PAM-1 aminopeptidase prevents neurodegeneration in *Caenorhabditis elegans*.

The pathologies associated with neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease, and Huntington's disease are due in part to aberrant protein and aggregation and inclusion body formation. Protein aggregates are normally cleared through autophagic mechanisms, and disruption of the constituents of these pathways trigger neurodegeneration in a number of animal models. For example, mutation of the cytosolic puromycin-sensitive aminopeptidase (Psa) in flies results in the age dependent accumulation of protein aggregates and the degeneration of GFP-labeled neurons *in vivo*.

PAM-1 is the *Caenorhabditis elegans* orthologue of Psa. PAM-1 has known functions in mediating fertility and lifespan, but little assessment its role in preventing neurodegeneration in worms has been done. We are utilizing a neuron-specific GFP transgene to monitor the development of neurological abnormalities in the GABA neurons in wild-type and mutant *pam-1* strains over the course of their lifespan. These abnormalities will be characterized in reference to age of the organisms. We hypothesize that the *pam-1* mutant worms will accrue abnormalities at a faster rate than the wild-type worms; and preliminary trials have supported this hypothesis thus far. Abnormalities such as blebs, breaks, and branching are apparent. The development of simple model systems that mimic human pathology may provide useful and powerful reagents in dissecting complex diseases.