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POSTER PRESENTATIONS

P-1026

A methodological approach using rAAV vectors encoding nanobody-based biologics to evaluate ARTC2.2 and P2X7 *in vivo*

Henri Gondé[1], Mélanie Demeules[1], Romain Hardet[1], Allan Scarpitta[1], Marten Junge[2], Carolina Pinto Espinoza[2], Rémi Varin[1], Friedrich Koch Nolte[2], Olivier Boyer[1], Sahil Adriouch[1]

[1]*Normandie University, UNIROUEN, INSERM, U1234, Pathophysiology, Autoimmunity, Neuromuscular Diseases and Regenerative THERapies, Rouen, France*
[2]*Institute of Immunology, University Medical Center Hamburg-Eppendorf, Hamburg, Germany*

On murine T cells, mono-ADP ribosyltransferase ARTC2.2 catalyzes posttranslational modification, ADP-ribosylation at arginine residues of various surface proteins when nicotinamide adenine dinucleotide (NAD+) is released into the extracellular compartment. Covalent ADP-ribosylation of the P2X7 receptor by ARTC2.2 thereby represents an additional mechanism of activation, complementary to its triggering by extracellular ATP. P2X7 is a multifaceted receptor that may represents a potential target in inflammatory, and neurodegenerative diseases, as well as in cancer. We present herein an experimental approach using intramuscular injection of recombinant AAV vectors (rAAV) encoding nanobody-based biologics targeting ARTC2.2 or P2X7. We demonstrate the ability of these *in vivo* generated biologics to potently and durably block P2X7 or ARTC2.2 activities *in vivo*, or in contrast, to potentiate NAD+- or ATP-induced activation of P2X7. We additionally demonstrate the ability of rAAV-encoded functional heavy chain antibodies (hcAb) to elicit long-term depletion of T cells expressing high levels of ARTC2.2 or P2X7. Our approach of using rAAV to generate functional nanobody-based biologics *in vivo* appears promising to evaluate the role of ARTC2.2 and P2X7 in murine acute as well as chronic disease models.

Keywords: Engineering of antibodies and nanobodies, immune regulation and therapy, immunological techniques

P-1027

Characterization of conducting airway phagocytic cells that internalize SARS-CoV-2 receptor binding domain-coated 100-nanometer particles in conducting airways of mice

Julia Vavilova[1], Andrey Bogorodskiy[2], Elena Bolkhovitina[1], Ivan Okhrimenko[2], Valentin Borshchevskiy[2], Marina Shevchenko[1]

[1]*Department of Immunology, Shemyakin and Ovchinnikov Institute of Bioorganic Chemistry, Russian Academy of Sciences, Moscow, Russia*
[2]*Research Center for Molecular Mechanisms of Aging and Age-Related Diseases, Moscow Institute of Physics and Technology, Dolgoprudny, Russia*

SARS-CoV-2 penetrates the epithelial barrier of the respiratory tract and invading the airways immune cells can either provide the defense or facilitate virus dissemination. In this study, we compared the phagocytic potential of conducting airway immune cells in response to 100-nanometer fluorescent particles in the presence or absence of peptide – receptor-binding domain (RBD) of SARS-CoV-2. Mice received 100 nm carboxylate-modified fluorescent particles dissolved in phosphate buffer (PBS) or 0,1 % solution of RBD of SARS-CoV-2. Control mice received particles in 0,1% solution of bovine serum albumin (BSA) and 0,1 % solution of RBD of SARS-CoV-2 without particles. Particles were applied to mice oropharyngealy in a dose of 10 million particles per mouse. Conducting airway was dissected and subjected to immunohistochemistry as a whole mount. Specimens were analyzed using confocal laser-scanning microscopy. 24 hours after the particle application slight infiltration of $CD11b^+$ phagocytes was detected in conducting airways of mice that received particles both in protein or peptide solution or alone. Infiltrated airways $CD11b^+$ phagocytes did not participate in the uptake of particles dissolved in PBS or BSA. These particles were mostly ingested by $CD11c^+CD169^+$ cells in the luminal side of the airway epithelium. RBD of SARS-CoV-2 solution alone stimulates $CD11b^+$ phagocytes infiltration to the conducting airway. Dissolved in RBD of SARS-CoV-2 solution particles were ingested by both $CD11c^+CD169^+$ cells and $CD11b^+$ phagocytes. Thus, RBD of SARS-CoV-2 stimulates $CD11b^+$ phagocytes infiltration and 100 nm particle uptake in conducting airways.

The work was supported by RFBR, project 20-04-60311.

Keywords: Inflammatory disease, innate host defence, viral infections, visualizing immune responses

P-1028

The mithralog EC-7072 induces chronic lymphocytic leukemia cell death by targeting tonic B-cell receptor signaling

Seila Lorenzo Herrero[1], Christian Sordo Bahamonde[1], Ángel R. Payer[2], Ana P. González Rodríguez[2], Esther González García[3], Alejandro López Soto[4], Segundo Gonzalez[1]

[1]*Department of Functional Biology, University of Oviedo, Oviedo, Spain; Instituto Universitario de Oncología del Principado de Asturias (IUOPA), Oviedo, Spain; Instituto de Investigación Sanitaria del Principado de Asturias (ISPA), Oviedo, Spain*
[2]*Department of Hematology, Hospital Universitario Central de Asturias (HUCA), Oviedo, Spain; Instituto Universitario de Oncología del Principado de Asturias (IUOPA), Oviedo, Spain; Instituto de Investigación Sanitaria del Principado de Asturias (ISPA), Oviedo, Spain*
[3]*Department of Hematology, Hospital de Cabueñes, Gijón, Spain*
[4]*Department of Biochemistry and Molecular Biology, University of Oviedo, Oviedo, Spain; Instituto Universitario de Oncología del Principado de Asturias (IUOPA), Oviedo, Spain; Instituto de Investigación Sanitaria del Principado de Asturias (ISPA), Oviedo, Spain*

In this study, we evaluate the therapeutic potential of the mithralog EC-7072 and its impact on leukemia B-cell homeostasis in chronic lymphocytic leukemia (CLL). Peripheral blood mononuclear cells (PBMCs) isolated from untreated patients with CLL fulfilling the diagnosis criteria for this malignancy and healthy donors were analyzed. RNA-sequencing analyses of CLL cells treated with EC-7072 were performed. The effect of EC-7072 on cell viability, apoptosis or BCR-signaling was analyzed by flow cytometry and western blotting. Herein, we demonstrate that the mithralog EC-7072 displays high *ex vivo* cytotoxic activity against leukemia cells from CLL patients independently from high-risk prognostic markers and *IGHV* mutational status. EC-7072 is significantly less toxic against T cells and NK cells. EC-7072 directly triggered caspase-3-dependent leukemia cell apoptosis, which was not abrogated by microenvironment-derived factors that sustain leukemia cell survival. RNA-sequencing analyses revealed a dramatic EC-7072-driven reprograming of the transcriptome of CLL cells, including a wide downregulation of multiple components and targets of the BCR signaling pathway. EC-7072 exerted similar or higher antileukemic activity than that of several available CLL therapies and displayed additive or synergistic interaction with these drugs in killing CLL cells. Our findings provide evidence that EC-7072 induces leukemia cell death by hampering BCR signaling in CLL cells. Further, the compound enhances the antileukemic activity of approved therapeutic agents, hence opening the question of whether EC-7072 may be a potential novel standalone or combination therapeutic option for patients with CLL and other B-cell malignancies.

Keywords: B lymphocytes, cancer immunology, cell death, RNAseq

P-1029

Preclinical evaluation of a novel rosmarinic acid derivative on the pathogenesis of type 1 diabetes in a mouse model

Ivan Koprivica[1], Natalija Jonić[1], Dimitris Diamantis[2], Christina Papaemmanouil[2], Dragica Gajić[1], Goran Stegnjaić[1], Bojan Jevtić[1], Tamara Saksida[1], Đorđe Miljković[1], Andreas Tzakos[2], Ivana Stojanović[1]

[1]*Institute for Biological Reseaerch "Siniša Stanković" - National Institute of the Republic of Serbia, University of Belgrade, Belgrade, Serbia*
[2]*Section of Organic Chemistry & Biochemistry, Department of Chemistry, University of Ioannina, Ioannina, Greece*

Rosmarinic acid (RA) is a polyphenol compound that naturally occurs in plants of the *Lamiaceae* family. A novel rosmarinic acid derivative (RAd) has been developed and tested in the animal model of type 1 diabetes (T1D) and the animal model of multiple sclerosis, experimental autoimmune encephalomyelitis (EAE). T1D was induced in male C57BL/6 mice using streptozotocin that was applied intraperitoneally for five consecutive days. EAE was induced in Dark Agouti (DA) rats by subcutaneous injection of autologous spinal cord homogenate. For T1D, intraperitoneal administration of RAd (10 mg/kg bw) began from the first streptozotocin injection and continued for 20 days, while for EAE, subcutaneous administration of RAd (28 mg/kg bw) started with the first clinical signs of the disease and continued for 15 days. RAd-treated mice exhibited lower incidence of T1D (monitored up to 45 days from the disease induction), and fluorescent histochemical analysis showed that their pancreatic islets expressed more insulin. Additionally, RAd ameliorated EAE in DA rats. In T1D, RAd treatment significantly down-regulated the proportions of $CD11b^+$ and $CD11c^+$ myeloid cells in the immune cell infiltrates in the pancreas, detected on day 10 after T1D induction. However, the proportions of cells of adaptive immunity ($CD4^+$, $CD8^+$, Th1, Th17) were comparable between the groups. These results suggest that chemically modified RA shows great promise for anti-inflammatory approaches in autoimmune and inflammatory diseases, while our previous research illustrated that unmodified RA exerted no effect on T1D pathogenesis.

Keywords: Animal models, autoimmunity, diabetes, drugs for immune modulation, immune regulation and therapy, multiple sclerosis