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Editorial

# Enzymes as Biomolecular Drug Targets

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## DESCRIPTION

Most of the enzymes and proteins are controlled by their quaternary structure and also by their relation with in homoand/or hetero-oligomer complexes. From this we can say that the interaction between two proteins that is protein-protein interaction can act as good biological target for achieving maximum therapeutic activity in the subject of drug delivery. We can achieve this either by blocking the protein function or by modulating the protein function. There a large number of oligomeric structures available in the Protein Data Bank that represents increasing interest in proteins that function as multimeric complexes. In this editorial, we focus on the particular topic of homodimeric enzymes that help act as biological drug targets. There is complex enthusiasm in drugs that inhibit dimerization of a functionally obligate homodimeric enzyme. Peptides and peptidomimetics are the two dimerization inhibitors that are developed. Based on the sequences that are present between protein-protein interactions both peptides and peptidomimetics act at the point of protein-protein interfaces. HIV protease and HIV integrase are the examples of inhibitors. To study the mechanism of action of enzyme inhibitors and binding sites of inhibitors we need to understand the different techniques for different proteins [1].

There are different components in the cell system that possesses different functions in order to maintain cellular metabolism. Macromolecules inside the body interact with other cellular components or with other macromolecular to perform the cellular activities. Most of the proteins inside the cell interact with other proteins or nucleic acid to control and regulate the cellular functions like cell signal transduction, immunocomplex formation, gene expression regulation, and structural component aggregation in the cell [2].

The structural dimensions of the protein surfaces include the protein-protein interactions that help in guiding during the molecular recognition. The divergence in protein complexes makes them more complicated to locate the association process in the general context of mechanisms that regulate protein folding. We can inhibit the dimeric enzymes with the help of ligand binding at their interfaces. The drawback in this process was the properties of the ligand may change during the ligand binding process that makes it difficult to access which results in difficulty of identification of the mechanism of inhibition [3].

After identifying the key residues for dimerization, subsequent task is to identify potential ligand binding sites at the monomer-monomer interface. Small molecules that bind in clefts, preferably near the hot spot residues, can potentially prevent dimerization or disturb the dimeric structure. A wide spread of programs are developed for detecting ligand binding sites in proteins [4]. Some interface grooves and clefts form only upon association with the binding partner; thus, monomer-monomer interfaces are often adaptive. While the pliability and plasticity of a protein-protein interface can provide novel binding sites for little molecules, they also pose a challenge for structure-based pocket detection. Virtual screening (VS) may be a popular computational technique wont to look for novel ligands or lead structures from large (virtual) compound databases [5].

## CONCLUSION

Among all the biological drug targets that have been discovered and identified in this genomic era enzymes have been representing more and more opportunities for regulation of most of the pathways in the cells. This can be achieved due to the new mechanism of inhibition which can be as a result of interfering with the monomeric and dimeric equilibrium.

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