ABSTRACT
INVESTIGATIONS INTO GERMLINE RESPONSE MECHANISMS DURING MODERATE TEMPERATURES STRESS IN *CAENORHABDITIS ELEGANS*

Kristen Ann Quaglia, B.S.

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In organisms as diverse as flies, nematodes, and mammals, as temperature increases, fertility decreases. Thus, organisms must have mechanisms to maintain the ability to produce fit offspring despite experiencing moderately elevated temperatures. The goal of this dissertation is to investigate germline apoptosis as a response mechanism in *Caenorhabditis elegans* to buffer fertility and progeny fitness during moderate temperature stress.

In chapter III, I investigate the impact of moderate temperature stress on fertility, oocytes, and progeny fitness when apoptosis is absent or elevated above basal levels. I show that the presence of apoptosis at an intermediate level removes germ cells that would fail to develop into viable progeny while retaining enough germ cells to supply cytoplasmic resources to developing oocytes. Additionally, using 12 wild strains, I show that an intermediate induction of apoptosis during temperature stress is associated with the highest fertility and progeny fitness, suggesting that the induction of apoptosis as a stress-response mechanisms is a balance between removing low-quality germ cells and producing enough cytoplasmic resources for fit progeny.

In chapter IV, I report on underlying genetic contributions to the variability in fertility and apoptosis using a novel recombinant inbred line panel. I performed linkage mapping which identified a quantitative trait locus associated with variation in fertility level and found the induction of apoptosis is largest in lines containing one of the parental alleles.

In Chapter V, I examine the mechanism that activates the canonical apoptosis pathway during moderate temperature stress. I show that the induction of apoptosis is dependent on the synapsis checkpoint, and asynapsed nuclei are culled via apoptosis. Additionally, I demonstrate that the canonical apoptosis pathway is triggered through parallel pathways: CED-13 inhibits CED-9 while LIN-35 and the DREAM complex down regulate *ced-9* expression.

Collectively, this dissertation demonstrates the induction of apoptosis in response to moderately elevated temperatures functions to both remove low-quality germ cells and to supply additional cytoplasmic resources to remaining germ cells. As elevated temperatures become more prevalent due to prolonged climate change, investigation into germline stress-response strategies is imperative for the survival of species.