Isolation of mutant *Caenorhabditis elegans* suppressor to α-Integrin Subunit Deficiency

The *ina-1*-encoded integrin α subunit is essential to *Caenorhabditis elegans* development, due to its role in complex processes such as morphogenesis, neuron migration, and cell signaling. Studies have shown that when this gene is mutated, many larvae die, and the worms that do survive display inactivity and morphogenic defects. Specifically, α-integrins function within heterodimeric integral membrane proteins and facilitate cellular and organismal processes via interactions between the cell's cytoskeleton and other cell surfaces and the extracellular matrix. Similar to *C. elegans*' *ina-1* gene, humans possess 13 *ina-1* homologs (ITGA3, ITGA6, and ITGA7), which are integral to normal human development; defects in the human α-integrin genes are linked to congenital muscular dystrophy, epidermolysis bullosa and cancer. *C. elegans* are a model organism to better understand integrin subunits and their important roles in both nematode development and human disorders due to their small number of α- integrin genes, which simplifies their genetic analysis. In this study, we generated and isolated mutants *C. elegans* that suppressed the integrin subunit deficiency by treating them with ethyl methanesulfonate (EMS). Mutants expressing suppressor mutations were characterized by more successful development, viability, and movement. The phenotypes were analyzed, and the mutants displaying these characteristics were isolated and cultured to form a colony of worms that contained the suppressor mutation. In the future, we will genetically analyze the suppressors of ina-1 mutations to elucidate their genetic basis and their important implications in human homologs.