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## Characterization of *Caenorhabditis elegans* F07A5.4, human ortholog of Olfactomedin 1

Sade K. Thomas, Karunambigai Kalichamy, and Martin Hudson\*

Accurate control of nervous system development is critical for normal brain patterning, and defects in this process can lead to neurological disorders such as schizophrenia and Autism Spectrum Disorder. The transcription factor neurogenin is necessary for the development of neural subtypes and is a deeply conserved across species. However, the transcriptional targets of neurogenin are poorly understood, creating an imperative for further study. We have used the nematode *Caenorhabditis elegans* as a model to better understand *ngn-1*/neurogenin function. Previous work from our lab revealed that *ngn-1* plays a role in nerve ring architecture, and neural cell fate specification. In addition, *ngn-1* mutants have an array of neuromuscular defects such as sluggish, uncoordinated movement and precocious egg laying. To help identify downstream targets of *ngn-1*, we performed a comparative transcriptome on messenger RNA isolated from wild type and *ngn-1* mutants. We discovered that *F07A5.4* transcript levels are significantly lower in *ngn-1* mutants. *F07A5.4* is an ortholog of Human olfactomedin (*Olfm1*), which is a secreted glycoprotein in the conserved olfactomedin family (OLF). In rodents, OLF domain proteins play important roles in neurogenesis, neural crest formation, and cell adhesion. We hypothesize that *F07A5.4* is required for establishing normal nervous system architecture in the worm. To investigate this, we have identified alleles from the Million Mutation Project that mutate conserved amino acids in the *F07A5.4* protein sequence. This project aims to characterize the phenotype of those alleles and to establish the normal function of *F07A5.4* in *C. elegans* nervous system development.