INTRODUCTION
Intravascular thrombosis is one of the major cardiovascular complications associated with higher rate of morbidity and mortality, both in the developed and developing countries. Epidemiological data shows that there is constant increase in death rate in developing countries due to these cardiovascular complications. Intravascular thrombosis leads to other pathological complications such as myocardial infarction (MI), ischemia, deep vein thrombosis (DVT), unstable angina, coronary artery disease (CAD), restenosis, stroke and disseminated intravascular coagulation (DIC). Various pathological conditions such as atherosclerosis, hyperlipidemia, hypertension, hyperhomocystenemia predispose a person to intravascular thrombosis while aging and smoking habit also promote intravascular thrombosis.

Onset of thrombosis is a complex phenomenon; it seems to be initiated as a result of vascular dysfunction. Primarily plasma proteins, blood cells, vascular endothelium and blood rheology play also a significant role in the development of thrombosis. **Rudolf Virchow (1858)** suggested that dysregulation in following three factors leads to intravascular thrombosis.

1. Blood flow alterations/pertubations
2. Vascular endothelial dysfunction
3. Hypercoagulability

Vascular endothelium actively maintains the anti-adhesive surface of blood vessels by releasing nitric oxide (NO), PGI₂ (prostacyclin) and by expressing thrombomodulin. Endothelial denudation due to vascular injury or rupture of
atherosclerotic plaque exposes the sub endothelial collagens and vWF along with decrease in NO and PGJ2 availability leading to platelet aggregation and finally thrombus formation.

Simultaneously clotting factors also get activated by the tissue factor released from the injured site, leading to the generation of thrombin and formation of insoluble fibrin to stabilize the thrombus. With improved understanding of the pathogenesis of thrombosis, newer targets are being identified which might help in the prevention of intravascular thrombosis. Total prevention of thrombotic complications with the existing therapies is thus not as effective as desired. Need to develop newer anti-thrombotic molecules with better safety and efficacy profile using novel approaches to newly identified targets is therefore approachable. Present study was undertaken to delineate the mechanism of action and efficacy of newly identified anti-thrombotic compounds at Central Drug Research Institute from synthetic and natural sources in various in vitro test systems and animal models.