Abstract

The BDNFTrkB signaling system which plays a major role in the regulation of neuronal activity. This neuronal regulation influences the potential role of this system in the therapeutic efficacy of many neurological and psychiatric disorders. Despite these roles decreased levels of BDNF are associated with diabetes and obesity which can be regulated by increasing the insulin sensitivity and glucose tolerance. BDNF mediated signaling through phosphoinositide 3-kinase (PI3K) pathway plays a key role in insulin sensitivity. These beneficial effects of BDNF as antidiabetic agents can be enhanced by the activating BDNFTrkB signaling. These multiple functionality of BDNFTrkB complex relies on the protein-protein interactions where those interactions are important in designing pharmacological targets. This apporach focus the use of BDNF peptides in place of BDNF protein in binding to the TrkB receptor. Protein–peptide docking studies were performed to know the interactions of the TrkB and BDNF peptides. Docking studies were done using ZDOCK pro (Accelrys Discovery studio). The interacting amino acids residues identified at the binding site were THR 306, LYS 308, CYS 5 ASP 370.

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REFERENCES


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