COMPUTACIONAL ANALISYS OF SUPEROXIDE DISMUTASE MUTATIONS

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SOD (Cu,Zn-superoxide dismutase) is known to be a locus of mutation in familial amyotrophic lateral sclerosis (FALS). Although five mutations located at Gly93 (G93A, G93C, G93D, G93R, G93V) have been implicated in FALS progress, only G93R revealed to be deleterious to sod activity. By using *in silico* analyses, we investigated whether there would be any differences between the three-dimensional protein conformation of wild-type and FALS mutants Cu,Zn-SOD. For this purpose, it was created theoretical models for all G93 mutants through homology modeling performed by Geno3D. The models predicted were aligned to wild type PDB structure using MATRAS, a program for protein 3D structure comparison. In addition, an extensive phylogenetic analysis revealed that Gly93 region is high conserved among primates and yeast. According to our results, only G93R led to a drastic alteration of 3D structure of Cu,Zn-SOD, correlating the conformational change with activity loss found in this FALS mutant.