Project Title: Evolutionary Path of a Non-canonical Start Codon in the *Drosophila Fmr1* Gene

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The human fragile X messenger ribonucleoprotein (*FMR1)* gene encodes fragile X mental retardation protein (FMRP), a RNA binding protein which functions as a chaperone to mRNA during nuclear export to the cytoplasm. *Drosophila melanogaster* contains an ortholog of this gene, and has been a model organism for better understanding its role in behavior and in the development of neural anatomy. The *D. melanogaster Fmr1* gene has multiple coding isoforms and previous research has demonstrated the existence of a non-canonical start codon present in a subset of those isoforms. Non-canonical start codons are understood to be less efficient at translation initiation than standard start codons, but are thought to provide an alternative mechanism for isoform diversification and protein function. Our project aims to further understand the evolutionary path of the non-canonical start codon in the *Drosophila* genus through sequence analysis of the *Fmr1* gene in different species. We first annotated the gene in various *Drosophila* species using *D. melanogaster* as a reference species. We used RNA sequence data from the target species and localized homology searching as determinants for a non-canonical start codon in species. Our results suggest evidence for the utilization of a non-canonical start codon in *Fmr1* isoforms in a subset of closely related *Drosophila* species but a lack of evidence in species more distantly related, indicating its recent evolution as a gene expression mechanism. These results raise additional questions about translation initiation of, and the diversification of, FMRP within eukaryotic genomes.