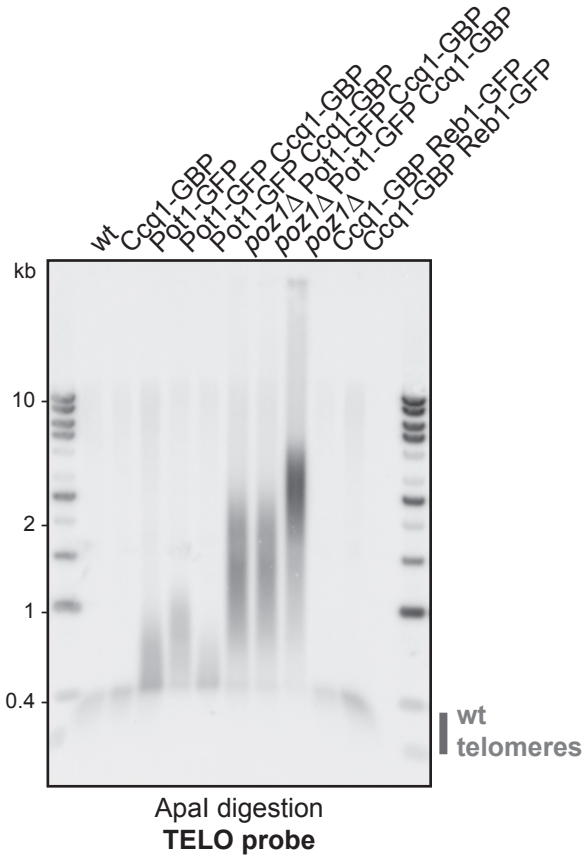
A)

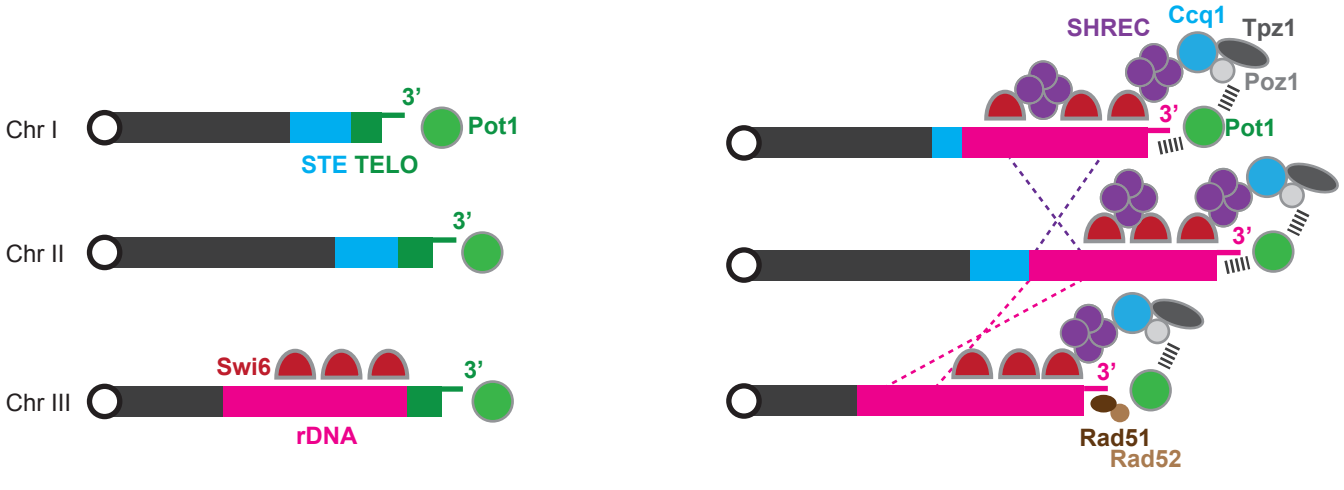


B)

COMPETITIVE GROWTH

	n	L	O	HAATI^{rDNA}	HAATI^{STE}
trt1Δ	10	0%	0%	100%	0%
tazΔ trt1Δ	10	100%	0%	0%	0%
poz1Δ trt1Δ	10	10%	90%	0%	0%
poz1-W209A trt1Δ	10	0%	0%	100%	0%
poz1Δ dcr1Δ trt1Δ	10	0%	90%	0%	10%

C)



WT

HAATI^{rDNA} (*trt1Δ*)

SUPPLEMENTAL FIGURE S1. Telomere length in cells harbouring Shelterin components artificially tethered to rDNA or each other

A) Genomic DNA extracted from the indicated strains was digested with ApaI, which cuts just inside the telomeres of Chr I and II, and analyzed by Southern blotting with a TELO probe derived from ApaI digestion of pNSU70 (see Materials and Methods).

B) The *poz1-W209A* mutation does not prevent HAATI formation. Distribution of survivor types arising from the indicated genotypes under competitive growth conditions. 'n' indicates number of populations analysed per each genomic background. HAATI formation was scored as described in Figure 1.

C) Schematic representing mechanisms of Pot1 recruitment in WT and HAATI^{rDNA}. In WT cells, Pot1 is recruited by the ds telomere binding proteins as well as the ss telomeric overhang. In HAATI^{rDNA}, the rDNA sequences associate with SHREC, which binds Ccq1, which binds Tpz1 and Poz1, in turn concentrating Pot1 at the rDNA terminus. Simultaneously, local Rad51-mediated processes facilitate the handover of replication stalling-induced ss rDNA to the locally concentrated Pot1.