H3K27me3 is required to prevent sister somatic gene expression in the ascidian germline

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During ascidian early embryogenesis, the Pem protein suppresses phosphorylation of serine2 of the C-terminal domain (CTD-pSer2) of RNA polymerase II in germline, thereby globally repressing the transcription and ectopic somatic programs. At around the 110-cell stage, the decrease of Pem protein level acts as a pre-requisition for the initiation of zygotic germline gene expression, whereas the somatic transcripts are still faithfully repressed. Thus, we were curious to determine how transcriptionally active germline distinguishes the regulation of germline specific genes from somatic gene expression.

Here we report that H3K27me3, a transcriptional repression marker, becomes enriched in the ascidian *Halocynthia roretzi* germline from the 64-cell stage. Furthermore, we show that H3K27me3 deficient germline in a Pem knockdown background ectopically expresses muscle actin gene around 110-cell stage, but not other tissue-specific genes, suggesting that germline takes on its sister lineage program. Consistent with it, we confirmed that this favored ectopic muscle program depended on a maternal muscle determinant, Macho-1. Finally, depletion of H3K27me3 or Pem did not affect each other.

Altogether our results indicate that *Halocynthia* germline employs H3K27me3-dependent mechanism to firmly protect the germline from taking somatic program. The present study is a new proof supporting a conserved transition from Pol II CTD-based transcriptional repression by localized maternal factors to chromatin-based repression with respect to protect germline fate across animal species.