Medical Immunology Campus Erlangen

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

Newsletter
Summer 2016

CONTENT

FRIEDRICH-ALEXANDER
UNIVERSITÄT
ERLANGEN-NÜRNBERG

EDITORIAL

Dear colleagues and friends,

A couple of weeks ago one of the key immunological research consortia at the FAU has come to an end: the Collaborative Research Center (CRC) 643 “Strategies of Cellular Immune Intervention”, which had been continuously funded by the German Research Foundation (DFG) for the maximum period of 12 years from July 1, 2004 until June 30, 2016. Considering its tremendous impact on the development of preclinical and clinical immunological research in Erlangen, it is more than appropriate to have a look back and to acknowledge the initiator and the speakers of this CRC.

In April 2001, Gerold Schuler, head of the Department of Dermatology, put forward the idea of a new CRC directed towards the identification of targets and strategies for immune modulation in the areas of infection, inflammation and malignancies. His vision was to establish a consortium with a clear clinical orientation, which aims to transfer research results obtained in the laboratory into clinical application. While clinical immunology already had a long tradition in Erlangen as exemplified by the highly successful earlier CRC263, Gerold was convinced that a true “bench-to-bedside” concept needs to entail more than clinically relevant projects and a mere declaration of future perspectives. Being an expert on dendritic cells, he was inspired by the dream to use these cells for vaccination and immunotherapy. The *in vitro* optimization of the immunogenicity of the cells should be accompanied by clinical trials. Gerold´s ideas fell on fruitful ground in Erlangen. Already in Spring 2002 –, a full CRC application with 16 projects was submitted, and in July 2002 the on-site review took place. Although the reviewers were enthusiastic about the concept and the consortium, the DFG senate initially declined the installation of the CRC based on the usual competitive financial situation and critique on the thematic focus of some projects. However, half a year later the DFG representatives indicated to Gerold that they would be happy to reassess a revised application, which was then approved in May 2004. In the following 12 years of funding, the CRC not only lived up to the expectations, but also succeeded in the initiation of several clinical trials studying the effect of dendritic cells, B cells and T cells.

Prof. Dr. med. Gerold Schuler during the final symposium of the CRC643 on July 16, 2016

On behalf of all former members of the CRC I would like to cordially thank Gerold Schuler as well as the deputy spokesman of the CRC643, Alexander Steinkasserer, for their enthusiasm, commitment and work throughout the entire funding period. It was a pleasure for us to have been part of this consortium, which undoubtedly further catalyzed new research initiatives in Erlangen such as the CRC1181. Special thanks also go to Brigitte Wölfel and Liliana Bodin for their untiring administrative support of the CRC.

I wish you all a relaxing summer break and hope to see you back in October for the new round of our national and international guest seminar series.

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

SCIENTIFIC HIGHLIGHTS

Identification of a central role of NFATc1 in IL-9 mediated allergic asthma

Increased expression of nuclear factor of activated T cells 1 drives IL-9-mediated allergic asthma

SONJA KOCH, NINA SOPEL AND SUSETTA FINOTTO
DEPARTMENT OF MOLECULAR PNEUMOLOGY, FAU ERLANGEN-NÜRNBERG, UNIVERSITÄTSKLINIKUM ERLANGEN

Nuclear factor of activated T cells (NFAT) is a family of transcription factors activated by dephosphorylation mediated by Ca^{++}-activated calcineurin. NFAT coordinates different aspects of lymphocytes and mast cell development and activation. We reported recently that targeted deletion of NFATc1 in T cells ($NFATc1^{fl/fl}xCD4^{Cre}$) resulted in inhibition of TH2 and TH17 differentiation and reduced serum levels of ovalbumin (OVA)-specific IgE, associated with decreased *Batf* expression, a transcription factor essential for immunoglobulin class-switching, that cooperates with the transcription factor interferon regulatory factor 4 (IRF4) at the promoter of different genes relevant for asthma.

In the present study, we found that children from the PreDicta cohort with asthma and with a positive skin test result had significantly increased expression of *NFATC1* and *IRF4* mRNA compared with healthy control subjects from the same study (panel A). These observations were confirmed by analyzing blood cells in the Asthma BRIDGE study. Both NFATc1 and IRF4 also positively influenced IL-9 production of TH9 cells. Consistently, we also found increased IL-9 levels in the PBMCs of the children with allergic asthma (panel A).

The functional relevance of NFATc1 in asthmatic patients was supported by our observations in $NFATc1^{fl/fl}xCD4^{Cre}$ mice, where *Nfatc1/A* mRNA expression perfectly correlated with OVA-specific IgE levels and resulted in downregulation of IL-9 and *Cd40l* in $CD4^+$ T cells (panel B) and mast cell function (panel C). Therefore, targeting NFATc1 in T lymphocytes might ameliorate the allergic phenotype seen in asthmatic patients by reducing IL-9, IgE and mucosal mast cell function.

S. Koch, A. Graser, H. Mirzakhani, T. Zimmermann, VO. Melichar, M. Wölfel, DC. Croteau-Chonka, BA. Raby, ST. Weiss, S. Finotto. 2016. Increased expression of nuclear factor of activated T cells 1 drives IL-9-mediated allergic asthma. J Allergy Clin Immunol.;137(6):1898 – 1902.e7. doi: 10.1016/j.jaci.2015.11.047.

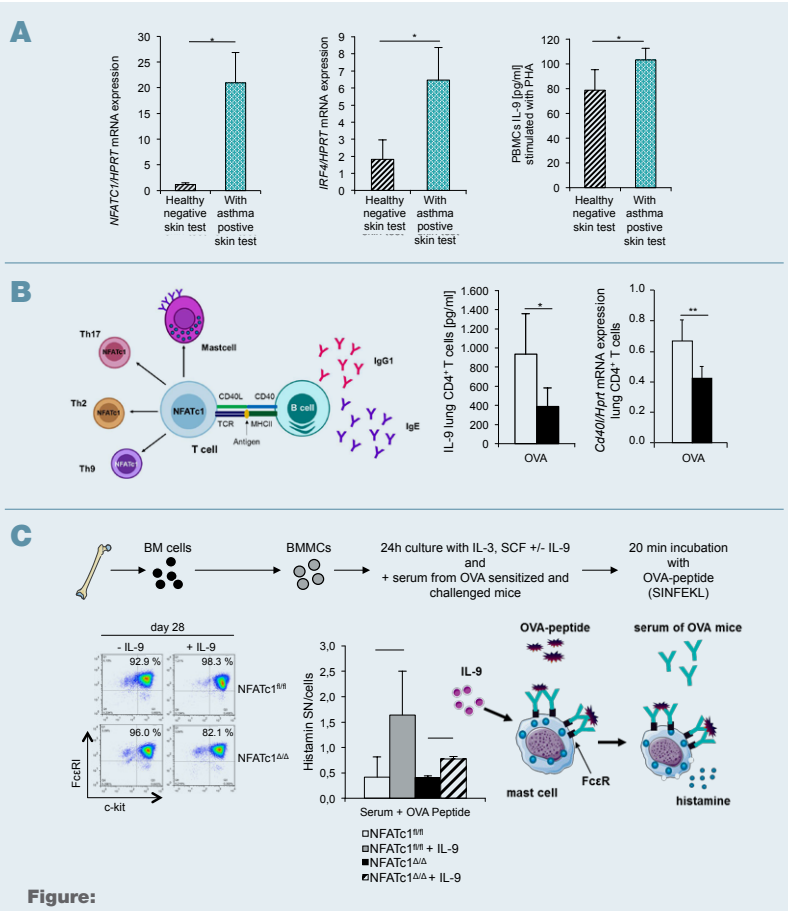


Figure:

A) Increased expression of *NFATC1*, *IRF4* and IL-9 in atopic children with asthma. Children were subdivided into healthy children with negative skin test results and asthmatic children with positive skin test results. mRNA expression was analyzed by using quantitative real-time PCR in RNA isolated from whole blood. Statistical significance was evaluated with the Student t test. *P ≤ .05. Data are presented as means ± SEMs.

B) Cartoon showing the central role of NFATc1 in T helper cell differentiation and the influence on the downstream B cells and mast cells involved in the allergic reaction. Decreased IL-9 protein and *Cd40l* mRNA production in lung $CD4^+$ T cells isolated from $NFATc1^{fl/fl}xCD4^{Cre}$ mice after allergen sensitization and challenge. Statistical significances were evaluated with the Student t test. *P ≤ .05, **P ≤ .01. Data are presented as means ± SEMs.

C) Experimental design for bone marrow–derived mast cell (BMMC) differentiation (upper panel) and histamine release (lower panel). Decreased mast cell numbers and activation in $NFATc1^{fl/fl}xCD4^{Cre}$ mice, as compared to $NFATc1^{fl/fl}$ control mice. Mast cell numbers ($c\text{-}kit^+Fc\varepsilon RI^+CD123^+$ cells) were analyzed by using flow cytometry, histamine release was measured by means of ELISA. Mast cells differentiated from $Nfatc1^{fl/fl}xCD4^{Cre}$ mice received serum from OVA-sensitized and challenged $Nfatc1^{fl/fl}xCD4^{Cre}$ mice, whereas mast cells from $Nfatc1^{fl/fl}$ mice received serum from OVA-treated $Nfatc1^{fl/fl}$ mice. Statistical significances were evaluated with the Student t test. *P ≤ .05. Data are presented as means ± SEMs.

SCIENTIFIC HIGHLIGHTS

Helminth infection inhibits inflammatory arthritis

Th2 and eosinophil responses suppress inflammatory arthritis

ALINE BOZEC
DEPARTMENT OF INTERNAL MEDICINE 3, FAU ERLANGEN-NÜRNBERG, UNIVERSITÄTSKLINIKUM ERLANGEN

Rheumatoid arthritis (RA) is a chronic autoimmune disease characterized by synovial inflammation and bone erosion. Strikingly, this disease hardly resolves and usually handicaps the patients during their entire life. Hence, ineffective resolution is a major clinical challenge.

T helper type 2 (Th2) immune responses are generally known to mediate host defense against helminths. In contrast, not much information is available on the role of Th2 responses during inflammatory diseases such as arthritis.

To study the influence of Th2 immune responses on the course of inflammatory arthritis, we used the *Nippostrongylus brasiliensis* (Nb) infection model, which causes a transient activation of the Th2 pathway, in combination with two mouse arthritis models, the K/BxN serum-induced arthritis and the chronic arthritis hTNF transgenic mice. We observed that Nb infection led to a strong inhibition of inflammation and a protection from bone loss in both arthritis models. This inhibitory effect was due to an activation of Th2 cells in the spleen, an accumulation of eosinophils and a shift from pro- to anti-inflammatory macrophages accompanied by a decreased number of neutrophils in the synovium of Nb infected mice compared to non-infected controls. Using genetically modified mice, we found that this protective effect was dependent on IL-4/IL-13-induced STAT6 activation in hematopoietic cells. Finally, we could demonstrate the presence of these pathways in human disease by detecting GATA3-positive cells and eosinophils in the joints of rheumatoid arthritis patients. Taken together, our results revealed that the activation of the Th2 pathway effectively alleviates the course of inflammatory arthritis and also display a new strategy to treat this disease.

Z. Chen, D. Andreev, K. Oeser, B. Krljanac, A. Hueber, A. Kleyer, D. Voehringer, G. Schett, and A. Bozec. 2016. Th2 and eosinophil responses suppress inflammatory arthritis. Nature Communications 7:11596.

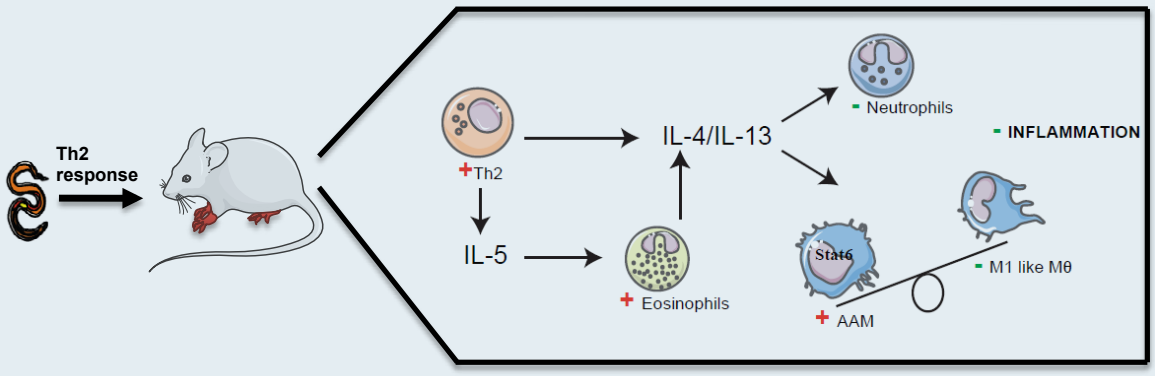


Figure: Schematic overview of cellular interplay during helminth-induced arthritis inhibition.

Nippostrongylus brasiliensis (Nb) infections leads to an activation of the Th2 pathway and an accumulation of Th2 cells and eosinophils in the joints. Induction of the cytokines interleukin-4 and interleukin-13 causes a STAT6 dependent shift from pro- to anti-inflammatory macrophages and a decreased number of neutrophils in the joints, which prompts a strong inhibition of inflammatory arthritis.

SCIENTIFIC HIGHLIGHTS

AnkG activity is controlled by intracellular localization

The activity of the Coxiella burnetii anti-apoptotic effector AnkG depends on p32 and Importinα1-mediated intracellular trafficking

WALTER SCHÄFER AND ANJA LÜHRMANN
INSTITUTE FOR CLINICAL MICROBIOLOGY, IMMUNOLOGY AND HYGIENE, FAU ERLANGEN-NÜRNBERG, UNIVERSITÄTSKLINIKUM ERLANGEN

The obligate intracellular, Gram-negative bacterium *Coxiella burnetii* is the causative agent of the zoonotic disease Q-fever. Q-fever is often a mild flu-like illness, but can develop into an interstitial pneu-monia or hepatitis. Furthermore, the infection may also become chronic which is potentially fatal. Im-portantly, an effective therapy of chronic Q fever still has to be established. *Coxiella* pathogenesis depends on a functional type IV secretion system (T4SS). The T4SS effector AnkG inhibits pathogen- induced host cell apoptosis, which is believed to be important for the establishment of a persistent infection.

Recently, we found that during *C. burnetii* infection AnkG is injected into the host cell in a T4SS-dependent manner. AnkG accumulated inside the host cell nucleus, the organelle of its activity. The localization of AnkG was regulated by intracellular trafficking, which was controlled by the host cell proteins p32 and importin α1. AnkG primarily targeted the mitochondria to sense host cell apoptotic stress and then captured p32 to be transported to the nucleus. However, the ability to bind to p32 was not sufficient to get transported into the nucleus. AnkG additionally had to bind to importin α1 to migrate into the nucleus.

Taken together, our results clearly demonstrated that during *C. burnetii* infection AnkG is imported into the nucleus to prevent host cell death. While we could show that AnkG activity was controlled by intracellular trafficking events, the precise biochemical activity of AnkG still has to be elucidated.

W. Schafer, R.A. Eckart, B. Schmid, H. Cagkoylu, K. Hof, Y.A. Muller, B. Amin, and A. Luhrmann. 2016. Nuclear trafficking of the anti-apoptotic Coxiella burnetii effector protein AnkG requires binding to p32 and Importin-alpha1. Cellular Microbiology Doi:10.1111/cmi.12634

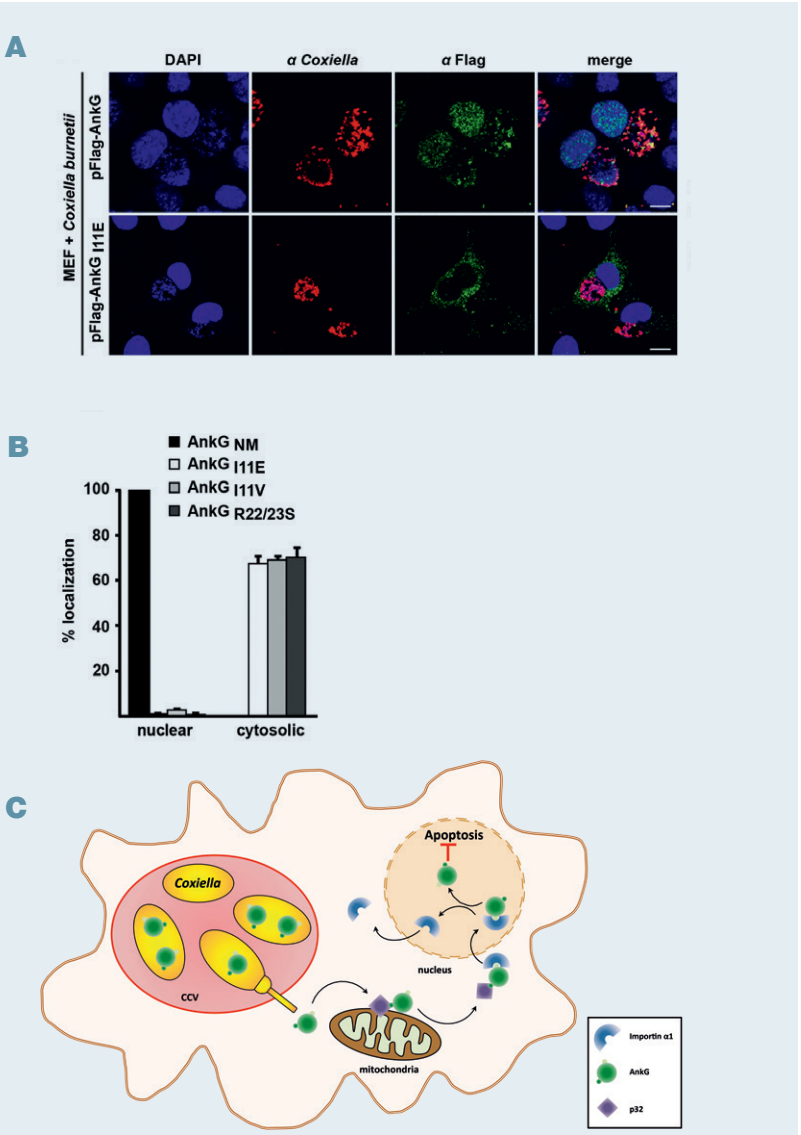


Figure:

Mouse embryonic fibroblasts (MEFs) were infected with *Coxiella burnetii* containing indicated plasmids. MEFs were fixed 48 hours post-infection and were stained with antibodies specific for Flag (green), *Coxiella* (red), and with DAPI (blue).

A) Representative confocal micrographs are shown. Scale bars, 10µm.

B) Intracellular localization of Flag-AnkG, Flag-AnkGI11E, Flag-AnkGI11V and Flag-AnkGR22/23S were quantified by confocal microscopy.

C) Once *Coxiella burnetii* has established a replicative vacuole (CCV), it translocates the type IV secretion substrate AnkG into the host cell cytoplasm. AnkG associates with mitochondria, where it binds to the host cell protein p32. Together with p32 AnkG traffics towards the host cell nucleus. After binding to importin α1, AnkG gets imported into the host cell nucleus. Inside the nucleus AnkG displays its anti-apoptotic activity.

SCIENTIFIC HIGHLIGHTS

mTORture in human ALPS: more than Fas deficiency?!

Hyperactive mTOR pathway promotes lymphoproliferation and abnormal differentiation in Autoimmune Lymphoproliferative Syndrome

SIMON VÖLKL, ANDREAS MACKENSEN
DEPARTMENT OF INTERNAL MEDICINE 5, HEMATOLOGY AND ONCOLOGY, FAU ERLANGEN-NÜRNBERG, UNIVERSITÄTSKLINIKUM ERLANGEN

The autoimmune lymphoproliferative syndrome (ALPS) is a rare human disorder of dysregulated lymphocyte homeostasis due to defects in the Fas signaling cascade. ALPS often manifests in childhood resulting in chronic benign lymphoproliferation with massive splenomegaly and lymphadenopathy, autoimmune manifestations and accumulation of TCR$\alpha\beta^+$ CD4$^-$CD8$^-$ double-negative T (DNT) cells. Although DNT cells represent a diagnostic hallmark of the disease, their role and ontogeny remains unclear.

We found that despite their terminally differentiated phenotype, pathognomonic DNT cells exhibit substantial mitotic activity *in vivo*. Notably, hyperproliferation of ALPS DNT cells was associated with increased basal and activation-induced phosphorylation of serine-threonine kinases Akt and mTOR. Moreover, increased mitotic activity and hyperactive mTOR signaling was also observed in recently defined CD4$^+$ or CD8$^+$ precursor DNT cells, indicating abnormal programming of Fas-deficient T cells before the DNT-stage. The mTOR inhibitor rapamycin abrogated survival and proliferation of ALPS DNT cells *in vitro* and reduced proliferation and abnormal differentiation *in vivo*. In contrast, DNT cells from ALPS patients treated with the immunosuppressive drug mycophenolate mofetil (MMF) retained the aberrant phenotype and mitotic activity, indicating that pharmacological regulation of the mTOR pathway might offer a superior therapeutic option than MMF therapy. Taken together, our results identify the mTOR pathway as a major regulator of lymphoproliferation and aberrant differentiation in human Fas deficiency.

S. Volkl, A. Rensing-Ehl, A. Allgauer, E. Schreiner, M.R. Lorenz, J. Rohr, C. Klemann, I. Fuchs, V. Schuster, A.O. von Bueren, N. Naumann-Bartsch, E. Gambineri, K. Siepermann, R. Kobbe, M. Nathrath, P.D. Arkwright, M. Miano, K.D. Stachel, M. Metzler, K. Schwarz, A.N. Kremer, C. Speckmann, S. Ehl, and A. Mackensen. 2016. Hyperactive mTOR pathway promotes lymphoproliferation and abnormal differentiation in autoimmune lymphoproliferative syndrome. Blood 128:227–238.

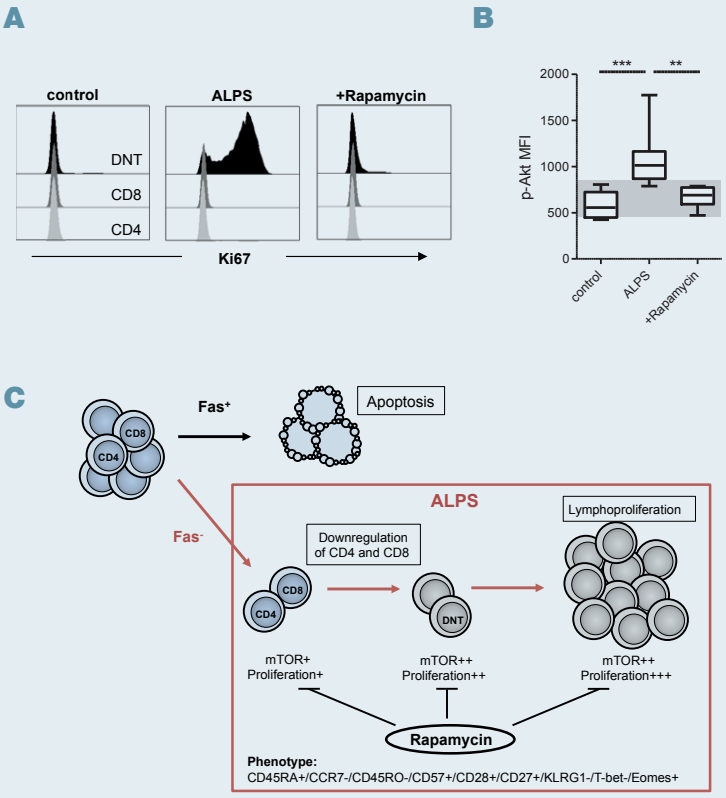


Figure:

A) Expression of Ki67 was determined in CD4$^+$ (light grey), CD8$^+$ (dark grey) and DNT cells (black) from healthy controls, ALPS patients and ALPS patients treated with rapamycin.

B) Phosphorylation of protein kinase Akt was analyzed in DNT cells from healthy controls, ALPS patients and ALPS patients under rapamycin therapy. Grey belt depicts the interdecile range of healthy donor T cells.

C) Based on our observation we propose the following model for the development of aberrant DNT cells in ALPS: Under normal conditions, CD4$^+$ or CD8$^+$ T cells can be eliminated by Fas/FasL interaction (black arrow). The majority of T cells in ALPS-FAS patients show normal differentiation and normal mTOR activation despite their defect in Fas signaling. Notably, a small fraction of CD4$^+$ and CD8$^+$ T cells show an abnormal differentiation and transcriptional program, enhanced mitotic activity and hyperactive mTOR signaling (red arrow). Aberrant CD4$^+$ and CD8$^+$ T cells downregulate the coreceptor and convert to DNT cells, while the expression profile remains constant. A hyperactive mTOR pathway in Fas-deficient cells leads to expansion of pathognomonic DNT cells and chronic lymphoproliferation. Rapamycin therapy is able to control mTOR activity in both DNT cells and their precursors, resulting in reduced proliferation and increased apoptosis of aberrant cells.

SCIENTIFIC HIGHLIGHTS

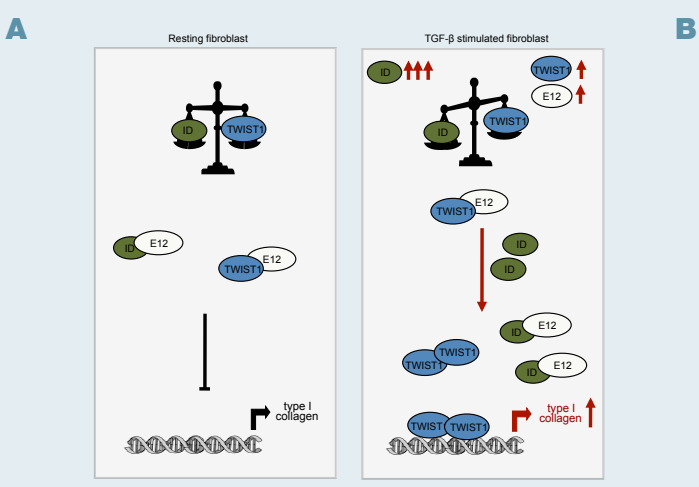
TWIST1 in fibrosis

Composition of TWIST1 dimers regulates fibroblast activation and tissue fibrosis

JÖRG DISTLER DEPARTMENT OF INTERNAL MEDICINE 3,
FAU ERLANGEN-NÜRNBERG, UNIVERSITÄTSKLINIKUM ERLANGEN

Balanced activation of fibroblasts is essential for normal wound healing. Failure in appropriate control of fibroblast activation, however, leads to progressive accumulation of extracellular matrix and fibrotic disease as in systemic Sclerosis (SSc). The molecular mechanisms, which regulate the activation of resident fibroblasts in physiologic and pathologic tissue responses, are incompletely understood. Defining such pathways might be essential to develop effective treatment for fibrotic diseases. Transforming growth factor-β (TGF-β) is a master-regulator of mesenchymal repair responses that remains persistently activated in fibrotic diseases. In the present study, we demonstrate that an imbalance in TWIST1 homo- and heterodimers amplifies TGF-β signaling and promotes activation of resident fibroblasts in SSc. Persistently increased TGF-β signaling in SSc shifts the balance from inhibitory TWIST heterodimers to pro-fibrotic TWIST1 homodimers, which foster the transcription of pro-fibrotic TGF-β target genes. Targeted inactivation of TWIST1 normalizes TGF-β signaling, de-activates SSc fibroblasts and inhibits fibrosis in several experimental models. Regulation of TWIST1 dimerization thus serves as a molecular switch to regulate TGF-β signaling and fibroblast activation and targeting of TWIST1 homodimers may be a novel approach for the treatment of fibrosis in SSc.

K. Palumbo-Zerr, A. Soare, P. Zerr, A. Liebl, R. Mancuso, M. Tomcik, B. Sumova, C. Dees, C.W. Chen, T. Wohlfahrt, T. Mallano, A. Distler, A. Ramming, K. Gelse, C. Mihai, O. Distler, G. Schett, and J.H. Distler. 2016. Composition of TWIST1 dimers regulates fibroblast activation and tissue fibrosis. Annals of Rheumatic Diseases doi: 10.1136/annrheumdis-2015-208470



Schematic summary of the role of Twist1 in fibroblast activation
(a) Resting fibroblast in the absence of TGF-β: The levels of Twist1, E12/E47 and Id proteins are in balance and E12/E47/Twist1 and E12/E47/Id heterodimers dominate. **(b) Fibroblast exposed to TGF-β:** The upregulation of E12/E47 in fibroblasts is outweighed by an even more pronounced induction of Id proteins. Id proteins have great affinity for E12/E47 leading to shift of Twist1/E12/E47 heterodimers to Id/E12/E47 complexes and Twist1/Twist1 homodimers. Direct binding of Twist1/Twist1 homodimers stimulates the transcription of profibrotic target genes such as col 1a1 and col 1a2.

PEOPLE

Prof. Dr. Aline Bozec

Prof. Aline Bozec received the Heinz Maier-Leibnitz award by the Deutsche Forschungsgemeinschaft (DFG)

Dr. Bozec, head of an Emmy Noether independent junior research group at the Department of Medicine 3, Universitätsklinikum Erlangen, was honored by the DFG with one of the most important national research awards for young academics, the Heinz Maier-Leibnitz award. The French-born biochemist has been studying the key transcription factors and signal pathways involved in the activation and differentiation of osteoclasts, osteoblasts and fat cells. During her research career which has led her from France to Spain, then Austria and since 2012 to Erlangen, she made fundamental discoveries regarding the regulation of bone growth and resorption. Moreover, she was able to identify key events concerning the modulation of regenerative mechanisms in bone marrow stem cell niches and their significance during health and disease.

The Heinz Maier-Leibnitz award worth 20.000€ was handed over to Aline Bozec during a festive occasion in Bonn on May 18, 2016.

NEWS AND UPDATES

The members of the CRC643 on the occasion of the final symposium on July 16 and 17, 2016

July 15 – 16, 2016 FINAL SYMPOSIUM OF THE CRC643 “Strategies of Cellular Immune Intervention”

After 12 years of funding by the Deutsche Forschungsgemeinschaft, the CRC643 came to a formal end on June 30, 2016 (see also the Editorial of this issue). The research group leaders, postdocs, doctoral students and technicians of the 19 projects from the last funding period met at the Novotel hotel in Erlangen for a two day symposium together with national and international guest speakers. Everybody very much enjoyed the final wrap up of the central research themes of the consortium and thanked the spokesmen Gerold Schuler and Alexander Steinkasserer for running this highly successful CRC.

April 29, 2016 DAY OF IMMUNOLOGY

This year´s international Day of Immunology themed “Pirates of the Immunobay – Adventure Immune System” was organized by doctoral students of the DFG research training groups GK1660 and IRTG TRR130. Every year at the end of April, the EFIS plans this event to inform the public about various immunological topics such as allergies, autoimmune diseases, vaccination, or immune responses towards pathogens and tumors.

This year, everything was based on a pirate adventure to arouse children´s interest. For this, a short story and painting book was created about the “treasure of life” and how it was saved from the evil pirates who attack to steal the treasure.

This book was available at a kids` station with different games and handicraft work. Furthermore, several pathogens could be observed at a microscopy station. A poster presentation informing about general functions of the immune system, history of vaccination and common pathogens was displayed and participants could win little prizes at a raffle. The “Deutsche Rote Kreuz” was also present with a service station offering free check-ups.

All in all, it was a very successful event with a lot of positive feedback by the visitors. We would be pleased to welcome many interested people to the next Day of Immunology 2017.

Written by Anne Hahn, Alexandra Weise, Tobit Steinmetz

Doctoral students of the GK1660 and IRTG TRR130 at the Hugenottenplatz, Erlangen on Day of Immunology 2016 Source: Alexandra Weise

UPCOMING EVENTS

Immunological Colloquium of the Medical Immunology Campus Erlangen – Winter 2016

Tuesdays, 5.15 pm

18. 10. 2016
Prof. James J. Lee
Mayo Clinic, Scottsdale, Arizona, USA
Eosinophils in Health and Disease: Regulators of Local Tissue Immune Responses

25.10. 2016
Prof. Nicola Harris
École polytechnique fédérale de Lausanne, Switzerland
Title to be announced

08.11. 2016
Joachim Kalden Lecture 2016
Prof. Fiona Powrie
Kennedy Institute of Rheumatology, University of Oxford, UK
Gut reactions: Immune pathways in the intestine in health and disease

29.11.2016
Prof. Bernd Schmeck
Philipps-Universität Marburg, Klinik für Pneumologie
Modeling RNA-networks in host-pathogen interaction

06. 12. 2016
Prof. Paul Crocker
School of Life Sciences, University of Dundee, UK
Regulation of neutrophil and macrophage functions by the murine inhibitory lectin, Siglec-E

20. 12. 2016
Prof. Andrew Mellor
Augusta University Cancer Center, Augusta, GA, USA
Exploiting DNA as an immune adjuvant to treat cancer & auto-immune syndromes

10. 01. 2017
Prof. Andrea Alimonti
Institute of Oncology Research, Bellinzona, Switzerland
Title to be announced

31. 01. 2017
PD Dr. Gernot Schabbauer
Institute of Physiology, Medical University of Vienna, Austria
Title to be announced

07. 02. 2017
Prof. Daniela Finke
Developmental Immunology, University Children's Hospital of Basel and Department of Biomedicine, Basel, Switzerland
Title to be announced

Further Conferences and Events of Interest

September 9 – 11, 2016
5th International Symposium – Regulators of Adaptive Immunity
Erlangen
www.gk-symposium.de

September 11 – 14, 2016
68. Jahrestagung der DGHM
Ulm
www.dghm-kongress.de

September 21 – 23, 2016
30th Annual Conference of the European Macrophage and Dendritic Cell Society (EMDS)
Amsterdam, The Netherlands
www.dghm-kongress.de

September 27 – 30, 2016
46th Annual Meeting of the German Society for Immunology
Hamburg
www.immunology-conference.de

October 5 -7, 2016
Interdisciplinary Forum on Virulence Mechanisms of Phyto- and Human-Pathogenic Fungi

Institute of Microbiology – Clinical Microbiology, Immunology and Hygiene

This international symposium brings together renowned experts of molecular mycology to discuss distinct as well as common principles of fungal virulence and infections affecting plants and patients. Besides a plenary talk given by Prof. Dr. Regine Kahmann from the MPI of Terrestrial Microbiology in Marburg, regular sessions with invited speakers, short talks by upcoming researchers in the field, and a poster exhibition will provide an up-to-date overview on this exciting topic of infection research.

www.mikrobiologie.uk-erlangen.de/kongress/

October 9 – 14, 2016
8th Autumn School ›Current Concepts in Immunology‹
Merseburg
www.herbstschule.de

Medical Immunology Campus Erlangen

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

Publisher
Medical Immunology Campus Erlangen
An Interdisciplinary Center of the Friedrich-Alexander-Universität Erlangen-Nürnberg

Dr. rer. nat. Sonja Pötzsch (Scientific Coordinator)

Mikrobiologisches Institut – Klinische Mikrobiologie, Immunologie und Hygiene

Universitätsklinikum Erlangen
Friedrich-Alexander-Universität Erlangen-Nürnberg
Wasserturmstraße 3/5 · 91054 Erlangen

Phone +49. 9131. 85. 225 71
Fax +49. 9131. 85. 225 73

Mail Sonja.Poetzsch@uk-erlangen.de
www.mice.uni-erlangen.de

Conceptual Design and Editor
Dr. rer. nat. Sonja Pötzsch V.i.S.d.P.

Subscription via Email to:
Sonja.Poetzsch@uk-erlangen.de

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

FRIEDRICH-ALEXANDER UNIVERSITÄT ERLANGEN-NÜRNBERG