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The DSL ligand APX-1 is required for normal ovulation in *C. elegans*

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ABSTRACT:
DSL ligands activate the Notch receptor in many cellular contexts across metazoa to specify cell fate. In addition, Notch receptor activity is implicated in post-mitotic morphogenesis and neuronal function. In *C. elegans*, the DSL family ligand APX-1 is expressed in a subset of cells of the proximal gonad lineage, where it can act as a latent proliferation-promoting signal to maintain proximal germline tumors. Here we examine *apx-1* in the proximal gonad and uncover a role in the maintenance of normal ovulation. Depletion of *apx-1* causes an endomitotic oocyte (Emo) phenotype and ovulation defects. We find that *lag-2* can substitute for *apx-1* in this role, that the ovulation defect is partially suppressed by loss of *ipp-5,* and that *lin-12* depletion causes a similar phenotype. In addition, we find that the ovulation defects are often accompanied by a delay of spermathecal distal neck closure after oocyte entry. Although calcium oscillations occur in the spermatheca, calcium signals are abnormal when the distal neck does not close completely. Moreover, oocytes sometimes cannot properly transit through the spermatheca, leading to fragmentation of oocytes once the neck closes. Finally, abnormal oocytes and neck closure defects are seen occasionally when *apx-1* or *lin-12* activity is reduced in adult animals, suggesting a possible post-developmental role for APX-1 and LIN-12 signaling in ovulation.

Keywords:
somatic gonad; Emo; spermatheca; reproduction; Notch; LIN-12