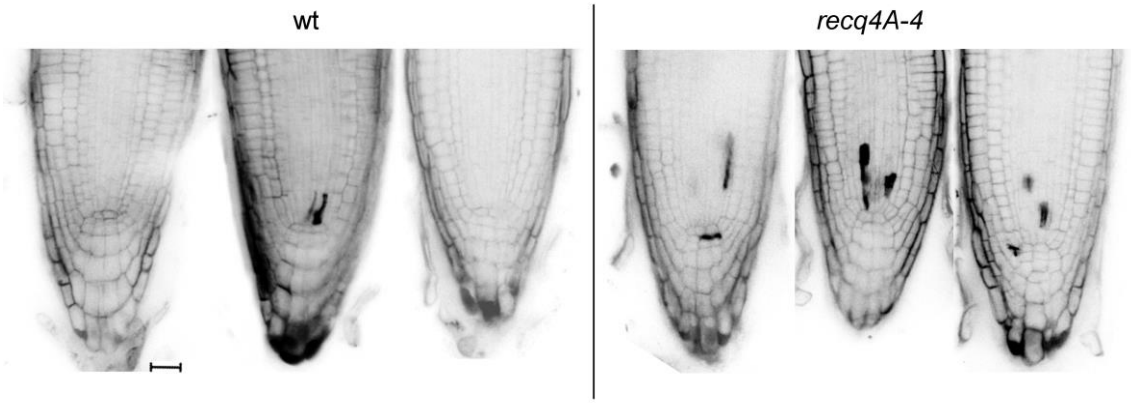


Supplemental Figure 3. Epistasis analysis of root tip stem cell death after *cis*-platin induction.

To elucidate the interplay of RTEL1, FANCM and MUS81 in DNA intrastrand crosslink repair, root tip stem cell death was analyzed in single and double mutants of the respective genes.

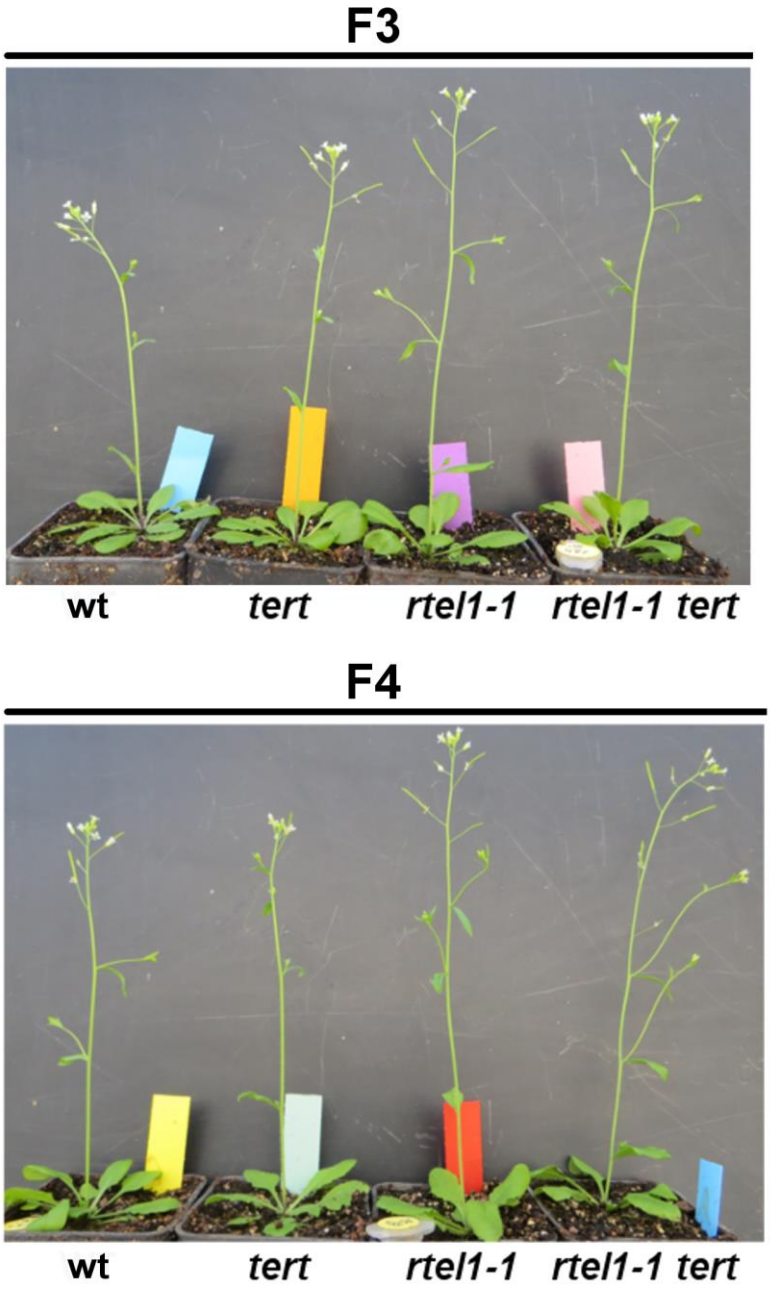
(A) Representative micrographs of PI-stained root tips of the wild-type, the *rtel1-1*, *fancm-1*, *mus81-1* single mutants and the *rtel1-1 fancm-1* and *rtel1-1 mus81-1* double mutants after induction with 35 µM *cis*-platin are shown. Bar corresponds to 20 µm.

(B) Quantification of roots containing at least one dead stem cell belonging to either vascular, TA or non-vascular stem cells after induction with 35 µM *cis*-platin. Columns correspond to the mean values (n=3), error bars represent the standard deviations.

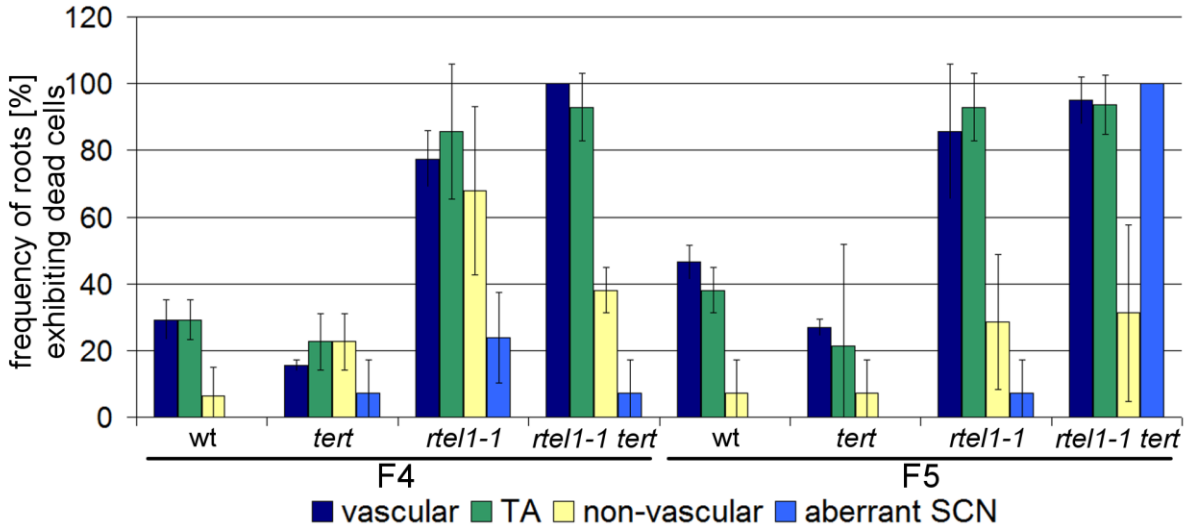


Supplemental Figure 4. Root tip stem cell death after *cis*-platin induction in *recq4A-4*.

Representative micrographs of PI-stained root tips of the wild-type and line *recq4A-4* after induction with 35 µM *cis*-platin are shown. Bar corresponds to 20 µm.



Supplemental Figure 5. Normal development of *rtel1-1 tert* in F3 and F4.
In contrast to the growth defect of *rtel1-1 tert* in F5 (Figure 9), the double mutant line did not show any difference in growth compared to either of the single mutants or the wild-type in F3 and F4.



Supplemental Figure 6. Analysis of root tip stem cell death after *cis*-platin induction in *rtel1-1 tert*.

Quantification of the roots of the wild-type and the *tert*, *rtel1-1* and *rtel1-1 tert* mutants in F4 and F5 containing at least one dead vascular, TA or non-vascular stem cell after treatment with 35 µM *cis*-platin. Columns correspond to the mean values (n=3), error bars represent the standard deviations.