Medical Immunology Campus Erlangen

An Interdisciplinary Center
of the Friedrich-Alexander-Universität
Erlangen-Nürnberg

Newsletter Winter 2023
CONTENT

Friedrich-Alexander-Universität
Erlangen-Nürnberg

EDITORIAL

Dear colleagues and friends,

On November 22, Prof. Dr. med. Martin Röllinghoff, emeritus professor of the Medical Faculty and former director of the Institute of Clinical Microbiology, Immunology and Hygiene from 1983 until 2007, died after a serious illness. Martin Röllinghoff was the founding father and driving force of infectious disease immunology in Erlangen and thereby strongly contributed to making immunology a central field of research at FAU. The recruitment and support of young, talented investigators was an important concern for him. Many of his scholars became professors, heads of departments or directors of institutes. Even after his retirement, he followed the development of the research landscape in Erlangen with great interest. We will deeply miss his advice, his friendship as well as regular attention of our guest seminars. A detailed obituary honoring his life's work will follow.

A few days ago, the *Medical Immunology Campus Erlangen* also received a number of good news. First, Georg Schett, head of the Medical Clinic 3 for Rheumatology and Clinical Immunology, is one of this year's recipients of the Gottfried Wilhelm Leibniz-Award of the German Research Foundation, the most prestigious research award in Germany. Second, Claudia Günther and Beate Winner were successful with their clinical research group application "Immune checkpoints of gut-brain communication in inflammatory and neurodegenerative diseases", which will be funded by the DFG from 2023 onwards for four years. Third, the second funding period of the Transregio 374 "Tubulus system and interstitium of the kidney: (patho-)physiology and crosstalk", which contains a number of immunological projects and represents a joint consortium of the University of Regensburg and the FAU with Kerstin Amann and Mario Schiffer as vice-spokespersons, has also been approved. My warmest congratulations to all of them!

As presented in the members' assembly on December 1, the *Medical Immunology Campus Erlangen*, which has been an "Interdisciplinary Center" of FAU since 2009, is supposed to be re-structured and transformed into a "Profile Center" of FAU. The necessary paperwork has been submitted to the university leadership, including the required new statues as well as an agreement on future objectives. We await a final decision in January.

At the end of the year 2022, I would like to inform you that after long-lasting discussions the board of MICE has decided to shift our weekly guest seminar series – whenever possible – from 5.15 p.m. to a more family-friendly time (i.e. 12.30 p.m.). We will closely monitor whether the number of attending PIs will increase. Lately, neither seminars in presence nor via ZOOM were attended by more than 5 to 10 PIs, even when well-known international speakers gave a talk.

Although the corona pandemic is not yet over and several other viruses are currently causing a winter wave of respiratory infections, I wish you and your families a Merry Christmas, some relaxing days of vacation and all the best for 2023.

Christian Bogdan

Prof. Christian Bogdan
Chairman of The Medical Immunology Campus Erlangen

SCIENTIFIC HIGHLIGHTS

Protective mucosal immunity against SARS-CoV-2 after heterologous systemic prime-mucosal boost immunization

Mucosal booster immunizations provide local immunity at viral entry site

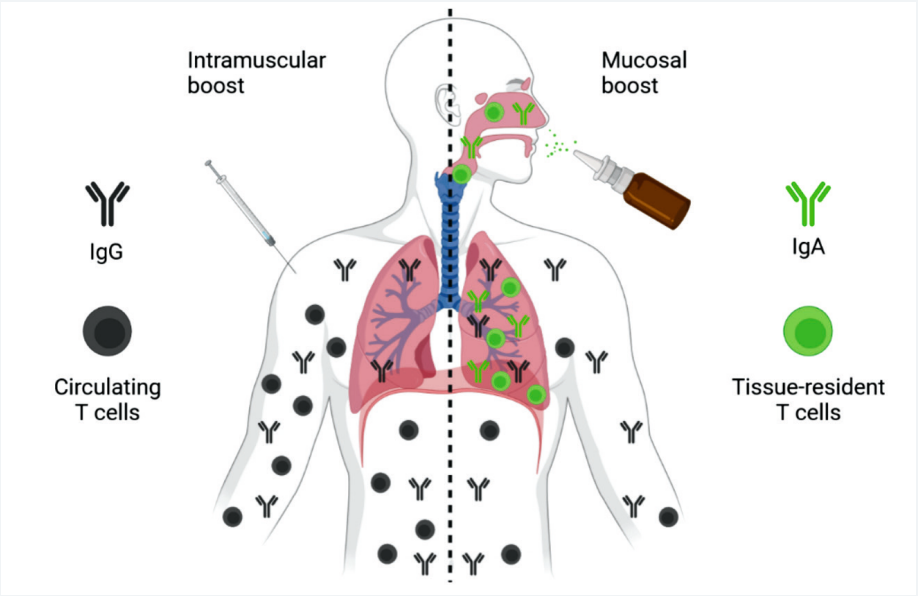
DENNIS LAPUENTE, MATTHIAS TENBUSCH
INSTITUTE OF CLINICAL AND MOLECULAR VIROLOGY, UNIVERSITÄTSKLINIKUM ERLANGEN, FAU ERLANGEN-NÜRNBERG

Several SARS-CoV-2 vaccines are currently in use and have demonstrated efficacy. In the light of waning immune responses and the emergence of novel variants, effective boost modalities are needed to maintain or even enhance immunity. While the currently approved vaccines induce systemic immune responses, they do not evoke a significant mucosal immunity in form of secretory immunoglobulin A or tissue-resident memory T cells (T_{RM}) in the respiratory tract. Inducing this front-line immunity might further reduce breakthrough infections and virus transmission.

In our recent study, we report that intranasal vaccinations with adenovirus 5 and 19a vectored vaccines following a systemic mRNA priming result in strong systemic and mucosal immunity in mice. While conventional prime-boost vaccination schemes with two intramuscular mRNA doses established high levels of circulating T cell memory and neutralizing antibodies in serum, a single intramuscular mRNA prime followed by a mucosal boost additionally induced strong spike protein-specific IgA and $CD103^{+}CD69^{+}$ $CD8^{+}$ T_{RM} responses in the respiratory tract. Mucosal neutralization of virus variants of concern (Alpha, Beta, Gamma, Delta) was also enhanced by an intranasal boost. Finally, we demonstrated that the heterologous mRNA prime/intranasal Ad5 boost strategy is not inferior to the common gold standard of two intramuscular mRNA immunizations in protecting mice from infection. Ongoing studies investigate the potential benefits of a mucosal booster vaccination against infections with SARS-CoV-2 strains that are controlled less efficiently by vaccine-induced systemic immunity such as the current Omicron variants.

In conclusion, mucosal booster immunizations with adenoviral vector vaccines represent a promising way to establish mucosal immunity at the viral entry site, while increasing systemic responses as well. Clinical studies are warranted to evaluate the potential of these mucosal vaccinations in humans.

Lapuente D., Fuchs J., Willar J, Antao A.V., Eberlein V, Uhlig N., Issmail L., Schmidt A., Oltmanns F., Peter AS, Mueller-Schmucker S., Irrgang P., Frædrich K., Cara A., Hoffmann M., Pöhlmann S., Ensser A., Pertl C., Willert T., Thirion C., Grunwald T., Überla K., Tenbusch M.
Nat Commun. 2021 Nov:12(1):6871.



Two consecutive intramuscular mRNA immunizations result in pronounced systemic T cell and IgG responses. In contrast, a mucosal boost with adenoviral vectors after an initial mRNA priming provokes a balanced immunity consisting of systemic responses as well as mucosal IgA and tissue-resident memory T cells in the respiratory tract.

SCIENTIFIC HIGHLIGHTS

Innate PD-L1 limits T cell-mediated adipose tissue inflammation

Dendritic cell expression of PD-L1 prevents pro-inflammatory immune responses during obesity

CHRISTIAN SCHWARTZ, VIVIANE SCHMIDT
INSTITUTE OF CLINICAL MICROBIOLOGY, IMMUNOLOGY AND HYGIENE, UNIVERSITÄTSKLINIKUM ERLANGEN, FAU ERLANGEN-NÜRNBERG

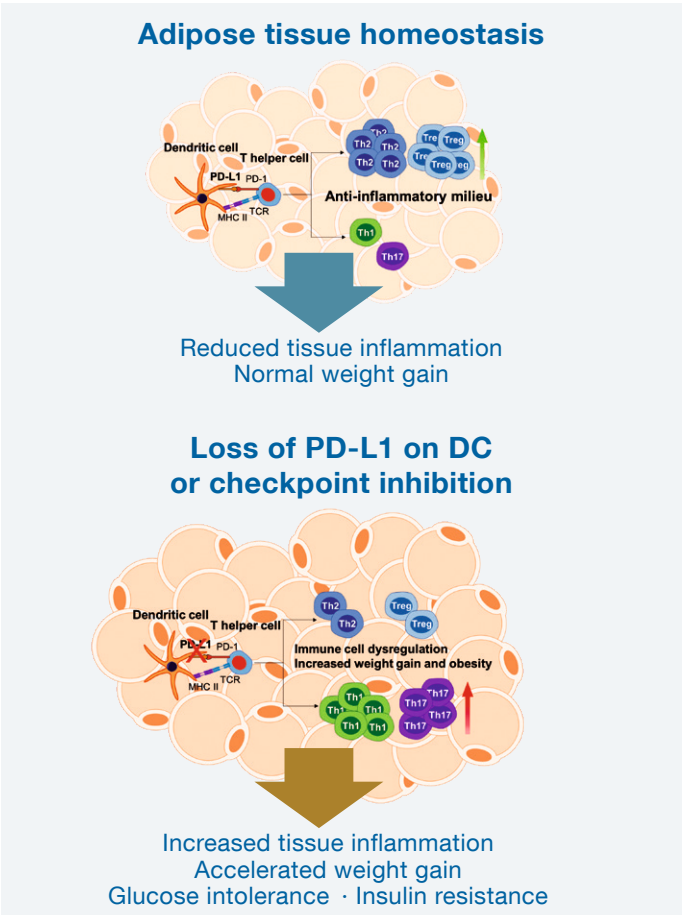
Immune cells in white adipose tissue switch to a pro-inflammatory phenotype during obesity. However, the underlying mechanisms are not well understood. Recently, we discovered a role for the immune checkpoint protein programmed death ligand 1 (PD-L1) in reducing inflammation in adipose tissue and limiting diet-induced obesity.

We compared weight gain after feeding a high-fat diet (HFD) in PD-L1$^{-/-}$ mice with that of wild-type (WT) mice. Mice genetically lacking PD-L1 on all cells gained more weight and had more inflammation in their tissues. PD-L1$^{-/-}$ mice also had lower glucose tolerance and increased insulin resistance compared to WT mice.

PD-L1 was upregulated on immune cells, including group 2 innate lymphoid cells (ILC2), dendritic cells (DC), macrophages, and T helper (Th) cells in adipose tissue of obese mice compared to non-obese controls. Using mice with selective knockout of PD-L1 on these immune cell populations, we found that PD-L1 loss on DCs (PD-L1$^{fl/fl}$DCCre mice) was responsible for accelerated weight gain during diet-induced obesity. These PD-L1$^{fl/fl}$DC^{Cre} mice had significantly increased levels of Th1 cells and significantly reduced levels of regulatory T cells in their adipose tissue, leading us to hypothesize that PD-L1:PD-1 signaling between DCs and T cells reduces adipose tissue inflammation and obesity by inhibiting the polarization of T cells towards the Th1 subset.

In order to confirm the relevance of these findings for humans, we compared the expression of PD-L1 mRNA transcripts in adipose tissue from lean individuals and individuals with obesity. Here, we found that PD-L1 correlated with the patient's body-mass-index. Furthermore, we found that PD-L1 protein was upregulated in immune cells isolated from adipose tissue of patients with obesity.

Treatment of obese mice with anti-PD-L1 monoclonal antibodies resulted in increased weight gain and polarization of T cells towards Th1 and Th17 subsets. When obese mice were switched to a control diet, PD-L1 inhibition resulted in a statistically significant decrease in glucose tolerance compared to isotype controls. Based on these results, we think that checkpoint inhibition therapy could affect the metabolic health of cancer patients.



PD-L1 on DC limits pro-inflammatory T cells in adipose tissue and limits obesity. Under steady state conditions, PD-L1 on DC supports adipose tissue homeostasis by interacting with T helper cells via PD-1. This interaction inhibits Th1 proliferation and promotes anti-inflammatory T cell polarization. Loss of PD-L1 on aggravates inflammation and accelerates weight gain and metabolic disease.

Thus, PD-L1 on DCs poses an important checkpoint for T cells, limiting their pro-inflammatory potential and thereby maintaining adipose tissue homeostasis. Further investigations on the DC subsets involved in adipose tissue homeostasis and the impact of checkpoint inhibition therapies in cancer on obesity and metabolic health will provide important insights into a field of continuous clinical relevance.

Schwartz C, Schmidt V, Deinzer A, Hawerkamp HC, Hams E, Bayerlein J, Röger O, Bailer M, Krautz C, El Gendy A, Elshafei M, Heneghan HM, Hogan Æ, O'Shea D, Fallon PG. Innate PD-L1 limits T cell-mediated adipose tissue inflammation and ameliorates diet-induced obesity. Sci Transl Med. 2022 Mar 9;14(635):eabj6879

SCIENTIFIC HIGHLIGHTS

IL-33-induced metabolic reprogramming controls the differentiation of alternatively activated macrophages and the resolution of inflammation

IL-33 promotes a mitochondrial rewiring of macrophages and the consecutive IL-4-independent differentiation of AAMs and the resolution of inflammation upon tissue injury.

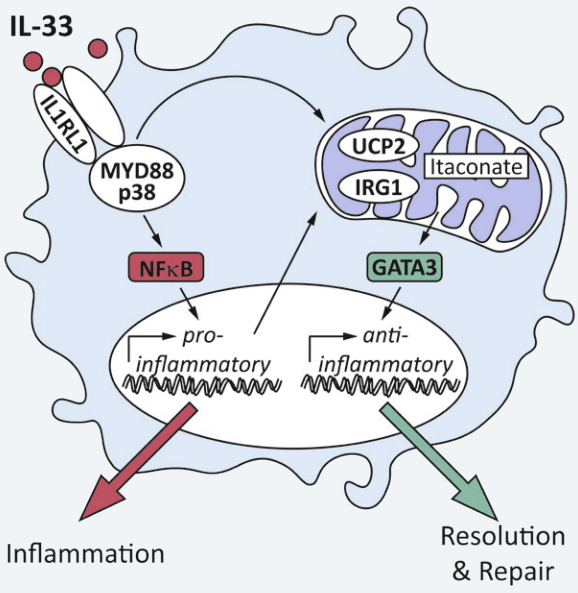
MARIA FAAS, GERHARD KRÖNKE
DEPARTMENT OF INTERNAL MEDICINE 3, UNIVERSITÄTSKLINIKUM ERLANGEN, FAU ERLANGEN-NÜRNBERG

Inflammation is a natural and vital response of our immune system to danger signals and tissue damage. The inflammatory process helps to eliminate the respective trigger, for example bacteria, and to set repair mechanisms in motion. Equally important, however, is a timely and coordinated resolution of this inflammatory response, otherwise there is a risk of developing a chronic inflammatory disease. Alternatively-activated macrophages (AAMs) play an important role during resolution of inflammation and tissue repair. However, molecular pathways controlling their differentiation have remained incompletely understood.

Recently, we found that the alarmin IL-33 sequentially triggered early expression of pro-inflammatory genes and subsequent differentiation into AAMs. Global analysis of underlying signaling events revealed that IL-33 induced a rapid metabolic rewiring of macrophages that involved uncoupling protein-2-mediated mitochondrial reprogramming, resulting in uncoupling of the respiratory chain and an increased production of the metabolite itaconate. This metabolic reprogramming subsequently promoted the alternative polarization of macrophages via the transcription factor GATA3. Deletion of GATA3 in mononuclear phagocytes in turn abrogated the differentiation of IL-33 induced AAMs and tissue repair upon tissue damage.

Our data indicate an IL-4-independent and GATA3-dependent pathway in mononuclear phagocytes that controls alternative activation and the resolution of inflammation. These data additionally emphasize the dual role of macrophages during inflammation, and revealed novel molecular targets to potentially impede inflammation-associated tissue damage, and simultaneously improve resolution of inflammation and tissue repair. Since the findings are based on a muscle injury model, it remains to be determined whether GATA3 also contributes to the plasticity and function of mononuclear phagocytes in other disease settings, such as parasite infection, metabolic disorders, cancer or Alzheimer's disease.

Faas M, Ipseiz N, Ackermann J, Culemann S, Grüneboom A, Schröder F, Rothe T, Scholtysek C, Eberhardt M, Böttcher M, Kirchner P, Stoll C, Ekici A, Fuchs M, Kunz M, Weigmann B, Wirtz S, Lang R, Hofmann J, Vera J, Vœhringer D, Michelucci A, Mougiakakos D, Uderhardt S, Schett G, Krönke G.
IL-33-induced metabolic reprogramming controls the differentiation of alternatively activated macrophages and the resolution of inflammation. Immunity. 2021 Nov 9;54(11):2531-2546.e5. doi: 10.1016/j.immuni.2021.09.010. Epub 2021 Oct 12. PMID: 34644537.



The alarmin IL-33 induces early expression of pro-inflammatory genes and the subsequent differentiation into AAMs via a rapid mitochondrial rewiring in macrophages. Uncoupling of the respiratory chain via UCP2 promotes itaconate production as well as GATA3 activation, leading to a pro resolving AAM phenotype fostering tissue repair.

SCIENTIFIC HIGHLIGHTS

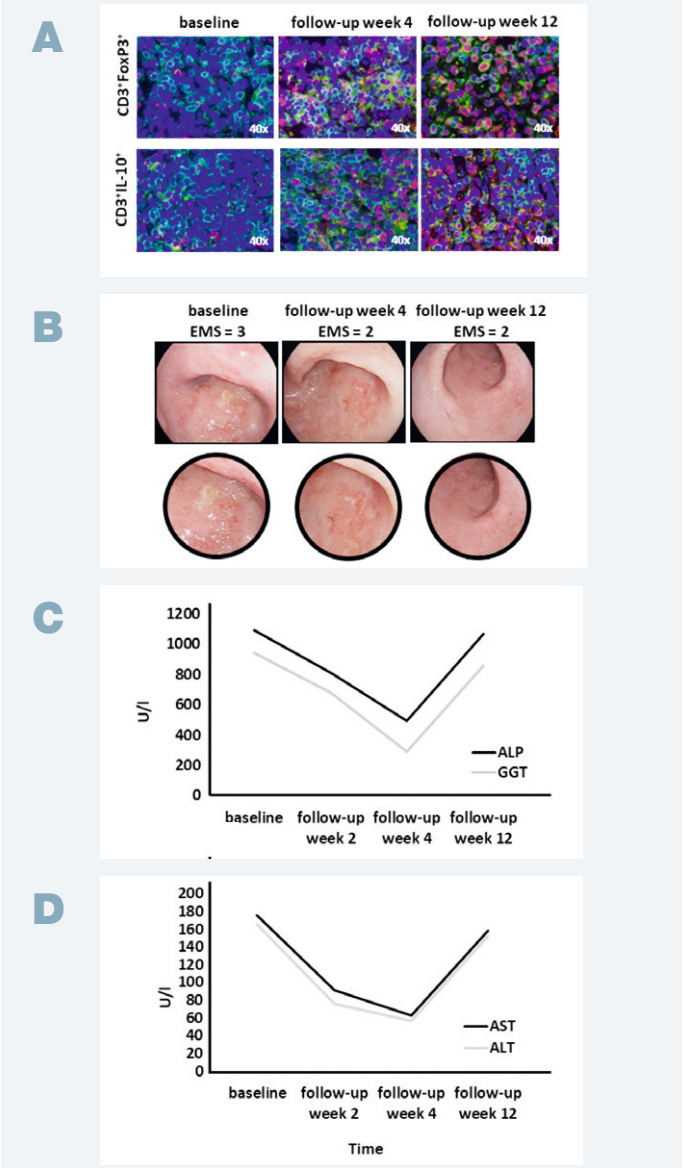
Treatment of refractory ulcerative colitis with concomitant primary sclerosing cholangitis with autologous regulatory T cells

Adoptively transferred regulatory T cells migrate to the gut and induce clinical, biochemical, endoscopic and histological signs of response until week 12 after adoptive transfer.

CAROLINE BOSCH-VOSKENS
DEPARTMENT OF DERMATOLOGY, UNIVERSITÄTSKLINIKUM ERLANGEN, FAU ERLANGEN-NÜRNBERG

Ulcerative colitis (UC) is a systemic disease that can manifest itself not only in the gut but also in extra-intestinal organs, including the skin, joints, eyes and hepatobiliary tract. An excess of intestinal effector T cells and an insufficient expansion of mucosal regulatory T cells (Tregs) is thought to drive inflammation in the gut. We have developed a protocol to produce autologous polyclonal Tregs intended for clinical use and currently perform a dose-finding clinical trial in patients with UC. We here report the unique observation that a single infusion of autologous Tregs induced a clinical, biochemical, endoscopic and histological response in a patient with UC and concomitant primary sclerosing cholangitis (PSC). PSC is an immune-mediated chronic liver disease with no effective therapy available and the close association with UC implicates the existence of a common liver-gut immune axis that drives these diseases. In the reported patient, clinical improvement of symptoms was long lasting and associated with a regression of ulcerations besides a substantial increase in FoxP3-expressing and IL-10 expressing Tregs in the gut. Although the corresponding improvement in PSC-specific laboratory values was transient, our findings strongly indicate that Tregs may have wide therapeutic application in chronic inflammatory disorders associated with an imbalance between effector and Treg subsets.

Voskens C, Stoica D, Rosenberg M, Vitali F, Zundler S, Ganslmayer M, Knott H, Wiesinger M, Wunder J, Kummer M, Siegmund B, Schnoy E, Rath T, Hartmann A, Hackstein H, Schuler-Thurner B, Berking C, Schuler G, Atreya R, Neurath M. Gut 2022 Apr 15:gutjnl-2022-327075



Enrichment of CD3⁺FoxP3⁺ and CD3⁺IL-10⁺ cell populations is associated with a regression of mucosal ulcerations in the gut and a transient improvement of liver values after treatment with autologous regulatory T cells. (**A**) Immunohistochemistry showing the enrichment of CD3⁺FoxP3⁺ and CD3⁺IL-10⁺ cells in the gut, (**B**) endoscopic examination showing a regression of ulcerations, (**C**) transient improvement of ALP, GGT, (**D**) AST and ALT levels after Treg transfer. ALP, alkaline phosphatase; GGT, gamma-glutamyl transferase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; Treg, regulatory T cell.

SCIENTIFIC HIGHLIGHTS

Sialic acids on B cells are crucial for their survival and provide protection against apoptosis

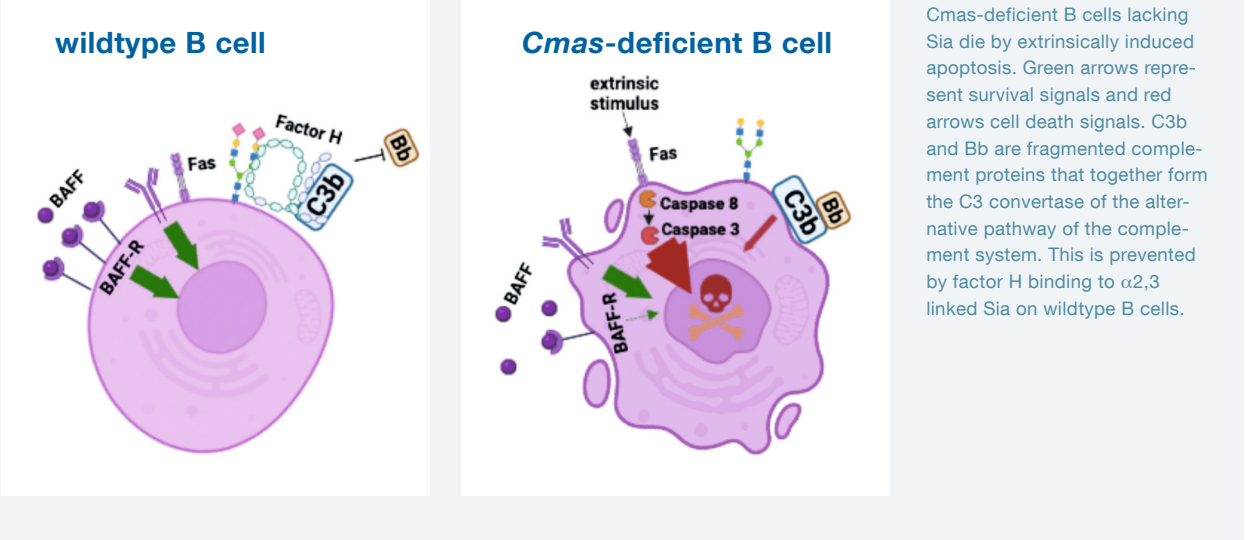
Sialic acids are essential for B cell survival

MICHAEL SCHMIDT, LARS NITSCHKE
DEPARTMENT OF BIOLOGY, FAU ERLANGEN-NÜRNBERG

Sialic acids (Sia) are 9-carbon sugars expressed on all vertebrate cells as the terminal carbohydrates of glycoproteins and glycolipids. These Sia containing glycans are also called sialoglycans. Because of their exposed location and diversity in derivates and binding linkage, they fulfil a lot of different functions. Sialoglycans on cellular surfaces are important for cell migration, control of the complement system, and by binding to the inhibitory receptors CD22 and Siglec-G on the same B cell surface they also regulate B cell activation. Previous mouse models with mutations in *CD22* and *Siglecg* from our group showed that sialoglycan binding of these receptors is crucial for regulation of their inhibitory function. *CD22*- and *Siglecg* -deficient mice also develop lupus-like autoimmune disease upon ageing. However, a model lacking all ligands for *CD22* and Siglec G on the B cell surface has not been generated so far.

In our present work we analyzed mice with a B cell-specific deletion of CMP-sialic acid synthase (*Cmas*), a gene essential for the synthesis of sialoglycans. The B cells of these mice lost all sialoglycans. Surprisingly, conditional *Cmas*-deficient mice showed a severe B cell deficiency in secondary lymphoid organs, leading to hardly any remaining peripheral B cells, no plasma cells and no serum antibodies. It was previously published that the fluid-phase regulatory complement protein factor H can bind to α2,3 linked sialoglycans and thereby prevents opsonization and destruction of host cells by the alternative pathway of the complement system (Blaum BS et al., Nat. Chem. Biol. 2015). In order to test, whether complement activation is mechanistically involved in the B cell deficiency of *Cmas*-deficient mice, we crossed these mice with mice lacking the central complement protein C3. Complement C3-deficiency rescued the phenotype of *Cmas*-deficient mice only marginally, demonstrating a largely complement-independent mechanism. B cells of *Cmas*-deficient mice also showed impaired upregulation of the B cell survival receptor BAFF-receptor. Additionally, B cells of *Cmas*-deficient mice had high levels of activated caspase 3 and processed caspase 8, which could be further induced by the death receptor Fas, indicating ongoing apoptosis. Overexpression of Bcl2 did not rescue this phenotype, pointing at an extrinsic apoptosis process. These results show that sialoglycans on the B cell surface are crucial for B cell survival by counteracting several death-inducing pathways.

Linder AT, Schmidt M, Hitschfel J, Abeln M, Schneider P, Gerardy-Schahn R, Münster-Kühnel AK, Nitschke L.
Sialic acids on B cells are crucial for their survival and provide protection against apoptosis. Proc Natl Acad Sci USA. 2022 Jun 21;119(25):e2201129119. doi: 10.1073



Cmas-deficient B cells lacking Sia die by extrinsically induced apoptosis. Green arrows represent survival signals and red arrows cell death signals. C3b and Bb are fragmented complement proteins that together form the C3 convertase of the alternative pathway of the complement system. This is prevented by factor H binding to α2,3 linked Sia on wildtype B cells.

PEOPLE

Prof. Dr. rer. nat. Alexander Steinkasserer

m^4 Award for Prof. Alexander Steinkasserer

Dr. Dmytro Royzman and Prof. Dr. Alexander Steinkasserer received the m4 Award in the context of the BioEntrepreneurship Summit 2021

The Department of Immune Modulation, headed by Prof. Dr. Alexander Steinkasserer, focuses on the biological and therapeutic function of the immune checkpoint molecule CD83. Especially, the soluble isoform of the CD83 protein (sCD83) exerted immune-modulatory and pro-resolving capacities in preclinical autoimmune models of arthritis, inflammatory bowel disease and multiple sclerosis, but also transplant rejections were significantly reduced in murine skin-, heart- and cornea transplantation models. Recent data extended this knowledge also with respect to cutaneous wound healing (DOI: 10.3389/fimu.2022.1012647). Concomit-antly, also a boosting effect on hair regrowth was observed, thus providing a potential new approach for the future treatment of wound healing and hair loss.

The “Bayerisches Staatsministerium für Wirtschaft, Landesentwicklung und Energie” awarded this research now with the m4 Award 2021, with a total sum of 500.000 €. Thus, within the next two years, the group of Alexander Steinkasserer and his co-workers aim to translate this preclinical data into the human system and to elucidate the underlying molecular and cellular mechanisms. Beyond that, toxicity and pharmacological studies are planned.

We congratulate Prof. Steinkasserer and his team on the m^4 award and wish them much success for their future studies.

Prof. Dr. med. Stefan Uderhardt

European Research Council (ERC) grant for Prof. Dr. med. Stefan Uderhardt

Prof. Dr. med. Stefan Uderhardt, who works at Department of Medicine 3 at Universitätsklinikum Erlangen, has been awarded with an ERC Starting grant comprising approximately 1.5 million euros for his project “NEXUS – Network Synergies in Tissue Homeostasis and Stromal Prevention of Inflammatory Disease”.

Prof. Dr. med. Stefan Uderhardt

Stefan Uderhardt studied medicine at FAU and began to train as a specialist in rheumatology and clinical immunology at Department of Medicine 3 under Prof. Dr. Georg Schett. Following a five year research stay at the renowned National Institutes of Health (NIH) in the USA, he has been in charge of his own research group at the Universitätsklinikum Erlangen in conjunction with the Optical Imaging Centre Erlangen (OICE) since 2020 and has been an assistant professor for rheumatology at FAU since 2021. Recently, he received a W2 professorship for integrative tissue immunology.

We congratulate Prof. Uderhardt on his ERC grant and wish him all the best for his future research.

PEOPLE

PD Dr. med. Kilian Schober

FAU T cell researcher receives several honors

PD Dr. med. Kilian Schober

In recent years, the importance of T cells has been very much appreciated because of their protective role against pandemic viruses, but also because of their use as “living drugs” in the fight against cancer or autoimmune diseases. The junior research group of PD Dr. med. Kilian Schober at the Institute of Clinical Microbiology, Immunology and Hygiene (Director: Prof. Dr. med. Christian Bogdan) aims to understand human antigen-specific T-cell responses and develops novel forms of T cell-based therapies. To this end, Dr. Schober’s team combines state-of-the-art technologies for tracking T cells (single-cell sequencing) with innovative methods of genetic engineering using CRISPR/Cas9. For his outstanding work, Kilian Schober was awarded the prestigious Postdoctoral Award in Immunology by the Robert Koch-Foundation in November 2021 and the GlaxoSmithKline Foundation Science Award in July 2022. Recently, Dr. Schober was also elected into the Young Council of the Bavarian Academy of Sciences.

We congratulate Dr. Schober on these honors!

Dr. rer.nat. Christian Schwartz

Dr. rer. nat. Christian Schwart received BMBF funding “Obesity ImpAcT”

Within the framework of the call for proposals “Junior Research Groups in Infection Research” of the German Federal Ministry of Education and Research (BMBF), Dr. rer. nat. Christian Schwartz, research group leader at the Institute of Clinical Microbiology, Immunology and Hygiene (Director: Prof. Dr. Christian Bogdan), was able to acquire a 5 year-grant endowed with 2.3 Mio €. The aim of his work is to find out why wound healing and tissue regeneration are poorer in overweight and obese people, and why the antimicrobial immune response is impaired. To this end, Dr. Schwartz’s research group is using infection models to study tissue repair mechanisms in the lungs, intestines, and skin. Through collaboration with physicians at the UK Erlangen, another aim is to substantiate these results with clinical samples from people with and without obesity, with the ultimate goal to develop new treatment approaches.

Dr. rer. nat. Christian Schwartz

Dr. Schwartz studied biology at the Eberhard Karls University in Tübingen. After his diploma thesis at LMU Munich, he came to Erlangen in 2011 together with his PhD supervisor Prof. David Vöhringer (Department of Infection Biology). After his PhD on the role of basophils during helminth infections and other type 2 immune responses, he joined the Translational Immunology Group of Prof. Padraic Fallon at Trinity College Dublin, Ireland, as a PostDoc in 2015 and became an EMBO Fellow shortly after. Since 2019, he has been leading his own research group at the Microbiology Institute and was recently awarded the status of FAU Junior Research Group Leader.

We sincerely congratulate Dr. Schwartz and wish much much success in his work on the funded project.

PEOPLE

Prof. Dr. rer. nat. Christoph Becker,

Prof. Dr. med. Gerhard Krönke,

Prof. Dr. med. Andreas Mackensen

Continuation of the funding of three DFG research consortia

This year, three research consortia at the University Hospital Erlangen and at FAU were very positively re-evaluated and will receive further funding by the DFG:

• Collaborative Research Center TRR241 "Immune-Epithelial Communication in Inflammatory Bowel Diseases" (2022 to 2026; spokesperson: Prof. Dr. rer. nat. Christoph Becker, Medical Clinic 1)

• Research Unit 2886 "PANDORA – Pathways triggering AutoimmuNity and Defining Onset of early Rheumatoid Arthritis" (2022 – 2025; spokesperson: Prof. Dr. med. Gerhard Krönke, Medical Clinic 3)

• TRR221 "Controlling graft-versus-host and graft-versus-leukemia immune reactions after allogenic stem cell transplants" (2022 – 2025; spokesperson: Prof. Dr. med. Andreas Mackensen, Medical Clinic 5)

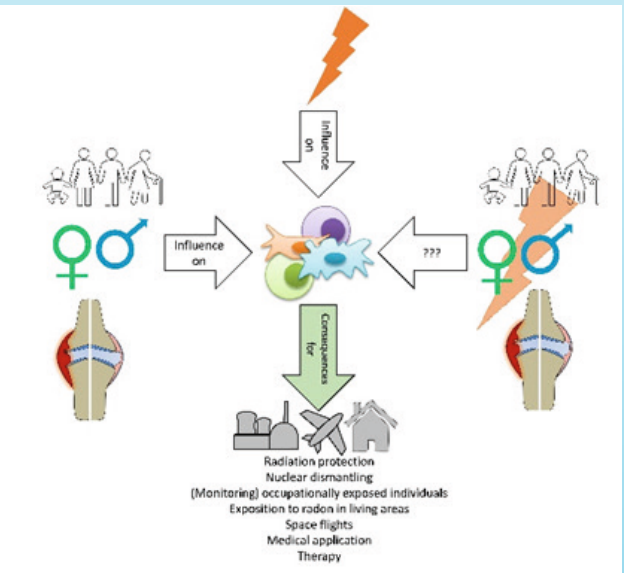
PEOPLE

Dr. rer.nat. Lisa Deloch

Dr. rer. nat. Lisa Deloch obtained funding by the Federal Ministry of Education and Research (BMBF)

As part of the highly competitive call for Junior Research Groups from the natural and engineering sciences "Kreativer Nachwuchs forscht für die Nukleare Sicherheits-, Strahlen- und Rückbauforschung (NukSiFutur)" by the Federal Ministry of Education and Research (BMBF), Dr. Lisa Deloch managed to obtain funding of 1.8 million euros for a junior research group located in Erlangen. The Radiation Osteoimmunology working group she leads at the Erlangen Radiation Clinic (Director: Prof. Dr. Rainer Fietkau) in the Translational Radiation Biology Laboratory (Head: Prof. Dr. Udo Gaipl) will be able to assess the effects of various radiation sources on the immune system as well as on the bone metabolism over the next 5 years. The group will further examine the influence of age, gender and the basic inflammatory status. Within this framework, three PhD students in the natural and engineering sciences can be recruited to create competence in radiation research. Existing European collaborations with France, Hungary and Sweden, among others, are also being intensified by carrying out some of the experiments with specific radiation sources on site.

We congratulate Dr. Deloch on this funding and wish her good luck and success for her future work.



Age, gender and inflammatory status have an impact on the immune system. While radiation itself can modulate the immune system, it has not yet been clarified what influence ionizing radiation has on the immune system and on bone metabolism, in dependence of the above-mentioned factors.

NEWS AND UPDATES

Joachim Kalden Lecture 2022

The Medical Immunology Campus Erlangen honors Dr. Anne O'Garra

The Joachim Kalden Lecture 2022, which took place on July 5, 2022, was delivered by Dr. Anne O'Garra, The Francis Crick Institute, London, United Kingdom.

Dr. Anne O'Garra's main research interest lies in the regulation of the immune response during immune challenge and infection. In this context, her lab aims to identify immune cells, pathways and targets of protection and pathogenesis determining disease outcome in mice and men. Particularly, the group studies the regulation of cytokine-driven pathways in myeloid cells and T cells leading to or controlling pathology in various models of infection. A major focus lies on the investigation of cellular and gene expression changes at mucosal sites, specifically in (i) the lung and airways after infection with *M. tuberculosis* as well as (ii) in the gut and periphery after infection with *H. hepaticus*, *T. gondii* or *E. coli*.

Anne O'Garra was born in Gibraltar and graduated at the MRC National Institute for Medical Research (MRC NIMR), London, United Kingdom (today part of The Francis Crick Institute) in the middle of the 80s of the last century. After a postdoctoral fellowship in Palo Alto, USA, she went back to the MRC NIMR to become the head of the Division of Immunoregulation and later on the associated research director of the Francis Crick Institute, London. Since 2019, she is concentrating more on her research again at her own request and is currently principal group leader at the Crick Institute and Professor of Infection Immunology at the Imperial College, London.

Anne O'Garra is author of more than 230 scientific publications, which have received broad attention (h-index 100, more than 56 000 citations). Dr. O'Garra is scientific board member of various high-ranking journals and various scientific organizations and holds several patents.

In her lecture, Anne O'Garra focused on the immune response to *Mycobacterium tuberculosis* and highlighted her findings on the expression and deleterious function of type I interferons in tuberculosis. She also presented exciting findings on the differential transcriptional signature in active versus latent TB.

Dr. Anne O'Garra holding the certificate of the Joachim Kalden Lecture together with Prof. Christian Bogdan (spokesman of the *Medical Immunology Campus Erlangen* and Director of the Institute of Clinical Microbiology, Immunology and Hygiene).

https://www.mice.fau.de/veranstaltungen/joachim-kalden-lecture/

"Type I interferons protect against viruses and cancer, but frequently exacerbate bacterial infections."

Anne O´Garra

NEWS AND UPDATES

Prof. Dr. rer. nat. Claudia Günther, Dr. rer. nat. Ulrike Steffen

Appointment as new members of the executive board of the Medical Immunology Campus Erlangen

It is with great pleasure that the *Medical Immunology Campus Erlangen* welcomes two excellent scientists as new board members: Prof. Dr. rer. nat. Claudia Günther (Medicine 1) and Dr. Ulrike Steffen (Medicine 3).

Prof. Dr. rer. nat. Claudia Günther studied Biology in Mainz and graduated in the Department of Medicine 1, FAU Erlangen-Nürnberg. Afterwards, she became a group leader of the "Laboratory of Mucosal Infection Biology" and finished her habilitation for experimental medicine in 2017. Since 2020, she holds a W2 professorship in the Department of Medicine 1. Claudia Günther is the author of more than 50 scientific publications. In 2018, she received the *Thiersch Award* of the Medical Faculty of the FAU, and in 2022 the *German Medical Award*.

We congratulate Claudia Günther on becoming a member of the executive board of the *Medical Immunology Campus Erlangen* and very much look forward to working together.

Dr. Ulrike Steffen (née Harre) studied Biochemistry in Leipzig before she started her dissertation at the FAU Erlangen-Nürnberg in the lab of Professor G. Schett, where she graduated with *summa cum laude*. Since 2016 she is a group leader at the Department of Medicine 3, FAU Erlangen-Nürnberg and is author of more than 35 scientific publications.

We congratulate Ulrike Steffen on her appointment as a board member and very much look forward to her participation in the *Medical Immunology Campus Erlangen*.

As new members of the executive board Prof. Günther will give a presentation within the seminar series of the *Medical Immunology Campus Erlangen* on January 10, 2023, while we will welcome Dr. Steffen in the summer term 2023.

Prof. Dr. rer. nat. Claudia Günther

Dr. rer. nat. Ulrike Steffen

New: Immunological Colloquium Tuesdays at 12:30 pm

There is another novelty at the *Medical Immunology Campus Erlangen:* Starting in January 2023, the Immunological Colloquium will routinely take place on Tuesdays at 12:30 pm. With this, we would like to enable all parents of children in need of care to also participate in the seminar.

UPCOMING EVENTS

Immunological Colloquium of the Medical Immunology Campus Erlangen – Winter 2022/23

Tuesdays, 12.30 pm

10.01.2023
Prof. Claudia Günther
Medical Clinic 1, Mucosal Infection Biology,
Universitätsklinikum Erlangen

17.01.2023 online 5:15 pm
Prof. Alicia Ponte-sucre
Lab of Molecular Physiology,
Universidad Central de Venezuela,
Physiological Sciences,
Instituto de Medicina Experimental,
Caracas, Venezuela

24.01.2023
Dr. Moritz Gaidt
IMP - Research Institute of Molecular Pathology,
Campus-Vienna-Biocenter, Vienna, Austria

07.02.2023
Prof. Adam Croft
Rheumatology Research Group,
Institute of Inflammation and Ageing (IIA),
University of Birmingham, UK

28.02.2023
Prof. Christoph Wilhelm
Institute of Clinical Chemistry and Clinical
Pharmacology, Universitätsklinikum Bonn

For latest information's please visit:
https://www.mice.fau.de/veranstaltungen/immunologisches-kolloquium/

Conferences and Events of Interest 2023

January 12 – 14, 2023 · Braunschweig
Meeting AK Vakzine

February 01 – 03, 2023 · Waischenfeld
Meeting AK Dendritische Zellen

March 05 – 10, 2023 · Ettal
DGfI Spring School of Immunology

March 08, 2023
International Women`s Day 2023
Online meeting

March 22 – 24, 2023 · Burg Rothenfels
Meeting AK Infektionsimmunologie

March 27 – 29, 2023 · Meschede
Meeting AK Biologie der B-Lymphozyten

March 27 – 29, 2023 · Hamburg
Meeting AK Komplementsystem

March 28 – 31, 2023 · Ulm
Annual Meeting of the Society for Virology

April 20 – 22, 2023 · Halle/Saale
Meeting AK Tumorimmunologie TIMO2023

April 22 – 24, 2023 · Copenhagen, Denmark
Springtime School 2023
Skin homeostasis and inflammation

April 29, 2023
Day of Immunology

May 03 – 05, 2023 · Mainz
20th CIMT Annual Meeting

May 04 – 07, 2023 · Gdańsk, Poland
8th European Congress of Virology EUSV

May 11 – 15, 2023 · Washington, DC, USA
Annual Meeting of the American Association of Immunologists

June 22 – 23, 2023 · Schloss Rauischholzhausen
Meeting AK Allergie und Immunologie

June 28 – 30, 2023 · Erlangen
International Symposium RTG2740

June 29 – July 01, 2023 · Marburg
Meeting AK T-Zellen

July 05 – 08, 2023 · Davos, Switzerland
17th World Immune Regulation Meeting

September 26 – 29, 2023 · Strassbourg, France
Joint Meeting of SFI and DGFI

October 05 – 07, 2023 · Erlangen
International Symposium RTG2599

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We are looking forward to suggestions for the next MICE newsletter. Please send material to: Ilka.Knippertz@uk-erlangen.de

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