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Effect of a cell binding domain mutation in the *unc-52* gene of *C. elegans*

Caenorhabditis elegans provides a significant canvas for research due to their sequenced genome, recorded molecular pathways, simple structure and comparative systematic components useful in modeling human diseases. The *unc-52* gene in *Caenorhabditis elegans* encodes a homologouge for the extracellular matrix proteoglycan perlecan**.** *UNC-52* constitutes a structural basement membrane protein which plays an important role in myofilament organization, and a regulator of growth-factor signaling in the body wall muscle cells. To determine the phenotypic effect formed from the presence of *unc-52* mutation, we utilized the CRISPR-Cas9 gene-editing technology to mutate the amino acid sequence of the *unc-52* gene. We edited the cell-binding domain of *unc-52* and produced RGE (arg-gly-glu) from RGD (arg-gly-asp). We injected 52 N2 worms, and successfully generated several homozygous alleles in the *C. elegans* where the RGD sequences had been transformed to RGE. *C. elegans* was observed after treatment, and successfully mutated genes produced *severely paralyzed uncoordinated* worms in the surviving phenotype specimens proceeding CRISPR-cas 9 gene editing. Previous experiments with the *unc-52* gene have shown a number of different mutations causing frameshift mutations and nonsense mutations which have been lethal to the organism. The RGD sequence we aim to mutate has been shown to mediate interactions with cell-surface integrins, as well as function in the development of myofilament lattice assembly. A severe phenotypic defect arising from the mutated genes would prove specifically the importance of the RGD sequence in the development of the cytoskeleton and cellular interactions, as well as demonstrate a non-lethal mutant in the *unc-52* gene.