β-III spectrin is a member of the spectrin meshwork in neurons formed by heterodimeric units of αII-spectrin and each of four β-spectrins (I-IV), which then form tetramers that crosslink F-actin to form periodic spectrin-actin arrays along axons and dendrites. β-spectrins also bind their molecular partners ankyrins to, together, stabilize ion channels, cell adhesion molecules, and membrane receptors. β-III spectrin, in addition to the other spectrins, is involved in various cellular processes, including axonal growth and maintenance, intracellular transport, and signaling transactivation. Unsurprisingly, mutations in β-III spectrin underlie neurodevelopmental, neurogenerative, and psychiatric disorders. Specifically, mutations in SPTBN2, the gene for β-III spectrin, have been found to be associated with infantile-onset spinocerebellar ataxias, global developmental delays and cognitive impairment. However, the pathogenic mechanisms are not fully understood. In order to better understand how β-III spectrin mutations manifest cellurally, we characterized various mutations in β-III spectrin including L426del, L253P, R437Q/G/W, and R480W through transfection of mutagenized GFP-β-III spectrin in HEK293T cells and evaluation of protein expression levels and binding interactions through Western blots and immuno- precipitation assays. Our preliminary findings indicate that two of the mutations show decreased protein expression levels relative to WT. Further studies are necessary to substantiate this data. All three mutations also seemed to demonstrate no significant disruption to β-III spectrin-αII-spectrin binding. However, further experimental data is needed to confirm or deny this observation.