Abstracts of the

BIO 2014 Congress

1st Congress of the Polish Biochemistry, Cell Biology, Biophysics and Bioinformatics

Warsaw, Poland
September 9th–12th 2014

49th Meeting of the Polish Biochemical Society

12th Conference of the Polish Society for Cell Biology

16th Meeting of the Polish Biophysical Society

7th Meeting of the Polish Bioinformatics Society

Abstracts of the

BIO 2014 Congress

Warsaw, Poland
September 9th–12th 2014

Honorary Patronage:

HONORARY PATRONAGE
OF THE MAYOR OF WARSAW

Mayor of Warsaw,
Hanna Gronkiewicz-Waltz

Mazovia.
heart of Poland

Marshal of the Mazowieckie
Voivodeship,
Adam Struzik

UNIVERSITY
OF WARSAW

Rector of University of Warsaw,
Professor Marcin Pałys

Warsaw University of Life Sciences
SGGW

Rector of Warsaw University of
Life Sciences,
Professor Alojzy Szymański

P7.42

Personalized therapy of chronic lymphocytic leukemia

Małgorzata Rogalińska[1], Paweł Góralski[2], Jerzy Z. Błoński[3], Jan Barciszewski[4], Karolina Tarnowska[1], Marta Wawro[1], Katarzyna Woźniak[5], Aneta Kocewa-Chyła[6], Henryk Piekarski[2], Tadeusz Robak[3], Zofia M. Kilianska[1]

[1]University of Lodz, Faculty of Biology and Environmental Protection, Department of Cytobiochemistry, Łódź, Poland; [2]University of Lodz, Faculty of Chemistry, Department of Physical Chemistry, Łódź, Poland; [3]Medical University of Lodz, Department of Hematology, Łódź, Poland; [4]Polish Academy of Science, Institute of Bioorganic Chemistry, Poznań, Poland; [5]University of Lodz, Faculty of Biology and Environmental Protection, Department of Molecular Genetics Łódź, Poland; [6]University of Lodz, Faculty of Biology and Environmental Protection, Department of Thermobiology, Łódź, Poland
e-mail: Małgorzata Rogalińska <gosiar@wp.pl>

B-chronic lymphocytic leukemia (CLL) is a common type of leukemia in Europe and North America with growing incidence of disease for younger people.
The general goal of therapies directed towards lymphoproliferative disorders, like CLL, is elimination of leukemic cells by apoptosis induction. Due to unpredictable clinical picture and personal patient's differences in anti-cancer treatment sensitivity, establishing optimal therapy for this type of leukemia sometimes reflects difficulties. Therefore, a special importance in case of patient's resistance to therapy is to search drug administration with potency to eliminate *in vitro* leukemic cells from patient's peripheral blood.
The comparative analysis of CLL cells incubated with anticancer agents (purine analogs combine with alkylating agent mafosfamide or monoclonal antibody – rituximab) by cytometric analysis (cell viability, apoptosis level), thermal profiles by differential scanning calorimetry, and protein expression related to apoptosis (PARP and Mcl-1), could be useful in the most effective drug treatment choice or even monitoring patient's treatment *in vivo*.
Because of personal differences between patients in disease dynamics and the response to drugs registered to cure of this type of leukemia, it seems to be important to personalize therapy by choosing potentially effective type of treatment with ability to induce apoptosis before its *in vivo* administration to reduce ineffective patient's response to anticancer therapy.

P7.43

Mechanism of inhibition of human platelet aggregation by Lys-plasminogen

Yana M. Roka-Moiia, Dmytro D. Zhernossekov

Palladin Institute of Biochemistry of the National Academy of Sciences of Ukraine, Department of Enzyme Chemistry and Biochemistry, Ukraine
e-mail: Yana Roka-Moya <yanulia@bk.ru>

Plasminogen/plasmin system takes part not only in fibrinolysis but also in regulation of functional state of different cells. Platelet membrane can provide a surface for assembly of plasminogen and its activators. On the surface of certain blood cells Glu-plasminogen is transformed into Lys-form, which possesses open conformation and can be more easily activated with the plasmin formation. We showed that exogenous Lys-plasminogen but not its native Glu-form inhibits platelet aggregation stimulated by ADP, thrombin and collagen. The aim of this work is to define the role of certain structural domains of plasminogen molecule in the observed inhibitory effect.One of the possible mechanisms of plasminogen interaction with the surface receptors is the binding of carboxyl-terminal lysines by lysine-binding sites (LBS) of plasminogen molecule. In our experiments lysine analogue, 6-aminohexanoic acid abolishes above mentioned inhibitory effect.Serine protease inhibitor, aprotinin (5.5 IU/ml) does not make any influence on the inhibitory effect of Lys-plasminogen during thrombin-induced platelet aggregation. The used concentration of aprotinin has no effect on platelet aggregation.Preincubation of washed platelets with K1-3, K4 and K5 taken into equimolar concentration with Lys-plasminogen completely abolishes inhibitory effect of the last one. The plasminogen kringle K5 added to the reaction mixture (washed platelets and 1.2 μM Lys-plasminogen) even at concentration 0.12 μM recovers platelet aggregation till 80%, whereas K1-3 and K4 at this concentration reach only near 60% as compared as control aggregation level. Plasminogen kringles K1-3, K4 and their combination have no influence on aggregation of washed platelets. There was no change in platelet aggregation in case of mini-plasminogen (K5 plus serine protease domain), where catalytic activity was inhibited by aprotinin or p-nitrophenyl-p'-guanidine benzoate.The observed effect of plasminogen kringles on the inhibition of platelet aggregation by Lys-plasminogen let us suggest that LBS of plasminogen kringles provide the plasminogen binding to the adhesive proteins of platelet surface (e.g. thrombospondin, vitronectin and fibrinogen). These proteins are secreted from α-granules and remain bound to platelet surface. The binding of plasminogen kringles to these proteins may lead to the disturbance of protein-protein interaction which is the necessary condition for efficient platelet aggregation.

Abstracts of the

BIO Congress

Warsaw, Poland
September 9th–12th 2014

Contents