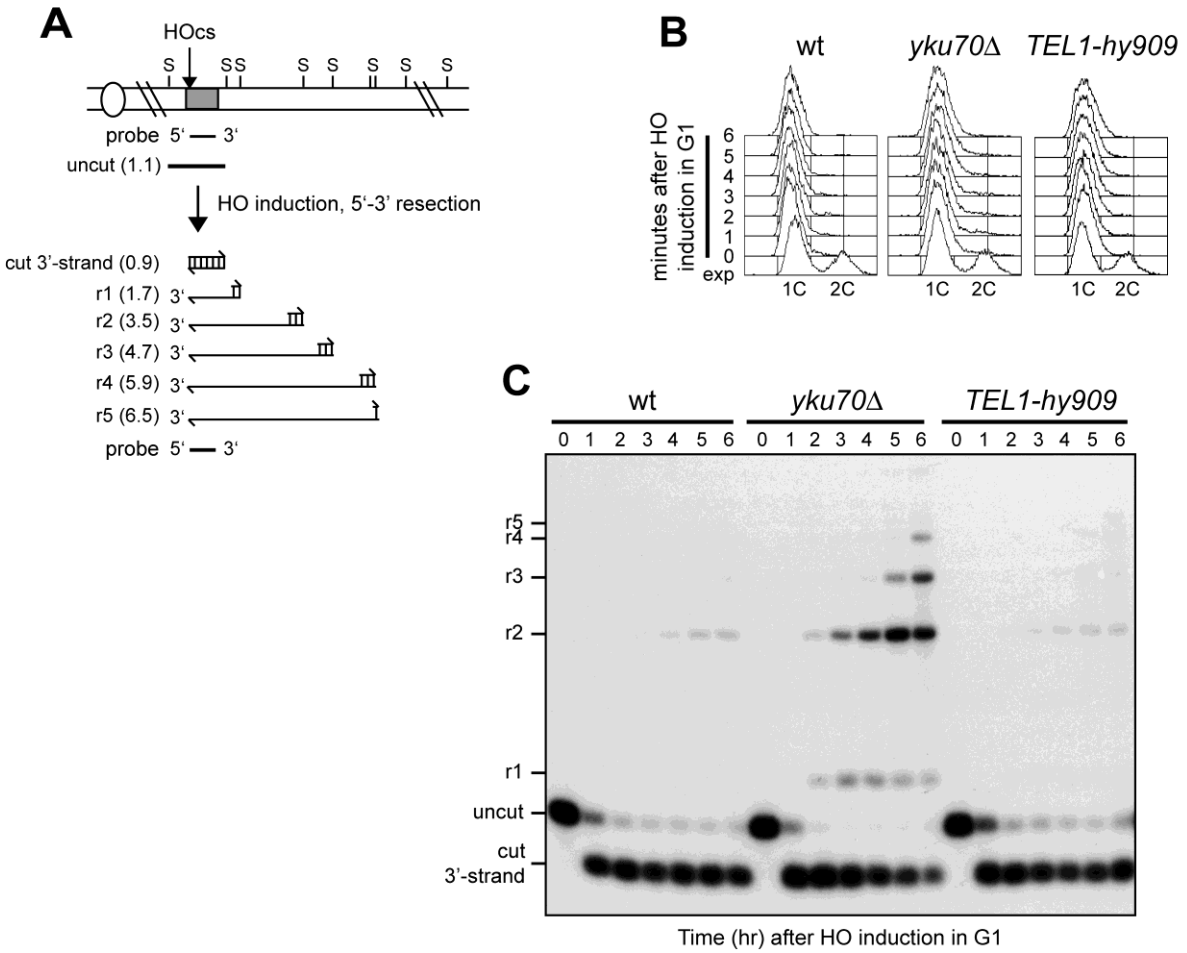

FIG. S1. The *TEL1-hy909* mutation does not enhance nucleolytic processing in G1 of an irreparable HO-induced DSB at the *MAT* locus. (A) Schematic representation of the system used to detect generation of 3'-ended single-stranded resection products at an HO-induced DSB generated at the *MAT* locus in G1-arrested cells, which were unable to repair this DSB because they lacked the homologous donor sequences *HML* and *HMR*. Gel blots of SspI-digested genomic DNA separated on alkaline agarose gel are hybridized with the indicated single-stranded riboprobe that anneals to the unresected strand. 5'-3' resection progressively eliminates SspI sites (S), producing larger SspI fragments (r1 through r5) detected by the probe. (B-C) HO expression was induced at time zero by galactose addition to α-factor-arrested wild type JKM139 and its derivative mutant strains, all carrying the system depicted in (A), which were then kept arrested in G1 with α-factor. (B) FACS analysis of DNA content. (C) Analysis of ssDNA formation as described in (A). The expected resection products were almost undetectable in both wild type and *TEL1-hy909* G1-arrested cells, while they were clearly formed in similarly treated *yku70*Δ cells, which we analyzed as a control because they are known to allow Cdk1-independent ssDNA generation at DSB ends (1).

References

1. Clerici, M., D. Mantiero, I. Guerini, G. Lucchini, and M. P. Longhese. 2008. The Yku70-Yku80 complex contributes to regulate double-strand break processing and checkpoint activation during the cell cycle. EMBO Rep. **9:** 810-818.