Summary

Inheritance of stable genomes is a prerequisite for species maintenance. Germ cells invoke efficient meiotic checkpoint signaling and the DNA damage responses (DDR) to ensure numerical and structural genomic integrity. Canonical DDR induce cell cycle arrest, DNA repair, and apoptosis to encounter exogenous or endogenous genotoxic assaults. Herein, we investigate whether known somatic stress responsive factors in *Caenorhabditis elegans* affect the regulation of germ cell apoptosis and maintenance of heritable genomic integrity. We show that intestinal PMK-1/p38 MAPK signaling regulates DNA damage-induced apoptosis in germ cells through the transcriptional activator ATF-7. Intestinal PMK-1 is also required for germ cell apoptosis in response to meiotic DNA damage inflicted by defective synaptonemal complex formation in *syp-2(ok307)* mutant animals. We identify T24B8.5, a putative secreted peptide to mediate this non-cell-autonomous control of DNA damage-induced germ cell apoptosis. Furthermore, we demonstrate that intestinal PMK-1 signalling regulates germ cell apoptosis in response to somatic heat stress. Importantly, this transient heat stress leads to a robust increase in X chromosome missegregation and aneuploidy in the *pmk-1(km25)* mutant, resulting in a high incidence of male (HIM) phenotype which can be rescued by intestinal PMK-1. Consistently, single-worm whole genome sequencing (WGS) revealed enhanced aneuploidy as a result of compromised PMK-1 stress signaling in the following generation of *syp-2(ok307); pmk-1(km25)* worms. Taken together, in this PhD dissertation, we suggest that somatic stress surveillance mechanisms control germ cell apoptosis and heritable genomic stability.