SESSION VII

FUNCTIONS OF BLOOD
HEMOSTASIS

Thursday (September 16, 2021; 15:30 – 18.00)

Chair:

Prof. Ewa Chabielska
Department of Biopharmacy, Medical University of Bialystok, Poland

Assoc. Prof. Andrzej Mogielnicki
Department of Pharmacodynamics, Medical University of Bialystok, Poland

DETAILED SESSION VII SCHEDULE

Opening lectures (Thursday, September 16, 2021; 16:30 – 17:30; *virtual stream A*)

S7.L1 THE ROLE OF PLATELET JUNCTION ADHESION MOLECULE-A IN HAEMOSTASIS AND ATHEROSCLEROSIS. **T. Przygodzki** (Department of Blood Clotting Disorders, Department of Biomedical Sciences, Medical University of Lodz, Lodz, Poland).

S7.L2 STRIATIN - A NOVEL MEDIATOR OF STEROID HORMONES EFFECTS IN HEMOSTASIS. **A. Gromotowicz-Poplawska[1], N. Marcinczyk[1], R. Flaumenhaft[2,3], J.R. Romero[3,4], G.H. Williams[3,4], E. Chabielska[1]** ([1]Department of Biopharmacy, Medical University of Bialystok, Bialystok, Poland, [2]Division of Hemostasis and Thrombosis, Beth Israel Deaconess Medical Center, Boston, USA, [3]Harvard Medical School, Boston, USA, [4]Division of Endocrinology, Diabetes, and Hypertension, Brigham and Women's Hospital, Boston, USA).

Oral presentations (Thursday, September 16, 2021; 17:30 – 18:00; *virtual stream A*)

S7.L3 THE EFFECTS OF PROTAMINE SULFATE ON THE HEMOSTASIS, CARDIOVASCULAR AND RESPIRATORY FUNCTIONS IN DIFFERENT ANIMAL MODELS. **J. Miklosz[1],B. Kalaska[1], P. Podlasz[2], M. Chmielewska-Krzesinska[2], M. Zajaczkowski[3], M. Rusak[4], A. Kosinski[3], D. Pawlak[1], A. Mogielnicki[1]** ([1]Department of Pharmacodynamics, Medical University of Bialystok, Bialystok, Poland, [2]Department of Pathophysiology, Forensic Veterinary Medicine and Administration, University of Warmia and Mazury in Olsztyn, Olsztyn, Poland, [3]Department of Clinical Anatomy, Medical University of Gdansk, Gdansk, Poland, [4]Department of Haematological Diagnostics, Medical University of Bialystok, Bialystok, Poland).

Session summary

Poster presentations (Thursday, September 16, 2021; 15:30 – 16:25; *virtual stream C*)

S7.P1 CONTRIBUTION OF GASEOUS TRANSMITTERS TO THE OZONE EFFECT ON BLOOD OXYGEN TRANSPORT FUNCTION UNDER HYPOCAMPIC CONDITIONS. **V. Zinchuk[1], E. Biletskaya[1], A. Muravyov[2]** ([1]Grodno State Medical University, Grodno, Belarus, [2]Yaroslavl State Pedagogical University named after K.D. Ushinsky, Yaroslavl, Russia).

S7.P2 THE INFLUENCE OF ANTIMICROBIAL NEUTROPHIL EXTRACT AND PENTOXIFYLLINE ON OVINE NEUTROPHILS ISOLATED DURING THE INSERTION TITANIUM IMPLANT IN A SHEEP MODEL. **J. Zdziennicka[1], J. Wessely-Szponer[1], T. Szponder[2], M. Latalski[3]** ([1]Sub-Department of Pathophysiology, Department of Preclinical Veterinary Sciences, Faculty of Veterinary Medicine, University of Life Sciences, Lublin, Poland, [2]Department and Clinic of Animal Surgery, Faculty of Veterinary Medicine, University of Life Sciences, Lublin, Poland, [3]Department of Pediatric Orthopedics, Medical University, Lublin, Poland).

S7.P3 IMPACT OF FUNCTIONALIZED SILVER NANOPARTICLES ON AGGREGATION OF HUMAN BLOOD PLATELETS. **J. Hajtuch[1], E. Tomczyk[2], M. Wojcik[2], M.J. Santos-Martinez[3], I. Inkielewicz-Stepniak[1]** ([1]Department of Pharmaceutical Pathophysiology, Medical University of Gdansk, Gdansk, Poland, [2]Faculty of Chemistry, University of Warsaw, Warsaw, Poland, [3]School of Pharmacy and Pharmaceutical Sciences, Trinity College Dublin, Dublin 2, Ireland).

S7.P4 NEUTROPHIL-TO-LYMPHOCYTE RATIO AS A FACTOR PREDICTING RADIOTHERAPY INDUCED ORAL MUCOSITIS AND OVERALL SURVIVAL IN HEAD NECK CANCER PATIENTS TREATED WITH RADIOTHERAPY. **I. Homa-Mlak[1], A. Brzozowska[2], R. Mlak[1], A. Szudy-Szczyrek[3], T. Malecka-Massalska[1]** ([1]Department of Human Physiology, Medical University of Lublin, Lublin, Poland; [2]Department of Oncology, Medical University of Lublin, Lublin, Poland, [3]Department of Hematooncology and Bone Marrow Transplantation, Medical University of Lublin, Lublin, Poland).

S7.P5 UTILITY OF THE PLATELET-ENDOTHELIAL CELL ADHESION MOLECULE 1 (PECAM-1) AS A MARKER OF PLATELET ACTIVITY IN THE FLOW CHAMBER MODEL OF THROMBOSIS IN ANIMAL AND HUMAN STUDY. **N. Marcinczyk[1], T. Misztal[2], A. Gromotowicz-Poplawska[1], T. Rusak[2], E. Chabielska[1]** ([1]Department of Biopharmacy, Medical University of Bialystok, Bialystok, Poland, [2]Department of Physical Chemistry, Medical University of Bialystok, Bialystok, Poland).

S7.P6 THE ROLE OF LDG CELLS AS A POTENTIAL FACTOR IN THE DEVELOPMENT AND INTENSITY OF INFLAMMATION IN PSORIASIS. **W. Domerecka[1], I. Homa-Mlak[1], R. Mlak[1], A. Wilinska[2], T. Malecka-Massalska[1]** ([1]Department of Human Physiology, Medical University of Lublin, Lublin, Poland, [2]Department of Clinical Genetics, Medical University of Lublin, Lublin, Poland).

S7.P7 CHANGES IN ARTERIAL OXYGEN SATURATION IN HEALTHY PERSONS DURING BACK MASSAGE PROCEDURE. **P. Radziejowski[1], M. Radziejowska[1], V. Dychko[2], O. Romaniv[1]** ([1]Department of Innovations and Safety Management Systems, Faculty of Management, Czestochowa University of Technology, Czestochowa, Poland, [2]Department of Physical Therapy, Physical Education and Biology, Donbass State Pedagogical University, Slavyansk, Ukraine).

S7.L1

THE ROLE OF PLATELET JUNCTIONAL ADHESION MOLECULE-A IN HAEMOSTASIS AND ATHEROSCLEROSIS

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Junctional adhesion molecule-A (JAM-A) is a transmembrane protein which belongs to the immunoglobulin superfamily of cell adhesion molecules. JAM-A is present in epithelial cells, endothelial cells, leukocytes and blood platelets. In epithelial and endothelial cells it takes part in formation of tight junctions. In these structures, molecules of JAM-A located on adjacent cells form homodimers and thus take part in stabilization of cellular layer integrity. In leukocytes, JAM-A was shown to play role in their transmigration through the vascular wall. Paradoxically, the function of JAM-A in blood platelets, where it was primarily discovered, is much less understood. Blood platelets are most often considered in terms of two biological processes: haemostasis and development of atherosclerosis. Existing evidence allow to assume that platelet pool of JAM-A takes part in both of them. The studies of platelet JAM-A involvement in haemostasis provide ambiguous results. Functional blockade of homophilic interactions of JAM-A inhibited platelet aggregation induced by selected agonists. Preliminary studies suggest that such functional blockade decreased thrombus formation at high shear rates. On the contrary, genetic deletion of JAM-A in mouse platelets resulted in their hyper-reactivity. This was explained by the finding that JAM-A allowed to maintain the αIIbβ3 integrin in a quiescent state. Therefore, the protein seems to play a complex, plausibly regulatory role in platelet aggregation. The potential role of platelet JAM-A in the process of development of atherosclerosis is substantiated by several observations. Upon endothelium activation, JAM-A translocates from tight junctions to the luminal surface of the vascular wall. There, it can facilitate recruitment of flowing cells to the vascular wall. Such a phenomenon has been shown for monocytes and for platelets. The functional blockade of homophilic interactions of JAM-A diminished platelet adhesion to inflamed endothelium in static *in vitro* conditions as well as *in vivo*. What is more, JAM-A has been shown to take part in deposition of pro-atherogenic platelet-derived microparticles on inflamed endothelium. Finally, the functional blockade of JAM-A decreased growth of atherosclerotic plaques and prolonged survival in mouse model of atherosclerosis. Therefore JAM-A, similarly to other molecules such as P- selectin, seems to be a factor connecting platelets and inflamed vascular wall in the context of atherogenesis. Understanding of the role of platelet JAM-A in haemostasis and atherosclerosis may lead to development of novel antithrombotic anti-atherosclerotic strategies.

S7.L2

STRIATIN - A NOVEL MEDIATOR OF STEROID HORMONES EFFECTS IN HEMOSTASIS

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Recent data suggest that striatin, a caveolin-1-binding protein, is a mediator of steroid hormones effects in cardiovascular system (CVS) and serves as a novel link between the actions of the mineralocorticoid (MR) and estrogen (ER) receptors. It was shown that striatin colocalizes with the MR and that MR activation increases striatin levels in endothelial cells. On the other hand, lowering striatin levels in endothelial cells reduces aldosterone, MR-dependent nongenomic signalling. A similar role in nongenomic steroid receptor signaling has been described for striatin ER-dependent nongenomic effects, suggesting that striatin is involved in the estrogen-mediated protection of arteries after injury. The contribution of striatin to the nongenomic steroid receptor signalling in CVS is well documented, however the role of striatin in hemostasis is still unknown. Although, there are some data suggesting a potential role of striatin in aldosterone-mediated thrombotic response. It was demonstrated that individuals who carry rs2540923, a single nucleotide polymorphic gene variant of striatin, exhibit salt sensitivity of blood pressure (BP). A mouse model of striatin deficiency showed that the mechanisms for salt sensitivity of BP is related to reduced striatin levels, increased aldosterone levels, enhanced vasoconstriction, decreased vascular relaxation and reduced eNOS expression. We showed previously in animal models of thrombosis, that increased aldosterone levels enhances thrombotic response in the mechanism related to platelet and coagulation activation, fibrinolysis inhibition, reduced eNOS expression and vascular relaxation as well. These results suggest that in humans and rodent models, striatin deficiency plays a role in vascular thrombotic response mediated by excess aldosterone. Recently, we showed that striatin deficiency in mice was associated with increased aldosterone level and significant increases in laser-induced thrombotic process, in a mechanism that was likewise associated with increased platelet accumulation and fibrin deposition. These results demonstrate a novel protective role of striatin in aldosterone-mediated hemostatic effects and suggest that subjects who carry the polymorphic striatin gene variant may have a procoagulant phenotype and as such are at increased risk of thrombotic events.

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S7.L3

THE EFFECTS OF PROTAMINE SULFATE ON THE HEMOSTASIS, CARDIOVASCULAR AND RESPIRATORY FUNCTIONS IN DIFFERENT ANIMAL MODELS

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Protamine-induced thrombocytopenia is an immuno-hematological disorder described in patients treated with heparin and protamine during cardiopulmonary bypass. It was proposed that protamine and heparin form multimolecular complexes which may induce production of platelet-activating antibodies, and thus severe thrombocytopenia and thromboembolic complications. However, the link between protamine, heparin, platelets and thrombosis is not clear. The aim of the study was to elucidate the effect of protamine and its heparin complexes on platelets and to investigate its possible early and late thromboembolic complications. All procedures involving animals were approved by the Local Ethical Committee. We explored the toxicity of protamine and its complexes with heparin in zebrafish and rodents. Male Wistar rats and BALB/c mice were divided into 4 groups treated with vehicle, heparin, protamine alone and together with heparin once a week for 5 weeks. The bone marrow and the heart histology, platelet count and aggregation in blood, activated partial thromboplastin time (aPTT) in plasma, cardiac troponin T type 2, P-selectin, thrombopoetin, platelet factor 4 and β-thromboglobulin concentrations in serum were assessed in mice. Thrombus weight, platelet count and aggregation in blood, aPTT, prothrombin time, fibrinolysis indicators, D-dimers, fibrinogen, prostacyclin metabolite and anti-Xa activity in plasma were assessed in rats with electrically induced arterial thrombosis. In the acute experiment, platelet aggregation, their number and cardiovascular and respiratory functions were evaluated during one hour from single administration of tested agents. The involvement of nitric oxide, cationicity of protamine and hERG channels in the above effects was investigated. We found a short-term antiplatelet activity and long-term platelet-independent antithrombotic activity of protamine. Protamine and heparin complexes do not cause cardio-respiratory failure, but an overdose of protamine may affect blood pressure and respiratory parameters. Above effects seems to be charge-dependent and involve enhanced release of nitric oxide.

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S7.P1

CONTRIBUTION OF GASEOUS TRANSMITTERS TO THE OZONE EFFECT ON BLOOD OXYGEN TRANSPORT FUNCTION UNDER HYPOCAPNIC CONDITIONS

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Ozone (O_3) improves tissue oxygenation that enable to use it in hypoxic conditions, which are often accompanied by hypocapnia. The contribution of gaseous transmitters to the effect of ozone (6 mg/L) on blood oxygen transport function under hypocapnic conditions (4.2% CO_2; 5.3% O_2, 90.5% N_2) *in vitro* experiments at exposure of 30 min was studied. The following parameters of blood oxygen transport function were determined: oxygen partial pressure (SO_2), blood oxygen saturation (SO_2), hemoglobin affinity for oxygen according p50 value (with pO_2 equal 50%). Ozonised isotonic sodium chloride solution in a volume of 1 ml and donors of nitrogen monoxide and hydrogen sulfide gaseous transmitters were added to blood samples (nitroglycerin at a final concentration of 0.05 mmol/L and sodium hydrosulfide at a final concentration of 0.38 mmol/L). Introducing O_3 into the blood samples led to an increase in the main parameters of the blood oxygen transport function, such as SO_2, pO_2, $p50_{real}$, $p50_{stand}$ and the shift of the oxyhemoglobin dissociation curve to the right compared to control group. When treated with hypocapnic gas mixture, these parameters decreased compared to the control. Incubation of the blood samples in hypocapnic conditions enhanced the effect of O_3 on blood oxygen transport function parameters. Nitroglycerin caused a significant increase in this effect under given conditions of pO_2 and SO_2 that is parameters increased compared to the group of preliminary hypocapnia with the ozone addition. The $p50_{real}$ parameter increased and the shift of the oxyhemoglobin dissociation curve to the right became more pronounced. Sodium hydrosulfide did not have this effect. Concentration of NO_3^-/NO_2^- and H_2S in blood plasma at action of O_3 under conditions of hypocapnia did not change compared to the group in which only ozone was administered. The addition of nitroglycerin and sodium hydrosulfide under these conditions led to a significant increase of NO_3^-/NO_2^- and H_2S compared with group ozonised under hypocapnic conditions. Thus, the experimental data prove the contribution of these gaseos transmitters to the modification of the blood oxygen transport function.

Acknowledgements: This work was supported by international projects No. M20P-428 – BRFFR and No. 20-515-00019-RFBR.

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S7.P2

THE INFLUENCE OF ANTIMICROBIAL NEUTROPHIL EXTRACT AND PENTOXIFYLLINE ON OVINE NEUTROPHILS ISOLATED DURING THE INSERTION TITANUM IMPLANT IN A SHEEP MODEL

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Titanium (Ti) is the metal commonly used in orthopedic field. Titanium is highly resistant to corrosion. However, Ti ions might slowly diffuse into surrounding tissue where they would be transported into circulation and may interact with blood cells, causing their excessive activation. Autologous neutrophil extract (AMP) was previously considered as factor to decrease of excessive response of leukocytes. Pentoxifylline (PTX) is a competitive non-selective phosphodiesterase inhibitor, which acts anti-inflammatory, enhances microcirculation, blood flow and tissue oxygenation. It also stimulate bone formation and could be considered in management of osseointegration. The aim of this study was to assess of neutrophil *in vitro* response to implantation of biomaterial into the tibia with or without treatment with AMP or PTX. The study was conducted on 8 sheep, females, BCP local breed, 4 months old, from the Bezek Experimental Farm. The procedure consisted of inserting a Ti implant into the proximal tibial physis. Blood sampling necessary to obtain AMP was done 7 days before implantation. For the determination of neutrophil activity, blood was collected at three time points: 7 days before implantation, 1 h and 24 h after implantation. The secretory activity of neutrophils was estimated on the basis of the degranulation and free radicals generation at above time-points, after *in vitro* stimulation with 20 µg/mL AMP or PTX added to final concentrations of 0, 1, and 100 µg/ml of culture of ovine neutrophils. The obtained results show that the addition of AMP and PTX in concentration of 1 µg/ml to the neutrophil suspension decrease of activity of neutrophils. Our study showed that AMP and PTX added at the stated concentrations to the neutrophil suspension isolated during implantation of a Ti implant into the proximal tibial physis reduces the pro-inflammatory response of neutrophils.

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S7.P3

IMPACT OF FUNCTIONALIZED SILVER NANOPARTICLES ON AGGREAGTION OF HUMAN BLOOD PLATELETS

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Nanomedicine is a relatively new field of science and technology. Specifically designed functionalized nanoparticles can impart enhanced cellular internalization ability, non-cytotoxicity, and improved payload binding capacity necessary for effective intracellular delivery. Among metal nanoparticles, silver nanoparticles (AgNPs) are emerging as an attractive tool for many nanomedical applications. They are endowed with anticancer, antibacterial, antifungal and antiviral properties. The field of the nanopharmaceutical development of antithrombotic drugs, has not been explored yet. Based on our previous research, we decided to evaluate the effect of functionalized AgNPs on platelet aggregation and cytotoxicity on human cells. We hypothesized that AgNPs, a known antimicrobial agent, can be used as blood-compatible, ideal material in medical devices or as a drug delivery system. The aim of the research was the synthesis of functionalized AgNPs (glutathione (GSH), polyethylene glycol (PEG), lipoic acid (LA)), evaluation of cytotoxicity and determination of interactions between AgNPs and platelets. A quartz crystal microbalance was used to measure the effect of AgNPs on platelet aggregation. Flow cytometry was used to determine surface platelet receptors. The lactate dehydrogenase assay was used to evaluate the potential cytotoxicity of AgNPs against human platelets, endothelial cells. ELISA tests were used to measure the levels of thromboxane B_2 (TXB_2) and the metalloproteinases released by platelets. All tested functionalized AgNPs inhibited platelet aggregation, increased in total P-selectin and GPIIb/IIIa, TXB_2 formation, release of metalloproteinases at non-toxic concentrations. The results of our research indicate that functionalized AgNPs can potentially be used as an antiplatelet agent in the design of medical materials and equipment.

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S7.P4

NEUTROPHIL-TO-LYMPHOCYTE RATIO AS A FACTOR PREDICTING RADIOTHERAPY INDUCED ORAL MUCOSITIS AND OVERALL SURVIVAL IN HEAD NECK CANCER PATIENTS TREATED WITH RADIOTHERAPY

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Epithelial tumors in the head and neck area (head and neck cancer-HNC) are ones of the most frequently tumors with as many as 650,000 new cases every year. The treatment of HNC patients include surgery, radiotherapy (RT), chemotherapy (CTH) or the combination of these methods. Radical RT, often combined with chemotherapy (C-RT), leads to complications including severe acute radiation reaction in the area of mucosa (oral mucositis - OM). OM occurs in the majority of irradiated patients (80%), which constitutes a serious problem in everyday clinical practice. The objective of this research conducted in HNC patients was the assessment of the relationship between neutrophil-to-lymphocyte ratio (NLR) the incidence of severe RT induced OM as well as overall survival (OS). The study involved 207 patients in advanced stages (III–IV) of HNC. RTOG/EORTC scale was used to assess OM. The pre-treatment NLR was specified as the absolute neutrophil count divided by the absolute lymphocyte count. Starting from 2nd to 7th week of RT we observed significant, positive correlation between NLR values and OM grade. From 2nd to 7th week of RT higher NLR values were related with significant increases (from 2 to over 24-fold) in the risk of occurrence of more severe OM (multivariate analysis confirmed its independent influence). Moreover, multivariate analysis for survival revealed that both higher TNM stage (HR=1.84; p=0.0043) and higher NLR values (HR=1.48; p=0.0395) were independent prognostic factors. We conclude that NLR is a simple and accurate parameter useful in the evaluation of the risk of more severe OM as well as independent prognostic factor of OS in patients subjected to RT due to HNC.

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S7.P5

UTILITY OF THE PLATELET-ENDOTHELIAL CELL ADHESION MOLECULE 1 (PECAM-1) AS A MARKER OF PLATELET ACTIVITY IN THE FLOW CHAMBER MODEL OF THROMBOSIS IN ANIMAL AND HUMAN STUDY

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Platelet endothelial cell adhesion molecule 1 (PECAM-1) is considered as an antithrombotic molecule. In our previous study we introduced the parameter PECAM-1/thrombus ratio which indicates the proportion of PECAM-1 in a laser-induced thrombus in a mouse mesenteric vein. The higher the PECAM-1/thrombus ratio is, the less activated platelets in thrombus are. The present study aimed to assess the utility of PECAM-1/thrombus ratio in a model of thrombus formation on collagen fibers under controlled flow (flow chamber model) which can reflect arterial or venous conditions. This approach extends the possibility of determining the PECAM-1/thrombus ratio to human blood. Flow chamber model enables the observation of thrombotic process *ex vivo* and *in vitro* as well as simultaneous assessment of platelet activity (expressed as the PECAM-1/thrombus ratio) and platelet aggregation (expressed as the thrombus area). In our preliminary study with mice we have shown that the antiplatelet drug acetylsalicylic acid (ASA, 30 mg/kg, i.v.) increased PECAM-1/thrombus ratio by 32.7% (n=7, p <0,001 vs. VEH) and decreased thrombus area by 76.8% (n=7, p <0.001 vs. VEH). We showed for the first time that PECAM-1/thrombus ratio is suitable parameter in the platelet activation assessment in flow chamber model. Experiments with human blood are performed.

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S7.P6

THE ROLE OF LOW-DENSITY GRANULOCYTES CELLS AS A POTENTIAL FACTOR IN THE DEVELOPMENT AND INTENSITY OF INFLAMMATION IN PSORIASIS

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Two populations of cells are observed in human peripheral blood, i.e. polymorphonuclear leukocytes (PMN), with their characteristic granular structure, and peripheral blood mononuclear cells (PBMC). Low-density granulocytes (LDG) are neutrophils which, after separation by a density gradient, remain in the peripheral blood mononuclear fraction of PBMCs. Post-inflammatory LDGs can damage endothelial cells and release a large amount of tumor necrosis factor (TNF) as well as type I and II interferons. They are characteristic of autoimmune diseases, including psoriasis, which occurs in about 2% of the population. The inflammatory process in the course of psoriasis is systemic and manifests itself mainly on the skin, but also affecting internal organs. The diagnosis and monitoring of the disease is based on the clinical picture. The assessment of disorders of other organs requires additional tests. In the study, it was observed that in the PBMC fraction of patients with psoriasis, LDG cells appear in a much greater number, on average about 6 times higher than in healthy people from the control group. In patients suffering from psoriasis, it was on average about 2.77% of the PBMC population, while healthy donors had on average 0.46% of LDG. It is worth noting that in patients suffering from psoriasis, this population was up to approx. 32% of PBMCs, compared to healthy donors, whose maximum was approx. 3.5%. It was also shown that this population is characterized by an increased ability to release the MPO enzyme in psoriasis patients compared to healthy people (28% vs. 0.73%). In sick patients, an increased activity of MPO is observed, on average about 39 times higher than in healthy donors. Presumably, LDGs are an epiphenomenon of ongoing inflammation, and not the primary cause of psoriasis pathogenesis. It is possible that LDG cells present in abundance at inflammatory sites play an active role in the development and maintenance of autoimmune responses. To establish the potential of LDG as a biomarker in inflammatory autoimmune diseases, larger groups should be analyzed to discover potential correlations with disease severity or prognosis.

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S7.P7

CHANGES IN ARTERIAL OXYGEN SATURATION IN HEALTHY PERSONS DURING BACK MASSAGE PROCEDURE

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The aim of this study was to assess changes in arterial oxygen saturation (S_aO_2) in healthy persons during a procedure of back massage. Relevance of this studies is justified by the fact that it is crucial to control the level of S_aO_2 when massaging persons who recovered from COVID-19 in order to assess efficiency of any therapeutic measures, including physiotherapy, for instance massage therapy. The studies included 3 women and 3 men aged 43.5 ± 1.5 years, BMI=24.84 ± 2.03 kg/m^2. All subjects signed a written consent to take part in the study (permission of the Kazimiera Milanowska College of Education and Therapy Bioethical Commission, Resolution No. 006/2018/2019 of 10.05.2019). The intensity of the classical back massage was controlled with thermovision pictures (thermovision camera Flir E6, Estonia) taken before and after the procedure what enabled, through the assessment of temperature distribution in the massaged area, to even out the level of stimulation caused by the mechanical influence of manual massage (the increase in the massaged area temperature was 3.02 ± 0.38°C). The procedure lasted for 20 minutes. The massage was conducted according to the rules of the safe classical massage methodology. Oxygen saturation and heart rate were measured with pulse oximeter (OxyShuttle, USA) before the procedure, with a patient lying down, during the procedure, and within 5 minutes after the procedure was completed. The baseline S_aO_2 was 96.83 ± 0.75%. The highest deviation from the baseline was observed during the massage of left and right side intercostal muscles (7th and 14th minute of the procedure, respectively); S_aO_2 decreased to 94.0 ± 0.63% (7th minute, left side) and 94.16 ± 0.75% (14th minute, right side). Decrease in S_aO_2 to 94.5 ± 0.55% was also noted at the completion of the procedure, after employing the tapotement techniques (hacking and cupping). Five minutes after the massage, the level of S_aO_2 increased to 97.0 ± 0.63%. Changes in S_aO_2 observed during the massage procedure can indicate that during the massage the vesicular ventilation to blood flow velocity ratio changes similarly to low intensity physical activity due to increased temperature of the massaged area.

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