LPS Award was presented to Caroline Sartain (Wolfner lab) in May 2012 for the best G&D paper.

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The poly(A) polymerase GLD2 is required for spermatogenesis in Drosophila melanogaster.

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Source

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Abstract

The DNA of a developing sperm is normally inaccessible for transcription for part of spermatogenesis in many animals. In Drosophila melanogaster, many transcripts needed for late spermatid differentiation are synthesized in pre-meiotic spermatocytes, but are not translated until later stages. Thus, post-transcriptional control mechanisms are required to decouple transcription and translation during spermatogenesis. In the female germline, developing germ cells accomplish similar decoupling through poly(A) tail alterations to ensure that dormant transcripts are not prematurely translated: a transcript with a short poly(A) tail will remain untranslated, whereas elongating the poly(A) tail permits protein production. In Drosophila, the ovary-expressed cytoplasmic poly(A) polymerase WISPY is responsible for stage-specific poly(A) tail extension in the female germline. Here, we examine the possibility that a recently derived testis-expressed WISPY paralog, GLD2, plays a similar role in the Drosophila male germline. We show that knockdown of Gld2 transcripts causes male sterility, as GLD2-deficient males do not produce mature sperm. Spermatogenesis up to and including meiosis appears normal in the absence of GLD2, but post-meiotic spermatid development rapidly becomes abnormal. Nuclear bundling and F-actin assembly are defective in GLD2 knockdown testes and nuclei fail to undergo chromatin reorganization in elongated spermatids. GLD2 also affects the incorporation of protamines and the stability of dynamin and transition protein transcripts. Our results indicate that GLD2 is an important regulator of late spermatogenesis and is the first example of a Gld-2 family member that plays a significant role specifically in male gametogenesis.