CONCLUSION
Conclusion

The work embodied in this thesis demonstrates the mechanism of action of the CDRI compounds having significant antithrombotic action in various animal models of thrombosis. CDRI test compounds such as 99/353, S000-20, S001-556, S002-329 and S002-333 exhibited antithrombotic activity by interfering at different receptors on platelets. 99/353 seems to act through GPIIb/IIIa receptor antagonism as 99/353 inhibited aggregation by most of the inducers that employ different signaling pathways for platelet activation. 99/353 also possessed significant antithrombotic efficacy better than standard drug aspirin and also exhibited nominal vasodilatory effects and lesser adverse effects on bleeding time.

Semi-synthetic compound S001-556 seems to act through COX mediated pathway, as it significantly inhibited arachidonic acid induced platelet aggregation. Moreover, it was more effective than aspirin in various animal models of thrombosis.

S000-20 inhibited both collagen and thrombin mediated platelet aggregation without having any effect on clotting factors. It has also been found to be quite effective against intravascular thrombosis in various animal models of thrombosis. Moreover, analogs of S000-20 had more inhibitory effect against thrombin and collagen mediated platelet aggregation.

Other test compounds like S002-329 and S002-333 were also found to act by interfering with collagen mediated platelet activation and exhibited potent antithrombotic activity in various animal models. Herbal product, Curcuma oil also exhibited anti-platelet activity which conferred antithrombotic actions. It also enhanced the inhibitory effect on platelets with increase in the duration of treatment. The test compounds thus possessed promising antithrombotic effects and seem to be novel therapeutic agent to prevent thrombosis related cardiovascular disorders.