**Tata Institute of Fundamental Research**
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# Webinar

## Understanding bio-molecular recognition of various biological systems

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In solution, molecules collide and interact with one another many number of times and get dispersed in most of the cases because the products formed by these interactions are short-lived, weak and energetically non-favourable. But when the featured surface of one molecule is complementary to those of its neighbour/partner i.e, when the generated attractive forces between the molecule and its partner outweigh the repulsive forces and the entropic costs of being together, then long-lived, stronger and energetically favourable interactions are established leading to formation of stable complexes. These specific complexes have significant biological significance. Molecular recognition refers to the process of such binding or aggregation between the molecules. Understanding the forces and mechanisms that drive the formation of complexes will yield many insights and allows rational design of molecules that interact in desired ways.

I will start my discussion by showing the heterogeneity among carbohydrate force fields for an amylose dodecamer. Out of 4 commercially available force fields, we observe GLYCAM06 and CHARMM show good agreement with experimental observations. Using GLYCAM06, parameters for LacNAc a bi-saccharide were obtained, which helped us in observing successful binding of LacNAc and Galectin-3 protein. By following similar protocol, we simulated bound complex of Galectin with LacNAc derived ligand. By analysing both simulation data, we are able to pin point the reason by which LacNAc derivative shows increased affinity to Galectin-3. Next, I will talk about how small length peptides made up of 10 β-amino-acids disrupt a bacterial mimic bi-layer to cause water poration through it. Drug resistance is a common phenomenon where a target protein decreases its binding affinity with a drug molecule by mutating a single or multiple amino-acid residues. One such know mutation is gate-keeper mutation of T338M residues in Src-kinase protein. Here I discuss about a computational work flow where we design and characterise a set of kinase inhibitors which are derived from second generation kinase inhibitor RL-45. These derived inhibitors show increased binding affinity with wild type and mutated Src-kinase.

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***11:00 AM***