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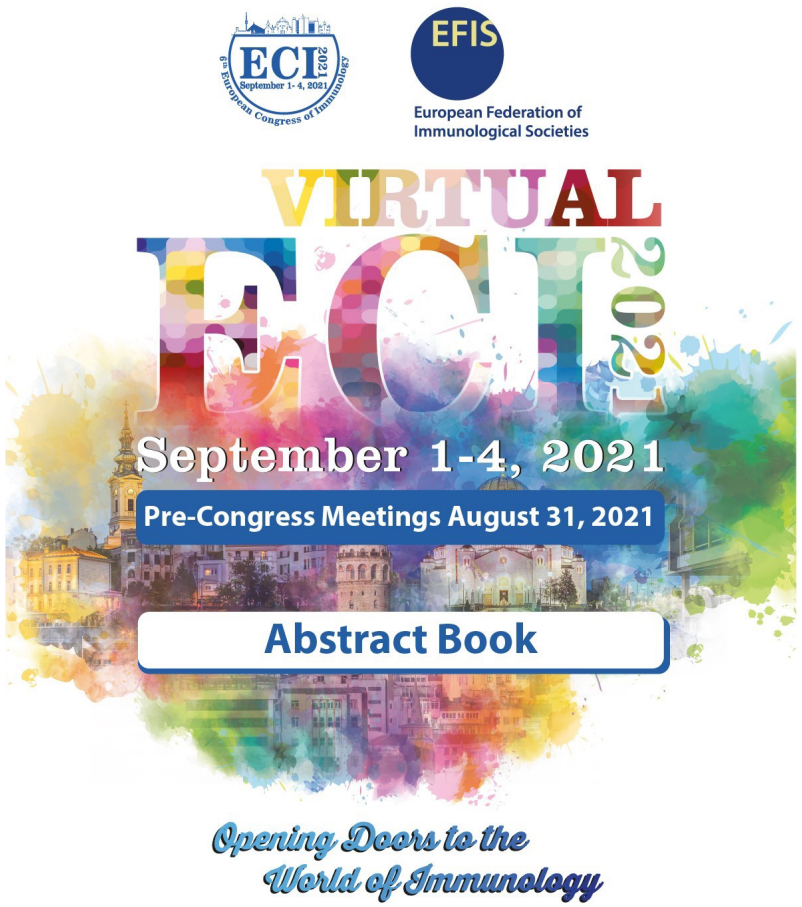


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# Abstracts of ECI 2021

# 6th European Congress of Immunology

1-4 September 2021

Virtual meeting

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# POSTER PRESENTATIONS

## P-0808

### Germline STAT3-activating mutations from autoimmunity and lymphoid malignancy perturb mouse and human T lymphocytes

**Etienne Masle Farquhar,** Timothy Peters, Ghamdan Al Eryani, Kathryn Payne, Geetha Rao, Mandeep Singh, Katherine Jackson, Robert Brink, Stuart Tangye, Joanne Reed, Christopher Goodnow

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Signal transducer and activator of transcription 3 (STAT3) is a latent transcription factor with pleiotropic roles in hematopoietic and non-hematopoietic cells, that regulates gene expression downstream of cell surface cytokine and hormone receptors. Heterozygous germline loss-of-function *STAT3* mutations lead to the primary immunodeficiency hyper-IgE syndrome (HIES) while somatic gain-of-function *STAT3* mutations recur in human solid organ malignancies and non-Hodgkin lymphoma. Recently, germline heterozygous gain- of-function mutations in *STAT3* were shown to result in early-onset and multi-organ autoimmunity with aspects of immunodeficiency. Affected individuals share characteristics with autoimmune lymphoproliferative syndrome (ALPS) and immunodysregulation polyendocrinopathy enteropathy X-linked (IPEX) syndrome, including reduced T regulatory cell numbers and suppressive activity. They present with variable early-onset autoimmune symptoms including type 1 diabetes, juvenile-onset rheumatoid arthritis, gut enteropathies and autoimmune cytopenias. Whilst many effects of STAT3 loss-of-function on immune cells have been described, the mechanisms behind autoimmunity and immunodeficiency in patients with STAT3-activating mutations remain unclear. Here, we present a detailed characterisation of T cell development and maturation in young and old mice on two different backgrounds with Crispr-engineered germline activating mutations in two domains of STAT3. We use mixed chimeras, flow cytometric analysis, T cell receptor deep-sequencing and high-throughput single-cell transcriptomics to reveal cell-extrinsic and -autonomous roles of STAT3 activation in T cells in autoimmunity versus immune malignancy. To our knowledge, this is the first report of mice with *Stat3* germline activating mutations identical to those in autoimmunity or malignancy. We validate our key findings in humans with gain-of-function germline *STAT3* mutations and childhood-onset autoimmune disease.

**Keywords:** Ageing, animal models, autoimmunity, cytokines and mediators, immunodeficiency

## P-0809

### Report on lower anti-pneumococcus IgG1 levels in men, especially over the age of fifty

Sanja Knežević[1], Dejana Kosanović[1], Irena Živković[1], Vesna Ilić[2], Luka Dragačević[1], Ljiljana Hajduković[3], **Rajna Minić[1]**

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*Streptococcus pneumoniae*, the pneumococcus is an important human pathogen. It possesses a capsule which has been thoroughly studied, since it is an important factor in virulence. In this work *S. pneumoniae* ATCC 6301, belonging to Serogroup 1, was used as a model organism to determine the levels of different IgG isotypes specific to this bacterium in different sex and age groups. For this purpose an in-house ELISA was used. Although IgG2 is a major antibacterial antibody subclass, acting against polysaccharide antigens, the level of pneumococcus-specific IgG2 did not differ from the level of IgG2 specific to several tested lactic acid bacteria (LAB). On the other hand pneumococcus specific IgG1 level was significantly higher to the level of LAB specific IgG1. This is most likely a consequence of previous encounters with pathogenic pneumococcal microorganism and a distinguishing characteristic of this encounter. By analyzing different sex and age groups we found significantly lower anti-pneumococcus IgG1 levels in men, implying lower protection against invasion with pneumococcus, which possibly translates to other encapsulated bacteria. The Covid-19 pandemic has identified males as being at increased risk of hospital admissionand death and lower levels of anti-pneumococcus antibodies might be a contributing factor. Hence, we advocate the immunization with conjugate pneumococcal vaccines in male individuals, especially over the age of 50.

**Keywords:** Adaptive immunity, ageing, antibody

## P-0810

### A case of primary hemophagocytic syndrome seen in adult age

**Kemal Fidan[1],** Muzaffer Keklik[1], Neslihan Mandacı Şanlı[1], Gülşah Akyol[1], Mustafa Baydar[1], Serhat Çelik[1], Zeynep Tuğba Güven[1], Hatice Gözde Demir[2], Ali Ünal[1]

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Hemophagocytic lymphohistiocytosis (HLH) is an immune disorder with high mortality due to uncontrolled activation of T lymphocytes and macrophages and excessive proliferation of inflammatory cytokines. A 37-year-old female patient diagnosed with pimer HLH is presented here. A 37-year-old female patient, was hospitalized with complaints of fever, weakness, and joint pain. Laboratory tests; Hemoglobine: 6.8 gr/dl, Wbc: 1.06x103/uL, plt: 8x103/uL neutrophil: 0.73x103/uL, lymphocyte: 0.18x103/uL, fibrinogen: 160.18 mg/dl, ALT: 118 u/L, LDH: 302 u/L, total bilirubin: 1.72 mg /dl,albumine: 2.76 g/dl, BUN: 15 mg/dl, creatinine: 0,73 mg/dl, CRP: 32.81 mg/L, triglyceride: 405 mg/dl, ferritin: 2820 mg/dl. On abdominal USG, the liver was 167mm, spleen 200mm. The bone marrow pathology result was consistent with hemophagocytosis. The patient's NK cytotoxicity was low.As a result of the genetic whole exon sequencing performed upon the presence of a family history; PRF1 / NM-001083116 / EXZON 3, C.560C> G p (Pro187Arg) came as homozygous. HLH 2004 protocol was started with the diagnosis of familial hemophagocytic syndrome. Allogeneic bone marrow transplant was planned for the patient. Pulse steroid was initiated and plasma exchange was performed due to diffuse alveolar hemorrhage in thoracic tomography performed upon the development of dyspnea, tachypnea and tachycardia at the 7th week of the treatment. The patient died at the 8th week of the treatment. In adult patients, primary hemophagocytic syndrome should be considered in differential diagnosis, treatment should be initiated after diagnosis and bone marrow transplantation should be performed as soon as possible.

**Keywords:** Autoimmunity, cytokines and mediators, granulocytes, immunodeficiency, lymphoid organs, macrophage

## P-0812

### What about the cytokines for the etiology of idiopathic granulomatous mastitis?

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Few things are known about the etiology of Idiopathic Granulomatosis Mastitis. It was associated with autoimmunity, but no significant evidence was determined. In this study, we aimed to investigate the roles of cytokines in the etiopathogenesis of Idiopathic Granulomatosis Mastitis. Idiopathic Granulomatous Mastitis patients in active or remission who admitted to the breast diseases outpatient clinic and healthy volunteers were included prospectively in the present study. The IL-1β, IFN-α2, IFN-γ, TNF-α, MCP-1, IL-6, IL-8, IL-10, IL-12p (p70), IL-17A, IL-18, IL-23 and IL-33 values were measured with Flow Cytometry. The blood samples were taken before the treatment in active Idiopathic Granulomatosis Mastitis group. The ages, physical examination findings, menopausal conditions, smoking conditions, and treatment methods were also evaluated. A total of 32 patients, including 19 active and 13 remission patients, and 18 controls, were included in the present study, which made a total of 50 people. The mean age was 37.18±7.15. The IL-1β, TNF-α, IL-10 and IL-18 values were lower in patients with Idiopathic Granulomatosis Mastitis than the control group. Granulomatosis Mastitis patients smoked more than control. When the active patients, remission patients, and control group were evaluated together, no significant differences were detected. In our study, the low rate of cytokines which are important for autoimmune and granulomatous reactions, especially cytokines associated with Th1 and Th17, in patients with idiopathic granulomatous mastitis suggests that these cytokines may not play a role in the etiopathogenesis, and that different mechanisms may be effective.

**Keywords:** Adaptive immunity, autoimmunity, chronic inflammation and fibrosis, cytokines and mediators