Medical Immunology Campus Erlangen

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

Newsletter Fall/Winter 2021
CONTENT

FAU
Friedrich-Alexander-Universität
Erlangen-Nürnberg

EDITORIAL

Dear colleagues and friends,

Earlier this year, two scientific giants, doyens and emeritus professors of the *Medical Immunology Campus Erlangen* passed away: Prof. Dr. med. Dr. h.c. mult. Joachim Kalden, who was director of the Medical Clinic III from 1977 until 2006, and Prof. Dr. med. Bernhard Fleckenstein, who led the Institute of Clinical and Molecular Virology from 1978 until 2015. Both of them have shaped the immunological and infectious disease research landscape in Erlangen for decades. Due to their extraordinary professional commitment, their visions and their impressive leadership qualities, basic, translational and clinical immunology became one of the key research areas at FAU and UKER with high national and international reputation. Jochen and Bernhard have trained and supported generations of students, doctoral candidates, researchers and clinicians. Many of the current members of the *Medical Immunology Campus Erlangen* have profited during their careers from Jochen's and Bernhard's friendship and mentorship. Even after their retirement, they frequently took part in the guest seminars of the Campus. We all will miss their kind attitude, their encouraging attention and their scientific advice.

The COVID-19 vaccination campaign that started between Christmas 2020 and New Year following the rapid development, authorization and recommendation of highly effective vaccines, had initially led to a remarkable change in the course of the SARS-CoV-2 pandemic: fully vaccinated persons showed a drastically reduced risk to develop severe or even lethal COVID-19. Consequently, the number of hospitalizations, admissions to intensive care units and deaths due to COVID-19 went down dramatically, most notably amongst elderly people. Currently, approximately 75 to 80% of the entire population in Germany exhibit SARS-CoV-2-immunity, due to vaccination or natural immunization following infection. However, the remaining 20 to 25% of our population are still unprotected. Furthermore, in elderly and immunocompromised persons it has become clear that protection against SARS-CoV-2 induced by two vaccine doses is waning, or has been insufficient in the first place. In addition, it turned out that unvaccinated people will not only infect each other, but can also acquire the virus from vaccinated contact persons, because the COVID-19 vaccines do not confer sterile immunity. All these factors as well as the premature abolition of the hygiene regulations by the political decision makers during the summer have contributed to the current worrying COVID-19 wave in Germany. I can only appeal to all of you to return to the previously practiced hygiene measurements, to make sure that all vulnerable persons in your families immediately receive the necessary 3rd vaccine dose, to register for your own booster vaccination five to six months after the 2nd dose, and to encourage unvaccinated people to change their mind. As of November 15, students are allowed to return to FAU for attending lectures or practical courses in person, provided they have a »2G« or »3G« status (vaccinated, recovered or tested) and wear an FFP2 mask.

Prof. Christian Bogdan
Chairman of The Medical Immunology Campus Erlangen

SCIENTIFIC HIGHLIGHTS

β2-microglobulin triggers NLRP3 inflammasome activation in tumor-associated macrophages to promote multiple myeloma progression.

β2m-mediated lysosomal damage leads to inflammasome activation

HEIKO BRUNS
DEPARTMENT OF INTERNAL MEDICINE 5, FAU ERLANGEN-NÜRNBERG, UNIVERSITÄTSKLINIKUM ERLANGEN

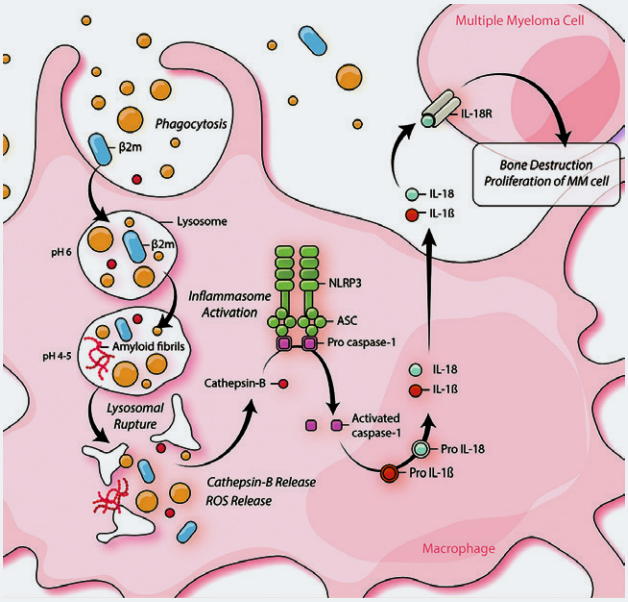
Uncontrolled inflammasome activation by damage-associated molecular patterns (DAMPs) can lead to metabolic pathologies, neurologic disorders and autoimmune disease. Importantly, the NLRP3 inflammasome is also involved in tumorigenesis. However, the endogenous molecular trigger remained to be elucidated.

Multiple myeloma (MM) is an incurable B-cell malignancy characterized by accumulation of malignant plasma cells in the bone marrow (BM), lytic bone lesions and a remarkable ability to manipulate the BM environment. It is widely accepted that dysregulated inflammatory and immunological processes in the tumor microenvironment are not mere bystanders but that invading leukocytes and tumor-associated macrophages (TAMs) are central for the initiation and progression of MM. Consequently, the identification of endogenous mediators of these inflammatory processes can open novel therapeutic avenues against major pathological features of MM.

In our work, we discovered that beta-2-microglobulin (β2m) is an important driver in the initiation of inflammation in TAMs. While under physiological conditions β2m is generated at a constant rate, elevated β2m serum levels are observed in a range of autoimmune, renal and hematological diseases. Accumulation of phagocytosed β2m in the lysosomes of TAMs resulted in β2m amyloid aggregation causing lysosome rupture. This activated the NLRP3 inflammasome and ultimately resulted in the production of active interleukin (IL)-1β and IL-18 in MM patients. Interestingly, macrophages from mice deficient in NLRP3 exhibited impaired β2m-induced IL-1β and IL-18 production. Accordingly, depletion or silencing of β2m in MM cells abrogated inflammasome activation in a murine MM model. Most importantly, β2m-induced tumor growth and osteolytic bone destruction were strongly attenuated after specific ablation of NLRP3 or IL-18.

Our findings provide insight into the molecular processes underlying the inflammatory conditions of MM and suggests that therapeutic inhibition of the NLRP3 inflammasome might reduce the severity of myeloma disease.

Hofbauer D., Mougiakakos D., Broggini L., Zaiss M., Büttner-Herold M., Bach C., Spriewald B., Neumann F., Bisht S., Nolting J., Zeiser R., Hamarsheh S., Eberhardt M., Vera J., Visentin C., De Luca C.M.G., Moda F., Haskamp S., Flamann C., Böttcher M., Bitterer K., Völkl S., Mackensen A., Ricagno S., Bruns H. Immunity. 2021 Aug 10;54(8):1772–1787.



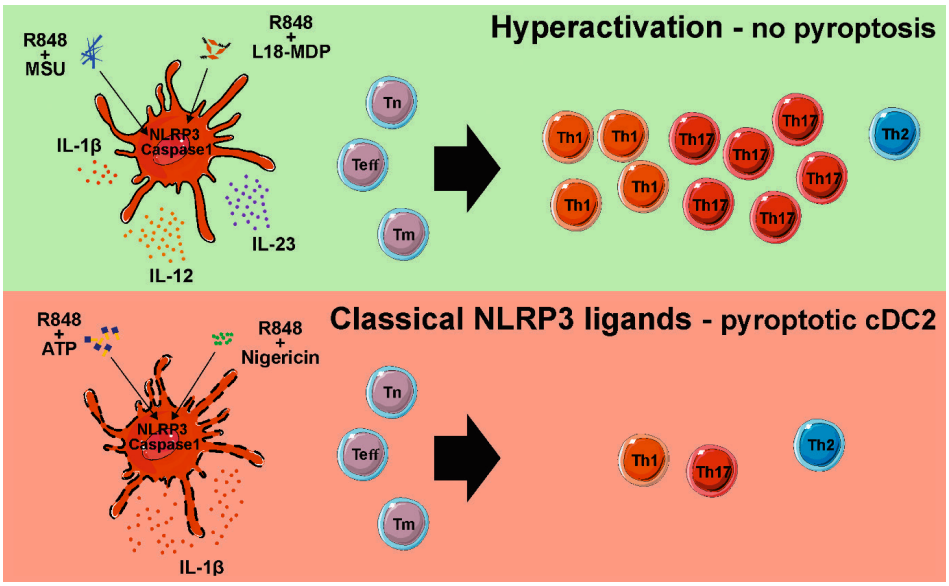
Accumulation of phagocytosed β2m in the lysosomes of TAMs resulted in β2m amyloid aggregation and subsequent lysosome rupture. This led to the activation of the NLRP3 inflammasome and ultimately resulted in the production of active IL-1β and IL-18 in MM patients, which in turn promoted progression and severity of disease in a MM mouse model.

SCIENTIFIC HIGHLIGHTS

Human conventional dendritic cells type 2 (cDC2) can activate the NLRP3 inflammasome without induction of pyroptosis

Select NLRP3 ligands induce a hyperactivated state in human cDC2

LUKAS HEGER, LUKAS HATSCHER, DIANA DUDZIAK
DEPARTMENT OF DERMATOLOGY FAU ERLANGEN-NÜRNBERG, UNIVERSITÄTSKLINIKUM ERLANGEN



Concept of hyperactivation vs. pyroptosis in the regulation of T cell responses. Ligands that weakly activated the NLRP3 inflammasome in human cDC2 (upper panel), such as L18-MDP and MSU, induced simultaneous secretion of IL-1β and IL-12 family members. Thereby, cDC2 induced stronger T cell responses with a Th1/Th17 phenotype. In contrast, ligands strongly activating the NLRP3 inflammasome, such as ATP and Nigericin, induced mainly IL-1β and led to cell death of the cDC2. Thereby, T cell proliferation induced by cDC2 was decreased.

Activation of the inflammasome is typically accompanied by induction of pyroptosis - a programmed form of inflammatory cell death. Here, formation of inflammasomes leads to activation of caspase-1, which cleaves, in addition to pro-IL-1β and -IL-18, gasdermin D. Subsequently, this results in pore formation, the release of mature IL-1β and IL-18 and in cell death. As the main role of dendritic cells (DCs) is the initiation and regulation of immune responses, it is counter-intuitive that DCs undergo pyroptosis after engulfment of pathogens that frequently express inflammasome ligands. Further, it was still under debate whether bona fide DCs are able to form functional inflammasomes or whether this is restricted to the monocyte lineage.

In our present study, we provided evidence that bona fide human cDC2 were able to react to stimulation with NLRP3 ligands, while the other DC subsets, cDC1 and plasmacytoid DCs, were rather inert to most tested inflammasome ligands. We further showed that cDC2s did not undergo pyroptosis in response to rather weak NLRP3 ligands such as L18-MDP or monosodium urate (MSU), while strong damage-associated molecular patterns such as ATP induced pyroptosis. In line with that, hyperactive cDC2 simultaneously secreted IL-1β in combination with IL-12 family cytokines and other inflammatory cytokines, while cDC2 undergoing pyroptosis were unable to secrete cytokines other than IL-1β. Thus, exposure to cDC2 to weak NLRP3 ligands set them into a hyperactivated state and resulted in an enhanced capacity to induce Th1/Th17 responses.
As inflammasomes are crucial players in several chronic inflammatory diseases, hyperactive cDC2 might be involved in the pathogenesis of these diseases, making cDC2 potential targets for treatment. Moreover, induction of hyperactivation in DCs could be beneficial for the therapy of tumors as it might break tolerance induced in DCs by the tumor microenvironment.

Hatscher L., Lehmann C.H.K., Purbojo A., Onderka C., Liang C., Hartmann A., Cesnjevar R., Bruns H., Gross O., Nimmerjahn F., Ivanović-Burmazović I., Kunz M., Heger L., Dudziak D. 2021 Apr 27;14(680):eabe1757.

SCIENTIFIC HIGHLIGHTS

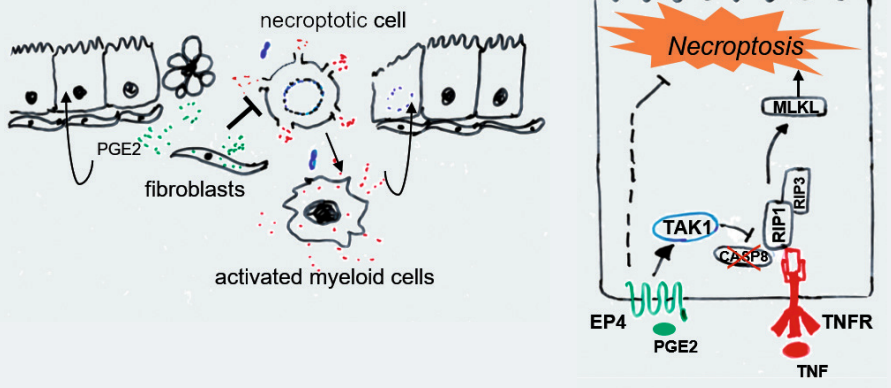
E-type prostanoid receptor 4 drives resolution of intestinal inflammation by blocking epithelial necroptosis

E-type prostanoid receptor 4 blocks epithelial necroptosis

JAY V. PATANKAR
DEPARTMENT OF MEDICINE 1, FAU ERLANGEN-NÜRNBERG, UNIVERSITÄTSKLINIKUM ERLANGEN

Epithelial cells lining the intestine are short-lived and metabolically agile cells that perform crucial innate immune functions forming a tight protective barrier. In patients suffering from inflammatory bowel disease (IBD) and in experimental models of colitis, these epithelial cells die in various ways leading to a breach of the barrier. One mode of cell death termed ›necroptosis‹ is triggered by the cytokine TNF and causes cells to spill out their intracellular contents into the tissue. This initiates proinflammatory changes, which will fuel the chronicity of inflammation in organs that are constantly exposed to microbial and environmental challenges, thereby creating a vicious cycle. Although recent research efforts have uncovered the mechanistic components that orchestrate necroptotic cell death, little is known about the endogenous signaling molecules, which halt this process under healthy conditions and allow for resolution of inflammation. In our recent report, we uncovered that the activation of the epithelial E-type prostanoid receptor 4 (EP4) via its endogenous ligand PGE2 can block epithelial necroptosis in mouse and patient-derived intestinal 3D organoids. Together with our Canadian partners, we were able to show that a novel selective agonist for the EP4 receptor, EP4-D, blocks the progression of colitis and reduces the number of necroptotic cells *in vivo*. Interestingly, treatment of mice, which had developed colitis, with EP4-D led to a resolution-like transcriptomic alteration in their colon tissues. Finally, IBD patients with an elevated expression of the PTGER4 gene, which encodes the EP4 receptor and is a known risk gene for Crohn's disease, showed improved flare-free survival. Mechanistically, we identified TAK1 as a signaling node, which is common to TNFR signaling and EP4 receptor signaling through which EP4 suppressed the activation of the RIP1, an upstream factor that controls necroptotic death. Our data show that during barrier breach, prostanoids derived from first responders and from dying cells can act in a paracrine manner on neighboring epithelial cells to protect them from the consequences of proinflammatory cytokines such as TNF. In the next phase of this project, we aim to investigate how this endogenous mechanism fails upon chronification in IBD patients.

Patankar J.V., Müller T.M., Kantham S., Acera M.G., Mascia F., Scheibe K., Mahapatro M., Heichler C., Yu Y., Li W., Ruder B., Günther C., Leppkes M., Mathew M.J., Wirtz S., Neufert C., Kühl A.A., Paquette J., Jacobson K., Atreya R., Zundler S., Neurath M.F., Young R.N., Becker C. Nat Cell Biol. 2021 Jul;23(7):796–807.



Activation of the EP4 receptor blocks intestinal epithelial cell (IEC) necroptosis via TAK1 dependent and independent mechanisms. (**A**) Ectopic death of IECs causes barrier breach and invasion of microbes (blue doublets) into the lamina propria activating myeloid cells, which produce proinflammatory factors such as TNF. TNF triggers further necroptosis of susceptible IECs causing more proinflammatory activation of immune cells driving IEC necroptosis (vicious cycle), which is inhibited by PGE2 derived from activated fibroblasts (**B**) Signaling cascades initiated at the TNFR trigger necroptosis under specific conditions. PGE2 drives EP4 signaling, which converges on TAK1, repressing RIP1 phosphorylation and therefore inhibits the activation of MLKL, the terminal executioner of necroptosis.

PEOPLE

Prof. Dr. med. Christian Bogdan

Prof. Dr. med. Christian Bogdan is the spokesman of the new Research Training Group 2740 »ImmunoMicroTope« that was approved by the DFG in May 2021

Prof. Dr. med. Christian Bogdan

The German Research Foundation has approved the new Research Training Group (RTG) 2740 »ImmunoMicroTope – Microenvironmental, Metabolic and Microbial Signals Regulating Immune Cell-Pathogen Interactions" for an initial period of four and a half years with a total of 6.1 million €. From January 1, 2022, onwards, 14 new doctoral students will be trained in eleven highly innovative scientific projects. The research consortium aims to investigate host- or microbe-derived factors that shape the microenvironment in the infected tissue and thereby regulate the function of immune cells and/or the persistance of non-viral infectious agents like bacteria, fungi or parasites. Spokesman of the RTG 2740 is Prof. Dr. med. Christian Bogdan, Director at the Institute for Clinical Microbiology, Immunology and Hygiene, University Hospital Erlangen.

Prof. Dr. rer. nat. Diana Dudziak

Paul Langerhans Award for Prof. Dr. rer. nat. Diana Dudziak

Our sincere congratulations go to Professor Diana Dudziak who received this year's Paul Langerhans Award of the Arbeitsgemeinschaft Dermatologische Forschung e.V. (ADF e.V.) endowed with 15.000 € for her work on novel dendritic cell-based tumor therapies. The Paul Langerhans Award has been awarded annually since 2003 to highly qualified dermatological scientists in Germany, Austria or Switzerland who are in the middle of their academic career and who have continuously performed excellent and innovative research over the past years.

Prof. Dr. rer. nat. Diana Dudziak has worked at the Helmholtz Zentrum München – German Research Center for Environment and Health- and at Rockefeller University in New York. Since March 2008 she leads her own independent research group »Biology of dendritic cells« at the Department of Dermatology at the University hospital Erlangen.

Prof. Dr. rer. nat. Diana Dudziak

PEOPLE

Source: Pressestelle des UKER

The foundation of our **Medical Immunology Campus Erlangen** *in 2009 goes back to an idea of Bernhard during the preparation of an FAU excellence cluster application.*

Obituary for Prof. Dr. med. Bernhard Fleckenstein (1944 – 2021)

The Friedrich-Alexander Universität at Erlangen-Nürnberg and the University Hospital Erlangen mourns the death of the former Chair of the Institute of Clinical and Molecular Virology.

On May 4, 2021, the FAU and University Hospital Erlangen has lost an exceptional personality who dedicated his academic career to oncogenic herpesviruses and other tumor viruses, the promotion of the field of virology, and the training and support of young scientists: Prof. Dr. med. Bernhard Fleckenstein.

Born in Würzburg in 1944, Bernhard studied medicine at the universities of Freiburg/Breisgau and Vienna (1963–1969). After passing his state examinations in medicine in Freiburg in 1969, he worked as a medical assistant at hospitals in Karlsruhe and Lübeck. In 1970, he was awarded his M.D. degree in the field of physiology at the University of Freiburg. Therafter, he became scientific assistant at the Institute for Hygiene and Microbiology at the University of Göttingen. During this time, he started his virological research and developed great enthusiasm for tumor viruses, notably the newly discovered rhadinoviruses, a subgroup of the gammaherpesvirinae. In 1972, Bernhard joined the group of Professor Harald zur Hausen at the Institute of Clinical Virology at FAU, where he received his habilitation degree in virology in 1975 and continued to work as a Privatdozent until 1976. From 1976 until 1978, he was Associate Professor of Microbiology and Molecular Genetics at Harvard Medical School in Boston and headed the Department of Microbiology at the New England Regional Primate Research Center, where he discovered the tumorigenic effect of viral DNA derived from oncogenic primate herpesviruses. In 1978, at the age of only 34 years, Bernhard returned to Erlangen and was appointed as Full Professor for Virology at FAU and Director of the Institute of Clinical and Molecular Virology. He led the institute for 37 years until his retirement in 2015, after having turned down attractive offers from the University of Freiburg and the LMU in Munich. One major scientific achievement of his research group in Erlangen was the immortalization of human T lymphocytes by Herpesvirus saimiri. Later on, he established the Collaborative Research Center 466 »Lymphoproliferation and viral immunodeficiency«, which was funded by the DFG from 1996 until 2007. Between 1997 and 2001 and again between 2005 and 2008, Bernhard served as Dean of the Medical Faculty at FAU. In addition, he was founding president of the German Society for Virology and founding member and first secretary general of the European Society for Virology. He belonged to numerous national and international scientific bodies, including the Academy of Sciences and Literature in Mainz and the National Academy of Sciences Leopoldina. For his achievements, Bernhard received various awards and honors, including the Max Planck Prize (1991), the Aronson Prize of the State of Berlin (1991), the Ludwig Aschoff Prize of the University of Freiburg (2004), the Federal Cross of Merit on Ribbon (2003) and the Bavarian Order of Merit (2006).

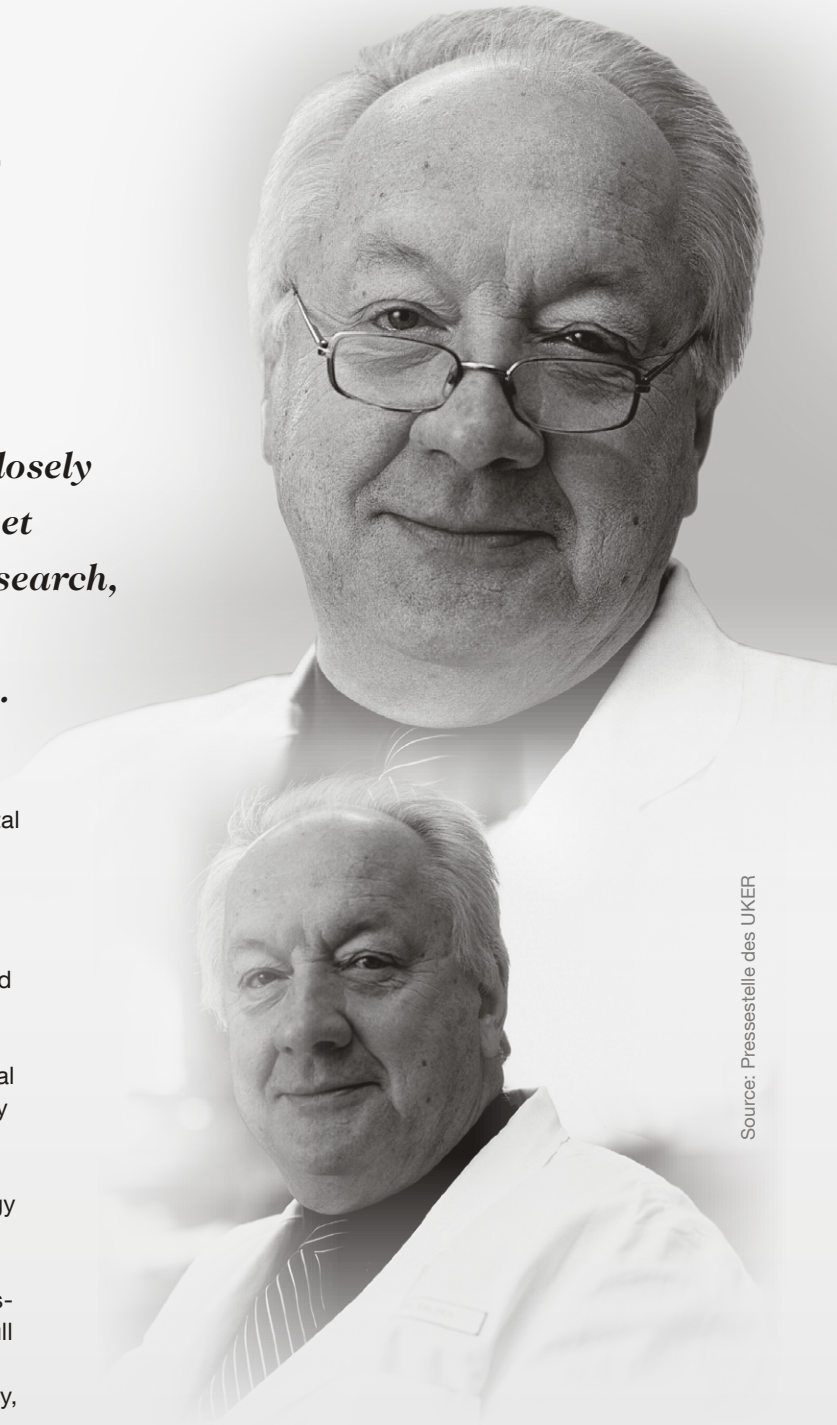
The foundation of our *Medical Immunology Campus Erlangen* in 2009 goes back to an idea of Bernhard during the preparation of an FAU excellence cluster application. After 12 years of existence of the Campus, we are very grateful for Bernhard's initiative to bring together all immunologists in Erlangen, across the different clinical fields and research disciplines, in a virtual center. He will remain in our memories as an outstanding person, whose enthusiasm, charisma, commitment and strategic foresight had a very strong impact on the development of FAU and the University Hospital Erlangen.

PEOPLE

Obituary for Prof. Dr. med. Dr. h.c. mult. Joachim R. Kalden (1937 – 2021)

The Friedrich-Alexander Universität Erlangen-Nürnberg and the University Hospital Erlangen mourns the loss of the former director of the Medical Clinic III for Rheumatology and Clinical Immunology.

Those, who had the opportunity to closely interact with him, will never forget his enthusiasm for immunological research, his liveliness, humor, empathy and his support of young scientists.



Source: Pressestelle des UKER

With the passing of Prof. Dr. med. Dr. h.c. mult. Joachim R. Kalden on Februar 6, 2021, the FAU and the University Hospital Erlangen lost one of their most prominent members and an outstanding physician and teacher who devoted his life to immunology, clinical science and medicine.

Jochen was born 1937 in Marburg. After passing his university entrance exam in Frankenberg/Eder in 1958, he studied medicine in Freiburg/Breisgau, Marburg and Tübingen, where he obtained his doctoral degree for an experimental thesis in allergology in 1966. From 1967 until 1970, he was postdoctoral fellow at the MRC Clinical Endocrinology Unit at the University of Edinburgh. Here, he started his work on the immunopathogenesis of myasthenia gravis, which he later continued as a research assistant in internal medicine and clinical immunology at Hanover Medical School (MHH). After his habilitation and board certificate in internal medicine in 1974, he was senior physician in the Department of Clinical Immunology and Transfusion Medicine at the MHH. In 1977, he was appointed as Full Professor for Clinical Immunology and Rheumatology at FAU and Head of the Institute and Polyclinic of Clinical Immunology, which later became the Medical Clinic III for Rheumatology and Clinical Immunology. Jochen helped to establish two Max Planck Research Groups in Erlangen in 1987 and was spiritus rector and spokesperson of the Collaborative Research Center 263 (»Infection, Inflammation and Autoimmunity«), which was funded by the DFG for 12 years from 1991 until 2002. Jochen was also the initiator of the Interdisciplinary Centre for Clinical Research (IZKF), which started in 1996 following a BMBF funding call, and established the Department of Molecular Immunology in 1997. From 1983 until 1990, he was President of the German Society of Immunology (DGfI) and responsible for the organization of the 7th International Conference of Immunology in Berlin in 1989. From 1993 to 1994, he served as President of the German Society for Rheumatology, and from 2001 until 2003 as President of the European League against Rheumatism (EULAR). His scientific accomplishments are also reflected by honorary doctorates from the Charité Berlin, the Medical Faculty of Lund and the Hanover Medical School as well as by several awards such as the International Prize of the Japan College for Rheumatology in 2005. In 1996, he received the Federal Cross of Merit on Ribbon and in 1999 the Bavarian Order of Merit. Since 1999, he has been a member of the Bavarian Academy of Sciences and Humanities, and in 2002 he became member of the National Academy of Sciences Leopoldina.

After his retirement in 2006, Jochen continued to be a sought-after opinion leader and advisor in clinical immunology and rheumatology. Those in Erlangen, who had the opportunity to closely interact with him over the years, will never forget his enthusiasm for immunological research, his liveliness, humor and empathy, his support of young scientists, the legendary after-seminar dinners with guest speakers in the »Karzer« and other inns in Erlangen, or the commemorable international conferences on »Advances in Targeted Therapy« at pleasant sites around the world. His unique personality as an outstanding scientist, teacher and visionary and as a nestor of clinical immunology and rheumatology in Erlangen and Germany will be dearly missed.

NEWS AND UPDATES

Immunological Colloquium of the Medical Immunology Campus Erlangen – Winter 2021/22

Tuesdays, 5.15 pm

16.11.2021 **12.30 p.m.**
Prof. Veit Rothhammer
Heisenbergprofessur für Neuroimmunologie, Universitätsklinikum Erlangen, FAU
Role of glial cells in autoimmune CNS inflammation

23.11.2021
Prof. Michael Hölzel
Institute of Experimental Oncology, University Hospital Bonn
Modelling and exploring principles of T-cell therapy

30.11.2021
Prof. Dirk Elewaut
VIP-Ugent Center for Inflammation Research, Gent, Belgium
Linking mechanics to inflammation

07.12.2021
Joachim Kalden Lecture 2021
Prof. Ursula Grohmann
Università degli Studi di Perugia, Perugia, Italy
Indoles: very busy (and not indolent) molecules at work in immune regulation

14.12.2021
Prof. Marc Hübner
Institut für Medizinische Mikrobiologie, Immunologie und Parasitologie (IMMIP), Bonn
Eosinophils – essential players in immune defense, pathology and treatment

11.01.2022
Prof. Sarina Ravens
Medizinische Hochschule Hannover, Institut für Immunologie, Hannover
to be announced

18.01.2022
Prof. Martin Kriegel
Universitätsklinikum Münster, Sektion Rheumatologie & Klinische Immunologie, Münster
to be announced

25.01.2022
Prof. Florent Ginhoux
Singapore Immunology Network (SIgN), A(*)STAR; Translational Immunology Institute, SingHealth/ Duke-NUS Academic Medical Centre, Singapore; Shanghai Institute of Immunology, Shanghai Jiao Tong University School of Medicine, Shanghai, China
to be announced

01.02.2022
Prof. Reinaldus Toes
Leiden University, LUMC Main Building, Leiden, Netherlands
to be announced

08.02.2022
Dr. Johannes Schlachetzki
University of California, San Diego, Dept. of Cellular & Molecular Medicine, USA
to be announced

15.02.2022
Prof. Pedro Moura Alves
Ludwig Institute for Cancer Research Ltd, University of Oxford, UK
to be announced

22.02.2022
Prof. Manolis Pasparakis
University of Cologne, CECAD Research Center, Institute for Genetics, Cologne
to be announced

Conferences and Events of Interest

November 15 – 17, 2021
Weimar
Meeting des AK Signaltransduktion

November 22 - 23, 2021
Ghent, Belgium
Translational Immunology

November 23 – 26, 2021
Ibaraki, Japan (Hybrid)
EMBO Workshop: Bacterial membrane vesicles: Biogenesis, functions and medical applications

November 28 – December 1, 2021
Edinburgh, UK
Society for Immunology Congress 2021

January 12 – 14, 2022
Braunschweig (Hybrid)
Meeting des AK Vakzine

January 28 – 29, 2022 · Essen
3rd International Symposium on Tumor-Host Interaction in Head and Neck Cancer and 11th Symposium of the Working Group Oncology

February 16 – 18, 2022
Kassel
21th International AEK Cancer Congress

February 23 – 26, 2022
Berlin
35. Deutscher Krebskongress 2020

March 3 – 5, 2022
Sonthofen
Meeting des AK Biologie der B-Lymphozyten

March 23 – 25, 2022
Schloß Tutzing
Meeting des AK Komplementsystem

March 30 – 31, 2022
Waischenfeld
Meeting des AK Dendritische Zellen

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