

Abstracts of the

BIO 2014 Congress

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

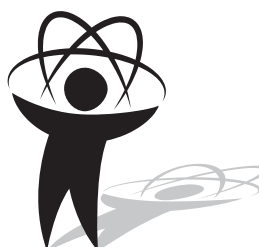
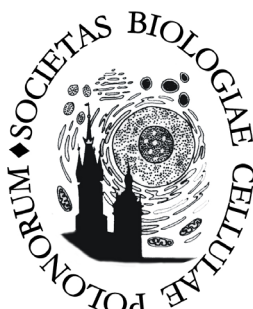
**Warsaw, Poland
September 9th–12th 2014**

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
Rector of Warsaw University of
Life Sciences,
Professor Alojzy Szymański

P7.42

Personalized therapy of chronic lymphocytic leukemia

Małgorzata Rogalińska¹, Paweł Góralski², Jerzy Z. Błoński³, Jan Barciszewski⁴, Karolina Tarnowska¹, Marta Wawro¹, Katarzyna Woźniak⁵, Aneta Kocewa-Chyła⁶, Henryk Piekarski², Tadeusz Robak³, Zofia M. Kilińska¹

¹University of Lodz, Faculty of Biology and Environmental Protection, Department of Cytobiochemistry, Łódź, Poland; ²University of Lodz, Faculty of Chemistry, Department of Physical Chemistry, Łódź, Poland; ³Medical University of Lodz, Department of Hematology, Łódź, Poland; ⁴Polish Academy of Science, Institute of Bioorganic Chemistry, Poznań, Poland; ⁵University of Lodz, Faculty of Biology and Environmental Protection, Department of Molecular Genetics Łódź, Poland; ⁶University 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 μ M Lys-plasminogen) even at concentration 0.12 μ M 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

Plenary Lectures	1
Parnas Lecture	1
Benedykt Wladyka	
FEBS National Lecture	4
Aaron Ciechanover	
Session 1. Stem Cells Bioengineering for Therapeutic Translation	5
organized by: Leonora Bużańska, Alicja Józkwicz	
Session 2. Heterogeneity, Plasticity and Microenvironment of Cancer	25
organized by: Janusz Siedlecki, Magdalena Chechlińska	
Session 3. Cytoskeleton, Intracellular Transport and Cell Motility	61
organized by: Michał Witt, Andrzej Kasprzak	
Session 4. Bioinformatics and Computational Biology	74
organized by: Janusz Bujnicki	
Session 5. Lipids: Metabolism and Biological Functions	108
organized by: Ewa Świeżewska	
Session 6. Genomics and Epigenomics	116
organized by: Marta Koblowska	
Session 7. Protein Functions in Cellular Signaling	142
organized by: Marta Miączyńska	
Session 8. Development and Ageing	180
organized by: Maria A. Ciemerych-Litwinienko, Ewa Sikora	
Session 9. Plant Cell Biology and Biochemistry	199
organized by: Agnieszka Mostowska, Przemysław Wojtaszek	
Session 10. Electrophysiology and Electrochemical Sensors	221
organized by: Krzysztof Dołowy	
Session 11. EMBO and Poland: Metabolic Disorders: External and Internal Signaling	226
organized by: Dorota Włoga, Agnieszka Dobrzyń	
Session 12. Cellular and Molecular Neurobiology – New Ideas and New Technologies	241
organized by: Witold Konopka, Marta B. Wiśniewska	
Session 13. Biospectroscopy and Oxidative Stress	253
organized by: Wiesław Gruszecki	
Session 14. Bioenergetics	267
organized by: Adam Szewczyk, Jerzy Duszyński, Lech Wojteczak	
Session 15. Biotechnology	281
organized by: Joanna Kruszewska, Katarzyna Tońska	
Symposium EMBO and Poland	318
organized by: Leszek Kaczmarek	
Authors Index	327