ISSN 0014-2980 · EJIMAF51 (Supplement S1) · Vol.51 · Supplement S1 · August 2021

50th anniversary

European Journal of Immunology

Supplement S1

www.eji-journal.eu

WILEY

DOI: 10.1002/eji.202170200

European Journal of Immunology

Abstracts of ECI 2021

6th European Congress of Immunology

1-4 September 2021

Virtual meeting

European Journal of Immunology Volume 51, Suppl. 1, August 2021

This abstract book can be searched using the PDF search function to look, for example, for the Abstract number if applicable or author name.

To cite an Abstract, please use the following format:
Abstract title. Authors. Conference: 6th European Congress of Immunology. Abstracts. Location Virtual.
Date September 1-4, 2021. Eur. J. Immunol. 2021. 51, S1, page number(s). Meeting Abstract number [the Abstract number if applicable can be found above the title]

This Abstract book was corrected after online publication on 25 August 2021 by including two Abstracts that were missing from the initial supplied copy

DOI: 10.1002/eji.202170200

ABSTRACT REVIEWERS

The organizers of the 6th European Congress of Immunology would like to extend their special thanks to all abstract reviewers for their contribution and time they dedicated to the success of the congress:

Adriana Narcisa Munteanu	Romania
Ahmet Eken	Turkey
Akif Turna	Turkey
Aleksandra Popov Aleksandrov	Serbia
Alexandra Siorenta	Greece
Alexandra Tsirogianni	Greece
Alisa Gruden-Movsesijan	Serbia
Amiram Ariel	Israel
Andreas Radbruch	Germany
Anita Milicic	United Kingdom
Anna Sediva	Czech Republic
Anne Caignard	France
Arpad Lanyi	Hungary
Arzu L. Aral	Turkey
Aslı Gelincik Akkor	Turkey
Aude Magerus	France
Ayça Sayı Yazgan	Turkey
Ayça Kıykım	Turkey
Ayşegül Atak Yücel	Turkey
Batu Erman	Turkey
Biljana Božić Nedeljković	Serbia
Bojan Polić	Croatia
Carsten Watzl	Germany
Ceren Çıracı	Turkey
Cezmi Akdiş	Switzerland
Danka Grcevic	Croatia
Delphine Sauce	France
Dicle Güç	Turkey
Diana Dudziak	Germany
Diğdem Yöyen Ermiş	Turkey
Djordje Miljkovic	Serbia
Dominik Filipp	Czech Republic
Duygu Sağ	Turkey
Ed Lavelle	Ireland
Edit Buzas	Hungary
Elif Karakoç Aydıner	Turkey
Erika Jensen-Jarolim	Austria
Eva Martinez Caceres	Spain
Eva Untersmayr	Austria
Gerhard Wingender	Turkey
Goran Marjanović	Serbia
Güher Saruhan Direskeneli	Turkey
Günnur Deniz	Turkey
Güneş Esendağlı	Turkey
Haluk Barbaros Oral	Turkey
Haner Direskeneli	Turkey
Hanna Jarva	Finland
Hannes Stockinger	Austria
İhsan Gürsel	Turkey
Iva Hafner Bratkovic	Slovenia
Ivan Pilipović	Serbia
Ivana Stojanovic	Serbia
Ivanka Tsacheva	Bulgaria
Jacques Nunes	France
James Brewer	United Kingdom
Jelena Djokic	Serbia
Jordi Cano Orchando	Spain
Jüergen Wittmann	Germany
Katerina Tarassi	Greece
Kingston Mills	Ireland
Krassimira Todorova	Bulgaria
Krisztina Buzas	Hungary
Lazar Popovıć	Serbia
Luis Graça	Portugal
Luke Foster	United Kingdom
Marcela Vlkova	Czech Republic
Marco A. Cassatella	Italy
Marcos Lopez-Hoyos	Spain
Marieke Van Ham	The Netherlands
Marinela Bostan	Romania
Marta Szell	Hungary
Martin Schwarzer	Czech Republic
Martina Colicchia	United Kingdom
Maxim Nosenko	Russia
Mayda Gürsel	Turkey
Michael Lohoff	Germany
Mira Barda-Saad	Israel
Micha Berger	Israel
Milada Sirova	Czech Republic
Mirjam Heemskerk	The Netherlands
Mirjana Nacka-Aleksić	Serbia
Muhlis Cem Ar	Turkey
Nada Pejnovic	Serbia
Nataša Ilić	Serbia
Nemanja Jovicic	Serbia
Noushine Mossadegh-Keller	France
Peter Heeringa	The Netherlands
Sergej Tomić	Serbia
Serkan Belkaya	Turkey
Silke Appel	Norway
Slavko Mojsilovic	Serbia
Soren Hayrabedyan	Bulgaria
Srdja Janković	Serbia
Sylvia Knapp	Austria
Şefik Alkan	Turkey
Tal Shay	Israel
Tamara Saksida	Serbia
Tanja Džopalić	Serbia
Tolga Sütlü	Turkey
Trevor Owens	Denmark
Uğur Sezerman	Turkey
Wilfried Ellmeier	Austria
Yıldız Camcıoğlu	Turkey
Zimmer Jacques	Luxembourg
Zoltán Prohászka	Hungary
Zsolt Komlosi	Hungary

DOI: 10.1002/eji.202170200

POSTER PRESENTATIONS

P-1002

The possible cytotoxic function of natural killer cells of patients infected with SARS-CoV-2

Nilgun Akdeniz[1], Fatma Betul Oktelik[1], Murat Kose[2], Ilhan Tahralı[1], Yusuf Metin Gelmez[1], Umut Can Kucuksezer[1], Esin Aktas Cetın[1], Gunnur Denız[1]

[1]*Aziz Sancar Institute of Experimental Medicine, Department of Immunology, Istanbul University Istanbul, Turkey*
[2]*Istanbul Faculty of Medicine, Department of Internal Medicine, Division of Infectious Diseases, Istanbul University Istanbul, Turkey*

Natural Killer (NK) cells are critical components of the innate immune response against virus-infected cells. In this study, the possible cytotoxic role of NK cells in SARS-CoV-2 infection causing the ongoing COVID-19 pandemic was investigated. The patients diagnosed with COVID-19 (n=31; mild:11, moderate:11, severe:9) and healthy subjects (n=8) were enrolled in this study. CD56dimCD16+ and CD3-CD56brightCD16+/- NK cell subsets and their CD38 expression as well as perforin and Granzyme B levels were investigated by flow cytometry. Higher percentage of total NK cells and CD3-CD56dimCD16+ NK cell subset were detected in mild patients than those in other cases and healthy individuals. CD3-CD56dimCD16+CD38+ and CD3-CD56brightCD16+/-CD38+ NK cells were significantly increased in mild and moderate COVID-19 patients in comparison with healthy subjects. All forms of COVID-19 patients had significantly decreased in perforin expression in comparison with healthy subjects. In mild COVID-19 patients, Granzyme B expression of NK cells was significantly increased whereas CD107a expression was significantly decreased compared to healthy subjects. According to our findings, a general decrease in perforin expression of NK cells was observed in all patient groups while CD38 and Granzyme B expression levels were increased especially in mild COVID-19 patients. These findings confirm that CD38 is associated with NK cytotoxicity and further indicate that NK cytotoxic activity varies in the early and late phases of the disease.

Keywords: Innate immunity, NK cells, viral infections

P-1022

Transgenic overexpression of galectin-3 in pancreatic β cells attenuates type 1 diabetes in mice: synergistic antidiabetic effect with exogenous IL-33

Nemanja Jovicic[1], Ivica Petrovic[2], Nada Pejnovic[3], Biljana Ljujic[4], Marina Miletic Kovacevic[1], Sladjana M. Pavlovic[5], Ilija Jeftic[2], Aleksandar Djukic[2], Ivan M. Srejovic[6], Dragica Selakovic[6], Vladimir L. Jakovljevic[7], Miodrag L. Lukic[5]

[1]*Department of Histology and embryology, Faculty of Medical Sciences, University of Kragujevac, Serbia*
[2]*Department of Pathophysiology, Faculty of Medical Sciences, University of Kragujevac, Serbia*
[3]*Department of Immunology, Siniša Stanković Institute for Biological Research, University of Belgrade, Serbia*
[4]*Department of Genetics, Faculty of Medical Sciences, University of Kragujevac, Serbia*
[5]*Center for Molecular Medicine and Stem Cell Research, Faculty of Medical Sciences, University of Kragujevac, Serbia*
[6]*Department of Physiology, Faculty of Medical Sciences, University of Kragujevac, Serbia*
[7]*Department of Physiology, Faculty of Medical Sciences, University of Kragujevac, Serbia, Department of Human Pathology, I.M. Sechenov First Moscow State Medical University, Russia*

Galectin 3 (gal 3) has diverse roles in inflammatory and autoimmune diseases. There is evidence that galectin 3 plays a role in both, type 1 and type 2 diabetes. While the role of Gal-3 expression in immune cells in experimental type 1 diabetes has been already studied, the importance of the overexpression of Gal-3 in the target β cells is not defined. We used 10-12 weeks old C57/BL6 male mice (WT) and C57/BL6 mice with transgenically enhanced Gal-3 expression in pancreatic β cells (TG). Both groups, received STZ for 5 consecutive days at a dose of 40 mg/kg ip. Mice received exogenous mouse IL-33 (0.4 μg/injection) i.p., 12th, 14 th, 16 th, and 18 th day after the disease induction. Control animals were treated with intraperitoneally PBS + citrate buffer or IL-33 + citrate buffer. The overexpression of Gal-3 protected β cells from apoptosis and attenuated MLD-STZ induced hyperglycemia, glycosuria and ketonuria. The cellular analysis of pancreata and draining lymph nodes showed that Gal- 3 overexpression significantly decreased the number of proinflammatory cells without affecting T regulatory cells. The application of exogenous IL-33, attenuates the development of disease, by increasing the presence of regulatory FoxP3+ ST2+ cells, and competely abrogate diabetogenesis. We demonstrated the potential synergistic effect of exogenous IL-33 and TG overexpression of Gal-3 in β cells Not only enhanced expresion of Gal-3 in β cells reduced T cell mediated autoimmune inflammatory disease, but also exogenous IL-33 application had powerful terapeutic effect in TG mice.

Keywords: Animal models, autoimmunity, cytokines and mediators, diabetes, drugs for immune modulation

P-1110

Immunoregulatory properties of acridines derived from anti-malaria drug quinacrine

Qian Wei[1], Johannes Landskron[2], Jo Klaveness[3], Rafi Ahmad[4], Kjetil Taskén[1]

[1]*Department of Cancer Immunology, Institute of Cancer Research, Oslo University Hospital, 0424 Oslo, Norway;K.G. Jebsen Centre for Cancer Immunotherapy, Institute of Clinical Medicine, University of Oslo, 0317 Oslo, Norway*
[2]*Centre for Molecular Medicine Norway, Nordic EMBL Partnership, University of Oslo, 0318 Oslo, Norway*
[3]*School of Pharmacy, University of Oslo, 0317 Oslo, Norway*
[4]*Dept. of Biotechnology, Inland Norway University of Applied Sciences, 2318 Hamar, Norway*

FoxP3$^+$ regulatory T cells (Tregs) are a critical subset of CD4$^+$ T cells responsible for regulating immune homeostasis by suppressing excessive anti self-antigen immunity. The suppressive function of FoxP3$^+$ Treg cells also facilitates tumor cell proliferation due to inhibition of anti-tumor immunity. The key lineage-defining transcription factor FoxP3 in Tregs turns out to be a potential candidate target for anti-tumor treatment. Here, we identified the anti-malaria drug, quinacrine, as an inhibitor of FoxP3 expression in a cell-based high-throughput screening of a drug repurposing library using CD3$^+$ T cells from human healthy donors. Based on the structure of quinacrine, we established a small library containing quinacrine-like acridines by *in silico* prediction and functional verification. A subset of acridine analogues were found to have even more potent inhibitory effect on Treg suppression of effector T cells, by down-regulation of FoxP3 and critical Treg markers. This rescued proliferation and cytokine production of effector T cells. TCR signaling pathways analyzed by phospho-flow revealed that, these acridines could suppress FoxP3$^+$ Tregs more efficiently than other effector T cells. By SPR analysis, we found direct interactions between acridines and FoxP3, which could then interfere with FoxP3-DNA binding activity. The effect appeared selective for FoxP3 as other Forkhead proteins were not affected by these acridines, neither *in vitro* nor *in vivo*. Mice experiments further revealed anti-tumor properties of the several quinacrine-like acridines. We conclude that selected acridines could provide valuable toolsfor assessing Treg functions such as their role in tumor immune evasion.

Keywords: Cancer immunology, drugs for immune modulation, immune regulation and therapy, immunotherapy, regulatory cells