Structure based Prediction of Stability for Human β-Glucronidase Mutants

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Mucopolysaccharidosis or Sly syndrome, caused by deficiency of the β-glucuronidase enzyme show wide clinical variability, with severe mental retardation, severe somatic involvement, death at an early age), skeletal abnormalities, mild delay in neuropsychomotor development, mild organomegaly, onset at adolescence, very mild somatic involvement. A form associated with fetal hydrops is also frequent. Many different disease-causing mutations have been identified in patients from Japan, Europe, the United States, Northern Africa, Kuwait, India, Mexico and Chile. There are a lists of mutations found in β-glucuronidase enzyme like mutation in exon 3 of the β-glucuronidase gene that produces a Leu → Phe substitution (L176F). We have created *in-sililico* mutations and minimized the energy of these mutated proteins using various bioinformatics tools and also with online tools like MUSTER, LOMETS and modweb. Further we take one suitable model of each and run CONACT program.