Theoretical studies on the anti-inflammatory activity of hyperoside with the cyclooxygenesa 2 enzyme

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Arachidonic acid and its metabolites (prostaglandins and leukotrienes) are important intracellular messengers which play an important role in pain and inflammation regulatory pathways. Cyclooxygenase-2 (COX-2) is the key enzyme in the biosynthesis of prostaglandins from aracidonic acid, which are key mediators of inflammation. COX-2 catalyzes the conversion of arachidonic acid to prostaglandin H_2, the precursor of PGs and thromboxane.

The aim of our study was to perform molecular docking of hyperoside with the COX-2 enzyme.

A molecular docking study was conducted using the tool known as AutoDockTools 1.5.6. Genetic algorithm parameters were applied for ligand interaction, with 10 runs of this criterion. COX-2 (PDB ID: 1ddx) structure was obtained from PDB database. The resolution of 1svc was 3.0 Å. The ligand structures of hyperoside (CID_5281643) was obtained from PubChem database. The active site of the docking protein was identified utilizing the Computed Atlas for Surface Topography of Proteins. As a standard was taken diclofenac sodium. We applied the following classification of selectivity: inhibition concentration (IC)50<0.001 mM (high selective); 0.05>IC50>0.01 (medium selective); IC50>0.05 mM (low selective).

The hyperoside had a high value of free energy value (-12.36 kcal/mol), whereas IC50 was 0.0000009 mmol, so hyperoside belong to high selective inhibitor. Comparing result with diclofenac sodium standard, the affinity of hyperoside was 53% more than of diclofenac sodium (-5.76 kcal/mol, IC50 – 0.06 mmol).

It was established that hyperoside is a potentially medium selective inhibitor of COX-2. So, the extract with hyperoside can be applied for developing a new anti-inflammatory drugs.