Abstract Book

5th European Congress of Immunology

Editors:

ECI2018 Congress President
Prof. Dr. Marieke van Ham
Head Dept. of Immunopathology
Sanquin Research and SILS, Faculty of Science, University of Amsterdam
Plesmanlaan 125, 1066 CX Amsterdam, The Netherlands
m.vanham@sanquin.nl

ECI2018 Scientific Program Committee President
Prof Dr. Jaques Neefjes
Leiden University Medical CenterDepartment of Cell & Chemical Biology
P.O. Box 9600 , 2300 RC Leiden, The Netherlands
j.j.c.neefjes@lumc.nl

ECI2018 Local Organizing Committee President
Janneke N. Samsom, PhD
Erasmus University Medical Center
Laboratory of Pediatric Gastroenterology
P.O. Box 2040, 3000 CA Rotterdam, The Netherlands
j.samsom@erasmusmc.nl

ECI2018 and EFIS President's Office
Christina Helbig, PhD
Plesmanlaan 125, 1066 CX Amsterdam, The Netherlands
helbig@efis.org

Graphic Design: Christina Helbig

Coordination:

Wiener Medizinische Akademie GmbH
Alser Strasse 4, 1090 Vienna, Austria
+43 1 405 13 83 30
eci2018@medacad.org
www.eci2018.org

GUIDELINES TO READ THE ABSTRACT BOOK

The scientific program is divided into 5 main topic tracks:

Track A: Immune development and differentiation
Track B: Tumor immunology and therapy
Track C: Autoimmunity, allergy and transplantation
Track D: Infections and microbial immune regulation
Track E: Immunomics – Technical advances and big data

There are 10 types of sessions generated from invited and submitted abstracts:

Keynote Lectures (KL): oral presentations by invited keynote speakers
Symposia (S): oral presentations by invited speakers
Joint Symposium (JS): oral presentations by invited speakers
Educational Sessions (EDU): oral presentations by invited speakers
Men and Women in Immunology (MWI): oral presentations by invited speakers
EFIS President's Symposium (EP): oral presentations by invited speakers
Bright Sparks Workshop (BS): oral presentations from selected abstracts
Workshop (WS): oral presentations from selected abstracts
Late Breaking Hot Topics (HT): oral presentations from selected abstracts
Guided Poster Session (P): poster presentations from selected abstracts

How to read the presentation numbers – for example: WS.B1.06.04

WS.B1.06.04: **WS** stands for one of the following session types

KL:	Keynote Lecture	EP:	EFIS President's Symposium
S:	Symposium	BS:	Bright Spark
JS:	Joint Symposium	WS:	Workshop
EDU:	Educational Session	HT:	Late Breaking Hot Topic
MWI:	Men and Women in Immunology	P:	Poster Session

WS.B1.06.04: **B** stands for one of the following tracks:

A:	Immune development and differentiation
B:	Tumor immunology and therapy
C:	Autoimmunity, Allergy and Transplantation
D:	Infections and microbial immune regulation
E:	Immunomics - Technical advances and big data

WS.B1.06.04: **1** indicates the subtopic within the respective track

WS.B1.06.04: **06** indicates the chronological order of sessions within the respective subtopic

WS.B1.06.04: **04** indicates the chronological order of presentations within the respective session

Thus, WS.B1.04.04 indicates the fourth talk in workshop 04 of subtopic B1!

ANNOTATIONS

In the following we are publishing the abstracts as submitted by the authors.
Missing session numbers represent sessions with no abstracts associated. Missing presentation numbers represent withdrawn or embargoed abstracts which have not been received as per date of publication.

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P.C2.05.13
OCA-B in the pathogenesis of Type 1 Diabetes

***H. Kim**, A. Shakya, C. German, D. Tantin;*
The University of Utah, Salt Lake City, United States.

Introduction: Type 1 diabetes (T1D) is an autoimmune disease caused by the destruction of insulin-producing beta cells in the pancreas. The mechanisms of T1D pathogenesis remain incompletely understood. Oct1 is a sequence-specific DNA binding transcription factor that in T cells potently regulates target gene expression with a co-factor, OCA-B. Both proteins control CD4+ T cell memory. Importantly, pathogenic CD8+ memory T cell frequency correlates with severity of insulitis in the non-obese-diabetic (NOD) mouse. The strongest OCA-B expression is found in pancreas-infiltrating-islet-reactive CD4+ T cells. Moreover, GWAS studies associate SNPs at multiple Oct1/OCA-B binding sites with T1D.
Materials and Methods: To study OCA-B effects on CD8+ T cell memory, WT or OCA-B KO naïve CD8+ T cells were cultured with a-CD3/28 for 2days. Subsequently, the cells were rested for 8 days without stimulus, and were re-stimulated for a recall response. To investigate impacts of OCA-B in the pathogenesis of T1D, NOD mice were injected with newly developed OCA-B inhibitor peptides or control.
Results: OCA-B loss in CD8+ T cells failed to induce recall responses in vitro. Strikingly, the inhibitor peptides reversed the elevated blood glucose in NOD mice. T cell infiltration and cytokine production in the pancreas was reduced compared to NOD mice treated with control peptide.
Conclusions: OCA-B regulates both CD4+ and CD8+ T cell memory and plays a role in T1D pathogenesis. To elucidate the mechanisms more deeply, OCA-B germline KO and T cell-specific OCA-B deleted NOD mice have been generated.

P.C2.05.14
Ethyl pyruvate treatment enhances regulatory T cell proliferation and function in type 1 diabetes

***I. Koprivica**, M. Vujičić, T. Saksida, D. Gajić, I. Stojanović;*
Institute for Biological Research "Siniša Stanković", Belgrade, Serbia.

Type 1 diabetes (T1D) is an autoimmune disease in which a strong inflammatory response causes insulin-producing pancreatic β-cell death. Ethyl pyruvate (EP), a stable pyruvate derivate, has exerted antioxidant and anti-inflammatory properties in several disease models. To test its therapeutic potential in T1D, EP was administered intraperitoneally to C57BL/6 mice with multiple low-dose streptozotocin (STZ)-induced T1D. EP treatment decreased T1D incidence and reduced the infiltration of cells into the pancreatic islets. *Ex vivo* analysis by flow cytometry showed that the EP treatment didn't change the number of immune cells in the spleen, pancreatic lymph nodes (PLN) or pancreatic mononuclear infiltrates (PMNI), nor the relative percentages of Th1, Th17 and Th2 cells. However, EP treatment increased the levels of regulatory T cells (Treg) in PLN and PMNI. After the EP treatment, all PLN Treg were $GITR^+CD127^-$, and an increase was noted in the percentage of $CD101^+$Treg, indicating a stronger suppressive activity. That was confirmed by an *in vitro* suppression assay, in which Treg from EP treated mice showed a higher capacity to supress effector T cell proliferation.
The number of $CXCR3^+$Treg and the presence of CD11a and CD62L per cell increased, which might imply an increase in Treg migration into the pancreas. However, a rise in the presence of $Ki67^+$Treg suggested that EP treatment also promotes Treg proliferation. These results show that EP treatment reduces T1D incidence in C57BL/6 mice by enhancing Treg proliferation, suppressive capacity, and recruitment into the pancreas. *This research is supported by MESTD, Republic of Serbia (#173013)*

P.C2.05.15
The role of IL-23TH17 pathway in Inflammatory bowel disease

***Y. Lakhoua Gorgi**[1], M. Majdoubi[1], J. Abdellatif[1], M. Jellouli[1], T. Dhaouadi[1], L. Mouelhi[2], T. Ben Abdallah[1], I. Sfar[1];*
[1]Research Laboratory in Immunology of Renal Transplantation and Immunopathology (LR03SP01), Tunis, Tunisia, [2]Department of Gastroenterology Charles Nicolle hospital, Tunis, Tunisia.

AIMS: Investigate a possible association between the functional polymorphisms: IL-17RC rs708567, IL-17F rs2397084 and IL-23R rs11209026 and the susceptibility to IBD and define the impact of these genetic variants on the IBD clinical forms **METHODS**: A cross-sectional case-control study involving 178 patients with IBD (108 CD and 70 UC) and 100 healthy control subjects was made. The molecular analysis was performed by PCR-RFLP. An ELISA tests were used to process to the quantitative determination of serum IL-17F and IL-23 cytokines.
RESULATS: Quantitatively, the mean level of IL-17F was significantly higher in patients than in controls (p = 0.003) and in CD patients with stricturing complications compared to other clinical forms (p = 0.006). The serum IL-23 levels were similar in patients and controls. The genotype G/G of IL-17RC polymorphism and the A/G genotype of IL-17-F SNP's seem to be associated to a higher occurrence of IBD and to the positivity of the ANCA antibodies in RCH patients in Tunisians (p = 0.049 and p = 0.008). For the R381Q IL-23R polymorphism, the frequency of the A allele was significantly higher in controls than in IBD patients (p <**0,0001)** suggesting a protective role of this SNP. **CONCLUSION:** The results of our study highlight the key role of IL-23Th17 pathway in the pathophysiological mechanisms of IBD. The prognostic interest of these markers deserved to be confirmed by further prospective studies on a larger cohort and by a functional analysis of IL-17F and IL-23mRNA expression

P.C2.05.16
Sulforaphane inhibits inflammatory responses of primary human T-cells by increasing ROS and depleting glutathione

***J. Liang**[1], B. Jahraus[1], E. Balta[1], J. Ziegler[1], K. Hübner[1], N. Blank[2], B. Niesler[3], G. Wabnitz[1], Y. Samstag[1];*
[1]Institute of Immunology, Heidelberg, Germany, [2]Department of Rheumatology, Heidelberg, Germany, [3]Department of Human Molecular Genetics, Heidelberg, Germany.

Sulforaphane (SFN), a compound in plants of the brassicaceae family, was reported to suppress cancer cell growth. Information about the relevance of SFN for human T-cells is limited. This is surprising as T-cells play a critical role in tumor control. We, therefore, investigated the effects of SFN on human untransformed peripheral blood T-cells. While SFN did not show cytotoxic effects and did not interfere with early T-cell activation, i.e. the formation of a mature immune synapse, it affected later activation events as upregulation of CD69 and CD25. The inhibitory effects of SFN could be rescued by thiol-containing antioxidants. In line with that finding, SFN led to an increase of intracellular reactive oxygen species (ROS) and a marked decrease of glutathione. Consistently, increased global cysteine sulfenylation was detected. Importantly, a major target for SFN-mediated protein oxidation was STAT-3, a transcription factor involved in the regulation of TH17-related genes. Moreover, costimulation-induced STAT-3 phosphorylation was significantly inhibited by SFN. In an unbiased gene expression signature analysis, we indeed found that TH17-related genes were predominantly inhibited by SFN. Since IL-17 and ROS regulation may be attractive targets for treating rheumatoid arthritis (RA), we tested the effect of SFN on whole blood from RA patients and found an increase in intracellular ROS levels in T-cells. Moreover, costimulation-induced expression of IL-17 was markedly decreased in SFN-treated T-cells from RA patients. Taken together, our study shows that SFN may act as a promising substance for therapeutic immune suppression via regulating the redox balance in human T-cells.

P.C2.05.17
Novel biomarkers of disease combined with ligand-directed targeted therapy for the control of autoimmune arthritis

*S. H. Venkatesha[1,2], S. Dudics[1,2], R. R. Meka[1,2], **K. D. Moudgil**[1,2];*
[1]University of Maryland School of Medicine, Baltimore, Baltimore, United States, [2]Baltimore Veterans Affairs Medical Center, Baltimore, United States.

Introduction: Rheumatoid arthritis (RA) affects millions of people worldwide. A key event here is the breakdown of self-tolerance leading to anti-self reactivity and tissue dysfunction/damage in the synovial joints. Current biomarkers for the diagnosis and prognosis of RA have inherent limitations. Similarly, the presently used drugs against RA are potent but their prolonged use is associated with severe adverse effects. Thus, there is a need for new biomarkers of disease and novel approaches to improve the therapeutic index of anti-arthritic drugs.
Materials and Methods: We addressed both these issues using the adjuvant-induced arthritis (AA) model of RA. We examined the micro-RNA (miRNA) expression profiles of the peripheral lymphoid cells of untreated/ treated arthritic and control rats, using miRNA-microarray. The data was subjected to statistical and bioinformatics analyses.
Results: We identified 8 specific miRNAs that were upregulated upon arthritis development. Collectively, these miRNAs targeted T cell response, angiogenesis, and bone remodeling pathways, but
individual miRNA specifically affecting Th17/Treg differentiation was also identified. Six of above 8 miRNAs were inhibited following arthritis treatment. For RA therapy, we employed a novel peptide
ligand that preferentially homes to inflamed joints after systemic administration, to direct drug-entrapping liposomes into the joints. Arthritic rats treated with these liposomes showed markedly
reduced severity of arthritis and decreased systemic toxicity compared to rats treated with free drug.
Conclusion: Our study has unraveled novel biomarkers of disease development and therapeutic response as well as an improved targeted therapy for autoimmune arthritis.
(Acknowledgement: grants from NIH and Veterans Affairs)