MAPPING SUPPRESSION OF PREMATURE SPERM ACTIVATION IN *C. ELEGANS*

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The sperm of the roundworm *Caenorhabditis elegans* move by a crawling motion, in contrast to mammalian sperm which exhibit a swimming motion. This method of locomotion makes the sperm of *C. elegans* an excellent model for examining cell motility. In order to become motile, the sperm must undergo the process of activation, during which the sperm change morphologically and gain the ability to crawl. Because *C. elegans* is an androdioecious species, the sperm of both the male and hermaphrodite must activate. Previous work has shown that each sex relies on a distinct genetic pathway to activate its sperm, though both pathways are present in sperm from both males and hermaphrodites. In males, two protein factors, SWM-1 and TRY-5, have been shown to modulate sperm activation. SWM-1 activity is required to delay activation while spermatids reside in the male's seminal vesicle. In this project, we utilized two methods to identify additional protein factors that may have a controlling role in the activation process. The first method was mapping by outcrossing. A strain containing a mutation in the *swm-1* gene, which caused premature activation of the spermatids while still inside the seminal vesicle of the male, had previously been exposed to a mutagen and lines showing suppression of the premature activation phenotype were isolated. A round of outcrossing to a polymorphic strain of was followed by the pooling of male worms expressing the suppressed phenotype. We then sequenced the entire genomes of these worms and identified genomic regions linked to the suppression phenotype. The second method involved sequencing suppressed strains without any mapping. This strategy relied on the filtering of common variants, variants inconsistent with our mutagen of choice, and variants unlikely to influence sperm activation out of the total variant list, leaving only mutations in genes with a relatively high likelihood of affecting the activation process. Both of these variant identification methods have yielded short lists of variants of interest that can now be tested for causality using rescue experiments.