Medical Immunology Campus Erlangen

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

Newsletter
Winter 2016 · Spring 2017

CONTENT

FRIEDRICH-ALEXANDER UNIVERSITÄT ERLANGEN-NÜRNBERG

EDITORIAL

Dear colleagues and friends,

In June 2016, the Federal Government and the Federal States of Germany decided to continue and expand the Excellence Initiative to further strengthen high-level research at German universities. At the end of September 2016, the funding line "Cluster of Excellence" (CE) was officially announced by the DFG and the German Council of Science and Humanities. Already in July 2016, Prof. Achim Hornegger, President of FAU, had invited representatives of all FAU faculties to present innovative research fields that appeared promising for cluster applications, which were then consolidated during a two day retreat in October. With this editorial we would like to briefly update all members of the *Medical Immunology Campus Erlangen* on the scientific rationale and central goal of the CE preproposal *ImmunoPhysics* that has been submitted to the DFG on March 31.

Immune-mediated diseases are a particular challenge in medicine because they are frequently chronic and difficult to treat. Although numerous experimental and clinical studies have revealed the key immunological players that drive inflammatory responses, the parameters that account for the process of disease chronification are still incompletely defined. During a joint train ride in April 2014, we had a first intense discussion on the urgent need for new quantitative technologies in immunological research in order to gain insights into the molecular nature of the tissue micromilieu during inflammatory processes. During the July 2016 meeting, we proposed that in addition to classical proinflammatory immune cells and cytokines, non-immune, i.e. physicochemical and biophysical factors are critical for the generation of a pathological immune response. This idea became the central hypothesis of our cluster application and is supported by prior observations at FAU that microenvironmental factors such as tonicity, hypoxia or lipid metabolites affect the function of immune cells (e.g. *Nat Medicine* 15, 545-552, 2009; *J Invest Dermatol* 134, 2339-2346, 2014; *Cell Metab* 21, 493-501, 2015; *J Clin Invest* 125, 1944-1954, 2015; *Immunity* 43, 817-829, 2015; *New Engl J Med* 376, 2017 in press). Furthermore, the basic biomechanical properties of cells as well as the physicochemical composition of their membranes are also affected during chronic inflammations. The cluster *ImmunoPhysics* pursues the vision to tackle these physicochemical factors with the ultimate goal to develop a new understanding of chronic inflammatory diseases and to define entirely novel therapeutic targets. The strong immunological, biophysical and technical expertise at the FAU along with the *Max Planck Institute for the Science of Light* and the planned *Max-Planck-Zentrum für Physik und Medizin* forms the scientific and structural backbone for this research endeavour.

We invite all members of the *Medical Immunology Campus Erlangen* to share their thoughts and ideas with us for further refinement of the *ImmunoPhysics* concept and to keep their fingers crossed that we will be invited in September to prepare a full proposal.

Prof. Georg Schett | Prof. Christian Bogdan | Prof. Vahid Sandoghdar

SCIENTIFIC HIGHLIGHTS

Blocking GATA-3 reduces colitis in mice

Rectal Delivery of a DNAzyme That Specifically Blocks the Transcription Factor GATA3 Reduces Colitis in Mice

VANESSA POPP · BENNO WEIGMANN · MARKUS F. NEURATH
DEPARTMENT OF MEDICINE 1, FAU ERLANGEN-NÜRNBERG, UNIVERSITÄTSKLINIKUM ERLANGEN

Inflammatory bowel disease (IBD) is composed of two major disorders: ulcerative colitis (UC) and Crohn's disease (CD). Previous studies have highlighted an important pathogenic role in these diseases of both the innate and the adaptive immune system. Within the adaptive immune system, UC patients showed upregulated T_H2 cell-derived cytokines causing mucosal inflammation.

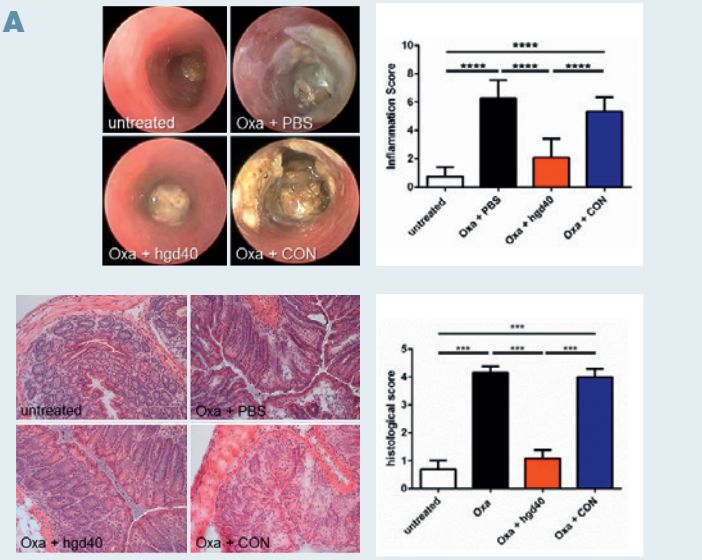
GATA-3 is the major transcription factor of T_H2 - cell differentiation. By activation of production of pro-inflammatory cytokines like IL-5 and IL-13, GATA-3 is considered as the pacemaker of Type 2 immune response-mediated UC.

Patients with UC showed a significant higher GATA-3 expression in the lamina propria cells compared to healthy controls correlating with inflammation intensity. Functional analyses revealed a protection of conditional GATA-3 CD4 KO mice compared to control mice in an experimental model of colitis induced by rectal administration of oxazolone. These results were supported by miniendoscopic analysis and histological colon sections. Next, we targeted GATA-3 expression by using a GATA-3-specific DNAzyme (hgd40) to inhibit mRNA expression.

We observed a protective effect of hgd40 in the oxazolone colitis when it was applicated prophylactically before inflammation induction, but also when it was applied therapeutically when the inflammation was already established in the colon. Mice that received the DNAzyme showed no inflammation in the colon compared to mice that received a placebo control. By analyzing the production of cytokines, we found reduced levels of important inflammatory cytokines in mice that revealed the GATA-3 DNAzyme.

Taking these results together, we believe that the GATA-3 DNAzyme emerges as a novel approach for therapy in human ulcerative colitis.

Popp V., Gerlach K., Mott S., Turowska A., Garn H., Atreya R., Lehr H.A., Ho I.C., Renz H., Weigmann B. and Neurath M.F. 2017. Rectal Delivery of a DNAzyme. That Specifically Blocks the Transcription Factor GATA3 and Reduces Colitis in Mice. Gastroenterology 152:176-192 e5.



Figures:

A) Rectal administration of the GATA-3 specific DNAzyme hgd40 ameliorates signs of colonic inflammation in mice during the oxazolone-induced colitis model indicated with miniendoscopy images, inflammation scoring and H&E staining of colonic cryosections

B) The *Gata-3* mRNA level in T_H2 cells is significantly downregulated after hgd40 administration. Due to this reduced transcription factor level, the production of inflammatory cytokines like IL-13, IL-6, IL-5 and IL-9 is suppressed.

SCIENTIFIC HIGHLIGHTS

Mechanistic insights in TNF-mediated pathogen control

TNF restricts arginase 1 expression by epigenetic regulation and thereby facilitates NO production by type 2 NO synthase at the site of infection

KATRIN PADUCH · CHRISTIAN BOGDAN · ULRIKE SCHLEICHER
INSTITUTE FOR CLINICAL MICROBIOLOGY, IMMUNOLOGY AND HYGIENE, FAU ERLANGEN-NÜRNBERG, UNIVERSITÄTSKLINIKUM ERLANGEN

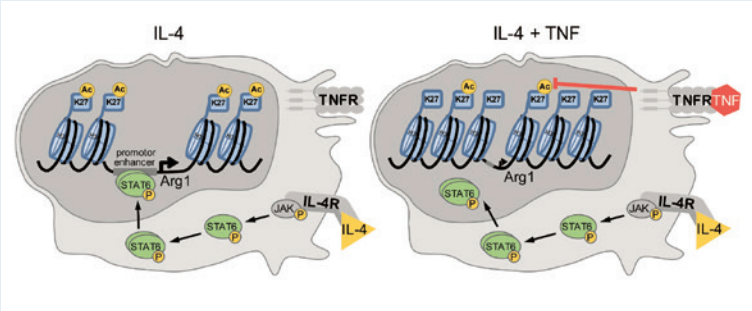
The pro-inflammatory cytokine tumor necrosis factor (TNF) plays a key role in initiating protective immunity to infections with intracellular pathogens, but the underlying mechanisms are incompletely understood.

In infections caused by the intracellular parasite *Leishmania (L.) major* effective control requires an interferon (IFN)γ-based immune response including activation of type 2 nitric oxide (NO) synthase (NOS2). The activity of NOS2, which metabolizes L-arginine to citrulline and antimicrobial NO, can be modulated by host cell-derived arginase 1 (Arg1), a competing enzyme that hydrolyses L-arginine into urea and ornithine. In the absence of TNF otherwise self-healing C57BL/6 mice succumbed to *L. major* infection despite an intact IFNγ-response and a sustained expression of NOS2 in the infected tissues.

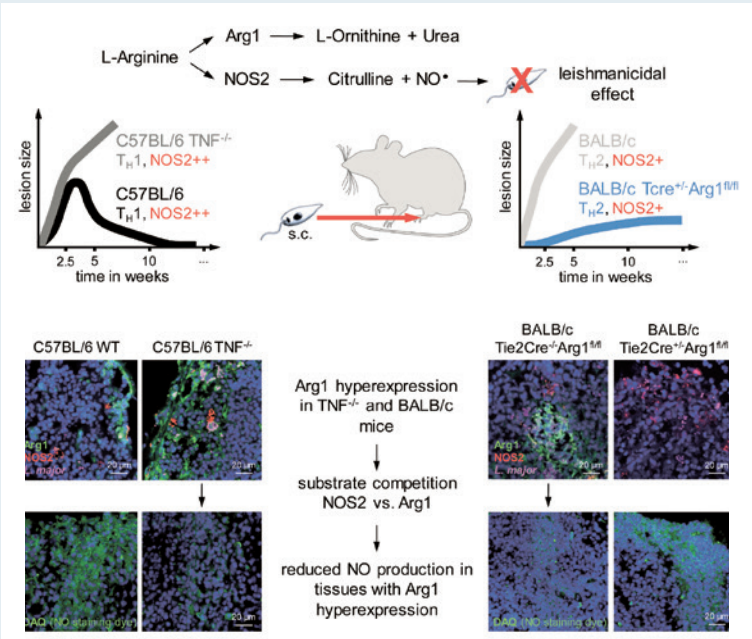
In our work, we showed that TNF acts as a negative regulator of interleukin (IL)-4-induced Arg1 in macrophages and dendritic cells. TNF neither suppressed phosphorylation nor nuclear translocation of STAT6, the key transcription factor for IL-4-mediated Arg1 induction, but reduced the acetylation of histone residues that are critical for the opening of the promoter and regulatory region of the Arg1 gene locus and thereby impaired the binding of STAT6 to the DNA. TNF-deficiency in *L. major*-infected C57BL/6 mice caused hyperexpression of Arg1 which led to an increased frequency of Arg1/NOS2 double-positive cells and a reduced release of NO at the site of infection. Conversely, in *L. major*-infected BALB/c mice deletion of Arg1 unleashed the production of NO and protected them from an otherwise lethal infection, although their Th2 response was maintained.

In summary, our data identify a novel pathway by which TNF confers protection against intracellular pathogens. Understanding the molecular mechanisms of TNF-mediated antimicrobial defence might help to develop adjunct therapies to prevent infections in patients with autoimmune diseases treated with TNF-antagonists.

Arg1 expression *in vitro* in myeloid cells



Leishmania major* infection *in vivo



Schleicher U., Paduch K., Debus A., Obermeyer S., Konig T., Kling J.C., Ribechini E., Dudziak D., Mougiakakos D., Murray P.J., Ostuni R., Korner H. and Bogdan C. 2016. TNF-Mediated Restriction of Arginase 1 Expression in Myeloid Cells Triggers Type 2 NO Synthase Activity at the Site of Infection. *Cell Rep* 15:1062-75.

SCIENTIFIC HIGHLIGHTS

CD83 modulates B cell activation and is required for thymic CD4 T cell selection

The CD83 molecule modulates B cell activation and germinal center responses and is required for thymic CD4 T cells selection via the inhibition of March8-mediated MHC II degradation in thymic cortical epithelial cells.

LENA KRZYZAK[1] · ANDREAS WILD[1] · THOMAS H. WINKLER[2] · LARS NITSCHKE[2]
ALEXANDER STEINKASSERER[1]

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[2] DIVISION OF GENETICS, DEPARTMENT OF BIOLOGY, FAU ERLANGEN-NÜRNBERG

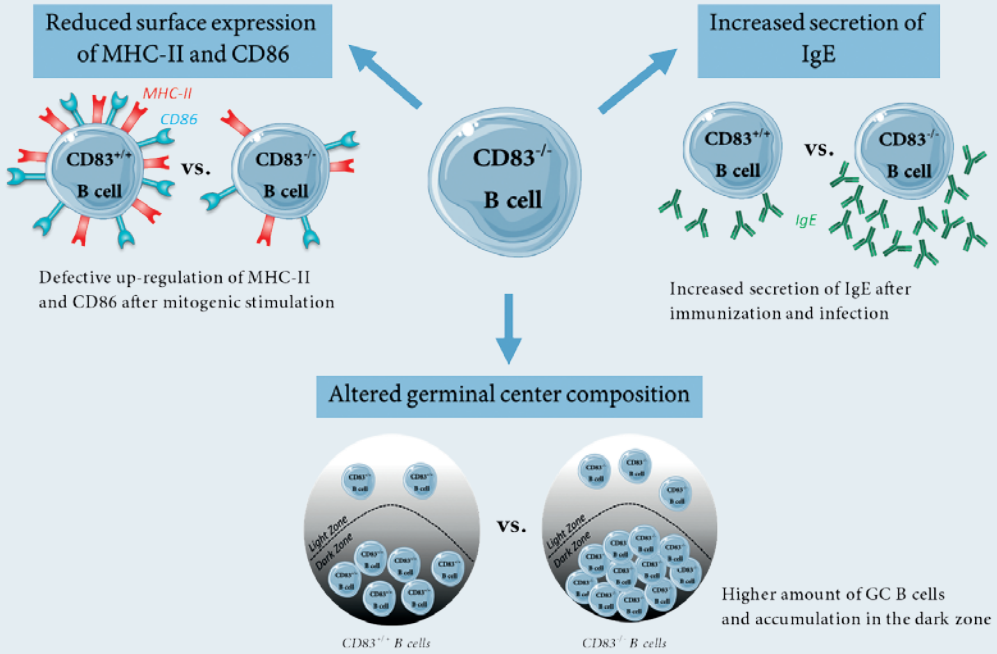


Figure:

Left: Defective upregulation of MHC II and CD86 on CD83 cKO B cells. Purified splenic B cells were stimulated and analysed by FACS.

Right: Increased secretion of IgE after immunization with NP-KLH or infection with *B. burgdorferi.*

Middle: Increased GC B cells and accumulation in the dark zone in CD83 cKO mice after immunization.

CD83 is one of the best maturation markers for dendritic cells. In the B cell lineage, CD83 is expressed on activated B cells and on light zone (LZ) B cells during the germinal center (GC) reaction. To analyze the function of CD83 on B cells during GC responses, we generated B cell specific CD83 conditional-knock-out (CD83 B-cKO) mice. CD83 B-cKO B cells showed defective upregulation of MHCII and CD86 expression and impaired proliferation. Analyses of GC responses after immunization revealed a shift in dark zone (DZ) and LZ B cell numbers, with increased B cells in the DZ of CD83 B-cKO mice (see figure). This effect was not accompanied by alterations in IgG level responses. However, enhanced IgE responses were observed in CD83 B-cKO mice. In addition, we observed a strong competitive disadvantage of CD83-cKO B cells in GC responses in mixed bone marrow chimeras. Furthermore, infection of mice with *B. burgdorferi* revealed defects in bacterial clearance of CD83 B-cKO mice with a shift towards a Th2 response, underlined by a strong increase in IgE titers. These results show that CD83 is important for B cell activation, modulates GC-composition and IgE antibody responses *in vivo*.

Furthermore, CD83 complete-knockout mice showed a strong reduction of $CD4^+$ T cells. In collaboration with Prof. Ludger Klein from Munich, we found that the transmembrane domain of CD83 is necessary and sufficient for thymic CD4 T cell selection, via the inhibition of MARCH8 induced MHC-II degradation. Thus, CD83 plays an important role not only in the modulation of dendritic cells but also for B cells and thymic T cell selection.

Krzyzak L., Seitz C., Urbat A., Hutzler S., Ostalecki C., Gläsner J., Hiergeist A., Gessner A., Winkler T.H., Steinkasserer A., Nitschke L. 2016. CD83 Modulates B Cell Activation and Germinal Center Responses. *J Immunol* 196:3581-94.

von Rohrscheidt J., Petrozziello E., Nedjic J., Federle C., Krzyzak L., Ploegh H.L., Ishido S., Steinkasserer A. and Klein L. 2016. Thymic CD4 T cell selection requires attenuation of March8-mediated MHCII turnover in cortical epithelial cells through CD83. *J Exp Med* 213:1685-94.

SCIENTIFIC HIGHLIGHTS

Siglec-H protects from virus-triggered severe systemic autoimmunity

Siglec-H is a receptor on plasmacytoid dendritic cells which inhibits type I interferon responses

HEIKE SCHMITT · THOMAS H. WINKLER · LARS NITSCHKE
DIVISION OF GENETICS, DEPARTMENT OF BIOLOGY, FAU ERLANGEN-NÜRNBERG

It is controversial whether virus infections can contribute to the development of autoimmune diseases. Type I interferons (IFNs) confer antiviral effects during virus infections, but have also been implicated in the pathogenesis of systemic lupus erythematosus (SLE). Type I IFNs are mainly produced by plasmacytoid dendritic cells (pDCs). The secretion of typeI IFN by pDCs is modulated by Siglec-H, a DAP12- associated receptor on pDCs. We showed that Siglec H-deficient pDCs produce more of the typeI interferon IFN-α *in vitro* and that Siglec-H ko mice produce more IFN-α after murine cytomegalovirus (mCMV) infection *in vivo*. Siglec-H ko mice developed a severe form of systemic lupus-like autoimmune disease with strong kidney nephritis several weeks after a single mCMV infection (panels A and B). This induction of systemic autoimmune disease after virus infection in Siglec-H ko mice was accompanied by a type I IFN signature and fully dependent on type I IFN signaling. These results show that Siglec-H normally serves as modulator of type I IFN responses after infection with a persistent virus and thereby prevents induction of autoimmune disease (panel C).

This work was done by Heike Schmitt, a PhD student/postdoc in the lab of Lars Nitschke, in a close cooperation with the lab of Thomas Winkler. Further groups involved were C. Daniel (Nephropathology) and Sophia Sonnewald (Division of Biochemistry), both FAU. The work was supported by the DFG through RTG 1660, CRC 1181 and TRR130.

Schmitt H., Sell S., Koch J., Seefried M., Sonnewald S., Daniel C., Winkler T.H., Nitschke L. 2016. Siglec-H protects from virus-triggered severe systemic autoimmunity. *J Exp Med* 213:1627-44.

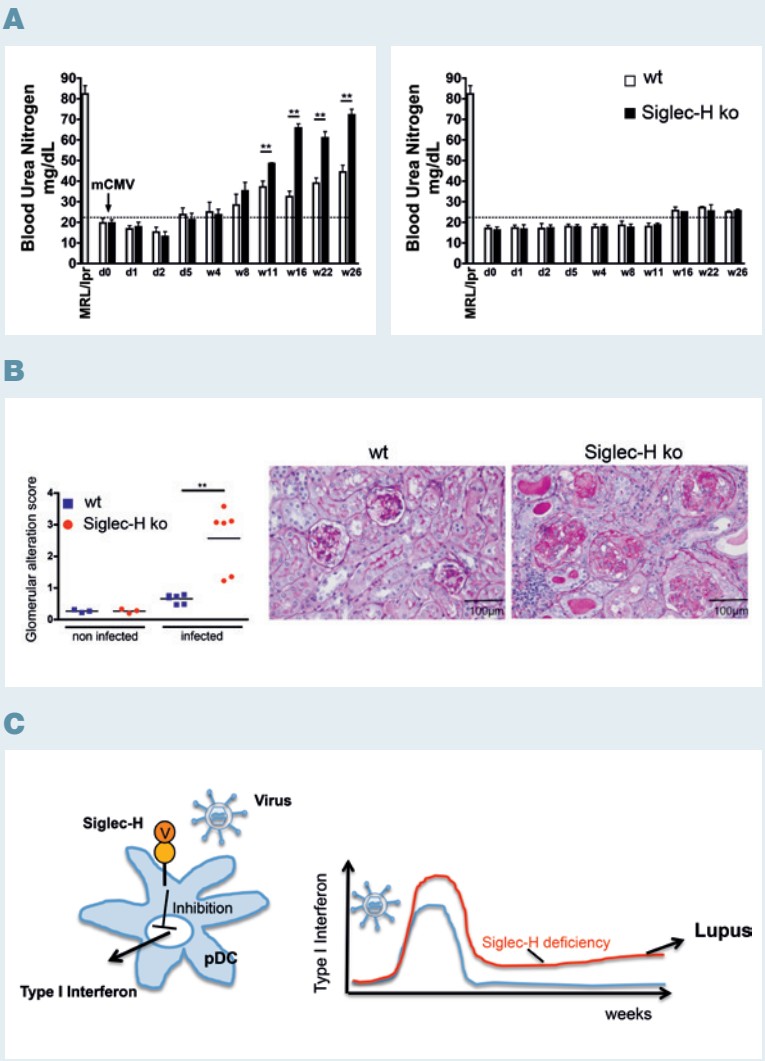


Figure:

A) Urea nitrogen contents in the blood of mCMV-infected mice (left) or uninfected mice (right) were determined using an enzymatic BUN kit. Increased BUN levels indicate a dysfunction of the kidney.

B) Strong kidney damage in Siglec-H KO mice 26 weeks after mCMV infection. Severe glomerular alterations that are typical for kidney nephritis were observed in mCMV-infected Siglec-H KO mice.

C) Model for the role of Siglec-H on plasmacytoid dendritic cells: Siglec-H inhibits type I interferon production, which is induced by a persistant virus infection. Siglec-H KO mice develop a strong lupus-like disease several weeks after virus infection due to ongoing type I interferon production.

SCIENTIFIC HIGHLIGHTS

A novel mechanism of nuclear lamina disassembly

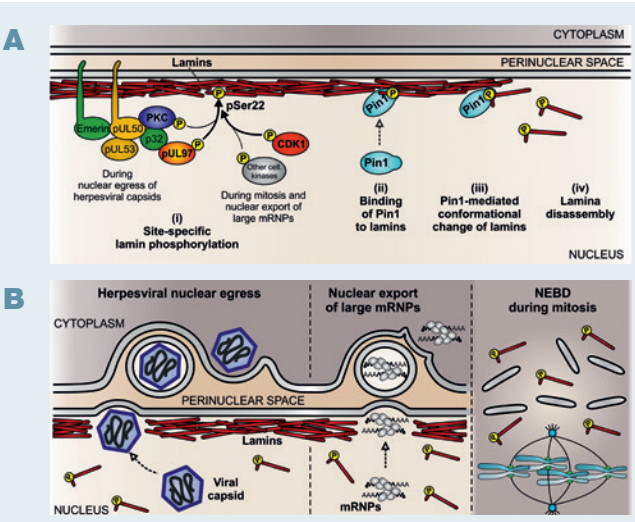
The prolyl isomerase Pin1 promotes the herpesvirus-induced phosphorylation-dependent disassembly of the nuclear lamina required for nucleocytoplasmic egress

JENS MILBRADT INSTITUTE FOR CLINICAL AND MOLECULAR VIROLOGY, FAU ERLANGEN-NÜRNBERG, UNIVERSITÄTSKLINIKUM ERLANGEN

Viruses often adopt preexisting cellular pathways to promote their own replication. In this regard, the recently discovered alternative mechanism for the nuclear export of large messenger ribonucleoprotein (mRNP) complexes is particularly noteworthy. This process is mechanistically similar to the nuclear egress of herpesviruses, which appear to utilize cellular pathways and effectors to release assembled capsids from the host nucleus. While vesicle formation and scission events at nuclear membranes are now increasingly understood in greater detail, the precise mechanism of the preceding disassembly of the nuclear lamina still awaits a defined molecular characterization.

In our study, we used herpesviruses in their property to induce a nucleocytoplasmic viral capsid export for our investigation of nuclear lamina disassembly. We identified a mechanism that promotes lamina disassembly by a conformational change of lamins, mediated by the cellular isomerase Pin1 in a phosphorylation-dependent manner. Intriguingly, Pin1 appeared to control the rearrangement of phosphorylated lamins and their transient displacement from the nuclear lamina. Our study suggests that Pin1 functions as a major regulatory effector of lamina disassembly and thus determines the nuclear egress pathway of herpesviruses.

J. Milbradt, C. Hutterer, H. Bahsi, S. Wagner, E. Sonntag, A.H.C. Horn, B.B. Kaufer, Y. Mori, H. Sticht, T. Fossen, M. Marschall. 2016 The Prolyl Isomerase Pin1 Promotes the Herpesvirus-Induced Phosphorylation-Dependent Disassembly of the Nuclear Lamina Required for Nucleocytoplasmic Egress. PLoS Pathogens 12: e1005825



Putative conserved mechanism of Pin1-induced nuclear lamina disassembly

A) Our findings suggest a functional role of Pin1 leading to disassembly of the nuclear lamina in response to herpesviral or inherent cellular stimuli. In both cases, Ser22-specific lamin phosphorylation generates a Pin1-binding motif (i). After binding (ii), Pin1-mediated isomerization then induces a conformational change in the lamin N-terminus (iii) resulting in local lamina disassembly (iv).

B) Cellular processes which require disassembly of the nuclear lamina. *mRNP*, messenger ribonucleoprotein complexes; *NEBD*, nuclear envelope breakdown.

PEOPLE

Prof. Raja Atreya

Prof. Raja Atreya has been appointed as Heisenberg Professor at the Friedrich-Alexander-Universität Erlangen-Nürnberg

Dr. Atreya, junior professor and senior physician at the Department of Medicine 3, Universitätsklinikum Erlangen, has received the renowned Heisenberg professorship granted by the DFG (German Research Foundation). The Heisenberg programme of the DFG aims to support outstanding researchers and to increase their reputation in the field. Also, after five years, if the candidate proves successful, the Heisenberg Professorship will be converted in a permanent professorship at the FAU. Atreya's Heisenberg professorship entitled "Translational immunology in inflammatory bowel diseases (IBD)" is dedicated to the development of novel therapeutic approaches for diseases like Morbus Crohn and Colitis ulcerosa. This involves not only basic research on the pathogenesis of the diseases but also clinical research trials in patients with IBD.

Prof. Atreya studied Medicine at the University of Mainz where he also received his M.D. in the lab of Prof. Markus Neurath. In August 2010, Dr. Atreya came to Erlangen and was appointed junior professor and later became head of the outpatient clinic and the research clinic. In 2015, Prof. Atreya was awarded the Paul-Ehrlich-und-Ludwig-Darmstädter-Nachwuchspreis for his discovery of a diagnostic signal spray which can be applied during colonoscopy to detect mTNF expressing cells in the human gut and thus predict the outcome of a response to an anti-TNF therapy.

NEWS AND UPDATES

Joachim Kalden Lecture 2016

Prof. Fiona Powrie from the University of Oxford, UK was the eighth honoree for the Joachim Kalden Lecture of the *Medical Immunology Campus Erlangen*

The Joachim Kalden Lecture 2016 of the *Medical Immunology Campus Erlangen* took place on November 8, 2016 and was delivered by Professor Fiona Powrie, Director of the Kennedy Institute at the University of Oxford, UK. Dr Powrie's main research interests focus on the interaction between the intestinal microbiota and the host immune system and how it collapses in inflammatory bowel disease (IBD) and cancer. Fiona Powrie's work has identified the functional role of regulatory T cells in intestinal homeostasis and shed light on their development and mechanism of action. She has also shown that both adaptive and innate immune mechanisms contribute to intestinal inflammation and identified the IL-23 pathway as a pivotal player in the pathogenesis of chronic intestinal inflammation.

Fiona Powrie received many scientific distinctions like the Ita Askonas Award from the European Federation of Immunological Societies for her contribution to immunology in Europe and the Louis-Jeantet Prize for Medicine in 2012. She was elected a Fellow of the Royal Society in 2011, an EMBO fellow in 2013 and a Fellow of the British Academy of Medical Sciences in 2014.

In her lecture entitled "Gut reactions: Immune pathways in the intestine in health and disease", Prof. Powrie talked about the development and function of different types of regulatory T cells in IBD. She summarized the work of her group on IL-23 and the GM-CSF-receptor β in different models of mouse colitis and illustrated how eosinophils change from tissue protective to tissue damaging cells under the influence of IL-5 (important for eosinophil survival) and GM-CSF (necessary for eosinophil activation). Finally, she presented new data on the role of IL-33 in colitis and the overexpression of oncostatin M in human IBD. Mice lacking oncostatin M showed an ameliorated course of colitis.

From left: Prof. Joachim Kalden (former Director of the Department of Medicine III), Prof. Fiona Powrie holding the certificate of the Joachim Kalden Lecture and Prof. Christian Bogdan (spokesman of the *Medical Immunology Campus Erlangen* and Director of the Institute of Clinical Microbiology, Immunology and Hygiene). Source: Prof. S. Krappmann

Special Seminar by Prof. Jacques Miller, Melbourne, Australia

On October 14, just before the beginning of the winter seminar series 2016/17, a very special guest, Jacques Miller from Melbourne, Australia, visited us in Erlangen and held an extraordinary seminar on "Thymus function revealed" for the *Medical Immunology Campus Erlangen*. The immunologist Prof. Miller is famous for identifying the function of the thymus and discovering that T and B cell lymphocytes have different origins and roles. Many peers believe that for his funamental findings, Prof. Miller has long been overdue for the Nobel Prize in Medicine.

During his talk, Dr. Miller gave fascinating insights into the discovery of T lymphocytes and the early research on their function.

Born in Nice, Professor Miller spent his early years in France, Switzerland and China before his family moved to Sydney in 1941. Professor Miller went to St Aloysius' College and later studied medicine at Sydney University. After working in England and the USA, Professor Miller returned to Melbourne in 1966, where he was hired as a research director and was later appointed director of the illustrious Walter and Eliza Hall Institute of Medical Research in Melbourne, Australia. Semi-retired since 1996, 86 year old Prof. Miller is still involved in immunological research.

UPCOMING EVENTS

Immunological Colloquium of the Medical Immunology Campus Erlangen – Summer 2017

Tuesdays, 5.15 pm

02. 05. 2017
Prof. Matthias Tenbusch
Klinische und Molekulare Virologie, Universitätsklinikum Erlangen, FAU
Title to be announced

09. 05. 2017
Univ.-Prof. Mathias Hornef
Institut für Medizinische Mikrobiologie, Universitätsklinik der RWTH Aachen
Ontogeny of the intestinal mucosal host response to infection

23. 05. 2017
Prof. Christian Schulz
Klinikum der Universität München, Ludwig-Maximilians-Universität München
Macrophage development – does origin matter?

30. 05. 2017
Prof. Alfred Zippelius
Department of Biomedicine, University Hospital Basel, Switzerland
Cancer Immunotherapy: Strategies for personalization and combination approaches

13. 06. 2017
PD Dr. Stella Autenrieth
Universitätsklinikum Tübingen
Modulation of dendritic cells by bacterial pathogens

20. 06. 2017
Prof. Martin Staege
Universitätsklinikum Halle (Saale)
Gene Expression Music applications in immunology and cancer research

27. 06. 2017
Prof. Christian Münz
Institute of Experimental Immunology, University of Zürich, Switzerland
Infection and immune control of human tumor viruses in vivo

04. 07. 2017
Prof. Burkhard Ludewig
Institut für Immunbiologie, Kantonspital St. Gallen, Switzerland
Stromal cell – innate lymphoid cell interaction

11. 07. 2017
Prof. Christoph Wilhelm
Institute of Clinical Chemistry and Clinical Pharmacology, University Hospital Bonn
Metabolic Regulation of ILC Mediated Barrier Immunity

18. 07. 2017
Prof. Hanspeter Pircher
Institute for Immunology, University Medical Center Freiburg
Antibodies and tissue-resident memory T cells in LCMV infection

Further Conferences and Events of Interest

May 20, 2017 (10 am – 4 pm)
Day of Immunology 2017

To celebrate the "International Day of Immunology" doctoral students of the DFG research training groups GK1660 and IRTG TRR130 are arranging an exciting and versatile program to educate the public about aspects of the human immune system. At the Hugenottenplatz in Erlangen they will provide information about the immune system, vaccination and infection. Especially children are welcome to discover our fascinating immune system and its enemies by playing fun games and winning attractive prices.

September 21 – 23, 2017
31st Annual Conference of the European Macrophage and Dendritic Cell Society (EMDS)

Madrid, Spain www.macrophage.de

September 12 – 19, 2017
47th Annual Meeting of the German Society for Immunology

Erlangen

Dear colleagues and friends,

The 47th Annual Meeting will be held in the Heinrich-Lades-Halle located in the center of Erlangen.

We cordially invite everyone to submit abstracts in spring 2017 and to join us for this exciting event. The highlight of the 2017 meeting will be the celebration of the 50th birthday of our Society. To salute this special occasion, the organizing committee has compiled an exciting scientific program highlighting current research topics in basic and translational immunology in plenary sessions, main symposia, and workshops. A poster exhibition "50 years DGfI – 50 discoveries" and a Festschrift will commemorate the history of our Society and the highlights of Immunology in Germany. We will finish with an unforgettable birthday party on the last evening of the congress.

Please visit the congress website www.immunology-conference.de for detailed information.

We hope to make this special meeting a memorable event!

Sincerely yours,
Hans-Martin Jäck Congress President

Confirmed Invited Speakers

Friederike Berberich-Siebelt Würzburg/DE • Gordon Brown Aberdeen/GB • Katrin Busch Heidelbg./DE
Dirk H. Busch Munich/DE • Marco A. Cassatella Verona/IT • Adelheid Cerwenka Heidelberg/DE
Gérard Eberl Paris/FR • Markus Feuerer Heidelberg/DE • Irmgard Förster Bonn/DE
Reinhold Förster Hannover/DE • Dale Ian Godfrey Melbourne/AU Christopher Goodnow Canberra/AU
Francesca Granucci Milano/IT • Olaf Groß Munich/DE • Anja Hauser Berlin/DE
Vigo Heissmeyer Planegg-Martinsried/DE • Veit Hornung Munich/DE • Samuel Huber Hamburg/DE
Jonathan Jantsch Regensburg/DE • Julia Jellusova Freiburg/DE • Erika Jensen-Jarolim Vienna/AT
Axel Kallies Parkville Victoria /AU • Stefan Kaufmann Berlin/DE • Manfred Kopf Schlieren/CH
Marina Kreutz Regensburg/DE • Anne Krug Planegg-Martinsried/DE • Michael Lohoff Marburg/DE
Rudolf A. Manz Lübeck/DE • Tak Wah Mak Toronto/CA • Bastian Opitz Berlin/DE
Klaus Dieter Pfeffer Düsseldorf/DE • Klaus Rajewsky Berlin/DE • Felix Randow Cambridge/GB
Harald Renz Marburg/DE • Claude-Agnes Reynaud Paris/FR • Shimon Sakaguchi Kyoto/JP
Wolfgang Schamel Freiburg/DE • Alexander Scheffold Berlin/DE • Georg Schett Erlangen/DE
Andreas Strasser Bundoora Victoria/AU • Victor Tybulewicz London/GB
Reinhard Edmund Voll Freiburg/DE • Ari Waisman Mainz/DE • Jürgen Wienands Göttingen/DE
Sho Yamasaki Fukuoka/JP • Wayne M. Yokoyama St. Louis MO/US • Dietmar Zehn Freising/DE

September 23 – 30, 2017
19th International Summer School on Immunology

Hvar, Croatia www.febs-immunology-course.org

Medical Immunology Campus Erlangen

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