



Figure 4. RNAi-mediated cotranscriptional assembly of heterochromatin in *S. pombe*. Transcription of pericentric repeats gives rise to long noncoding RNAs that are processed into primary small RNAs by Dicer-dependent and -independent pathways. (A) A small RNA loaded onto the RITS complex targets the nascent noncoding RNA by base-pairing interactions. This leads to the recruitment of the RDRC (RNA-directed RNA polymerase complex) and conversion of the targeted RNA into dsRNA, which is diced into siRNAs by Dicer. The resulting duplex siRNA is loaded onto the Argonaute chaperone (ARC) complex and converted into single-stranded siRNA after cleavage and release of the passenger strand in the RITS complex. The mature RITS complex containing single-stranded siRNA can now target additional noncoding RNAs completing a positive feedback loop. The RITS complex also recruits the CLRC H3K9 methyltransferase complex to chromatin via interactions with the Rik1 subunit of CLRC and Stc1, an adaptor protein. (B) H3K9 methylation stabilizes the association of RITS with chromatin and also provides binding sites for HP1 proteins (Swi6 and Chp2). Swi6 facilitates the recruitment of RDRC and degradation by the exosome (C), whereas Chp2 recruits the SHREC complex containing the Clr3 HDAC promotes TGS by mechanisms that remain to be defined (D). In addition to TGS, efficient silencing requires cotranscriptional RNA degradation (CTGS) by RNAi-dependent (A, dicing and slicing) and RNAi-independent (C, TRAMP/exosome degradation) mechanisms. Dicer-independent priRNAs contribute to low levels of H3K9 methylation (E) and may trigger siRNA amplification (A). The 3' ends of priRNAs and siRNAs are trimmed by the Triman exonuclease (A,E). Black tapered arrows indicate enzymatic activity.