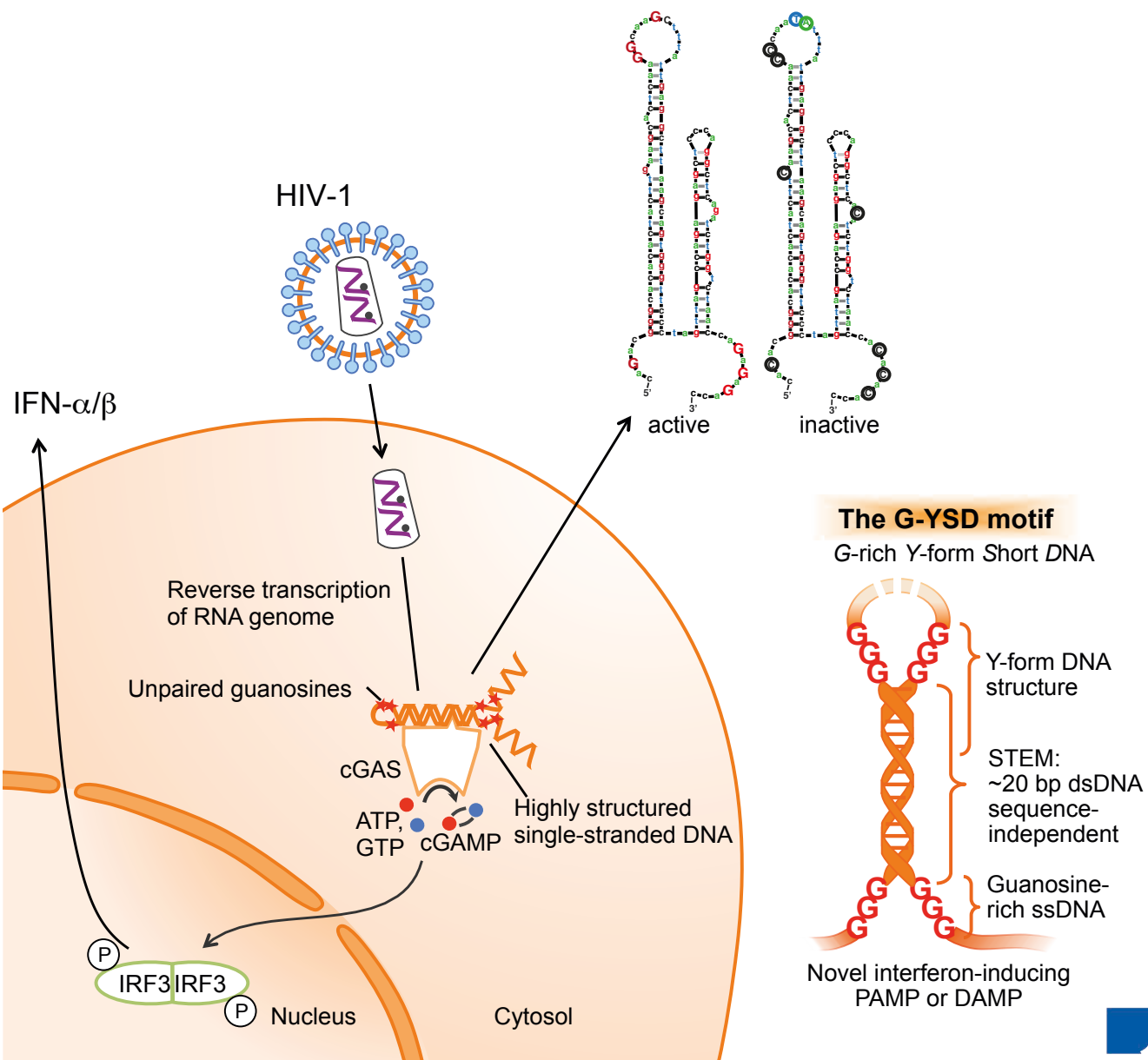
ImmunoSensation

the immune sensory system Bonn cluster of excellence

ANNUAL REPORT 2015



universitätbonn

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universitätsklinikumbonn
LIMES Life & Medical Sciences Institute
DZNE German Center for Neurodegenerative Diseases within the Helmholtz Association
caesar center of advanced european studies and research
DFG

OVERVIEW

Preface

Preface

The Cluster of Excellence “ImmunoSensation”, funded by the German Research foundation, DFG, proudly presents you its third annual report. The year 2015 was a great success with many scientific and structural achievements, and we are deeply grateful for the continued support of the DFG, the University of Bonn, the German Center of Neurodegenerative Diseases, DZNE, and the Max Planck-associated Center of Advanced European Studies and Research, caesar.
Scientific achievements and the constant striving for scientific excellence are at the heart of our endeavor. The main section of this report is therefore dedicated to presenting our most important publications from the last year in the research fields of innate immune sensing, T cell memory formation, neuro-immunology, cellular orchestration in the lymphatic system, and cancer immunology. These outstanding contributions are fruits of the collaborative effort of our Cluster members including senior authors Martin Schlee, Gunther Hartmann, Percy Knolle, Andreas Zimmer, Wolfgang Kastenmüller and Michael Hölzel.
At the core of our Cluster of Excellence are people and their research activities, and these greatly benefit from intellectual academic exchange. We proudly announce the recruitment of several outstanding scientists as new Cluster members over the last year. These are: Christoph Wilhelm who was recruited as Professor for Immunopathology from the NIH, USA in September 2015, Sven Wehner, who was recruited as Professor for Immunopathophysiology from the University of Amsterdam, Andreas Schlitzer, who has been awarded the prestigious Emmy-Noether fellowship by the DFG in November 2015 and joined the LIMES Institute from A-Star, Singapore, and Annkristin Heine and Zeinab Abdullah, who established two new Cluster Junior Research groups.

Prof. Waldemar Kolanus (Vice-Speaker) and Prof. Gunther Hartmann (Speaker)
(picture by J. Saba UKB)

Preface

We have also been able to continue our support of young scientists and expand the international scope of our programs. Cluster member Christian Kurts spearheaded the effort to establish a new DFG International Research Training Group (IRTG) for PhD students, and, in November 2015, the “Bo&MeRanG” program, or Bonn & Melbourne Research and Graduate training group, was established. Focusing on “Myeloid Antigen Presenting Cells and the Induction of Adaptive Immunity”, the program will promote scientific collaboration between leading immunologists from ImmunoSensation and the University of Melbourne. In addition to promoting scientific outreach, this program ideally complements our research focus. The Cluster contributes broad expertise in immune sensing receptors and molecular and transcriptional immune regulation, whereas Melbourne is a leading international center in the field of immunological defense against infection. Moreover, PhD students selected for the program will have the unique opportunity to obtain instruction and their degree from both universities. The DFG will fund this new joint international graduate college at the Universities of Bonn and Melbourne for 4.5 years starting in April 2016. The new graduate school will be located at the ImmunoSensation headquarters of the Bonn Institutes of Immunosciences and Infection (BI[3]) and will move into the new Biomedical Center II (BMZII) currently being constructed on the campus Venusberg.
Furthermore, our ImmunoSensation student training program, the IITB (International Immunology Training Program Bonn), is now fully developed, and its offers include outstanding training opportunities in a wide spectrum of immunology-related research areas as well as travel fellowships for attending international meetings and lab rotations worldwide.
The Cluster is now fully represented on social media. One great example is the “ImmunoSensation Blog”, a framework in which students and members of the Cluster regularly exchange information and expertise.
The international scope of our activities has become also more visible through our contributions to important meetings and symposia. The International Symposium on DAMPs and HMGB1 was established by the international HMGB1 group of scientists with the goal of better understanding the mechanisms of DAMP and HMGB1 biology and their implications for disease. This year, we hosted the meeting under Gunther Hartmann’s organization, making it the first time a German site was selected to host this international event. The meeting was met with a resounding international response, and we are proud that we could welcome so many internationally renowned experts in innate immunity to Bonn, including Charles Dinarello, Tak Mak, Marco Bianchi and Michael Lotze who all gave fantastic talks.
In addition, the Mini-Symposium “Epigenetics and Chromatin Structure” took place at the LIMES Institute as a part of the LIMES Women in Science (WiS) program in March 2015. This program is sponsored through the DFG-funded collaborative research centers SFB 704, SFB 645 and TRR 83 at the University of Bonn. Among other initiatives, the LIMES-WiS program aims to enhance the visibility of successful female scientists as role models for career development. LIMES-WiS is also linked with the gender equality program of ImmunoSensation, and we are proud to be part of this initiative. The Mini-Symposium was organized by Cluster members Irmgard Förster and Joachim Schultze and focused on the topic of epigenetics to provide a state-of-

Preface

the-art overview of recent achievements within this rapidly expanding field of research.Furthermore, the important structural impact of our Cluster has become more and more visible through the establishment of joint structures with partner institutions: for example, Joachim Schultze, the head of ImmunoGenomics at LIMES Institute of the Faculty of Natural Sciences, was co-appointed as a group leader at the Genomics Department of the DZNE in September 2015.
We are also very proud that several members of ImmunoSensation have received prestigious awards and honors (see page 153) during the last year. The Gottfried Wilhelm Leibniz Prize is the most renowned research prize in Germany and was awarded to Cluster member Frank Bradke for his work on neuroregeneration. Congratulations to Frank and all of the other awardees for their outstanding scientific discoveries!
Last but not least, our Cluster member Michael Hoch from LIMES Institute has been appointed as the one-hundred-forty-third Rector of our University. We wish him good luck and great success in this prestigious and demanding position!

We hope that this short excerpt of our activities will tempt you to read more about our mission in science and in public outreach. Welcome to ImmunoSensation!

Kind regards and best wishes,

Prof. Gunther Hartmann and
Prof. Waldemar Kolanus

Research Area A: Immune Sensing Receptors and Modulators

Prof. U. Benjamin Kaupp, PhD
center of advanced european studies and research (caesar)

Prof. Eicke Latz, MD PhD
Institute of Innate Immunity
Medical Faculty
University of Bonn

Introduction

Innate immune cells have a critical role in the rapid recognition of and response to microbial infections and changes in normal tissue homeostasis, such as tissue damage, tumor growth or metabolic derangements. To perform these vital tasks, innate immune cells are equipped with a battery of germline-encoded signaling receptors. These molecular sensors can recognize microbial substances as well as endogenous molecules that appear in the host during disturbances of tissue homeostasis. Nucleic acids represent one class of molecules that can alert the immune system to a microbial infection or that signify damage or stress in cells and tissues. A range of innate immune signaling molecules have evolved to recognize nucleic acids, and these molecules are connected to key signaling pathways that can drive potent pro-inflammatory or anti-viral immune responses. The last decade of research in the immune sensing of nucleic acids has uncovered many complex molecular mechanisms which allow nucleic acid sensing receptors to distinguish physiologically-occurring nucleic acids from those appearing during infections or cellular stress. One fundamental principle of this process is the use of specific “molecular tags” on or with the nucleic acids themselves. These “tags” can be specific sequences and structural features within nucleic acids, covalent modifications or combination thereof, and their presence either “disables” or “licenses” recognition by a range of nucleic acid sensors. Research in the ImmunoSensation Cluster in the last year has uncovered critical details of how RIG-I and cGAS, two key cytoplasmic sensors of RNA and DNA, can distinguish between microbial and host-derived nucleic acids. Martin Schlee and his colleagues were able to discover that a simple covalent modification to RNA, N12’O-methylation, could “tag” RNA as endogenous and prevents RIG-I-mediated immune activation. This relatively simple mechanism is a key checkpoint in the recognition of cytosolic RNA. In a separate study, Schlee and colleagues could also identify a novel DNA structure which activates cGAS, which they coin “Y-DNA”. These cGAS ligands require critical guanosines and are single-stranded, thus overturning two previous paradigms concerning cGAS sensing: (1) that it is inherently sequence independent and (2) that it critically requires long double-stranded DNA. These studies provide yet another piece to the great puzzle of how nucleic acid sensors monitor subtle molecular details in nucleic acids.

A conserved histidine in RIG-I controls immune tolerance to N1-2'O-methylated self RNA

RNA genome-based viruses are some of the most highly pathogenic and emergent currently known (e.g. Influenza, yellow/dengue/Lassa fever, Ebola disease virus). The cell-autonomous and systemic antiviral immune responses against RNA viruses are initiated by cytosolic RNA sensing innate immune receptors, i.e. RIG-I and/or MDA5. These receptors are expressed in most cell types, including somatic cells. The detection of a few viral RNA molecules within abundant host RNA in the cytosol is exquisitely challenging and usually requires the sensing of unusual structures or modifications of viral RNAs. While MDA5 recognizes (>300nt) long RNA molecules which are double-stranded or build extensive secondary structures, RIG-I can be activated by much shorter double-stranded RNA molecules (>19bp) provided that di- or triphosphates are present at the 5'end ((p)pp-dsRNA), (Hornung et al. 2006; Pichlmair et al. 2006; Schlee et al. 2009; Schlee 2013; Goubau et al. 2014). Like most viral RNAs, endogenous mRNA

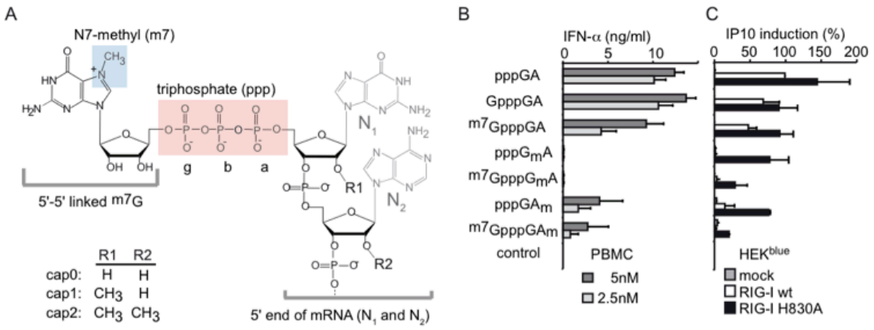

Figure 1 His830 in the RIG-I RNA binding domain prevents recognition of endogenous RNA bearing a N1-2'O-methyl group as a marker of "self"

A: Eukaryotes' mRNA cap (m7GpppNmNm): N1 5'ppp5' linked to G with N7-methylation. N1/N2 are 2'O-methylated in higher eukaryotes' mRNA (cap2). B: Chloroquine treated PBMC stimulated with the indicated synthetic RNA hybridized with complementary RNA. IFN-α production was analyzed 20h after stimulation. C: RIG-I(wt) or RIG-I(H830A) was overexpressed in HEK293blue cells and stimulated with ppp-dsRNA ligands as in B. IP10 production was analyzed 20h after stimulation. m=2'O-methyl.

bears a triphosphate moiety at its 5' end, but, upon maturation in the nucleus, eurkaryotic mRNAs is subjected to 5' capping. The cap structure contains the triphosphorylated 5' end which is then 5'-5' linked to a guanosine methylated at N7 (m7G) (**Figure 1**). This m7G cap is prerequisite for the translation of protein from most eukaryotic mRNAs whether yeast or vertebrates. To date, the m7G cap was considered the primary inhibitor of RIG-I activation via endogenous mRNA. However, this conclusion was based on studies which made use of RIG-I ligands produced by enzymatic *in vitro* transcription (IVT). Later, it was discovered that IVT also produced unintended immostimulatory side products which distorted the interpretation of stimulation experiments. In the featured study (Schuberth-Wagner

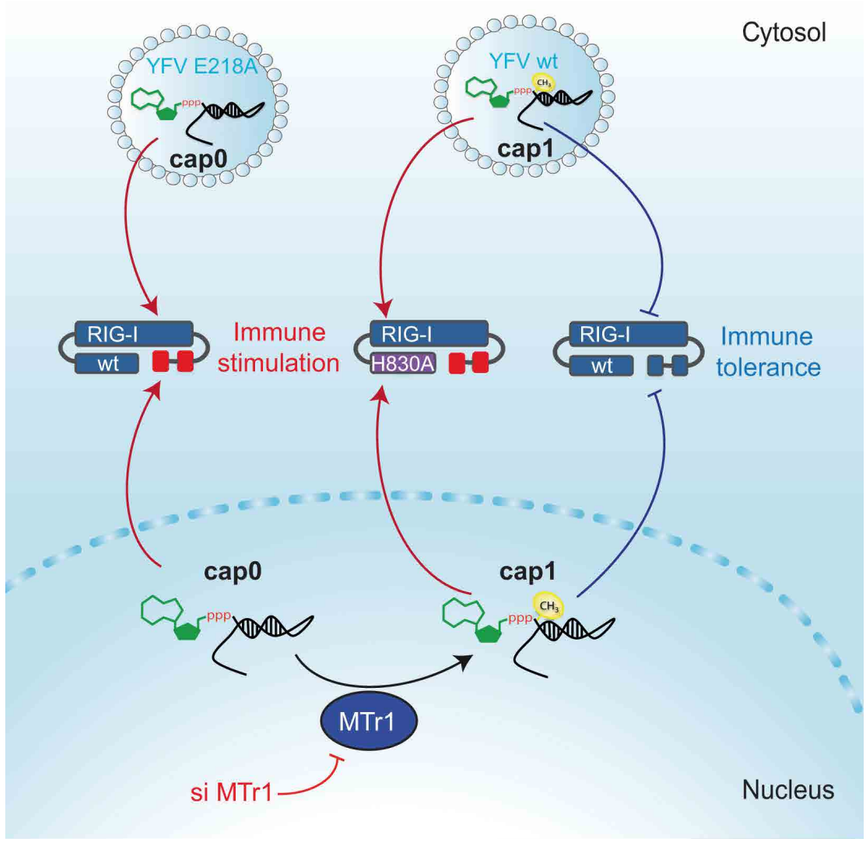

Figure 2 Graphical Summary of 'A conserved histidine in RIG-I controls immune tolerance to N1-2'O-methylated self RNA'.

et al. 2015), we instead used defined synthetic pppRNA and unexpectedly found that m7G capped RNA (m7GpppRNA) still profoundly activated RIG-I. This observation raised the question what feature of mRNA, if not the m7G cap, is actually responsible for mRNA immunotolerance against RIG-I? Higher eukaryotic mRNA cap structures also manifest 2'O-methyl modifications at N1 (cap1) or N1 and N2 (cap2) (Banerjee 1980) (**Figure 1**), and we discovered that this 2'O-methylation of the 5'-terminal nucleotide (N1) could completely inhibit RIG-I activation. Using structure-guided mutations, we could identify a highly conserved histidine (His830) in the C-terminal RNA binding domain of RIG-I, which was dispensable for RIG-I activation but mediated sterical exclusion of N1-methylated RNA. Accordingly, we observed that endogenous RNA could stimulate RIG-I if either endogenous cap1 2'O-methylation was inhibited or if His830 was mutated. Strikingly, we also found that RNA viruses exploit this very tolerance mechanism to avoid recognition by RIG-I. The 100% conservation of this "licensing" histidine position in RIG-I among vertebrates can even be found in sea anemone RIG-I, which highlights the fundamental role of this self-RNA tolerance mechanism in evolution. As

such, this is the first study showing the biological relevance of endogenous cap 2'O-methylation and the corresponding enzymes for immune tolerance of self-RNA by RIG-I, an innate immune receptor which is expressed in nearly every cell of our bodies.

by Martin Schlee

Reference Publication

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Ann Kristin Bruder, Dr. Martin Schlee (picture by Dr. Anna-Maria Herzner)

Sequence-specific activation of the DNA sensor cGAS by Y-form DNA structures as found in primary HIV-1 cDNA

The type I interferon response is an essential defense mechanism against viruses. Therefore, many viruses have evolved mechanisms to evade their detection by the pattern recognition receptors that initiate a type I interferon response. HIV-1 exploits the human proteins, TREX-1 and SAMHD1 to avoid recognition. Under physiological conditions, these proteins act as counterregulators of the Type-I IFN response to avoid autoinflammation. However, they also hinder the accumulation of cytosolic DNA that arises during the replication cycle of this retrovirus (Puigdomènech et al. 2013; Yan et al. 2010). Recent research has demonstrated that the so-called elite controllers, HIV-1 positive patients who control viral titers without any medical treatment, have reduced TREX-1 and SAMHD1 activity in innate immune cells, enabling the recognition of HIV-1 by cGAS, the cytosolic pattern recognition receptor responsible for DNA recognition and subsequent type-I-IFN induction (Martin-Gayo *et al.*; Gao *et al.* 2013).

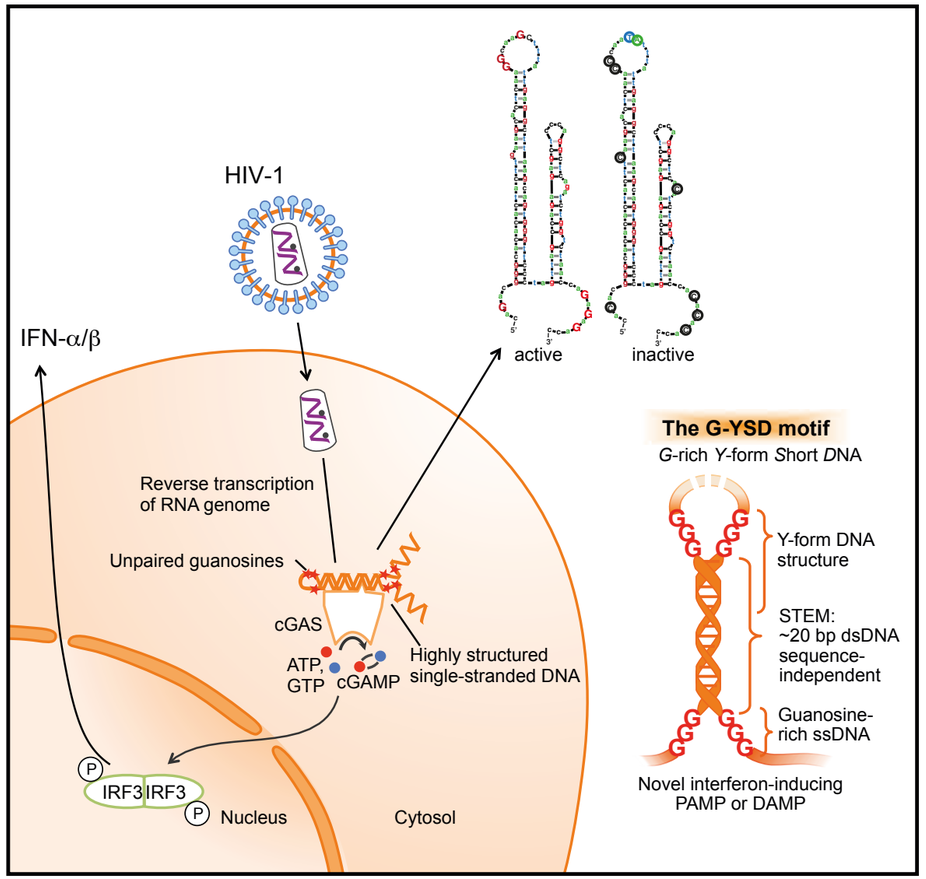

Figure 3 Graphical summary of 'Sequence-specific activation of the DNA sensor cGAS by Y-form DNA structures as found in primary HIV-1 cDNA'.

In our research, we examined conditions under which SAMHD1 activity is suppressed, and we could observe an increased accumulation of single-stranded DNA during early HIV-1 infection. In addition, these conditions also induced a robust, cGAS-dependent type-I-IFN response. We then generated lentiviral particles containing a genetically modified version of the HIV-1 reverse transcriptase with an impaired production of double-stranded (ds)DNA, and this also lead to enhanced type-I-IFN induction. These findings were unexpected, since all previously published data, including crystallization and *in-vitro*-activation studies of cGAS, had demonstrated that only paired, double-stranded DNA with a minimum of 40 base pairs, but not unpaired, single-stranded DNA, could activate cGAS and induce type-I-IFN. While others had already shown that double-stranded stretches of ssDNA could also activate type-I-IFN via base-pairing within the secondary structure, in HIV ssDNA, the predicted secondary structures in base-paired stretches still seemed too short for recognition by cGAS. Surprisingly, we found that the recognition of HIV ssDNA was not dependent on its base-paired regions but instead the short, single-stranded stretches in between them. Furthermore, this recognition depended on the presence of guanosines within the unpaired stretches. This was unanticipated, since up to then, DNA recognition by cGAS had been considered sequence-independent. However, we observed that this sequence independence was only true for base-paired but not for unpaired DNA. Furthermore, we found that base-paired DNA of any sequence, even as short as 12-20 bp, induced a robust type-I-IFN response if it was flanked with three consecutive guanosines at each possible 3' or 5' end. Other flanks, such as cytidines, adenosines or thymidines were inactive. We termed this sequence-specific cGAS recognition motif in branched DNA G-ended Y-form short DNA (G-YSD) (**Figure 3**).

Our study describes a completely new type of cGAS ligand. YSD is a minimal, sequence-specific motif enabling the recognition of single-stranded DNA with base-paired stretches within their secondary structures. Recognition of this motif enables sensing of early HIV-1 infection. Further research will be necessary to investigate the importance of this motif in the recognition of other forms of pathogenic and endogenous DNA.

by Martin Schlee

Reference Publication

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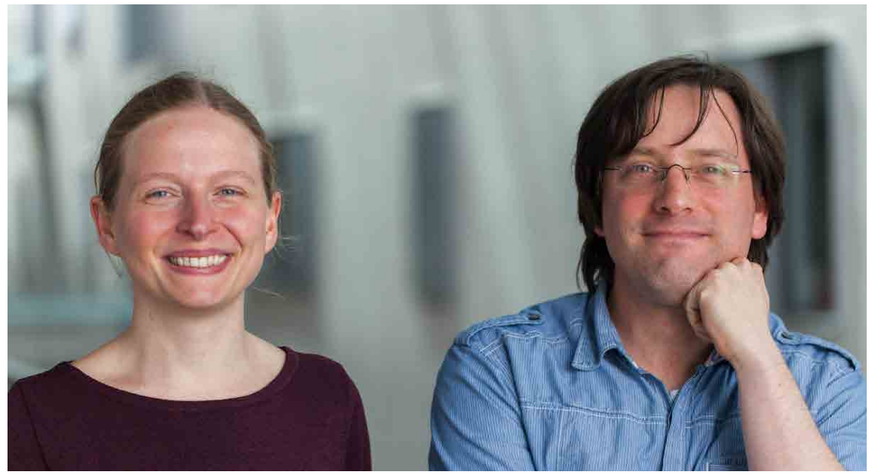

Dr. Anna-Maria Herzner, Dr. Martin Schlee (picture by Dr. Jonas Doerr)

Research Area B: Local Context Sensing

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Prof. Percy Knolle, MD
Institute of Molecular Medicine
University of Bonn
Institute of Molecular Immunology
Technical University Munich

Prof. Waldemar Kolanus, PhD
Molecular Immunology & Cell Biology
Life and Medical Sciences Institute (LIMES)
University of Bonn

Introduction

In response to an infection with pathogens, the immune system mounts a balanced response to assure control of infection while usually avoiding the onset of immunopathology in tissues or lymphoid organs. Innate immunity provides a first line of protection against infectious microorganisms through a variety of mechanisms, including the generation of inflammation and the recruitment of phagocytic immune cells and other cell populations, some of which can mount immediate effector functions. Innate immune activation is also required for the subsequent programing of pathogen-specific T cell responses, where $CD8^+$ T cell driven immunity plays a predominant role and, in particular, eventual T cell-dependent cytotoxicity. $CD8^+$ T cell differentiation is an integrated process which links multiple signals from the local, i.e. organ-specific, microenvironment and from the cell populations capable of (cross)-presenting antigens derived from infectious microorganisms. Co-stimulatory molecules on antigen (cross)-presenting cells (APC) are essential to the priming of naïve $CD8^+$ cells. After clearance of the primary infection, $CD8^+$ T cells usually undergo apoptosis. However, some of these activated killer T cells survive and are termed memory $CD8^+$ T cells, based on their ability to survive and slowly proliferate in the absence of cognate antigens for long periods of time via IL-7 and IL-15-mediated signals. At present, no single marker exists for memory T cells, but they are operationally characterized by the expression of Bcl2, IL7Rα (CD127) and CD27. Memory T cells are traditionally classified into two subsets: central memory T cells, which reside in lymphoid tissues, and effector memory T cells in peripheral tissues. However, this coarse classification does not fully appreciate the apparent plasticity and functional sub-specialization within T memory compartments. The following summary of a paper published by Percy Knolle and colleagues (Böttcher *et al.*, Nat Comm. 2015) describes a new functional marker which allows the discrimination between proliferating and effector-type memory $CD8^+$ T cells via flow-cytometry, employing the marker CX3CR1.

Identification of a new marker that stratifies memory T cells with proliferative potential from those with direct effector function

Induction of CD8 T cell memory is critical for the protection of the organism against subsequent encounters with the same pathogen. The induction of memory CD8 T cells is the result of immune sensing which leads to inflammation and the programming of naïve into memory CD8 T cells. Conventionally, memory T cells have been classified according to their anatomic localization, with central memory T cells residing in lymphoid tissues and effector memory T cells in peripheral tissues, such as liver, lung or skin. Thus, localization of memory CD8 T cells to lymphoid or peripheral tissues is believed to correlate with proliferative capacity and effector function.
Scientists from the Institute of Experimental Immunology, together with the LIMES Institute, the Max Planck Institute of Biochemistry in Munich and the Clinic for Internal Medicine II at the University Hospital Freiburg, have identified a new molecular marker that stratifies memo-

ry T cells into those with direct effector function or with proliferative potential. Transcriptome and proteome profiling of memory CD8 T cells identified a differentially up-regulated expression of the fractalkine receptor CX3CR1 on memory CD8 T cells that can exert direct cytotoxic effector function and kill their target cells. Such CX3CR1 expression characterized memory CD8 T cells with direct cytotoxic effector function in both, mice and humans. Conversely, those memory CD8 T cells that did not express CX3CR1 produced high amounts of the cytokine IL-2 and showed proliferative potential upon restimulation. Thus, CX3CR1 expression was predicted to correlate with the anatomic distribution of memory CD8 T cells, i.e. CX3CR1^{+} effector memory T cells were expected in peripheral tissues and CX3CR1^{-} central memory CD8 T cells in lymphoid tissues. Surprisingly, CX3CR1^{+} memory CD8 T cells were detected close to the subcapsular sinus in lymph nodes, thus contradicting the accepted notion that only central memory CD8 T cells resided within secondary lymphoid tissues such as lymph nodes. Moreover, these CX3CR1^{+} memory CD8 T cells remained in lymph nodes for extensive time periods and their slow velocity and scanning of their environment was indicative of antigen-presenting cells. This indicates that these memory CD8 T cell population with direct effector function in the lymph nodes may have a role in the rapid initiation of CD8 recall responses that is most efficiently triggered by antigen derived from dead cells. The correlation of CX3CR1 expression with effector function also allowed the quantification of antigen-specific memory CD8 T cells with effector function during viral infection. In mouse models of acute resolved and chronic LCMV infection, resolved infection demonstrated higher CX3CR1 expression on virus-specific memory CD8 T cells than chronic infection. Similarly, the number of CX3CR1^{+} hepatitis C virus-specific CD8 T cells was lower in patients with chronic Hepatitis C compared to the number of Cytomegalovirus-specific CD8

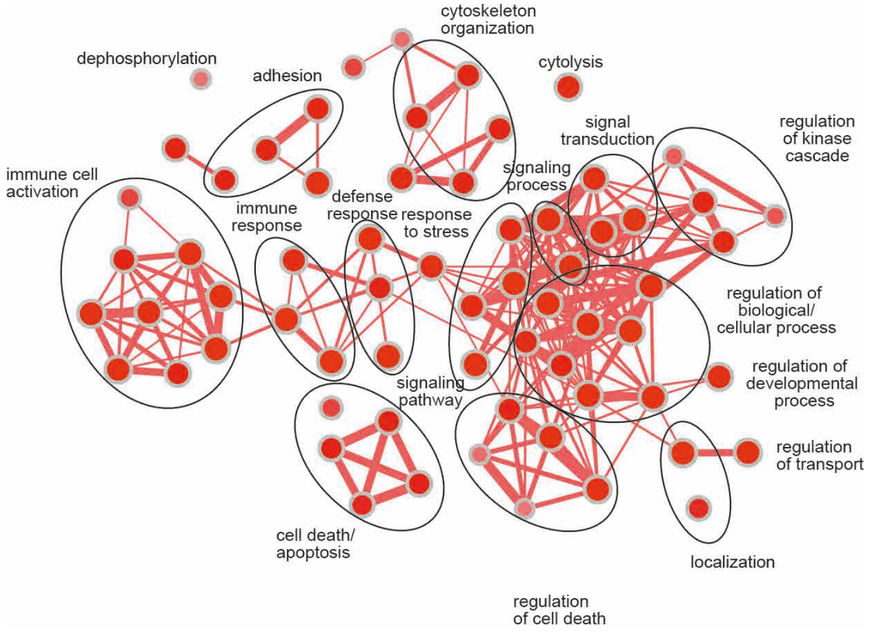

Figure 1 Model outline for the generation of organ specific, local T cell memory in the liver.

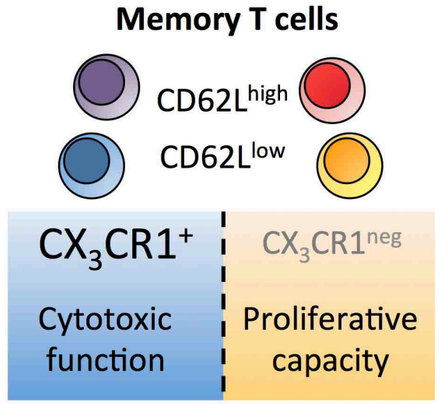

Figure 2 Model for the distinction of memory T cells upon expression of the marker CX3CR1 in a group with proliferative potential and with direct effector function.

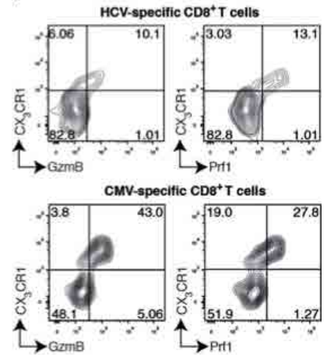

Figure 3 Presence of CX3CR1^{+} CD8^{+} T cells during acute resolved and chronic viral infection. HCV-specific CD8^{+} T cells (top) and CMV-specific CD8^{+} T cells (bottom).

Prof. Percy Knolle

T cells, which could efficiently controlled cytomegalovirus infection. Furthermore, CX3CR1 expression was a more accurate predictor of effector function in memory CD8 T cells than expression of the co-inhibitory receptor PD1. The identification of CX3CR1 as a marker for the functional classification of memory CD8 T cells will prove useful in future studies to determine the dynamics of immunity during chronic infection or cancer and thus improve T cell-based immunotherapies.

by Percy Knolle

Reference Publication

Jan P. Böttcher, **Marc Beyer**, Felix Meissner, **Zeinab Abdullah**, Jil Sander, Bastian Höchst, Sarah Eickhoff, Jan C. Rieckmann, Caroline Russo, Tanja Bauer, Tobias Flecken, Dominik Giesen, Daniel Engel, Steffen Jung, Dirk H. Busch, Ulrike Protzer, Robert Thimme, Matthias Mann, **Christian Kurts**, **Joachim L. Schultze**, **Wolfgang Kastenmüller** & **Percy A. Knolle** (2015) **Functional classification of memory CD8+ T cells by CX3CR1 expression.** Nature Communications Sep 5; 6:8306.

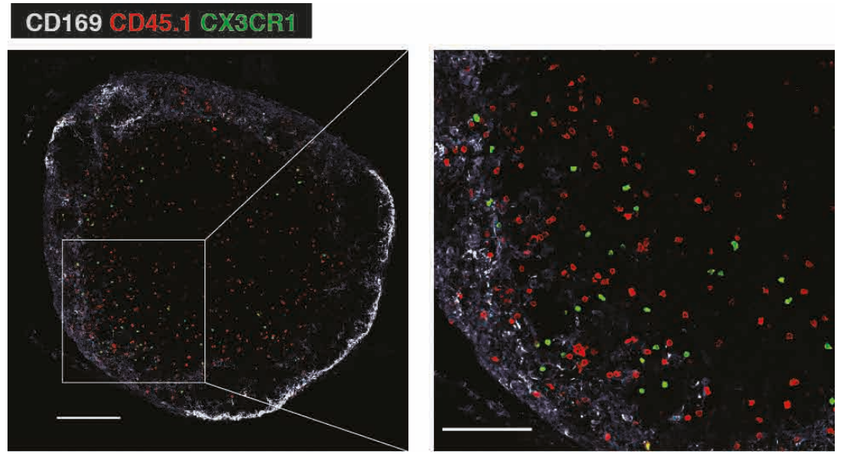

Figure 4 CX3CR1 identifies a distinct population of CD8^{+} memory T cells in lymph nodes. Confocal immunofluorescence images of popliteal lymph nodes harbouring CX3CR1^{neg} and CX3CR1^{pos} memory OT-I CX3CR1-GFP T cells.

Research Area C: Metabolic Sensing and Nervous-Immune System Interactions

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Life and Medical Sciences Institute (LIMES)
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Prof. Andreas Zimmer, PhD
Institute of Molecular Psychiatry
Medical Faculty
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Introduction

In this research area of the Cluster, we address how metabolites are sensed to evoke specific immune responses and how this affects the nervous system to bring about changes in physiology and behavior.

In the Hoch lab, the metabolites studied are ceramides, a special class of lipids, in both Drosophila and mouse. Drosophila has a single ceramide synthase family member (named Schlank), which is involved in ceramide synthesis and fat metabolism. Mammals have six ceramide synthases, of which CerS5 shows the highest homology to Drosophila Schlank. Hoch and his researchers could show that the specific ceramide generated by CerS5, namely C16:0, promotes diet-induced obesity and insulin resistance. Since two ceramide synthases are involved in C16:0 production, CerS5 and CerS6, a double knockout was then generated, thus completely abrogating synthesis of C16:0 ceramide. These mutants will be used for further studies on the influence of C16:0 on fat metabolism, physiology and inflammation.

The Zimmer lab has had a long-standing interest in another lipid signaling system: the endocannabinoid system. This system includes the cannabinoid receptors, CB1 and CB2, the endogenous cannabinoid molecules (eCBs), anandamide (AEA) and 2-arachidonoylglycerol (2-AG), and the enzymes that lead to eCB release. Although CB1 was first discovered as the receptor mediating the psychoactive effects of the exogenous cannabinoid Δ9-tetrahydrocannabinol (THC) from Cannabis sativa, this sensing of eCBs by CB1 and CB2 is involved in a myriad of physiological processes, including the regulation of neuronal activity, inflammation and aging.

The Pankratz lab investigates how bacterial infection influences the neural physiology and feeding behavior of an organism, using Drosophila as a model. Drosophila are an ideal model for identifying novel sensors that detect bacteria and their metabolites, and how this message is signaled to the brain to effect an appropriate behavioral response, such as stopping food intake. On the one hand, Drosophila brains have a relative “numerical simplicity”. On the other, there are already unparalleled genetic tools available for monitoring and manipulating the activity of Drosophila neurons. The Pankratz lab has mapped the motor, neuroendocrine and sensory cells that mediate chemosensory and feeding responses. This has led to the identification of neurons in the brain that detect bitter compounds and make animals move away from a “bitter” food source. Interestingly, some of the neurons and neuroendocrine cells may be involved in detecting food-borne bacteria. Work is underway to determine the molecular nature of the receptors in these cells that sense bacteria and their metabolites.

Lipid metabolism and innate immune receptor signaling

We could recently demonstrate important roles for Ceramide synthases (CerSs) in lipid homeostasis[1,2,3]. However, CerSs is also involved in immunoregulatory circuits, which we now intend to analyze. Ceramide Synthases (CerS) comprise an enzyme family, which is involved in the production of ceramides, an important class of lipids. CerS enzymes are in the center of sphingolipid *de novo* synthesis. Many pathogenic changes are associated with changes in ceramide and/or sphingolipid levels. Recently, we could demonstrate that the single Drosophila ceramide synthase family member Schlank plays a key role in regulating both ceramide *de novo* synthesis and body fat regulation. All CerS family members share a lag1p motif, which is required for ceramide synthesis and many CerS have a homeodomain. In synthesis of ceramide, CerSs acylate a sphingoid long-chain base (LCB) with a fatty acyl-CoA of distinct length to generate (dihydro) ceramide, the backbone of all complex sphingolipids[4]. The number of CerS orthologs in a given species varies considerably. Whereas mammals express six CerSs, Drosophila encodes for only one CerS. The six mammalian CerSs show characteristic substrate specificities towards different acyl-CoAs. CerS5 and 6 share a common substrate specificity towards long chain CoAs (C14:0, C16:0, C18:0) generating C14:0 to C18:0-ceramide. As the murine CerS5 protein shares the highest sequence homology with Drosophila Schlank, we generated and analyzed CerS5 knockout mice. Our group discovered that C16:0-ceramide generated by ceramide synthase 5 (CerS5) promotes diet-induced obesity and insulin resistance[2]. C16:0-ceramide accumulates especially in white adipose tissue after high fat diet (HFD) feeding. However, CerS5 knockout mice are protected from HFD-induced C16:0-ceramide accumulation. Deficiency of CerS5 results in ameliorated HFD-induced weight gain. Moreover, CerS5 KO mice are also protected from HFD-induced obesity and insulin resistance[5]. Of note, CerS5 knockout mice also show decreased pro-inflammatory gene expression in adipose tissue.

Ceramide synthases in immune regulation

Metabolic disorders exhibit strong inflammatory characteristics, and, conversely, inflammation is associated with metabolic alterations[6]. Ceramide has been identified as a lipid damage-associated molecular pattern molecule activating the NLRP3 inflammasome in the context of obesity and aging. Previous studies used exogenous C6:0-ceramide to investigate ceramide induced inflammasome activation. The endogenous ceramide molecules and its metabolic synthesis route have not yet been identified. We have analyzed the sphingolipidome in isolated primary murine bone marrow derived macrophages in response to TLR (Toll-Like Receptor) activation and subsequent inflammasome activation. We found increased concentrations of ceramide and monohexosylceramide in response to treatment with the inflammasome activators ATP, nigericin, Imject Alum and palmitate, and also subsequent to prolonged TLR4 activation. These data indicate that sphingolipid homeostasis is disrupted in the context of inflammasome activation on a cellular level. Now, we plan to analyze the endogenous production of ceramides in the context of diet-induced obesity and its impact on the consequence of adipose tissue homeostasis and inflammation. All studies performed to date have failed to link endogenous *de novo* ceramide synthesis in the myeloid lineage with inflammasome activation in obesity. However, targeting ceramide synthesis in the myeloid lineage may only provide limited information, since macrophages do not only synthesize ceramides *de novo* but can also take up extracellular sphingolipids from other sources, such as membrane fragments, thus constitutively replenishing their intracellular pools.

In order to investigate the endogenous production of ceramides and its impact on adipose tissue homeostasis and inflammation, we have generated mice which are unable to synthesize C16:0-ceramide by crossing CerS5 and CerS6 deficient mice to delete both genes responsible for C16:0-ceramide production. These mice are born in sub-Mendelian ratios and suffer from increased lethality shortly after birth, but, CerS5/6 double deficient mice that survive the perinatal period continue their development without increased lethality. Surprisingly, these mice show no obvious phenotypes except lowered relative body weight and adipose tissue mass even without a HFD. We plan to reintroduce the ability to synthesize C16:0-ceramide by bone marrow transfer with hematopoietic stem cells from wild type and single (CerS5 or CerS6) deficient mice and analyze the consequences on adipose tissue biology under normal and HFD conditions. Using this approach, we can discriminate between effects of endogenous myeloid ceramide production and sphingolipids that are taken up by macrophages from non-myeloid cells. To analyze the acute and chronic inflammatory response to HFD in adipose tissue, we plan to develop a flow cytometry analysis of adipose tissue composition, which captures a wide range of cell types from the myeloid lineage, T and B cell populations, NK cells and adipocyte precursor cells. The effect of altered sphingolipid synthesis on the cellular adipose tissue composition has not yet been determined.

by Reinhard Bauer

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Metabolic Sensing and Nervous-Immune System Interactions

The endogenous cannabinoid system in neuroinflammation, metabolism and aging

The endogenous cannabinoid system (ECS) is a lipid signaling system that was originally discovered through studies on the chemistry of Cannabis sativa extracts and its major psychoactive constituent Δ9-tetrahydrocannabinol (THC). This constituent activates G-protein-coupled receptors, known as type 1 or type 2 cannabinoid receptors (CB1, CB2). CB1 is prominently expressed in the central nervous system, whereas CB2 is mainly present in the periphery. The endogenous ligands of CB1 and CB2, the so-called endocannabinoids (eCBs), are N-arachidonoylethanolamine (AEA) and 2-arachidonoylglycerol (2-AG). Both are metabolites of membrane phospholipids but are produced by independent biochemical pathways. 2-AG (and possibly also AEA) is a retrograde synaptic messenger that can prevent the development of excessive neuronal activity, a regulatory action of major importance. The ESC is not only important for brain physiology but also in many other tissues. It is thought to be a key regulator of tissue homeostasis.

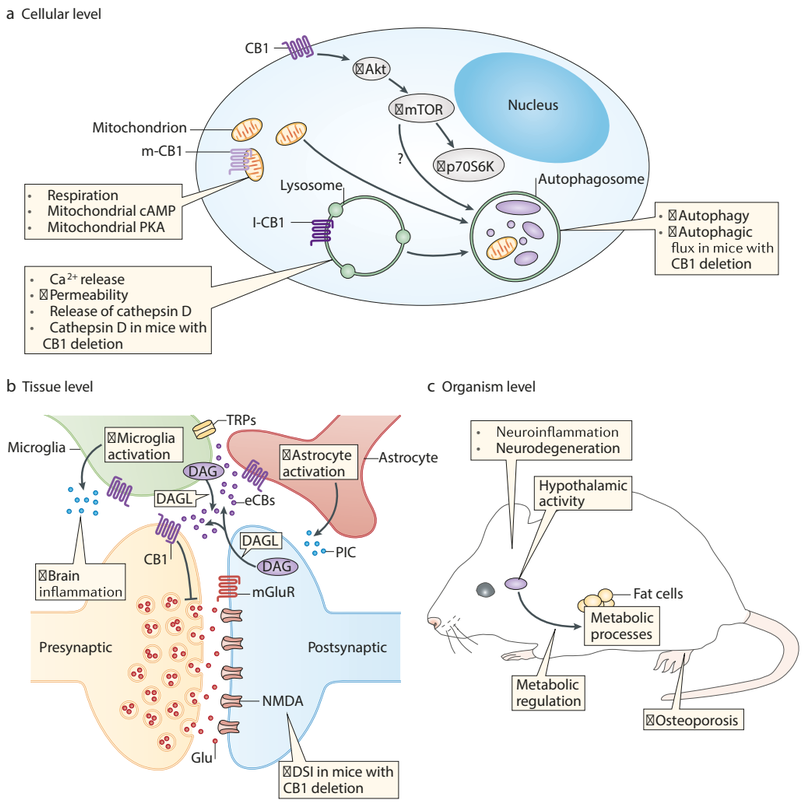

Figure 1 Age-related changes at different system levels and their regulation by endocannabinoid signaling. Ageing is accompanied by changes in cellular processes, impairments in the integration of cellular activities, and deficits in physiological functions. From Di Marzo, Stella, Zimmer Nature Reviews Neuroscience (2015).

The ECS is activated under inflammatory conditions, resulting in an increased production of eCBs and changes in the expression of cannabinoid receptors. Thus, expression of CB2 receptors is often low in healthy tissues, including the central nervous system, but strongly induced under pathological conditions such as neuropathic pain or in neurodegenerative disorders. This activation is important to counterbalance the inflammatory processes and necessary to restore tissue homeostasis (1). A reduced endocannabinoid tone can thus aggravate neuroinflammation and result in the development of neuropsychiatric symptoms (2-4). The CB2 receptor in particular seems to be at the nexus of inflammation and neuronal activity (5).

The activity of the ECS is not constant throughout life. It peaks during adolescence and slowly declines during aging (6). It has been suggested that the decreased endocannabinoid tone in older individuals contributes to some symptoms of the aging process (Di Marzo *et al.*, 2015; **Figure 1**). Indeed, animals with disrupted cannabinoid receptors show an accelerated appearance of indicators of brain aging such as neuronal loss and chronic neuroinflammation, bone loss and atrophy of the subdermal fat layer (7-9). Polymorphisms in the CB2 receptor in humans also result in an enhanced age-related bone loss and an increased vulnerability to develop psychiatric disorders. We are currently investigating the precise cellular and molecular mechanisms involved in the regulation of the endocannabinoid tone and are exploring the therapeutic potential of ECS modulators for age-related symptoms.

by Andreas Zimmer

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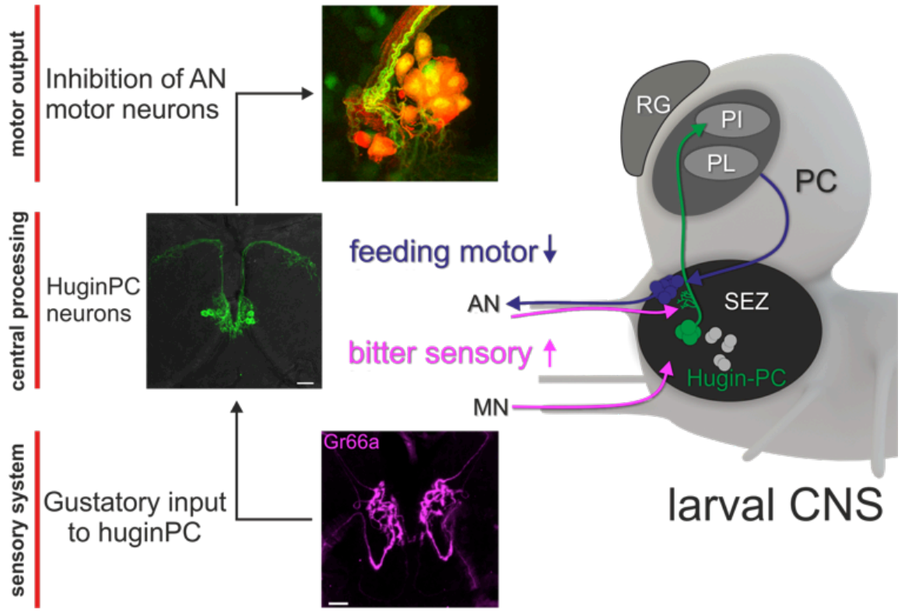

Figure 2 Identification of feeding motor neurons and taste circuits mediating bitter aversion

To understand the regulation of feeding behavior, motor neurons responsible for the control of food intake in Drosophila were identified (the AN motor neurons). In parallel, it could be shown that a cluster of eight interneurons in the brain (hugin-PC neurons) receive sensory input from bitter gustatory receptor neurons (gustatory receptor 66a). These interneurons express the neuropeptide hugin (homolog of mammalian neuromedin U) and their activity leads to a decrease of feeding motor patterns (adapted and modified from Hückesfeld *et al.*, 2015). Bitter taste is commonly associated with toxic or even pathogenic substances like bacteria, and current research is exploring whether bitter class of gustatory receptors can also sense bacteria/bacterial metabolites to alter feeding behavior.

Taste circuits mediating bitter and food aversion behavior

In addition to direct anti-microbial mechanisms, an organism uses many other strategies to detect and defend itself against harmful substances, such as avoidance (Medzhitov et al, 2012). Bitter taste is normally associated with detection of harmful substances and evokes an avoidance behavioral response, such as moving away from the source. Such reactions are mediated by specific sets of taste receptors that usually reside in the mouth region. However, recent finding indicate that bitter taste receptors are found internally as well, such as in the gut, and may be involved in detecting harmful bacteria, thus raising the possibility these taste receptors in the gut may also influence the brain to undergo aversion behavior in response to bacterial infection. Moreover, it also poses the question whether similar regions in the brain might be acted upon by perception of bitter taste from the internal and external environments.

Our lab has been addressing how bacteria influence behavior using Drosophila melanogaster. The brain, and its interaction with the periphery are complex: there are 10,000 neurons in fly larval brain, but this is still far few fewer than the 70,000,000 in the mouse and 85,000,000,000 in humans. In addition, there are powerful tools available to monitor and modulate Drosophila neuronal activity. Thus, Drosophila provides an ideal model system to study the molecular mechanisms underlying brain-gut-immune interactions. In previous work, we have investigated motor programs in the Drosophila brain for the suppression of food intake and induction of avoidance (Schoofs et al, 2014; Please see publication summary in ImmunoSensation Annual Report 2014.). In a recent publication (Hückesfeld et al, 2015), we were able to contribute fundamental information necessary to further studies on this subject by mapping the motor components that underlie feeding behavior. Using a photoactivatable GFP system, we characterized the motor neurons of the Drosophila larval feeding motor system, which allowed us to then make use of a genetically-encoded calcium indicator GCaMP3 to monitor the activity of the identified motor neurons and compare them to electrophysiological recordings. In addition, we were able to characterize the ipsi- and contralateral innervation patterns of the cibarial dilator musculature (CDM) by single Antennal Nerve (AN) motor neurons. Using a temperature-sensitive inhibitor in glutamatergic neurons, shibireTS, we could then block synaptic transmission from the AN to CDM, allowing us to modulate and inhibit feeding behavior. Moreover, we determined that the central pattern generator for larval feeding in Drosophila is located in the subesophageal zone. Altogether, our study will facilitate the identification of upstream cellular elements and CNS modulators that effect feeding activity. Current work focuses on interneurons expressing the neuropeptide hugin which, when activated, inhibit feeding and potentially process bitter taste information within the Drosophila larval brain. Further studies are currently addressing whether gustatory receptors are able to mediate the taste of bacteria in order to change feeding behavior.

Metabolic Sensing and Nervous-Immune System Interactions

Reference Publication

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Prof. Michael Pankratz, Sebastian Hückesfeld

Research Area D: Integration of Immune Sensory System Input on the Cellular and Subcellular Level

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Introduction

Over the last two years, important advances have been made in our understanding of the role of signal integration in the myeloid compartment. Together with colleagues from Singapore, Memphis and New York, Joachim L. Schultze has outlined a new multidimensional concept of macrophage ontogeny, activation and function.

New insights into the multidimensional concept of macrophage ontogeny, activation and function.

Ginhoux F*, **Schultze JL***, Murray PJ*, Ochando J*, Biswas SK*. Nat Immunol. 2016 Jan;17(1):34-40. published online 17 December 2015, doi: 10.1038/ni.3324. * all authors contributed equally

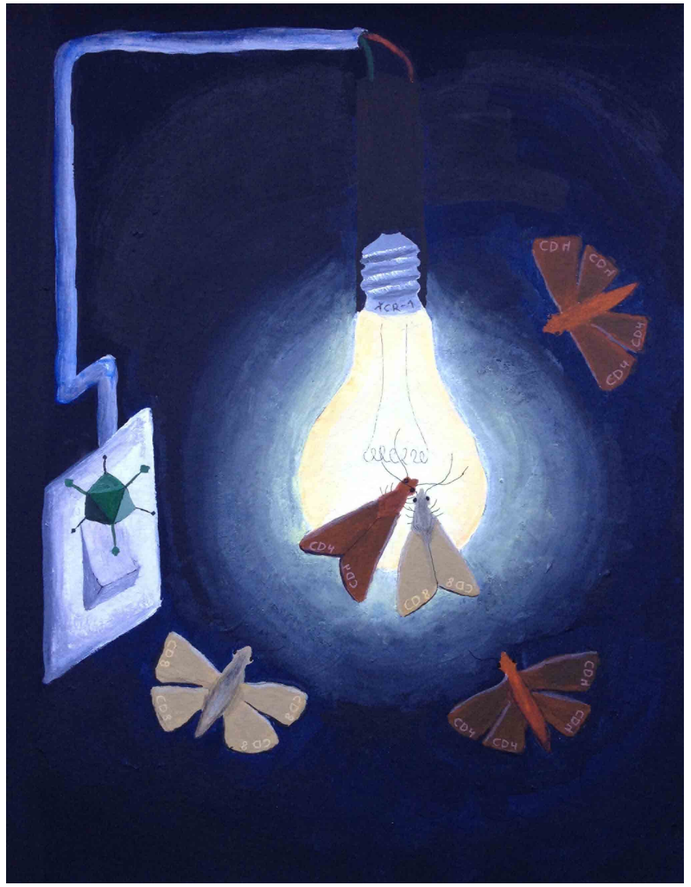
Macrophage biology has been the subject of intensive research for many years, yet we are only beginning to appreciate the intricacies of their development and function. Previous systematic classifications of macrophages were based on a polar dichotomy between inflammatory (M1) and anti-inflammatory (M2) states. However, new advances, such as transcriptome-based networks and epigenetic analysis, have compelled researchers to reconsider this dichotomous approach. In their study, Joachim L. Schultze and his colleagues propose a multidimensional, stimulus-oriented model of macrophage activation and phenotypic classification. One of the great benefits of such a system is that it can take into consideration both the ontogeny and spatiotemporal microenvironment of individual cells. However, deconvoluting complicated cell populations will also require expert use of cutting-edge technologies. Here, the authors provide a “roadmap” for the unbiased, high-resolution analysis of macrophage populations from specific tissues in homeostasis and during disease, including a variety of single-cell sequencing techniques and deep immunophenotypic assessment with cytometry by time-of-flight mass spectrometry (CyTOF).
Given the preeminent role macrophages play in tissue homeostasis, disease and repair, these cells are undoubtedly exceptionally attractive targets for immunomodulatory therapies. Thus, better understanding their heterogeneity will allow research to bypass the “roadblocks” posed by the oversimplifications of the polar activation model, to the benefit of both basic science and clinical therapeutic strategies.

The orchestration of cellular interactions during the activation of cytotoxic lymphocytes.

In order to mount a potent adaptive immune response, immune cells need to interact and communicate, an essential prerequisite for lymphocyte activation, proliferation and differentiation. In the context of humoral responses, CD4 T cells are first activated by dendritic cells (DC) in secondary lymphoid organs leading to an altered chemokine receptor expression pattern and subsequent migration to the T-cell/ B-cell border. Here, CD4 T cells interact with and provide help to antigen-presenting B cells, thereby initiating a complex order of events that is called the germinal center reaction (Victora et al. 2012).
It is well established that CD4 T cells do not only help B cells to produce neutralizing antibodies but also support the formation a functional memory T cell compartment from cytotoxic CD8 T cells (Castellino et al. 2006). Until recently, the spatiotemporal dynamics of those interactions, i.e. when and where CD4 T cells provide help, remained unclear. Importantly and in contrast to B cells, CD8 T cells cannot directly interact with CD4 T cells but instead do this using DCs as a proxy. In our recent work, we examined which subset of DC serves as the critical proxy for CD4 T cells to help CD8 T cells. Surprisingly, we found that, in context of viral infections, CD4 and CD8 T cells are initially activated separately by different DC. Once both lymphocyte subsets have become activated, they seek out XCR1 DC, which we have identified as the critical platform that brings CD4 and CD8 T cells together.
Altogether, our study has elucidated the spatitemporal dynamics of CD4 help to cytotoxic CD8 T cells and identified XCR1 DC as non-redundant DC subset that serves as a communication platform to mediate helper signals to CD8 T cells. Our current findings provide a framework for the design of novel vaccines that aim to elicit a potent cellular immune response as required for anti-tumor therapy and a variety of other applications.

by Wolfgang Kastenmüller

f.l.t.r.: Prof. Wolfgang Kastenmüller, Anna Brewitz, Karl Komander and Sarah Eickhoff (picture by K. Kastenmüller)



This oil on canvas image shows moth (CD4 and CD8) being attracted to light (XCR1) that is operated by a viral particle. It illustrates our central finding that XCR1 DC act as a critical platform that brings CD4 and CD8 T cells together to allow for the delivery of help. This “menage à trois” is important for optimal cytotoxic T cell responses in the context of viral infections. Image created by Dr. Marianne Janas.

Integration of Immune Sensory System Input on the Cellular and Subcellular Level

Reference Publication

Eickhoff S, Brewitz A, Gerner MY, Klauschen F, Komander K, Hemmi H, **Garbi N**, Kaisho T, Germain RN, **Kastenmuller W**. 2015. **Robust Anti-viral Immunity Requires Multiple Distinct T Cell-Dendritic Cell Interactions.** Cell 162: 1322-37.

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Research Area E: Consequences of Immune Sensing for Sterile Inflammation *in vivo*

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Introduction

Inflammation during infection is essential to pathogen clearance and tissue repair. However, immune sensors can also induce inflammation in response to “sterile conditions”, such as tissue damage, a process with the inherent potential of inducing chronic inflammatory states. Such chronic sterile inflammation drives the pathogenesis of many human diseases, including atherosclerosis, and Alzheimer’s disease. In addition, it participates in the etiology of many cancers by promoting neoangiogenesis and tumor stroma development. Within research area E, we aim to unravel the underlying mechanisms of sterile inflammation in order to better understand these illnesses and develop novel therapeutic strategies. In their previous work, Cluster members Michael Hölzel and Thomas Tüting could link pro-inflammatory signals, particularly TNF-α, to relapse in melanoma after adopted T cell therapy (Landsberg *et al.*, 2012). Immune-cell derived TNF-α induced reversible melanoma dedifferentiation and “phenotype switching”, allowing cells to stop expressing melanocytic antigens, escape recognition by cytotoxic T cells and promoting relapse. In their recent work (Riesenberg *et al.*, 2015), they have further elucidated this phenomenon by characterizing the molecular pathways through which TNF-α-induced dedifferentiation takes effect. Their study, which is summarized in this chapter (see page 38), describes a novel feed-forward mechanism by which TNF-α induces the activity of the c-Jun, which, in turn, suppresses the melanocytic lineage transcription factor *microphthalmia-associated transcription factor* (MITF). MITF is the central transcription factor of the “melanocyte phenotype” with its reduction leading to dedifferentiation. Thus, this new insight into MITF signaling provides new approaches to the targeted immunotherapy of melanoma.

Another important disease in the context of sterile inflammation is Alzheimer’s disease (AD), and the role of inflammation in AD has been the focus of the research of Cluster members Michael Heneka, Annett Halle and Eicke Latz for some time. In the Lancet Neurology review “Neuroinflammation in Alzheimer’s disease”, these researchers as well as other luminaries in the fields of inflammation and neurology, clearly state and support that neuroinflammation contributes to AD progression just as much as Aβ plaques. In this review, they provide insight into the cellular mechanisms of AD, the mediators and modulators of neuroinflammation, and new approaches to analyse, characterise and finally treat AD with the help of anti-inflammatory drugs. In “Innate immunity in Alzheimer’s disease”, a review published in Nature Immunology, Michael Heneka, Eicke Latz, and Douglas Golenbock, a member of the Scientific Advisory Board of the Cluster, also shed light on the role of innate immune sensing in AD. Here, they detail the newest advances in our understanding of the pathogenesis of Alzheimer’s disease and the causal role of inflammatory responses.

Altogether, Cluster research groups have continued to contribute to our understanding of chronic sterile inflammation in human disease, and their research has contributed to the identification of putative therapeutic targets for common inflammatory diseases and cancer.

Heneka M, [...] **Latz E**, **Halle A**, [...], Kummer P
Neuroinflammation in Alzheimer’s Disease
Lancet Neurology (2015)

Heneka M, Golenbock D & **Latz E**
Innate Immunity in Alzheimer’s Disease
Nature Immunology (2015)

Tumors are complex entities that are characterized by aberrant tissue architectures. In addition to tumor cells, non-malignant cell types such as stroma, endothelial and in particular immune cells critically contribute to tumorigenesis. Currently, our main research focus is malignant melanoma, an aggressive type of skin cancer that originates from pigment-producing melanocytes. New types of therapy including the use of oncogenic signaling inhibitors and immunotherapeutic approaches have remarkably improved the prognosis of patients with metastatic melanoma. In recent years and particularly within the first funding period of our Cluster of Excellence ImmunoSensation, we have discovered pivotal roles for reciprocal interactions between melanoma and immune cells in the tumor microenvironment[1-4]. Recruited immune cells evoke inflammatory responses in the tumor tissue and thereby critically change the biological behavior of the melanoma cells, a phenomenon also known as phenotypic plasticity[5]. Specifically, inflammatory signals such as the cytokine TNF-α are capable of rapidly inducing the dedifferentiation of melanoma cells, i.e. downregulation of the pigmentation gene program and acquisition of migratory neural crest progenitors properties. In our research, we have demonstrated how this inflammation-induced plasticity of melanoma cells can cause resistance to T cell immunotherapy[1] and promote metastatic progression in response to UV irradiation[3] of the skin. However, it has remained unclear how inflammatory signals are coordinated within melanoma cells to orchestrate phenotype switching in a pro-inflammatory microenvironment. It has also remained unexplained whether or to what degree different melanoma cell states instruct the immune cell composition of the tumor microenvironment. To address these important questions, we performed a series of transcriptomic and functional genomics approaches and found that the melanocytic lineage transcription factor MITF (*microphthalmia-associated transcription factor*) acts as potent repressor of inflammatory signaling in melanoma cells[4]. As MITF is the key regulator of the differentiation/pigmentation gene program, our finding clearly links dedifferentiation of melanoma cells to the acquisition of an inflammatory cell state with important implications for the tumor and immune cell cross-talk in the microenvironment[6]. Specifically, we identified a reciprocal antagonism between MITF and c-Jun, a stress-inducible transcription factor and component of the so-called AP-1 complex that promotes cytokine gene expression (**Figure 1**). We showed that pro-inflammatory cytokines like TNF-α instigate gradual suppression of MITF expression through c-Jun. However, MITF, itself, binds to the c-Jun regulatory genomic region, acting as a transcriptional repressor, and, therefore, its reduction increases c-Jun expression, which in turn amplifies cytokine expression. This feed-forward mechanism turns poor, peak-like transcriptional responses to TNF-α into progressive and persistent cytokine and chemokine induction by melanoma cells. This explains why inflammatory MITF^{low}/c-Jun^{high} syngeneic mouse melanomas strongly recruit myeloid immune cells into the tumor microenvironment, a phenotype that could be recapitulated in matched human patient specimens. Our findings suggest that myeloid cell-directed therapies may be useful for MITF^{low}/c-Jun^{high} melanomas to counteract their tumor growth-promoting and immunosuppressive functions. In summary, understanding the reciprocal interactions between melanoma and immune cells is important for the rational design of new clinical studies that evaluate immunotherapeutic approaches, as different immune cell compositions of the tumor microenvironment will require different combinatorial strategies.

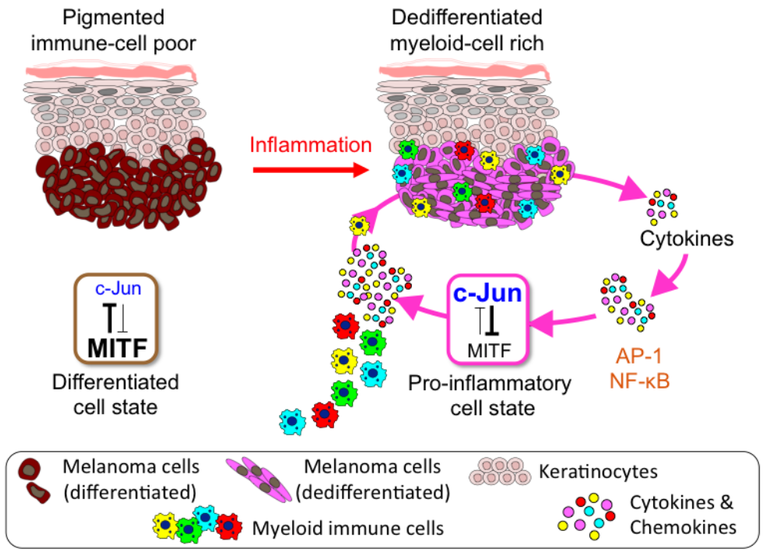

Figure 1 Summary of our findings showing how the antagonism between MITF and c-Jun controls cytokine responsiveness of melanoma cells and thereby the immune cell composition of the tumor microenvironment. Figure from Hölzel *et al.*, Trends in Immunology.

by Michael Hölzel

Reference Publication

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Consequences of Immune Sensing for Sterile Inflammation *in vivo*

Prof. Michael Hölzel

New Developments

Technical Platform:
Transgenic Service

Prof. Andreas Zimmer
Institute of Molecular Psychiatry
University of Bonn

Dr. David-Marian Otte
Haus für experimentelle Therapie (HET)
University of Bonn

New Members

Prof. Matthias Geyer, PhD
Prof. Christoph Wilhelm, PhD
Dr. Zeinab Abdullah, PhD
Dr. Annkristin Heine, MD
PD Dr. Marc Beyer, MD
Dr. Martin Schlee, PhD
Dr. Andreas Schlitzer, PhD
Prof. Sven Wehner, PhD

The Transgenic Service Core Facility

Genetically modified mice in biomedical research

The Transgenic Service Core Facility

Transgenic mouse models are a crucial tool for the understanding of gene and protein functions. At the Transgenic Service Core Facility (TGS), we have expert scientific and technical knowledge in the generation of genetically modified mice using a variety of different techniques. The core facility produces transgenic mice for a variety of applications. Thus, mice can be designed to overexpress proteins, to express tagged proteins or to express genes in a tissue or time-dependent manner using specific Cre mouse lines. Furthermore, we are currently establishing the generation of knockout or knockin mice using the CRISPR/Cas9-mediated gene editing approach. CRISPR/Cas9 is a new and very powerful tool for editing genomes which is based on the immune system of bacteria and archaea. The system consists of the Cas9 nuclease or nickase and a single guide RNA (sgRNA). sgRNA is approximately 20 nucleotides in length and escorts the Cas9 to the target sequence, where it generates double or single-stranded DNA breaks. When these breaks are repaired by the cell, mutations can occur in a process known as non-homologous end joining. To generate transgenic mice with CRISPR/Cas9, the sgRNA and Cas9 (RNA or protein) are injected into a zygote. Moreover, co-injection of a DNA fragment with homology to the sequences flanking the double-strand break can produce mutant alleles with inserts such as loxP sites. An illustration of the experimental procedure for the CRISPR/Cas9-mediated gene editing is shown in **Figure 1**.

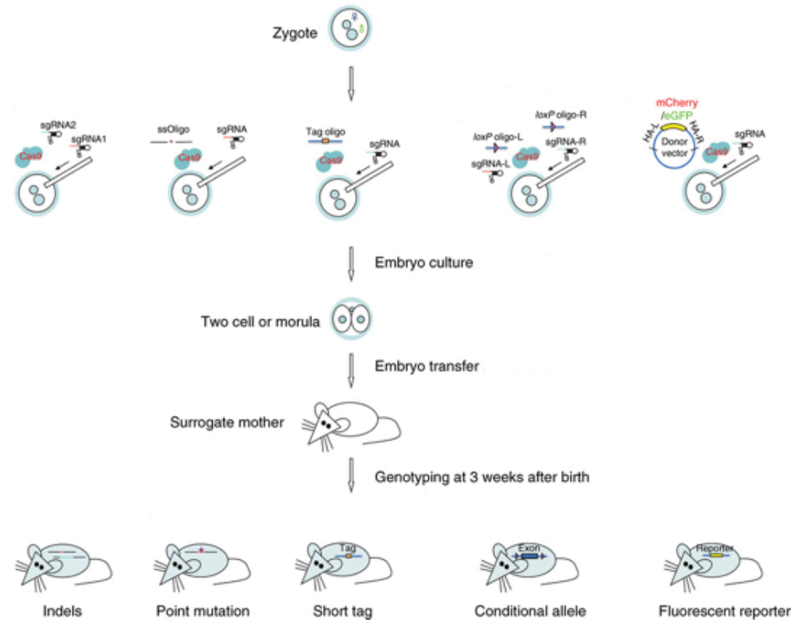

Figure 1 Schematic representation of gene editing options using the CRISPR/Cas9 system. Adapted from Yang *et al.*, 2014 Nature Protocols.

Ancillary services

The Transgenic Service Core Facility offers support, advice and counseling for non-specialists in recombinant techniques, mouse breeding, gene-targeting experiments and the regulatory approval of certain animal experiments. Additionally, we archive genetically modified mice as embryos frozen in liquid nitrogen and also have the capability of rederiving mouse strains from frozen stocks (sperm or 2-cell stages). On demand, we can also import mouse strains from other institutes and companies into our animal facility via embryo transfer.

Equipment and mice

The TGS is located in a modern, state-of-the-art animal facility at the clinical campus. We keep our genetically modified mice in a separate holding area with limited access to avoid contaminations. All mice are kept in internally ventilated cages (IVCs), and their well-being is monitored on a daily basis. Adjacent to the holding cages, we run the microinjection unit, which is equipped with specialized microscopes with micromanipulators to inject nucleic acids and proteins. Here, we also perform the embryo cryopreservation. Within the laboratory area of the facility, we run a molecular biology laboratory with state-of-the-art methods for the generation of plasmids and RNA.

Reference

Generating genetically modified mice using CRISPR/Cas-mediated genome engineering. Yang H, Wang H, Jaenisch R. Nat Protoc. 2014 Aug;9(8):1956-68. doi: 10.1038/nprot.2014.134. Epub 2014 Jul 24.

The Transgenic Service Core Facility

Prof. Andreas Zimmer
by Thomas Mauersberg

Dr. David-Marian Otte

New Developments - Member

We are proud to have been able to recruit eight outstanding scientists as new Cluster members over the last year: Christoph Wilhelm and Matthias Geyer, who were recruited for Cluster professorships in 2015, Annkristin Heine and Zeinab Abdullah, who established two new Cluster junior research groups, Marc Beyer, Martin Schlee, Andreas Schlitzer and Sven Wehner, who are new Cluster members. (For more information on all of these scientists and their research, please see below.) We would also like to congratulate Veit Hornung and Thomas Tüting for their appointments to the faculty of the Gene Center Munich and the University Hospital Magdeburg, respectively. Although no longer in Bonn, they will remain members of the Cluster for the remaining funding period. We wish them all the best and great success in their new positions.

Veit Hornung Immune Biochemistry, LMU Munich
by Johann Saba, UKB-Ukom

Thomas Tüting Dermatology, University Hospital Magdeburg
by Johann Saba, UKB-Ukom

Matthias Geyer
Cluster chair for Structural Immunology

The immune system is permanently defending the human body against a wide range of different pathogens. A common response to these harmful stimuli is inflammation. The cytosolic receptor protein NLRP3 is one member of the family of Nod-like receptors (NLRs) that sense inflammation. NLRP3 recognises pathogen-associated and danger-associated molecular patterns in immune cells. These molecular patterns lead to activation of NLRP3 and the formation of a high-molecular assembly, called the inflammasome. The Geyer lab is using a broad variety of molecular, biochemical and structural techniques to study the effects of activation and nucleotide binding in NLRs. The correlation of structure-function relationships in NLRP3 provides insights into the the molecular assembly of the inflammasome and yields key sites for the regulation and targeted inhibition of this immune receptor. In addition, the lab studies the regulation of transcription

Prof. Matthias Geyer
by Johann Saba, UKB-Ukom

kinases Cdk7 to Cdk13 and analyses how in eukaryotic cells the transition from transcription initiation to productive elongation is mediated. The malfunction of transcriptional elongation leads to multiple diseases as many forms of cancer, leukaemia, HIV infection and myocardial hypertrophy. Matthias Geyer is using a broad range of biochemical methods to determine protein-protein interactions as well as interactions to lipids, nucleic acids and small molecular ligands. The laboratory is mostly applying X-ray crystallography but also uses NMR spectroscopy and electron microscopy for the structural analysis of protein complexes.

Prof. Christoph Wilhelm
by Johann Saba, UKB-Ukom

Christoph Wilhelm
Cluster chair for Immunopathology

The mucosal immune system maintains the intestinal barrier in the face of a diverse array of challenges, including the uptake of nutrition, the interaction with commensal microbiota, and the defense against chronic helminth infections and bacterial or viral pathogens. An important component of this barrier defense mechanism are innate lymphoid cells (ILC). ILC are a recently discovered lymphocyte population, and their functions are not yet fully understood. Three distinct ILC subsets have been reported to reside in the gut, lung and skin. Type 1 ILC (ILC1) express the transcription factor T-bet, produce the cytokine interferon (IFN)-γ and are implicated in protecting against intracellular pathogens such as Toxoplasma gondii. ILC2 express the transcription factor GATA-3 and produce the cytokines IL-5, IL-9, IL-13 and amphiregulin. ILC3 are characterized by the expression of the transcription factor Roryt and the cytokine IL-22. Christoph Wilhelm found that vitamin A deficiency (a severe form of malnutrition) leads to the loss ILC3 producing IL-22 but a reciprocal increase in ILC2 secreting IL-13. This switch in ILC subsets resulted in impaired anti-bacterial immunity with increased gastro-intestinal pathology but also enhanced immunity to helminth infections, despite a previously reported dysfunction in T and B cells during vitamin A deficiency. The important implication of this discovery is that, unlike the adaptive immune response, ILC-mediated barrier immunity is able to adapt to malnutrition by switching to an ILC2-mediated response and maintaining the gut barrier. Such dynamic regulation by dietary components away from ILC3-dominated barrier immunity to ILC2-mediated responses and repair mechanisms could be beneficial in the context of chronic infections with tissue dwelling parasites. Both parameters, malnutrition and helmith infections, are largely absent in the westernized world, which is instead plagued by a multitude of inflammatory diseases. Hence investigating the protective function of ILC in the context of malnutrition could lead to new opportunities for manipulating their function for therapeutic purposes.

Zeinab Abdullah
Cluster junior research group

Chronic inflammation in the liver can affect the function of immune cells leading to impaired immune responses against new pathogenic infections. The research focus of the Abdullah lab is on the impact of chronic inflammation during liver fibrosis on the function of hepatic myeloid cells and how this effects the host immune response against microbial pathogens during chronic liver inflammation. You can find an interview with Zeinab Abdullah on page 51.

Dr. Zeinab Abdullah
by Johann Saba, UKB-Ukom

Annkristin Heine
Cluster junior research group/ Clinical translational group

Annkristin Heine and her group are working to improve cytotoxic T cell mediated anti-tumor responses by harnessing cross-presentation. Her clinical translational research group aims at integrating laboratory and clinical investigation of immunotherapies. An additional focus of the Heine group is on understanding the immunomodulatory effect of novel anti-cancer drugs and the molecular and cellular regulatory mechanisms of immunotherapeutic anti-tumor approaches. With their work, the Heine lab would like to help translate basic research investigations into effective cancer treatments. You can find an interview with Annkristin Heine on page 53.

Dr. Annkristin Heine
by Johann Saba, UKB-Ukom

New
Developments
-
Member

PD Dr. Marc Beyer
by Johann Saba, UKB-Ukom

Marc Beyer

Marc Beyer and his group are interested in deciphering the molecular mechanisms governing T cell and myeloid cell functionality using modern genomics or transcriptomics methods, such as genome editing via TALENs and CRISPR/Cas9 or single-cell transcriptomics, and investigating important epigenetic and posttranscriptional regulations. They employ human cells and model systems to study the transcriptional events necessary for the suppressive activity of regulatory T cells. The current focus of their research is the role of Satb1, a global chromatin organizer and transcription factor, in $CD4^+$ T cells and the prostaglandin E2 (PGE2) metabolizing enzyme hydroxyprostaglandin-dehydrogenase (HPGD) in regulatory T cells as well as signal integration resulting in immune cell exhaustion.

Dr. Martin Schlee
by Johann Saba, UKB-Ukom

Martin Schlee

Research in the Schlee lab focuses on understanding the mechanisms of immune recognition of nucleic acids. Anti-viral immune responses rely on nucleic acid receptors of the innate immune system that distinguish pathogenic nucleic acids from endogenous ones. This distinction is important because an insensitive recognition can lead to increased spread of infection while an excessive immune detection of nucleic acids leads to autoimmune diseases. Thus, recognition of nucleic acids relies on the unusual localization, structure or modifications of pathogenic nucleic acids which can be detected by distinct immune receptors. Moreover, to avoid immune recognition, some viruses incur RNA modifications shielding them from recognition by nucleic acid receptors. Recently, Martin Schlee and his group have helped identify and characterize the recognition motifs of cytosolic DNA and a cytosolic RNA receptors as well as viral modifications that can prevent their recognition. You can find an interview with Martin Schlee on page 56.

Andreas Schlitzer

The focus of Andreas Schlitzer‘s research is on the developmental processes leading to the many different forms of specialized dendritic cell and monocyte subsets during homeostasis and disease. The Schlitzer lab approaches this question from different angles with state-of-the-art techniques, such as single cell mRNA sequencing, multi-colour fish and advanced flow cytometry. Understanding the etiology of the different functional states of dendritic cells and monocytes will help to identify relevant steps in the onset and progression of many diseases.

Dr. Andreas Schlitzer
by Johann Saba, UKB-Ukom

New
Developments
-
Member

Sven Wehner

Sven Wehner and his lab aim to elucidate the underlying mechanisms of acute and chronic intestinal inflammatory disorders, with a focus on the (neuro)-immunological mechanisms that contribute to functional disturbances of the gastro-intestinal tract. Currently, the Wehner lab is investigating the neuroimmunology of chronic inflammatory bowel diseases and how intestinal inflammation is triggered. The identification of novel neuro-immune modulatory pathways is an important step towards understanding the mechanisms of acute and chronic intestinal inflammatory diseases.

Prof. Sven Wehner
by Johann Saba, UKB-Ukom

caesar

center of advanced
european studies
and research

DZNE
German Center for
Neurodegenerative Diseases
within the Helmholtz Association

Interviews

Dr. Zeinab Abdullah, PhD
Institute of Experimental Immunology
Medical Faculty
University of Bonn

Dr. Annkristin Heine, MD
Medical Clinic III for Hematology and Oncology
and Institute of Experimental Immunology
Medical Faculty
University of Bonn

Dr. Martin Schlee, PhD
Institute of Clinical Chemistry and Clinical Pharmacology
Medical Faculty
University of Bonn

Dr. Karin Pelka, PhD
Institute of Innate Immunity
Medical Faculty
University of Bonn

Philipp Schlegel
Molecular Brain Physiology and Behavior
Life and Medical Sciences Institute (LIMES)
University of Bonn

Lorenz Omran
Carl-von-Ossietzky Secondary School, Bonn

Interview with Dr. Zeinab Abdullah

Cluster Junior Research Group Leader

Dr. Zeinab Abdullah
Institute of Experimental Immunology

ImmunoSensation:
You are a new member of the ImmunoSensation Cluster and group leader of a junior research group. What is your main research focus and how is it related to the ImmunoSensation Cluster?

Zeinab Abdullah:
The overall goal of our research is to understand how chronic inflammation can affect the outcome of infection and immune cell function. Specifically, our research focuses on mechanistic studies to understand the impact of chronic liver inflammation on the function of hepatic myeloid cells and lymphocytes in host defence and adaptive immune responses against *de novo* infections.

ImmunoSensation:
You recently published a paper in Nature Immunology. Congratulations! Could you give us a short overview of the major findings?

Zeinab Abdullah:
Using *in vivo* mouse models for chronic viral infections that mirror the situation in humans and combined approaches, such as genomics, bioinformatics and cellular immunology, we have identified TNF signaling in T cells to be the underlying mechanism for T cell dysfunction during chronic viral infections. Moreover, we could show that TNFα promotes the induction of multiple inhibitory pathways related to T cell dysfunction including PD-1 and CTLA-4. Strikingly, *in vivo* blockade of TNF using the FDA approved drug infliximab re-invigorated CD4 and CTL function and reduced viral loads to background levels. These findings open new avenues for future therapeutic strategies for chronic viral inflammatory conditions with TNFα involvement.

ImmunoSensation:
The work was done in conjunction with another Cluster group (Prof. J. L. Schul-

tze). How does the ImmunoSensation Cluster foster scientific collaborations?

Zeinab Abdullah:
The ImmunoSensation Cluster fosters collaborations and scientific exchange by organising joint meetings and scientific retreats in which Cluster PIs can present their work, concepts and technologies, and these can greatly benefit other projects.

ImmunoSensation:
How do you think could we further enhance networking and collaborations within the Cluster?

Zeinab Abdullah:
In my opinion organizing joint seminars between different groups within the Cluster will enable the connection of expert knowledge from different fields, such as neuroscience, bioinformatics & genomics immunology and will make state-of-the-art techniques in these fields available to all scientists involved. All this facilitates fruitful interactions.

ImmunoSensation:
What kind of advice would you give young students who would like to pursue a career in science?

Zeinab Abdullah:
Follow your passion and desire to discover things. Discuss your findings. Don't be afraid to question things, and get input from others. Communicate and interact with others to move your work forward. Collaborate with others because you will not be able to do it all on your own.

ImmunoSensation:
What future innovation could have the highest impact on your research area?

Zeinab Abdullah:
In collaboration with the lab of Prof. JL Schultze we are conducing single cell RNA-sequencing to be able to dissect the interplay between intrinsic cellular processes and extrinsic stimuli such as the local environment or neighboring and their impact on the outcome of the immune responses in the liver.
We would like also to make use of the outstanding expertise of Prof. W. Kastenmüller in intravital microscopy to gain better insights on the interaction between different immune cell population in the liver.

Interview with Dr. Annkristin Heine

Cluster Junior Research Group Leader

Dr. Annkristin Heine
Medical Clinic III for Hematology and Oncology and Institute of Experimental Immunology

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ImmunoSensation:
You are leader of a newly established junior research group within the Cluster. What is the focus of your research and how does it relate to the Cluster?

Annkristin Heine:
The aim of my research is the identification of novel immunotherapeutic strategies with a special focus on dendritic cell licensing and chemokine receptor modulation. A further interest of mine is the monitoring of immune responses and immunomodulatory effects of novel anti-cancer drugs in patients and mice. Due to my work as a senior physician at the Medical Clinic III for Oncology, Hematology and Rheumatology, I am confronted with clinically relevant questions on a daily basis. It fascinates me to translate these problems into scientific questions and to try to identify the underlying mechanisms of what I observe clinically. This kind of research is very inspiring to me. I believe that my research focus fits well into the Cluster because it offers a direct translation from basic immunologic ideas into clinically meaningful therapeutic or diagnostic options. And, of course, most of these questions cannot be answered without the support of the excellent immunologists within the Cluster.

ImmunoSensation:
What advantages do you think will it have to work as a researcher within the ImmunoSensation Cluster?

Annkristin Heine:
The most striking advantage of being a Cluster member is having the opportunity to meet all of the great immunologists in the Cluster and establish new, productive collaborations. The Cluster provides a really unique and energetic atmosphere. In addition, becoming part of the Cluster also was a very important step in my scientific career. It has enabled me to

start my own junior research group and employ a very skilled technician and a motivated PhD student. Without them, I couldn't pursue my further career as a clinician scientist, which of course means a lot of time not only in the lab but also in the clinic. Moreover, the Cluster also offers lots of additional support for its members, such as interesting seminars, great talks and meetings with fascinating scientists.

ImmunoSensation:
You published successfully in several different journals in 2015. What was your personal highlight?

Annkristin Heine:
I don't think that it was one, single publication but rather the honor of receiving the Lisec-Artz-Preis for the best junior scientist in tumor immunotherapy in 2015 that was my personal highlight. Seeing my work acknowledged was very motivating.

ImmunoSensation:
Your independent junior research group is associated with the Medical Clinic III for Hematology and Oncology. What do you think are the advantages of working as a clinician and conducting research in the field of immunology?

Annkristin Heine:
I love to work as a clinician and do basic research at the same time. In the clinic, the direct contact with patients and their diseases brings up so many questions and riddles every day. In the laboratory, I have a great opportunity to discuss these important questions with basic scientists and develop new scientific approaches to solving these problems. As an example, I treat many patients who have received allogeneic bone marrow transplantation, many of whom develop severe graft-versus hose disease. The whole process of stem cell transplantation is a fanatastic example of "pure" translational immunology, and it is fascinating to understand the underlying mechanisms of this process and develop new scientific ideas based on careful, detailed observation of my patients.

ImmunoSensation:
How do you think could the Cluster encourage more women to stay in science?

Annkristin Heine:
There are great women who are fantastic scientists. However, even though they love their job, a lot of them would like to start a family one day. In my opinion, many women themselves, but also many supervisors, still believe that a woman cannot handle both a family and serious scientific research efficiently. However, there are many examples of successful women who manage both. For me, personally, it is quite a relief to know that there are attractive offers for child care or emergency babysitting support within the Cluster.

ImmunoSensation:
What future innovation could have the highest impact on your research area?

Annkristin Heine:
When I was first interested in tumor immunology and vaccination therapies in 2004, many researchers thought that tumor immunotherapy was obsolete. Talks or poster presentations dealing with tumor immunology at the big congresses were always during the last sessions when most attendees had already checked out. I am happy that I was not too discouraged at the time and stuck to the topic. Now, it is almost overwhelming how big the "hype" surrounding tumor immunology is. Innovations like checkpoint inhibitors have fully changed the lives of tumor patients. It is a really exciting time to be in the field!

Impression 7th DAMPs and HMGB1 Meeting 2015, Bonn

Interview with Dr. Martin Schlee

Bonfor Junior Research Group Leader

Dr. Martin Schlee
Institute of Clinical Chemistry and Clinical Pharmacology

ImmunoSensation:
What is your main research area, and how does it connect to the ImmunoSensation Cluster?

Martin Schlee:
The focus of my research is the antiviral immune response of the innate immune system. Here, nucleic acid receptors play a particularly central role, in that they initiate and control the antiviral immune response of the infected organism. The detection of pathogenic RNA/DNA by nucleic acid receptors is based on recognition of unusual RNA/DNA localization, structure and modifications, also known as pattern recognition motifs. The challenge for the innate immune system is that it should, ideally, detect pathogenic nucleic acids as sensitively as possible, yet also perform this task specifically, without false activation by the endogenous nucleic acids. A lack of sensitivity would favor the spread of infection. However, excessive immune detection of nucleic acids can lead to autoimmune diseases such as systemic lupus erythematosus (SLE). My research area immunorecognition of nucleic acid is thus directly in the focus of the Cluster of Excellence ImmunoSensation.

ImmunoSensation:
2015 has been a really successful year for you. What was your personal highlight 2015?

Martin Schlee:
We were able to publish two projects in high ranking international journals. However, these projects were started a couple of years ago. The paper about immunotolerance of endogenous RNA was submitted the first time in December 2010. There is always the question whether you should take a risk and follow a project with high potential until its acceptance in a high ranking journal or if you should publish a study while it is still not quite as mature in a journal with less attention to be on the safe side. In some cases, the last 10% of the work takes up 90% of the whole work time. I am really glad and grateful that we were lucky enough to see our hard work and patience rewarded.

ImmunoSensation:
What important findings led to your two publications in 2015?

Martin Schlee:
The projects are described in more detail as research highlights in this issue (see page 14). In brief, we found that a simple methylation, which the smallest known covalent modification in nature, at the 5'cap of mRNA prevents recognition of endogenous RNA by the innate immune receptor RIG-I, a master sensor of viral infections. RIG-I is responsible for the detection of and antiviral defense against most RNA viruses and initiates a signaling cascade towards either type I IFN induction or apoptosis after sensing of viral RNA. Since most cells have RIG-I, absence of this mRNA methylation would lead to a permanent immune stimulation of all tissues. Of note, this methylation has no direct impact of RNA dependent protein translation. Thus, at the moment, this methylation appears to have mainly immunomodulatory functions and serves as a marker of "self" RNA. Another aspect of this work was that RIG-I has a highly-conserved, bulky amino acid in its RNA binding pocket. This residue is dispensable for RIG-I activation but prevents binding of endogenous methylated mRNA. Amazingly, this self-tolerance mechanism in RIG-I is 100% conserved all the way back to simpler invertebrate species like sea anemones.
In another project, we identified a new DNA recognition motif for the cytosolic innate immune receptor cGAS. Like RIG-I, the DNA sensor cGAS is a master sensor of viruses, albeit of viruses that generate viral DNA during their replication cycle. This includes DNA viruses (e.g. Herpes-, Papilloma-, Polyoma- and Poxviridae) with a double stranded DNA genome but also viruses that produce single stranded DNA, like lentiviruses (e.g. HIV). We found that the sensitivity of cGAS activation by ssDNA is tremendously enhanced when structures are formed that bear G rich single-stranded DNA adjacent to short base paired structures. Such ssDNA structures are formed in the cytosol during the early phase of HIV replication.

ImmunoSensation:
You have been conducting research in Bonn since 2006. How did the ImmunoSensation Cluster influence your research?

Martin Schlee:
One of my PhD students, Ann Kristin Bruder, has been funded by the Cluster since 2015. She is one of the first authors of the manuscript on N1-2'O-Methylation and RIG-I. In addition, many of my collaborators are also Cluster members.

ImmunoSensation:
What do you think are emerging topics in the field of innate immunology?

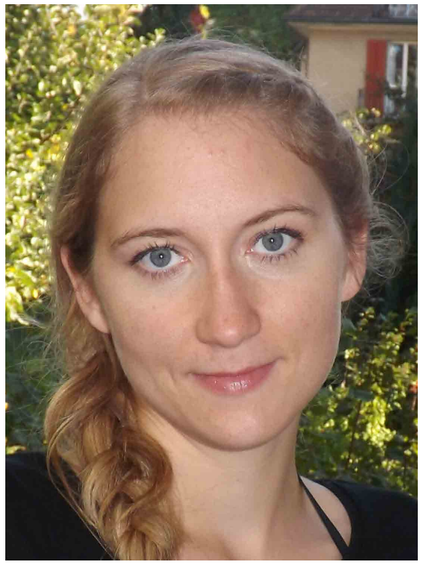
Martin Schlee:
I think that there is still a lot to learn about how viruses exploit and manipulate post-transcriptional regulation mechanisms of gene expression.

ImmunoSensation:
What new innovations have had the greatest impact on your research area?

Martin Schlee:
A new technique, CRISPR/Cas9, which exploits a pathway of the bacterial immune system that guides nucleases to specific target DNA sequences, has enormously facilitated genome editing even in tissue culture cells. This has tremendously accelerated the progress in our research area in the last years.

Interview with Dr. Karin Pelka

Former - PhD Student in the lab of Prof. Eicke Latz



Dr. Karin Pelka
Institute of Innate Immunity

ImmunoSensation:
Karin, you just finished your PhD in the lab of Prof. Eicke Latz. What brought you to Bonn?

Karin Pelka:
I came to Bonn to study Molecular Biomedicine. Back then, not so many universities offered a study program like this. To be honest, coming from the south of Germany, universities like Freiburg, Erlangen or Ulm were on the top of my list initially. However, during the interviews, Bonn just blew me away. I could feel the passion for science, the excitement for this new study program, and the great atmosphere among the students.

ImmunoSensation:
Tell us a bit more about your PhD. What was the main research focus of your doctoral thesis?

Karin Pelka:
I did my PhD under the supervision of Eicke Latz at the Institute of Innate Immunity. I was working on a protein called UNC93B1. UNC93B1 is a key-regulator of nucleic acid sensing Toll-like receptors (TLRs). These receptors are crucial in the host defense against viruses and bacteria by sensing pathogen-derived nucleic acids. Detection of host-derived nucleic acids, however, can lead to autoinflammatory diseases. During my PhD, we looked into the mechanisms by which UNC93B1 regulates these TLRs. We made some quite unexpected discoveries which we currently try to publish.

ImmunoSensation:
What do you think were the benefits of conducting your PhD in the ImmunoSensation Cluster?

Karin Pelka:
I was already quite far in my PhD when the Cluster graduate program (IITB) with all the workshops and seminars started. I would definitvely advise future generations to make use of these offers. For me, the benefits were mostly to be among highly motivated people who work on a wide variety of scientific aspects and have complementary expertise. I was also involved in organizing a Cluster Girls' Day and a Cluster Night of Science which was fun.

ImmunoSensation:
Where are you heading now after finishing your PhD?

Karin Pelka:
To Boston. I am very excited to do my postdoc in Nir Hacohen's lab at the Broad Institute.

ImmunoSensation:
If you had unlimited research funds, what scientific problem would you tackle?

Karin Pelka:
The amount of funding actually would not change what I want to work on. I am fascinated by the complex regulation of immune responses. The fact that the immune system plays a crucial role in many diseases such as infectious diseases, autoimmunity, metabolic diseases, and cancer, makes it a very relevant and versatile research topic. Having unlimited research funds would mostly change how I perform research. In many ways, science feels a little bit like gambling to me. Depending on the available resources, you have to find the right strategy. With little money, you have to divide your big goal into very small and rather safe steps, use affordable technologies, and avoid spending money on risky projects. Unlimited research funds enable you to use high-end technologies, take on projects on any scale, check out creative shortcuts, be bold, share openly, and go for high-risk high-gain projects. If done in a clever way, this can of course greatly accelerate your research. I was very lucky to do my PhD with Eicke Latz where I got some sense for this second approach towards science. Now, I am very excited and slightly scared to join the Broad Institute where this approach is taken to another level.

Interview with Philipp Schlegel

PhD student in the lab of Prof. Michael Pankratz

ImmunoSensation:
You conducted your PhD thesis at the Life and Medical Sciences Institute. What brought you to Bonn in the first place?

Philipp Schlegel:
I did my Diploma in biology in Bonn and by the time I finished my thesis in the lab of Prof. Pankratz working with the lab had been a lot of fun but the projects between me and my colleagues were just about to really take off. Fortunately, Prof. Pankratz offered me a PhD position and so I decided to stay and follow up on the findings of my diploma thesis.

ImmunoSensation:
What was the main research focus of

Philipp Schlegel
Department for Molecular Brain Physiology and Behavior (LIMES)

your PhD in Prof. Pankratz' lab?

Philipp Schlegel:
The lab collaborates with scientists from the U.S. to generate a map of the wiring of the larval Drosophila brain, a so-called connectome, based on EM-sections of an entire nervous system. I reconstructed and analyzed the circuitry for neurons that produce a conserved neuropeptide which is involved in the regulation of a range of behaviors, including pathogen avoidance.

ImmunoSensation:
What do you think were the benefits of conducting your PhD in the ImmunoSensation Cluster?

Philipp Schlegel:
I am a neurobiologist at heart. Consequently, it comes natural to me to approach the topic of brain-immune interactions from the brain's point of view but it also means that I am less familiar with immunology. This is why the various workshops, meetings, etc. organized by the ImmunoSensation Cluster have been tremendously helpful to get in touch with experts in this field and discuss how the brain integrates immune-relevant signals to initiate an appropriate behavioral response.

ImmunoSensation:
Where are you heading now after finishing your PhD?

Philipp Schlegel:
I will be joining a team of scientists in Cambridge who want to tackle the adult Drosophila brain similar to what I did for my PhD in the larva. For starters, I will train and supervise a team of people that are going reconstruct the connectome of the adult fly. With these data we are going to investigate the circuitry that is involved in learning, memory and innate behavior.

ImmunoSensation:
If you had unlimited research funds, what scientific problem would you tackle?

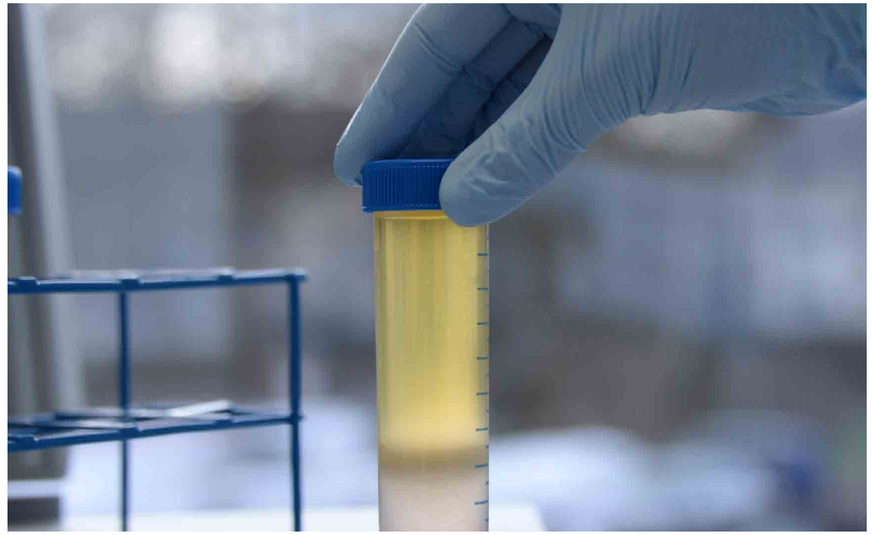
Philipp Schlegel:
There is an infinite number of scientific problems that are in dire need of scientific attention. But instead of focusing on a single one of them, I'd rather invest into modernizing the way scientific research is conducted: create incentives to publish everything (including negative results) as open-source and open-access, improve the reviewing process, establish more meaningful metrics than the journal impact factor - to name but a few.

Interview with Lorenz Omran

Carl-von-Ossietzky Secondary School Bonn

ImmunoSensation:
You decided to pursue research for your eleventh-grade term paper (Facharbeit) in the lab of Prof. Hartmann. How did you get the idea to work in a real lab, and why did you pick the topic of immunology and Prof. Hartmann's lab?

Lorenz Omran:
Chemistry is one of my main subjects, and it's also my favorite one. That's why I chose to write my "Facharbeit" in a scientific field. However, I really wanted to do something new and hands-on rather than summarizing theoretical things that have already been discovered. And, since it turned out that such a project would be impossible at my school, I started looking for an alternative. I'm also interested in medicine and had already done an internship in a large medical device company (Medtronic) in Maastricht. I found the idea of combining medicine and chemistry very attractive. And, since I live close to the University Hospital in Bonn, I decided to try and send an application to the Institute of Clinical Chemistry and Clinical Pharmacology. Prof. Hartmann was so kind as to offer me the chance to take part in one of his team's projects. The project sounded very interesting, so I gladly accepted his offer and started reading about this particular area of immunology, the STING/cGAMP pathway.



Lorenz Omran working in the lab

ImmunoSensation:
How did you like working in an immunology lab and what about it in particular?

Lorenz Omran:
At first, it was quite challenging and sometimes a bit frustrating to learn how limited my school knowlege in biology really was, and the English scientific terminology didn't make it easier, but with the support of Prof. Hartmann and Dr. Herzner, one of his team members --and also motivation from my girlfriend and my family-- I started to make progress in my understanding of the subject and found that it got more and more interesting. The most exciting thing was that I was allowed to take part in real scientific research and that the team, who made me feel as if I really was part of it, were the first to ever discover these things, which I think is a great feeling, even if it's just a small part in the overall research.

ImmunoSensation:
What was the topic of your "Facharbeit" and what results could you find?

Lorenz Omran:
The title of my term paper was: "Einfluss oxidativer Veränderung von DNA-Oligonukleotiden auf die Immunantwort". In a nutshell: DNA in cells belongs mainly in the nucleus, but definitely not into the cytosol. Therefore, whenever a cell detects DNA in its cytosol, it is usually a sign for a viral infection and is fought by an immune response. The goal of the study was to come up with a DNA-oligonucleotide which stimulates the immune response and thereby strengthens the autoimmune system. Recent research showed that the y-stem structure of the DNA-oligos in the

early HIV virus was highly stimulatory. We tested seven different modifications, using human white blood cells and measured the interferon levels to determine the immune response. As a conclusion, one can say: different modifications of the oligos differed in their stimulating effects, depending on the concentration of the oligos in the cells.

ImmunoSensation:
You are finishing school now. What are your plans afterwards?

Lorenz Omran:
Luckily, I still have one more year at school, as I don't yet know exactly what I want to do afterwards. However, I know for sure that I want to work in the area of natural sciences.

ImmunoSensation:
After experiencing the working atmosphere in an immunology lab, can you imagine yourself being a scientist?

Lorenz Omran:
I definitely find the idea of working in a scientific lab very attractive.

Impressions Cluster Science Days 2015


universitätbonn
LIMES
Life & Medical Sciences Institute

Cluster Coordination Report

Events

The Cluster Coordination Office is in charge of the implementation and financial administration of Cluster events, the coordination of the graduate program, gender and family support and public relations. Within this section, we would like to give an overview of the organized events for students and Cluster scientists.

ImmunoSensation hosts 7th International Symposium on DAMPs and HMGB1

September 10-12, 2015

7th International DAMPs and HMGB1 Symposium

Background

Damage (or danger)-associated molecular pattern molecules, or DAMPs, are endogenous molecules, such as proteins and nucleic acids and metabolites, which can function as pro-inflammatory danger signals upon changing their subcellular localization or after their release into the extracellular space. As endogenous stress signals, DAMP release and sensing are pivotal to the initiation and perpetuation of the inflammatory response, both in sterile inflammation and in the host defense against pathogens. High-mobility group box 1 (HMGB1) is a prototypical DAMP. Inside the cell, HMGB1 is a nuclear protein that participates in chromatin remodeling and transcriptional regulation. However, upon a variety of physico-chemical or biological insults, HMGB1 is released into the extracellular space, where it triggers inflammatory responses via its receptors TLR2, TLR4 or RAGE. Via these pathways, HMGB1 can induce dendritic cell maturation, the production of pro-inflammatory cytokines, and the upregulation of the expression of cell adhesion molecules, processes vital to the immune response. However, excessive or inappropriate HMGB1 release has also been implicated in the sterile inflammatory processes associated with many human diseases, including ischemia, immune disorders, neurodegenerative diseases, metabolic disorders, and cancer.

The International Symposium on DAMPs and HMGB1 was established by the international HMGB1 group of scientists with the goal of better understanding the mechanisms of DAMP and HMGB1 biology and their implications for disease. This year was the first time that Bonn was selected to host the symposium, and all of the local organizers, including Gunther Hartmann (local chair), Michael Hoch, Waldemar Kolanus, Christian Kurts, Eicke Latz and Andreas Zimmer, are members of ImmunoSensation. We are very proud that the Cluster was given the opportunity to host such an important meeting and that we could welcome so many internationally renowned experts in innate immunity to Bonn, including Charles Dinarello, Tak Mak and Kevin Tracey, who all agreed to be keynote speakers.

7th International Symposium on DAMPs and HMGB1

Day 1

On Thursday, September 10, Gunther Hartmann officially opened the meeting and welcomed the participants to the Biomedical Center at Bonn Venusberg. The first session then began with Ulf Andersson's keynote address, which provided fascinating insight into current research and open questions about HMGB1 biology. Afterwards, the session then continued with a variety of talks on the structure and function of HMGB1 with a focus on its role in health and disease. The first day concluded with the speakers' dinner cruise along the river Rhine. While enjoying the beautiful sunset, Marco Bianchi was awarded the Angelika Bierhaus Memorial Award for his outstanding contributions to our understanding of the biology of HMGB1. Prof. Dr. Angelika Bierhaus, University of Heidelberg, devoted her life to understanding the molecular mechanisms of chronic diseases and discovered that many of these were associated with the Receptor for Advanced Glycation Endproducts (RAGE), which is a major receptor for HMGB1 and other DAMPs. The evening offered not only a spectacular view of the Siebengebirge and fantastic food but also lively scientific discussions and great expectations for the two days to come.

Day 2

Friday, September 11, was all about DAMPs, their immunobiology, and their clinical implications in sterile inflammation and tumor immunology. The first session of the day was chaired by the HMGB1 expert Helena Erlandsson and focused on DAMPs and sterile inflammation. The session began with an excellent keynote address from Charles Dinarello, who offered a new perspective on the role of IL-1α as a DAMP in conjunction with the inflammatory response to HMGB1. The next session was on the immunobiology of DAMPs and was chaired by the eminent TLR researcher, Hermann Wagner. The keynote address was held by Tak Mak, who described HMGB1 as a "double-edged sword" in inflammation. The final session of the day focused on DAMPs in tumor immunology and was chaired by the noted thoracic oncologist, Michele Carbone. The keynote address by Michael Lotze was "HMGB1, Cancer and Immunity: RAGE, rage against the dying of the light".
After the sessions, a podium discussion on HMGB1 and IL-1 sought a consensus on how those two related DAMPs molecules impact the immunopathogenesis of acute and inflammatory diseases. In the evening, the official conference dinner and party was hosted at the Godesburg, Bad Godesberg, Bonn, leaving the participants with colourful impressions of Bonn and its surroundings.

Day 3

On the final day of the 7th International Symposium on DAMPs and HMGB1, the meeting moved to an extraordinary location: the "Wasserwerk", the former plenary hall of the German Federal Parliament. ImmunoSensation invited all participants to discuss clinical and therapeutic implications of DAMPs and HMGB1. This historic location was also used to confer the poster awards.

Poster awards

Selina K. Fassl, Institute of Immunology, University of Münster, Germany for the poster entitled "Pyrin and PSTPIP1, mutated in FMF, PAPA-, and PAMI syndrome, are involved in the hypersecretion of DAMPs MRP8/14"
Ravikumar Sitapara, Department of Pharmaceutical Sciences, College of Pharmacy and Health Sciences, St. John's University, Queens, NY, USA for the poster entitled "The α7 nicotinic acetylcholine receptor agonist GTS-21 attenuates hyperoxiainduced acute lung injury by decreasing hyperacetylation and the release of nuclear HMGB1"
Thomas Zillinger, Institute of Clinical Chemistry and Clinical Pharmacology, University Hospital of Bonn, Germany for the poster entitled "ADAR1 controls recognition of self-RNA by MDA5"

This wrapped up the official part of the meeting, but the social activities continued. The Cluster organized bag lunches and bikes for a long tour along the river Rhine. The tour ended at the Chairman's private house, where everyone was invited to a barbecue garden party.
The next International Symposium on DAMPs and HMGB1 will take place in Liverpool in 2017, and we are sure that many Cluster members will attend.

7th International Symposium on DAMPs and HMGB1

Bike Tour on Day 3

3rd Cluster Science Days

November 2- 3, 2015

Organization Committee

3rd Cluster Science Days

The Cluster Science Days have been held every year since 2013. This intramural scientific meeting is firmly focused on young scientists and their research, offering a forum for young investigators to network and exchange ideas. This year, the Steering Committee decided to entrust several PhD students and young postdocs with the organization of the 3rd Cluster Science Days, which was a great success.
This year 85 abstracts were submitted by young scientists, from which 25 talks and 23 speed talks were selected. These talks were organized according to the research areas within the Cluster, which, in itself, was a daunting task. Over the years the Cluster research areas have "grown together" and many research projects incorporated aspects of several areas at once. Nonetheless, the talks gave an excellent overview of the current research topics within the Cluster and were also complemented by keynote addresses and poster sessions for each area. One of the new features implemented by the organizing team was a technical session to introduce the work of the Cluster platforms and to offer a close-up look at new technical developments within the labs of the Cluster groups. The sessions helped to enhance exchange of technical knowledge and technical expertise between all participating Cluster scientists. Another new feature were the evening events implemented to give PhD students the possibility to discuss scientific career options. Internationally recruited group leaders and professors from the Cluster and some of the Cluster's alumni were invited for round table discussions and an informal get-together. IITB students from all groups used the opportunity to discuss how to be successful in the highly competitive world of science. As usual, the posters and talks from the participants were of excellent quality, and it was difficult to decide who should win the poster and talk prizes. This year we wanted to involve the audience more in the decision process, and the poster prizes were democratically chosen with all participants voting.

3rd Cluster Science Days

November 2- 3, 2015

Best Talk:

Donald Guu
'Space and Time in Leukocyte Migration'

Best Poster:

Jesuthas Ajendra
'NOD2$^{-/-}$ mice are more susceptible to infection with *Litomosoides sogmodontis* due to an impaired early protective immune response'

Lorenz Fülle
'Inflammation dependent upregulation of CCL17 in hippocampal neurons and its influence on neuro-immune interactions'

Monika Plescher
'Establishing single-cell transcriptomic analysis of microglia in Alzheimer's disease'

f.l.t.r.: Prof. Gunther Hartmann, Lorenz Fülle, Monika Plescher, Jesuthas Ajendra, Dr. Donald Guu and Prof. Waldemar Kolanus

Joint ASI-DGfI Workshop Canberra (AUS)

December 3- 4, 2015

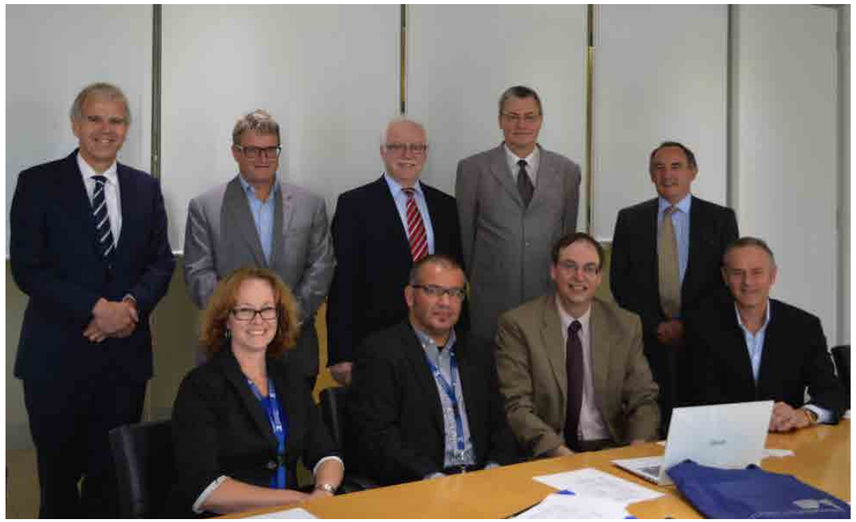
Delegates of the first joint ASI-DGfI Workshop in Canberra, Australia

The first joint workshop of the German and the Australasian Societies for Immunology was held in Canberra, Australia under the joint organization of Christian Kurts and Catherine Drescher (Immuno-Sensation) and Sammy Bedoui (University of Melbourne) and Anselm Enders (Australian National University). The workshop was associated with the 45th Annual Scientific Meeting of the ASI, held from Nov 29-Dec 3, 2015 in Canberra. The second ASI-DGfI meeting will be held in conjunction with the annual DGfI meeting in 2017 in Erlangen.
The satellite ASI-DGfI workshop was the perfect follow-up to a very successful ASI meeting, fostering new German-Australasian scientific collaborations, and full of inspiring discussions. In particular, the application of stochastic modeling has led to a re-evaluation of “old, accepted truths” in the field, such as the asymmetric cell division model.
The focus of the joint workshop was “immune regulation in infections and immune mediated diseases”, with delegations of outstanding German and Australasian immunologists in attendance. A full meeting report can be found in the European Journal of Immunology, Volume 46, Issue 2, pages 265–268, February 2016.

Establishment of the DFG-funded IRTG “Bo&MeRanG“ with Melbourne, Australia

by Prof. Christian Kurts

Cluster scientists have teamed up with leading immunologists from Melbourne University, the premier University of Australia, to establish an International Research Training Group (IRTG) for PhD students. Participants will receive instruction from scientists from both universities. The common research focus within the program is the acquired immune defense against infections, which ideally complements the expertise of “ImmunoSensation”. The DFG will fund this new joint international graduate college “Bo&MeRanG” at the Universities of Bonn, and Melbourne for 4.5 years starting in April 2016. Bo&MeRanG stands for **Bo**nn & **Me**lbourne **R**esearch **an**d **G**raduate Training group. The topic synergizes with the Cluster of Excellence “ImmunoSensation”, which focuses on the innate immune response in sterile inflammation. The Cluster boasts great expertise on immune sensors and molecular and transcriptional immune regulation, whereas Melbourne is a leading international center in the field of immunological defense against infection and in research on rare immune cell populations. Combining these areas of expertise will allow the scientists to perform more effective research and broaden the teaching curriculum in immunology and infectiology. The new graduate school



Signing the joint teaching agreement in Melbourne, February 2015.

Standing from left to right: Geoff McColl (Head of Melbourne Medical School), Dick Strugnell (Pro-Vice Chancellor for Graduate Research, University of Melbourne), Max Baur (former Dean of the Bonn Medical Faculty), Nicolas Wernert (President of the Senate of the University of Bonn, now Dean of the Bonn Medical Faculty), Jim McCLuskey (Vice Chancellor, University of Melbourne).

Seated from left to right: Liz Hartland (Head of the Dept. of Microbiology & Immunology, Melbourne), Sammy Bedoui (Bo&MeRanG Co-Speaker, Melbourne), Christian Kurts (Bo&MeRanG speaker. Bonn), Ian van Driel (Associate Dean, Melbourne School of Graduate Research)

will be located at the ImmunoSensation headquarters at the Bonner Institutes of Immunology and Infectious Diseases (BI[3]) in the planned BMZ II research building to be erected from 2017 on the Venusberg. According to Prof. Christian Kurts, the speaker of the graduate school: “The synergistic combination of the expertise of outstanding scientists from the Universities of Bonn and Melbourne is a great advantage in the international competition for outstanding students and excellence in research, because it will give our students an even better education and research opportunities.” He knows Melbourne and the immunologists there well, because he received his own immunological training at the Walter and Eliza Hall Institute in Melbourne almost 20 years ago. The students will spend about 2/3 of their PhD studies in one of the 15 participating immunological laboratories in Bonn and one third, at least 1 year, in a partner laboratory in Melbourne. Scientists have designed “tandem research projects”, which cover topics that require the expertise of the both laboratories. The students are trained in two disciplines and benefit from the strengths of two research universities and of their coordinated teaching programs. A similar PhD program will be set up at the University of Melbourne, led by the German-born scientist Sammy Bedoui, who trained at the Hannover Medical School. The students from Melbourne will come to Bonn while the German students are doing research Downunder. According to Dr. Bedoui, “We are extremely pleased that our long-standing scientific cooperation can be supported by this international-class doctoral program, thanks to the funding by the DFG and the Universities of Bonn and Melbourne. This program of scientific exchange between Bonn and Melbourne will not only advance science, but will also provide the graduate students involved with the opportunity to live and work in the partner country.” After successful completion of the program, students will receive a joint PhD (Doctor of Philosophy) title from both universities. The common-research theme of the new Research Training Group are ‘myeloid antigen presenting cells’ (APC) and their control over T lymphocytes. This interaction is essential to the adaptive immune response and is required for both vaccination and pathogen defense. In addition, dysregulation of this process has been implicated in many autoimmune and autoinflammatory diseases, including type I diabetes, atherosclerosis, kidney and liver infections and multiple sclerosis. Thus, the myeloid APC / T-cell interaction is of great interest for both basic science and the development of therapeutic approaches. The more than 30 scientists involved in “Bo&MeRanG” have greatly contributed to our knowledge in this field In early spring 2016, the first cohort of 15 German and 15 Australian students will be recruited and start to work on their tandem projects. While some will start in Bonn, others will depart immediately for the other side of the globe. Regardless, true to the name of the program, they will all leave Bonn, go to Australia and come back again --hopefully taking new knowledge and great memories of Australia back with them!

Pro-Vice Chancellor Dick Strugnell, Melbourne, and Dean Nicolas Wernert, Bonn

The Peter Doherty Institute for Infection and Immunity at the University of Melbourne

International Immunology Training Program Bonn (IITB)

The International Immunology Training Program Bonn (IITB) continued its great range of scientific and social activities to offer in 2015. In addition, we were also able to expand our program in 2015. The IITB now also offers travel grants, the students’ network day and scholarships for the DGfI Autumn School.

1. IITB Travel Grants

IITB students can now apply for travel fellowships granted twice a year on a competitive basis. These fellowships are offered to promote scientific exchange and give IITB students the opportunity to present their Cluster-related research to an international audience. We now offer three different forms of travel grants:

Travel grant 1: Scientific exchange
IITB members working in a Cluster laboratory can apply for travel grant to visit a lab abroad to learn a new method or technique that is important to their project.

Travel grant 2: Meetings and Seminars
IITB members who wish to participate in an international meeting to present their data and broaden their scientific network can apply for this type of Cluster travel grant. The Cluster offers financial support to cover travel costs and participation fees.

Travel grant 3: Blog support
The Cluster also offers support to non-IITB members like Master’s students or young postdocs from Cluster groups. This travel grant is used to promote other young Cluster scientists outside of the IITB and encourage them to participate in the newly established ImmunoSensation Blog. Cluster scientists can apply for financial assistance for research visits abroad or an international meetings if they are willing to write about their experiences there afterwards for the ImmunoSensation Blog.The first round of travel fellowships was granted in October 2015 to the following IITB members:

Sarah Eickhoff, Kastenmueller lab, CFCD Annual Meeting: DC ontogeny, functional specialization and targeting, Paris, France

Alena Grebe, Latz lab, Toll 2015 Targeting of Innate Immunity, Marbella, Spain

Christoph Heuser, Kurts lab, CD1-MR1, Mantra Lorne, Australia

Florian Hoss, Latz lab, Toll 2015 Targeting of Innate Immunity, Marbella, Spain

Katarzyna Jobin, Kurts lab, Kidney Week, San Diego, USA

Patricia Korir, Hoerauf lab, European congress of Immunology, Vienna, Austria

Bettina Nadorp, Soreq lab (Israel), ImmunoSensation workshop “Data Analysis” Bonn, Germany

Stefanie Riesenberg, Hoelzel lab, Society for Melanoma Research, San Francisco, USA

Juan F. Rodriguez Alcazar, Latz lab, Toll 2015 Targeting of Innate Immunity, Marbella, Spain

Shani Shenhar-Tsarfaty, Soreq lab (Israel), ImmunoSensation workshop “Data Analysis” Bonn, Germany

If you want to read about the experiences of the travel grant recipients, you can visit the ImmunoSensation Blog on http://www.immunosensation.de/blog/. We look forward to continuing the travel grant program in 2016.

International Immunology Training Program Bonn (IITB)

2. IITB students' network meeting

The Cluster hosted the first IITB students' network meeting in May 2015. The network meeting was jointly organized by the Cluster Coordination Office and a team of IITB students. The purpose of this meeting was to enhance networking between IITB students, inform students about new IITB activities and workshops and a "Young Academy", which gave PhD students the opportunity to talk to experienced postdocs about challenges on the way to finishing their PhD. The integrated soft skill workshop "Elevator pitch", the "Young academy" and the social evening afterwards were all very popular. Due to the success of the meeting, we now plan to offer it on a yearly basis.

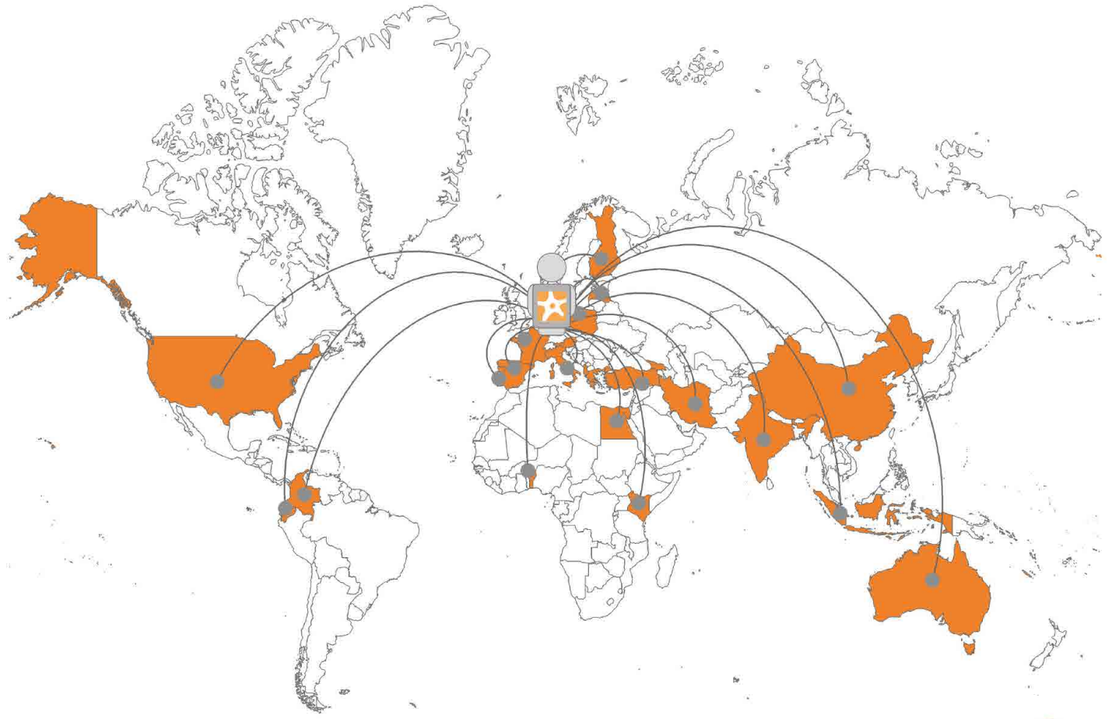


Figure 1 Countries (orange) IITB PhD students originate from.

3. The "International" in IITB

IITB members come from many different countries (**Figure 1**). The level of internationalization is roughly 25%, and the trend is rising. ImmunoSensation would like to foster international exchange in both directions: (1) PhD students are recruited via internationally visible networks and job announcements like the DAAD supported PhDGermany, Euraxess or Academic Keys; (2) to prepare German PhD students for an international work environment, the Cluster offers IITB travel grants and actively supports IITB members that are looking for research opportunities abroad.
One important measure taken to increase the number of international PhD students was our successful establishment of the DFG-funded international graduate training group "Bo&MeRanG". You can read more about this initiative on page 73. In addition, we have also been able to offer our long-term partners from the Edmund and Lily Safra Center for Brain Sciences (Hebrew University Jerusalem) the opportunity to participate in the IITB workshop "Data Analysis" and to present their work on cholinergic signalling and regulation in inflammation in a mini seminar series on December 9, 2015 in Bonn. A short report how our guests from Israel have experienced their trip to Germany, the workshop and the seminar series can be found on page 80.

4. Cooperation with the DGfI Autumn School

In today's scientific environment, pioneering research can only be performed by highly interdisciplinary teams. Reflecting the diversity of the Cluster, IITB students also have very diverse backgrounds in life and medical sciences, ranging from biochemistry, pharmacy, biology and biomedicine to mathematics and bioinformatics, and it is important to give these students the opportunity to advance their skills and acquire a structured background in immunology. In addition to the seminars offered to IITB students in Bonn, the Cluster has started a cooperation with the Autumn School on "Current concepts in immunology", hosted annually by the German Society of Immunology (DGfI). In October 2015, ImmunoSensation sent 10 outstanding PhD students to participate in the Autumn School in Merseburg. The registration fee and travel costs for these students were provided for via travel fellowships. In return, these students presented their research at the Autumn School, and they are also expected to share their newly acquired knowledge with other IITB members. After receiving extremely positive feedback from the participants in the 2015 program, the Cluster has decided to continue this cooperation in 2016.

International Immunology Training Program Bonn (IITB)

In addition to these new developments, the IITB program continued its well-established meet-the-experts events, the Career Café and its scientific and soft skill workshops. Furthermore, some of the IITB students have started a Stammtisch and a discussion group the “Immunology Study Group”, a student initiative to review the most important concepts of innate and adaptive immunity.

August 20, 2015
Career Café Miltenyi

September 24, 2015
Presentation Skills Workshop
Dr. Rick Scavetta

September 28, 2015
Adobe Illustrator Workshop
Dr. Kristina Koch

October 05, 2015
Meet-the-Expert
Prof. David Tarlinton
Walter and Eliza Hall Institute of Medical Research

October 26, 2015
Career Café Participants

International Immunology Training Program Bonn (IITB)

International Immunology Training Program Bonn (IITB)

by Immunology Study Group

WHAT: Immunology Study Group

WHEN: every week Wednesday, 6-7 pm

WHERE: BMZ, first floor

WHY: The Immunology Study Group is a self-organized weekly meeting that aims to improve and enhance the common knowledge of its members in immunology. It is prepared by and for PhD and Master’s students who want to learn more about immunology, as well as have a wider perspective of the field to better develop their own research. People attending the sessions have diverse expertise, which gives every member the opportunity to learn from the other.

A group member, who is in charge of preparing a previously assigned topic and organizing a discussion around it, chairs every meeting. Topics are picked up based on the structure of the book “Janeway’s Immunobiology” from Kenneth Murphy. After completing every part of the book, the field is deeper explored by selecting a pile of further reading about the most interesting discussed topics. With the aim of solving some of the posed questions during our discussions, we plan to host a meeting with experienced scientists who can clarify these issues.

International Immunology Training Program Bonn (IITB)

Field Report: Data Analysis Workshop

From Jerusalem to Bonn: Workshop with Dr. Rick Scavetta

by Dr. Shani Shenhar-Tsarfaty and Bettina Nadorp

We are both members of the laboratory of Prof. Hermona Soreq at The Hebrew University in Jerusalem, Israel, which is part of the International ImmunoSensation Cluster, Bonn. Last autumn, we were awarded with Cluster Travel Fellowships in order to attend a highly relevant workshop organized by the International Immunology Training Program Bonn (IITB) on the topic of "Data Analysis". In addition, we were invited to give a talk on our research interests and to have dinner with Cluster members. This is a report on our experiences and interactions with Cluster members during our visit to Bonn. First of all, we would like to introduce ourselves, our research interests, our goals when attending this workshop and the focus of research at the laboratory of Hermona Soreq, where we both work. In Prof. Hermona Soreq's laboratory, we are focusing on the molecular regulators of human stress reactions that enable immediate survival yet entail long-term damage spanning neuromuscular, neurodegenerative, and inflammatory diseases. More specifically, we are interested in the brain-to-body communication pathways that control the cholinergic signaling, which plays a causative role in these immediate reactions and in many diverse diseases. Dr. Shani Shenhar-Tsarfaty is a PostDoc in the laboratory. Her research focuses on the bio-medical aspects of the cholinergic system and involves genomic and biochemical analyses of human volunteer cohorts as well as molecular biology interference studies in live mice and cell cultures. In her current research, Shani explores cholinergic regulation at different post-transcriptional levels in multiple clinical models such as stroke, myocardial infarction, metabolic disorders and fear of terrorism. Bettina Nadorp is a PhD student in the laboratory. Her research interests involve microRNAs (miRNAs) in brain-immune communication, and specifically miRNAs that contribute to the regulation of the cholinergic signaling pathway. Many of these miRNA regulators are primate-specific (Nadorp and Soreq, 2014). Bettina's work is primarily focused on the primate-specific miRNA hsa-miR-608 (Hanin et al., 2014), but also includes other evolutionarily conserved miRNAs, such as miR-132. Currently, Bettina is working on the characterization of a 'humanized' transgenic mouse strain carrying the primate-specific microRNA-608 and its human-derived acetylcholinesterase binding site. Our motivation for attending the data analysis workshop was learning how to better cope with large data sets, such as high-throughput sequencing data or large cohort studies. The workshop was a very intense, three days' training for graduate students of the ImmunoSensation Cluster in Bonn. During this workshop we learned how to use the "R" statistical programming environment for data analysis purposes. Throughout the workshop, instructor Dr. Rick Scavetta taught us not only how to perform data manipulation and statistical modelling but also how to "think" in R, i.e. building our result files in the best way for future statistical analysis, and gave us hands-on experience in analyzing our own data. The workshop was structured so that it was interesting from the beginning to the end. Instead of starting with the very basic language, Dr. Scavetta showed us from the beginning how we can use "R" for the purpose of analysis with highly motivating hands-on training. This is a completely unique approach to learning "R" that we had never experienced before. By using our own data for the analysis during the workshop, we were able to achieve new insights into our data with the skilled help of Dr. Scavetta. After the workshop, we really felt like we had actually achieved our motivational goal, and we can now start using "R" for analyzing our own data in a fast and effective manner. On the evening of the last day of the workshop, we held a Cluster seminar hosted by Prof. Heneka. We both presented some recently published data and ongoing projects, and our talks were titled "The Cholinergic signaling impairment in neuro-inflammatory syndromes" and "CholinomiRs: From anxiety and inflammation to humanized Cholinergic regulation in mice", respectively. We were really excited to present our data and got very good feedback and advice from Cluster members and students. We also enjoyed our dinner with Cluster members and could benefit from fruitful discussions and planning of future collaborations. Altogether, we had a really great time in Bonn, and we would like to thank the leadership and members of the ImmunoSensation Cluster for enabling us to be part of this workshop and for giving us the opportunity to present our work.

International Immunology Training Program Bonn (IITB)

Rappaport Prize for Prof. Soreq - Tel Aviv

Rappaport Prize Ceremony March 15, 2015

Numerous awards and honours mark Professor Hermona Soreq's achievements, including the Kay Prize for Innovative Research, the U.S. Army Science Award for Excellence, Chancellor's Distinguished Lectureship from the University of California at Berkley and the Israeli Ministry of Health Prize for Excellence in Biomedical Research. Prof. Soreq has published over 200 peer reviewed research papers in internationally acclaimed journals, book chapters and monographs. Prof. Soreq holds a PhD degree in biochemistry from the Weizmann Institute, an honorary PhD in Chemistry from the University of Stockholm, Sweden, an honorary professorship in the Maimonides University, Buenos Aires, Argentina, and completed her post-doctoral studies at Rockefeller University, in New York. Prof. Soreq was nominated for the Rappaport Prize for Excellence in Medical Research. The prize was presented at the Tel Aviv Museum of Art on March 15, 2015. The prize was established by the Rappaport family to promote visionary, groundbreaking and innovative research with therapeutic ramifications that significantly promote human health.

International Immunology Training Program Bonn (IITB)

Teaching ‘big data’ to young scientists

Prof. Joachim Schultze on how to teach young immunologists

One of the major assets of the Cluster is the use of cutting-edge genomic technologies and computational approaches to better understand immune cell type and function, and one of our important goals is teaching young scientists these techniques in genomic and computational biology. In a commentary for Nature Immunology, Joachim L. Schultze outlines the structural and educational changes required to educate young scientists in high-throughput technologies. Teaching ‘big data’ analysis to young immunologists.

In this article, major considerations for scientists interested in big data immunology are provided. These include learning a computer language, participating in special undergraduate and graduate courses in computational biology, genomics and systems immunology, understanding the big data–driven circle of systems immunology, attending summer or winter schools on computational biology, and spending time on computational skills.

Schultze JL. Nat Immunol. 2015 Sep;16(9):902-5. doi: 10.1038/ni.3250.

Prof. Joachim L. Schultze

Students’ Initiative: IITB Blog

The ImmunoSensation Blog connects the Cluster’s young investigator community

by Klara Höning

ImmunoSensation Blog
the young investigators science blog

Pursuing your PhD in the ImmunoSensation Cluster of Excellence can be so much more than just pipetting and toiling in the lab from before sunrise to long after sunset. Inspired by the wish to learn more about what is going on within the Cluster and to bring the young investigators together to a lively community we, a team of enthusiastic PhD students, started the ImmunoSensation Blog in spring 2015. With the ImmunoSensation Blog we have established a platform for young investigators and everyone interested in immunology to explore the diverse range of topics, knowledge and people within the Cluster. Furthermore, we want to make people aware of all the fantastic opportunities the Cluster is offering for our research and scientific careers. Last year, the newly started blog connected scientists from all over the world. It had visitors from more than 50 countries. This platform presents new scientific findings, information on which research topics other students are working on, how to accelerate your career or which workshops or courses to take. The Blog answers these questions in categories like “How was...”, “Meet the Expert”, “Hard Science”, “Events” and “Short Profiles” within about 50 articles to date, with more coming out every week. There has been great interest in contributions to the “Meet the Expert” category with articles reporting on how to promote your scientific career as well as “Hard Science” articles featuring research topics from the Cluster of Excellence using new media formats. The “Event” posts, reporting about the Cluster Science Days and research abroad, have also received a lot of attention. Furthermore, a survey among the blog’s readers returned us very positive feedback on the articles as well as the webpage design. The readers also commented on their interest on articles featuring research and people from the ImmunoSensation Cluster or Cluster events. One important ongoing goal for us as the blog team is to further strengthen the young investigators’ network with additional events and personal meetings beyond the online platform. The next main challenge will be to spread the idea and to get more Cluster students and members involved in the project. We have already started a campaign to create more awareness of the blog and its opportunities. Altogether, having started the blog initiative, the first “blog year” was a very enriching year for me. I have enjoyed being part of a motivated team with the aspiration of realizing the great potential of the Cluster’s young investigator network to form a lively community. The team has built up an online platform and has created a set of articles on diverse topics. With the ImmunoSensation Blog I have discovered a lot about the fascinating diversity the ImmunoSensation Cluster of Excellence offers and together with my team I am enthusiastically looking forward to the future development of our blog!

International Immunology Training Program Bonn (IITB)

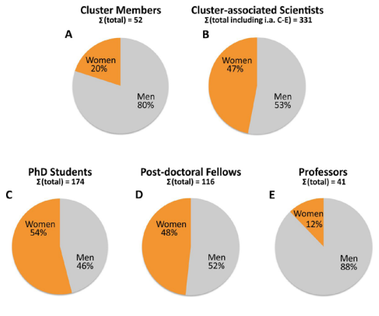
Family Support & Gender Equality

Our Goals for 2015

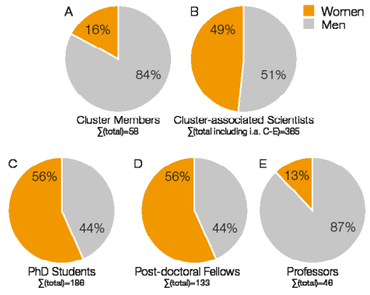
In the 2014 ImmunoSensation Annual Report, we referred to a “leaking pipeline“, a metaphor for the discrepancy between the number of talented women entering the life-science field and those who later on assume senior leadership positions. Stopping this leak remains a major priority for ImmunoSensation.

ImmunoSensation in Numbers

In 2015, the number of Cluster-associated scientists increased once again. Within this group we have the same percentage of female to male scientists (B). There are 56% (C) female scientists at the PhD level and 56% (D) at the PostDoc level, which means an increase of female scientists in both groups.
Although the number of female scientists drops, as expected, at the professorial level to 13% (E), ImmunoSensation considers positive that the number did not drop under the 12% from 2013. At the Cluster member level the percentage of women has declined from 20% in 2013 to 16% (A) in 2015. In order to counteract this trend, promote female representation and reward their hard work, ImmunoSensation’s Steering Committee appointed two excellent female scientists to Junior Research Group Leaders within ImmunoSensation.



Distribution of male and female researchers 2013



Distribution of male and female researchers 2015

Appointment of Two Independent Female Junior Research Group Leaders

Dr. Zeinab Abdullah (right) from the Institute of Experimental Immunology (IEI) and Dr. Annkristin Heine (left) from the Institute of Experimental Immunology (IEI) & the Medical Clinic III have become Junior Research Group Leaders within ImmunoSensation.

Cluster member Dr. Heine received the Lisec-Artz award for outstanding cancer research in 2015. For more information about Dr. Abdullah and Dr. Heine, please read the interviews on pages 51 and 53 respectively.

MeTra: Support for Career Development

In order to offer female scientists long-term career support, ImmunoSensation is participating in the networking program (MeTra) for young women in academia organized by Dr. Martina Pottek from the University of Bonn.
Two female scientists from ImmunoSensation took part in the program in 2015, one from the group of Dr. Dagmar Wachten (caesar) and the other from the group of Prof. Dr. Volkmar Gieselmann. ImmunoSensation has already registered another two young researchers for 2016, and we are looking forward to continuing our cooperation with the University of Bonn.

Annual Network Meeting of the Clusters of Excellence: Gender Equality

The annual network meetings within the Clusters of Excellence are always a first-rate opportunity to exchange information and expertise. The goal of these meetings is to raise awareness of the meaning of gender equality questions. The Clusters agree that family-friendly science is a key factor to successful science. In August 2015 ImmunoSensation hosted the meeting in Bonn and we are looking forward to more fruitful meetings.

Family-friendly Science

ImmunoSensation is aware that we have a long way to go until we reach 100% family-friendly science, but we are very pleased about the participation we have had in our programs in the last three years and the feedback we have received. An overview of our programs is provided below.

Support for Scientists on Parental Leave

In 2015 we supported two female scientists, Dr. Julia Vorac and Dr. Verena Schütte from the LIMES-Institute during their pregnancies and parental leave by providing student assistants.
Julia Vorac: “During my pregnancy a student assistant was working in our working group 12 hours a week, for 9 months in total, beginning in the fourth month of my pregnancy. Being pregnant meant that I was no longer allowed to perform particular experiments, such as those involving carcinogenic chemicals. Thus, it was very important for me to get someone as soon as possible who was able to take over these parts of my experiments. With the help of Prof. Förster and Dr. Astrid Draffehn, we could hire a student assistant to help. It was definitely great to get this help already during my pregnancy and not have to wait until my maternity leave. This gave my student assistant the opportunity to learn about the experiments while I was still there, so that she could independently complete experiments during my maternity leave. Also, the flexibility of choosing the best time period for the employment of the student is ideal. However, in my opinion some points might need some improvement. Because of the administrative requirements at the university, there was a certain lag period before the student could start working. Furthermore, the information about what kind of financial options are actually available for pregnant women should be more accessible. Some colleagues and supervisors are not even aware of the fact that financial resources, specifically for pregnant women and young parents, are available.“
Verena Schütte: “During my parental leave, I was fortunate to have access to a student assistant (SHK), who was financed by ImmunoSensation. For my research, this was a great support since it kept my practical work in the lab from

Family Support & Gender Equality

Family Support & Gender Equality

completely shutting down and after 6 months I could take over the running experiments. One reason why this worked so well is that we had a reliable student assistant who had performed his Bachelor thesis in our lab and was already engaged in the project. The organization and administration was done very quickly and without any problems. I can strongly recommend and do appreciate this form of support. Thanks ImmunoSensation!" We are glad that this measure provides rapid help and is now widely accepted although there is potential for improvement. In particular, we are taking measures to make sure that female scientists are better informed of the resources available to them.

Childcare Tailored to the Needs of Scientists

ImmunoSensation cooperates with the "pme Familienservice", which provides childcare for emergencies, such as the public childcare (Kita) strike this year, and holiday care during school holidays. The holiday care programs are tailored to the children's needs; the children are supervised in suitable age groups, and the childcare ratio is above-average. If there is a meeting within Germany, it is even possible to bring your child along and benefit from the local pme service since there are pme offices all over Germany. César Evaristo from Christian Kurts' group at the IEI used pme during the Kita strike this year, and he was very happy with the service, because it was very convenient and easy to use, and the children were happy with it - which is the most important thing. In 2015, ImmunoSensation has continued its cooperation with "Max and Mary", the English-speaking nursery, which is located at the Venusberg. The long opening hours and the flexible registration (no fixed deadlines as opposed to public childcare facilities) are key benefits for our scientists, especially for those who come from abroad. To encourage even the youngest of future scientists, ImmunoSensation will visit Max and Mary in 2016 for the "Day of Immunology". We hope to awaken their interest in science and inspire the next generation of immunologists!

Children and the Future of Science

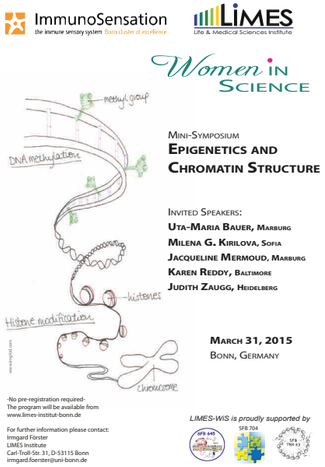
ImmunoSensation would like to share its enthusiasm for immunology with people of all ages and levels of knowledge. For the second time, the ImmunoSensation Cluster of Excellence participated in "Girls' Day". Five 14 to 15 year-old girls visited the Cluster at the University Hospital Bonn. After a short overview of ImmunoSensation's history of origins and the structure from Nicole Dahms, the girls spent the rest of the day in the labs of the Institute of Neurology and Institute of Medical Microbiology, Immunology and Parasitology (IMMIP) at the University Hospital Bonn. The Cluster would especially like to thank the female scientists Angelika Griep, Dr. Beatrix Schumak and Stephanie Schwartz who made this day a very special experience for the girls. One of this year's participants even came back to Prof. Heneka's lab for an internship during her summer school holidays. Nora Korber from the Liebfrauenschule Gymnasium in Cologne said: "After spending my Girls' Day at ImmunoSensation, I'm even more sure that I want to be a scientist one day, too. I'm interested in studying chemistry after finishing school." All in all, we hope that we have inspired a few future scientists in the last year and will continue to do so in the future. We have planned to continue our programs for children in 2016 and will also include the nursery "Max and Mary" (see above) in our children's program. ImmunoSensation is aware that these measures/activities are only small steps, but as you can see we cast our net wide, very wide and we hope to thereby enhance chances for the scientific world.

Participants of the Girls' Day 2015 with Nicole Dahms (left)
by Johann Saba - UKom

Women in Science Mini-Symposium

March 31, LIMES Institute

By Prof. Irmgard Förster



On March 31, 2015, the Mini-Symposium „Epigenetics and Chromatin Structure“ took place at the LIMES Institute in the frame of the LIMES Women in Science (WiS) program. This program is sponsored through the DFG-funded collaborative research centers SFB 704, SFB 645 and TRR 83 at the University of Bonn. Among other initiatives, the LIMES-WiS program aims to enhance the visibility of successful female scientists as role models for career development. LIMES-WiS is also linked with the gender equality program of the Excellence Cluster “ImmunoSensation”. The Mini-Symposium was organized by Irmgard Förster, Joachim Schultze and Waldemar Kolanus and focused on the topic of epigenetics to provide a state-of-the-art overview on recent achievements within this rapidly expanding field of research. In particular, it highlighted the importance of chromatin organization and positioning of genetic information within the nucleus for regulation of gene transcription. Karen Reddy from the John Hopkins School of Medicine, Baltimore, provided a fascinating introduction to the importance of the nuclear lamina underlying the inner nuclear membrane in chromosomal gene silencing using the example of the immunoglobulin heavy chain locus. Judith Zaugg, a bioinformatician from the European Molecular Biology Laboratories (EMBL) in Heidelberg, presented her recent work on the quantification of single nucleotide

Podium Discussion on gender equality

Round Table Discussion

polymorphisms identified in genome wide association studies, in particular their appearance in non-coding regions of the human genome. After lively discussions during the lunch break Jaqueline Mermoud (Institute of Molecular Biology and Tumor Research (IMT), University of Marburg) addressed the necessity of *de novo* chromatin silencing and appropriate histone modifications during the process of DNA replication. The influence of environmentally induced epigenetic modifications on cellular ageing was then discussed by Milena Georgieva Kirilova from the Bulgarian Academy of Science in Sofia. The last presentation of the Symposium was given by Uta-Maria Bauer, who is also working at the IMT, University of Marburg. She introduced a different class of histone modifying enzymes, namely the family of protein arginine methyltransferases (PRMTs). After the scientific program the invited speakers participated in an inspiring podium discussion on gender equality together with Michael Hoch, the rector of the University of Bonn, Margret Bülow, a young postdoc at the LIMES Institute, and Irmgard Förster as head of the LIMES-WiS program. Facing the obvious discrepancy between the high proportion (60-70%) of female undergraduate students in biomedicine and the apparent underrepresentation of women in leading academic positions, the discussants first tried to identify the main reasons for this unbalance. Obviously the problem is not restricted to Germany but similarly occurs in many other countries around the world. While the double challenge of raising children and being a competitive scientists certainly represents a major hurdle, the level of self-confidence appears to be different between male and female scientists, often preventing women from entering leadership positions. With lively participation of the audience, some key measures were proposed to improve gender equality in academic positions. Among others, these included the inclusion of childcare facilities for employees directly within bigger University institutions similar to that existing in the EMBL, Heidelberg; equal participation of parents in child raising; a higher proportion of tenure track positions; and early recruitment of female scientists into challenging professional tasks and responsibilities to build up self-confidence. Clearly, increasing the visibility of successful female scientists in meetings like this will help to encourage women to further pursue a research career.

Public Relations

Immuna and Immuno visit the Villa Hammerschmidt

We are proud that ImmunoSensation could participate in the Open Day at the Villa Hammerschmidt on June 14, 2015 (11am-6 pm). The Open Day in the Villa Hammerschmidt is a family-friendly event that gives the public the opportunity to visit the official residence of the Federal President Joachim Gauck. The ”Chancellor’s Bungalow” and parts of the Federal Chancellery were also open to visitors then, as well. A diverse program for families was also offered at the event, including contributions from caesar (center of advanced european studies and research), the Hausdorff Center for Mathematics and ImmunoSensation. ImmunoSensation offered its “sensing game”, which was immensely popular at the “9th Night of Science” in 2014. Participants test their tactile senses in order to better understand how our immune system discriminates between harmful and harmless, again. Immuna and Immuno were so busy explaining the immune system to the visitors that they did not have much of a chance to see the Villa, but they promised to be back!

Open Day at Villa Hammerschmidt

‘Sensing Game’

Social Media and Hompage

128 Follower
(2014: 90)

49 Follower
(2014: 25)

ImmunoSensation
the immune sensory system Bonn cluster of excellence

1375 Visitors
(2014: 1086)

universitätbonn ImmunoSensation
the immune sensory system Bonn cluster of excellence

The Cluster Times

The ImmunoSensation Newsletter / Issue n°1 / December 2015

We proudly present...

Dear members, associates and friends,

We warmly welcome you to our first issue of *The Cluster Times*, the brand new newsletter of the ImmunoSensation Cluster of Excellence, now served regularly.

We searched the web for "the purpose of newsletters" and most answers claimed newsletters should disseminate informatiwon and promote unity of a group while being a tool for representation and advertisement.

This is not wrong, just a bit apathetic. *The Cluster Times* has the goal to help you staying abreast of the Cluster while being entertained. It was created to inform about the latest Cluster tidings, to announce and remind of upcoming events and - last but not least - communicate our outstanding science in an enjoyable fashion.

Now, please enjoy reading and feel free to contact us any time.

Merry Christmas and a Happy New Year!

Yours,
Cluster Coordination Office

First Issue of ‘The Cluster TImes’ released December 2015

Childrens’ University

In 2015, our programs for children included another beloved tradition: the participation in the “Kinderuni” (the University of Bonn) for children aged 8 - 13. This time Professor Eicke Latz presented ImmunoSensation to this young audience. His topic was “Das Abwehrsystem des Körpers: Die Polizei in Dir” (or “The immune system: your very own internal police force”), with lively discussions between Professor Latz and the children. In 2016, the torch will be passed on to Dr. Annett Halle (caesar). For more information about Prof. Latz (see page 192) and Dr. Halle (see page 181), please see their biosketches.

List of ImmunoSensation Seminars, Seminars of Cluster Cooperation Partners and Meetings

ImmunoSensation Seminars 2015

ImmunoSensation Seminar: Exosome and RNA secretion by helminths
March 04, 2015
Dr. Amy Buck, The University of Edinburgh, Division of Infection and Pathway Medicine, UK

ImmunoSensation Seminar: Regulation of myeloid cell development and host-pathogen interactions by the canonical miRNA pathway
March 09, 2015
Dr. Nikoletta Papadopoulou, Institute for Med. Microbiology, Immunology and Hygiene, University of Cologne, Germany

ImmunoSensation Seminar: Dynamics and function of the master transcriptional factor Bcl6 in Follicular T helper cells
March 16, 2015
Weiwei Ma, Group of Prof. Hai Qi, Tsinghua University, Beijing, China

ImmunoSensation Seminar: New roles for immune components in immunity to helminths
March 23, 2015
Dr. William Horsnell, Institute of Infectious Disease and Molecular Medicine & Dept. of Clinical Laboratory Sciences, University of Cape Town, South Africa

ImmunoSensation Seminar: Regulatory T cells, IL-2 and type-1-diabetes
April 20, 2015
Dr. Emma Hamilton-Williams, Translational Research Institute, Diamantina Institute, The University of Queensland, Australia

ImmunoSensation Seminar: Dynamic regulation of permissive histone modifications and GATA3 underpin acquisition of Granzyme A in CD8+ T cells
April 24, 2015
Michelle Nguyen, Department of Microbiology & Immunology, The Peter Doherty Institute for Infection and Immunity, The University of Melbourne, Australia

ImmunoSensation Seminar: Induction of regulatory B cells by schistosome egg antigens
May 12, 2015
Dr. Simone Häberlein, Department of Parasitology, Leiden University Medical Center, Netherlands

ImmunoSensation Seminar: A surprising connection: Invariant T cells, vitamins, bacteria and wild mice
June 30, 2015
Prof. Olivier Lantz, Immunité et Cancer, Institut Curie Paris, France

ImmunoSensation Seminars 2015

ImmunoSensation Seminar: The parasitic worm product ES-62: a starting point for novel anti-inflammatory drug development
August 25, 2015
Prof. William Harnett, Strathclyde Institute of Pharmacy and Biomedical Sciences, University of Strathclyde, Scotland

ImmunoSensation Seminar: Elastases and related serine proteases of neutrophils: intensifiers of defense and destruction
September 22, 2015
Dr. Dieter Jenne, Institute of Lung Biology and Disease (iLBD), Comprehensive Pneumology Center (CPC), Helmholtz Zentrum München, Germany

ImmunoSensation Seminar & Meet-the-expert: Controlling the Input and Output of Germinal Centers: Roles for Myb and PRMT1
October 5, 2015
Prof. David Tarlinton, Walter and Eliza Hall Institute of Medical Research, Melbourne, Australia

ImmunoSensation Seminar: From mice to men: MicroRNA regulators of stress-induced cholinergic signaling
October 6, 2015
Prof. Hermona Soreq, The Edmond and Lily Safra Center for Brain Sciences, The Hebrew University of Jerusalem, Israel

ImmunoSensation Seminar: RIG-I like receptors and autoimmune diseases
October 9, 2015
Prof. Hiroki Kato, Institute for Virus Research, Kyoto University, Japan

ImmunoSensation Technical Seminar: Deutsches Schutz-/ Patentrecht
October 22, 2015
Rüdiger Mull, Rheinische Friedrich-Wilhems-Universität Bonn, Germany

ImmunoSensation Seminar: Rhythmic control of leukocyte migration
November 24, 2015
Dr. Christoph Scheiermann, Walter-Brendel-Zentrum für Experimentelle Medizin, Ludwig-Maximilian-Universität München, Germany

ImmunoSensation Seminar & Meet-the-expert: Inducing monocyte function during acute gastrointestinal infection
December 01, 2015
Dr. John Grainger, Manchester Collaborative Centre for Inflammation Research (MCCIR), Inflammatory cell function and conditioning during infection, The University of Manchester, England

ImmunoSensation Seminars 2015

ImmunoSensation Seminar: What turns Aire on? Transcriptional programs controlling the expression of the Autoimmune Regulator gene
December 03, 2015
Yonatan Herzig, Jakub Abramson's lab, Weizmann Institute of Science, Israel

ImmunoSensation Seminar: The Cholinergic signaling impairment in neuro inflammatory syndromes
December 09, 2015
Dr. Shani Shenhar-Tsarfaty, The Edmond and Lily Safra Center for Brain Sciences, The Hebrew University of Jerusalem, Israel

ImmunoSensation Seminar: CholinomiRs: From anxiety and inflammation to humanized Cholinergic regulation in mice
December 09, 2015
Bettina Nadorp, The Edmond and Lily Safra Center for Brain Sciences, The Hebrew University of Jerusalem, Israel

Seminars Cooperation Partners 2015

Bonn Lecture Series in Neuroscience: Astrocytic and neuronal large-pore (hemi) channels: Activation and permeability
January 13, 2015
Nanna MacAulay, Ph.D. Associate Professor, Institute of Cellular and Molecular Medicine, University of Copenhagen, Denmark

SFB 704 Seminar: Therapeutic T cell vaccination through antigen targeting into cross-presenting dendritic cells
January 20, 2015
Prof. Richard Kroczek, Molekulare Immunologie, Robert Koch-Institut, Berlin, Germany

Bonn Lecture Series in Neuroscience: Functional organisation of connection strength in neocortical circuits
January 29, 2015
Prof. Dr. Thomas Mrsic-Flogel, Associate Professor in Neuroscience, Biocenter, University of Basel, Switzerland

Bonn Lecture Series in Neuroscience: Plasticity of hippocampal mossy fiber synapses
February 19, 2015
Prof. Dr. Dr. Michael Frotscher, Center for Molecular Neurobiology, Institute for Structural Neurobiology, University Medical Center Hamburg-Eppendorf, Germany

Seminars Cooperation Partners 2015

Women in Science Seminar Series:
Mini Symposium: Epigenetics and Chromatic Structure, see page 88
March 31, 2015
LIMES Institute, Bonn, Germany

Women in Science Seminar Series:
Exploring the Molecular Signature of Mammalian Touch and Pain- Insights into Health and Disease
April 20, 2015
Dr. Manuela Schmidt, Somatosensory Signaling, Max Planck Institute of Experimental Medicine, Göttingen, Germany

Bonn Lecture Series in Neuroscience: Degradation of Tau Protein by Selective Autophagy
May 11, 2015
Gail V. Johnson, University of Rochester Medical Center, N.Y., USA

Bonn Lecture Series in Neuroscience: Bridging the scales: Spiking networks for biological and artificial neural computation
May 15, 2015
Dr. Raoul-Martin, Memmesheimer Department for Neuroinformatics, Donders Center, Radboud University Nijmegen, Netherlands

Bonn Lecture Series in Neuroscience: Synaptic tenacity: When everything changes, do things really stay the same?
May 18, 2015
Prof. Dr. Noam E. Ziv, Technion, Dept. of Physiology, Rappaport Faculty of Medicine and Network Biology Research Laboratorie, Haifa, Israel

SFB 704 Seminar: Transcutaneous Immunization – Regulation of local inflammatory responses to generate systemic tumor immunity
May 19, 2015
PD Dr. Markus P. Radsak, III. Medizinische Klinik, Universität Mainz, Germany

Bonn Lecture Series in Neuroscience: Fluorescence Trails Microscopy: A Multifocal Imaging Technique for Fast Optical Recordings of Neuronal Activity
June 12, 2015
Matthew Shtrahman, Departments of Neurology and Neurobiology, UCLA David Geffen School of Medicine, USA

Bonn Lecture Series in Neuroscience: Netrin-ephrin synergy in motor axon guidance
June 17, 2015
Artur Kania, PhD, Director, Neural Circuit Development Laboratory, Institut de recherches cliniques de Montréal (IRCM), Montréal, Canada

Bonn Lecture Series in Neuroscience: The molecular logic of axon guidance
June 22, 2015

Seminars Cooperation Partners 2015

Prof. Dr. Rüdiger Klein, Director, Max-Planck-Institute of Neurobiology, Department of Molecular Neurobiology, München-Martinsried, Germany

Bonn Lecture Series in Neuroscience: Synapses and Dementia?
June 22, 2015
Prof. Dr. Dietmar Schmitz, Deutsches Zentrum für Neurodegenerative Erkrankungen (DZNE), Charité - Universitätsmedizin Berlin, Germany

Bonn Lecture Series in Neuroscience: GABAergic action and the activity-dependent dendritic inhibition in the primary gate of hippocampus
June 23, 2015
Dr.Yu-Chao Liu, Institute of Neuroscience, National Yang-Ming University, Taipei 112, Taiwan, China

Bonn Lecture Series in Neuroscience: Towards understanding positive symptoms of DiGeorge syndrome and schizophrenia
July 03, 2015
Dr. Stanislav S. Zakharenko, Developmental Neurobiology St. Jude Children's Research Hospital Memphis, USA

Bonn Lecture Series in Neuroscience: Microtubules during nervous system development and disease
July 16, 2015
Peter W. Baas, Dept. Neuroscience, Drexel University, Philadelphia, PA, USA

Bonn Lecture Series in Neuroscience: Deconstructing complexity through spectral imaging and metagenomics: Systems analysis of the oral microbiome
July 23, 2015
Gary G. Borisy, Dept. Microbiology, Forsyth Institute Cambridge, MA, USA

Bonn Lecture Series in Neuroscience: Physiology and functional effects of tDCS and related techniques
July 28, 2015
Prof. Dr. Michael A. Nitsche, Dept. Clinical Neurophysiology, University Medicine Göttingen

Bonn Lecture Series in Neuroscience: Medical Text Analytics for Neurodegenerative Diseases
August 12, 2015
Daniel Damian, Centre Leenaards de la Memoire - CHUV, University Hospital of Lausanne, Switzerland

Bonn Lecture Series in Neuroscience: Recent progress toward a high-performance neural prosthesis
September 16, 2015
Prof. Dr. Andrew Schwartz, Department of Neurobiology, University of Pittsburgh, USA

Seminars Cooperation Partners 2015

Bonn Lecture Series in Neuroscience: Functional imaging hippocampal microcircuits for spatial navigation and learning
September 24, 2015
Attila Losonczy, Department of Neuroscience, Mortimer B. Zuckerman Mind Brain Behavior Institute, Kavli Institute for Brain Science, Columbia University, USA

Bonn Lecture Series in Neuroscience: The Cell Biology and Biochemistry of Microtubule Nucleation
September 28, 2015
Prof. Dr. Gary Brouhard, Department of Biology, McGill University, Montréal, Quebec, Canada

SFB 704 Seminar: Myeloid cells, T cells and Legionella infection in the lung
October 06, 2015
Prof. Ian van Driel, Department of Biochemistry and Molecular Biology, University of Melbourne, Australia

Bonn Lecture Series in Neuroscience: Chaperone- and Translocon-modulated Folding Pathways of Single Membrane Proteins
October 09, 2015
Daniel Müller, Dept. Biosystems Science and Engineering, ETH-Zürich, Basel, Switzerland

Bonn Lecture Series in Neuroscience: Bi-directional control of epileptic networks by the thalamus
October 15, 2015
Prof. Jeanne T. Paz, Gladstone Institute of Neurological Disease & Neurology at UCSF, San Francisco, USA

Bonn Lecture Series in Neuroscience: The potential role of microglia in neuroprotection and depression
October 23, 2015
Prof. Dr. Knut Biber, Department of Psychiatry and Psychotherapy, University Hospital Freiburg, Germany

Bonn Lecture Series in Neuroscience: Building Memories: Sparse Coding and Learning of Associations by Single Neurons in the Human Brain
October 26, 2015
Matias J Ison, University of Leicester, UK

Bonn Lecture Series in Neuroscience: Sensor-basierte Bewegungsanalyse beim Parkinson Syndrom
October 27, 2015
Dr. Jochen Klucken, Hospital for Neurology, University Erlangen, Germany

Bonn Lecture Series in Neuroscience: Joint longitudinal and survival analysis. Application to the Spinocerebellar Ataxia data

Seminars Cooperation Partners 2015

October 28, 2015
Dr. Sophie Tezenas du Montcel, Department of Biostatistics and Medical Informatics, Paris, France

SFB 704 Seminar: Physiologic and immunopathologic features of single cells and whole organs revealed by 2-Photon and lightsheet microscopy
November 11, 2015
Prof. Matthias Gunzer, Institut für Experimentelle Immunologie und Bildgebung, Universität Duisburg-Essen, Germany

Bonn Lecture Series in Neuroscience: Serum protein biomarkers of the latent dementia variable d(delta)
November 11, 2015
Donald R. Royall, Department of Psychiatry, The University of Texas Health Science Center, San Antonio, TX, USA

Bonn Lecture Series in Neuroscience: Astrocytic contribution to synaptic transmission
November 19, 2015
Prof. Stéphane H. Oliet, Neurocentre Magendie, University of Bordeaux, Bordeaux, France

Bonn Lecture Series in Neuroscience: Ionenkanalmodulation in der Multiplen Sklerose – Fakten und Phantasien
December 01, 2015
Prof. Dr. Sven Meuth, Klinik für Neurologie, Universität Münster, Germany

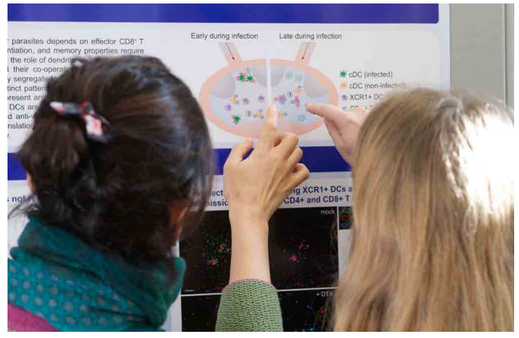
Bonn Lecture Series in Neuroscience: An ion channel which regulates immune surveillance of the brain by microglia
December 10, 2015
Dr. Christian Madry, Department of Neuroscience, Physiology and Pharmacology, UK

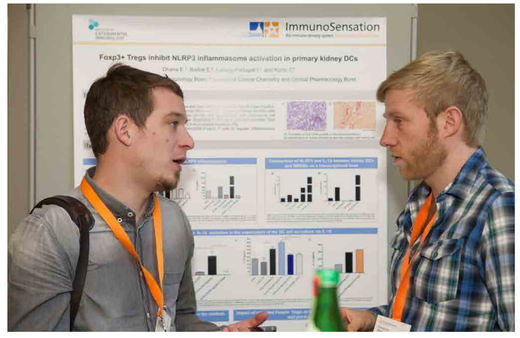
Meetings 2015

7th International Symposium on DAMPs and HMGB1
September 10 - 12, 2015
University Hospital Bonn and Wasserwerk Bonn, Germany

3rd Cluster Science Days 2015
November 02 & 03, 2015
University Hospital Bonn, Germany

Impressions Cluster Science Days 2015





Impressions Cluster Science Days 2015

Publications

ImmunoSensation Publication List 2015

Al Haj A, Mazur AJ, Radaszkiewicz K, Radaszkiewicz T, Makowiecka A, Stopschinski BE, Schönichen A, **Geyer M**, Mannherz HG. Distribution of formins in cardiac muscle: FHOD1 is a component of intercalated discs and costameres. **Eur. J. Cell Biol.** 2015 Feb 1;94(2): 101-113

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Model of the BMZ II, University Hospital Bonn
by Ludes Generalplaner GmbH

Prizes & Distinctions

Prizes and Distinctions awarded within ImmunoSensation 2015

2015 Prof. Frank Bradke has been awarded the Gottfried Wilhelm Leibniz prize.
Cluster member Prof. Bradke is the head of the “Axon Growth and Regeneration” research group at the German Center for Neurodegenerative Diseases (DZNE). The award includes prize money of 2,5 million €.

2015 Prof. Veit Hornung from the Institute of Molecular Medicine, University Hospital Bonn has been announced by EMBO as one of 58 outstanding researchers in the field of life sciences that have been elected to its membership.

2015 Prof. Veit Hornung received ERC (European Research Council) Consolidator Grant for his project “Genetic Dissection of Innate Immune Sensing and Signaling” (GENESIS).

2015 Prof. Thomas Tüting (Department of Dermatology) received the Photodermatology Research Award.
This international award is being sponsored by La Roche-Posay Laboratoire Pharmaceutique Germany. The prize is awarded biennially.

February 2015 Prof. Achim Hörauf and his research team (Institute of Medical Microbiology, Immunology and Parasitology at the University Hospital Bonn) has been awarded the “Memento Preis für vernachlässigte Krankheiten” 2015.
The award was launched by Ärzte ohne Grenzen e.V., Brot für die Welt, DAHW Deutsche Lepra, Tuberkulosehilfe e.V. and BUKO Pharma-Kampagne in 2014. The Memento Preis is awarded annually. This year the prize giving ceremony took place in Berlin on February 25, 2015.

February 2015 Prof. Hermona Soreq (Hebrew University) received the “Rappaport Prize” for her Excellence in Medical Research.
The Rappaport family established the prize in order to promote visionary, groundbreaking and innovative research with therapeutic ramifications that significantly promote human health. Cluster member Hermona Soreq received $60,000 for her research on the neurotransmitter acetylcholine’s role in both health and disease in the brain and other organs.

March 23, 2015 Prof. Waldemar Kolanus was elected as executive Director of the LIMES Institute.
ImmunoSensation’s vice speaker Prof. Waldemar Kolanus was officially elected as the new executive Director of the LIMES Institute.

April 2015 Prof. Achim Hörauf has been awarded the BioRegionen Innovation Prize of 2015.
Cluster member Prof. Achim Hörauf (Institute of Medical Microbiology, Immunology & Parasitology, University Hospital Bonn) and his team received the BioRegionen Innovation Prize 2015 for their remarkable invention in the field of tropical infectious diseases.

Prizes and Distinctions awarded within ImmunoSensation 2015

April 29, 2015 Inauguration of Cluster member Prof. Michael Hoch as rector of the University of Bonn.
The former excecutive Director of the LIMES Institute is the 143. rector of the Rheinsche Friedrich-Wilhelms-Universtität Bonn.

May 2015 Prof. Christian Kurts from the Institute of Experimental Immunology, University Hospital Bonn became an elected member of the German National Academy of Sciences, Leopoldina.

September 30 - October 03, 2015 Dr. Anna-Maria Herzner received the Paula Pitha Award.
Anna-Maria Herzner from the Institute of Clinical Chemistry and Clinical Pharmacology received the Paula Pitha Award for the Best Abstract on Interferon Research at the TOLL2015 Conference (http://www.toll2015.org), which took place in Marbella, Spain. More than 400 abstracts had been submitted.

October 30, 2015 Dr. Beate Henrichfreise has been awarded the PHOENIX Pharmazie Wissenschaftspreis.
The prize is being awarded in the categories pharmacology and clinical pharmacy, pharmaceutical biology, pharmaceutical chemistry and pharmaceutical technology. Beate Henrichfreise from the Insitute of Medical Microbiology, Immunology & Parasitology, University of Bonn received the prize in the field “pharmaceutical biology” with her publication “AmiA is a penicillin target enzyme with dual activity in the intracellular pathogen Chlamydia pneumoniae” (Nature Communications. 5, 4201, 2014).

November 2015 Dr. Annkristin Heine received the Lisec-Artz award.
Cluster member Dr. Annkristin Heine from the Medical Clinic III and Institute of Experimental Immunology received the Lisec-Artz award for her outstanding cancer research.

November 2015 Prof. Christian Kurts is speaker of the DFG funded Bonn and Melbourne International Research and Training Group (Bo&MeRanG) that will start in April 2016 and covers the topic „Myeloid Antigen Presenting Cells and the Induction of Adaptive Immunity”.

November 27, 2015 DFG Review Board Election 2015.
Three Cluster members have been elected as members of the DFG review boards for the 2016-2019 term. The DFG review boards scientifically evaluate proposal to fund research projects in their respective subject areas.
The following ImmunoSensation members have been elected: **Prof. Achim Hörauf** (Institute of Medical Microbiology, Immunology & Parasitology, University Hospital Bonn) for subject area Medical Microbiology, Parasitology, Medical Mycology, and Hygiene, Molecular Infection Biology, **Prof. Irmgard Förster** (LIMES Institute, University of Bonn) for the subject area Immunology, **Prof. Christian Kurts** (Institute of Experimental Immunology, University Hospital Bonn) for the subject area Nephrology.

Inauguration of Prof. Michael Hoch as rector of the University of Bonn
by Volker Lannert

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Annual Report 2015
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ImmunoSensation Member List

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ImmunoSensation Member List

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Turtles at the Poppelsdorfer Schloss University of Bonn
by Dr. Thomas Mauersberg

Biosketches

New Members

Dr. Zeinab Abdullah, PhD

Institute of Experimental Immunology

new Member since 2015

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Research Expertise

The overall goal of our research is to understand the molecular and cellular mechanisms by which chronic inflammation can affect outcome of infection and immune cell function. Specifically, the research focuses on mechanistic studies to understand the impact of chronic inflammation during liver fibrosis on the function of hepatic myeloid cells in host defence and adaptive immune responses against de novo infections.

Education / Training

University of Cologne, Immunology, PhD, 2007
University of Bonn, Biology, Diploma Thesis, 2004

Appointments / Positions Held

Since 2015
Group Leader at the Institute of Experimental Immunology, University of Bonn, Germany
2012 - 2015
Senior Group Leader at the Institutes of Molecular Medicine and Experimental Immunology, University of Bonn, Germany
2009 - 2012
Junior Research Group Leader of Bonfor at the Institutes of Molecular Medicine and Experimental Immunology, University of Bonn, Germany
2008 - 2009
Postdoctoral Research Fellow, Institute of Medical Microbiology, Immunology and Hygiene, University of Cologne, Germany

Honors / Awards

2014
DFG TRR57 “Organ Fibrosis: From Mechanisms of Injury to Modulation of Disease” (Female Scientist Support Program)
2012
DFG TRR57 “Organ Fibrosis: From Mechanisms of Injury to Modulation of Disease” (Female Scientist Support Program)

10 Most Relevant Publications for Dr. Zeinab Abdullah

1. Beyer M. *, **Abdullah Z.** *, Chemnitz J. M. *, M. Daniela, Sander J., Lehmann C., Thabet Y., Shinde, P. V., Schmidleithner L., Köhne M., Trebicka J., Schierwagen R., Hofmann A. Popov A., Lang K. S., Oxenius A., Buch T., Kurts C., Heikenwälder M., Fätkenheuer G., Lang P. A., Hartmann P., Knolle P. A. *, Schultze J. L. *. (2016). TNF impairs CD4+ T-cell mediated immune control in chronic viral infection. Nat Immunology
2. Hasenberg, A., Hasenberg, M., Mann, L., Neumann, F., Borkenstein, L., Stecher, M., Kraus, A., Engel, D. R., Klingberg, A., Seddigh, P., **Abdullah, Z.**, Klebow, S., Engelmann, S., Reinhold, A., Brandau, S., Seeling, M., Waisman, A., Schraven, B., Gothert, J. R., Nimmerjahn, F., Gunzer, M. (2015): Catchup: a mouse model for imaging-based tracking and modulation of neutrophil granulocytes. Nat Methods 12, 445-452.
3. Bottcher, J. P., Beyer, M., Meissner, F., **Abdullah, Z.**, Sander, J., Hochst, B., Eickhoff, S., Rieckmann, J. C., Russo, C., Bauer, T., Flecken, T., Giesen, D., Engel, D., Jung, S., Busch, D. H., Protzer, U., Thimme, R., Mann, M., Kurts, C., Schultze, J. L., Kastenmuller, W., Knolle, P. A. (2015): Functional classification of memory CD8(+) T cells by CX3CR1 expression. Nat Commun 6, 8306.
4. M. J. Wolf, Adili, A., Piotrowitz, K., **Abdullah, Z.**, Boege, Y., Stemmer, K., Ringelhan, M., Simonavicius, N., Egger, M., Wohlleber, D., Lorentzen, A., Einer, C., Schulz, S., Clavel, T., Protzer, U., Thiele, C., Zischka, H., Moch, H., Tschop, M., Tumanov, A. V., Haller, D., Unger, K., Karin, M., Kopf, M., Knolle, P., Weber, A. and Heikenwalder, M. (2014): Metabolic activation of intrahepatic CD8+ T cells and NKT cells causes nonalcoholic steatohepatitis and liver cancer via cross-talk with hepatocytes. Cancer cell, 549-64
5. **Abdullah, Z.** and Knolle, P. A. (2014): Scaling of immune responses against intracellular bacterial infection. The EMBO journal, 2283-94
6. L. R. Huang, Wohlleber, D., Reisinger, F., Jenne, C. N., Cheng, R. L., **Abdullah, Z.**, Schildberg, F. A., Odenthal, M., Dienes, H. P., van Rooijen, N., Schmitt, E., Garbi, N., Croft, M., Kurts, C., Kubes, P., Protzer, U., Heikenwalder, M. and Knolle, P. A. (2013): Intrahepatic myeloid-cell aggregates enable local proliferation of CD8(+) T cells and successful immunotherapy against chronic viral liver infection. Nature immunology, 574-83
7. **Abdullah Z.**, Schlee, M., Roth, S., Mraheil, M. A., Barchet, W., Bottcher, J., Hain, T., Geiger, S., Hayakawa, Y., Fritz, J. H., Civril, F., Hopfner, K. P., Kurts, C., Ruland, J., Hartmann, G., Chakraborty, T. and Knolle, P. A. (2012): RIG-I detects infection with live Listeria by sensing secreted bacterial nucleic acids. The EMBO journal, 4153-64
8. **Abdullah Z.**, Geiger, S., Nino-Castro, A., Bottcher, J. P., Muraliv, E., Gaidt, M., Schildberg, F. A., Riethausen, K., Flossdorf, J., Krebs, W., Chakraborty, T., Kurts, C., Schultze, J. L., Knolle, P. A. and Klotz, L. (2012): Lack of PPARgamma in myeloid cells confers resistance to Listeria monocytogenes infection. PloS one, e37349
9. **Abdullah Z.**, Saric, T., Kashkar, H., Baschuk, N., Yazdanpanah, B., Fleischmann, B. K., Hescheler, J., Kronke, M. and Utermohlen, O. (2007): Serpin-6 expression protects embryonic stem cells from lysis by antigen-specific CTL. Journal of immunology, 3390-9
10. A. Popov*, **Abdullah, Z*.**, Wickenhauser, C., Saric, T., Driesen, J., Hanisch, F. G., Domann, E., Raven, E. L., Dehus, O., Hermann, C., Eggle, D., Debey, S., Chakraborty, T., Kronke, M., Utermohlen, O. and Schultze, J. L. (2006): Indoleamine 2,3-dioxygenase-expressing dendritic cells form suppurative granulomas following Listeria monocytogenes infection. The Journal of clinical investigation, 3160-70
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PD Dr. Marc Beyer, MD

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Research Expertise

Marc Beyer's scientific focus is on the characterization of immunoregulatory mechanisms in myeloid and T cells. Based on transcriptomic and epigenetic discovery his group uses genetic engineering to study fundamental molecular mechanisms of immunoregulation.

Education / Training

University of Bonn, Germany, Life & Medical Sciences, Habilitation, 2015
University of Cologne, Germany, Medicine, MD thesis, 2005
University of Heidelberg, Germany, Bioinformatics, Postgraduate program, 2004
University of Cologne, Germany, Medicine, Medicine Fellow 2002 - 2004
University of Cologne, Germany, Medicine, MD, 2002

Appointments / Positions Held

Since 2014
Group leader, University of Bonn, Germany
2008 - 2013
Senior postdoctoral research fellow, University of Bonn, Germany
2004 - 2007
Postdoctoral research fellow, University of Cologne, Germany
2002 - 2004
Medicine fellow, University of Cologne, Germany

10 Most Relevant Publications for PD Dr. Marc Beyer

1. **Beyer M**, Abdullah Z, Chemnitz JM, Maisel D, Sander J, Lehmann C, Thabet Y, Shinde PV, Schmidleithner L, Köhne M, Trebicka J, Schierwagen R, Hofmann A, Popov A, Lang KS, Oxenius A, Buch T, Kurts C, Heikenwalder M, Fätkenheuer G, Lang PA, Hartmann P, Knolle PA, Schultze JL. 1. Tumor-necrosis factor impairs CD4(+) T cell-mediated immunological control in chronic viral infection. Nat Immunol. 2016 Mar 7
2. Schmidt SV, Krebs W, Ulas T, Xue J, Baßler K, Günther P, Hardt AL, Schultze H, Sander J, Klee K, Theis H, Kraut M, **Beyer M**, Schultze JL. The transcriptional regulator network of human inflammatory macrophages is defined by open chromatin. Cell Res. 2016 Feb.
3. Schultze JL, **Beyer M.** Myelopoiesis Reloaded: Single-Cell Transcriptomics Leads the Way. Immunity. 2016 Jan 19.
4. Böttcher JP, **Beyer M**, Meissner F, Abdullah Z, Sander J, Höchst B, Eickhoff S, Rieckmann JC, Russo C, Bauer T, Flecken T, Giesen D, Engel D, Jung S, Busch DH, Protzer U, Thimme R, Mann M, Kurts C, Schultze JL, Kastenmüller W, Knolle PA. Functional classification of memory CD8(+) T cells by CX3CR1 expression. Nat Commun. 2015 Sep 25.
5. Krebs W, Schmidt SV, Goren A, De Nardo D, Labzin L, Bovier A, Ulas T, Theis H, Kraut M, Latz E, **Beyer M**, Schultze JL. Optimization of transcription factor binding map accuracy utilizing knockout-mouse models. Nucleic Acids Res. 2014 2014 Dec 1;42(21):13051-60.
6. Xue J, Schmidt SV, Sander J, Draffehn A, Krebs W, Quester I, De Nardo D, Gohel TD, Emde M, Schmidleithner L, Ganesan H, Nino-Castro A, Mallmann MR, Labzin L, Theis H, Kraut M, **Beyer M**, Latz E, Freeman TC, Ulas T, Schultze JL. Transcriptome-based network analysis reveals a spectrum model of human macrophage activation. Immunity. 2014 Feb 20;40(2):274-88.
7. Sommer D, Peters A, Wirtz T, Mai M, Ackermann J, Thabet Y, Schmidt J, Weighardt H, Wunderlich FT, Degen J, Schultze JL, **Beyer M**. Efficient genome engineering by targeted homologous recombination in mouse embryos using transcription activator-like effector nucleases. Nat Commun. 2014 Jan 13;5:3045.
8. **Beyer M**, Mallmann MR, Xue J, Staratschek-Jox A, Vorholt D, Krebs W, Sommer D, Sander J, Mertens C, Nino-Castro A, Schmidt SV, Schultze JL. High-resolution transcriptome of human macrophages. PLoS One. 2012;7(9):e45466.
9. **Beyer M**, Schumak B, Weihrauch MR, Andres B, Giese T, Endl E, Knolle PA, Classen S, Limmer A, Schultze JL. In vivo expansion of naïve CD4+ CD25(high) FOXP3+ regulatory T c ells in patients with colorectal carcinoma after IL-2 administration. PLoS One. 2012;7(1):e30422.
10. **Beyer M**, Thabet Y, Müller RU, Sadlon T, Classen S, Lahl K, Basu S, Zhou X, Bailey-Bucktrout SL, Krebs W, Schönfeld EA, Böttcher J, Golovina T, Mayer CT, Hofmann A, Sommer D, Debey-Pascher S, Endl E, Limmer A, Hippen KL, Blazar BR, Balderas R, Quast T, Waha A, Mayer G, Famulok M, Knolle PA, Wickenhauser C, Kolanus W, Schermer B, Bluestone JA, Barry SC, Sparwasser T, Riley JL, Schultze JL. Repression of the genome organizer SATB1 in regulatory T cells is required for suppressive function and inhibition of effector differentiation. Nat Immunol. 2011 Aug 14;12(9):898-907.

Prof. Matthias Geyer, PhD

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new Member since 2014

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Institute of Innate Immunity
Department of Structural Immunology
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Research Expertise

The Geyer lab is interested in the regulation of transcription and the molecular mechanisms that govern immune receptor activation. We use a variety of techniques from molecular biology and biochemistry to structural biology to analyze interaction between proteins, RNA, lipids, and ligands. The transcription cycle is regulated by cyclin-dependent kinases that phosphorylate the RNA polymerase II. We analyze the transition from transcription initiation to productive elongation in eukaryotic cells. We study the molecular and structural mechanisms that determine the activity and regulation of transcription-controlling kinases, as well as their inhibition by small molecular compounds. We recently also focused on the analysis of receptor activation of NLRP3 and formation of the NLRP3/ASC/caspase inflammasome. Besides NACHT-domain containing proteins, Toll-like receptors, RIG-I and the cGAS-STING pathway mediate the immune-recognition of pathogens. We aim at identifying target sites in these immune regulators that allow for the specific interference with the immune system, e.g., by small molecular compounds.

Education / Training

University of Heidelberg, Germany, Biochemistry, Habilitation, 2006
University of Heidelberg, Germany, Biophysics, PhD, 1995
University of Heidelberg, Germany, Physics, Diploma, 1991

Appointments / Positions Held

2014 - present
Group leader Structural Immunology, University of Bonn, Germany
2012 - 2014
Group leader Physical Biochemistry, Research center caesar, Bonn, Germany
2003 - 2012
Group leader, Department of Physical Biochemistry, Max Planck Institute of Molecular Physiology, Dortmund, Germany
2001 - 2002
Visiting Scientist, Computational and Structural Biology Programme, European Molecular Biology Laboratory, Heidelberg, Germany
1998 - 2001
Research associate at the Howard Hughes Medical Institute, Dept. of Medicine, Microbiology and Immunology, University of California at San Francisco, San Francisco, USA
1995 - 1998
Research fellow in the Dept. of Biophysics, Max-Planck-Institute for Medical Research, Heidelberg, Germany

Honors / Awards

2008
Editorial Board Member: Cytoskeleton
2001
Habilitation fellowship of the Peter and Traudl Engelhorn-Stiftung, Penzberg
1998
Long-term fellowship of the European Molecular Biology Organization (EMBO), Heidelberg
1995
Postdoctoral fellowship of the German Science Foundation (DFG)

10 Most Relevant Publications for Prof. Matthias Geyer

1. Greifenberg AK, Hönig D, Pilarova K, Düster R, Bartholomeeusen K, Bösken CA, Anand K, Blazek D, **Geyer M** (2016). Structural and functional analysis of the Cdk13/Cyclin K complex. Cell Rep. 14, 320–331.
2. Kühn S, Erdmann C, Kage, F, Block, J, Schwenkmezger L, Steffen A, Rottner K, **Geyer M** (2015). Structure of the FMNL2–Cdc42 complex yields insights in lamellipodia and filopodia formation. Nat. Commun. 6: 7088.
3. Bösken CA, Farnung L, Hintermair C, Merzel Schachter M, Vogel-Bachmayr K, Blazek D, Anand K, Fisher RP, Eick D, **Geyer M** (2014). The structure and substrate specificity of human Cdk12/Cyclin K. Nat. Commun. 5: 3505.
4. Eick D, **Geyer M** (2013). The RNA polymerase II carboxy-terminal domain (CTD) code. Chem. Rev. 113, 8456–8490.
5. Czudnochowski N, Bösken CA, **Geyer M** (2012). Serine-7 but not serine-5 phosphorylation primes RNA polymerase II CTD for P-TEFb recognition. Nat. Commun. 3: 842.
6. Vollmuth F, **Geyer M** (2010). Interaction of propionylated and butyrylated histone H3 lysine marks with Brd4 bromodomains. Angew. Chem. Int. Ed. Engl. 49, 6768–6772.
7. Gerlach H, Laumann V, Martens S, Becker CF, Goody RS, **Geyer M** (2010). HIV-1 Nef membrane association depends on charge, curvature, composition and sequence. Nat. Chem. Biol. 6, 46–53.
8. Anand K, Schulte A, Vogel-Bachmayr K, Scheffzek K, **Geyer M** (2008). Structural insights into the cyclin T1-Tat-TAR RNA transcription activation complex from EIAV. Nat. Struct. Mol. Biol. 15, 1287–1292.
9. Nekrep N, Jabrane-Ferrat N, Wolf HM, Eibl MM, **Geyer M**, Peterlin BM (2002). Mutation in a winged-helix DNA-binding motif causes atypical bare lymphocyte syndrome. Nat. Immunol. 3, 1075–1081.
10. Antz C, **Geyer M**, Fakler B, Schott MK, Guy HR, Frank R, Ruppersberg JP, Kalbitzer HR. (1998). NMR structure of inactivation gates from mammalian voltage-dependent potassium channels. Nature 385, 272–275.

Dr. Annkristin Heine, MD

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Research Expertise

Dr. Heine and her group are interested in the mechanisms governing antigen cross-presentation and cytotoxic T cell induction in the context of anti-tumor immune responses. Effective cancer vaccines should not only induce strong and durable T cell responses, but also optimally modulate the balance between suppressive and stimulatory factors in the tumor microenvironment. The aim of her research is the identification of novel immunotherapeutic strategies that accomplish both, with a special focus on different pathways of dendritic cell licensing and chemokine receptor modulation.

Education / Training

University of Bonn, Germany, Internal Medicine, Hematology, Oncology, Medical Specialist, 2013
University of Tübingen, Germany, Clinical Immunology, MD thesis, 2006
University of Tübingen, Germany; University of Bordeaux, France; Mount Sinai School of Medicine, New York, USA, MD, 2006

Appointments / Positions Held

since 2013
Senior Physician in the Medical Clinic III for Oncology, Hematology and Rheumatology, University of Bonn, Germany
since 2015
Group leader of a clinical-translational junior research group, Institute of Experimental Immunology and Medical Clinic III, University of Bonn, Germany
2013 - 2014
Group leader of a BONFOR junior research group, Institute of Experimental Immunology, University of Bonn, Germany
2010 - 2012 Postdoctoral fellow, Institute of Experimental Immunology, University of Bonn, Germany

Honors / Awards

2015
Lisec-Artz-price for best junior scientist in the field of oncology
2014
Poster Prize for best abstract in the category “Immunotherapy“ of the German Society for Hematology and Oncology
2012
BONFOR young scientists award in the category “post-doc“
2006
Carl-Liebermeister prize for excellent scientific research of medical students

10 Most Relevant Publications for Dr. Annkristin Heine

1. **Heine A**, Schilling J, Grünwald B, Krüger A, Gevensleben H, Held SAE, Garbi N, Kurts C, Brossart P, Knolle P, Diehl L and Höchst B. The induction of human myeloid-derived suppressor cells through hepatic stellate cells is dose-dependently inhibited by the tyrosine kinase inhibitors nilotinib, dasatinib and sorafenib, but not sunitinib. Cancer Immunol Immunotherapy 2016 Mar;65(3):273-82
2. Rittig SM, Haentschel M, Weimer KJ, **Heine A**, Muller MR, Brugger W, Horger MS, Maksimovic O, Stenzl A, Hoerr I, Rammensee HG, Holderried TA, Kanz L, Pascolo S, Brossart P. Long-term survival correlates with immunological responses in renal cell carcinoma patients treated with mRNA-based immunotherapy. Oncoimmunology 2015 Oct 29;5(5)
3. Held SAE*, **Heine A***, Kesper AR, Beckers A, Wolf D, Brossart P. Interferon gamma modulates sensitivity of CML cells to tyrosine kinase inhibitors. Oncoimmunology, in press
4. **Heine A**, Held SAE, Daecke SN, Riethausen K, Flores C, Kurts C and Brossart P. The VEGF-receptor inhibitor axitinib impairs dendritic cell phenotype and function. PlosOne, 2015 Jun 4;10(6):e0128897
5. Schonberg K, Rudolph J, Vonnahme M, Parampalli Yajnanarayana S, Cornez I, Hejazi M, Manser A, Uhrberg M, Verbeek W, Koschmieder S, Brummendorf TH, Brossart P, **Heine A**, Wolf D. JAK inhibition impairs NK cell function in myeloproliferative neoplasms. Cancer Res. 2015 Apr 1. pii: canres.3198.2014
6. Parampalli Yajnanarayana S, Stübig T, Cornez I, Alchalby H, Schönberg K, Rudolph J, Triviai I, Wolschke C, **Heine A**, Brossart P, Kröger N, Wolf D. JAK1/2 inhibition impairs T cell function in vitro and in patients with myeloproliferative neoplasms. Br J Haematol. 2015 Mar 30
7. **Heine A**, Brossart P, Wolf D. Ruxolitinib is a potent immunosuppressive compound: is it time for anti-infective prophylaxis? Blood. 2013 Nov 28;122(23):3843-4
8. **Heine A**, Held SAE, Daecke SN, Wallner S, Yajnanarayana SM, Kurts C, Wolf D and Brossart P. The JAK-inhibitor Ruxolitinib impairs dendritic cell function in vitro and in vivo. Blood. 2013 Aug 15;122(7):1192-202
9. Held SAE, Duchardt KM, Tenzer S, Rückrich T, von Schwarzenberg K, Bringmann A, Schild HJ, Driessen C, Brossart P and **Heine A**. Imatinib mesylate and nilotinib affect MHC-class I presentation by modulating the proteasomal processing of antigenic peptides. Cancer Immunol Immunother. 2012 Nov 25.
10. **Heine A**, Grünebach F, Holderried T, Appel S, Weck MM, Dörfel D, Sinzger C, Brossart P. Transfection of dendritic cells with in vitro-transcribed CMV RNA induces polyclonal CD8+- and CD4+-mediated CMV-specific T cell responses. Mol Ther. 2006 Feb;13(2):280-8.

*These authors contributed equally

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Research Expertise

Michael Hölzel has long-standing research expertise in the field of tumor biology and functional genomics with a particular focus on neural crest derived tumors such as melanoma. Currently his group investigates how the immune system crosstalks with the tumor cells in response to danger and proinflammatory signals released by therapy-induced tumor tissue injury. A central hypothesis is that this reciprocal communication drives therapy relapse due to rewiring of survival and differentiation pathways in tumor cells. This knowledge is critically needed for the rational combination of immunotherapies and targeted signal transduction inhibitors in the clinic.

Education / Training

University of Munich, Germany, Medicine, MD thesis, 2004
University of Munich, Germany, Medicine MD, 2003

Appointments / Positions Held

2012 - present
W2 Professor, Unit for RNA Biology, Institute of Clinical Chemistry and Clinical Pharmacology, University of Bonn, Germany
2007 - 2011
Postdoctoral Fellow, Laboratory of Rene Bernards, The Netherlands Cancer Institute, Amsterdam, The Netherlands
2003 - 2006
Residency Hematology/Oncology, University Hospital Munich (LMU), Germany

Honors / Awards

2002
Scholarship „Harvard-Munich Alliance“
1999
Scholarship “Studienstiftung des deutschen Volkes”

Most Relevant Publications for Prof. Michael Hölzel

1. Riesenberg R, Groetchen A, Siddaway R, Bald T, Reinhardt J, Smorra D, Kohlmeyer J, Renn M, Phung B, Aymans P, Schmidt T, Hornung V, Davidson I, Goding CR, Jönsson G, Landsberg J, Tüting T, **Hölzel M** (2015) MITF and c-Jun antagonism interconnects melanoma dedifferentiation with pro-inflammatory cytokine responsiveness and myeloid cell recruitment. Nature Communications. 6:8755´
2. **Hölzel M**, Landsberg J, Glodde N, Bald T, Rogava M, Riesenberg S, Becker A, Jönsson G, Tüting T. (2015epub) A Preclinical Model of Malignant Peripheral Nerve Sheath Tumor-like Melanoma Is Characterized by Infiltrating Mast Cells. Cancer Res. 76(2):251-63.
3. Bald, T., Landsberg, J., Lopez-Ramos, D., Renn, M., Glodde, N., Jansen, P., Gaffal, E., Steitz, J., Tolba, R., Kalinke, U., Limmer, A., Jönsson, G., **Hölzel, M.**, Tüting T (2014). Immune-cell poor melanomas benefit from PD-1 blockade after targeted type I IFN activation. Cancer Discovery, 2014 Mar 3. Epub ahead of print.
4. Bald, T., Quast, T., Landsberg, J., Rogava, M., Glodde, N., Lopez-Ramos, D., Kohlmeyer, J., Riesenberg, S., van den Boorn-Konijnenberg, D., Hömig-Hölzel, C., Reuten, R. Schadow, B., Weighardt, I., Wenzel, D., Helfrich, I., Schadendorf, D., Bloch, W.,. Bianchi, M.E., Koch, M., Fleischmann, B.K., Förster, I., Kastenmüller, W., Kolanus, W., **Hölzel, M.***, Gaffal, G.*, Tüting, T* (*corresponding authors). (2014). Ultraviolet radiation-induced inflammation promotes angiotropism and metastasis in melanoma. Nature, 507, 109-13.
5. Landsberg, J., Kohlmeyer, J., Renn, M., Bald, T., Rogava, M., Cron, M., Fatho, M., Lennerz, V., Wölfel, T., **Hölzel, M.**, Tüting, T. (2012) Melanomas resist T-cell therapy through inflammation-induced reversible dedifferentiation. Nature, 490, 412–416.
6. Huang, S., **Hölzel, M.**, Knijnenburger, T., Schlicker, A., Roepman, P., McDermott, U., Garnett, M.J., Grernrum, W., Sun, C., Prahallad, A., Groenendijk, F.H., Mittempergher, L., Nijkamp, W., Neefjes, J., Salazar, R., Ten Dijke, P., Uramoto, H., Tanaka, F., Beijersbergen, R.L., Wessels, L.F., Bernards, R. (2012) MED12 controls the response to multiple cancer drugs through regulation of TGFβ receptor signaling. Cell, 151, 937–950.
7. **Hölzel, M.***, Huang, S.*, Koster, J., Ora, I., Lakeman, A., Caron, H., Nijkamp, W., Xie, J., Callens, T., Asgharzadeh, S., Seeger, RC., Messiaen, L., Versteeg, R., Bernards, R. NF1 is a tumor suppressor in neuroblastoma that determines retinoic acid response and disease outcome. Cell, 2010; 142, 218–229.
8. **Hölzel M**, Orban M, Hochstatter J, Rohrmoser M, Harasim T, Malamoussi A, Kremmer E, Längst G, Eick D. (2010) Defects in 18 S or 28 S rRNA processing activate the p53 pathway. J Biol Chem., 285, 6364-70.
9. Huang S, Laoukili J, Epping MT, Koster J, **Hölzel M.,** Westerman BA, Nijkamp W, Hata A, Asgharzadeh S, Seeger RC, Versteeg R, Beijersbergen RL, Bernards R. (2009) ZNF423 is critically required for retinoic acid-induced differentiation and is a marker of neuroblastoma outcome. Cancer Cell, 15, 328-40.

*These authors contributed equally

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Research Expertise

The scientific focus of his group are cellular interactions and cell-cell communication in the context of acute infections. Central techniques are live intravital imaging and histocytometry.

Education / Training

Laboratory of Systems Biology NIH/USA,
Ronald N. Germain, 2008 - 2012
Technical University of Munich, Germany, Specialization Infectious Disease, 2008
Technical University of Munich, Germany, Medicine, MD thesis, 2003
Technical University of Munich, Germany, Medicine, 1997 - 2002
Universities of Regensburg, Germany, Medicine, 1995 - 1997

Appointments / Positions Held

2013
Associate Professor, University of Bonn, Germany
2008 - 2012
Postdoctoral Fellow, NIH/Besthesda USA
2002 - 2008
Clinical Fellow/Post-Doc, Technical University of Munich, Germany

Honors / Awards

2012
CIG - Best Paper Award NIH
2003
Dietmar-Zumpf-Promotions Preis

10 Most Relevant Publications for Prof. Wolfgang Kastenmüller

1. Eickhoff S, Brewitz A, Gerner MY, Klauschen F, Komander K, Hemmi H, Garbi N, Kaisho T, Germain RN, **Kastenmuller W.** 2015. Robust Anti-viral Immunity Requires Multiple Distinct T Cell-Dendritic Cell Interactions. Cell 2015 Sep 10;162(6):1322-37
2. Franklin BS, Bossaller L, De Nardo D, Ratter JM, Stutz A, Engels G, Brenker C, Nordhoff M, Mirandola SR, Al-Amoudi A, Mangan MS, Zimmer S, Monks BG, Fricke M, Schmidt RE, Espevik T, Jones B, Jarnicki AG, Hansbro PM, Busto P, Marshak-Rothstein A, Hornemann S, Aguzzi A, **Kastenmuller W**, Latz E. The adaptor ASC has extracellular and 'prionoid' activities that propagate inflammation. Nat Immunol: 2014 Aug;15(8):727-37.
3. Honda T, Egen JG, Lammermann T, **Kastenmuller W**, Torabi-Parizi P, Germain RN. 2014. Tuning of antigen sensitivity by T cell receptor-dependent negative feedback controls T cell effector function in inflamed tissues. Immunity 40: 235-47
4. Bald T, Quast T, Landsberg J, Rogava M, Glodde N, Lopez-Ramos D, Kohlmeyer J, Riesenberg S, van den Boorn-Konijnenberg D, Hömig-Hölzel C, Reuten R, Schadow B, Weighardt H, Wenzel D, Helfrich I, Schadendorf D, Bloch W, Bianchi ME, Lugassy C, Barnhill RL, Koch M, Fleischmann BK, Förster I, **Kastenmuller W**, Kolanus W, Hölzel M, Gaffal E, Tüting T. Ultraviolet-radiation-induced inflammation promotes angiotropism and metastasis in melanoma. Nature. 2014 Mar 6;507(7490):109-13.
5. Lämmermann T, Afonso PV, Angermann BR, Wang JM, **Kastenmuller W**, Parent CA, Germain RN.: Neutrophil swarms require LTB4 and integrins at sites of cell death in vivo. Nature. 2013 Jun 20;498(7454):371-5.
6. **Kastenmuller W.**, Brandes, M., Wang, Z., Herz, J., Egen YG., Germain, RN.: Peripheral pre-positioning and local CXCL9-mediated guidance orchestrate rapid memory CD8+ T cell responses in the lymph node. Immunity, 2013, Jan 24:
7. Naik S, Bouladoux N, Wilhelm C, Molloy MJ, Salcedo R, **Kastenmuller W**, Deming C, Quinones M, Koo L, Conlan S, Spencer S, Hall JA, Dzutsev A, Kong H, Campbell DJ, Trinchieri G, Segre JA, Belkaid Y. 2012. Compartmentalized control of skin immunity by resident commensals. Science 337: 1115-9
8. **Kastenmuller W.**, Torabi-Parizi, P., Subramanian, S., Lammermann, T., Germain, R.N.: A spatially-organized multicellular innate immune response in the lymph node limits the systemic spread of tissue-invasive pathogens. Cell, 2012, Sep 14; 150(6):1235-48.
9. Gerner, M.Y., **Kastenmuller W.**, Ifrim, I., Kabat J., Germain, R.N.: Histo-Cytometry: in situ multiplex cell phenotyping, quantification, and spatial analysis applied to dendritic cell subset analysis in lymph nodes. Immunity, 2012 Aug 24;37(2):364-76.
10. **Kastenmuller W**, Gasteiger G, Gronau JH, Baier R, Ljapoci R, Busch DH, Drexler I. 2007. Cross-competition of CD8+ T cells shapes the immunodominance hierarchy during boost vaccination. J Exp Med 204: 2187-98

Dr. Martin Schlee, PhD

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Rheinische Friedrich-Wilhelms-University Bonn, University Hospital, Institute of Clinical Chemistry and Clinical Pharmacology

E-Mail: martin.schlee@uni-bonn.de

Research Expertise

The focus of Martin Schlee's research group is immune recognition and immune tolerance of viral and endogenous nucleic acids. Nucleic acid receptors of the innate immune system initiate and control the antiviral immune response of the infected organism. The detection of pathogenic RNA/DNA by nucleic acid receptors is based on recognition of unusual RNA/DNA localization, structure and modifications, so-called pattern recognition motifs. The challenge here is that the innate immune system detects sensitively pathogenic nucleic acids without false activation by the endogenous nucleic acids. While an insensitive recognition favors the spread of infection, an excessive immune detection of nucleic acids leads to autoimmune diseases. The group has identified and characterized recognition motifs of the cytosolic DNA receptor cGAS and the cytosolic RNA receptor RIG-I and endogenous as well as viral RNA modifications that prevent recognition by RIG-I.

Education / Training

University of Munich, Germany, Biochemistry, PhD, 2003
University of Bielefeld, Germany, Biochemistry, Diploma, 1999

Appointments / Positions Held

2006 - present
Group Leader, Institute of Clinical Chemistry and Clinical Pharmacology, University of Bonn, Germany
2005 - 2006
PostDoc, Division of Clinical Pharmacology, University of Munich, Germany
2003 - 2005
PostDoc, Institute of Clinical Molecular Biology and Tumor Genetics, Helmholtz Center Munich, Germany

Honors / Awards

2009
Bonfor Junior research Group award

10 Most Relevant Publications for Dr. Martin Schlee

1. Herzner AM, Hagmann CA, Goldeck M, Wolter S, Kübler K, Wittmann S, Gramberg T, Andreeva L, Hopfner KP, Mertens C, Zillinger T, Jin T, Xiao TS, Bartok E, Coch C, Ackermann D, Hornung V, Ludwig J, Barchet W, Hartmann G and **Schlee M.** Sequence-specific activation of cGAS by Y-form DNA structures as found in primary HIV-1 cDNA. Nat Immunol. 2015. 16(10):1025-33
2. Schuberth-Wagner C, Ludwig J, Bruder AK, Herzner AM, Zillinger T, Goldeck, M, Schmidt T, Schmid-Burgk L, Kerber R, Wolter S, Stümpel JP, Roth A, Bartok E, Drosten C, Coch C, Hornung V, Barchet W, Kümmerer BM, Hartmann G, **Schlee M.** A conserved histidine in the RNA sensor RIG-I controls immune tolerance to N1-2'O-methylated self RNA. Immunity. 2015. 43(1):41-51
3. Goubau D, **Schlee M***, Deddouche S, Pruijssers AJ, Zillinger T, Goldeck M, Schuberth C, Van der Veen AG, Fujimura T, Rehwinkel J, Iskarpatyoti JA, Barchet W, Ludwig J, Dermody TS, Hartmann G, Reis e Sousa C. Antiviral immunity via RIG-I-mediated recognition of RNA bearing 5'-diphosphates. Nature. 2014. 514(7522):372-5 (JIF 41.3)
4. Coch C, Lück C, Schwickart A, Putschli B, Renn M, Höller T, Barchet W, Hartmann G, **Schlee M.** A Human In Vitro Whole Blood Assay to Predict the Systemic Cytokine Response to Therapeutic Oligonucleotides Including siRNA. PLoS One. 2013. 8: e71057. doi:10.1371/journal.pone.0071057
5. Hagmann CA, Herzner AM, Abdullah Z, Zillinger T, Jakobs C, Schuberth C, Coch C, Higgins PG, Wisplinghoff H, Barchet W, Hornung V, Hartmann G, **Schlee M.** RIG-I detects triphosphorylated RNA of Listeria monocytogenes during infection in non-immune cells. PLoS One. 2013. 8(4): e62872
6. Abdullah Z, **Schlee M***, Roth S, Mraheil MA, Barchet W, Böttcher J, Hain T, Geiger S, Hayakawa Y, Fritz JH, Civril F, Hopfner K-P, Kurts C, Ruland J, Hartmann G, Chakraborty T and Knolle PA. RIG-I detects infection with live Listeria by sensing secreted bacterial nucleic acids. EMBO J. 2012. 31, 4153 – 4164.
7. Coch C, Busch N, Wimmenauer V, Hartmann E, Janke M, Abdel-Mottaleb MMA, Lamprecht A, Ludwig J, Barchet W, **Schlee M*** , Hartmann G*. Higher activation of TLR9 in plasmacytoid dendritic cells by microbial DNA compared with self-DNA based on CpG-specific recognition of phosphodiester DNA. Journal of Leukocyte Biology. 2009. 86: 663-670,
8. **Schlee M**, Roth A, Hornung V, Hagmann CA, Wimmenauer V, Barchet W, Coch C, Janke M, Mihailovic A, Wardle G, Juranek S, Kato H, Kawai T, Poeck H, Fitzgerald KA, Takeuchi O, Akira S, Tuschl T, Latz E, Ludwig J, Hartmann G. Recognition of 5' Triphosphate by RIG-I Helicase Requires Short Blunt Double-Stranded RNA as Contained in Panhandle of Negative-Strand Virus. Immunity. 2009. 31(1): 25-34
9. **Schlee M**, Hölzel M, Bernard S, Mailhammer R, Schuhmacher M, Reschke J, Eick D, Marinkovic D, Wirth T, Rosenwald A, Staudt LM, Eilers M, Baran-Marszak F, Fagard R, Feuillard J, Laux G, Bornkamm GW. C-myc activation impairs the NF-kappaB and the interferon response: implications for the pathogenesis of Burkitt's lymphoma. Int J Cancer. 2007 120(7):1387-95
10. **Schlee M**, Krug T, Gires O, Zeidler R, Hammerschmidt W, Mailhammer R, Laux G, Sauer G, Lovric J, Bornkamm GW. Identification of Epstein-Barr virus (EBV) nuclear antigen 2 (EBNA2) target proteins by proteome analysis: activation of EBNA2 in conditionally immortalized B cells reflects early events after infection of primary B cells by EBV. J Virol. 2004 78(8):3941-52

Dr. Andreas Schlitzer, PhD

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Research Expertise

The aim of our research is to understand the complexity and function of dendritic cells and monocytes during health and disease. A major focus lies on the developmental processes leading to the functional specialization of dendritic cell subsets and monocytes. To analyse these highly heterogeneous compartments we use state of the art technologies such as single cell mRNA sequencing, multi-colour fish and advanced flow cytometry. Taken together we are investigating how the development of dendritic cells and monocytes shapes their functional specialization during homeostasis and disease.

Education / Training

Technical University of Munich, Germany, Immunology, PhD, 2012
University of Manchester, UK, Immunology, Master of Science, 2008
University of Marburg, Germany, Molecular Biology, Bachelor of Science, 2007

Appointments / Positions Held

2015 - present
Emmy Noether research group leader, Myeloid cell biology, University of Bonn, Germany
2012 - 2015
Postdoctoral Fellow, SIngapore Immunology Network, Singapore

Honors / Awards

2015
Bright Sparks award of the German Immunologists association
2013 - 2016
Junior Invstigator fellowship, Biomedical research council, Singapore

10 Most Relevant Publications for Dr. Andreas Schlitzer

1. Paul, F., Arkin, Y., Giladi, A., Jaitin, D. A., Kenigsberg, E., Keren-Shaul, H., …, **Schlitzer, A.**, et al. (2015). Transcriptional Heterogeneity and Lineage Commitment in Myeloid Progenitors. Cell, 1–16.
2. **Schlitzer, A.**, Sivakamasundari, V., Chen, J., Bin Sumatoh, H. R., Schreuder, J., Lum, J., et al. (2015). Identification of cDC1- and cDC2-committed DC progenitors reveals early lineage priming at the common DC progenitor stage in the bone marrow. Nat Immunol, 1–13. http://doi.org/10.1038/ni.3200
3. **Schlitzer, A.**, McGovern, N., & Ginhoux, F. (2015). Dendritic cells and monocyte-derived cells: Two complementary and integrated functional systems. Seminars in Cell & Developmental Biology. http://doi.org/10.1016/j.semcdb.2015.03.011
4. Becher, B.*, **Schlitzer, A.***, Chen, J., Mair, F., Sumatoh, H. R., Teng, K. W. W., et al. (2014). High-dimensional analysis of the murine myeloid cell system. Nat Immunol, 15(12), 1181–1189. doi:10.1038/ni.3006. Co-first author
5. McGovern*, **Schlitzer, A.***, N., Gunawan, M., Jardine, L., Shin, A., Poyner, E., et al. (2014). Human dermal CD14+ cells are a transient population of monocyte-derived macrophages. Immunity, 41(3), 465–477. doi:10.1016/j.immuni.2014.08.006. Co-first author
6. **Schlitzer, A.**, & Ginhoux, F. (2014). Organization of the mouse and human DC network. Current Opinion in Immunology, 26, 90–99. doi:10.1016/j.coi.2013.11.002
7. Jakubzick, C., Gautier, E. L., Gibbings, S. L., Sojka, D. K., **Schlitzer, A.**, Johnson, T. E., et al. (2013). Minimal differentiation of classical monocytes as they survey steady-state tissues and transport antigen to lymph nodes. Immunity, 39(3), 599–610. doi:10.1016/j.immuni.2013.08.007
8. **Schlitzer, A.**, McGovern, N., Teo, P., Zelante, T., Atarashi, K., Low, D., et al. (2013). IRF4 transcription factor-dependent CD11b+ dendritic cells in human and mouse control mucosal IL-17 cytokine responses. Immunity, 38(5), 970–983. doi:10.1016/j.immuni.2013.04.011
9. **Schlitzer, A.**, Heiseke, A. F., Einwächter, H., Reindl, W., Schiemann, M., Manta, C.- P., et al. (2012). Tissue-specific differentiation of a circulating CCR9- pDC-like common dendritic cell precursor. Blood, 119(25), 6063–6071. doi:10.1182/blood- 2012-03-418400
10. **Schlitzer, A.**, Loschko, J., Mair, K., Vogelmann, R., Henkel, L., Einwächter, H., et al. (2011). Identification of CCR9- murine plasmacytoid DC precursors with plasticity to differentiate into conventional DCs. Blood, 117(24), 6562–6570. doi:10.1182/blood- 2010-12-326678

Prof. Sven Wehner, PhD

Department of Surgery

new Member since 2015

Rheinische Friedrich-Wilhelms-University Bonn, Department of Surgery, Immune Pathophysiology

E-Mail: sven.wehner@ukb.uni-bonn.de

Research Expertise

The aim of our research is to identify (neuro)-immunological mechanisms contributing to acute (postoperative) and chronic functional disturbances of the GI tract. In a series of publications our group demonstrated that interactions of resident immune cells and the enteric nervous system trigger postoperative bowel wall inflammation. This local neuroinflammation can be effectively modified by intrinsic and extrinsic neurotransmitters. With international collaborators we also investigate the neuroimmunology of chronic inflammatory bowel diseases. Currently, we are aiming to understand the initial trigger mechanisms of intestinal inflammation as well as to identify novel neuro-immune modulatory pathways. This will help us to gather a comprehensive understanding of acute and chronic intestinal inflammatory disorders.

Education / Training

University of Bonn, Molecular Medicine, venia legendi, 2012
University of Bonn, Cell Biology, PhD thesis, 2003
University of Bonn, Biology, Diploma, 1999

Appointments / Positions Held

2016 - present
Full Professor for Immune Pathophysiology, University of Bonn, Germany
2013 - 2015
Asociate Professor, AMC, Amsterdam, Netherlands
2006 - 2013
Independent workgroup leader and laboratory head, Department of Surgery, University Bonn, Germany
2003 - 2006
Postdoctoral Fellow, Department of Surgery, University of Bonn, Germany
1999 - 2003
Research Scientist at Celonic GmBH, Research Center Jülich; Doctoral Student, Institute of Cell Biology, University of Bonn, Germany

Honors / Awards

2013
'Poster of Distinction; Digestive Diesease Week, May 19-22 San Diego
2007
Best of Abstracts, Detusche Gesellschaft für Chirurgie
2005
Best of Abstracts, Deutsche Gesellschaft für Chirurgie'

10 Most Relevant Publications for Prof. Sven Wehner

1. Ochoa-Cortes F, Turco F, Linan-Rico A, Soghomonyan S, Whitaker E, **Wehner S**, Cuomo R, Christofi FL. Enteric Glial Cells: A New Frontier in Neurogastroenterology and Clinical Target for Inflammatory Bowel Diseases. 2016 Feb;22(2):433-49
2. Stein K, Stoffels M, Lysson M, Schneiker B, Dewald O, Krönke G, Kalff JC, **Wehner S**. A role for 12/15-lipoxygenase-derived proresolving mediators in postoperative ileus: protectin DX-regulated neutrophil extravasation. J Leukoc Biol. 2016 Feb;99(2):231-9.
3. Hong GS, Schwandt T, Stein K, Schneiker B, Kummer MP, Heneka MT, Kitamura K, Kalff JC, **Wehner S**. Effects of macrophage-dependent peroxisome proliferator-activated receptor λ signalling on adhesion formation after abdominal surgery in an experimental model. Br J Surg. 2015 Nov;102(12):1506-16.
4. Stoffels B, Hupa KJ, Snoek A, van Bree A, Stein K, Schwandt T, Vilz T, Lysson M, van´t Veer, Hornung V, Kalff JC, de Jonge WJ, **Wehner S.** Interleukin-1 receptor signaling contributes to postoperative ileus by targeting enteric glial cells. Gastroenterology 2014, 146(1): 176-87.
5. Glowka TR, Steinebach A, Schwandt T, Kranz A, Kalff JC, **Wehner S**. CGRP release during abdominal surgery mediates small bowel inflammation and postoperative ileus in a macrophage dependent Neurogastroenterol Motil, 2015, 27(7):1039-49.
6. Vilz TO, Sommer MD, Kahl P, Pantelis D, Kalff JC, **Wehner S**. Oral CPSI-2364 treatment prevents postoperative ileus in swine without impairment of anastomotic healing. Cell.Physiol.Biochem , 2013;32(5):1362-1373.
7. Schwandt T, Schumak B, Jüngerkes F, Gielen G, Schmidbauer P, Klocke K, Staratscheck-Jox A, van Rooijen N, Kraal G, Ludwig-Portugall I, **Wehner S**, Kalff JC, Kirschning , Coch C, Kalinke U, Wenzel J, Kurts C, Zawatzky R, Holzmann B, Layland L, Schultze J, Burgdorf S, Haan J, Knolle J, Franken L, Weber O, Limmer A. Expression of type I interferon by splenic macrophages suppresses adaptive immunity during sepsis. EMBO J, 2012; 31(1):201-13.
8. Engel DR, Koscielny A **Wehner S**, Maurer J, Schiwon M, Franken L, Schumak B, Limmer A, Sparwasser T, Hirner T, Knolle PA, Kalff JC KurtsC. T helper type 1 memory cells disseminate postoperative ileus over the entire intestinal tract. Nature Medicine 2010, 16(12), 1407-13.
9. **Wehner S**, Straesser S, Pantelis D, Sielecki T, de la Cruz VF, Hirner A, Kalff JC. Inhibition of p38 MAP kinase pathway as a target for prophylaxis of postoperative ileus in mice. Gastroenterology 2009;136(2):619-629.
10. **Wehner S**, Behrendt FF, Lyutenski B, Lysson M, Bauer AJ, Kalff JC. Inhibition of Macrophage Function Prevents Intestinal Inflammation and Postoperative Ileus in Rodents. Gut 2007; 56(2):176-85.

Prof. Christoph Wilhelm, PhD

Institute of Clinical Chemistry and Clinical Pharmacology

new Member since 2015

Rheinische Friedrich-Wilhelms-University Bonn, Institute of Clinical Chemistry and Clinical Pharmacology
E-Mail: christoph.wilhelm@uni-bonn.de

Research Expertise

Prof. Wilhelm has longterm experience in the research of a unique group of immune cells termed innate lymphoid cells (ILC). His scientific focus is to understand how these cells protect the barrier sites of our body such as the lung, skin and gut and to understand the maintenance of ILC mediated barrier immunity in the context of malnutrition. The overall aim of this research is to understand the link between westernization and the growing health problem of chronic inflammatory conditions such as inflammatory bowl disease (IBD) and asthma.

Education / Training

National Institute for Medical Research, London, UK, Immunology, PhD, 2011
University of Munich, Germany, Biology MSc (Diploma), 2007

Appointments / Positions Held

2015 - present
Assistant Professor for Immunopathology, University of Bonn, Germany
2015 - present
Principal Investigator, Department of Medicine, University of Bonn, Germany
2011- 2015
Postdoctoral Fellow, National Institutes of Health, USA

Honors / Awards

2015
NRW-Return Programme, funding to establish an independent research programme
2014
NIH Fellow Award for Research Excellence (FARE award)
2014
The CIG best paper award, Best publication in cytokine research for postdoctoral fellow working at the NIH and FDA
2014
Keystone Symposia travel fellowship for oral presentation at the Keystone Symposia 2014
2013
Society of Mucosal Immunology travel fellowship
2012
NIH Fellow Award for Research Excellence (FARE award)
2012
Human Frontiers Science program postdoctoral fellowship
2010
British Society for Immunology travel fellowship for the International Congress of Immunology
2010
European Federation of Immunological Societies travel fellowship for the International Congress of Immunology
2009
European Federation of Immunological Societies travel fellowship for the European Congress of Immunology
2008
MUGEN travel fellowship for ENII-MUGEN Immunology Summer School
2007
Medical Research Council (MRC) Ph.D. studentship

10 Most Relevant Publications for Prof. Christoph Wilhelm

1. Zhong C, Cui K, **Wilhelm C**, Hu G, Mao K, Belkaid Y, Zhao K, Zhu J.GATA3 dictates NKp46+ T-bet/RORλt-co-expressing ILC3 development and regulates ILC3 homeostasis and function. Nature Immunology 2016.
2. Askenase MH, Han SJ, Byrd AL, Morais da Fonseca D, Bouladoux N, **Wilhelm C**, Konkel JE, Hand TW, Lacerda-Queiroz N, Su XZ, Trinchieri G, Grainger JR, Belkaid Y. Bone-Marrow-Resident NK Cells Prime Monocytes for Regulatory Function during Infection. Immunity 2015.
3. Naik, S., N. Bouladoux, J.L. Linehan, S.J. Han, O.J. Harrison, **C. Wilhelm**, S. Conlan, S. Himmelfarb, A.L. Byrd, C. Deming, M. Quinones, J.M. Brenchley, H.H. Kong, R. Tussiwand, K.M. Murphy, M. Merad, J.A. Segre, and Y. Belkaid. 2015. Commensal-dendritic-cell interaction specifies a unique protective skin immune signature. Nature 2015.
4. **Wilhelm C***, Spencer SP*, Yang Q, Hall JA, Bouladoux N, Boyd A, Nutman TB, Urban JF Jr, Wang J, Ramalingam TR, Bhandoola A, Wynn TA, Belkaid Y. Adaptation of innate lymphoid cells to a micronutrient deficiency promotes type 2 barrier immunity. Science 2014 *Primary authors contributed equally to this work.
5. Turner JE*, Morrison PJ*, **Wilhelm C**, Wilson M, Ahlfors H, Renauld JC, Panzer U, Helmby H and Stockinger B. (2013) IL-9-mediated survival of type 2 innate lymphoid cells promotes damage control in helminth-induced lung inflammation. J Exp Med *Primary authors contributed equally to this work.
6. Naik S, Bouladoux N, **Wilhelm C**, Molloy MJ, Salcedo R, Kastenmuller W, Deming C, Quinones M, Koo L, Conlan S, Spencer S, Hall JA, Dzutsev A, Kong H, Campbell DJ, Trinchieri G, Segre JA, Belkaid Y. (2012) Compartmentalized control of skin immunity by resident commensals. Science
7. **Wilhelm C**, Turner JE, Van Snick J, and Stockinger B. The many lives of IL-9: a question of survival? Nature Immunology 2012.
8. **Wilhelm C**, Hirota K, Stieglitz B, Van Snick J, Tolaini M, Lahl K, Sparwasser T, Helmby H and Stockinger B. (2011) An IL-9 fate reporter demonstrates the induction of an innate IL-9 response in lung inflammation. Nature immunology
9. Li Y, Innocentin S, Withers DR, Roberts NA, Gallagher AR, Grigorieva EF, **Wilhelm C** and Veldhoen M. (2011) Exogenous stimuli maintain intraepithelial lymphocytes via aryl hydrocarbon receptor activation. Cell
10. Veldhoen M, Uyttenhove C, van Snick J, Helmby H, Westendorf A Buer J, Martin B, **Wilhelm C** and Stockinger B. (2008) Transforming growth factor-beta 'reprograms' the differentiation of T helper 2 cells and promotes an interleukin 9-producing subset. Nature immunology

Hofgarten University of Bonn
by Frank Luerweg

Biosketches

Core Members

Dr. Ashraf Al-Amoudi, PhD

German Centre for Neurodegenerative Diseases (DZNE) and
center of advanced european studies and research (caesar)

German Centre for Neurodegenerative Diseases (DZNE) and center of advanced european studies and research (caesar)

E-Mail: ashraf.al-amoudi@dzne.de

Research Expertise

Structural biology, cryo-electron tomography, intercellular adhesion junctions, synapses, neurodegenerative diseases.

Education / Training

University of Lausanne, Switzerland, Life science/EM Structural, Biology, PhD, 2004
University of Lausanne, Switzerland, Physics, Science diploma, 1999
Birzeit University, West Bank, Palestine, Physics, B.Sc, 1997

Appointments / Positions Held

Jan 2010 - present
Group leader, Cryo-Electron Microscopy and Tomography in neurodegenerative diseases, German Centre for Neurodegenerative Disease (DZNE) and center of advanced european studies andresearch (caesar), Bonn
2005 - 2009
Postdoctoral fellow, Structural and Computational Biology Unit, European Molecular Biology Laboratory (EMBL), Heidelberg, Germany
2004 - 2005
Postdoctoral, Laboratory of Ultrastructure Analysis University of Lausanne, Switzerland

Honors / Awards

2014 - 2017
SFB, Transregio Collaborative Research, TRR83
2012 - 2017
Cluster of Excellence, ImmunoSensation, DFG
2010 - 2014
Wellcome Trust and MRC Career Development Awards - declined
2007 - 2009
Marie Curie Intra-European Fellowship
2006 - 2007
EMBO Fellowship
2005
Prize of excellence for young researchers for the PhD work, Faculty of Biology and Medicine, University of Lausanne, Lausanne, Switzerland

10 Most Relevant Publications for Dr. Ashraf Al- Amoudi

1. Gunkel M, Schöneberg J, Alkhaldi W, Irsen S, Noé F, Kaupp UB, **Al-Amoudi A**. 2015. Higher-order architecture of rhodopsin in intact photoreceptors and its implication for phototransduction kinetics. pii: S0969-2126(15)00039-8. doi: 10.1016/j.str.2015.01.015.
2. Franklin BS, Bossaller L, De Nardo D, Ratter JM, Stutz A, Engels G, Brenker C, Nordhoff M, Mirandola SR, **Al-Amoudi A**, Mangan MS, Zimmer S, Monks BG, Fricke M, Schmidt RE, Espevik T, Jones B, Jarnicki AG, Hansbro PM, Busto P, Marshak-Rothstein A, Hornemann S, Aguzzi A, Kastenmüller W, Latz E. 2014. The adaptor ASC has extracellular and 'prionoid' activities that propagate inflammation. Nat Immunol. 727-37.
3. **Al-Amoudi A**, Frangakis AS. 2013. Three-dimensional visualization of the molecular architecture of cell-cell junctions in situ by cryo-electron tomography of vitreous sections. Methods Mol Biol 961: 97-117.
4. **Al-Amoudi A**, Castaño-Diez D, Devos DP, Russell RB, Johnson GT, Frangakis AS. The three-dimensional molecular structure of the desmosomal plaque. Proc. Natl. Acad. Sci. 2011,108, 6480-5.
5. **Al-Amoudi A**, Diez DC, Betts MJ, Frangakis AS. 2007. The molecular architecture of cadherins in native epidermal desmosomes. Nature 450: 832-7.
6. Castano-Diez D, **Al-Amoudi A**, Glynn AM, Seybert A, Frangakis AS. 2007. Fiducial-less alignment of cryo-sections. J Struct Biol 159: 413-23.
7. **Al-Amoudi A**, Studer D, Dubochet J. 2005. Cutting artefacts and cutting process in vitreous sections for cryo-electron microscopy. J Struct Biol 150: 109-21.
8. **Al-Amoudi A**, Dubochet J, Norlen L. 2005. Nanostructure of the epidermal extracellular space as observed by cryo-electron microscopy of vitreous sections of human skin. J Invest Dermatol 124: 764-77.
9. Norlen L, **Al-Amoudi A.** 2004. Stratum corneum keratin structure, function, and formation: the cubic rod-packing and membrane templating model. J Invest Dermatol 123: 715-32.
10. **Al-Amoudi A**, Chang JJ, Leforestier A, McDowall A, Salamin LM, Norlen LP, Richter K, Blanc NS, Studer D, Dubochet J. 2004. Cryo-electron microscopy of vitreous sections. EMBO J 23: 3583-8.

Prof. Regina C. Betz, MD

Institute of Human Genetics

Rheinische Friedrich-Wilhelms-Universität Bonn
Institute of Human Genetics
E-Mail: regina.betz@uni-bonn.de

Research Expertise

The aim of our research is the identification and functional characterization of genes for monogenic and genetically complex hair loss disorders with a major focus on the autoimmune disorder alopecia areata (AA). We have the largest sample of AA patients available worldwide, which includes a current total of more than 2.200 individuals of middle European origin. We have been able to demonstrate the contribution of the HLA-complex and the genes PTPN22, TRAF1/C5 , CTLA4, IL13 and KIAA0350 to the disease risk using candidate gene studies. By the use of genome-wide association studies, meta-analyses, immunochips and functional studies, we just recently identified HLA-DR as a key etiologic driver for AA as well as two loci outside the HLA-region: ACOXL/BCL2L11 and GARP. Future analyses and functional studies will contribute to a comprehensive understanding of AA.

Education / Training

University of Bonn, Germany, Human Genetics, Habilitation, 2009
University of Bonn, Germany, Human Genetics, Medical Specialist, 2007
Karolinska Institute, Stockholm, Sweden University of Saarland Clinical Medicine, Medical license, 1999
University of Saarland, Germany, Medicine, MD thesis, 1998

Appointments / Positions Held

2015 - present W2 Professorship, Institute of Human Genetics, University of Bonn, Germany
2010 - 2015 Heisenberg Professorship, Institute of Human Genetics, University of Bonn, Germany
2009 - 2010 Research Scientist, Institute of Human Genetics, University of Bonn, Germany
2004 - 2009 Independent Head of a Junior Research Group, Institute of Human Genetics, University of Bonn, Germany
2002 - 2004 Postdoctoral fellow, Department of Medical Genetics, University of Antwerp, Belgium
2000 - 2002 Postdoctoral fellow, Institute of Human Genetics, University of Bonn, Germany
1999 - 2000 Research Scientist, Institute of Human Genetics, University of Bonn, Germany

Honors / Awards

2010 - 2015 Heisenberg-Professorship from the DFG
2004 - 2009 Emmy Noether Independent Junior Research Group (DFG)
2008 PRO-SCIENTIA-Sponsorship Award of the Eckhart-Buddecke-Foundation for the advancement of basic medical research
2008 EP-Patent application 07 01 8871.9: “Maintenance of hair growth and treatment of hair loss.” (together with Prof. Nöthen, S. Pasternack Dipl.-biol., and Dr. Al Aboud)
2008 Lecture Prize at the Annual Meeting of the European Hair Research Society in Genoa, Italy
2006 Gottron-Just-Scientific Prize of the University and City of Ulm, Germany
2002 - 2004 Flemish Research Council Postdoctoral Fellowship
2000 - 2002 DFG Postdoctoral Fellowship

10 Most Relevant Publications for Prof. Regina Betz

1. **Betz RC**, Petukhova L, Ripke S, [..] Clynes D, de Bakker PIW, Nöthen MM, Daly MJ, Christiano AM: Meta-analysis of genome-wide association studies in alopecia areata resolves HLA associations and reveals two new susceptibility loci. Nat Commun, Jan 2015 22, 6:5966
2. Basmanav FB, Oprisoreanu AM, Pasternack SM, Thiele H, Fritz G, Wenzel J, Größer L, Wehner M, Wolf S, Fagerberg C, Bygum A, Altmüller J, Rütten A, Parmentier L, El Shabrawi-Caelen L, Hafner C, Nürnberg P, Kruse R, Schoch S, Hanneken S, **Betz RC**. 2014. Mutations in POGLUT1, encoding protein O-glucosyltransferase 1, cause autosomal dominant Dowling-Degos disease. Am J Hum Genet 94:135-143.
3. Pasternack SM, Refke M, Paknia E, Hennies HC, Franz T, Schäfer N, Fryer A, van Steensel M, Sweeney E, Just M, Grimm C, Kruse R, Ferrándiz C, Nöthen MM, Fischer U, **Betz RC**. 2013. Mutations in SNRPE, encoding a core protein of the spliceosome, cause autosomal-dominant hypotrichosis simplex. Am J Hum Genet 92:81-87.
4. Jagielska D, Redler S, Brockschmidt FF, Herold C, Garcia Bartels N, Hanneken S, Eigelshoven S, Refke M, Barth S, Giehl KA, Kruse R, Lutz G, Wolff H, Blaumeiser B, Böhm M, Blume-Peytavi U, Becker T, Nöthen MM, **Betz RC**. 2012. A follow-up study of a genome-wide association scan in alopecia areata: replication of previously identified loci and identification of IL13 and KIAA0350 as new susceptibility loci supported with genome-wide significance. J Invest Dermatol 132:2192-2197.
5. Wen Y, Liu Y, Xu Y, Zhao Y, Hua R, Wang K, Sun M, Li Y, Yang S, Zhang XJ, Kruse R, Cichon S, **Betz RC**, Nothen MM, van Steensel MA, van Geel M, Steijlen PM, Hohl D, Huber M, Dunnill GS, Kennedy C, Messenger A, Munro CS, Terrinoni A, Hovnanian A, Bodemer C, de Prost Y, Paller AS, Irvine AD, Sinclair R, Green J, Shang D, Liu Q, Luo Y, Jiang L, Chen HD, Lo WH, McLean WH, He CD, Zhang X. 2009. Loss-of-function mutations of an inhibitory upstream ORF in the human hairless transcript cause Marie Unna hereditary hypotrichosis. Nat Genet 41: 228-33.
6. Pasternack SM, von Kugelgen I, Aboud KA, Lee YA, Ruschendorf F, Voss K, Hillmer AM, Molderings GJ, Franz T, Ramirez A, Nurnberg P, Nothen MM, **Betz RC**. 2008. G protein-coupled receptor P2Y5 and its ligand LPA are involved in maintenance of human hair growth. Nat Genet 40: 329-34.
7. Hillmer AM, Brockschmidt FF, Hanneken S, Eigelshoven S, Steffens M, Flaquer A, Herms S, Becker T, Kortum AK, Nyholt DR, Zhao ZZ, Montgomery GW, Martin NG, Muhleisen TW, Alblas MA, Moebus S, Jockel KH, Brocker-Preuss M, Erbel R, Reinartz R, **Betz RC**, Cichon S, Propping P, Baur MP, Wienker TF, Kruse R, Nothen MM. 2008. Susceptibility variants for male-pattern baldness on chromosome 20p11. Nat Genet 40: 1279-81.
8. **Betz RC**, Planko L, Eigelshoven S, Hanneken S, Pasternack SM, Bussow H, Van Den Bogaert K, Wenzel J, Braun-Falco M, Rutten A, Rogers MA, Ruzicka T, Nothen MM, Magin TM, Kruse R. 2006. Loss-of-function mutations in the keratin 5 gene lead to Dowling-Degos disease. Am J Hum Genet 78: 510-9.
9. Levy-Nissenbaum E, **Betz RC**, Frydman M, Simon M, Lahat H, Bakhan T, Goldman B, Bygum A, Pierick M, Hillmer AM, Jonca N, Toribio J, Kruse R, Dewald G, Cichon S, Kubisch C, Guerrin M, Serre G, Nothen MM, Pras E. 2003. Hypotrichosis simplex of the scalp is associated with nonsense mutations in CDSN encoding corneodesmosin. Nat Genet 34: 151-3.
10. **Betz RC**, Schoser BG, Kasper D, Ricker K, Ramirez A, Stein V, Torbergsen T, Lee YA, Nöthen MM, Wienker TF, Malin JP, Propping P, Reis A, Mortier W, Jentsch TJ, Vorgerd M, Kubisch C. 2001. Mutations in CAV3 cause mechanical hyperirritability of skeletal muscle in rippling muscle disease. Nat Genet 28: 218-9.

Prof. Anton Bovier, PhD

Institute for Applied Mathematics

Rheinische Friedrich-Wilhelms-Universität Bonn
Institute for Applied Mathematics
E-Mail: bovier@uni-bonn.de

Research Expertise

The main focus of my work concerns the analysis of interacting stochastic systems of many components. This includes a special focus on models from statistical mechanics with an emphasis on disordered models, in particular spin glasses. Apart from classical aspects of equilibrium Gibbs measures I am particularly interested in aspects of long term dynamics such as metastability and aging. More recently I am also interested in application of methods from these areas in models of population genetics, ecology, and neurodegenerative diseases.

Education / Training

Technical University of Berlin, Germany
Mathematics, Habilitation, 1995
The Swiss Federal Institute of Technology (ETH), Zurich
Physics, Dr. sc. nat., 1986
University of Bonn, Germany
Physics, Diploma, 1981

Appointments / Positions Held

2008 - present
Full Professor, Institute for Applied Mathematics
University of Bonn, Germany
2003 - 2008
Full Professor, Mathematics, Technical University, Berlin, Germany
1994 - 2008
Laboratory Head, and 2nd Deputy Director
Weierstrass-Institute for Applied Analysis and Stochastics (WIAS), Berlin
1992 - 1995
Deputy Laboratory Head, WIAS, Berlin
1991 - 1992
Research Associate, Mathematics Department, Bochum University, Germany
1988 - 1991
Research Associate, Physics Department University of Bonn, Germany
1986 - 1988
Visiting Assistant Professor, Mathematics Department University of California, Irvine, CA, USA
1982 - 1986
Assistant, Institute for Theoretical Physics, ETH-Zurich

Honors / Awards

2014
Member of Selection Committee, Heinz-Maier-Leibnitz prize
2013
Elected Fellow, Institute of Mathematical Statistics
2012
Kloosterman Chair, University Leiden, NL
2010
Lady Davies Visiting Professor, Technion, Haifa, IL
2010
Plenary Speaker, Annual Meeting of the German Mathematical Association
2009
EURANDOM Chair, EURANDOM; Eindhoven, NL
2008
Member of the Selection Committee of the Minerva Foundation
2008
Member of the Review Board for Mathematics of the German Research Council
2006
Invited Speaker at the International Congress of Mathematicians, Madrid

10 Most Relevant Publications for Prof. Anton Bovier

1. **Bovier, A.** and den Hollander, F. 2015 Metastability: A Potential-Theoretic Approach. xxi + 581 pp. Grundlehren der mathematischen Wissenschaften Vol. 351, Springer, Charm.
2. Mayer, H, **Bovier, A**. 2015. Stochastic models of T-cell activation. J. Math. Biology 70: 99-132.
3. Arguin, L-P, **Bovier, A**. Kistler, N. 2013. The extremal process of branching Brownian motion. Prob. Theor. Rel. Fields: 157:535-574 .
4. Hölzel, M, **Bovier, A**, Tüting, T. 2013. Plasticity of tumor and immune cells: a source of heterogeneity and a cause for therapy resistance? Nature Reviews Cancer: 13: 365—376.
5. **Bovier, A**, Gayrard V., Svejda, A. 2013. Convergence to extremal processes in random environments and applications to extremal ageing in SK models. Probab Theor. Rel. Fields 157: 151–183.
6. Arguin, L-P, **Bovier, A**, Kistler, N. 2011. The genealogy of extremal particles of branching Brownian motion, Commun. Pure Appl. Math. 64: 1647--1676
7. **Bovier, A**. 2006. Statistical mechanics of disordered systems. A mathematical perspective, 312 + xiv pp, Cambridge Series in Statistical and Probabilistic Mathematics Cambridge University Press Vol. 18
8. **Bovier, A**, Gayrard V, Klein M. 2005. Metastability in reversible diffusion processes II. precise asymptotics for small eigenvalues. J Europ Math Soc 7: 69–99
9. Baake E, Baake M, **Bovier, A**, Klein M. 2005. An asymptotic maximum principle for essentially linear evolution models. J Math Biology 50: 83–114
10. Ben Arous G, **Bovier, A**, Gayrard V. 2003. Glauber dynamics of the random energy model. 2. Aging below the critical temperature. Commun. Math. Phys. 236: 1-54

Prof. Irmgard Förster, PhD

Life and Medical Sciences Institute (LIMES)

Rheinische Friedrich-Wilhelms-Universität Bonn
Life and Medical Sciences Institute (LIMES),
Immunology and Environment, Director
E-Mail: irmgard.foerster@uni-bonn.de

Research Expertise

Prof. Förster has special expertise in the functional characterization of macrophages and dendritic cells using conditional gene targeting techniques. She is interested in cell migration and immune regulation in barrier organs, and has profound experience with mouse models of atopic dermatitis, inflammatory bowel disease and bacterial infection.

Education / Training

University of Cologne, Germany, Genetics, PhD, 1988
University of Marburg, Germany, Human Biology, Diploma, 1985

Appointments / Positions Held

2012 - present
W3 Professor of Immunology and Environment Life and Medical Sciences (LIMES) Institute, University of Bonn
2005 - 2012
Laboratory Head of Molecular Immunology
IUF - Leibniz Institute for Environmental Medicine at the University of Düsseldorf, Germany
2004 - 2012
C3 Professor of Molecular Immunology, Heinrich-Heine-University Düsseldorf
1998 - 2004
Head of the Volkswagen Foundation Research Group
Institute for Medical Microbiology, Immunology and Hygiene and the second Medical Clinic, Technical University of Munich
1997 - 1998
Assistant Professor, Institute for Genetics, University of Cologne
1993 - 1997
Postdoctoral Research Fellow, Institute for Genetics, University of Cologne
1990 - 1993
Postdoctoral Research Fellow, University of California, San Francisco, USA
1988 -1990
Research Fellow, Institute for Genetics, University of Cologne

Honors / Awards

2016 - 2020 Member of the Scientific Committee of the HZI (Helmholtz-Zentrum für Infektionsforschung GmbH)
2016 - 2020 Member of the DFG Immunology Committee
Since 06/2012
Leibniz Chair at the IUF Düsseldorf
1994
Bennigsen Foerder Prize, Ministry of Science and Research of North Rhine-Westphalia
1991 - 1992
Research grant from the DFG
1985 - 1988
Research Scholarship from the Fritz Thyssen Stiftung

10 Most Relevant Publications for Prof. Irmgard Förster

1. Didiovic, S., Opitz, F.V., Holzmann, B., **Förster, I.**, Weighardt, H. 2015. Requirement of MyD88 signaling in keratinocytes for Langerhans cell migration and initiation of atopic dermatitis-like symptoms in mice. Eur J Immunol. 2015 Dec 23
2. Globisch, T, Steiner, N*, Fülle, L*, Lukacs-Kornek, V, Degrandi, D, Dresing, P, Alferink, J, Lang, R, Pfeffer, K, Beyer, M., Weighardt, H, Kurts, C, Ulas, T, Schultze JL and **Förster, I**. 2014. Cytokine-dependent regulation of denditic cell differentiation in the splenic microenvironment. Eur. J. Immunol. 44, 500-510.
3. Köhler, T, Reizis, B, Johnson, RS, Weighardt, H and **Förster, I**. 2012. Influence of hypoxia inducible factor 1a on dendritic cell differentiation and migration. Eur. J. Immunol. 42, 1226-1236.
4. Stutte S, Quast T, Gerbitzki N, Savinko T, Novak N, Reifenberger J, Homey B, Kolanus W, Alenius H and **Förster I**. 2010. Requirement of CCL17 for CCR7- and CXCR4-dependent migration of cutaneous dendritic cells. Proc. Natl. Acad. Sci. USA 107: 8736-41 .
5. Semmling V, Lukacs-Kornek V, Thaiss C, Quast T, Hochheiser K, Panzer U, Rossjohn J, Perlmutter P, Cao J, Godfrey D, Savage P, Knolle P, Kolanus W, **Förster, I*** and Kurts C*. 2010. Alternative cross-priming through CCL17/CCR4-mediated CTL attraction towards NKT cell-licensed dendritic cells. Nat. Immunol. 11: 313-20.
6. Buch T, Polic B, Clausen BE, Weiss S, Akilli Ö, Chang CH, Flavell R, Schulz A, Jonjic S, Waisman A and **Förster, I**. 2006. MHC class II expression through a hitherto unknown pathway supports T helper cell dependent immune responses: implications for MHC class II deficiency. Blood. 107, 1434-1444.
7. Alferink J*, Lieberam I*, Reindl W, Behrens A, Weiß S, Hüser N, Gerauer K, Ross R, Reske-Kunz A, Ahmad-Nejad P, Wagner H and **Förster, I**. 2003. Compartmentalized production of CCL17 in vivo: strong inducibility in peripheral dendritic cells contrasts selective absence from the spleen. J. Exp. Med. 197, 585-599.
8. Lieberam I and **Förster, I**. 1999. The murine beta-chemokine TARC is expressed by subsets of dendritic cells and attracts primed CD4+ T cells. Eur. J. Immunol. 29: 2684-2694.
9. Clausen BE, Burkhardt C, Reith W, Renkawitz R and **Förster, I**. 1999. Conditional gene targeting in macrophages and granulocytes using LysMcre mice. Transg. Res. 8: 265-277.
10. Takeda K*, Clausen BE*, Kaisho T, Tsujimura T, Terada N, **Förster, I*** and Akira S*. 1999. Enhanced Th1 activity and development of chronic enterocolitis in mice devoid of Stat3 in macrophages and neutrophils. Immunity. 10: 39-49.

*These authors contributed equally

Dr. Annett Halle, MD

center of advanced european studies and research (caesar)

center of advanced european studies and research (caesar), Max Planck Research Group „Neuroimmunology“
E-Mail: annett.halle@caesar.de

Research Expertise

Dr. Halle's group studies innate immune mechanisms and microglial function in Alzheimer's disease using cell culture techniques and mouse models of Alzheimer's disease.

Education / Training

Charité – University Medicine Berlin,
Medical Neuroscience, MD thesis, 2005
Free University Berlin, Humboldt University Berlin,
Clinical Medicine, MD, 2003

Appointments / Positions Held

2011 - present
Max-Planck Research Group leader
center of advanced european studies and research (caesar), Bonn, Germany
2009 - 2011
Research fellow and resident in Neuropathology
Department of Neuropathology, Charité – University Medicine Berlin, Germany
2005 - 2008
Postdoctoral fellow and instructor in Internal Medicine, Department of Infectious Diseases, University of Massachusetts, Worcester, USA
2003 - 2005
Medical dissertation and resident in Neurology, Department of Experimental Neurology, Charité – University Medicine Berlin, Germany
2000
Research internship, Department of Cell Biology, Harvard University, Boston, USA

Honors / Awards

2010
Ernst Jung-Career Award for Medical Research
2008
Lydia Rabinowitsch Fellowship for young scientists, Charité Berlin
2005 - 2007
Postdoctoral Fellowship, German Academic Exchange Foundation (DAAD)
2006
Young Scientist Award, Science Foundation Berlin, Germany
2006
Award for the best medical dissertation of 2005, Berlin Society for Psychiatry and Neurology
2005
Humboldt Prize (Prize for best dissertation of the year, Humboldt University Berlin)

10 Most Relevant Publications for Dr. Annett Halle

1. Schnaars M, Beckert H, **Halle A**. Assessing β-amyloid-induced NLRP3 inflammasome activation in primary microglia. Methods Mol Biol. 2013;1040:1-8.
2. Krabbe, G.*, **Halle, A.***, Matyash, V., Rinnenthal, J. L., Eom, G. D., Bernhardt, U., Miller, K. R., Prokop, S., Kettenmann, H. and Heppner, F. L., Functional impairment of microglia coincides with Beta-amyloid deposition in mice with Alzheimer-like pathology. PLoS One 2013. 8: e60921.
3. Heneka, M. T., Kummer, M. P., Stutz, A., Delekate, A., Schwartz, S., Vieira-Saecker, A., Griep, A., Axt, D., Remus, A., Tzeng, T. C., Gelpi, E., **Halle, A.**, Korte, M., Latz, E. and Golenbock, D. T., NLRP3 is activated in Alzheimer's disease and contributes to pathology in APP/PS1 mice. Nature 2013. 493: 674-678.
4. Stewart, C. R., Stuart, L. M., Wilkinson, K., van Gils, J. M., Deng, J., **Halle, A.**, Rayner, K. J., Boyer, L., Zhong, R., Frazier, W. A., Lacy-Hulbert, A., El Khoury, J., Golenbock, D. T. and Moore, K. J., CD36 ligands promote sterile inflammation through assembly of a Toll-like receptor 4 and 6 heterodimer. Nat Immunol 2010. 11: 155-161.
5. Siednienko, J., **Halle, A.**, Nagpal, K., Golenbock, D. T. and Miggin, S. M., TLR3-mediated IFN-beta gene induction is negatively regulated by the TLR adaptor MyD88 adaptor-like. Eur J Immunol 2010. 40: 3150-3160.
6. **Halle, A.,** Hornung, V., Petzold, G. C., Stewart, C. R., Monks, B. G., Reinheckel, T., Fitzgerald, K. A., Latz, E., Moore, K. J. and Golenbock, D. T., The NALP3 inflammasome is involved in the innate immune response to amyloid-beta. Nat Immunol 2008. 9: 857-865.
7. **Halle, A.***, Zhou, S*., Kurt-Jones, E. A., Cerny, A. M., Porpiglia, E., Rogers, M., Golenbock, D. T. and Finberg, R. W., Lymphocytic choriomeningitis virus (LCMV) infection of CNS glial cells results in TLR2-MyD88/Mal-dependent inflammatory responses. J Neuroimmunol 2008. 194: 70-82.
8. Hornung, V., Bauernfeind, F., **Halle, A.**, Samstad, E. O., Kono, H., Rock, K. L., Fitzgerald, K. A. and Latz, E., Silica crystals and aluminum salts activate the NALP3 inflammasome through phagosomal destabilization. Nat Immunol 2008. 9: 847-856.
9. Jain, V., **Halle, A.**, Halmen, K. A., Lien, E., Charrel-Dennis, M., Ram, S., Golenbock, D. T. and Visintin, A., Phagocytosis and intracellular killing of MD-2 opsonized gram-negative bacteria depend on TLR4 signaling. Blood 2008. 111: 4637-4645.
10. **Halle, A.***, Bermpohl, D.*, Freyer, D., Dagand, E., Braun, J. S., Bechmann, I., Schroder, N. W. and Weber, J. R., Bacterial programmed cell death of cerebral endothelial cells involves dual death pathways. J Clin Invest 2005. 115: 1607-1615.

*These authors contributed equally

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Institute of Clinical Chemistry and Clinical Pharmacology

Rheinische Friedrich-Wilhelms-Universität Bonn
Institute of Clinical Chemistry and Clinical Pharmacology, Director

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Research Expertise

The focus of research is the immunorecognition of nucleic acids, and its intersection with RNA interference. The group contributed to the immunobiology of TLR9 and CpG DNA, specifically the function of TLR9 in the human immune system. Furthermore, the group found that short interfering RNA molecules (siRNA) activate TLR7, and worked on the structural requirements for the detection of RNA by TLR7 and TLR8. The group identified the RNA ligand for RIG-I, and analyzed the signaling pathways of RIG-I, and resolved the crytal structure or RIG-I bound to its ligand 5′-triphosphate RNA. The group identified cyclic [G(2‘,5‘)pA(3“,5”)p] as the metazoan second messenger in the cGAS-STING pathway. The group applies immunostimulatory nucleic acids and siRNA for immunotherapy of cancer and viral infection.

Education / Training

University of Munich, Germany, Experimental Pharmacology and Toxicology Degree, 2006
University of Munich, Germany, Clinical Pharmacology, Degree, 2003
University of Munich, Germany, Clinical Pharmacology, Habilitation, 2001
University of Ulm, Germany, Clinical Genetics, MD thesis, 1994
University of Ulm, Germany, Clinical Medicine, MD, 1993

Appointments / Positions Held

2014 Founder Rigontec GmbH, Rigontec GmbH, Bonn, Germany
2013 - present Vice Speaker of DZIF Bonn-Cologne, University of Bonn, Germany
2012 - present Speaker of the DFG-ImmunoSensation Cluster of Excellence, University of Bonn, Germany
2008 - present Head of Research Committee BONFOR, University of Bonn, Germany
2007 - present Full Professor and Chair, Institute of Clinical Chemistry and Clinical Pharmacology with the Central Laboratory of the University Hospital, University of Bonn, Germany
2006 - present Member of the Steering Committee, Comprehensive Cancer Center Köln-Bonn (CIO), University of Bonn,
2005 Full Professor and Head, Division of Clinical Pharmacology, University of Bonn, Germany
2002 Assistant Professor, Division of Clinical Pharmacology, University of Munich, Germany
1999 - 2005 Research group: Therapeutic Oligonucleotides, University of Munich, Germany
1998 - 1999 Postdoctoral Fellow, Department of Internal Medicine, University of Iowa, USA
1995 Research Fellow, Division of Clinical Pharmacology, University of Munich, Germany
1994 Research Fellow, Department of Internal Medicine, University of Munich, Germany

Honors / Awards

2014 - present Elected Member of the German Academy of Sciences Leopoldina
2012 Gottfried-Wilhelm Leibniz-Preis
2011 - 2012 Elected President of the Oligonucleotide Therapeutics Society (OTS)
2011 Dr.-Friedrich-Sasse-Preis, Berliner Medizinische Gesellschaft/ GoBio-Award of the Federal Ministry for Education and Research (BMBF)
2010 Elected Vice Speaker of the SFB 670
2009 Elected member of the committee Krebstherapie-Studien of the German Cancer Aid (Deutsche Krebshilfe)
2007 Wilhelm-Vaillant-Award for Medical Sciences
2004 Ludwig-Heilmeyer-Award (Ludwig-Heilmeyer Society, Internal Medicine, Germany) / Biofuture Award, of the Federal Ministry for Education and Research (BMBF) / Georg-Heberer Award, Chiles Foundation, Portland
2000 Paul-Martini-Award / Award “Young Master” of the German Society for Hematology and Oncology

10 Most Relevant Publications for Prof. Gunther Hartmann

1. Herzner-AM, Hagmann CA, Goldeck M, Keßels S, Kübler K, Wittmann S, Gramberg T, Andreeva L, Hopfner KP, Mertens C, Zillinger T, Jin T, Xiao TS, Bartok E, Coch C, Ackermann D, Hornung V, Ludwig J, Barchet W, **Hartmann G*** and Schlee M*. Sequence-specific activation of cGAS by Y-form DNA structures as found in primary HIV-1 cDNA. Nat Immunol 2015 Oct; 16(10): 1025-33.
2. Schuberth-Wagner C, Ludwig J, Bruder AK, Herzner AM, Zillinger T, Goldeck M, Schmidt T, Schmid-Burgk JL, Kerber R, Wolter S, Stümpel JP, Roth A, Bartok E, Drosten C, Coch C, Hornung V, Barchet W, Kümmerer BM, **Hartmann G*** and Schlee M*. A conserved histidine in RIG-I controls immune tolerance to N1-2’O-methylated self RNA. Immunity, 2015 Jul 21;43(1):41-51.
3. Goubau D, Schlee M, Deddouche S, Pruijssers AJ, Zillinger T, Goldeck M, Schuberth C, Van der Veen AG, Fujimura T, Rehwinkel J, Iskarpatyoti JA, Barchet W, Ludwig J, Dermody TS, **Hartmann G**, Reis e Sousa C. Antiviral immunity via RIG-I-mediated recognition of RNA bearing 5’-diphosphates. Nature. 2014 Oct 16;514(7522):372-5
4. Gehrke N, Mertens C, Zillinger T, Wenzel J, Bald T, Zahn S, Tüting T, **Hartmann G**, Barchet W. Oxidative damage of DNA confers resistance to cytosolic nuclease TREX1 degradation and potentiates STING-dependent immune sensing. Immunity 2013 Sep 19;39(3):482-95.
5. Gao P, Ascano M, Wu Y, Barchet W, Gaffney BL, Zillinger T, Serganov AA, Liu Y, Jones RA, **Hartmann G**, Tuschl T, Patel DJ. Cyclic [G(2‘,5‘)pA(3“,5”)p] Is the Metazoan Second Messenger Produced by DNA-Activated Cyclic GMP-AMP Synthase. Cell 2013 May 23;153:1094-107.
6. Wang Y, Ludwig J, Schuberth C, Goldeck M, Schlee M, Li H, Juranek S, Sheng G, Micura R, Tuschl T*, **Hartmann G***, Patel DJ*. 2010. Structural and functional insights into 5‘-ppp RNA pattern recognition by the innate immune receptor RIG-I. Nat Struct Mol Biol 17:781-7.
7. Schlee M, Roth A, Hornung V, Hagmann CA, Wimmenauer V, Barchet W, Coch C, Janke M, Mihailovic A, Wardle G, Juranek S, Kato H, Kawai T, Poeck H, Fitzgerald KA, Takeuchi O, Akira S, Tuschl T, Latz E, Ludwig J, **Hartmann G**. 2009. Recognition of 5‘ triphosphate by RIG-I helicase requires short blunt double-stranded RNA as contained in panhandle of negative-strand virus. Immunity 31: 25-34.
8. Poeck H, Besch R, Maihoefer C, Renn M, Tormo D, Morskaya SS, Kirschnek S, Gaffal E, Landsberg J, Hellmuth J, Schmidt A, Anz D, Bscheider M, Schwerd T, Berking C, Bourquin C, Kalinke U, Kremmer E, Kato H, Akira S, Meyers R, Hacker G, Neuenhahn M, Busch D, Ruland J, Rothenfusser S, Prinz M, Hornung V, Endres S, Tuting T, **Hartmann G**. 2008. 5‘-Triphosphate-siRNA: turning gene silencing and Rig-I activation against melanoma. Nat Med 14: 1256-63.
9. Hornung V, Ellegast J, Kim S, Brzozka K, Jung A, Kato H, Poeck H, Akira S, Conzelmann KK, Schlee M, Endres S, **Hartmann G**. 2006. 5‘-Triphosphate RNA is the ligand for RIG-I. Science 314: 994-7.
10. Hornung V, Guenthner-Biller M, Bourquin C, Ablasser A, Schlee M, Uematsu S, Noronha A, Manoharan M, Akira S, de Fougerolles A, Endres S, **Hartmann G**. 2005. Sequence-specific potent induction of IFN-alpha by short interfering RNA in plasmacytoid dendritic cells through TLR7. Nat Med 11: 263-70.

* These authors contributed equallly

Prof. Michael Heneka, MD

Clinical Neurosciences Unit

Rheinische Friedrich-Wilhelms-Universität Bonn
Clinical Neurosciences Unit, Director

E-Mail: michael.heneka@ukb.uni-bonn.de

Research Expertise

Prof. Heneka is involved in basic science and translational research with focus on neurodegeneration and neuroinflammation. His major disease of interest and research topics include Alzheimer disease, amyotrophic lateral sclerosis, septic encephalopathy and multiple sclerosis. In clinical neurology, Prof. Heneka holds special expertise in neurodegenerative and autoimmune CNS disorders.

Education / Training

University of Bonn, Germany, Neurology, Professorial qualification (Habilitation), 2003
University of Bonn, Germany, Neurology, Specialty qualification, 2002
University of Tübingen, Germany, Medicine, MD, 1996

Appointments / Positions Held

2008 - present
Full Professor (W3) for Clinical Neurosciences, Head of the Clinical Research Group 177 of the DFG, University of Bonn
2004 - 2008
Full Professor (C3) for Molecular Neurology, University of Münster
2004
Senior Clinical Fellow in Neurology, University of Bonn
1999 - 2003
Resident in Neurology, University of Bonn
1996 - 1999
Resident in Neurology, University of Tübingen
1992 - 1996
Predoctoral research fellow in the Dept. of Pharmacology, University of Cologne

Honors / Awards

2013 - present
Associate Editor Neurology, Neuroimmunology and Neuroinflammation
2013
Hans und Ilse Breuer Award for Alzheimer Research
2012 - present
Editorial Board Molecular Neurobiology
2011
Christa Lorenz Award for Amyotrophic Lateral Sclerosis Research
2010 - present
Editorial Board Journal of Neurochemistry
2007 - present
Board Member of the Competence Network Degenerative Dementias (CNDD)
2008
Editorial board, Journal of Chemical Neuroanatomy
1998
Attempto Award - best Thesis of the University of Bonn

10 Most Relevant Publications for Prof. Michael Heneka

1. **Heneka MT,** Klockgether T, Feinstein DL. Peroxisome proliferator-activated receptor-gamma ligands reduce neuronal inducible nitric oxide synthase expression and cell death in vivo. J Neurosci 2000;20:6862-6867.
2. **Heneka MT**, Galea E, Gavriluyk V, Dumitrescu-Ozimek L, Daeschner J, O´Banion MK, Klockgether T, Feinstein DL. Noradrenergic depletion potentiates beta-amyloid induced cortical inflammation: Implications for Alzheimer’s disease. J Neurosci 2002;22:2434-2442.
3. **Heneka MT**, Dewachter I, Sastre M, Dumitrescu-Ozimek L, Cuiperi K, a gonist pioglitazone and ibuprofen reduces inflammation and Aβ 1-42 levels in APP V717I transgenic mice. Brain 2005;128:1442-1453.
4. Schütz B, Reimann J, Dumitrescu-Ozimek L, Kappes-Horn K, Landreth GE, Schürmann B, Zimmer A, **Heneka MT**. The oral antidiabetic pioglitazone protects from neurodegeneration and ALS-like symptoms in SOD1-G93A transgenic mice. J Neurosci 2005;25:7805-7812.
5. Sastre M, Dewachter I, Rossner S, Bogdanovic N, Rosen E, Borghraef P, Evert BO, Dumitrescu-Ozimek D, Thal DR, Landreth GE, Walter J, Klockgether T, Van Leuven F, **Heneka MT** (2006) NSAIDs suppress BACE1 gene expression by the activation of PPARγ. Proc Natl Acad Sci USA 2006;103:443-448.
6. **Heneka MT**, Ramanathan M, Jacobs AH, Dumitrescu-Ozimek L, Debeir T, Sastre M, Bilkei-Gorzo A, Zimmer A, Galldiks N, Hoehn M, Heiss WD, Klockgether T, Staufenbiel M. Locus ceruleus degeneration promotes Alzheimer pathogenesis in APP transgenic mice. J. Neurosci 2006;26:1343-1354.
7. Weberpals M, Hermes M, Hermann M, Kummer MP, Terwel D, Semmler A, Berger M, Schäfers M, **Heneka MT** (2009) NOS2 gene deficiency protects from sepsis-induced long-term cognitive deficits, J Neurosci, 29:14177-84.
8. **Heneka MT**, Nadrigny F, Regen T, Dumitrescu-Ozimek L, Terwel D, Jardanhazi-Kurutz D, Walter J, Kirchhoff F, Hanisch U, Kummer MP (2010) Locus ceruleus controls Alzheimer disease pathology by modulating microglial functions through norepinephrine. Proc. Natl. Acad. Sci. U.S.A., 107:6058-63.
9. Kummer MP, Hermes M, Delekarte A, Hammerschmidt T, Kumar S, Terwel D, Walter J, Pape HC, König, S, Roeber S, Jessen F, Klockgether T, Korte M, **Heneka MT** (2011) Nitration of tyrosine 10 critically enhances amyloid β aggregation and plaque formation. Neuron 71:833-44.
10. **Heneka MT**, Kummer MP, Stutz A, Delekate A, Schwartz S, Vieira-Saecker A, Griep A, Axt D, Remus A, Tzeng TC, Gelpi E, Halle A, Korte M, Latz E, Golenbock DT (2013) NLRP3 is activated in Alzheimer‘s disease and contributes to pathology in APP/PS1 mice. Nature. 493: 674-678.

Prof. Michael Hoch, PhD

Life and Medical Sciences Institute (LIMES)

Rheinische Friedrich-Wilhelms-Universität Bonn
Life and Medical Sciences Institute (LIMES), Program Unit Genetics, Developmental Biology & Molecular Physiology
E-Mail: m.hoch@uni-bonn.de

Research Expertise

Our aim is to identify new key regulators and genetic networks which control metabolism and cell and organ physiology. In particular, we elucidate the metabolism – innate immunity – gut microbiome axis, we investigate cellular (sphingo)lipid metabolism and body fat regulation, we study peroxisome and lysosome biogenesis and metabolic disorders (e.g. lipid storage diseases or neurodegeneration), and we analyse new regulators of cell-to-cell communication and tissue physiology. We use the fruit fly Drosophila, the mouse and zebra fish as genetic model organisms for our studies.

Education / Training

University of Munich, Germany, Developmental Biology PhD, 1992
University of Heidelberg, Germany, Biology Undergraduate (Dipl.), 1989

Appointments / Positions Held

2010
Visiting Research Professors, ASMeW Institute, Waseda University, Japan
2006 - present
Managing Director of the LIMES Institute, Chair Molecular Developmental Biology, LIMES Institute, University of Bonn, Germany
2000 - 2002
Director, Institute of Animal Physiology, University of Bonn, Germany
1999 - present
Full Professor, Chair of Molecular Developmental Biology LIMES Institute, University of Bonn, Germany
1996
Habilitation in Developmental Genetics & Cell Biology Technical University of Braunschweig, Germany
1994 - 1999
Group Leader, Dept. Mol. Developmental Biology (Head: Prof. H. Jäckle), Max Planck Institute for Biophysical Chemistry, Göttingen, Germany
1992 - 1994
Post-doc Fellow, Dept. Mol. Developmental Biology (Head: Prof. H. Jäckle), Max Planck Institute for Biophysical Chemistry, Göttingen, Germany

Honors / Awards

Since 2015 Rector of the University of Bonn
2006 - 2015 Managing Director of the LIMES (Life & Medical Sciences) Institute
2014 - 2015
Member of the Academic Senate of the University of Bonn
2013 - 2015
Member of the PhD fellowship selection committee of the German National Academic Foundation (Studienstiftung des deutschen Volkes)
2012 - 2015
Member of the Steering Committee of the ImmunoSensation Cluster of Excellence Bonn (German Research Foundation DFG)
2009 - 2015
Member of the Minerva Fellowship Committee of the Max Planck Society, Munich
2006 - 2009
Founding Head of the Section Molecular Biomedicine of the Faculty for Mathematics & Natural Science, University of Bonn
2005 - present
Speaker of the Collaborative Research Centre SFB 645 (German Research Foundation)
2003 - 2004
Head of the Section Biology of the Faculty for Mathematics & Natural Science, University of Bonn
2002 - 2004
Chairman of the Bonner Forum Biomedizin
2001 - 2004
Speaker of the Research Unit FOR 425, funded by the DFG
2000 - 2007
Member of the reviewer panel for the award of Post Graduate Fellowships of the DAAD (German Academic Exchange Service)
1996
Gerhard Hess Young Investigator Award (DFG)
1989 - 1992
PhD Fellowship of the Boehringer Ingelheim Fonds (Foundation for Basic Research in Medicine)
1986 - 1989
Member of the German National Academic Foundation (Studienstiftung des deutschen Volkes)

5 Most Relevant Publications for Prof. Michael Hoch

1. Mass E, Wachten D, Aschenbrenner AC, Voelzmann A, **Hoch M**. 2014. Murine Creld1 controls cardiac development through activation of calcineurin/NFATc1 signaling. Developmental Cell 28, 711-726. DOI: 10.1016/j.devcel.2014.02.012.
2. Becker T, Loch G, Beyer M, Zinke I, Aschenbrenner AC, Carrera P, Inhester T, Schultze JL, **Hoch M**. 2010. FOXO-dependent regulation of innate immune homeostasis. Nature 463: 369-73.
3. Bauer R, Voelzmann A, Breiden B, Schepers U, Farwanah H, Hahn I, Eckardt F, Sandhoff K, **Hoch M**. 2009. Schlank, a member of the ceramide synthase family controls growth and body fat in Drosophila. EMBO J 28: 3706-3716.
4. Behr M, Wingen C, Wolf C, Schuh R, **Hoch M**. 2007. Wurst is essential for airway clearance and respiratory-tube size control. Nat Cell Biol 9: 847-53.
5. Fuss B, Becker T, Zinke I, **Hoch M**. 2006. The cytohesin Steppke is essential for insulin signalling in Drosophila. Nature 444: 945-8.

Prof. Achim Hörauf, MD

Institute of Medical Microbiology, Immunology and Parasitology

Rheinische Friedrich-Wilhelms-Universität Bonn
Institute of Medical Microbiology, Immunology and Parasitology, Director
E-Mail: hoerauf@uni-bonn.de

Research Expertise

Prof. Hörauf is internationally renowned for his work in Tropical Medicine, in particular for pioneering new drug treatments against filariasis (a group of neglected tropical diseases). The new treatment exploits an endosymbiosis between worms and bacterial endosymbionts called Wolbachia, which are susceptible to some classes of antibiotics. Prof. Hörauf's group was the first to characterize a TLR2 ligand from Wolbachia and established that blindness in onchocerciasis (or 'river blindness') is dependent on innate immune reactions against Wolbachia. They were the first to detect regulatory T cells in humans in an infection. Modulation of host's immune responses is another focus of Prof. Hörauf's group, demonstrating that filarial infection and filarial antigen treatment protects against type 1 diabetes onset and improves glucose tolerance during obesity. Using expression quantitative trait loci analysis, the role of genetic factors in modulating cellular responses (to filarial antigens) and eventually filariae-caused pathology development is currently investigated.

Education / Training

University of Erlangen, Germany, Clinical Immunology, MD, 1989
University of Erlangen, Germany, Duke University, USA and Galaway University, Ireland
Clinical Immunology MD, with clinical rotations, 1989

Appointments / Positions Held

2003 - present
Full Professor and Director, Institute for Medical Microbiology, Immunology and Parasitology, University Hospital Bonn, Germany
2001 - 2003
Head, Department of Helminthology, Bernard Nocht Institute for Tropical Medicine, Germany
1995 - 2001
Independent Laboratory Head, Bernard Nocht Institute for Tropical Medicine, Germany
1990 - 1994
Fellow, Institute for Clinical Microbiology, Immunology and Hygiene, University of Erlangen, Germany

Honors / Awards

2015
The Innovation Award 2015 by BioRegionen
2015
Memento Prize for Neglected Diseases
2012
President of the Paul Ehrlich Society for Chemotherapy e.V.
2010
Coordinator for the partner BonnCologne, Dt. Zentrum für Infektionsforschung (DZIF) – German Center for Infectious Disease.
2002
Main annual award, German Society for Hygiene and Microbiology (DGHM)
2001
Martini-Prize (bi-annual) for best clinical research of the University Clinic, Eppendorf/Hamburg
1999
Main bi-annual award, German Society for Tropical Medicine (DTG)

10 Most Relevant Publications for Prof. Achim Hörauf

1. Gondorf F, Berbudi A, Buerfent BC, Ajendra J, Bloemker D, Specht S, Schmidt D, Neumann AL, Layland LE, **Hoerauf A**, Hübner MP. 2015 Chronic filarial infection provides protection against bacterial sepsis by functionally reprogramming macrophages. PLoS Pathog. 2015 Jan 22;11(1)
2. Mand S, Debrah AY, Klarmann U, Batsa L, Marfo-Debrekyei Y, Kwarteng A, Specht S, Belda-Domene A, Fimmers R, Taylor M, Adjei O, **Hoerauf A**. 2012. Doxycycline improves filarial lymphedema independent of active filarial infection: a randomized controlled trial. Clin Infect Dis. 55:621-30.
3. Specht S, Frank JK, Alferink J, Dubben B, Layland LE, Denece G, Bain O, Förster I, Kirschning CJ, Martin C, **Hoerauf A.** 2011. CCL17 controls mast cells for the defense against filarial larval entry. J Immunol 186:4845-52.
4. Taylor M, **Hoerauf A**, Bockarie M. 2010. Lymphatic filariasis and onchocerciasis. Lancet 376: 1175-85.
5. **Hoerauf A**. 2009. Mansonella perstans-the importance of an endosymbiont. N Engl J Med 361: 1502-4.
6. Specht S, **Hoerauf A**. 2007. Does helminth elimination promote or prevent malaria? Lancet 369: 446-7.
7. Taylor MJ, Makunde WH, McGarry HF, Turner JD, Mand S, **Hoerauf A**. 2005. Macrofilaricidal activity after doxycycline treatment of Wuchereria bancrofti: a double- blind, randomised placebo-controlled trial. Lancet 365: 2116-21.
8. Saint Andre A, Blackwell NM, Hall LR, **Hoerauf A**, Brattig NW, Volkmann L, Taylor MJ, Ford L, Hise AG, Lass JH, Diaconu E, Pearlman E. 2002. The role of endosymbiotic Wolbachia bacteria in the pathogenesis of river blindness. Science 295: 1892-5.
9. **Hoerauf A**, Mand S, Adjei O, Fleischer B, Buttner DW. 2001. Depletion of wolbachia endobacteria in Onchocerca volvulus by doxycycline and microfilaridermia after ivermectin treatment. Lancet 357: 1415-6.
10. **Hoerauf A**, Volkmann L, Hamelmann C, Adjei O, Autenrieth IB, Fleischer B, Buttner DW. 2000. Endosymbiotic bacteria in worms as targets for a novel chemotherapy in filariasis. Lancet 355: 1242-3.

Prof. Veit Hornung, MD

Gene Center and Department of Biochemistry

Gene Center and Department of Biochemistry Ludwig-Maximilians-University Munich
Rheinische Friedrich-Wilhelms-Universität Bonn
Institute of Molecular Medicine, Director
E-Mail: hornung@genzentrum.lmu.de

Research Expertise

Prof. Hornung has expertise in pattern recognition, innate immunology, macrophages, dendritic cells, RNA biology and genome engineering technologies.

Education / Training

University of Munich, Germany, Clinical Pharmacology MD thesis, 2004
University of Munich, LMU, including exchange rotations at Harvard University, USA, and University of Zürich, Switzerland, Clinical Medicine, MD, 2003

Appointments / Positions Held

since 2015
Chair of Immunobiochemistry, Gene Center and Department of Biochemistry, Ludwig-Maximilians-University Munich
2014 - 2015
Director (W3) Institute of Molecular Medicine, University Hospital, University of Bonn
2008 - 2013
Professor of Clinical Biochemistry, Institute for Clinical Chemistry and Clinical Pharmacology, University of Bonn, Germany
2006 - 2008
Postdoctoral research fellow, Division of Infectious Diseases and Immunology, University of Massachusetts, USA
2005 - 2006
Group leader, Division of Clinical Pharmacology University of Munich, Germany
2003 - 2005
Research Fellow, Division of Clinical Pharmacology, University of Munich, Germany

Honors / Awards

2015
Elected EMBO Member
2015
ERC Consolidator Grant
2013
Pettenkofer Prize of the Max von Pettenkofer Foundation
2010
Award for Basic Medical Research of the GlaxoSmithKline Foundation
2010
Paul-Martini-Prize of the Paul-Martini-Foundation
2009
ERC Starting Grant
2007
Heinz Maier Leibnitz Prize of the German Research Foundation
2006
Graduate-Scholarship of the Novartis-Foundation for Therapeutical Research
2002
Study Scholarship of the Munich-Harvard-Alliance
2000 - 2003
Fellow of the German National Academic Foundation

10 Most Relevant Publications for Prof. Veit Hornung

1. Ablasser, A., J. L. Schmid-Burgk, I. Hemmerling, G. L. Horvath, T. Schmidt, E. Latz and **V. Hornung.** Cell intrinsic immunity spreads to bystander cells via the intercellular transfer of cGAMP Nature, 2013; 503:530-534.
2. Ablasser, A., M. Goldeck, T. Cavlar, T. Deimling, G. Witte, I. Rohl, K. P. Hopfner, J. Ludwig and **V. Hornung.** cGAS produces a 2'-5'-linked cyclic dinucleotide second messenger that activates STING Nature, 2013; 498: 380-384.
3. Bartok, E., F. Bauernfeind, M. G. Khaminets, C. Jakobs, B. Monks, K. A. Fitzgerald, E. Latz and **V. Hornung.** iGLuc: a luciferase-based inflammasome and protease activity reporter Nat Methods, 2013; 10: 147-154.
4. Schmid-Burgk, J. L., T. Schmidt, V. Kaiser, K. Honing and **V. Hornung.** A ligation-independent cloning technique for high-throughput assembly of transcription activator-like effector genes Nat Biotechnol, 2013; 31: 76-81.
5. Duewell, P., H. Kono, K. J. Rayner, C. M. Sirois, G. Vladimer, F. G. Bauernfeind, G. S. Abela, L. Franchi, G. Nunez, M. Schnurr, T. Espevik, E. Lien, K. A. Fitzgerald, K. L. Rock, K. J. Moore, S. D. Wright, **V. Hornung*** and E. Latz*. NLRP3 inflammasomes are required for atherogenesis and activated by cholesterol crystals Nature, 2010; 464: 1357-1361.
6. Ablasser, A., F. Bauernfeind, G. Hartmann, E. Latz, K. A. Fitzgerald and **V. Hornung.** RIG- I-dependent sensing of poly (dA:dT) through the induction of an RNA polymerase III-transcribed RNA intermediate Nat Immunol, 2009; 10: 1065-1072.
7. **Hornung, V.**, A. Ablasser, M. Charrel-Dennis, F. Bauernfeind, G. Horvath, D. R. Caffrey, E. Latz and K. A. Fitzgerald. AIM2 recognizes cytosolic dsDNA and forms a caspase-1-activating inflammasome with ASC Nature, 2009; 458: 514-518.
8. **Hornung, V.***, F. Bauernfeind*, A. Halle, E. O. Samstad, H. Kono, K. L. Rock, K. A. Fitz- gerald and E. Latz. Silica crystals and aluminum salts activate the NALP3 inflammasome through phagosomal destabilization Nat Immunol, 2008; 9: 847-856.
9. **Hornung, V.**, J. Ellegast, S. Kim, K. Brzozka, A. Jung, H. Kato, H. Poeck, S. Akira, K. K. Conzelmann, M. Schlee, S. Endres and G. Hartmann. 5'-Triphosphate RNA is the ligand for RIG-I Science, 2006; 314: 994-997.
10. **Hornung, V.**, M. Guenthner-Biller, C. Bourquin, A. Ablasser, M. Schlee, S. Uematsu, A. Noronha, M. Manoharan, S. Akira, A. de Fougerolles, S. Endres and G. Hartmann. Sequence-specific potent induction of IFN-alpha by short interfering RNA in plasmacytoid dendritic cells through TLR7 Nat Med, 2005; 11: 263-270.

*These authors contributed equally

Prof. Jörg C. Kalff, MD

Department of Surgery

Rheinische Friedrich-Wilhelms-Universität Bonn
Department of Surgery, Director
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Research Expertise

The focus of research are the immunological consequences of operative trauma and their recognition and regulation in postoperative dysfunction of the gastrointestinal tract. The group described and elucidated the immunological pathomechanism of postoperative ileus. Furthermore, the group found that the gastrointestinal field effect - a panenteric inflammation following localized abdominal surgery - is mediated by an immunological response involving resident intestinal macrophages, mesenteric dendritic cells and memory TH1 cells.

Education / Training

University of Bonn, Germany, Surgery, Habilitation, 1999
University of Aachen, Germany, Intensive Care , MD thesis, 1988
University of Aachen, Germany, Clinical Medicine, MD, 1987

Appointments / Positions Held

2010 - present
Full Professor and Head, Dept. of Surgery, University of Bonn, Germany
2009
Head, Division of Transplant Surgery, University of Bonn, Germany
2003
Professor of Surgery, University of Bonn, Germany
1999 - 2001
Visiting Research Professor, Dept. of Medicine, University of Pittsburgh, USA
1995 - 1998
Research Fellow, Department of Surgery, University of Pittsburgh, USA
1995
Clinical Fellow, Department of Surgery, University of Bonn, Germany
1989
Resident, Department of Surgery, University of Bonn, Germany

Honors / Awards

2006
Fellow of the American College of Surgeons (FACS)
2003
Elected Speaker of the KFO 115
2000
Ferdinand Sauerbruch Award, Berlin, Germany
2000
Young Investigator Award, American Motility Society
2000
BONFOR Young Investigator Research Award

10 Most Relevant Publications for Prof. Jörg C. Kalff

1. Pantelis D, Beissel A, Kahl P, Vilz TO, Stoffels B, Wehner S, **Kalff JC.** 2011. Colonic anastomotic healing in the context of altered macrophage function and endotoxemia. Int J Colorect Dis 26:737-46.
2. Engel DR, Koscielny A, Wehner S, Maurer J, Schumak B, Limmer A, Sparwasser T, Hirner A, Knolle P, **Kalff JC***, Kurts C* (*joined corresponding authorship). 2010. T helper type 1 memory cells disseminate postoperative ileus over the entire intestinal tract. Nat Med. 16:1407-13.
3. Wehner S, Buchholz BM, Schuichtrup S, Rocke A, Schaefer N, Lysson M, Hirner A, **Kalff JC.** 2010. Mechanical strain and TLR4 synergistically induce cell-specific inflammatory gene expression in intestinal smooth muscle cells and peritoneal macrophages. Am J Physiol Gastrointest Liver Physiol 299:G1187-97.
4. Pantelis D, Kabba MS, Kirfel J, Kahl P, Wehner S, Buettner R, Hirner A, **Kalff JC.** 2010. Transient perioperative pharmacologic inhibition of muscularis macrophages as a target for prophylaxis of postoperative ileus does not affect anastomotic healing in mice. Surgery 148:59-70.
5. Wehner S, Straesser S, Vilz TO, Pantelis D, Sielecki T, de la Cruz VF, Hirner A, **Kalff JC.** 2009. Inhibition of p38 mitogen-activated protein kinase pathway as prophylaxis of postoperative ileus in mice. Gastroenterology 136:619-29.
6. Wehner S, Behrendt FF, Lyutenski BN, Lysson M, Bauer AJ, Hirner A, **Kalff JC.** 2007. Inhibition of macrophage function prevents intestinal inflammation and postoperative ileus in rodents. Gut 56:176-185.
7. Wehner S, Schwarz NT, Hundsdörfer R, Hierholzer C, Tweardy DJ, Billiar TR, Bauer AJ, **Kalff JC.** 2005. Induction of IL-6 within the rodent intestinal muscularis following intestinal surgical stress. Surgery 137:436-46.
8. Schwarz NT, **Kalff JC**, Turler A, Speidel N, Grandis JR, Billiar TR, Bauer AJ. 2004. Selective jejunal manipulation causes postoperative pan-enteric inflammation and dysmotility. Gastroenterology 126:159-69.
9. **Kalff JC**, Türler A, Schwarz NT, Schraut WH, Lee KKW, Tweardy DJ, Billiar TR, Simmons RL, Bauer AJ. 2003. Intra-abdominal activation of a local inflammatory response within the human muscularis externa during laparotomy. Ann Surg 237:301-15.
10. Schwarz NT, Engel BM, Eskandari M, **Kalff JC**, Grandis JR, Bauer AJ. 2002. Lipopolysaccharide preconditioning and cross-tolerance: the induction of protective mechanisms for rat intestinal ileus. Gastroenterology 123:586-98.

Prof. U. Benjamin Kaupp, PhD

center of advanced european studies and research (caesar)

center of advanced european studies and research (caesar), Molecular Sensory Systems, Scientific Director and Head of Department

E-Mail: u.b.kaupp@caesar.de

Research Expertise

Biophysics of sensory systems. Physiology of receptors and ion channels in cellular signaling. Cell motility and chemotaxis. Development of chemical tools for kinetic techniques in cell biology.

Education / Training

University of Osnabrück, Biophysics, Habilitation, 1983
Technical University of Berlin, Chemistry, PhD, 1979
University of Tübingen and Technical University of Berlin
Chemistry, Diploma, 1974

Appointments / Positions Held

2010 - 2011
Director of the Max-Planck-Institute for Neurological Research, University of Cologne, Germany
2008 - present
Professor of Molecular Neurobiology, University of Bonn, Germany
2008 - present
Scientific Director of caesar and Head of Department Molecular Sensory Systems, Center of Advanced European Studies and Research
2007 - present
Scientific Member of the Max-Planck-Society, Max-Planck-Society
2000 - present
Whitman Investigator, Marine Biological Laboratory (MBL), Woods Hole, USA
1988 - present
Professor of Biophysical Chemistry, University of Cologne, Germany
2006 - 2009
Director of the International Helmholtz Research School of Biophysics and Soft Matter, Research Centre Jülich
1988 - 2007
Director at the Institute of Neuroscience and Biophysics Research Centre Jülich
1987
Feodor-Lynen-Stipend at the Department of Medical Chemistry, University of Kyoto, Japan
1985 - 1988
Assistant Professor of Biophysics, University of Osnabrück
1982 - 1985
Hochschulassistent, University of Osnabrück
1981
Postdoctoral Fellow, SUNY Stony Brook, USA

Honors / Awards

2013 Member of the North Rhine-Westphalian Academy of Sciences
2005 Member of the “German Academy of Sciences Leopoldina”
1999 Novartis Lecture, University Regensburg
1999 Keynote lecturer on international conferences
1994 Alcon Research Award
1987 Feodor-Lynen-Fellowship
1978 Member of the Academic Senate Technical University Berlin

10 Most Relevant Publications for Prof. U. Benjamin Kaupp

1. Jansen V, Alvarez L, Balbach M, Strünker T, Hegemann P, **Kaupp UB**, Wachten, D. 2015. Controlling fertilization and cAMP signaling in sperm by optogenetics. eLife 4 10.7554/eLife.05161
2. Seifert R, Flick M, Bönigk W, Alvarez L, Trötschel C, Poetsch A, et al. 2015. The CatSper channel controls chemosensation in sea urchin sperm. EMBO J. 34:379-392.
3. Alvarez L, Dai L, Friedrich BM, Kashikar ND, Gregor I, Pascal R, **Kaupp UB**. 2012 The rate of change in Ca2+ concentration controls sperm chemotaxis J. Cell. Biol. 196, 653-663
4. Strünker T, Goodwin N, Brenker C, Kashikar ND, Weyand I, Seifert R, **Kaupp UB**. 2011. The CatSper channel mediates progesterone-induced Ca2+ influx in human sperm. Nature 471: 382-386
5. Schröder-Lang S, Schwärzel M, Seifert R, Strünker T, Kateriya S, Looser J, Watanabe M, **Kaupp UB**, Hegemann P, Nagel G. 2007. Fast manipulation of cellular cAMP level by light in vivo. Nat. Methods 4: 39-42
6. Strünker T, Weyand I, Bönigk W, Van Q, Loogen A, Brown JE, Kashikar ND, Hagen V, Krause E, **Kaupp UB**. 2006. A K+-selective cGMP-gated ion channel controls chemosensation of sperm. Nat Cell Biol 8: 1149-54
7. **Kaupp UB**, Solzin J, Hildebrand E, Brown JE, Helbig A, Hagen V, Beyermann M, Pampaloni F, Weyand I. 2003. The signal flow and motor response controling chemotaxis of sea urchin sperm. Nat Cell Biol 5: 109-17
8. Körschen HG, Beyermann M, Müller F, Heck M, Vantler M, Koch KW, Kellner R, Wolfrum U, Bode C, Hofmann KP, **Kaupp UB**. 1999. Interaction of glutamic-acid-rich proteins with the cGMP signalling pathway in rod photoreceptors. Nature 400: 761-6
9. Gauss R, Seifert R, **Kaupp UB**. 1998. Molecular identification of a hyperpolarization- activated channel in sea urchin sperm. Nature 393: 583-7
10. **Kaupp UB**, Niidome T, Tanabe T, Terada S, Bönigk W, Stühmer W, Cook NJ, Kangawa K, Matsuo H, Hirose T, et al. 1989. Primary structure and functional expression from complementary DNA of the rod photoreceptor cyclic GMP-gated channel. Nature 342: 762-6

Prof. Percy Knolle, MD

Institute of Molecular Immunology

**Technische Universität München (TU)
Institute for Molecular Immunology, Director
Rheinische Friedrich-Wilhelms-Universität Bonn
Institute of Molecular Medicine, Director (until 2012)**

E-Mail: percy.knolle@tum.de

Research Expertise

The focus of Prof. Knolle’s research group is on the molecular and cellular mechanisms governing local immune control in tissues. In his laboratory the relevance of local antigen presentation by organ-resident liver cells was demonstrated for induction of immune tolerance in naïve CD4 and CD8 T cells. The development of novel cell separation techniques allowed to study the mechanisms and functional relevance of different liver cell populations at a new level of resolution and to compare the immune function of these non-professional antigen presenting cells with myeloid professional antigen presenting cells such as dendritic cells or macrophages. His group has discovered novel stimulatory pathways that are initiated by unique immune sensory mechanisms in liver-resident antigen presenting cells that trigger local T cell immunity in the liver. The lab has developed an interest in local mechanisms determining regulation of CD4 T cell differentiation with particular reference to the impact of nuclear receptors that also impact on the metabolic state of T cells.

Education / Training

University of Mainz, Germany, Internal Medicine Specialist, 1997
German Cancer Research Centre, Heidelberg, Applied Immunology, MD thesis, 1990
Universities of Frankfurt, Paris, Birmingham (UK), Strasbourg, and Geneva, Medicine, MD, 1988

Appointments / Positions Held

2013 - present
Director Institute of Molecular Immunology, TU Munich
2013 - 2017
coopted Medical Faculty, University of Bonn, Germany
2006 - 2012
Vice-speaker of the SFB 704, University of Bonn, Germany
2002 - 2012
Director Institute of Molecular Medicine, University of Bonn, Germany
2002
Professor of Molecular Medicine and Immunology, University of Bonn, Germany
1997 - 2002
Independent Group Leader, Center of Molecular Biology Heidelberg (ZMBH), University of Heidelberg, Germany
1991 - 1997
Physician at the 1st Medical Department, University of Mainz, Germany
1990 - 1991
Postdoctoral Fellow, BASF Bioresearch, Corporation, Cambridge, USA

Honors / Awards

2001
Award by the Volkswagen Foundation (1.5 Million €)

10 Most Relevant Publications for Prof. Percy Knolle

1. Beyer M, Abdullah Z, Chemnitz JM, Maisel D, Sander J, Lehmann C, Thabet Y, Shinde PV, Schmidleithner L, Kohne M, Trebicka J, Schierwagen R, Hofmann A, Popov A, Lang KS, Oxenius A, Buch T, Kurts C, Heikenwalder M, Fatkenheuer G, Lang PA, Hartmann P, **Knolle PA***, Schultze JL*. Tumor-necrosis factor impairs CD4(+) T cell-mediated immunological control in chronic viral infection. Nat Immunol 2016;17:593-603.
2. Bottcher JP, Beyer M, Meissner F, Abdullah Z, Sander J, Hochst B, Eickhoff S, Rieckmann JC, Russo C, Bauer T, Flecken T, Giesen D, Engel D, Jung S, Busch DH, Protzer U, Thimme R, Mann M, Kurts C, Schultze JL, Kastenmuller W, **Knolle PA**. Functional classification of memory CD8(+) T cells by CX3CR1 expression. Nat Commun 2015;6:8306.
3. Knolle PA, Thimme R. Hepatic Immune Regulation and its Involvement in Viral Hepatitis Infection. Gastroenterology 2014, 146: 1193-207
4. Wolf MJ, Adili A, Piotrowitz K, Abdullah Z, Boege Y, Stemmer K, Ringelhan M, Simonavicius N, Egger M, Wohlleber D, Lorentzen A, Einer C, Schulz S, Clavel T, Protzer U, Thiele C, Zischka H, Moch H, Tschop M, Tumanov AV, Haller D, Unger K, Karin M, Kopf M, **Knolle PA***, Weber A*, Heikenwalder M*. Metabolic Activation of Intrahepatic CD8(+) T Cells and NKT Cells Causes Nonalcoholic Steatohepatitis and Liver Cancer via Cross-Talk with Hepatocytes. Cancer Cell 2014;26:549-64.
5. Böttcher JP, Schanz O, Garbers C, Zaremba A, Hegenbarth S, Kurts C, Beyer M, Schultze JL, Kastenmuller W, Rose-John S, **Knolle PA**. IL-6 trans-Signaling-Dependent Rapid Development of Cytotoxic CD8(+) T Cell Function. Cell Reports 2014;8:1318-27
6. Huang LR, Wohlleber D, Reisinger F, Jenne CN, Cheng RL, Abdullah Z, Schildberg FA, Odenthal M, Dienes HP, van Rooijen N, Schmitt E, Garbi N, Croft M, Kurts C, Kubes P, Protzer U, Heikenwalder M, **Knolle PA**. Intrahepatic myeloid-cell aggregates enable local proliferation of CD8(+) T cells and successful immunotherapy against chronic viral liver infection. Nat Immunol 2013;14:574-83.
7. Böttcher JP, Schanz O, Wohlleber D, Abdullah Z, Debey-Pascher S, Staratschek-Jox A, Hochst B, Hegenbarth S, Grell J, Limmer A, Atreya I, Neurath MF, Busch DH, Schmitt E, van Endert P, Kolanus W, Kurts C, Schultze JL, Diehl L, **Knolle PA**. Liver-Primed Memory T Cells Generated under Noninflammatory Conditions Provide Anti-infectious Immunity. Cell Reports 2013;3:779-95.
8. Abdullah Z, Schlee M, Roth S, Mraheil MA, Barchet W, Bottcher J, Hain T, Geiger S, Hayakawa Y, Fritz JH, Civril F, Hopfner KP, Kurts C, Ruland J, Hartmann G, Chakraborty T, **Knolle PA**. RIG-I detects infection with live Listeria by sensing secreted bacterial nucleic acids. EMBO J 2012;31:4153-64.
9. Wohlleber D, Kashkar H, Gartner K, Frings MK, Odenthal M, Hegenbarth S, Borner C, Arnold B, Hammerling G, Nieswandt B, van Rooijen N, Limmer A, Cederbrant K, Heikenwalder M, Pasparakis M, Protzer U, Dienes HP, Kurts C, Kronke M, **Knolle PA**. TNF-Induced Target Cell Killing by CTL Activated through Cross-Presentation. Cell Reports 2012;2:478-87.
10. Protzer U, Maini MK, **Knolle PA**. Living in the liver: hepatic infections. Nat Rev Immunol 2012;12:201-13.

Prof. Waldemar Kolanus, PhD

Life and Medical Sciences Institute (LIMES)

Rheinische Friedrich-Wilhelms-Universität Bonn
Life and Medical Sciences Institute (LIMES), Molecular Immunology & Cell Biology, Director
E-Mail: wkolanus@uni-bonn.de

Research Expertise

Prof. Kolanus and his group are interested in intracellular signal transduction events which control leukocyte adhesion, migration, and effector functions. The main emphasis of their current research activities lies in elucidating the role of integrin adhesion receptors and the cytoskeleton in the functional adaptation of leukocyte motility to specific microenvironments, some of which include force-dependent slow migration of immune cells on and across barriers, versus force-independent, fast migration in the interstitium.

Education / Training

University of Hannover, Molecular Biology, PhD, 1987
University of Hannover, Biology, Chemistry, State examination, 1984

Appointments / Positions Held

2002 - present
Full Professorship, Molecular Immunology,
University of Bonn
1999 - 2002
Associate Professor, Biochemistry,
University of Munich (LMU)
1999
Habilitation in Biochemistry, Faculty of Chemistry, University of Munich (LMU)
1994 - 1999
Independent Group Leader, Gene Center Munich, University of Munich (LMU)
1990 - 1993
Post-doc Fellow, Molecular Immunology, Harvard Medical School
1988 - 1990 Post-doc Fellow, Immunology, Hannover Medical School

Honors / Awards

2009
US Patent 20090105286, Low molecular inhibitors of cyohesin-family guanine nucleotide exchange factors
2007
US Patent 20070287153 - Methods for identification and validation of functional intracellular targets with intramers or in vivo selection
2004
US Patent 20040170990 - Intracellular nucleic acid inhibitors of small guanine nucleotide exchange factors
US Patent 20040029775 - Methods and compounds for influencing beta3-integrin- dependent intracellular processes
2003
US Patent 20030138410 - Targeted cytolysis of HIV-infected cells by chimeric CD4 receptor-bearing cells
2002
US Patent 20020176851 - Redirection of cellular immunity by protein-tyrosine kinase chimeras
1996
US Patent 6573362 - Cytohesin-PH peptides that affect the ability of integrins to adhere
1994
Munich Gene Center Junior Group Leader 5-year-Award, BMBF and University of Munich

10 Most Relevant Publications for Prof. Waldemar Kolanus

1. Bald T, Quast T, Landsberg J, Rogava M, Glodde N, Lopez-Ramos D, Kohlmeyer J, Riesenberg S, van den Boorn-Konijnenberg D, Hömig-Hölzel C, Reuten R, Schadow B, Weighardt H, Wenzel D, Helfrich I, Schadendorf D, Bloch W, Bianchi M.E, Lugassy C, Barnhill RL, Koch M, Fleischmann BK, Förster I, Kastenmüller W, **Kolanus W**, Hölzel M, Gaffal E, Tüting T. 2014.Ultraviolet-radiation-induced inflammation promotes angiotropism and metastasis in melanoma. Nature. 507,109-13.
2. Müller S, Quast T, Schröder A, Hucke S, Klotz L, Jantsch J, Gerzer R, Hemmersbach R, **Kolanus W**. Salt-dependent chemotaxis of macrophages. 2013 PLoS One. 16 :e73439.
3. Ulbricht A, Eppler FJ, Tapia VE, van der Ven PF, Hampe N, Hersch N, Vakeel P, Stadel D, Haas A, Saftig P, Behrends C, Fürst DO, Volkmer R, Hoffmann B, **Kolanus W**, Höhfeld J. Cellular mechanotransduction relies on tension-induced and chaperone-assisted autophagy., Curr Biol., 2013, 23, 430-435.
4. Quast T, Eppler F, Semmling V, Schild C, Homsi Y, Levy S, Lang T, Kurts C, **Kolanus W.** CD81 is essential for the formation of membrane protrusions and regulates Rac1-activation in adhesion-dependent immune cell migration., Blood, 2011, 118, 1818-1827.
5. Loer B, Bauer R, Bornheim R, Grell J, Kremmer E, **Kolanus W**, Hoch M. 2008. The NHLdomain protein Wech is crucial for the integrin-cytoskeleton link. Nat Cell Biol 10: 422-8.
6. Hafner M, Schmitz A, Grune I, Srivatsan SG, Paul B, **Kolanus W,** Quast T, Kremmer E, Bauer I, Famulok M. 2006. Inhibition of cytohesins by SecinH3 leads to hepatic insulin resistance. Nature 444: 941-4.
7. Shamri R, Grabovsky V, Gauguet JM, Feigelson S, Manevich E, **Kolanus W**, Robinson MK, Staunton DE, von Andrian UH, Alon R. 2005. Lymphocyte arrest requires instantaneous induction of an extended LFA-1 conformation mediated by endothelium- bound chemokines. Nat Immunol 6: 497-506.
8. Boehm T, Hofer S, Winklehner P, Kellersch B, Geiger C, Trockenbacher A, Neyer S, Fiegl H, Ebner S, Ivarsson L, Schneider R, Kremmer E, Heufler C, **Kolanus W**. 2003. Attenuation of cell adhesion in lymphocytes is regulated by CYTIP, a protein which mediates signal complex sequestration. EMBO J 22: 1014-24.
9. Geiger C, Nagel W, Boehm T, van Kooyk Y, Figdor CG, Kremmer E, Hogg N, Zeitlmann L, Dierks H, Weber KS, **Kolanus W**. 2000. Cytohesin-1 regulates beta-2 integrin-mediated adhesion through both ARF-GEF function and interaction with LFA-1. EMBO J 19: 2525-36.
10. **Kolanus W**, Nagel W, Schiller B, Zeitlmann L, Godar S, Stockinger H, Seed B. 1996. Alpha L beta 2 integrin/LFA-1 binding to ICAM-1 induced by cytohesin-1, a cytoplasmic regulatory molecule. Cell 86: 233-42.

Prof. Christian Kurts, MD

Institute of Experimental Immunology

Rheinische Friedrich-Wilhelms-Universität Bonn
Institute of Experimental Immunology, Director
E-Mail: ckurts@uni-bonn.de

Research Expertise

Prof. Kurts and his group are interested in the mechanisms governing antigen-presentation and the ensuing immune response in the defense against infections and in immune-mediated disease. Their main research projects focus on the mechanisms of antigen cross-presentation to cytotoxic CD8 T cells, peripheral immune tolerance of T and B lymphocytes against self antigens, and the role of dendritic cells in diseases, especially in kidney disease.

Education / Training

University of Göttingen, Germany
Medicine MD, 1991

Appointments / Positions Held

2015 Speaker GRK 2168 Bo&Merang (Bonn & Melbourne Research and Graduate training Group)
2013 Co-speaker SFB 704 “Molecular Mechanisms and Chemical Modulation of Local Immune Regulation“ University of Bonn, Germany
2012 Co-speaker SFB TR57 “Organ-Fibrosis”, representing the Bonn site, University of Bonn, Germany
2009 - present Director Institute of Experimental Immunology, University of Bonn, Germany
2003 - 2008 Full Professor of Molecular Immunology University of Bonn, Germany
2002 Visiting scientist, Stephen Schoenberger Group
La Jolla Institute for Allergy and Immunology, CA, USA
2000 - 2003 Research group leader, Dept. of Nephrology and Clinical Immunology, University of Aachen, Germany
1998 - 2000 Medical Officer and Research Fellow, Hannover Medical School, Germany
1997 - 1998 Postdoctoral Research Fellow, Dept of Microbiology, Monash Medical School, Melbourne, Australia
1995 - 1997 Postdoctoral Research Fellow, Thymus Biology Unit, The Walter and Eliza Hall Institute for Medical Research (WEHI), Melbourne, Australia
1991 - 1995 Medical Officer and Research Fellow, Hannover Medical School, Germany

Honors / Awards

2016
Elected member DFG Fachkollegium (Review Board) Inflammation/Nephrology
2014
Elected member German National Academy of Sciences Leopoldina
2012
Gottfried-Wilhelm-Leibniz-Prize of the DFG
2010
Hans-U.-Zollinger-Award of the German Society for Nephrology
2000
Heisenberg-Fellowship of the
Deutsche Forschungsgemeinschaft
1999
Sir Hans Krebs award for basic medical research
1992
Annual award of the German Society of Nephrology for best doctoral thesis
1986 - 1991
Fellowship Studienstiftung des deutschen Volkes (German National Academic Foundation)

10 Most Relevant Publications for Prof. Christian Kurts

1. Schiwon M, Weisheit C, Franken L, Gutweiler S, Dixit A, Meyer-Schwesinger C, Pohl J, Maurice NJ, Thiebes S, Lorenz K, Quast T, Fuhrmann M, Baumgarten G, Lohse MJ, Opdenakker G, Bernhagen J, Bucala R, Panzer U, Kolanus W, Gröne HJ, Garbi N, Kastenmüller W, Knolle PA, **Kurts C***, Engel DR*. 2014. Crosstalk between sentinel and helper macrophages permits neutrophil migration into infected uroepithelium. Cell, 156:456–68, (*joint senior authorship)
2. Kastenmüller W, Kastenmüller K, **Kurts C**, Seder RA. Dendritic Cell Targeted Vaccines - Hype or Hope? 2014. Nat Rev Immunol, 14(10):705-11
3. **Kurts C**, Panzer U, Anders HJ, Rees A. 2013. The immune system and kidney disease: basic concepts and clinical implications. Nat Rev Immunol, 13(10):738-53.
4. Hochheiser K, Heuser, C Krause TA, Teteris S, Ilias A, Weisheit C, Hoss F, Tittel AP, Panzer U, Knolle PA, Engel DR, Tharaux PL, **Kurts C**. 2013. Exclusive CX3CR1-dependence of kidney cortex dendritic cells identifies a therapeutic target in glomerulonephritis. J Clin Invest, 123(10):4242-54.
5. Gottschalk C, Damuzzo V, Gotot J, Kroczek R, Yagita H, Murphy KM, Knolle PA, Ludwig-Portugall I, **Kurts C**. 2013. Batf3-dependent renal lymph node DCs maintain immune-homeostasis against circulating antigens; J Am Soc Nephrol, 24:543-9.
6. Gotot J, Gottschalk C, Leopold S, Knolle PA, Yagita H, **Kurts C***, Ludwig-Portugall I*. 2012. Direct PD-1-mediated suppression of autoreactive B cells by regulatory T cells. PNAS 109(26):10468-7. (*joint senior authorship)
7. Tittel AP, Heuser C, Ohliger C, Yona S, Hämmerling GJ, Engel DR, Garbi N, **Kurts C**. 2012. Functionally relevant neutrophilia in CD11c-diphteria toxin receptor transgenic mice. Nat Methods 9(4):385-90.
8. Engel DR, Koscielny A, Wehner S, Maurer J, Schiwon M, Schumak B, Limmer A, Sparwasser T, Hirner A, Knolle PA, Kalff JC, **Kurts C**. 2010. Th1 memory cells disseminate postoperative ileus over the entire intestinal tract. Nat Med, 16(12): 1407–1413.
9. Semmling V, Lukacs-Kornek V, Thaiss CA, Quast T, Hochheiser K, Panzer U, Rossjohn J, Perlmutter P, Cao J, Godfrey DI, Savage PB, Knolle PA, Kolanus W, Forster I, **Kurts C**. 2010. Alternative cross-priming through CCL17-CCR4-mediated attraction of CTLs toward NKT cell-licensed DCs. Nat Immunol 11: 313-20.
10. **Kurts C**, Robinson BW, Knolle PA. 2010. Cross-priming in health and disease. Nat Rev Immunol 10: 403-14.

*These authors contributed equally

Prof. Eicke Latz, MD PhD

Institute of Innate Immunity

Rheinische Friedrich-Wilhelms-Universität Bonn
Institute of Innate Immunity, Director
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Research Expertise

The Latz Lab has a longstanding interest in deciphering the molecular mechanisms of innate immune receptor activation. In particular, the lab is interested in understanding how innate receptors interact with their ligands and how this molecular interaction leads to receptor activation. Recently, we have also focused on the molecular details of the mechanisms that lead to the activation of the NLRP3 and AIM2 inflammasome. The NLRP3 inflammasome can respond to a broad range of cellular stressors and to substances that indicate metabolic derangements such as aggregated peptides, crystals of monosodium urate (forming in gout) or crystals of cholesterol that are found in atherosclerotic plaques. One goal of the research is to translate the molecular understanding of innate immune receptor activation into the generation of molecular tools that could lead to the development of specific diagnostics for inflammatory materials. Another goal is to devise means to pharmacologically interfere with the activation of innate immune receptors in order to develop novel approaches to treat inflammatory diseases such as Alzheimer's disease or atherosclerosis.

Education / Training

Humbolt University of Berlin, Germany, PhD, 2001
Free University of Berlin, Germany, Molecular Medicine, Hematology, MD, 1998

Appointments / Positions Held

2009 - present Full Professor of Medicine, Founder and Director of the Institute of Innate Immunity, University of Bonn, Germany
2011- present Leader, Cooperation Unit Innate Immunity in Neurodegeneration, DZNE, Bonn, Germany
2003 - present Assistant Professor of Medicine UMass Medical School
2008 Adjunct Professor II, Institute of Cancer Research and Molecular Medicine, Norwegian University of Science & Technology
2007 Founder and Co-Director of UMassNanoMed, UMassNanoMed Institute
2003-2006 Assistant Research Professor, UMass Medical School
2001 - 2003 Postdoctoral Fellow, Division of Infectious Disease UMass Medical School
2001 Postdoctoral Fellow, Evans Biomedical Research Center, Boston University of Medicine
1999 - 2001 Research Fellow, Molecular Sepsis Research Laboratories, Charité University Hospital, Humboldt-University of Berlin
1998 - 2000 Internship and Residency (Intensive Care) Department of Surgery and Surgical Oncology, Charité University Hospital, Humboldt-University of Berlin
1998 Visiting Scientist, Department of Lipid Biochemistry, Merck Research Laboratories

Honors / Awards

2015 Head of the Scientific Advisory Board of the Max-Planck-Institute for Infection Biology in Berlin
2015 Highly Cited Researcher in Immunology
2015 Listed in the 'World's Most Influential Scientific Minds'
2014 Highly Cited Researcher in Immunology
2014 Listed in the 'World's Most Influential Scientific Minds'
2014 Kavli Fellow of the United States National Academy of Sciences (NAS)
2013 ERC Consolidator Grant

10 Most Relevant Publications for Prof. Eicke Latz

1. Zimmer S, Grebe A, Bakke S, Bode N, Halvorsen B, Ulas T, Skjelland M, De Nardo D, Labzin L, Kerksiek A, Hempel C, Heneka M, Hawxhurst V, Fitzgerald M, Trebicka J, Björkhem I, Gustafsson JA, Westerterp M, Tall AR, Wright SA, Espevik T, Schultze JL, Nickenig G, Lütjohann D, **Latz E**. Cyclodextrin promotes atherosclerosis regression via macrophage reprogramming. Science Translational Medicine, April 2016, 6;8 (333)
2. Franklin BS, Bossaller L, De Nardo D, Ratter JM, Stutz A, Engels G, Brenker C, Nordhoff M, Mirandola SR, Al-Amoudi A, Mangan MS, Zimmer S, Monks BG, Fricke M, Schmidt RE, Espevik T, Jones B, Jarnicki AG, Hansbro PM, Busto P, Marshak-Rothstein A, Hornemann S, Aguzzi A, Kastenmüller W & **Latz E**. The adaptor ASC has extracellular and 'prionoid' activities that propagate inflammation. Nat Immunol, 2014, Aug;15(8):727-37
3. De Nardo D*, Labzin LI*, Kono H, Seki R, Schmidt SV, Beyer M, Xu D, Zimmer S, Lahrmann C, Schildberg FA, Vogelhuber J, Kraut M, Ulas T, Kerksiek A, Krebs W, Bode N, Grebe A, Fitzgerald ML, Hernandez NJ, Williams BR, Knolle P, Kneilling M, Rocken M, Lutjohann D, Wright SD, Schultze JL* and **Latz E***. (2014). High-density lipoprotein mediates anti-inflammatory reprogramming of macrophages via the transcriptional regulator ATF3. Nat Immunol, 15(2), 152-160.
4. Heneka MT*, Kummer MP, Stutz A, Delekate A, Schwartz S, Vieira-Saecker A, Griep A, Axt D, Remus A, Tzeng TC, Gelpi E, Halle A, Korte M, **Latz E*** and Golenbock DT*. (2013). NLRP3 is activated in Alzheimer's disease and contributes to pathology in APP/PS1 mice. Nature, 493(7434), 674-678.
5. Duewell P*, Kono H*, Rayner KJ, Sirois CM, Vladimer G, Bauernfeind FG, Abela GS, Franchi L, Nunez G, Schnurr M, Espevik T, Lien E, Fitzgerald KA, Rock KL, Moore KJ, Wright SD, Hornung V* and **Latz E***. (2010). NLRP3 inflammasomes are required for atherogenesis and activated by cholesterol crystals. Nature, 464(7293), 1357-1361.
6. Hornung V, Ablasser A, Charrel-Dennis M, Bauernfeind F, Horvath G, Caffrey DR, **Latz E*** and Fitzgerald KA*. (2009). AIM2 recognizes cytosolic dsDNA and forms a caspase-1- activating inflammasome with ASC. Nature, 458(7237), 514-518.
7. Bauernfeind FG, Horvath G, Stutz A, Alnemri ES, MacDonald K, Speert D, Fernandes- Alnemri T, Wu J, Monks BG, Fitzgerald KA, Hornung V* and **Latz E***. (2009). Cutting edge: NF-kappaB activating pattern recognition and cytokine receptors license NLRP3 inflammasome activation by regulating NLRP3 expression. J Immunol, 183(2), 787-791.
8. Hornung V, Bauernfeind F, Halle A, Samstad EO, Kono H, Rock KL, Fitzgerald KA* and **Latz E***. (2008). Silica crystals and aluminum salts activate the NALP3 inflammasome through phagosomal destabilization. Nat Immunol, 9(8), 847-856.
9. **Latz E**, Verma A, Visintin A, Gong M, Sirois CM, Klein DC, Monks BG, McKnight CJ, Lamphier MS, Duprex WP, Espevik T and Golenbock DT. (2007). Ligand-induced conformational changes allosterically activate Toll-like receptor 9. Nat Immunol, 8(7), 772-779.
10. **Latz E**, Schoenemeyer A, Visintin A, Fitzgerald KA, Monks BG, Knetter CF, Lien E, Nilsen NJ, Espevik T and Golenbock DT. (2004). TLR9 signals after translocating from the ER to CpG DNA in the lysosome. Nat Immunol, 5(2), 190-198.

* These authors contributed equally

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Institute of Reconstructive Neurobiology

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Institute of Reconstructive Neurobiology
E-Mail: hneuman1@uni-bonn.de

Research Expertise

Neuroinflammation, mechanisms of inflammatory neurodegeneration, microglia-neuron interaction, Siglecs

Education / Training

Technical University Munich, Germany, Habilitation in Neuroimmunology, 1998
University (FernUniversität) of Hagen, Germany, Business Administration, 1994
University of Würzburg and University of Munich (LMU), Germany, Medicine, MD Thesis, 1991

Appointments / Positions Held

2004 - present
Head of the Neural Regeneration Group, University of Bonn, Germany
2001 - 2004
Head of the Neuroimmunology Group European Neuroscience Institute Göttingen, University Göttingen
1995 - 2001
Group leader, Department of Neuroimmunology, Max-Planck-Institute of Neurobiology, Martinsried
1992 - 1994
Research fellow, Department of Neuroimmunology Max-Planck-Institute of Psychiatry, Martinsried
1990 - 1992
Medical Internship, Department of Neurology, University Ulm, Germany

Honors / Awards

2009 - present
Contribution to several patents (EP2424976B1; EP2424977B1; EP2783691A1)
2005 - 2010
VIce-coordinator of the EU Integrated Project NeuroproMiSe
2002 - 2009
Managing Board member of the Institute of MS Research
2003 - 2008
Editorial Board member of ‚Stem Cells'
2007
DANA-Foundation-Award, Neuroimmunology-Program
1996
PCR-Award Boehringer Mannheim
1992
Research scholarship (German science foundation)

10 Most Relevant Publications for Prof. Harald Neumann

1. Bodea LG, Wang Y, Linnartz-Gerlach B, Kopatz J, Sinkkonen L, Musgrove R, Kaoma T, Muller A, Vallar L, Di Monte DA, Balling R and **Neumann H.** (2014). Neurodegeneration by activation of the microglial complement-phagosome pathway. J Neurosci. 2014 Jun 18;34(25):8546-56.
2. **Neumann H.** and Daly MJ. (2013). Variant TREM2 as risk factor for Alzheimer's disease. N Engl J Med. 2013 Jan 10;368(2):182-4.
3. Claude J, Linnartz-Gerlach B, Kudin AP, Kunz WS and **Neumann H.** (2013). Microglial CD33-related Siglec-E inhibits neurotoxicity by preventing the phagocytosis associated oxidative burst. J. Neurosci. 33(46):18270-6.
4. Zhang B*, Gaiteri C*, Bodea LG*, Wang Z, McElwee J, Podtelezhnikov AA, Zhang C, Xie T, Tran L, Dobrin R, Fluder E, Clurman B, Melquist S, Narayanan M, Suver C, Shah H, Mahajan M, Gillis T, Mysore J, MacDonald ME, Lamb JR, Bennett DA, Molony C, Stone DJ, Gudnason V, Myers AJ, Schadt EE, **Neumann H**, Zhu J, Emilsson V. (2013). Integrated systems approach identifies genetic nodes and networks in late-onset Alzheimer's disease. Cell. 2013 Apr 25;153(3):707-20.
5. Wang Y, **Neumann H.** 2010. Alleviation of neurotoxicity by microglial human Siglec-11. J Neurosci 30: 3482-8
6. Beutner C, Roy K, Linnartz B, Nappoli I, **Neumann H.** 2010. Generation of microglial cells from mouse embryonic stem cells. Nature Protocols: 5:1481-94
7. Takahashi K, Prinz M, Stagi M, Chechneva O, **Neumann H.** 2007. TREM2-transduced myeloid precursors mediate nervous tissue debris clearance and facilitate recovery in an animal model of multiple sclerosis. PLoS Med 4: e124
8. Stagi M, Gorlovoy P, Larionov S, Takahashi K, **Neumann H.** 2006. Unloading kinesin transported cargoes from the tubulin track via the inflammatory c-Jun N-terminal kinase pathway. FASEB J 20: 2573-5
9. Takahashi K, Rochford CD, **Neumann H.** 2005. Clearance of apoptotic neurons without inflammation by microglial triggering receptor expressed on myeloid cells-2. J Exp Med 201: 647-57
10. Stagi M, Dittrich PS, Frank N, Iliev AI, Schwille P, **Neumann H.** 2005. Breakdown of axonal synaptic vesicle precursor transport by microglial nitric oxide. J Neurosci 25: 352-62

Prof. Pierluigi Nicotera, MD PhD

German Centre for Neurodegenerative Diseases (DZNE)

German Centre for Neurodegenrative Diseases (DZNE), Scientific Director and Chairman

E-Mail: pierluigi.nicotera@dzne.de

Research Expertise

Prof. Nicotera's main research focus is on molecular mechanisms of cell death and neurodegeneration.

Education / Training

University of Pavia, Medical School, Italy, Cardiology Consultant, 1987
Karolinska Institute, Stockholm, Biochemical Toxicology PhD, 1986
University of Pavia, Medical School, Italy, Medicine MD, 1982

Appointments / Positions Held

April 2009 - present
Scientific Director & Chairman of the Executive Board, German Centre for Neurodegenerative Diseases (DZNE), Bonn, Germany
2002 - 2009
Director of the British Medical Research Council Toxicology Unit and Honorary Professor of Neuroscience (Dept. of Cell Physiology & Pharmacology), University of Leicester
2005 - 2008
Teaching Professor of Toxicology, Faculty of Pharmacy University of Siena, Italy
1996 - 2002
Foreign Adjunct Professor in Toxicology, Karolinska Institute, Stockholm, Sweden
1995 - 2000
C4 Professor of Molecular Toxicology, University of Konstanz, Germany
1989 - 1994
Senior University Lecturer, Karolinska Institute, Stockholm, Sweden
1992
Docent in Molecular Toxicology, Karolinska Institute, Stockholm, Sweden
1986 - 1989
Research assistant Professor, Department of Karolinska Institute, Stockholm, Toxicology, Sweden

Honors / Awards

2013
The Chancellor's Award Lecture in Neuroscience at LSU Neuroscience Center of Excellence, New Orleans, USA
2012
Honorary Citizenship and Key to the City of New Orleans
2010
The Cardano Prize University of Pavia and Rotary Club Pavia
2003
The Chancellor's Award Lecture in Neuroscience at LSU Neuroscience Center of Excellence, New Orleans, USA
2002
"Molecular switches in neuronal cell death" Lecture at the 37th Nobel Conference on Apoptosis, Stockholm
1999
The Jacob Hooisma Honorary Lecture at the 7th Meeting of the International Neurotoxicology Association, Leicester
1995
The EUROTOX Award Lecture, 1st G. Zbinden Memorial Lecture Award, Prague
1992
"Nuclear Calcium Signalling" Lecture at the 20th Nobel Conference on Calcium Signalling, Saltsjöbaden, Sweden
1992
The International Life Science Institute Research Foundation U.S.A. (ILSI), award

10 Most Relevant Publications for Prof. Pierluigi Nicotera

1. Michod, D., Bartesaghi, S., Khelifi, A., Bellodi, C., Berliocchi, L., **Nicotera P.**, and Salomoni, P. (2012) Calcium-Dependent Dephosphorylation of the Histone Chaperone DAXX Regulates H3.3 Loading and Transcription upon Neuronal Activation. Neuron 74(1):122-135
2. Ziviani E, Lippi G, Bano D, Munarriz E, Guiducci S, Zoli M, Young KW, **Nicotera P**. 2011. Ryanodine receptor-2 upregulation and nicotine-mediated plasticity. EMBO J 30(1): 194-204.
3. Regad T, Bellodi C, **Nicotera P**, Salomoni P. 2009. The tumor suppressor Pml regulates cell fate in the developing neocortex. Nat Neurosci 12: 132-40.
4. Berliocchi L, Fava E, Leist M, Horvat V, Dinsdale D, Read D, **Nicotera P**. 2005. Botulinum neurotoxin C initiates two different programs for neurite degeneration and neuronal apoptosis. J Cell Biol 168: 607-18.
5. Bano D, Young KW, Guerin CJ, Lefeuvre R, Rothwell NJ, Naldini L, Rizzuto R, Carafoli E, **Nicotera P**. 2005. Cleavage of the plasma membrane Na+/Ca2+ exchanger in excitotoxicity. Cell 120: 275-85.
6. Orrenius S, Zhivotovsky B, **Nicotera P**. 2003. Regulation of cell death: the calcium- apoptosis link. Nat Rev Mol Cell Biol 4: 552-6.
7. Schierle GS, Hansson O, Leist M, **Nicotera P**, Widner H, Brundin P. 1999. Caspase inhibition reduces apoptosis and increases survival of nigral transplants. Nat Med 5: 97-100.
8. Leist M, Single B, Castoldi AF, Kuhnle S, **Nicotera P**. 1997. Intracellular adenosine triphosphate (ATP) concentration: a switch in the decision between apoptosis and necrosis. J Exp Med 185: 1481-6.
9. Bonfoco E, Krainc D, Ankarcrona M, **Nicotera P**, Lipton SA. 1995. Apoptosis and necrosis: two distinct events induced, respectively, by mild and intense insults with N-methyl-D- aspartate or nitric oxide/superoxide in cortical cell cultures. Proc Natl Acad Sci U S A 92: 7162-6.
10. Ankarcrona M, Dypbukt JM, Bonfoco E, Zhivotovsky B, Orrenius S, Lipton SA, **Nicotera P**. 1995. Glutamate-induced neuronal death: a succession of necrosis or apoptosis depending on mitochondrial function. Neuron 15: 961-73.

Prof. Markus M. Nöthen, MD

Institute of Human Genetics

Rheinische Friedrich-Wilhelms-Universität Bonn Institute of Human Genetics, Director

E-Mail: markus.noethen@uni-bonn.de

Research Expertise

The identification of the genetic causes of inherited diseases, and a special focus on genetically complex and multifactorial phenotypes.

Education / Training

University of Bonn, Germany, Human Genetics, Habilitation, 1996
University of Bonn, Germany, Human Genetics, Medical Board Qualification, 1995
University of Würzburg, Germany, Internal Medicine, Medical thesis, 1992, University of Würzburg, Germany, Clinical Medicine, MD, 1989

Appointments / Positions Held

2008 - present
Director and Chair, Institute of Human Genetics, University of Bonn, Germany
2006 - 2014
Vice Dean for Research, Medical Faculty, University of Bonn, Germany
2004 - present
Alfried Krupp von Bohlen und Halbach Professor in Genetic Medicine, University of Bonn, Germany
2004 - present
Head, Department of Genomics, Life & Brain Center, University of Bonn, Germany
2001 - 2004
Head of Department and Chair of Medical Genetics, University of Antwerp, Belgium
1999 - 2001
Assistant Medical Director, Institute of Human Genetics University of Bonn, Germany
1996 - 2001
Assistant Professor, Institute of Human Genetics, University of Bonn, Germany
1991 - 1996
Postdoctoral Fellow, Institute of Human Genetics, University of Bonn, Germany
1990 - 1991
Internship, Institute of Human Genetics, University of Bonn, Germany

Honors / Awards

2016 - present National Genetic Diagnostic Commission (Permanent Guest on behalf of the German Medical Association)
2015 - present Life & Brain GmbH, Bonn (Scientific Director)
2015 - present Project Committee of the National e:Med Programme (Elected Member)
2014 - 2015 Project Committee of the National e:Med Programme (Spokesman)
2013 - present Scientific Advisory Board of the Leipzig Research Center for Civilization Diseases (LIFE) (Member)
2013 - present Scientific Advisory Board of the Dr. Margarete Fischer-Bosch Institute of Clinical Pharmacology (IKP) and the Robert Bosch Hospital (RBK) (Member)
2012 - present International Advisory Board of iPSYCH (Lundbeck Foundation) (Chair)
2011 - present European Society of Human Genetics (Elected Member of the Board)
2010 - 2012 Project Committee of the National Genome Research Network (Spokesman)
2010 - present Scientific Advisory Board of the Medical Research Council (MRC) Centre for Neuropsychiatric Genetics and Genomics, Cardiff, UK (Member)
2010 - present German Academy of Sciences Leopoldina (National Academy of Sciences)
2009 - present Hermann Emminghausen-Prize
2008 - 2013 Project Committee of the National Genome Research Network (Elected member)
2007 - present Institute of Science and Ethics, Bonn (Member of the Scientific Advisory Board)
National Foundation for Legasthenia and Dyscalculia (Member of the Medical Advisory Board)
2006 - present National Alopecia Areata Foundation (Member of the Medical Advisory Board) Task Force on Genetics, World Federation of Societies of Biological Psychiatry
2005 - present International Society of Psychiatric Genetics (Elected Member of the Board)

10 Most Relevant Publications for Prof. Markus M. Nöthen

1. Betz RC, ..., Redler S, ..., Heilmann S, …, **Nöthen MM**, Daly MJ, Christiano AM (2015) Genome-wide meta-analysis in alopecia areata resolves HLA associations and reveals two new susceptibility loci. Nat Commun 6:5966
2. Gockel I, Becker J, ..., **Nöthen MM***, Boeckxstaens GE, de Bakker PI, Knapp M, Schumacher J (2014) Common variants in the HLA-DQ region confer susceptibility to idiopathic achalasia. Nat Genet 46:901-904. doi: 10.1038/ng.3029.
3. Kim S, Becker J, ..., **Nöthen MM***, Müller-Myhsok B, Pütz B, Hornung V, Schumacher J (2014)Characterizing the genetic basis of innate immune response in TLR4-activated human monocytes. Nat Commun 5:5236. doi: 10.1038/ncomms6236.
4. Schizophrenia Working Group of the Psychiatric Genomics Consortium (2014) Biological insights from 108 schizophrenia-associated genetic loci. Nature 511:421-427. doi: 10.1038/nature 13595.
5. Ramirez A, van der Flier WM, Herold C, Ramonet D, Heilmann S, ..., **Nöthen MM*** (2014) SUCLG2 identified as both a determinator of CSF Aβ1-42 levels and an attenuator of cognitive decline in Alzheimer's disease. Hum Mol Genet 23:6644-6658. doi: 10.1093/hmg/ddu372.
6. Mühleisen TW, Leber M, Schulze TG, Strohmaier J, Degenhardt F, …, **Nöthen MM***, Cichon S (2014) Genome-wide association study reveals two new risk loci for bipolar disorder. Nat Commun 5:3339.
7. Cross-Disorder Group of the Psychiatric Genomics Consortium, … , **Nöthen MM**, … , Wray N. 2013. Genetic relationship between five psychiatric disorders estimated from genome-wide SNPs. Nat Genet 45:984-994.
8. Ludwig KU, Mangold E, Herms S, Nowak S, Reutter H,…, **Nöthen MM**. 2012. Genome-wide meta-analyses of nonsyndromic cleft lip with or without cleft palate identify six new risk loci. Nat Genet 44:968-971.
9. Cichon S, Mühleisen TW, Degenhardt FA, Mattheisen M,..., Schumacher J, Maier W, Propping P, Rietschel M, **Nöthen MM***. 2011. Genome-wide association study identifies genetic variation in neurocan as a susceptibility actor for bipolar disorder. Am J Hum Genet 88:372-381.
10. Birnbaum S, Ludwig KU,..., **Nöthen MM**, Mangold E*. 2009. Key susceptibility locus for nonsyndromic cleft lip with or without cleft palate on chromosome 8q24. Nat Genet 41: 473-477.

* Publications with more than 10 authors have been shortened

Prof. Natalija Novak, MD

Department of Dermatology and Allergy

Rheinische Friedrich-Wilhelms-Universität Bonn
Department of Dermatology and Allergy
E-Mail: natalija.novak@ukb.uni-bonn.de

Research Expertise

Pathophysiology of atopic dermatitis including genetic changes, regulation of the surface expression of the high affinity receptor for IgE on dendritic cells, role of IgE receptor bearing Langerhans cells and inflammatory dendritic epidermal cells, predictive factors for atopy in cord-blood, development and characterization of new therapeutic strategies for atopic dermatitis, role of dendritic cells in the oral and nasal mucosa.

Education / Training

University of Bonn, Germany, Medicine, MD., 1998

Appointments / Positions Held

2008 - present
Full Professor of Dermatology and Allergy, University of Bonn, Germany
2009
Board, Andrology, University of Bonn, Germany
2006 - 2007
Visiting scientist, Heisenberg-Fellowship, Swiss Institute of Allergy and Asthma Research, Davos, Switzerland
2004
Board, Allergy, University of Bonn, Germany
2003
Assistant Professor, Dermatology and Allergy, University of Bonn, Germany
Assistant to Medical Director, Dermatology, University of Bonn, Germany
Board, Dermatology, University of Bonn, Germany

Honors / Awards

2014
Allergopharma Award
2012
Henning-Löwenstein Award World Allergy Organization
2008
Phadia International Award on Allergy Research, Phadia
2007
Heisenberg-Professorship, German Research Council
2006
Heisenberg-Fellowship, German Research Council
Travel Award EAACI Vienna, Austria
2005
Heinz Maier-Leibnitz Award, German Research Council
Research Award Atopische Dermatitis, Dermatologikum Hamburg
Young Investigator Travel Award, ISAD Meeting Acachand
2004
Karl-Hansen Memorial Award, German Society for Allergology and Immunology (DGAKI)
2003
Fujisawa "Young Investigator Achievements Award in Atopic Dermatitis Research"
Award, Herbert-Reeck-Society
Honourable Mention Diploma Pharmacia Research Foundation
Travel Award EAACI Meeting, Davos, Switzerland
Erich-Hoffmann Memorial Award
2002
Herbert-Herxheimer Award, German Society for Allergology and Immunology (DGAKI)
2002
BONFOR Award

10 Most Relevant Publications for Prof. Natalija Novak

1. Yu CF, Peng WM, Oldenburg J, Hoch J, Bieber T, Limmer A, Hartmann G, Barchet W, Eis-Hubinger AM, **Novak N**. 2010. Human plasmacytoid dendritic cells support Th17 cell effector function in response to TLR7 ligation. J Immunol 184: 1159-67.
2. Allam JP, Würtzen PA, Reinartz M, Winter J, Vrtala S, Chen KW, Valenta R, Wenghoefer M, Appel T, Gros E, Niederhagen B, Bieber T, Lund K, **Novak N**. 2010. Phl p 3 resorption in human oral mucosa leads to dose-dependent and time-dependent allergen binding by oral mucosal Langerhans cells, attenuates their maturation, and enhances their migratory and TGF-β1 and IL-10 producing properties. J Allergy Clin Immunol 126: 638-45.
3. Gros E, Bussmann C, Bieber T, Forster I, **Novak N**. 2009. Expression of chemokines and chemokine receptors in lesional and nonlesional upper skin of patients with atopic dermatitis. J Allergy Clin Immunol 124: 753-60 e1.
4. Esparza-Gordillo J, Weidinger S, Folster-Holst R, Bauerfeind A, Ruschendorf F, Patone G, Rohde K, Marenholz I, Schulz F, Kerscher T, Hubner N, Wahn U, Schreiber S, Franke A, Vogler R, Heath S, Baurecht H, **Novak N**, Rodriguez E, Illig T, Lee-Kirsch MA, Ciechanowicz A, Kurek M, Piskackova T, Macek M, Lee YA, Ruether A. 2009. A common variant on chromosome 11q13 is associated with atopic dermatitis. Nat Genet 41: 596-601.
5. Kwiek B, Peng WM, Allam JP, Langner A, Bieber T, **Novak N**. 2008. Tacrolimus and TGF-beta act synergistically on the generation of Langerhans cells. J Allergy Clin Immunol 122: 126-32, 32 e1.
6. Weidinger S, Illig T, Baurecht H, Irvine AD, Rodriguez E, Diaz-Lacava A, Klopp N, Wagenpfeil S, Zhao Y, Liao H, Lee SP, Palmer CN, Jenneck C, Maintz L, Hagemann T, Behrendt H, Ring J, Nothen MM, McLean WH, **Novak N**. 2006. Loss-of-function variations within the filaggrin gene predispose for atopic dermatitis with allergic sensitizations. J Allergy Clin Immunol 118: 214-9.
7. **Novak N**, Valenta R, Bohle B, Laffer S, Haberstok J, Kraft S, Bieber T. 2004. FcepsilonRI engagement of Langerhans cell-like dendritic cells and inflammatory dendritic epidermal cell-like dendritic cells induces chemotactic signals and different T-cell phenotypes in vitro. J Allergy Clin Immunol 113: 949-57.
8. **Novak N**, Allam JP, Hagemann T, Jenneck C, Laffer S, Valenta R, Kochan J, Bieber T. 2004. Characterization of FcepsilonRI-bearing CD123 blood dendritic cell antigen-2 plasmacytoid dendritic cells in atopic dermatitis. J Allergy Clin Immunol 114: 364-70.
9. **Novak N**, Tepel C, Koch S, Brix K, Bieber T, Kraft S. 2003. Evidence for a differential expression of the FcepsilonRIgamma chain in dendritic cells of atopic and nonatopic donors. J Clin Invest 111: 1047-56.
10. **Novak N**, Bieber T, Katoh N. 2001. Engagement of Fc epsilon RI on human monocytes induces the production of IL-10 and prevents their differentiation in dendritic cells. J Immunol 167: 797-804

Prof. Michael J. Pankratz, PhD

Life and Medical Sciences Institute (LIMES)

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Department of Molecular Brain Physiology and Behavior, Director
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Research Expertise

Prof. Pankratz is an expert on the genetics of nutrient control, feeding behavior, gustation and neuroendocrine circuits in drosophila.

Education / Training

University of California, Los Angeles USA, Biochemistry PhD, 1986
Johns Hopkins University, USA, Biology BA, 1980

Appointments / Positions Held

2008 - present
Full Professorship, Molecular Brain Physiology, University of Bonn
2001 - 2007
Senior Group Leader, Genetics, Karlsruhe, Institute of Technology
2001
Habilitation, Genetics, Karlsruhe University
1997 - 2001
Group Leader, Institute of Genetics, Karlsruhe, Institute of Technology
1993 - 1997
Staff Scientist, Institute of Biophysical Chemistry, Max Planck Institute
1988 - 1992
Postdoctoral Fellow, Institute for Genetics and Microbiology, University of Munich
1987 - 1988
Postdoctoral Fellow, Institute for Developmental Biology Max Planck Institute

Honors / Awards

2003 - 2005
Member of the Scientific Advisory Board "Network of Molecular Nutrition Research", State of Baden-Württemberg, Germany
2000 - 2001
Consultant for Aventis

10 Most Relevant Publications for Prof. Michael Pankratz

1. Hückesfeld S, Schoofs A, Schlegel P, Miroschnikow A, **Pankratz MJ**. Localization of Motor Neurons and Central Pattern Generators for Motor Patterns Underlying Feeding Behavior in Drosophila Larvae. PLoS One 2015 Aug 7;10(8):e0135011.
2. Alekseyenko OV, Chan YB, Fernandez Mde L, Bülow T, **Pankratz MJ**, Kravitz EA. Single serotonergic neurons that modulate aggression in Drosophila. Curr Biol 2014 Nov 17;24(22):2700-7.
3. Schoofs A, Hückesfeld S, Schlegel P, Miroschnikow A, Peters M, Zeymer M, Spieß R, Chiang AS, **Pankratz MJ**. 2014. Selection of motor programs for suppressing food intake and inducing locomotion in the Drosophila brain. PLoS Biol, in press.
4. Bader R, Sarraf-Zadeh L, Peters M, Moderau N, Stocker H, Köhler K, **Pankratz MJ***, Hafen E. 2013. The IGFBP7 homolog Imp-L2 promotes insulin signaling in distinct neurons of the Drosophila brain. J Cell Science 126, 2571-2576.
5. Bülow M, Aebersold R, **Pankratz MJ***, Jünger M. 2010. The Drosophila FoxA Ortholog Fork Head Regulates Growth and Gene Expression Downstream of Target of Rapamycin. PLoS One 5(12): e15171.
6. Min KJ, Yamamoto R, Buch S, **Pankratz MJ**, Tatar M. 2008. Drosophila lifespan control by dietary restriction independent of insulin-like signaling. Aging Cell 7: 199-206.
7. Buch S, Melcher C, Bauer M, Katzenberger J, **Pankratz MJ**. 2008. Opposing effects of dietary protein and sugar regulate a transcriptional target of Drosophila insulin-like peptide signaling. Cell Metab 7: 321-32.
8. Melcher C, Bader R, **Pankratz MJ**. 2007. Amino acids, taste circuits, and feeding behavior in Drosophila: towards understanding the psychology of feeding in flies and man. J Endocrinol 192: 467-72.
9. Bader R, Colomb J, Pankratz B, Schrock A, Stocker RF, **Pankratz MJ**. 2007. Genetic dissection of neural circuit anatomy underlying feeding behavior in Drosophila: distinct classes of hugin-expressing neurons. J Comp Neurol 502: 848-56.
10.Melcher C, Bader R, Walther S, Simakov O, **Pankratz MJ**. 2006. Neuromedin U and its putative Drosophila homolog hugin. PLoS Biol 4: e68.

* Corresponding author

Prof. Joachim L. Schultze, MD

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Genomics & Immunoregulation, Director
E-Mail: j.schultze@uni-bonn.de

Research Expertise

Professor Schultze's current central expertise is at the interphase of immunoregulation and genomics, with a focus on transcriptional and epigenetic control of cell activation and immunoregulation, particularly in macrophages and monocytes.

Education / Training

University of Freiburg, Medicine Fellow, 1992 - 1993
University of Tübingen, Medicine, MD, 1991
University of Tübingen, Medicine, State examination, 1991

Appointments / Positions Held

2007 - present W3 Professorship, Genomics & Immunoregulation, University of Bonn
2002 - 2007 C3 Professorship, Tumor Immunology, University of Cologne
1997 - 2002 Instructor in Medicine, Adult Oncology, Daner-Farber Cancer Institute, MA, USA
1996 - 1997 Instructor in Medicine, Hematologic Malignancies, Daner-Farber Cancer Institute, MA, USA
1995 - 1996 Research Associate, Hematology, Daner-Farber Cancer Institute, MA, USA
1993 - 1995 Research Fellow, Hematology, Daner-Farber Cancer Institute, MA, USA

Honors / Awards

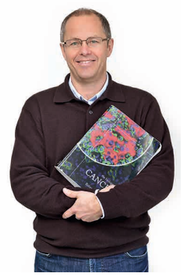
2015 - present Founding Director of the Platform for Genomics and Epigenomics at the University of Bonn and the DZNE
2012 - present Vice Dean for Research, Faculty for Mathematics and Natural Sciences, University of Bonn
2010 Patent: A method for lung cancer early detection and prognosis. Zander T, Schultze JL, Wolf J, Staratschek-Jox A, Debey-Pascher S, Eggle D, Boffetta P, Linseisen J.
2009 Patent: Anticancer Agent. Hoch M, Schultze JL, Loer B.
2009 Patent: Novel Marker Genes for regulatory T cells from human blood. Schultze JL, Beyer MD, Warner N, Hingorani R.
2002 Sofja-Kovalevskaja Award of the Alexander von Humboldt-Foundation
2000 Senior Investigator Award of the Multiple Myeloma Research Foundation
1999 Translational Research Award of the Leukemia & Lymphoma Society
1998 Special Fellowship Award of the Leukemia & Lymphoma Society
1997 Fellowship Award of the Lymphoma Research Foundation of America
1997 Travel Award Annual Meeting of the American Society of Hematology
1997 Leukemia Clinical Research Award (Deutsche Gesellschaft für Hämatologie und Onkologie)

10 Most Relevant Publications for Prof. Joachim L. Schultze

1. Beyer M, Abdullah Z, Chemnitz JM, Maisel D, Sander J, Lehmann C, Thabet Y, Shinde PV, Schmidleithner L, Köhne M, Trebicka J, Schierwagen R, Hofmann A, Popov A, Lang KS, Oxenius A, Buch T, Kurts C, Heikenwälder M, Fätkenheuer G, Lang PA, Hartmann P, Knolle PA, **Schultze JL**. TNF impairs CD4+ T-cell mediated immune control in chronic viral infection. Nat Immunol. 2016 Mar 7. [Epub ahead of print]
2. Schmidt SV, Krebs W, Ulas T, Xue J, Baßler K, Günther P, Hardt A-L, Schultze H, Sander J, Klee K, Theis H, Kraut M, Beyer M, **Schultze JL**. The transcriptional regulator network of human inflammatory macrophages is defined by open chromatin. Cell Res. 2016 Jan 5.
3. Ginhoux F, **Schultze JL**, Murray PJ, Ochando J, Biswas SK. New insights into the multidimensional concept of macrophage ontogeny, activation and function. Nat Immunol. 2015 Dec 17;17(1):34-40. All authors contributed equally
4. **Schultze JL**. Teaching 'big data' analysis to young immunologists. Nat Immunol. 2015 Aug 19;16(9):902-5.
5. Murray PJ, Allen JE, Biswas SK, Fisher EA, Gilroy DW, Goerdt S, Gordon S, Hamilton JA, Ivashkiv LB, Lawrence T, Locati M, Mantovani A, Martinez FO, Mege JL, Mosser DM, Natoli G, Saeij JP, **Schultze JL**, Shirey KA, Sica A, Suttles J, Udalova I, van Ginderachter JA, Vogel SN, Wynn TA. Macrophage activation and polarization: nomenclature and experimental guidelines. Immunity. 2014; 41(1):14-20 authors in alphabetical order except for P. Murray
6. Xue J, Schmidt SV, Sander J, Draffehn A, Krebs W, Quester I, De Nardo D, Gohel TD, Emde M, Schmidleithner L, Ganesan H, Nino-Castro A, Mallmann MR, Labzin L, Theis H, Kraut M, Beyer M, Latz E, Freeman TC, Ulas T, **Schultze JL**. Transcriptome-based network analysis reveals a spectrum model of human macrophage activation. Immunity. 2014; 40(2):274-88
7. De Nardo D, Labzin LI, Kono H, Seki R, Schmidt SV, Beyer M, Xu D, Zimmer S, Lahrmann C, Schildberg FA, Vogelhuber J, Kraut M, Ulas T, Kerksiek A, Krebs W, Bode N, Grebe A, Fitzgerald ML, Hernandez NJ, Williams BR, Knolle P, Kneilling M, Röcken M, Lütjohann D, Wright SD, **Schultze JL***, Latz E*. High-density lipoprotein mediates anti-inflammatory reprogramming of macrophages via the transcriptional regulator ATF3. Nat Immunol. 2014; 15(2):152-60 *Shared last author
8. Beyer M, Thabet Y, Müller RU, Sadlon T, Classen S, Lahl K, Basu S, Zhou X, Bailey-Bucktrout SL, Krebs W, Schönfeld EA, Böttcher J, Golovina T, Mayer CT, Hofmann A, Sommer D, Debey-Pascher S, Endl E, Limmer A, Hippen KL, Blazar BR, Balderas R, Quast T, Waha A, Mayer G, Famulok M, Knolle PA, Wickenhauser C, Kolanus W, Schermer B, Bluestone JA, Barry SC, Sparwasser T, Riley JL, **Schultze JL**. Repression of the genome organizer SATB1 in regulatory T cells is required for suppressive function and inhibition of effector differentiation. Nat Immunol. 2011; 12(9):898-907
9. Becker T, Loch G, Beyer M, Zinke I, Aschenbrenner AC, Carrera P, Inhester T, **Schultze JL**, Hoch M. FOXO-dependent regulation of innate immune homeostasis. Nature. 2010; 463(7279):369-73
10. Trojan A*, **Schultze JL***, Witzens M, Vonderheide RH, Ladetto M, Donovan JW, Gribben JG. Immunoglobulin framework-derived peptides function as cytotoxic T-cell epitopes commonly expressed in B-cell malignancies. Nat Med. 2000; 6(6):667-72 *Shared first author

Prof. Thomas Tüting, MD

Department of Dermatology and Allergy



Rheinische Friedrich-Wilhelms-Universität Bonn
Department of Dermatology and Allergy
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Research Expertise

Role of UV irradiation and the immune system in the pathogenesis of melanoma; mechanisms of melanoma metastasis and therapy resistance; preclinical and clinical evaluation of approaches combining immunotherapies and other treatment modalities for melanoma; development of novel genetic mouse models to study inflammation-induced phenotypic plasticity and reciprocal interactions between melanoma, immune and endothelial cells in the perivascular niche.

Education / Training

University of Frankfurt, Germany, MD, Thesis, 2000
University of Mainz, Germany, Dermatology and Allergic Diseases, Board Certification, 1998
University of Frankfurt School of Medicine, Medicine, MD, 1987

Appointments / Positions Held

2015 - present Professor and Chairman, Department of Dermatology, University of Magdeburg, Germany
2001 - present
Associate Professor and Laboratory Head, Experimental Dermatology, University of Bonn, Germany
2001 - present
Clinical work, General and Oncologic Dermatology, University of Bonn, Germany
1998 - 2001
Clinical and Scientific Work, Department of Dermatology, University of Mainz, Germany
1995 - 1997
Research Fellow in Tumor Immunology and Gene Therapy, Department of Surgery, University of Pittsburgh School of Medicine, Pittsburgh, PA
1991 - 1995
Residency in Dermatology and Allergic Diseases, Department of Dermatology, Military Hospital Koblenz and University of Mainz, Germany
1988 - 1991
Drafted as Airforce Medical Officer, Fighter- Bomber Wing 33, Cochem, Germany

Honors / Awards

2015
Photodermatology Research Award (Roche Posay)
2014
Arnold Rikli prize of the Jörg Wolff Stiftung
2014
German skin cancer research prize of the German skin cancer foundation
2009
Steigleder prize of the AG Dermatological Histology
2006
Translational Research prize of the AG Dermatological Research
2000
Research Award of the Erich Hoffmann Society, Bonn

10 Most Relevant Publications for Prof. Thomas Tüting

1. Baar M, Coquille L, Mayer H, Hölzel M, Rogava M, **Tüting T**, Bovier A. A stochastic model for immunotherapy of cancer. Sci Rep 6:24169, 2016.
2. Hölzel M, Landsberg J, Glodde N, Bald T, Rogava M, Riesenberg S, Becker AJ, Jonsson G, **Tüting T**. A preclinical model of malignant peripheral nerve sheath tumor-like melanoma is characterized by infiltrating mast cells. Cancer Res 76:251-63, 2015.
3. Riesenberg S, Groetchen A, Siddaway R, Bald T, Reinhardt J, Smorra D, Kohlmeyer J, Renn M, Phung B, Aymans P, Schmidt T, Hornung V, Davidson I, Goding CR, Jönsson G, Landsberg J, **Tüting T**, Hölzel M. MITF and c-Jun antagonism interconnects melanoma dedifferentiation with pro-inflammatory cytokine responsiveness and myeloid cell recruitment. Nat Commun 6:8755, 2015.
4. Bald T, Landsberg J, Lopez-Ramos D, Renn M, Glodde N, Jansen P, Gaffal E, Steitz J, Tolba R, Kalinke U, Limmer A, Jönsson G, Hölzel M, **Tüting T**. Immune-cell poor melanomas benefit from PD-1 blockade after targeted type I IFN activation. Cancer Discovery 4:674-87, 2014.
5. Bald T, Quast T, Landsberg J, Rogava M, Glodde N, Lopez-Ramos D, Kohlmeyer J , Riesenberg,S. van den Boorn-Konijnenberg D, Hömig-Hölzel C, Reuten R, Schadow B, Weighardt I, Wenzel D, Helfrich I, Schadendorf D, Bloch W, Bianchi ME, Koch M, Fleischmann BK, Förster I, Kastenmüller W, Kolanus W, Hölzel M, Gaffal E, **Tüting T.** Ultraviolet radiation-induced inflammation promotes angiotropism and metastasis in melanoma. Nature 507:109-13, 2014.
6. Hölzel M, Bovier A, **Tüting T.** Plasticity of tumour and immune cells: a source of heterogeneity and a cause for therapy resistance? Nat Rev Cancer. 13:365-76, 2013.
7. Gaffal E, Cron M, Glodde N, Bald T, Kuner R, Zimmer A, Lutz B, **Tüting T**. Cannabinoid 1 receptors in keratinocytes modulate proinflammatory chemokine secretion and attenuate contact allergic inflammation. J. Immunol. 190:4929-36, 2013.
8. Gehrke N, Mertens C, Zillinger T, Wenzel J, Bald T, Zahn S, **Tüting T**, Hartmann G, Barchet W. Oxidative damage of DNA confers resistance to cytosolic nuclease TREX1 degradation and potentiates STING-dependent immune sensing. Immunity. 39:482-95, 2013.
9. Landsberg J, Kohlmeyer J, Renn M, Bald T, Rogava M, Cron M, Fatho M, Lennerz V, Wölfel T, Hölzel H, **Tüting T**. Melanomas resist T-cell therapy through inflammation-induced reversible dedifferentiation. Nature. 490:412-416, 2012.
10. Kohlmeyer J, Cron M, Landsberg J, Bald T, Renn M, Mikus S, Bondong S, Wikasari D, Gaffal E, Hartmann G, **Tüting T**. Complete regression of advanced primary and metastatic mouse melanomas following combination chemoimmunotherapy. Cancer Res 69:6265-74, 2009.

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Prof. Andreas Zimmer, PhD

Institute of Molecular Psychiatry

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Institute of Molecular Psychiatry, Director
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Research Expertise

Prof. Zimmer and his groups are interested in the molecular mechanisms of neuropsychiatric disorders with a focus on addiction, pain and affective disorders, molecular biology of modulatory neurotransmitters, and the molecular biology of aging.

Education / Training

Max Planck Institute for Biophysical Chemistry,
Microbiology, PhD, 1989
Justus-Liebig Universität Giessen, Biology,
Diploma, 1986

Appointments / Positions Held

2006 - present
Professor of Molecular Psychiatry (W3), Director, Institute for Molecular Psychiatry, University of Bonn
2005 - 2006
Professor of Cell Biology (W3), University of Bielefeld
1999 - 2005
Professor for Molecular Neurobiology (C3), University of Bonn
1997 - 1999
Adjunct Professor, Department of Pharmacology, Georgetown University, Medical School, USA
1997 - 2000
Research Fellow, National Institute of Mental Health, USA
1995 - 1997
Visiting Research Fellow, National Institute of Mental Health, USA
1991 - 1995
Visiting Associate, National Institute of Mental Health, USA
1991 - 2000
Section Head, National Institute of Mental Health, USA
1989 - 1991
Postdoctoral researcher, DFG-Fellow, National Institute of Mental Health, USA

Honors / Awards

2000
U.S. Department of Health and Human Services Special Act or Service Award
1990 - 1992
DFG-Fellow
1987 - 1989
Max-Planck-Fellow
1989
PhD thesis awarded ‘summa cum laude’

10 Most Relevant Publications for Prof. Andreas Zimmer

1. Miró X, Meier S, Dreisow ML, Frank J, Strohmaier J, Breuer R, Schmäl C, Albayram O, Pardo-Olmedilla MT, Mühleisen TW, Degenhardt FA, Mattheisen M, Reinhard I, Bilkei-Gorzo A, Cichon S, Seidenbecher C, Rietschel M, Nöthen MM, **Zimmer A.** (2012). Studies in humans and mice implicate neurocan in the etiology of mania. Am J Psychiatry,169(9):982-90.
2. Gertsch J, Leonti M, Raduner S, Racz I, Chen JZ, Xie XQ, Altmann KH, Karsak M, **Zimmer A**. 2008. Beta-caryophyllene is a dietary cannabinoid. Proc Natl Acad Sci U S A 105: 9099-104.
3. Karsak M, Gaffal E, Date R, Wang-Eckhardt L, Rehnelt J, Petrosino S, Starowicz K, Steuder R, Schlicker E, Cravatt B, Mechoulam R, Buettner R, Werner S, Di Marzo V, Tuting T*, **Zimmer A***. 2007. Attenuation of allergic contact dermatitis through the endocannabinoid system. Science 316: 1494-7.
4. Bilkei-Gorzo A, Racz I, Valverde O, Otto M, Michel K, Sastre M, **Zimmer A**. 2005. Early age-related cognitive impairment in mice lacking cannabinoid CB1 receptors. Proc Natl Acad Sci U S A 102: 15670-5.
5. Nadeau JH, Balling R, Barsh G, Beier D, Brown SD, Bucan M, Camper S, Carlson G, Copeland N, Eppig J, Fletcher C, Frankel WN, Ganten D, Goldowitz D, Goodnow C, Guenet JL, Hicks G, Hrabe de Angelis M, Jackson I, Jacob HJ, Jenkins N, Johnson D, Justice M, Kay S, Kingsley D, Lehrach H, Magnuson T, Meisler M, Poustka A, Rinchik EM, Rossant J, Russell LB, Schimenti J, Shiroishi T, Skarnes WC, Soriano P, Stanford W, Takahashi JS, Wurst W, **Zimmer A.** 2001. Sequence interpretation. Functional annotation of mouse genome sequences. Science 291: 1251-5.
6. **Zimmer A**, Zimmer AM, Hohmann AG, Herkenham M, Bonner TI. 1999. Increased mortality, hypoactivity, and hypoalgesia in cannabinoid CB1 receptor knockout mice. Proc Natl Acad Sci U S A 96: 5780-5.
7. Hahn H, Wojnowski L, Zimmer AM, Hall J, Miller G, **Zimmer A**. 1998. Rhabdomyosarcomas and radiation hypersensitivity in a mouse model of Gorlin syndrome. Nat Med 4: 619-22.
8. Wojnowski L, Zimmer AM, Beck TW, Hahn H, Bernal R, Rapp UR, **Zimmer A**. 1997. Endothelial apoptosis in Braf-deficient mice. Nat Genet 16: 293-7.
9. Konig M, Zimmer AM, Steiner H, Holmes PV, Crawley JN, Brownstein MJ, **Zimmer A**. 1996. Pain responses, anxiety and aggression in mice deficient in pre-proenkephalin. Nature 383: 535-8.
10. **Zimmer A**, Gruss P. 1989. Production of chimaeric mice containing embryonic stem (ES) cells carrying a homoeobox Hox 1.1 allele mutated by homologous recombination. Nature
338: 150-3.

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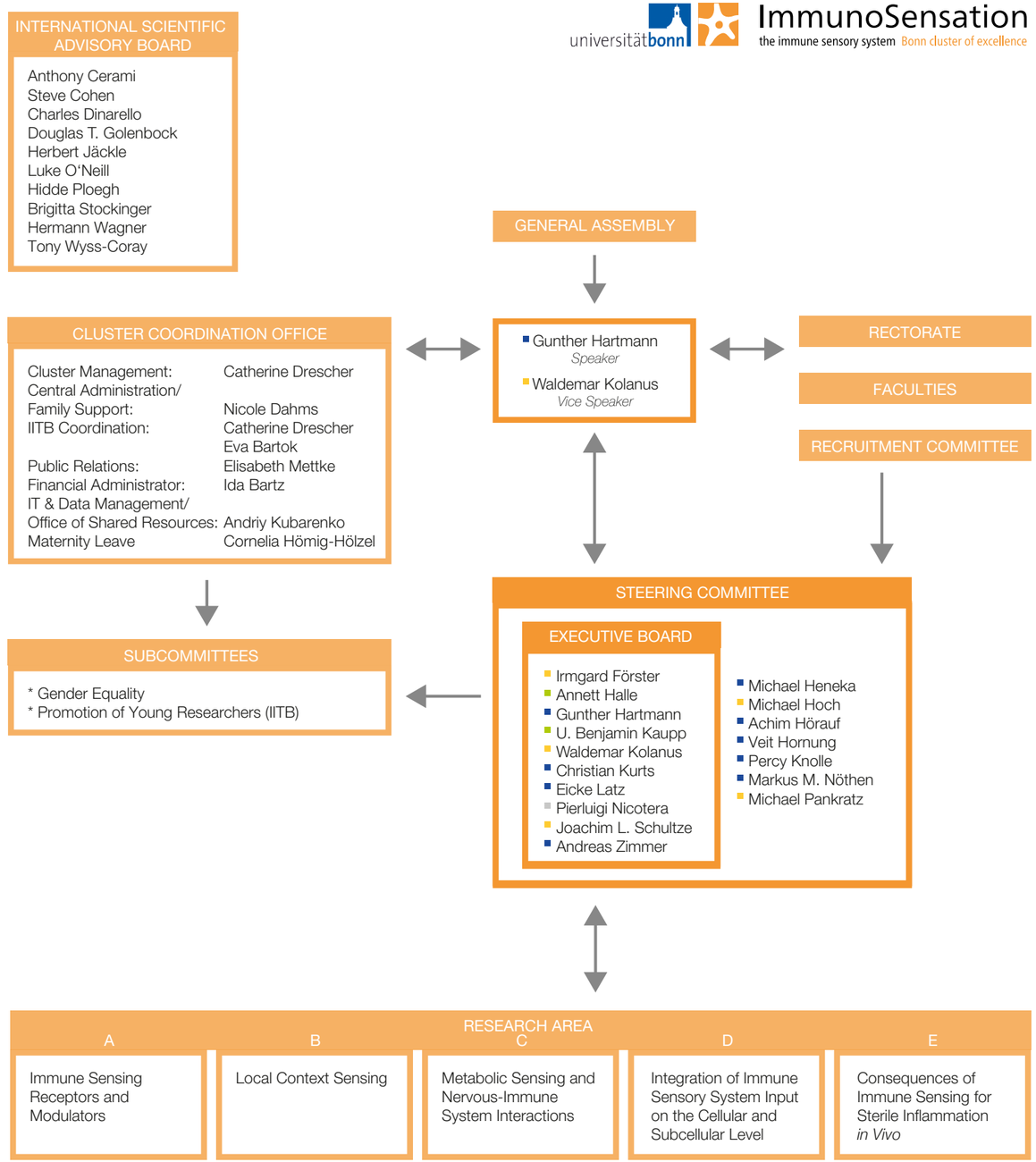
Main Building University of Bonn at night
by Frank Luerweg

Organigram

universitätbonn ImmunoSensation
the immune sensory system Bonn cluster of excellence



universitäts klinikumbonn
caesar center of advanced european studies and research
DZNE German Center for Neurodegenerative Diseases within the Helmholtz Association
LIMES Life & Medical Sciences Institute

Participating Institutions

Participating Institutions & CCO

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Neurodegenerative Diseases
Ludwig-Erhard-Allee 2
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Maternity Leave

Eva Bartok, MD
IITB support

Appendix: Shared Resources

Shared Resources Initiative

The Office of Shared Resources (OSR) has been part of the Cluster Coordination Office since 2014. The OSR acts as a central access point for resources that are shared on a scientific basis (collaboration), on a financial basis (usage of instruments and equipment) or within Core Facilities/ platforms. The OSR also provides a “Handbook of Shared Resources”, which provides users with an overview of the services available and how to access them. In 2015, the OSR implemented a regular Core Facility meeting with platform and facility managers, where ideas, problems and technical questions are discussed to further improve user services. All Core Facilities and platforms (**Figure 1**) are willing to share their expertise and instrumentation with Cluster members.

Shared Resources Core Unit

Institute of Clinical Chemistry and Clinical Pharmacology, Faculty of Medicine
Director: Prof. Gunther Hartmann

Contact: Dr. Andriy Kubarenko
a.kubarenko@uni-bonn.de
0228/287-51290

Flow Cytometry Core Facility

Institute of Molecular Medicine
Faculty of Medicine
Director: Prof. Veit Hornung

Contact: Dr. Elmar Endl
elmar.endl@uni-bonn.de
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Imaging Core Facility

Institute of Innate Immunity
Faculty of Medicine
Director: Prof. Eicke Latz

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Transgenic Core Facility

Haus für Experimentelle Therapie (HET), Faculty of Medicine
Director: Dr. Wolfgang Eichelkraut

Contact: Dr. David-Marian Otte
d.otte@uni-bonn.de
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Mass Spectrometry Core Facility

Institute of Biochemistry & Molecular Biology, Faculty of Medicine
Director: Prof. Volkmar Gieselmann

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Live Cell Imaging Platform

Molecular Immunology and Cell Biology
LIMES Institute
Director: Prof. Waldemar Kolanus

Contact: Dr. Thomas Quast
quast@uni-bonn.de
0228/73-62781

Next Generation Sequencing Core Facility

Institute of Human Genetics
Faculty of Medicine
Director: Prof. Markus Nöthen

Contact: Dr. rer. nat. Kerstin Ludwig
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0228/6885420

Genomics Platform

Genomics and Immunoregulation
LIMES Institute
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Figure 1 Shared Resources Initiative Team (December 2015).

Imprint

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