



Figure1-figure supplement 1. Mutations in the Rad51-binding domain of Rad52 suppress MMS and γ -ray sensitivity of Srs2-deficient cells. (A,B,C) Serial 10-fold dilutions of haploid strains that were isolated by random mutation screening (green) or that harbor Rad52 mutations created by directed mutagenesis (red). The tested strains are derivative of *rad52Δ* or *rad52Δ srs2Δ* null mutants transformed with empty Ycplac111 centromeric plasmid or with wild type or mutated versions of *RAD52*. (D,E) Survival curves of haploid cells bearing mutations from our collection. Cells were in the log phase of growth when exposed to γ -rays. Data are presented as the mean \pm SEM of at least 3 independent experiments. The *rad52-A371T*, *rad52-S374A* and *rad52-S387A* mutations cannot restore MMS resistance in Srs2-deficient cells, but they can reduce γ -ray sensitivity of Srs2-deficient cells. This difference could be explained by the constant formation of lesions in cells growing in MMS-containing plates, while exposure to γ -rays for a limited period of time creates only a definite number of lesions.