Ligands bind to G Protein-Coupled Receptors (GPCR) and induce a change in a remote part of the protein that leads to the binding of G proteins and a cellular response. Numerous structures of GPCRs show that the changes in the ligand pocket are very small compared to the much larger changes in the G protein site. This challenges the notion that ligand binding alone is responsible for the changes in the protein that lead to G protein binding and receptor activation. A possible involvement of internal waters, present in all GPCRs, could provide a mechanism for the connection between the ligand-binding pocket and the G protein-binding site. Using MD simulations and an enhanced inhomogenous fluid solvation theory we estimate the binding free energy of the internal waters and their contribution to the dynamics of protein complexes. The internal waters and the adjacent protein residues create a dynamical network of hydrogen bonds. Graph theoretical analysis of the network in a system with an antagonist shows that specific residues interrupt the network creating two isolated clusters preventing effective inter-site communication. In contrast, the presence of an agonist creates multiple pathways and increases network connectivity, establishing a possible allosteric link between the ligand and G protein.