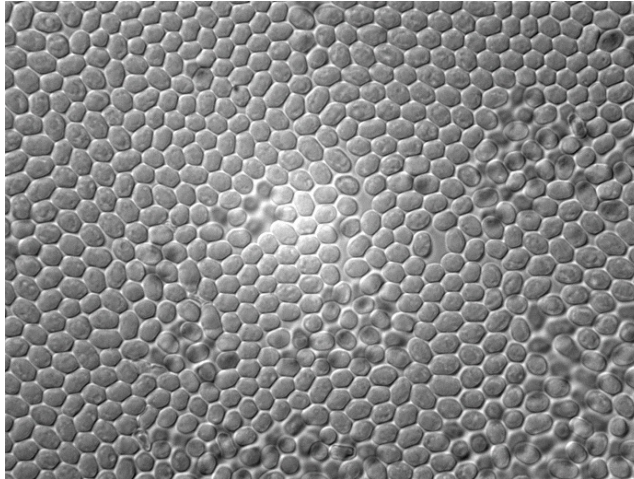




Let sleeping yeast lie: RNA interference in replication and quiescence



Heterochromatin comprises tightly compacted repetitive regions of eukaryotic chromosomes and has widespread roles in chromosome integrity, stability and silencing. The inheritance of heterochromatin requires RNA interference (RNAi), which guides modification of histones deposited on daughter strands upon DNA replication. In *S. pombe* pericentromeric heterochromatin the alternating arrangement of origins of replication and non-coding RNA transcribed during S phase provokes the collision of RNA polymerase with the replication machinery. We propose that collisions are resolved by co-transcriptional RNAi, which releases PolII, allows replication to complete, and couples the spreading of heterochromatin with fork progression. In the absence of RNAi, stalled forks accumulate damage and are repaired by homologous recombination without histone modification, and PolII accumulates at the 3' end of highly transcribed genes, and, surprisingly, at rRNA and tRNA genes as well as in heterochromatin. Recently, we have found that RNAi mutants ($\Delta dcr1$, $\Delta rdp1$, $\Delta ago1$) are impaired in the ability to enter quiescence and lose viability rapidly in G0. Suppressor screens have uncovered genes involved in transcription, heterochromatin and chromosome segregation. We propose that Dicer releases RNA polymerase I from rDNA in quiescent cells, and failure to release results in heterochromatinization and DNA damage.

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