Kennesaw State University
DigitalCommons@Kennesaw State University

Symposium of Student Scholars | 26th Annual Symposium of Student Scholars - 2022

SPR-5; MET-2 maternal reprogramming cooperates with the Dream Complex to regulate developmental cell fates

Jazmin Dozier
Kennesaw State University

Sandra Nguyen
Kennesaw State University

Brandon Carpenter
Kennesaw State University

Follow this and additional works at: https://digitalcommons.kennesaw.edu/undergradsymposiumksu

 Part of the Cell Biology Commons, Developmental Biology Commons, Genomics Commons, and the Molecular Genetics Commons

Dozier, Jazmin; Nguyen, Sandra; and Carpenter, Brandon, "SPR-5; MET-2 maternal reprogramming cooperates with the Dream Complex to regulate developmental cell fates" (2022). *Symposium of Student Scholars*. 416.
https://digitalcommons.kennesaw.edu/undergradsymposiumksu/spring2022/presentations/416

This Poster is brought to you for free and open access by the Office of Undergraduate Research at DigitalCommons@Kennesaw State University. It has been accepted for inclusion in Symposium of Student Scholars by an authorized administrator of DigitalCommons@Kennesaw State University. For more information, please contact digitalcommons@kennesaw.edu.

SPR-5; MET-2 maternal reprogramming cooperates with the Dream Complex to regulate developmental cell fates

Histone methylation is a post-transcriptional modification to the N-terminal tails of histone core proteins that regulates DNA accessibility, and consequently, gene expression. Like DNA, histone methylation can be inherited between generations, and is highly regulated during embryonic development. At fertilization, histone methylation must undergo maternal reprogramming to reset the epigenetic landscape in the new zygote. During maternal reprogramming of histone methylation in the nematode, *C. elegans*, H3K4me (a modification associated with active transcription) is removed by the H3K4 demethylase, SPR-5, and H3K9me (a modification associated with transcriptional repression) is subsequently added by the histone methyltransferase, MET-2. Recently, it was demonstrated that SPR-5; MET-2 maternal reprogramming antagonizes the H3K36 methyltransferase, MES-4, which maintains a transcriptional memory of a subset of germline genes between generations. Maternal loss of SPR-5 and MET-2 results in ectopic expression of MES-4 germline genes in somatic tissues and a severe developmental delay. Recently, exciting new literature suggest that the DREAM Complex, a transcriptional repressor complex that regulates cell cycle, also represses MES-4 germline genes in somatic tissues suggesting that the DREAM Complex and SPR-5; MET-2 maternal reprogramming may work together to prevent ectopic germline gene expression in somatic tissues and developmental delay. To test this hypothesis, we knocked down Dream Complex members LIN-35 and LIN-9 in *spr-5; met-2* mutants using RNA interference (RNAi). We found that loss of either LIN-35 or LIN-9 exacerbates the severe developmental delay that we normally observe in spr-5; met-2 mutants leading to a complete larval arrest. These findings suggest that the Dream Complex and SPR-5; MET-2 maternal reprogramming work together to ensure proper development and provide insight into how an evolutionary conserved transcriptional repressor complex cooperates with maternal reprogramming of histone methylation to regulate germline versus somatic cell fates.