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Atherothrombosis is one of the main causes of death worldwide. There are two stages of this process. First is a formation of atherosclerotic lesions which are a consequence of inflammatory alterations in structure of vascular wall. The second stage - thrombosis - occurs when atherosclerotic lesion becomes unstable and it ruptures. As a consequence of this process blood components responsible for blood clotting activate and thrombus starts to form in the lesion site. This process may finally lead to occlusion of blood vessel and cessation of blood flow. This is how ischemic strokes and myocardial infarctions occur. In order to decrease a probability of such an incident, various therapies have been implemented. Many of them are based on compromising of physiological mechanisms of blood clotting to limit the formation of thrombus. These therapies however, have side-effects. Better understanding of molecular mechanisms of atherothrombosis may lead to inventing of therapies devoid of the side effects. This project is aimed at understanding of one of the mechanisms which are behind the disease. There are strong evidence that one of the proteins located in blood platelet membranes plays a role in this process. Until now the role of this protein in blood platelets is barely understood. During implementation of the project we plan to evaluate what is the exact role of this protein during atherosclerosis and thrombosis. To achieve this goal we will use systems which mimic blood flow and which allow to study processes which occur during early phases of atherosclerotic lesions formation as well as during thrombus formation. Data obtained in the artificial systems will be validated in animal models of atherosclerosis and thrombosis.