ABSTRACT

Phosphorylation and *O*-GlcNAc modification act as functional switch of glutamate receptor AMPAR. *In silico* studies of AMPAR proposes that phosphorylation of AMPA receptor subunit GluR1 at certain Serine and therionine residues causes hippocampal LTP whereas of GluR2 promotes cereballar LTD. On the other hand *O*-GlcNAc on the same sites of GluR1and GluR2 promotes hippocampal LTD and cereballar LTP respectively. The *in silico* docking of 1YAE (template) and 2A5S (ligand) showed the best model of GluR1 ligand (glutamate) binding core.