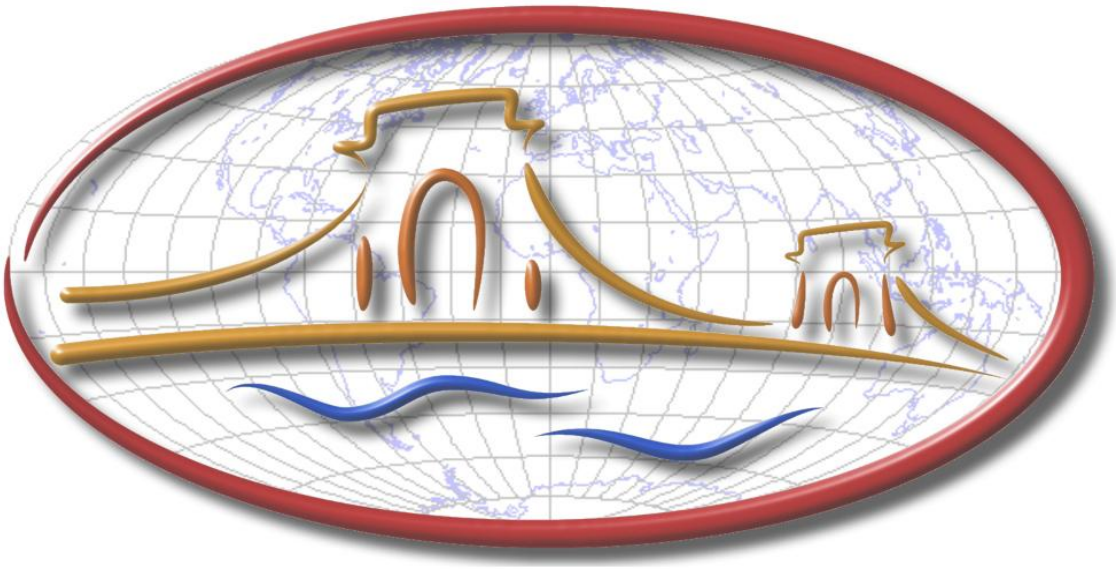

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Glu- and Lys-forms of plasminogen distinctly affect platelet aggregation, secretion and phosphatidylserine exposure

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Introduction. Plasminogen/plasmin system is known for its ability to support hemostatic balance of blood. However, plasminogen may be considered as an adhesive ligand and in this way affect the functioning of blood cells. The aim of this work was to investigate the influence of Glu- and Lys-form of plasminogen on the functioning of human platelets: aggregation, α-granule secretion and phosphatidylserine exposure.

Methods. Human platelets were obtained from human platelet-rich plasma by gel-filtration on Sepharose 2B. To estimate aggregation, platelets were stimulated with thrombin or collagen. Platelet secretion was studied using P-selectin antibodies conjugated with phycoerythrin. Phosphatidylserine exposure on the platelet surface was evaluated with FITC-conjugated annexin A5. Flow cytometry was used in both cases.

Results. Glu- and Lys-plasminogen have different impact on the platelet functioning. Exogenous Lys-plasminogen inhibits platelet aggregation, suppresses platelet α-granule secretion, but has no significant effect on phosphatidylserine exposure. Glu-plasminogen causes no effect on platelet aggregation but stimulates platelet secretion and increases phosphatidylserine exposure on the surface of thrombin- and collagen-activated human platelets.

Discussion. The obtained results showed the influence of plasminogen forms on the principal events of platelet functioning. Glu-plasminogen can be considered as a co-stimulator of agonist-induced platelet secretion and procoagulant surface formation. Meanwhile effects of Lys-plasminogen are probably directed at platelet-platelet interactions and not related to agonist-stimulated pro-apoptotic changes.

Conclusion. The observed different effects of Glu- and Lys-plasminogen on platelet aggregation, secretion and phosphatidylserine exposure can be explained by their structural peculiarities. The results should be taken into account at the treatment of cardiovascular patients, as both plasminogen forms appear to be natural modulators of the platelet function.

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Ethical approval. Research protocols were approved by the Ethical Committee of Palladin Institute of Biochemistry of NASU (from 3rd of November 2014, protocol N 10).