

The Neurofibromatosis Type 2 Molecule NFM-1 Controls Neural Migration in *C. elegans*

Aliani, Rana, Matthew Josephson, and Erik Lundquist, Department of Molecular Biosciences, University of Kansas

Cell migration plays an important role in the development of animals, including the development of nervous system. The migration of neuroblasts is essential for proper neuronal function. In *Caenorhabditis elegans*, the Q neuroblasts QR and QL are stem cells that give rise to multiple neurons including the sensory neurons AQR and PQR. QR and QL are born in the same region on the worm before migrating in opposite directions. QR migrates anteriorly, giving rise to AQR near the head. QL migrates posteriorly and gives rise to PQR, near the anus. A screen identified *nfm-1* as a gene required for Q cell migration. *nfm-1* is the human homolog of NF2/Neurofibromin. Neurofibromatosis type II is an autosomal dominant disease that occurs when one copy of NF2 is mutated. The disease causes unregulated glial growth and formation of tumors, the reason behind which is unknown. We report here that *nfm-1* mutations strongly reduce the ability of AQR to migrate anteriorly, with a slight effect on PQR, suggesting that *nfm-1* could be important in directed migration. Fluorescent microscopy and mosaic analysis suggest that *nfm-1* likely has a non-autonomous role in AQR migration. Further analysis shows that the *nfm-1* promoter is expressed in the posterior of the animal and excluded from the Q cells, consistent with a non-autonomous role for *nfm-1*. Our focus going forward is to determine genes that work with *nfm-1* to control migration. This will provide insight into how complex nervous systems develop and potentially identify the underlying cause of neurofibromatosis.