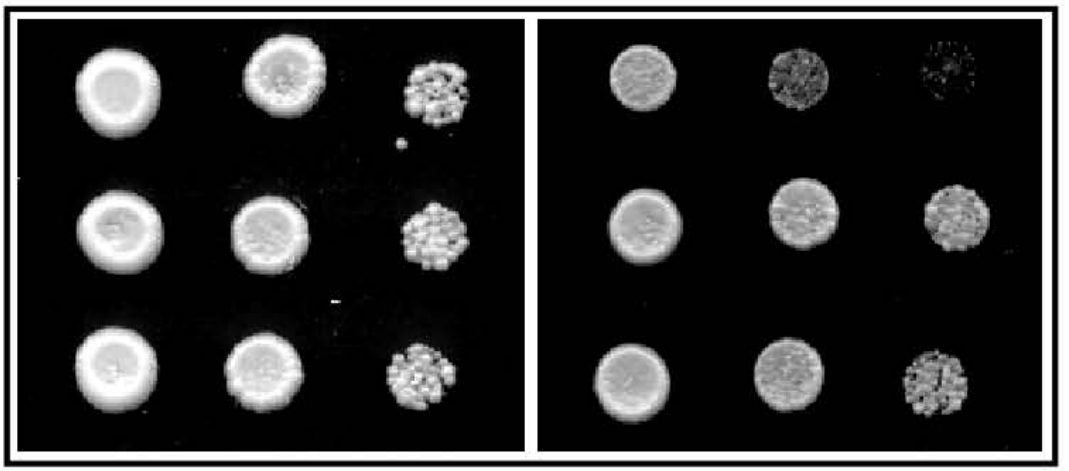
Figure legends (Supplementary figures).

Fig.S1. Silencing assays showing that non-fused, native ScFob1 caused rDNA silencing but not the native full length SbFob1 that was expressed from a single copy vector (pRS415) under the transcriptional control of ScFob1 promoter; **B**, 2D gel analysis showed that the plasmid based SbFob1 was biologically active because it caused normal fork arrest at Ter.

Fig.S2. 2D gel analysis of the replication intermediates of rDNA from the **A**, silencing strain with WT Fob1, **B**, tof1Δ derivative of the same strain and **C**, the tof1Δrrm3Δ double deletion of the same showing that fork arrest in the WT site was normal, in tof1Δ cells it was reduced by >90% and was partially restored in the tof1Δrrm3Δ cells (solid arrows). The open arrows show fork stalling at different regions of rDNA that is a characteristic of rrm3Δ.The data support the interpretation that the sweepase activity of Rrm3 was conserved in the silencing indicator strain.

A



WT/vector

fob1Δ/vector

fob1Δ/Sb FOB1

B

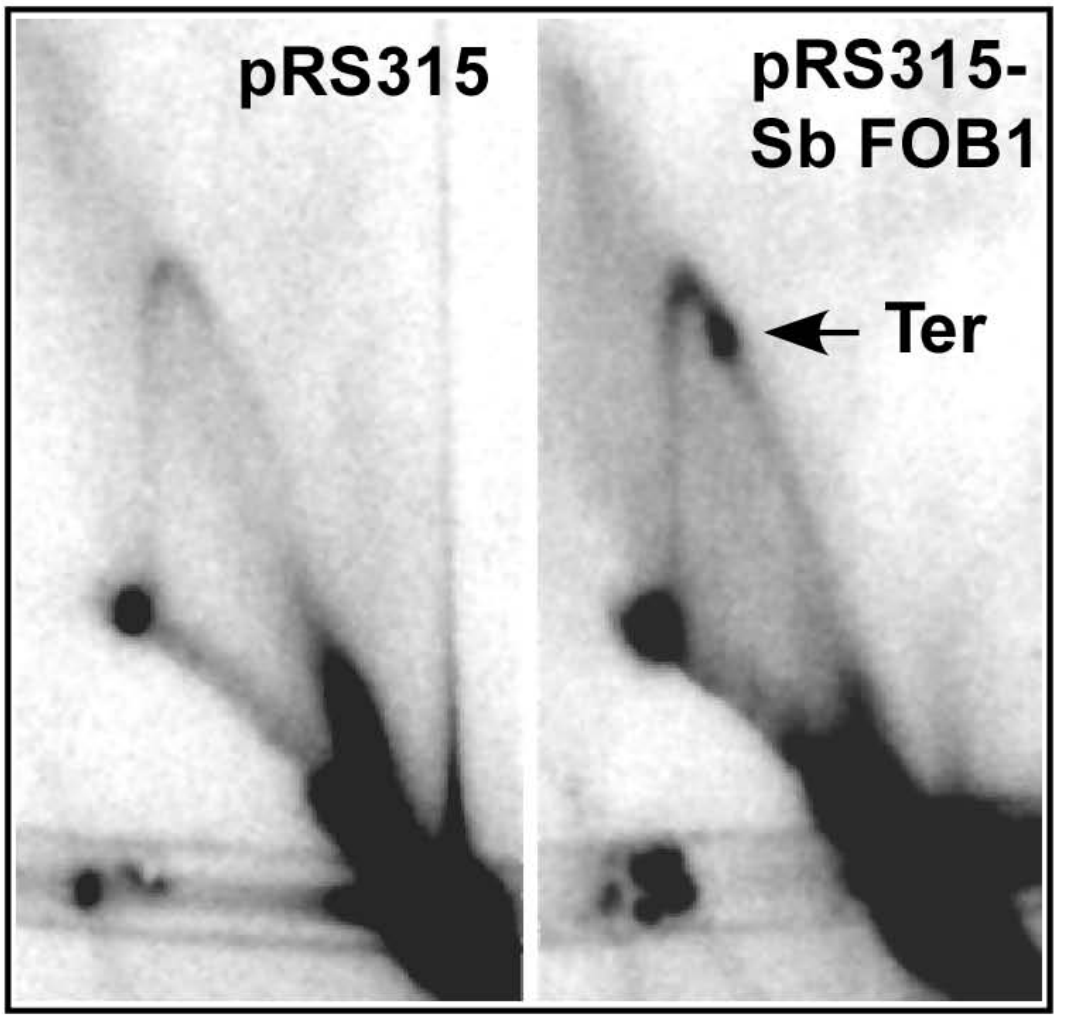


Fig. S1

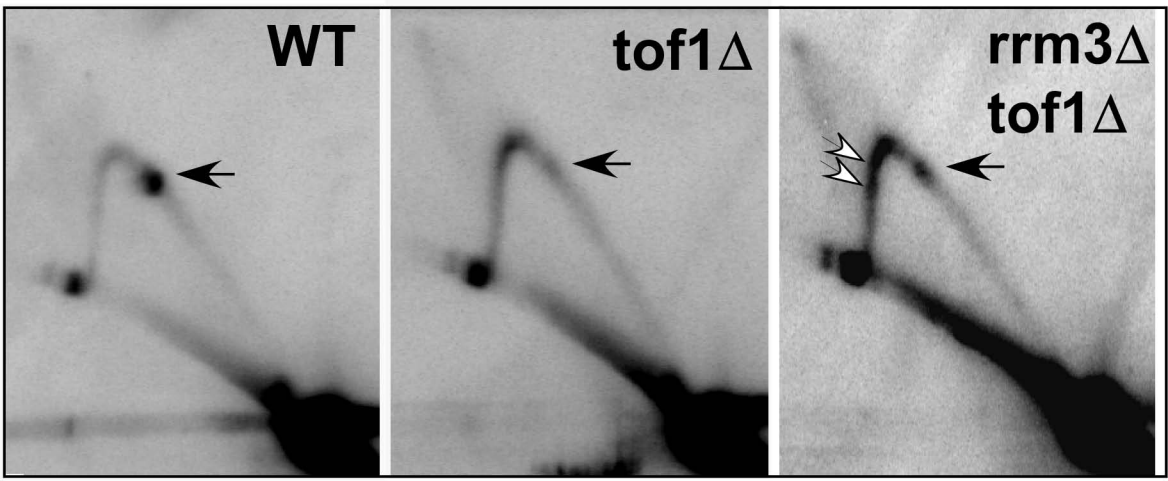


Fig.S2