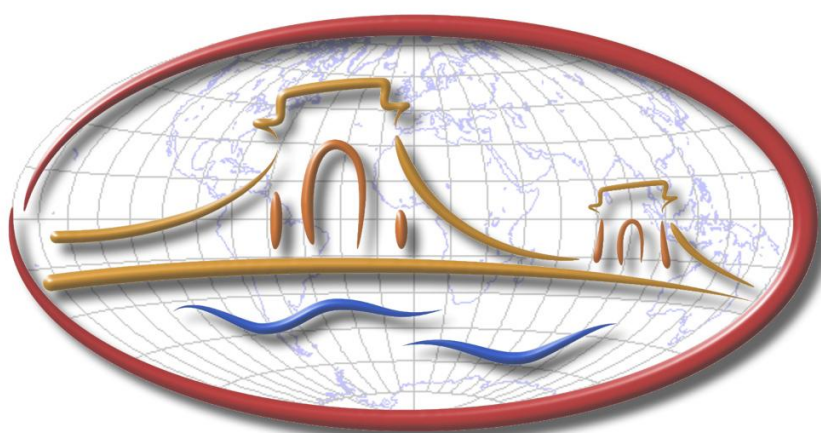




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# Bridges in Life Sciences 10th Annual Scientific Conference



**RECOOP HST ASSOCIATION**

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Centre for Advanced Materials and Nanotechnology



## Platelets as regulators of plasminogen activation system

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**Key words:** platelet, plasminogen activation, plasminogen activator inhibitor-1 (PAI-1), PAI-1 activity determination.

**Introduction.** Platelets bind plasminogen, its activators and contain PAI-1. Platelet membrane can be considered as a catalytic surface for plasminogen activation. The aim of this work is to investigate kinetic of plasminogen activation by tissue plasminogen activator (t-PA) in the presence of platelets and define inhibitory activity of platelet PAI-1.

**Methods.** Human platelets were isolated from blood of healthy volunteers. Human Glu-plasminogen was purified by lysine-sepharose chromatography. The rate of plasminogen activation by tPA was estimated by activity of new formed plasmin. Activity of platelet PAI-1 was determined by the method developed in our lab. It is based on the inhibition of plasminogen activation by tPA using bovine desAB-fibrin as stimulator.

**Results.** In the presence of resting platelets the catalytic efficiency of Glu-plasminogen activation increased in 8 times mainly thanks of Km decrease ( $0.184 \pm 0.066 \mu\text{M}$  compared with  $1.117 \pm 0.086 \mu\text{M}$  in cell free system), catalytic constant had no significant change. Thrombin treatment of platelets accompanied with further increase of catalytic efficiency of plasminogen activation more than in 30 times. Stimulating effect of platelets was not related with the presence of endogenous plasminogen or its activators. PAI-1 activity of lysates of resting platelets and releasates of thrombin- and collagen-stimulated platelets was  $2.04 \pm 0.70$ ,  $1.26 \pm 0.60$  and  $0.75 \pm 0.36 \text{ IU}/10^8$  cells, respectively.

**Conclusion.** Platelets stimulate plasminogen activation at physiological concentration of tPA. Platelets could be considered as sites of plasminogen activation and local plasmin generation. Platelets contain and release the active form of PAI-1 during activation. Quantitative assessment of its activity suggests that platelet PAI-1 makes a significant contribution to limiting of plasminogen activation and further fibrinolysis. The proposed method for quantifying PAI-1 activity in plasma and platelets can be used for evaluation of fibrinolytic potential in patients with cardiovascular diseases.

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Resrach protocols were approved by the Ethical Committee of Palladin Institute of Biochemistry of NASU (from the 4th of June 2014, protocol №2).

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