Medical Immunology Campus Erlangen

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

Newsletter
Summer 2014

CONTENT

FRIEDRICH-ALEXANDER
UNIVERSITÄT
ERLANGEN-NÜRNBERG

EDITORIAL

Dear colleagues and friends,

Prior to the summer break the Bavarian Government has just decided and announced that almost 600 million Euro will become available during the next four years to support the structural development of research and technology in the North of Bavaria. The FAU will particularly profit from this initiative, because approximately 115 Million Euro of the program will end up directly or indirectly at our university. These projects include the establishment of a *Helmholtz Institute for Renewable Energies* and the installation of a new *Max-Planck-Center for Physics and Medicine*. The latter will be a joint venture of the Max-Planck-Society and the Faculties of Medicine and Natural Sciences at the FAU. The *Max-Planck-Center* aims to apply innovative physical and mathematical methods to address medically relevant research questions. As a central topic, it is envisaged to measure the physical properties of the microenvironment surrounding individual cells in tissues with novel sensor and nanoscale imaging techniques that will be developed according to specific research ideas. It is not intended that the Max-Planck-Center is restricted to defined organs or research fields; instead, it will pursue broad and interdisciplinary approaches. Considering that the field of immunology currently experiences a revival of the analysis of biochemical and metabolic processes as well as a strong interest in the study of microenvironmental factors and their impact on immune responses, I am sure that members of the *Medical Immunology Campus Erlangen* will come up with exciting and challenging scientific questions to be addressed in the context of the new *Max-Planck-Center*.

During the past 12 weeks, we also received good news from the German Research Council (DFG). First, the *Research Training Group 1660* "Schlüsselsignale der adaptiven Immunität" has been approved for a second 4 ½ year funding period (2014 – 2019). Congratulations to its speaker Hans-Martin Jäck and the coordinator Anja Glanz, who certainly had to carry the largest burden of work for the reapplication, and to all researchers who have contributed to the program. Second, the DFG also positively evaluated the preproposal for a new *Collaborative Research Center CRC 1181* "Checkpoints for Resolution and Chronicity of Inflammation" that had been initiated by Georg Schett and Markus Neurath. Currently, the full proposal, which will contain 19 research projects and a central imaging platform, is prepared and will be submitted in autumn. Finally, Ivana Ivanovic-Burmazovic (Chair of Bioinorganic Chemistry) developed a concept for a new DFG Research Training Group together with several members of the *Medical Immunology Campus Erlangen* and the Natural Science Faculty, which will focus on inorganic redox signaling and prospects for the therapeutic manipulation of the *in vivo* redox status by small molecules. The respective preproposal has been sent to the DFG at the end of June and awaits evaluation by the reviewers.

I wish you all some relaxing days of vacation during the forthcoming weeks and hope to see you back at our guest seminars starting in the middle of October.

Christian Bogdan

Prof. Christian Bogdan
Chairman of the *Medical Immunology Campus Erlangen*

SCIENTIFIC HIGHLIGHTS

Kamikaze cells stop inflammation

Aggregated NETs limit neutrophil-driven inflammation by degrading cytokines and chemokines

CHRISTINE SCHAUER, MARKUS HOFFMANN, MARTIN HERRMANN
DEPARTMENT OF MEDICINE 3, UNIVERSITÄTSKLINIKUM ERLANGEN

... during early stages of neutrophil-driven inflammation NETs are formed and further neutrophils are attracted by cytokine release ...

... high neutrophil density leads to the formation of aggregated NETs (aggNETs) which degrade inflammatory mediators thereby orchestrating the resolution of inflammation ...

Gout is characterized by recurrent attacks of acute inflammation that causes sudden and severe pain, swelling and tenderness mostly affecting the joints of fingers and feet. The disease is triggered when uric acid converts into needle-shaped, pro-inflammatory monosodium urate (MSU) crystals. Surprisingly, inflammatory symptoms attenuate within a few days despite the ongoing presence of MSU in the affected tissues. In our manuscript, we describe a new mechanism that explains this formerly enigmatic process of self-limiting inflammation. In the presence of MSU, neutrophils cultured in low cell densities, representing the early phase of neutrophil-driven inflammation, eject their DNA decorated with antimicrobial proteins to create neutrophil extracellular traps (NETs). This process is dependent on an oxidative burst and accompanied by the release of pro-inflammatory cytokines/chemokines that attract further neutrophils and thus contributes to the inflammation at the beginning of an acute attack. In contrast, in areas of high cell densities, comparable with cells in tissue infiltrates during later stages of the attack, MSU stimulates the formation of high amounts of NETs that form felted, aggregated NETs (aggNETs). The latter degrade the inflammatory mediators via inherent serine proteases and thus initiate the process of self-limiting resolution of inflammation. In models of neutrophilic inflammation, Ncf1-knockout mice, which are NETosis-deficient, develop exacerbated and chronic disease that can be reduced by adoptive transfer of aggNETs. These findings confirm that aggNETs are responsible for the resolution of neutrophil-induced inflammation by degrading cytokines/chemokines and thus limiting further neutrophil recruitment and activation.

C. Schauer, · C. Janko · L.E. Munoz · Y. Zhao · D. Kienhofer · B. Frey · M. Lell · B. Manger · J. Rech · E. Naschberger · R. Holmdahl · V. Krenn · T. Harrer · I. Jeremic · R. Bilyy · G. Schett · M. Hoffmann · M. Herrmann. 2014. Aggregated neutrophil extracellular traps limit inflammation by degrading cytokines and chemokines. *Nature Medicine* 20:511–517.

FIGURE 1A

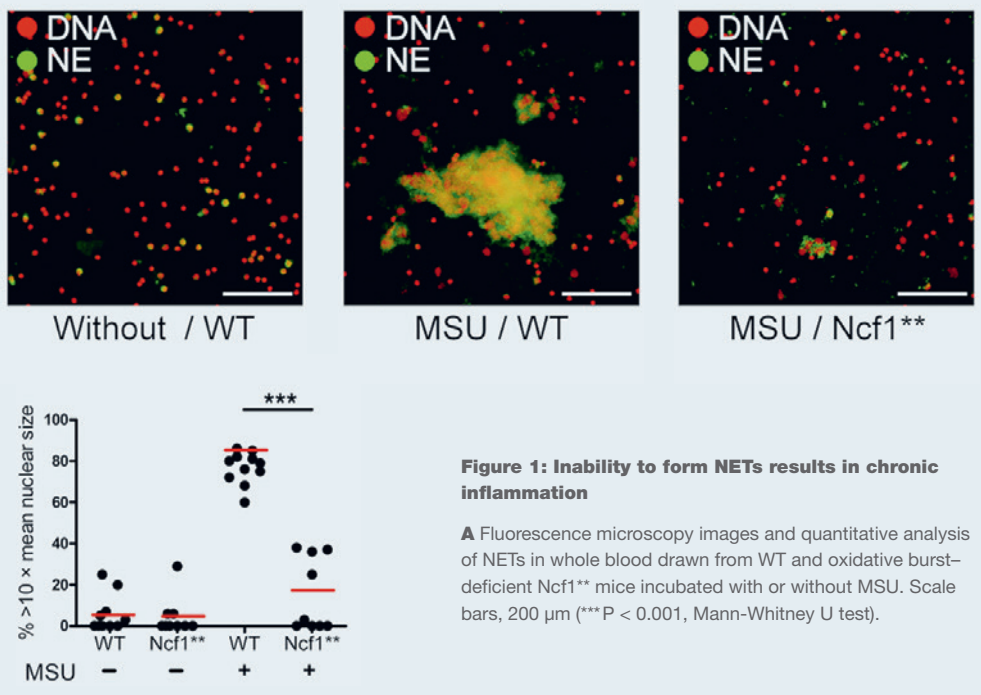


Figure 1: Inability to form NETs results in chronic inflammation

A Fluorescence microscopy images and quantitative analysis of NETs in whole blood drawn from WT and oxidative burst–deficient Ncf1** mice incubated with or without MSU. Scale bars, 200 µm (***P < 0.001, Mann-Whitney U test).

FIGURE 1

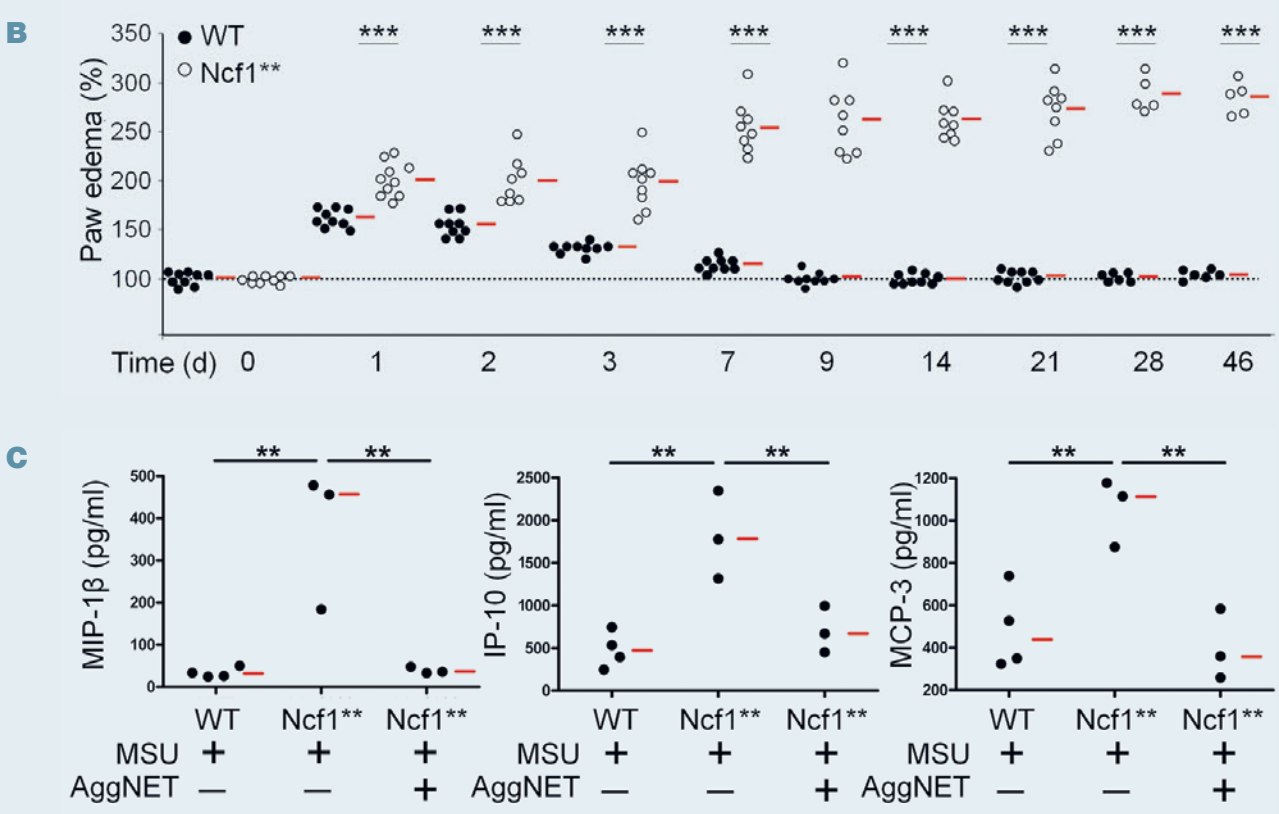


FIGURE 2

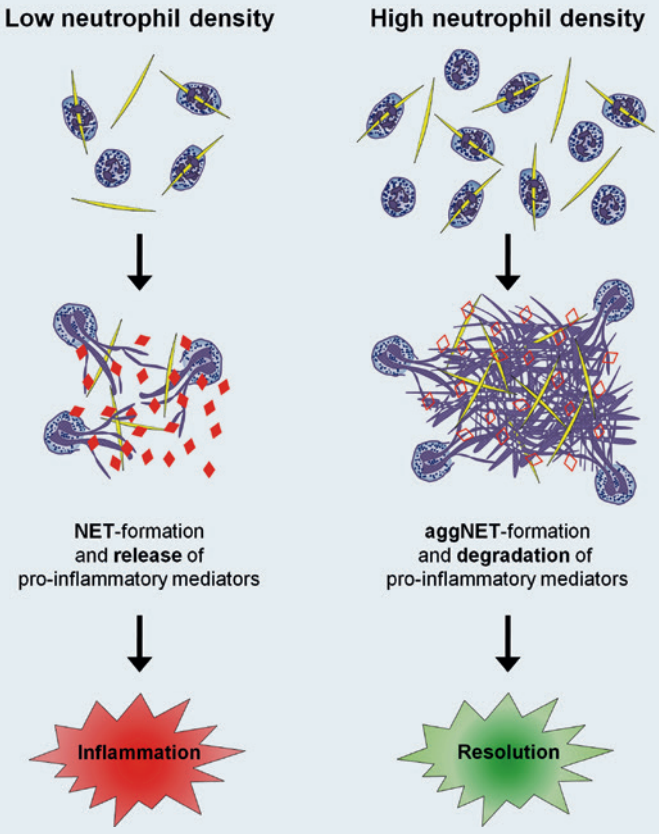


Figure 1: Inability to form NETs results in chronic inflammation

B Paw edema elicited by injection of MSU into the foot pads of Ncf1** and WT mice. The scatter plots show individual measurements and means of the relative thickness of the MSU-injected foot (***P < 0.001, Mann-Whitney U test).

C Inflammatory mediators in the lavage of air pouches of WT and Ncf1** mice after injection of MSU with (+) or without (–) preformed aggNETs (**P < 0.01, Student's t and Dunnett's post-hoc test).

Figure 2: Induction and resolution of inflammation by neutrophils

In low neutrophil densities, comparable to early stages of gouty arthritis, neutrophils induce single NETs, concomitant with the release of inflammatory mediators. Those chemokines/cytokines attract and activate further neutrophils resulting in high local neutrophil densities characteristic for the late stages of gouty arthritis. In high neutrophil densities cells form aggregated NETs (aggNETs) that trap and degrade inflammatory mediators and thus orchestrate the resolution of inflammation.

SCIENTIFIC HIGHLIGHTS

B cells are key for inflammation in Treg-deficient mice

An unexpected role for B cells and autoantibodies for the systemic autoimmune pathology in Scurfy mice

SUSANNE ASCHERMANN, FALK NIMMERJAHN CHAIR OF GENETICS, DEPARTMENT OF BIOLOGY, FAU

... Scurfy mice and humans with IPEX syndrom show mutations in the gene FoxP3 (critical for functionality of Tregs) leading to loss of immunological tolerance ...

... to study whether T helper cells are directly or indirectly responsible for tissue inflammation, we generated B cell deficient Scurfy mice ...

... we show that B cells and the production of autoantibodies play a major role for tissue inflammation ...

The transcription factor FoxP3 is critical for the development of functional regulatory T cells (Tregs). Mutations affecting FoxP3 function result in a loss of immunological tolerance in mice and humans. The resulting chronic autoimmune phenotype in Scurfy mice and in human patients with the immune dysregulation, polyendocrinopathy, enteropathy, X-linked (IPEX) syndrome is characterized by infiltrations of activated immune cells consisting of B cells, T cells, dendritic cells, monocytes and eosinophils into several organs such as the skin, lung, kidney and the liver, ultimately leading to organ failure and a premature death of affected individuals. The only curative therapy for human IPEX patients so far is allogeneic stem cell transplantation, which in many cases is hampered by the bad overall health of affected patients. Surprisingly, previous studies have shown that T helper cells but not cytotoxic T cells are critical for the disease pathology prompting us to study if this T cell subset is responsible directly for tissue inflammation or rather indirectly via the interaction with B cells or myeloid cells. By generating B cell deficient Scurfy mice, we show that B cells and the production of autoantibodies play a major role for skin, liver, lung and kidney inflammation and therapeutic depletion of B cells resulted in reduced tissue pathology and in prolonged survival of mice. In contrast, the absence of B cells did not impact systemic T cell activation and hyper-reactivity, indicating that autoantibody production by B cells may be a major factor for the autoimmune pathology in mice deficient for regulatory T cells.

S. Aschermann · C.H. Lehmann · S. Mihai · G. Schett · D. Dudziak · F. Nimmerjahn. 2013.
B cells are critical for autoimmune pathology in Scurfy mice. *Proceedings of the National Academy of Sciences of the United States of America* 110:19042–19047.

FIGURE

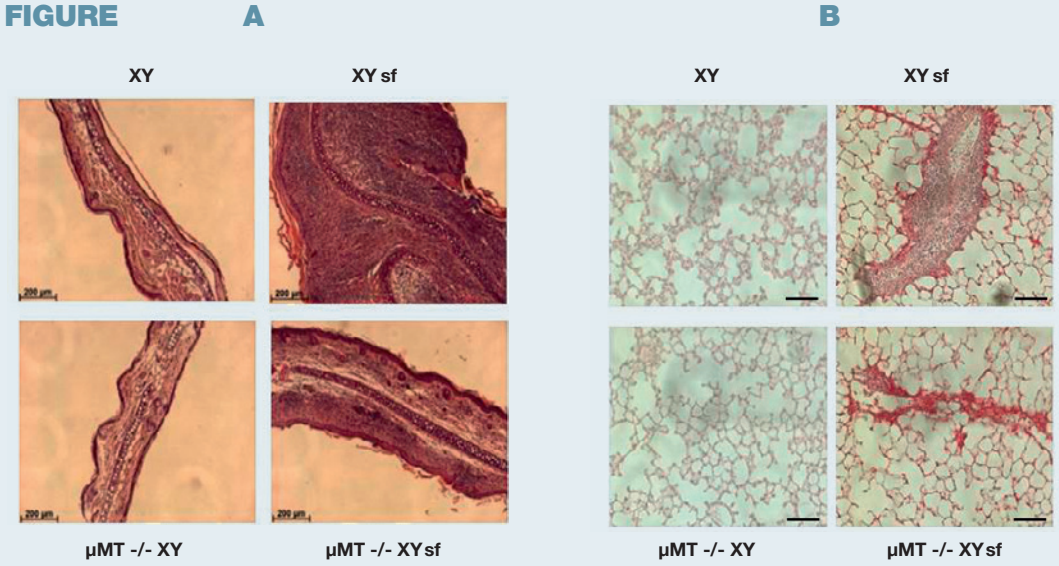


Figure B cells are critical for skin and lung inflammation in Scurfy mice Depicted are skin (**A**) and lung (**B**) tissue sections from C57BL/6 (**XY**), Scurfy (**XYsf**) and B cell deficient littermates (**µMT-/-**).

SCIENTIFIC HIGHLIGHTS

Prediction of response to biological therapy

In vivo molecular imaging using fluorescent anti-TNF antibodies predicts response to biological therapy in Crohn's disease

RAJA ATREYA DEPARTMENT OF MEDICINE 1, UNIVERSITÄTSKLINIKUM ERLANGEN

Anti-TNF antibodies have proven clinical efficacy in the treatment of Crohn's disease, but only a subgroup of patients responds to this therapy. A method to predict therapeutic response is much needed. Current data indicate that anti-TNF antibodies mediate their therapeutic effects via membrane-bound TNF (mTNF) expressing mucosal cells in Crohn's disease. We hypothesized that *in vivo* detection of such cells via fluorescent anti-TNF antibodies might be used to predict therapeutic efficacy.

Aim of our clinical study was to visualize mucosal mTNF expression *in vivo* using confocal laser endomicroscopy (CLE) with topical application of a Good Manufacturing Practice-conform fluorescent anti-TNF antibody to predict treatment outcome. 25 Crohn's disease patients with indication for anti-TNF therapy were prospectively included in this study. The fluorescent anti-TNF antibody was topically applied via a spraying catheter onto the inflamed mucosa of the patients during a colonoscopy prior to initiation of the anti-TNF therapy. Fluorescein expression on a cellular level, indicating mucosal mTNF+ cells, was identified and quantified via CLE. Patients with high amounts of mTNF+ cells showed significantly higher short-term response rates at week 12 (92%) upon subsequent anti-TNF therapy as compared to patients with low amounts of mTNF+ cells (15%). The mean number of mTNF+ cells per image in both groups of patients was significantly different (30 for anti-TNF responders and 11 for non-responders). The clinical response in the former patients was sustained over a follow-up period of one year and associated with mucosal healing on follow-up endoscopy. Sensitivity to predict the response was 92%, specificity 85% with an accuracy of 88%. These data indicate for the first time that molecular imaging with fluorescent antibodies *in vivo* has the potential to predict therapeutic responses to biological treatment and opens new avenues for personalized medicine.

R. Atreya · H. Neumann · C. Neufert · M.J. Waldner · U. Billmeier · Y. Zopf · M. Willma · C. App · T. Münster · H. Kessler · S. Maas · B. Gebhardt · R. Heimke-Brinck · E. Reuter · F. Dörje · T.T. Rau · W. Uter · T.D. Wang · R. Kiesslich · M. Vieth · E. Hannappel · M.F. Neurath. *In vivo* imaging using fluorescent antibodies to tumor necrosis factor predicts therapeutic response in Crohn's disease. *Nature Medicine.* 2014 Mar;20(3):313–8.

FIGURE

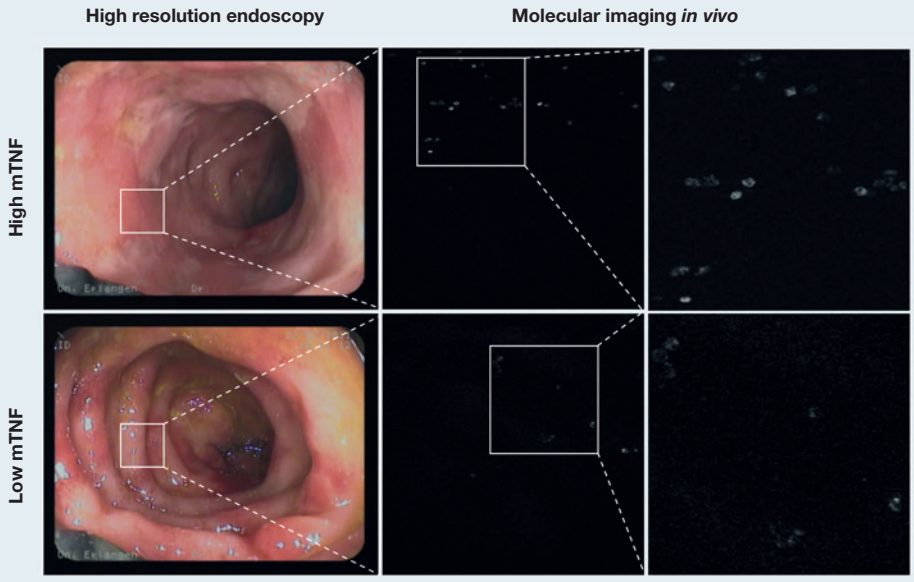


Figure: *In vivo* molecular imaging of intestinal mTNF-positive cells with fluorescent anti-TNF antibodies High-resolution endoscopic images of the inflamed mucosa of Crohn's disease patients. In spite of similar levels of endoscopic mucosal inflammation, molecular *in vivo* imaging with a fluorescent anti-TNF antibody revealed low (lower panel) and high (higher panel) numbers of mTNF-expressing immune cells in these patients (Figure is taken from the original article published in *Nature Medicine*)

SCIENTIFIC HIGHLIGHTS

Induction of antitumor responses by antigen targeting *in vivo*

Antigen Delivery to CD11c⁺CD8⁻ Dendritic Cells Induces Protective Immune Responses against Experimental Melanoma in Mice In Vivo

DIANA DUDZIAK DEPTARTMENT OF DERMATOLOGY, UNIVERSITÄTSKLINIKUM ERLANGEN

... antigen delivery to CD11c⁺CD8⁻ DCs induced a much less efficient CD8⁺ cytotoxic T cell response as opposed to CD11c⁺CD8⁺ DCs...

... unexpectedly, in the melanoma model, both CD11c⁺CD8⁺ and CD11c⁺CD8⁻ DCs induced protective as well as therapeutic immune responses ...

Dendritic cells (DCs) are key players for the induction of immune responses but also for the maintenance of peripheral tolerance. Therefore, DCs might be ideal target cells for immunotherapeutic approaches to improve (tumor) or dampen immune responses (autoimmune diseases). In the past, we have generated antibodies, which efficiently bind to uniquely expressed endocytic receptors on murine DC subpopulations (DEC205, DCIR2). By genetic modifications, these antibodies carry an antigen of choice and cannot bind to Fc receptors. Upon injection and binding to their endocytic receptors, the antigen targeting antibodies are internalized, and the antigens are processed and presented as peptide MHC complexes, thereby allowing an efficient activation of antigen specific T cells.

In a recent study, we delivered antigens to CD11c⁺CD8⁻ or CD11c⁺CD8⁺ DCs in the presence or absence of an immune stimulus in naïve animals. We found that antigen delivery to CD11c⁺CD8⁺ DCs induced a pronounced CD8+ cytotoxic response as well as CD4⁺ T cell responses. In contrast, the delivery of antigens to CD11c⁺CD8⁻ DCs was less efficient in inducing CD8⁺ cytotoxic T cell responses, but showed similar CD4⁺ helper T cell responses. The measured antibody titers against the engulfed antigen suggested that a mixed TH1/TH2 immune response was induced, which was independent of the DC subset that originally presented the antigen.

Next, we analyzed whether the induced immune responses would be protective for the outgrowth of melanoma cells. To this end, we used the B16F10 melanoma model, in which ovalbumin (OVA) was a surrogate antigen. We found that it did not matter, which of the DC subsets presented the antigen, as both CD11c⁺CD8⁺ and CD11c⁺CD8⁻ DCs induced protective as well as therapeutic immune responses. This finding was unexpected as CD11c⁺CD8⁻ DCs were less efficient inducers of cytotoxic CD8⁺ Tcells which usually are critical for the fighting against tumor cells .

This work was partly supported by the German Research Foundation, BayGene, ELAN and the Ria-Freifrau von Fritsch Stiftung.

K. Neubert · C.H. Lehmann · L. Heger · A. Baranska · A.M. Staedtler · V.R. Buchholz · S.Yamazaki · G.F. Heidkamp · N. Eissing · H. Zebroski · M.C. Nussenzweig · F. Nimmerjahn · D. Dudziak. 2014. Antigen delivery to CD11c⁺CD8⁻ dendritic cells induces protective immune responses against experimental melanoma in mice in vivo. *The Journal of Immunology* 192:5830-5838.

FIGURE 1

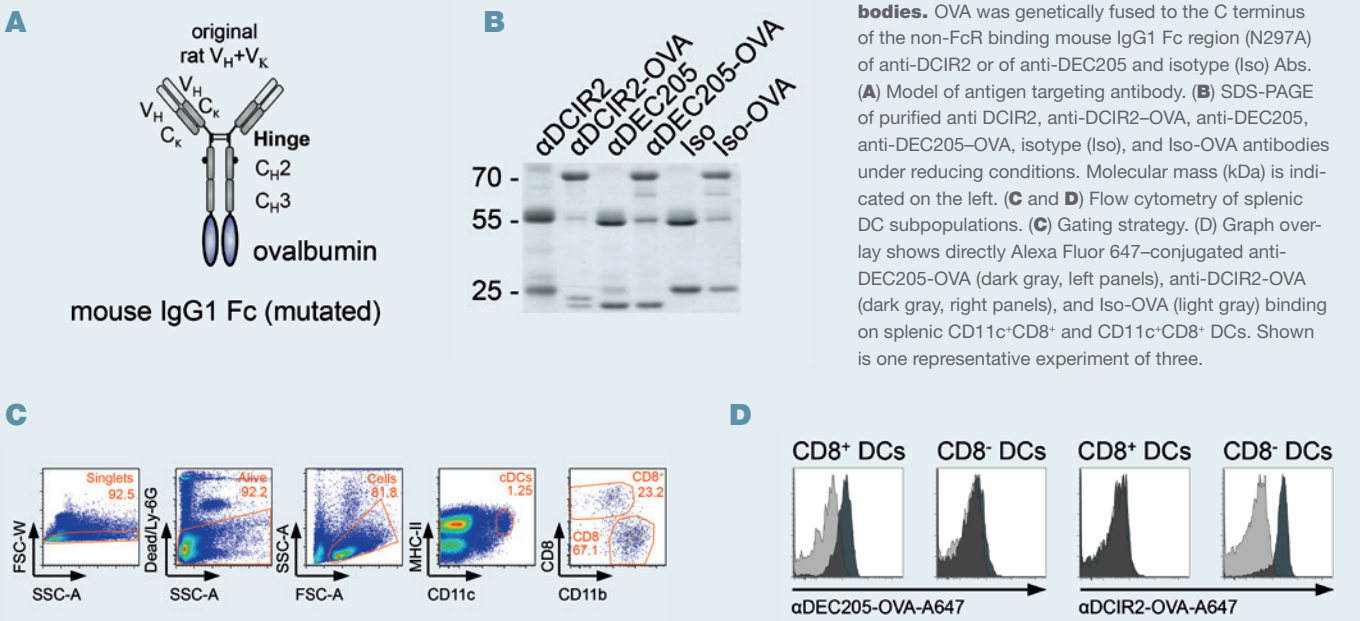


Figure 1: Production of antigen targeting antibodies. OVA was genetically fused to the C terminus of the non-FcR binding mouse IgG1 Fc region (N297A) of anti-DCIR2 or of anti-DEC205 and isotype (Iso) Abs. (**A**) Model of antigen targeting antibody. (**B**) SDS-PAGE of purified anti DCIR2, anti-DCIR2–OVA, anti-DEC205, anti-DEC205–OVA, isotype (Iso), and Iso-OVA antibodies under reducing conditions. Molecular mass (kDa) is indicated on the left. (**C** and **D**) Flow cytometry of splenic DC subpopulations. (**C**) Gating strategy. (D) Graph overlay shows directly Alexa Fluor 647–conjugated anti-DEC205-OVA (dark gray, left panels), anti-DCIR2-OVA (dark gray, right panels), and Iso-OVA (light gray) binding on splenic $CD11c^+CD8^+$ and $CD11c^+CD8^+$ DCs. Shown is one representative experiment of three.

FIGURE 2

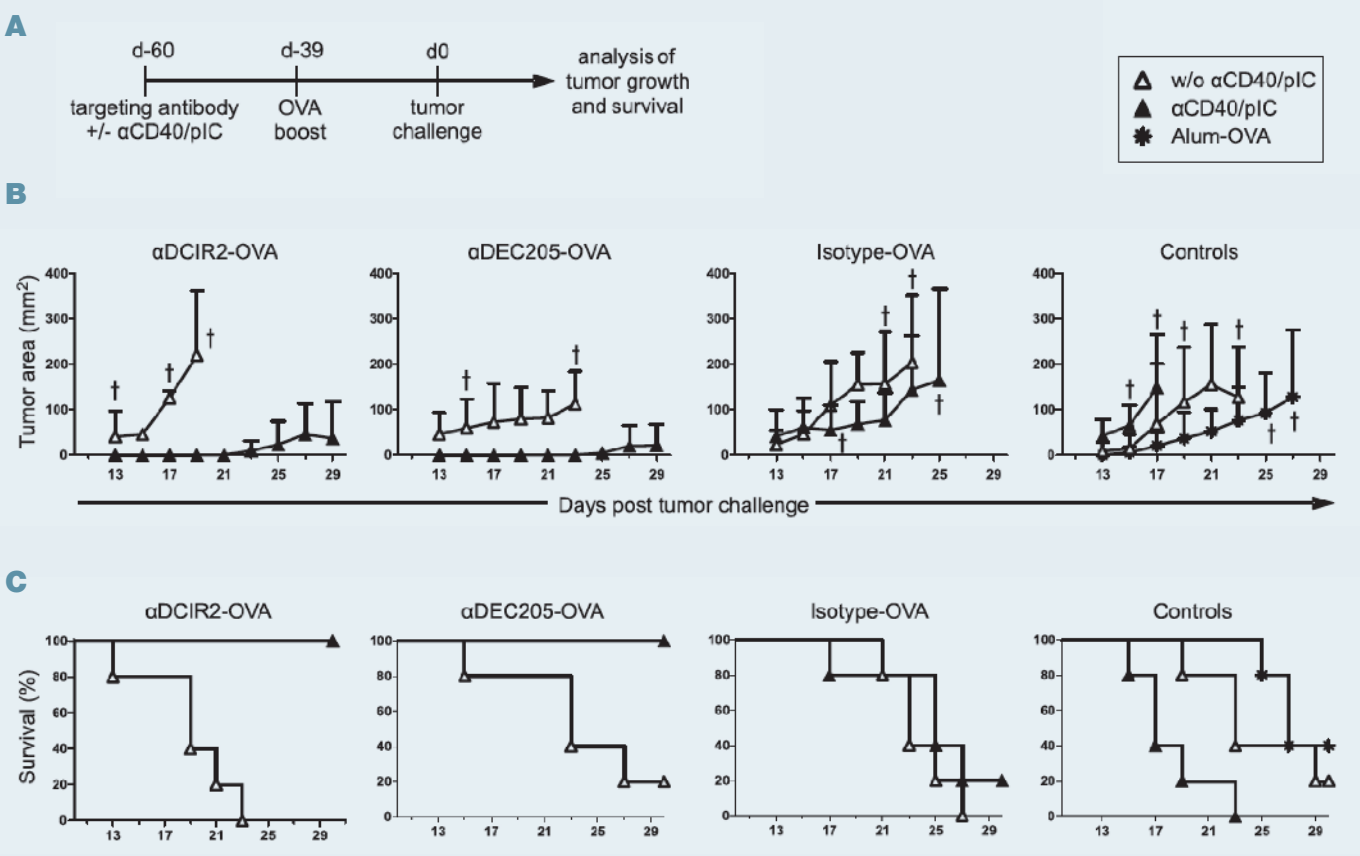


Figure 2: Preventive antigen targeting to $CD11c^+CD8^+$ and $CD11c^+CD8^-$ DCs reduces tumor cell growth and prolongs survival. Sixty days prior to tumor cell application, five C57BL/6 mice each were injected i.p. with 1 µg of anti-DCIR2-OVA, anti-DEC205-OVA, Iso-OVA, or PBS in the presence (filled) or absence (open) of the maturation stimuli 50 µg anti-CD40 Ab and 50 µg poly(I:C) (anti-CD40/pIC) or Alum-OVA (*). On day -39 (21 d after immunization), mice were boosted with 10 µg endotoxin-free soluble OVA protein and challenged with 2×10^5 B16F10-OVA cells on day 0. (**A**) Experimental setup. (**B**) Mean tumor sizes. Lines were discontinued when >50% of the mice needed to be sacrificed because of high tumor burden or died (†). Data are mean + SD. (**C**) Survival shown as Kaplan–Meier plots for five mice/group.

NEWS AND UPDATES

A new cellular immunotherapy tested for the first time in patients

Enhancing the humoral immune response after allogeneic stem cell transplantation

THOMAS WINKLER[1], MICHAEL MACH[2], JULIA WINKLER[3]

[1] CHAIR OF GENETICS, DEPARTMENT OF BIOLOGY, NIKOLAUS-FIEBIGER-ZENTRUM, FAU

[2] VIROLOGY INSTITUTE, UNIVERSITÄTSKLINIKUM ERLANGEN

[3] DEPARTMENT OF MEDICINE 5, UNIVERSITÄTSKLINIKUM ERLANGEN

The reconstitution of a functioning immune system after allogeneic stem cell transplantation takes months to years. Particularly memory B-lymphocytes reconstitute poorly with the current conditioning regimes. During the period of intense immune suppression, the patients are extremely susceptible to bacterial, fungal and, most importantly, viral infections. The adoptive transfer of B-lymphocytes from the stem-cell donor might significantly enhance humoral immunity for the patient.

In a cooperation of the groups of Michael Mach (Virology) and Thomas Winkler (Genetics) this concept of adoptive transfer of memory B-lymphocytes was evaluated in animal models in the context of infection and reactivation of cytomegalovirus within the DFG Sonderforschungsbereich 643 “Strategies of cellular immune intervention”. We have previously shown that adoptively transferred memory B-lymphocytes can protect immunodeficient mice from the lethal infection with the murine cytomegalovirus. In a collaboration with the Department of Medicine 5 (Andreas Mackensen and Julia Winkler) we went on to establish a large-scale protocol for the generation of human B-lymphocytes in the GMP-facility of the Department of Transfusion Medicine in Erlangen. This project was funded by the Bavarian Immunotherapy Network (BayImmuNet). After the development of a protocol for a phase I/IIa clinical trial for a first-in-man immunotherapy in patients after allogeneic stem cell transplantation, we obtained the permission by the Paul-Ehrlich-Institute and the local ethics committee in 2013.

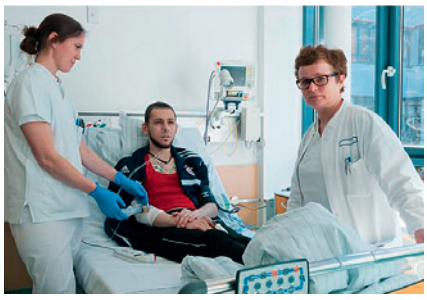
The aim of the study is to evaluate a new cellular therapy with B-lymphocytes regarding safety. A booster vaccination after B-lymphocyte transfer will evaluate the functionality of the transferred B-lymphocytes in the patient. We have now treated 2 patients here in Erlangen with this new immunotherapy (Figure 1). Dr. Julia Winkler is the principal investigator in this clinical trial sponsored by the Universitätsklinikum Erlangen. In these patients no adverse events were observed, particularly reactivation of EBV was not observed so far. FACS and ELISpot data 7 days after booster vaccination indicated an antigen-specific response of the memory B cells (Figure 2). In the near future, further patients will receive increasing numbers of B-lymphocytes and patients at the university hospitals of Regensburg, Würzburg and Essen will also be included in this trial. A total of 15 patients will be treated within this clinical trial supported entirely by the DFG (SFB643).

Trial is registered under clinicaltrial.gov: NCT02007811

FIGURE 1

Figure 1: A new cellular immunotherapy for the first time tested in patients in Erlangen

GMP-qualified B-lymphocytes are prepared from leukapheresis material from the original stem cell donor in a clean room of the Department for Transfusion Medicine in Erlangen.



The first patient included in the study receives the B-lymphocyte product at the Medical Department 5. Dr. Julia Winkler (right) is the principle investigator in this clinical trial

FIGURE 2

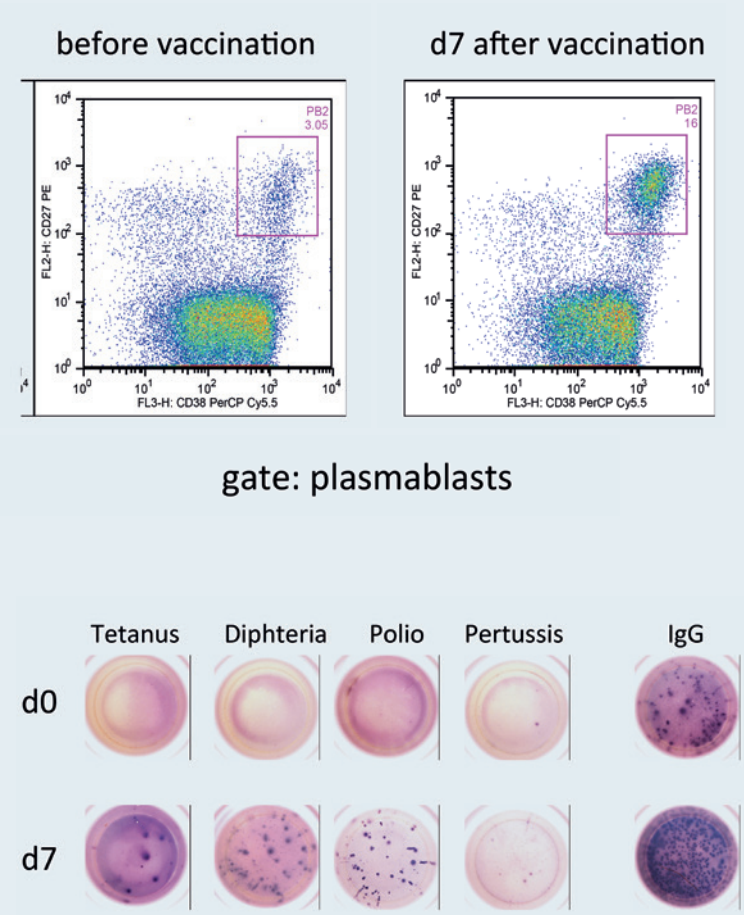


Figure 2: B-lymphocyte responses in the peripheral blood of the first study patient

Top: FACS analysis of B-lymphocytes isolated from the peripheral blood before and 7 days after booster vaccination. The frequency of antibody-secreting plasmablasts (as depicted with the gate) increased substantially.

Bottom: ELISpot analysis of B-lymphocytes isolated from the peripheral blood before and 7 days after booster vaccination. The frequency of vaccine-specific antibody-secreting cells, undetectable before vaccination, increased substantially for four vaccine antigens.

NEWS AND UPDATES

International Day of Immunology

On April 29, for the 3rd time doctoral students of the DFG research training groups GK1660, GKS-FB643 and IRTG TRR130 invited the public to celebrate the International Day of Immunology.

For this purpose, they organized an exciting and diversified program allowing adults and children to easily gain insight into the fascinating mechanisms with which our body defends itself against numerous pathogens.

Different immunological topics like vaccination, transplantation, allergy or autoimmunity were explained to the interested public. Young explorers had the opportunity to take part in various plays and games and to win attractive prices.

All in all, the Day of Immunology offers a platform where adults get to know the importance of the field of immunology in medicine and young scientists can impart their immunological knowledge and their enthusiasm for this interesting and important research field.

Text: Anna Maurberger

Prof. Nikolaus Fiebiger (Source: FAU)

Obituary for Prof. Nikolaus Fiebiger

The Friedrich-Alexander-Universität Erlangen-Nürnberg mourns the loss of a former president

With the passing of Prof. Nikolaus Fiebiger on April 6, 2014, the FAU lost one of its most distinguished and influential figures. Nikolaus Fiebiger, who lived to be 91 years, was not only longtime president of the FAU (1975 to 1990) but also responsible for many outstanding achievements during the 25-year "era Fiebiger", including the establishment of the technical faculty of the FAU and the recruitment of two Fraunhofer Institutes to Erlangen.

Born on August 7, 1922 in Langseifersdorf (Silesia), Nikolaus Fiebiger, like many young adults during that time, served in the Reich Labour Service after graduating from school. Afterwards he joined the German Airforce and later was held as a prisoner of war. In 1947, he started his studies of Physics at the TH Stuttgart which he interrupted for an employment with the company Bosch and a job at the Max Planck Institute of Physics of the Stratosphere in Hechingen. To finish his diploma and to pursue his doctoral degree, he returned to the TH Stuttgart, where he completed his PhD in 1957. After spending time as a researcher in the USA including two years at the renowned Brookhaven National Laboratory, he received his professorship in 1963 in Frankfurt/Main.

Prof. Fiebiger came to Erlangen in 1966 as Head of the Department for Experimental Physics and soon became rector (1969 – 1972) and prorector (1972/73) of the FAU and two years later was elected president which he remained until his retirement in 1990.

For his extraordinary achievements Nikolaus Fiebiger has been highly awarded on several occasions. In 1972, he received the Bavarian Order of Merit and in 1990 the Great Federal Cross of Merit. Also in 1990, the city of Erlangen declared him an honorary citizen. He also held the honors of the State Medal for Special Services for the Bavarian Economy, The European Medal of the State of Bavaria, the Helmut-Volz Medal and the Fraunhofer Medal.

To value his enthusiasm and commitment to promote medical research in Erlangen, in 2001, the Nikolaus-Fiebiger-Zentrum für Molekulare Medizin was named in his honor. This institute houses several research groups associated with the Medical Immunology Campus Erlangen.

Our sincerest condolences go out to his family. Nikolaus Fiebiger was a magnificent person who will be dearly missed.

PEOPLE

Foto: W. Rössler, Virologisches Institut

70th birthday of Prof. Dr. med. Bernhard Fleckenstein

We hereby send our warmest congratulations to Prof. Bernhard Fleckenstein, founder and former spokesperson of the *Medical Immunology Campus Erlangen*, who celebrated his 70th birthday on August 10. During his 36 years as director of the Institute of Molecular and Clinical Virology, Universitätsklinikum Erlangen, Bernhard Fleckenstein's unparalleled leadership and dedication has brought forward many successful scientists and outstanding scientific results in the fields of virology and immunology. Happy Birthday from all your friends and colleagues here at the Medical Immunology Campus Erlangen!

Prof. Jonathan Jantsch (Source: private)

Prof. Dr. med. Jonathan Jantsch

W2 professorship at the University of Regensburg

Jonathan Jantsch, former research group leader at the Institute of Clinical Microbiology, Immunology and Hygiene at the Universitätsklinikum Erlangen, was appointed as W2 professor at the University of Regensburg as of June 1, 2014. Jonathan Jantsch obtained his M.D. at the FAU in 2005 and was awarded the thesis award of the medical faculty (Staedtler-Promotionspreis). Between the years of 2005 and 2008, he received clinical training at the Department of Internal Medicine 4 and started to study the influence of microenvironmental factors (hypoxia and tonicity) on the immune response. In summer 2008, he joined the Microbiology Institute to continue his research work in infectious disease models and to complete a training in clinical microbiology. Prof. Jantsch was awarded the Young Investigator Award of the German Society for Microbiology and Hygiene in 2012.

The Microbiology Institute thanks Jonathan Jantsch for the past six years, during which he has not only done excellent research but has been a great colleague and friend to many coworkers. We sincerely wish Jonathan Jantsch the best of luck for his new position in Regensburg.

UPCOMING EVENTS

Immunological Colloquium of the Medical Immunology Campus Erlangen – Winter 2014/15 Tuesdays, 5.15 pm

14. 10. 2014
Awaiting confirmation of invited speaker

21. 10. 2014
Dr. Dietmar Zehn
Centre hospitalier universitaire vaudois (CHUV) Lausanne, Switzerland
"Title to be announced"

28. 10. 2014
Prof. Heinrich Körner
Menzies Research Institute Tasmania, Australia
"A New Function For An Old Acquaintance? Dissecting the Roles of TNF in Monocyte Differentiation."

04. 11. 2014
Prof. Victor Tybulewicz
MRC National Institute for Medical Research, London, UK
"Signals controlling B cell survival and T cell adhesion"

11.11. 2014
Awaiting confirmation of invited speaker

18. 11. 2014
Prof. Florian Greten
Georg-Speyer-Haus, Institut für Tumorbiologie und experimentelle Therapie
"Title to be announced"

25. 11. 2014
Prof.Burkhard Becher
University of Zurich, Institute of Experimental Immunology, Switzerland
"Title to be announced"

02. 12. 2014
Prof. Alexander Rösch
Universitätsklinikum Essen
"Title to be announced"

09. 12. 2014
Prof. Salomé LeibundGut-Landmann
Institute of Microbiology, ETH Zürich, Switzerland
"Interleukin-17-mediated host defense against fungal infection"

Joachim Kalden Lecture 2014

16. 12. 2014
Prof. Dontscho Kerjaschki
Clinical Institute of Pathology, Medical University Vienna, Austria
"Title to be announced"

13. 01. 2015
David Sancho, Ph.D.
CNIC- Fundación Centro Nacional de Investigaciones Cardiovasculares "Carlos III", Madrid, Spain
"Title to be announced"

20. 01. 2015
Anne Dudeck, PhD
Institute of Immunology, Technical University Dresden
"Title to be announced"

27. 01. 2015
Dr. Till Strowig
Helmholtz Center for Infection Research, Braunschweig
"Title to be announced"

23. 02. 2015
Prof. Sankar Ghosh
Columbia University, USA
"Title to be announced"

UPCOMING EVENTS

Further Conferences and Events of Interest

September 14 – 18, 2014
13th International Symposium on Dendritic Cells
Tours, France
www.dc-2014.com

September 17 – 20, 2014
44. Jahrestagung der Deutschen Gesellschaft für Immunologie DGfI 2014
Bonn
www.immunology-conference.de

October 2 – 4, 2014
28th Annual Conference of the European Macrophage and Dendritic Cell Society (EMDS)
Vienna, Austria
https://emds2014.univie.ac.at/home/

October 5 – 10, 2014
6th Autumn School: Current Concepts in Immunology – German Society for Immunology
Merseburg
The Autumn School "Current Concepts in Immunology" is organized by the German Society for Immunology and addresses master, diploma and first-year doctoral students. Comprehensive immunological knowledge will be imparted, and participants will be actively involved during student presentations, meet-the-speaker sessions and interactive discussions. Intense ex-change between lecturers and students will provide the possibility to set up a network of lecturers and students.
www.herbstschule.de

October 5 – 8, 2014
4. Gemeinsame Jahrestagung der Deutschen Gesellschaft für Hygiene und Mikrobiologie (DGHM)
Dresden
www.dghm-vaam-kongress.de

October 6 – 8, 2014
4th World Congress on Virology
San Antonio, USA
http://virology2014.conferenceseries.net/

March 8 – 13, 2015
11th Spring School on Immunology
Ettal
web.dgfi.org/spring-school/?q=spring-school

March 18 – 21, 2015
25th Annual Meeting of the Society for Virology
Bochum
www.virology-meeting.de

September 6 – 9, 2015
4th European Congress of Immunology
Vienna, Austria
www.eci-vienna2015.org

Medical Immunology Campus Erlangen

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

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Please note that the authors are responsible for the content of their contributions.

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

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