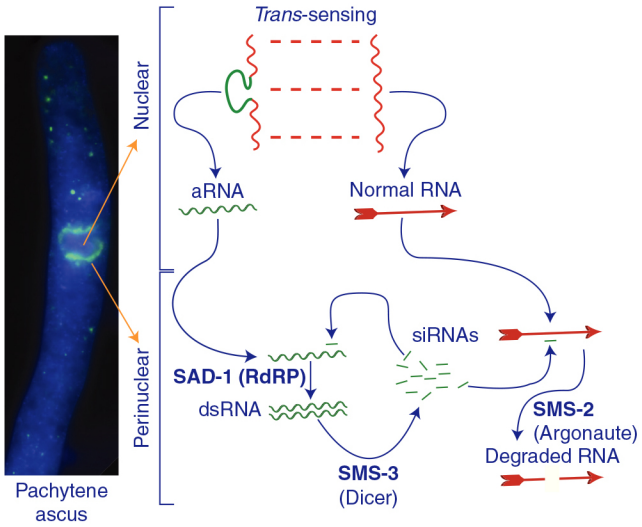

Figure 9. A model for meiotic silencing. An image of a developing ascus from a cross between parents engineered to contain paired copies of *sad-1*$^+$ fused to a reporter gene *gfp*$^+$(i.e., *sad-1*$^+$::*gfp*$^+$) at the Pachytene stage of meiosis I (*left*; DW Lee and R Aramayo, unpubl.). Inside this cell, the meiotic nucleus, delineated by its nuclear membrane, is surrounded by a perinuclear structure that supports the attachment of components of the meiotic silencing apparatus. Predicted nuclear and perinuclear steps in meiotic silencing are diagrammed (*right*). It is hypothesized that *trans*-sensing, a mechanism preceding silencing, identifies heterologous regions of interacting chromosomes. The degree of heterology determines the strength of the induction step, which presumably involves the synthesis of aRNA and its conversion to double-stranded RNA (dsRNA) by the SAD-1 RdRP, a perinuclear event. The presence of dsRNA triggers the initiation of the silencing process, which involves the conversion of the dsRNA trigger into siRNAs via the DCL-1/SMS-3 Dicer (initiation step), and use of these siRNAs primers and normal RNAs as templates, by SAD-1 RdRP to generate dsRNA (amplification cycle). The incorporation of the siRNAs, generated by both the initiation step and the amplification cycles, into the RNA-inducing silencing complex (RISC) directs the endonucleolytic cleavage of mRNA or ssRNA (single-stranded RNA).