SCOPE OF THE PRESENT WORK

Diabetes mellitus (type II), a chronic metabolic disorder, has become a major health challenge worldwide. Currently a variety of oral medications such as sulfonylureas, thiazolidinediones, meglitinides, biguanides and alfa-glucosidase inhibitors, which can be used alone or in combination to achieve the desired hypoglycemic effect, are available. Most of the reported studies on these drugs include hypoglycemic activity, mechanism of action, therapeutic uses, adverse effects and other pharmacokinetic parameters.

During recent years, there has been an increased emphasis on the biopharmaceutical properties and detailed physico-chemical characterization of various kinds of drug interactions in controlling the biological activity of pharmaceuticals. Such studies on the studied antidiabetic drugs are not available, although they are widely used therapeutic agents. The very poor aqueous solubility of most antidiabetic drugs give rise to potential drug development and drug delivery problems and leads to variable bioavailability. Most antidiabetic drugs are extensively bound to plasma proteins, a small change in the percentage of drug bound can cause large changes in the pharmacologically active free drug and consequent glycemic control. Binding of drugs to glycosylated plasma proteins, present in large amounts in diabetic patients, needs to be thoroughly investigated. Since combination and multiple drug therapy are commonly used in type II diabetes, competitive drug displacement interactions can provide useful information. Perusal of the literature shows that detailed studies on these and other physico-chemical characteristics of the hypoglycemic agents used in the present work are not available. In the present thesis, nine drug samples, taken from five categories of antidiabetic agents, have been selected. Following aspects, which play a key role in drug development and understanding the drug action at molecular level have been studied.

1. Drug estimation
2. Lipophilicity
3. Solubility enhancement
4. Drug-protein interaction
5. Drug-drug interaction