# Alumni Symposium 2022
# Robert Koch Postdoc Prize Awardees

## 25 years of Postdoc prizes

Robert-Koch-Stiftung

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# Welcome

Since 1998, the Robert Koch Foundation has honored young postdoctoral scientists for their outstanding research with the Robert Koch Postdoctoral Prizes in cooperation with the German Societies for Hygiene and Microbiology, Immunology and Virology. This year will mark the 25th occasion that these three Robert Koch Postdoctoral Prizes are awarded. So far, 75 individuals have received this distinctive honor, an entire generation of highly-rated scientists and outstanding experts in infectiology.

The Robert Koch Foundation has been eager to organize a get-together of this group from time to time in order to strengthen the contact to and among the awardees, and to provide a communication platform for the awardees to discuss their scientific development since receiving the prize. In 2016, the first highly successful Robert Koch PostDoc Alumni Symposium was held in the "Max Planck Institut für Infektionsbiologie". Today, we look forward to the second symposium of the Robert Koch Postdoctoral Prize alumni, this time at the "Humboldt-Universität". We expect approximately 30 presentations and intense discussions, after two years of the COVID-19 pandemic with essential contributions from virology, microbiology and immunology. In addition, this year´s Robert Koch Prize awardees Philip Felgner, Drew Weissman and Jörg Hacker will present at the symposium. It will be a unique event in the true heritage of Robert Koch.

We would like to thank the organizers of the symposium for making this event possible: Christian Drosten, Mathias Hornef and Max Löhning, who received the Robert Koch Postdoctoral Prizes in 2004; Katrin Moser and Sabine Timmermann for the organizational support. Finally, we would like to thank GlaxoSmithKline and BioNTech for their generous sponsoring of this event.

Wolfgang Plischke, Chairman of the Board of Directors
Andreas Radbruch, Deputy Chairman of the Board of Directors
and Chairman of the Scientific Advisory Council

# Welcome



It is our pleasure to welcome you to the Alumni Symposium of Robert Koch Postdoc Prize Awardees.

We had originally planned to welcome you in Berlin in autumn 2020. By then, the Corona pandemic showed its full impact on public life, travel, and personal meetings. Concurrently, the pandemic put our disciplines, virology, immunology, infection control and microbiology, into the limelight. Many of the Robert Koch Postdoc Prize alumni have contributed their expertise to studying and combatting the novel pandemic agent. In a short period of time, major progress has been made on our understanding of the biology and pathogenesis of SARS-CoV-2. However, it was thorough basic research performed over decades that enabled the swift development of innovative interventions such as mRNA vaccines that had their medical breakthrough as a critical component of our measures to restrict the burden of disease of COVID-19 during the pandemic.

And besides COVID-19, none of the other areas in our exciting field of research have lost their relevance and fascination. The program of the symposium reflects the wide range of expertises of the Robert Koch Postdoc Prize Awardees, with sessions on innate and adaptive immunity, microbiology, and virology.

We are particularly delighted to include in our program the Robert Koch lectures by this year's Robert Koch award winners, Phil Felgner and Drew Weissman, as well as the lecture by Jörg Hacker, who will be honored with the Robert Koch medal.

We are looking forward to two exciting days. Enjoy the science.

Christian Drosten
Mathias Hornef
Max Löhning

# Alumni Symposium 2022
# Robert Koch Postdoc Prize Awardees

10./11. November 2022
Heilig-Geist-Kapelle
of the Humboldt-Universität zu Berlin
Spandauer Straße 1
10178 Berlin

**Organizers**
Christian Drosten, Mathias Hornef, Max Löhning, Andreas Radbruch,
Sabine Timmermann and Katrin Moser

**Participants**
Alumni of the Robert Koch Post-doctoral Award, Robert Koch Awardees 2022,
Board of Directors, Scientific Advisory Council Members, and Sponsor Representatives

https://www.robert-koch-stiftung.de

# About the Heilig-Geist-Kapelle

The Heilig-Geist-Kapelle (Holy Spirit Chapel) was built together with the Heilig-Geist-Spital (Holy Spirit Hospital) around 1300 and is thus one of the oldest buildings in Berlin. It was first mentioned in 1313 in a deed of donation by the Knight Burghard Grevelhout. It was not until around 1600 that the chapel received its famous star-vaulted ceiling, instead of the previously unspectacular flat roof. When the Heilig-Geist-Spital gave way in 1825 to the new building of the Berlin Merchants' School, now known as the Faculty of Economics, the Heilig-Geist-Kapelle was used as a lecture hall from 1906. In 2005, the chapel underwent extensive restoration.

Photos: Humboldt-Innovation GmbH

Heilig-Geist-Kapelle
of the Humboldt-Universität zu Berlin
Spandauer Straße 1
10178 Berlin

# Program

## Thursday, 10th of November 2022

11:00 Registration and light lunch
12:00 Welcome: Wolfgang Plischke, Robert-Koch-Stiftung

### Innate Immunity

**Chairs:** Heidrun Moll, Julius-Maximilians-Universität Würzburg
Carsten Watzl, Leibniz-Institut für Arbeitsforschung an der TU Dortmund

12:05 Anne Krug, Ludwig-Maximilians-Universität München
Dendritic cell subsets in viral infection
12:25 Jost Enninga, Institut Pasteur, Paris
Intracellular niche formation of entero-invasive bacterial pathogens
12:45 Olaf Groß, Universitätsklinikum Freiburg
Small molecules triggering the NLRP3 inflammasome
13:05 Friedemann Weber, Justus-Liebig University Gießen
Viral Strategies against innate immunity ...
13:25 Melanie Brinkmann, Technische Universität Braunschweig
... reveal novel cellular restriction factors

13:45 Coffee break

### Microbiology

**Chairs:** Mathias Hornef, Universitätsklinikum Aachen
Peter Hammann, Helmholtz-Institut für Pharmazeutische Forschung des Saarlandes, HZI

14:15 Mathias Hornef, Universitätsklinikum Aachen
Host-Microbial Interaktion in the neonate Intestine
14:35 Ulrich Dobrindt, Westfälische Wilhelms-Universität Münster
Symptomatic infection vs. asymptomatic colonization – lessions from E. coli

14:55 Cynthia Sharma, Universität Würzburg
RNA-based regulation in virulence control and stress response of pathogenic bacteria

15:15 Bärbel Stecher-Letsch, Ludwig-Maximilians-Universität München
Synthetic communities to understand functions of the intestinal microbiome

15:35 Tanja Schneider, Universität Bonn
Bacterial targets for new antibiotics

15:55 Michael Sigal, Charité – Universitätsmedizin Berlin
Epithelial stem cells act as key sensors and effectors of mucosal infections

16:15 Coffee break

## Adaptive Immunity

**Chairs:** Max Löhning, Leibniz-Institut Deutsches Rheuma-Forschungszentrum Berlin
Lothar Wieler, Robert Koch-Institut Berlin

16:45 Astrid Westendorf, Universität Duisburg-Essen
Treg responses in the intestinal mucosa – inflammation versus regulation

17:05 Henning Grüll, Uniklinik Köln
Antibody-Mediated Prevention and Treatment of Viral Infections

17:25 Max Löhning, Deutsches Rheuma-Forschungszentrum Berlin, ein Leibniz-Institut
Immunity and immunopathology in antiviral immune responses

17:45 Kilian Schober, Friedrich-Alexander-Universität Erlangen-Nürnberg
T cell receptor-driven fate of pathogen antigen-specific T cell responses

18:05 Carsten Watzl, Leibniz-Institut für Arbeitsforschung an der TU Dortmund
Inside the Mind of a Serial Killer – Cytotoxic Mechanisms of Natural Killer Cells

18:25 End of day 1

19:00 Dinner
HABEL am Reichstag
Luisenstraße 19, 10117 Berlin

## Friday, 11th of November 2022

8:30 Robert Koch Lecture, Robert Koch Award Laureate 2022

Philip Felgner
University of California, Irvine, USA
150 Years of Vaccine Science

Introduction:
Peter Palese, Icahn School of Medicine at Mount Sinai, New York, USA

9:30 Robert Koch Lecture, Robert Koch Award Laureate 2022

Drew Weissman
University of Pennsylvania School of Medicine, USA
Nucleoside-modified mRNA-LNP Therapeutics

Introduction: Andreas Radbruch, Leibniz-Institut
Deutsches Rheuma-Forschungszentrum Berlin

10:30 Coffee break

11:00 Robert Koch Lecture, Robert Koch Gold Medal Laureate 2022

Jörg Hacker
Leopoldina – Nationale Akademie der Wissenschaften
Menschen, Seuchen und Mikroben – Infektionen als gesellschaftliche Herausforderung

Introduction: Wolfgang Plischke, Robert-Koch-Stiftung

### Virology, part 1

**Chairs:** Hans-Georg Kräusslich, Universitätsklinikum Heidelberg
Karl Ziegelbauer, Almirall R&D

11:30 Christian Drosten, Charité – Universitätsmedizin Berlin
Understanding fitness in emerging coronaviruses

11:50 Stefan Pöhlmann, Deutsches Primatenzentrum, Göttingen
Host cell entry of SARS-CoV-2 and its inhibition

12:10 Elke Mühlberger, Boston University
Does a dangerous cousin make you dangerous? Assessing the pathogenic potential of newly discovered zoonotic viruses that are closely related to known human pathogens

12:30 Michael Schindler, Universitätsklinikum Tübingen
Development of a novel class of broadly acting flavivirus assembly inhibitors

12:50 Matthias Dobbelstein, Universitätsmedizin Göttingen
A new job for cancer drugs – inhibitors of nucleotide biosynthesis interfere with the replication of the coronavirus SARS-CoV-2

13:10 Lunch break

## Virology, part 2

**Chairs:** Christian Drosten, Charité – Universitätsmedizin Berlin
Dominique Soldati-Favre, University of Geneva Medical School

14:10 Jens Bosse, Heinrich-Pette-Institut, Hamburg
Life is fluid: How (some) viruses use liquid-liquid phase separation to induce viral replication compartments

14:30 Gisa Gerold, Stiftung Tierärztliche Hochschule Hannover
To bend or not to bend: How RNA viruses hijack membrane protein complexes

14:50 Benedikt Kaufer, Freie Universität Berlin
Herpesvirus integration: Latency, endogenous viruses and cancer

15:10 Christine Goffinet, Charité – Universitätsmedizin Berlin
Understanding obstacles to HIV-1 cure: Antipodal role of innate immunity in the context of latency reversal.

15:30 Closing remarks and end of Symposium

16:30 **Robert Koch Foundation Prize Award Ceremony**
Berlin-Brandenburg Academy of Sciences and Humanities,
Gendarmenmarkt, Berlin

# Abstracts

In order of their contribution to the program.

Anne Krug
Ludwig-Maximilians-Universität München

# Dendritic cell subsets in viral infection

Vaccination with the live-attenuated yellow fever virus vaccine strain (YF-17D) provides life-long protection against infection and is a unique model for studying the immune response to an acute self-limiting RNA virus infection in humans. To elucidate the early innate immune events which precede the rapid generation of adaptive immunity, we investigated the response of blood dendritic cell (DC) and monocyte subpopulations to YF-17D in vivo by high-dimensional flow cytometry and bulk RNA-sequencing before and at multiple timepoints after vaccination. We detected transient activation and up-regulation of a common Interferon-stimulated gene signature in all DC and monocyte subsets on day 3 and 7 after vaccination as well as cell-type specific responses. Thus, the innate immune response to YF-17D vaccination is marked by concerted temporary activation of all circulating DC and monocyte subpopulations with distinct and overlapping gene expression programs. This well-coordinated innate response limits viral replication preventing vaccine-associated disease and is followed by rapid induction of protective antibody and T cell responses.

Frequencies and activation state of circulating DC and monocyte subsets were analyzed by flow cytometry in a cohort of hospitalized COVID-19 patients and in outpatients with a mild course of the disease. Compared to YF17D vaccination, patients with more severe COVID-19 showed low expression of costimulatory molecule CD86 in the cDC2, DC3 and monocytes. In contrast, non-hospitalized patients with mild COVID-19 showed an upregulation of CD86 similar to what was observed in YF17D vaccinees. The down-regulation of CD86 in severe COVID-19 was accompanied by upregulation of PD-L1. This altered phenotype of peripheral APCs coincided with a reduced capability of DC3 and monocytes isolated from the blood of COVID-19 patients to co-stimulate autologous T cell activation and proliferation in vitro. An increase of Ki67+ DCs alongside temporary reductions in cDC1 and cDC2 frequencies indicated a higher turnover of the blood DC compartment in both COVID-19 patients and YF17D vaccinees. Profound longer lasting depletion of circulating DCs was a characteristic feature of severe COVID-19 while the reduction of blood DCs was transient after YF17D vaccination. Functional impairment and delayed regeneration of DCs and monocytes may have consequences for susceptibility to secondary infections and therapy of COVID-19 patients.

Jost Enninga
Institut Pasteur, Paris

## Intracellular niche formation of entero-invasive bacterial pathogens

Enteroinvasive pathogens, such as Shigella and Salmonella, induce their uptake into non-phagocytic epithelial cells through the injection of effectors by the type-3-secretion system. The bacteria are ingested in tight bacterial-containing vacuoles (BCVs) that are surrounded by in situ formed infection-associated macropinosomes (IAMs). In contrast to previous reports, we have recently shown via novel 3D imaging techniques that macropinocytosis is not required for the entry of these bacterial pathogens, however the IAMs regulate their subsequent intracellular trafficking. In the case of Shigella, IAMs do not fuse with the BCV, and contact between these two compartments results in the destabilization of the BCV and membrane rupture. In the case of Salmonella two scenarios occur; IAMs either fuse with the BCV, which results in the generation of the Salmonella containing vacuole surrounded by Salmonella induced filaments (Sifs). Simple contact between IAMs and the BCV also promote vacuolar rupture in the case of Salmonella leading to cytoplasmic hyper-replication. Interestingly, BCV contacts with the surrounding compartments also dictates intravuolar bacterial growth or dormancy. We have performed ultra-structural studies, combined with dynamic imaging and proteomics of the involved compartments to identify the molecules that drive these complex interactions. This has shown a regulatoy network of Rab GTPases, the Exocyst complex, and SNAREs that is hijacked by injected bacterial effectors. I will describe how novel imaging technologies provide new insights into the intracellular niche formation of entero-invasive bacterial pathogens.

Olaf Groß
Universitätsklinikum Freiburg

## Small molecules triggering the NLRP3 inflammasome

Inflammasomes are intracellular protein complexes that control proteolytic maturation and secretion of inflammatory interleukin-1 (IL-1) family cytokines and are thus important in host defense. While some inflammasomes are activated simply by binding to pathogen-derived molecules, others, including those nucleated by NLRP3 and NLRP1, have more complex activation mechanisms that are not fully understood. We screened a library of small molecules to identify new inflammasome activators that might shed light on activation mechanisms. We find that clinical tyrosine kinase inhibitors (TKIs) including imatinib and masitinib activate the NLRP3 inflammasome. Mechanistically, these TKIs cause lysosomal swelling and damage, leading to cathepsin-mediated destabilization of myeloid cell membranes and cell lysis. This is accompanied by potassium (K+) efflux, which activates NLRP3. Both lytic cell death and NLRP3 activation but not lysosomal damage induced by TKIs are prevented by the cytoprotectant high molecular weight polyethylene glycol (PEG). Our study establishes a screening method that can be expanded for inflammasome research and immunostimulatory drug development, and provides new insight into immunological off-targets that may contribute to efficacy or adverse effects of certain TKIs.

Friedemann Weber
Justus-Liebig University Gießen

## Viral Strategies against innate immunity …

Type I interferons (IFN-alpha/beta), cytokines that belong to the so-called innate immunity , are produced by cells as a first response to virus infection ("IFN induction"). The secreted IFNs bind to their cognate receptor on neighbouring cells, and stimulate the expression of genes which have immunoregulatory or antiviral activity. The IFN system thus enables our body to rapidly sense virus infections, activate the immune response, and to form around the infection site a wall of cells that are in an antiviral state.

Intensive research of the last 20 years has enormously advanced our understanding of the IFN system. We now know that all nucleated cells express sensor proteins which are able to recognize virus-specific RNA structures and activate the signaling chain leading to IFN induction. However, it also got increasingly clear that viruses have evolved effective counterstrategies. Viral proteins, the so-called IFN antagonists, can disturb or even completely block all stages of the IFN response, e.g. IFN induction, IFN signaling, or expression or action of antiviral genes.

In our group, we investigate the interplay between IFN system and pathogenic RNA viruses, e.g. Rift Valley fever virus or SARS-coronaviruses. On one hand, we study how a cellular sensor protein can recognize an RNA structure as being viral, and on the other hand we are elucidating the astonishing variety of strategies by which viruses inhibit the IFN response. Insights obtained by such investigations can help to improve vaccines or antiviral therapies.

Melanie Brinkmann
Technische Universität Braunschweig

## … reveal novel cellular restriction factors

Human Cytomegalovirus (HCMV) infection causes only mild disease in immunocompetent individuals, but can cause severe complications in immunosuppressed individuals such as AIDS or transplant patients and is the major viral cause of birth defects. Our current understanding of immune control of HCMV infection is incomplete, and treatment options are limited. Type I interferons (IFNs) are potent cytokines which have long been considered as important effectors to rapidly counteract viral infection. Recent studies discovered a novel pathway for the innate immune defense during viral infection: the bone morphogenetic protein (BMP) signaling pathway [1]. Proteomic analyses indicated that HCMV may downregulate components of the BMP signaling machinery via the viral US18 and US20 proteins [2], suggesting deliberate manipulation of BMP-mediated signaling by HCMV due to its putative anti-viral effects. To assess the potential contribution of BMPs to control HCMV infection, we investigated whether BMPs inhibit HCMV replication and explored the effects of BMP stimulation on type I interferon (IFN) signaling and antiviral responses.

We found that pre-stimulation of primary human foreskin fibroblasts (HFF-1) with BMPs induces low levels of transcription of interferon-stimulated genes (ISGs), though without affecting HCMV replication. However, co-stimulation of HFF-1 with BMP9 and IFNβ prior to infection significantly enhanced the antiviral activity of IFNβ as compared to IFNβ pre-stimulation alone. Further experiments showed that BMP9 significantly enhances the phosphorylation of signal transducer and activator of transcription 1 (STAT1) and transcription of interferon regulatory factor 9 (IRF9) and STAT2, which are critical components of IFN-induced signaling. Moreover, we can show that HCMV US18 and US20 specifically antagonize BMP-mediated, but not IFN-mediated signaling, and their expression during HCMV infection impedes the responsiveness of HFF-1 to BMP9 stimulation, thus circumventing the potential effect of BMP9 on the antiviral host response to infection.

Taken together, our data reveal a previously underappreciated role of BMP9 as an important modulator of innate immunity and type I IFN signaling during HCMV infection.

[1] Eddowes et al., 2018, Nature Microbiology
[2] Fielding et al., 2018, eLife

Mathias Hornef
Universitätsklinikum Aachen

## Host-Microbial Interaktion in the neonate Intestine

The focus of my group's research is the interaction between commensal and pathogenic microorganisms with the mucosal immune system in the small intestine. In particular, we are interested in the situation of the neonate host that transits from the environmentally protected and sterile situation in utero to microbial and environmental exposure after birth on its way to establish a stable host-microbial homeostasis. This is reflected by many striking structural and functional differences between the neonate and adult intestinal epithelium and mucosal immune system.

Beside a better understanding the mutual interaction between commensal bacteria and the host we aim at identifying age-specific differences in the antimicrobial host response and susceptibility to infection with enteropathogenic microorganisms such as enteric Salmonella, enteropathogenic E. coli (EPEC), Listeria monocytogenes, rotavirus, and Giardia lamblia. These pathogens cause a very significant morbidity and – particularly in the infant population – also a significant mortality worldwide.

We hope that a better understanding of the feto-neonatal transition of the intestinal mucosa, the postnatal establishment of host-microbial homeostasis and the protective immune response to infection will contribute to reduce childhood mortality and reduce long-term sequelae.

Ulrich Dobrindt
Westfälische Wilhelms-Universität Münster

## Symptomatic infection vs. asymptomatic colonization – lessions from E. coli

We use urinary tract infection (UTI) and asymptomatic bacteriuria (ABU) as models to study the molecular mechanisms involved in different types of host-bacterium interaction leading to infection or colonization. Pathogens infect susceptible hosts and cause the immune system to react in an aggressive and self-damaging way, causing disease. Asymptomatic colonizers, on the other hand, successfully establish themselves in the host without provoking and harming it. During ABU, high bacterial numbers are present in the urinary bladder, without causing symptoms of UTI. It is not only interesting from the perspective of basic researchers to understand how symptomatic infections or asymptomatic colonizations arise. It is also exciting for applied research to find out how pathogens or asymptomatic colonizers interact with their hosts to search for new targets for therapy and prevention.

Bacterial adaptation strategies during long-term asymptomatic colonization include genomic decay and attenuation. In addition, the bacteria have also developed sophisticated mechanisms to actively manipulate the host in their favour. For example, asymptomatic bladder colonization of E. coli isolate 83972 can protect human carriers from superinfections by uropathogens. This strain actively modulates the host environment, down-regulating transcriptional activity, including that of pro-inflammatory genes. Uropathogenic E. coli can affect host gene expression by repressing the expression of key transcription factors in the host. At the level of chromatin dynamics, the host also responds differently to interaction with pathogens or asymptomatic colonizers.

These observations point to previously unknown strategies for bacterium-host interaction at mucosal membranes, the underlying molecular mechanisms of which have not yet been fully elucidated.

Cynthia Sharma
University of Würzburg

# A discovery in CRISPR-Cas biology in a bacterial pathogen enables multiplexed viral RNA detection

CRISPR-Cas systems are nowadays widely used for diverse biomedical and biotechnological applications, such as genome editing in eukaryotes. However, these systems are originally derived from prokaryotes, where they act as RNA-guided immune systems. An expanding diversity of such CRISPR-Cas systems in bacteria and archaea has been reported that protects them against invading nucleic acids, such as phages and plasmids. Here I will present, how we developed a new CRISPR-Cas9 based multiplexable RNA diagnostics platform, starting from a discovery that we made while studying biology of the CRISPR-Cas9 system in the food-borne pathogen Campylobacter jejuni [1].

During genome defense, CRISPR-Cas nucleases typically rely on CRISPR RNA (crRNA) guides encoded in repeat-spacer arrays associated with the system to recognize foreign genetic material. In Type II systems, another RNA component, the trans-activating crRNA (tracrRNA) hybridizes to the crRNAs to drive their processing and utilization by the Cas9 nuclease. While Cas9 typically binds and cleaves double-stranded DNA, we had uncovered that C. jejuni Cas9 (CjCas9) can also target endogenous RNAs in a crRNA-dependent manner based on a RIP-seq (co-immunoprecipitation combined with RNA-seq) approach [2]. While analyzing CjCas9-RNA complexes from additional C. jejuni strains, we recently discovered that the tracrRNA does not only bind to the crRNAs, but can also hybridize to certain cellular RNAs, such as mRNAs, leading to the formation of "non-canonical" crRNAs (ncrRNAs) [1]. While the function of these ncrRNAs in C. jejuni remains elusive, we demonstrated that these mRNA-derived fragments are capable of guiding DNA targeting by Cas9 in vitro and in vivo. Our discovery of ncrRNAs inspired the engineering of reprogrammed tracrRNAs that link the presence of any RNA-of-interest to DNA targeting with different Cas9 orthologs. This capability became the basis for a multiplexable diagnostic platform termed LEOPARD (Leveraging Engineered tracrRNAs and On-target DNAs for PArallel RNA Detection) [1]. LEOPARD can detect multiple RNAs of respiratory viruses in parallel and can distinguish different SARS-CoV-2 viral variants at single nucleotide resolution in patient samples. Overall, our study revealed crRNAs can originate from outside of CRISPR-Cas systems, and we have translated this finding into a multiplexable RNA detection platform.

References:

[1] Jiao C, Sharma S*, Dugar G*, Peeck NL, Bischler T, Wimmer F, Yu Y, Barquist L, Schoen C, Kurzai O, Sharma CM#, Beisel CL# (2021) Non-canonical crRNAs derived from host transcripts enable multiplexable RNA detection by Cas9. Science, (6545):941-948. **Equal contribution, #Corresponding authors*

[2] Dugar G, Leenay RT, Eisenbart SK, Bischler T, Aul BU, Beisel CL#, Sharma CM# (2018). CRISPR RNA-Dependent Binding and Cleavage of Endogenous RNAs by the Campylobacter jejuni Cas9. Molecular Cell 69, 893–905.

Bärbel Stecher-Letsch
Ludwig-Maximilians-Universität München

## Synthetic communities to understand functions of the intestinal microbiome

The mammalian gastrointestinal tract is a highly dynamic microbial ecosystem that controls its hosts health through its collective behavior. Microbial communities harbor hundreds of bacteria that form complex metabolic networks. Efficient metabolic interactions are essential for dietary breakdown, production of bioactive metabolites and pathogen exclusion. The lack of suitable model systems has limited our current understanding of the role of individual community members in host-microbiota interactions and resistance to infections. In my lab, we use synthetic bacterial communities that we can study in silico, in vitro and in gnotobiotic mouse models. We now understand, how individual strains within a bacterial consortium interact to influence complex microbiome functions such as colonization resistance to bacterial infections and provide necessary insight to develop strategies to steer microbial communities towards beneficial interactions promoting human health.

Volkhard Kempf
Universitätsklinikum Frankfurt

# Functional analysis of Bartonella pathogenicity factors might contribute to novel therapeutic concepts

Bacterial pathogenicity factors represent attractive targets for modulating the course of infections. Trimeric autotransporter adhesins (TAA) are important pathogenicity factors of Gram-negative bacteria and the prototypic TAA Bartonella adhesin A (BadA) from Bartonella henselae mediates bacterial adherence to endothelial cells (ECs). The molecular mechansisms by which the highly virulent bacterium B. bacilliformis mediates hemolysis (Oroya fever) are not resolved yet.

Deeper knowledge about the BadA- dependent mechanisms of host-cell adhesion of B. henselae and the mechanisms by which B. bacilliformis causes hemolysis might pave the way to novel therapeutic concepts. Experimental work revealed that BadA-host cell interactions mainly occur within the heparin-binding domains of fibronectin expressed on endothelial cells. The exact binding sites were identified by using high-throughput mass spectrometry and systematically generated B. henselae BadA mutants. BadA-domain-specific fibronectin binding was inhibited by the use of “anti-ligands”. Two B. bacilliformis proteins crucially involved in hemolysis were identified in functional assays.

Interactions between TAAs and the extracellular matrix may represent the key step for adhesion of human pathogenic bacteria to the host. Domain-specific interaction patterns of TAAs (also expressed by, e.g., multidrug resistant Acinetobacter baumannii) and the molecular understanding of the deadly Oroya-fever (caused by B. bacilliformis) opens the perspective to fight infections by anti-virulence-targeted antibiotics (“anti-ligands” and “anti-hemolysins”).

Tanja Schneider
Universität Bonn

## Bacterial targets for new antibiotics – Targeting a cell wall biosynthesis hot spot

The bacterial cell wall biosynthetic network is a very effective target for antibiotics. A most prominent target within the peptidoglycan biosynthesis pathway is the cell wall building block lipid II, which represents a particular "Achilles heel" for antibiotic attack. Lipid II is a unique non-protein target which is one of the structurally most conserved molecules in bacterial cells.

Nature invented a variety of different "lipid II binders" and their antibiotic activities can vary substantially depending on the compounds physicochemical and membrane-targeting properties, the binding site on lipid II, as well as the ability to interact with structurally similar precursors of other cell wall polymers, such as wall teichoic acid, capsule or arabinogalactan.

Besides the primary contact events, which result from initial drug-target interaction and direct binding of lipid II, sequestration of the ultimate cell wall building block can trigger multifaceted cellular events that all contribute to killing. These secondary events originate from the unique cellular role of lipid II, functioning as a structural and regulatory focus that directly and/or indirectly contributes to the organization of diverse cell wall biosynthetic processes.

The most recent addition to the portfolio of lipid II binding natural product antibiotics are teixobactin and newly identified teixobactin-like antibiotics (TLA), isolated from previously uncultured soil bacteria. Compared to all other lipid II binders investigated previously, teixobactin and TLAs appear unique in their mechanism of action and proved most refractory to resistance development.

Michael Sigal
Charité – Universitätsmedizin Berlin

## Interactions of H. pylori with gastric stem cells

H. pylori infection is linked to gastric diseases such as ulcers and cancer. We have identified that H. pylori not only colonize the stomach mucus but also invade deep into gastric glands, where they can directly interact with long-lived stem cells. I will elaborate on our recent findings that suggest that these gland-associated bacteria are key drivers of gastric inflammation and epithelial pathology.

Astrid Westendorf
Universität Duisburg-Essen

## Treg responses in the intestinal mucosa: inflammation versus regulation

The maintenance of the gut physiology depends largely on the immune regulatory network constantly rebalancing tissue homeostasis. The ability of regulatory T cells (Tregs) to home and expand in inflamed intestinal tissues and limit local damage is one of the most important features of this homeostatic network. However, the same regulatory mechanisms are also engaged in response to tissue remodeling and inflammation in the setting of a growing tumor. This in turn contributes to tumor immune evasion and resistance to immunotherapy. Therefore, targeting Tregs is an attractive strategy for cancer immunotherapy to restore and promote antitumor immune responses and potentially limit immune resistance. Given the contradictory role of Tregs in the gut, insights into specific features and the localization of tumor-associated Tregs are of high importance, when considering targeted antitumor therapies. Using a murine model of inflammation-induced colorectal cancer, we demonstrated that tumor-associated Tregs are mainly of thymic origin, inhibit antitumor immunity and are equipped with a specific set of molecules strongly associated with enhanced migratory properties. Particularly, a dense infiltration of Tregs in mouse and human colorectal cancer lesions correlated with increased expression of the orphan chemoattractant receptor GPR15. Interestingly, GPR15 expression was associated with elevated IL17 and TNF-α secretion, cytokines that are critical components of the inflammatory process involved in colorectal carcinogenesis. Gpr15 deficiency repressed Treg infiltration in colorectal cancer, which paved the way for enhanced antitumoral T-cell immunity and reduced tumorigenesis. Thus, it will be important to determine the extent to which GPR15+ Tregs contribute to colorectal cancer through the release of proinflammatory cytokines, the suppression of antitumor immune responses, or both mechanisms. In summary, our study underscore the relevance of the Treg migratory profile in supporting tumor development and progression and suggests GPR15 as a promising novel target for modifying T-cell-mediated antitumoral immunity in colorectal cancer.

Henning Gruell
Uniklinik Köln

## Antibody-Mediated Prevention and Treatment of Viral Infections

As indicated by the success against SARS-CoV-2, Ebola virus, or RSV, monoclonal neutralizing antibodies have a remarkable potential for the use in treatment and prevention of viral infections. However, pre-existing or de novo escape and viral diversity pose considerable challenges to antibody-mediated strategies. Focusing on antibodies targeting HIV-1 and SARS-CoV-2, preclinical and clinical advances in neutralizing antibodies and strategies for their effective application will be discussed.

Max Löhning
Deutsches Rheuma-Forschungszentrum Berlin, ein Leibniz-Institut

## Immunity and immunopathology in antiviral immune responses

In this talk, the role of the alarmin IL-33 and its receptor in the activation and differentiation of antiviral effector and memory T cells will be addressed. A second focus will be on the stability and plasticity of memory T helper cell subsets in immune responses to viruses.

Kilian Schober
Friedrich-Alexander-Universität Erlangen-Nürnberg

## T cell receptor-driven fate of pathogen antigen-specific T cell responses

Upon antigen encounter, individual T-cell clones are recruited into the immune response, clonally expand and differentiate into effector and memory subsets. This evolutionary process of adaptation to an antigen called clonal selection is a hallmark of the adaptive immune response. Surprisingly little is known about the functionality of antigen-specific T-cell clonotypes that are recruited into immune responses in vivo, particularly in humans. In this talk I will outline past and ongoing efforts to delineate T cell receptor-driven fate of pathogen antigen-specific T cell responses, which may guide the development of enhanced future vaccines and instruct our understanding of human T-cell biology.

Carsten Watzl
Leibniz-Institut für Arbeitsforschung an der TU Dortmund

# Inside the Mind of a Serial Killer – Cytotoxic Mechanisms of Natural Killer Cells

Natural Killer (NK) cells eliminate infected and tumor cells by releasing cytotoxic granules containing perforin and granzymes or by engaging death receptors that initiate caspase cascades. One NK cell can kill multiple target cells in a serial fashion. However, the orchestrated interplay between the cell death pathways remains poorly defined. Additionally, it is unclear which granzymes are used by NK cells to eliminate target cells. We used fluorescent localization reporters to simultaneously measure the activities of different granzymes or caspase-8 in tumor cells upon contact with human NK cells by live cell imaging.

We observed rapid GrzB-induced target cell death, originating from early established NK:target contacts. In contrast, cell death mediated by caspase-8 was slower and a result of late target cell engagements. This suggested a kinetic regulation of the two cytotoxic pathways during serial killing. We observed that NK cells switch from inducing GrzB-mediated cell death in their first killing events to a death receptor-mediated killing during subsequent tumor cell encounters. Investigating the use of the different granzymes we found that GrzB dominated most killing events of freshly isolated or activated human NK cells, while we also detected the activity of other granzymes. GrzK initiated target cell apoptosis was limited to freshly isolated NK cells. Additionally, we found individual killing events of activated NK cells where GrzA or GrzM activity was dominant.
This demonstrates that granzyme and death receptor-mediated cytotoxicity are differentially regulated during NK cell serial killing.

Christian Drosten
Charité – Universitätsmedizin Berlin

## Understanding fitness in emerging coronaviruses

The reconstruction of functional changes during virus emergence has become a major interest in evolutionary virology. The somewhat imprecise concept of “emergence” is used to capture the diverse changes that occur in zoonotic viruses while they are selected from diverse populations existing in animal reservoirs, adapt to new host environments such as in humans, and evade mounting population immunity in the case of sustained transmission. This contribution will present examples of evolution during coronavirus (CoV) emergence. During 2014/15, the MERS-CoV has increased its fitness even while still in the animal reservoir, camels, undergoing a change in innate immunity evasion that is effective in human cells. After host change, it is expected that a zoonotic virus adapts to the new host. However, for SARS-CoV the early human-to-human transmission chain involved a case of gene and function loss, providing a textbook example of neutral evolution. For SARS-CoV-2, adaptive functional changes occurred in humans even before establishment of wide antibody immunity, probably linked to the acquisition of prolonged infection stability in mucosal fluid environment. Studies of individual mutations in full SARS-CoV-2 background show that viral replicative fitness and antibody neutralization escape are separable functional traits that underwent mutual adjustments during evolution.

Stefan Pöhlmann
Deutsches Primatenzentrum, Göttingen

## Cell entry and antibody evasion of emerging SARS-CoV-2 variants

The coronavirus disease 2019 (COVID-19) pandemic claimed almost 20 million lives in 2020-2021 and continues to strain health systems and economies. Vaccines are the premier option to contain the pandemic but this approach is undermined by the constant emergence of SARS-CoV-2 variants that evade neutralizing antibodies. The research in my laboratory is focused on antibody evasion and host cell entry of emerging SARS-CoV-2 variants. I will present evidence that the Omicron subvariant BA.5, which is globally dominating at present, efficiently evades antibodies and, unlike previously circulating Omicron subvariants, robustly enters lung cells and replicates in lung tissue. Thus, BA.5 may have lost a restriction that is believed to be at least partially responsible for the attenuation of the Omicron variant. Further, data on antibody evasion of emerging variants that currently outcompete BA.5 will be presented and it will be discussed how changes in the entry strategy can promote antibody evasion.

Elke Mühlberger
Boston University

## Does a dangerous cousin make you dangerous? Assessing the pathogenic potential of newly discovered zoonotic viruses that are closely related to known human pathogens

Next generation sequencing has revealed the presence of numerous RNA viruses in animal reservoir hosts, including many closely related to known human pathogens. Despite their zoonotic potential, most of these viruses remain understudied due to not yet being cultured. While reverse genetic systems can facilitate virus rescue, this is often hindered by missing viral genome ends. A prime example is Lloviu virus (LLOV), a newly discovered filovirus circulating in bats in Europe that is closely related to the highly pathogenic Ebola virus. Using reverse genetics systems, we complemented the missing LLOV genomic ends and identified cis-acting elements required for LLOV replication that were lacking in the published sequence. We leveraged these data to generate recombinant full-length LLOV clones and rescue infectious virus. Similar to other filoviruses, recombinant LLOV (rLLOV) forms filamentous virions and induces the formation of characteristic inclusions in the cytoplasm of the infected cells, as shown by electron microscopy. Known target cells of Ebola virus, including macrophages and hepatocytes, are permissive to rLLOV infection, suggesting that humans could be potential hosts. However, inflammatory responses in human macrophages, a hallmark of Ebola virus disease, are not induced by rLLOV. Additional tropism testing identified pneumocytes as capable of robust rLLOV and Ebola virus infection. We also used rLLOV to test antivirals targeting multiple facets of the replication cycle. Rescue of uncultured viruses of pathogenic concern represents a valuable tool in our arsenal for pandemic preparedness.

Michael Schindler
Universitätsklinikum Tübingen

# Development of a novel class of broadly acting flavivirus assembly inhibitors

Flaviviruses pose a constant threat to emerge as epidemics or pandemics. As such, Dengue virus (DENV) is one of the world's fastest-growing infectious diseases. Using a FRET-based screening for capsid multimerization inhibitors, we identified a compound (C10) that shows excellent antiviral activity against Flaviviridae family members including HCV, DENV, West-Nile virus (WNV), Zika virus (ZIKV), Yellow-Fever virus (YFV) and Tick-borne encephalitis virus. In cell-based assays, C10 was non-cytotoxic at concentrations >100-fold higher than the IC50 (CC50>50µM; IC50=10-150nM). Since C10 was screened to bind and interfere with proper self-interaction of the flavivirus capsid protein, we hypothesized that the broad activity of C10 against Flaviviridae depends on structural similarities within flaviviral nucleocapsids.

In vitro experiments using recombinant HCV and DENV capsid protein showed the ability of C10 to establish a covalent interaction, inducing the formation of dimers, trimers, and higher molecular weight species. These oligomers formed at concentrations below the IC50, and the effect increased in a dose-dependent manner.

By structure-activity-relationship studies, a set of 45 C10-derivatives were designed, synthesized, and tested against HCV as well as DENV. In parallel, all compounds were evaluated in crosslinking assays. As a result, we observed a high correlation between the crosslinking ability of each compound and its activity in the cell-based assay. The activity and toxicity of C10 and two selected hit candidates (C45 and C46) were further characterized. IC50 values for different stages of HCV and DENV infection, as well as CC50 values, were determined. The calculated therapeutic index (Ti) showed that C45 is ~3-4 times better than C10 (Ti~330 HCV, ~870 DENV), being both very promising antiviral compounds. Moreover, toxicity of C10 and C45 were tested in vivo including zebrafish and mouse models, where different pharmacological parameters were evaluated. Finally, in vivo efficacy is tested in Flavivirus-infected mice. Altogether, C10 and C45 are promising molecules for further development as antiviral agents against virtually all members of the Flaviviridae family.

Matthias Dobbelstein
Universitätsmedizin Göttingen

# A new job for cancer drugs – inhibitors of nucleotide biosynthesis interfere with the replication of the coronavirus SARS-CoV-2

The COVID-19 pandemic still raises the need for improved antiviral treatment, preferably using drugs that are already in clinical use for different purposes. Besides virus-neutralizing nanobodies (Güttler et al., EMBO J 2021), we tested inhibition of nucleotide synthesis as a strategy to decrease the replication of viral RNA, thus diminishing the formation of virus progeny.

Methotrexate (MTX) has been used over decades for immunosuppression and cancer therapy. The drug inhibits dihydrofolate reductase and other enzymes required for the synthesis of nucleobases. Strikingly, the replication of SARS-CoV-2 was inhibited by MTX in therapeutically achievable concentrations, leading to up to 1000-fold reductions in virus progeny (Stegmann, Dickmanns et al., Virus Res 2021)

Inhibitors of Dihydroorotate dehydrogenase (DHODH) are in clinical use for similar purposes as MTX, and they interfere with the synthesis of pyrimidines. Our investigations revealed that DHODH inhibitors also reduce the replication of SARS-CoV-2. Moreover, these compounds strongly synergize with N4-Hydroxy-Cytidine, the active compound of the antiviral drug Molnupiravir (NHC). Mechanistically, inhibiting DHODH induces a lack of available pyrimidines, thus increasing the incorporation of NHC. The drugs also cooperated to alleviate COVID in animal models (Stegmann, Dickmanns et al., iScience 2022). We are now targeting CTP-Synthetase to further increase the efficacy of NHC and Molnupiravir. As an important caveat, however, we also found that NHC strongly enhances the occurrence of virus mutants, including escapers from neutralizing nanobodies.

Using inhibitors of nucleotide synthesis for treating SARS-CoV-2 infections still needs to be evaluated clinically, and the role of immunosuppression in disease progression awaits clarification. Within these limitations, however, our results are at least compatible with a possible use of such inhibitors in treating COVID-19. The seminar will raise the perspective of re-purposing cancer drugs to antagonize the spread of SARS-CoV-2.

Jens Bosse
Heinrich-Pette-Institut, Hamburg

## Life is fluid: How (some) viruses use liquid-liquid phase separation to induce viralreplication compartments

Recent progress has provided clear evidence that many RNA viruses form cytoplasmic biomolecular condensates mediated by liquid-liquid phase separation to form membrane-less compartments and facilitate their replication. In contrast, seemingly contradictory data exist for some DNA viruses, which replicate their DNA genomes in nuclear membrane-less replication compartments (RCs). Here, I will review the concept of liquid-liquid phase separation and elucidate the potential roles it plays in compartmentalizing viral replication. I will discuss the differences between the different virus groups in regard to condensate formation and propose a model of how liquid and homogenous early RCs convert into more heterogeneous RCs with complex properties over the course of infection.

Gisa Gerold
Stiftung Tierärztliche Hochschule Hannover

## To bend or not to bend: How RNA viruses hijack membrane protein complexes

The majority of emerging viruses posing a threat to human health are RNA viruses, which replicate their genomes in the cytoplasm. To protect the RNA genome from recognition by the innate immune system, RNA viruses evolved intricate mechanisms to re-shape cellular membranes into shielded replication compartments. Chikungunya virus, which causes debilitating arthritis, replicates in spherules at the plasma membrane. While the viral proteins involved in neck formation of spherules are well defined, host components remained elusive.

We found that the human tetraspanin CD81 is a critical replication factor for Chikungunya virus in fibroblasts and hepatocytes, two major replication sites of the virus. CD81 co-localizes with virus replication sites at the plasma membrane. The protein is dispensable for virus entry and release and is thus a bona fide replication factor. The closely related tetraspanin CD9 can partially replace the function of CD81 in virus replication. Murine CD81 similar to human CD81 supports virus genome replication, indicating that CD81 may be a cross-species host factor for Chikungunya virus. CD81 is known to stabilize membrane microdomains through cholesterol binding. By mutating the cholesterol binding site of CD81, we could show that cholesterol binding is critical for the host factor function of CD81. Finally, we show that human pathogenic viruses from the same family also rely on CD81 for replication.

Our work identifies the first human transmembrane protein hijacked by Chikungunya virus for its replication. We propose a model by which CD81 supports the formation of virus replication organelles through its cholesterol binding function. This work will spur future studies defining the minimum requirements for virus replication in human cells and may reveal targets for antiviral therapies.

Benedikt Kaufer
Freie Universität Berlin

## Herpesvirus integration: Latency, endogenous viruses and cancer

The establishment of latency allows herpesviruses to persist in the host for life. The dogma has been that all herpesviruses maintain their genome as a circular episome. However, several herpesviruses have recently been shown to integrate their genome into telomeres of latently infected cells. Among these are human herpesvirus 6A (HHV-6A), 6B (HHV-6B) and the highly oncogenic Marek's disease virus (MDV) that maintain their integrated virus genome in the absence of episomal DNA. Integration of HHV-6A/B also occurs in germ cells, resulting in individuals that harbor the integrated virus genome in every single cell of their body and transmit it to their offspring. This condition has been termed inherited chromosomally integrated HHV-6 (iciHHV-6). About 1% of the human population have this condition, while the biological and medical consequences for these individuals remain poorly understood. This presentation will highlight our recent advances in understanding herpesvirus integration and its role in pathogenesis.

Christine Goffinet
Charité – Universitätsmedizin Berlin

## Understanding obstacles to HIV-1 cure: Antipodal role of innate immunity in the context of latency reversal.

Cell-intrinsic innate immunity shapes the susceptibility and permissiveness of CD4+ T-cells to HIV-1 infection, and instructs the mounting of adaptive immune responses that can partially control HIV-1 replication. Vice versa, HIV-1 has evolved strategies to bypass or counteract these cellular defense mechanisms. Most of this knowledge has been gained in models of acute infection. The contribution of innate immunity to the establishment, maintenance and reversal of latency has not been adequately explored or is still under debate. However, cell-intrinsic immunity and its modulation by HIV-1 may exert a pivotal role in yet-to-be-developed strategies to effectively eliminate the replication-competent HIV-1 reservoir and thus functionally cure HIV-1/AIDS.

Here, by transcriptomics and functional analysis of CD4+ T-cells from aviremic patients with HIV-1 and immortalized T-cell models of HIV-1 latency, we studied the impact of latency-reverting agents (LRAs) on the cellular milieu and viral reactivation. Our work reveals quantitative and qualitative heterogeneity of individual provirus-containing clones in terms of HIV-1 reactivation. Furthermore, selected LRAs used in the context of pharmacological "shock-and-kill" approaches modulate cell-intrinsic responses by impairing both basal and IFN-induced expression of the majority of IFN-stimulated genes, resulting in facilitated HIV-1 reactivation. However, this LRA-induced reprogramming may hamper cellular processes driving immune recognition and killing of reactivating cells. Finally, identification and analysis of individual, HIV-1 transcript-positive CD4+ T-cells enabled us to define a set of genes whose expression correlated with HIV-1 RNA abundance, representing yet-to-be-characterized potential biomarkers of HIV-1 transcriptional reactivation.

# Robert Koch Lecture

Philip L. Felgner
University of California, Irvine, USA

## 150 Years of Vaccine Science

More than 1000 years ago there was a procedure called variolation that exposed scratched skin of naïve individuals to pus from the sores of smallpox infected people and there was evidence and growing acceptance that variolation conferred protection from smallpox infection. In 1796 Jenner first showed in 23 humans (including his newborn son) that cowpox virus collected from the pus of infected cows could be used instead of human smallpox pus to confer protection against smallpox infection. Fifty years after Jenner's death (circa 1880), a network of 'Vaccine Farms' began to be established that could eventually provide enough cowpox virus to vaccinate the world and 100 years later we rid the world of smallpox.

150 years ago (1880-1910) Pasteur and Koch were busy establishing germ theory and identifying specific microorganisms responsible for infectious diseases and plagues. Jenner's smallpox vaccination then provided a roadmap for developing effective vaccines against other microorganisms. The immunological mechanisms responsible for effective vaccines were unclear and Paul Ehrlich discovered serum factors he called antibodies that interfere with microorganism proliferation. The vaccine science developed by Koch, Pasteur and Ehrlich in the late 1890s was applied by a leader in modern vaccine science, Maurice Hilleman. Between 1950-70 Hilleman led development of 12 vaccines at Merck that we still give to our children today.

By any reasonable standard, in the 21st century the process for manufacturing vaccines based on technology first developed by Koch and Pasteur in the 19th century is primitive. The first step is to propagate 100s millions of doses of infectious microorganisms and do this without infecting the people involved in the manufacturing. For example, still today most of the seasonal influenza vaccine is produced by propagating

the predicted infectious seasonal influenza virus in chicken eggs in a process that takes 6 months. It takes one chicken egg to produce 1 vaccine dose against one influenza virus strain and four eggs for one dose of the quadrivalent influenza vaccine. For 200 million vaccine doses required to vaccinate the USA population every year, 800 million embryonated chicken eggs are required. One chicken produces 1 egg per day, so we need 10s million specific pathogen free chickens. Fortunately, the USA chicken egg industry produces 100 billion chicken eggs per year, so we have the capacity in the USA to produce 1 billion embryonated chicken eggs for influenza vaccine production every year.

Pharmaceutical product development and manufacturing is expensive and time consuming. A drug AZT developed in the 1960s for the treatment of HIV/AIDS received accelerated FDA approval in 1987. The drug developers worked fast and the time between the first demonstration that AZT was active against HIV in the laboratory and its approval was 25 months, the shortest period of drug development, until now.

The rapid pace of discovery, development, and FDA approval of the spectacular COVID mRNA/LNP vaccines in only ten months has taken vaccinologists, the scientific community, and the public by surprise. Within months after introduction and FDA approval of the vaccine, manufacturers Moderna and Pfizer produced and deployed billions of COVID vaccine doses. Then recently within months, they were able to deploy worldwide a modified version against newly emerging COVID variants. In addition to the spectacular efficacy of this modern vaccine technology the manufacturing of vaccines has finally moved from a 19th century technology into the 21st century.

Following introduction of the mRNA vaccine the public asked questions about safety of this new technology. The vaccine scientists often replied that the vaccine technology is not new and has been under development for 35 years. That answer prompted the next question, "Why did it take so long to make it available". These vaccines may seem to have suddenly emerged, but their advent is based on more than 35 years of scientific discovery, discourse and development, work from hundreds of scientists, numerous biotechnology companies and billions of public and private dollars invested enabling this effective response emerging from the scientific community so efficiently at this moment.

**Origins of the mRNA lipid nanoparticle vaccine**

Basic Lipid Nanoparticle (LNP) science began with the recognition of phospholipid vesicles called "liposomes" by Alec Bangham in 1965. Alec was a translationally oriented scientist who envisioned liposomes as 'magic bullets' that could deliver encapsulated drugs to the target cell. He attracted like-minded colleagues, students and post docs working to develop liposome drugs. The field attracted interest from investors in the 1980s and several liposomal drugs are approved.

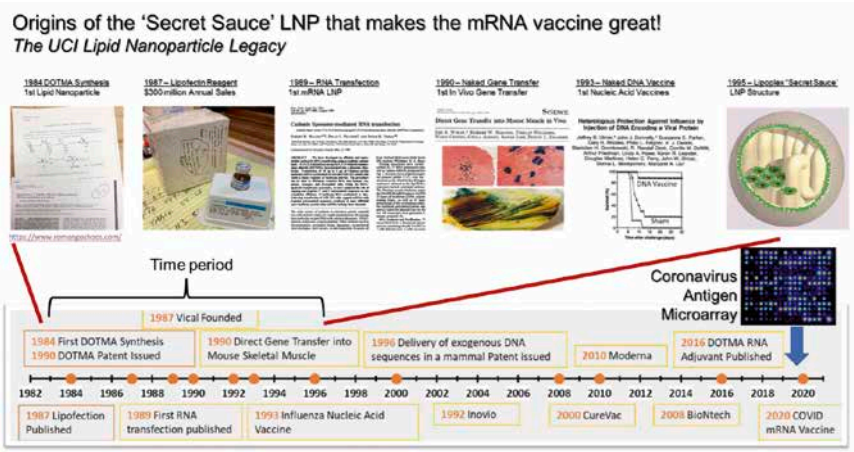
I began my post-doctoral training in biophysical sciences at the University of Virginia in Tom Thompsons laboratory. Translational science was the farthest from Tom mind. He was mystified how people could come up with uses for this technology. He had a beauti-

ful negatively stained electron micrograph of pure 70 nm diameter phospholipid vesicles next to his desk. He wanted to understand how the 4 nm thin bilayer membrane made up of lipid monomers is so remarkably stable. This membrane is necessary scaffold for life to exist so that individual cell remain discrete. He wanted to understand molecular dynamics of bilayer lipid membranes, how the molecules are organized, how the shape lipid molecules determine membrane stability, how strong is the bilayer membrane, and how mobile are the molecules. In the 1980s my basic science training converged with the nascent molecular biology, gene therapy, and immunology sciences and I made a series of discoveries that launched active fields of scientific research that did not exist before this work that is rooted in the biophysics, structure and hydrodynamic properties of emulsions, micelles and bilayer membranes.

In 1984, there were no positively charged bilayer forming lipids available in nature to prepare positively charged lipid vesicles. At the time, we understood the spatial requirements of lipid monomers necessary to form macromolecular lipid bilayer structures and stable liposomes. In collaboration with chemists at Syntex Research Institute we synthesized and characterized a series of positively charged bilayer forming lipids. These positively charged liposomes interact with the negatively charged surface of cultured cells and they enter the cells, and they have may applications for drug delivery. Cationic liposomes also interact spontaneously with negatively charged nucleic acid and self-assemble into a new highly organized stable structure called 'lipoplex.' This discovery led to a series of 6 surprising results that set the foundation for my work on nucleic acid vaccines and summarized in the timeline next page.

In the mid-1980s, the topic of gene therapy involved cloning a gene of interest into recombinant virus vectors, repurposing the virus's infection and gene delivery machinery. Another branch of scientists envisioned encapsulating nucleic acid into liposomes to chemically construct a fully synthetic virus-like particle from the ground up for gene therapy. The liposome approach had a technical challenge because the long dimension of plasmid DNA is greater than the internal diameter of a liposome. Consequently, DNA encapsulation efficiency is extremely poor. I predicted that positively charged liposomes would interact with negatively charged DNA and agglutinate to capture 100% of the DNA into aggregates. Surprisingly, my first experiments mixing DNA and cationic liposomes produced no obvious change in the appearance of the solution, no visible evidence of aggregation of the two oppositely charged macromolecules.

To prove that DNA interacts with cationic liposomes, I could show that centrifuged free DNA goes to the bottom of a sucrose density gradient, but the cationic lipid DNA complexes float. We were able to show that by simple extemporaneous mixing DNA and cationic liposomes reorganized to form a different highly organized 100 nm virus-like particle called 'lipoplex'. My team hypothesized the lipoplex would be a versatile platform to optimize by rational design with ligands and receptors to improve gene delivery properties of these synthetic virus-like particles when we got another




1. Mixing of positively charged liposomes with negatively charged nucleic acid was predicted to produce aggregation. Surprisingly, no change in turbidity was evident. Sucrose density gradient centrifugation was used to show that all of the input DNA floated to the top of the gradient along with the lipid, proving that they do interact as predicted. The lack of aggregation is a consequence of the lipid and DNA condensing to form a new stable structure called 'lipoplex' in which the fluid lipids change their packing to accommodate the nucleic acid. This synthetic self-assembled virus-like particle lacks an internal aqueous capture volume a characteristic of liposomes. Today we call these Lipid Nanoparticles.
2. We expected to add cell recognition ligands or antibodies to the lipoplex to achieve nucleic acid delivery to cells. Surprisingly, it worked right away without additional functionality. Encoded proteins were efficiently expressed using a simple transfection protocol. Simply mixing the cationic liposomes with DNA or mRNA, and adding the mixture to cultured cells resulted in expression of the encoded proteins.
3. It wasn't surprising that this convenient transfection reagent would be popular, but it is surprising how popular it became. Lipofectin and the similar reagent Lipofectamine sell $300 million/yr for Thermo Fisher.
4. I approached Syntex management to ask for approval to carry out an in vivo gene delivery study in mice using Lipofection technology. I was surprised when they declined, saying, "Gene Therapy is for the Year 2020".
5. I founded a start-up company with some faculty from UC San Diego to conduct the in vivo gene delivery study in mice and was surprised when it worked the first time. We could get reporter gene product levels in mouse muscle equivalent to that we get in cultured cells.
6. I tried to generate an immune response against HIV gp120 after intramuscular injection of DNA and mRNA encoding gp120 gene and was surprised when it worked the first time.

*The progression of science is like a fabric – threads of knowledge woven together over time to improve our understanding and to benefit humanity.*

surprise. The lipoplexes already worked to deliver functional nucleic without any additional added features. Instead, optimization was accomplished by high throughput screening of different molecules and formulations rather than by rational design. The criteria controlling transfection efficiency were related to the lipoplex poised at the transition between the bilayer and the fusogenic hexagonal-II phases which could be empirically optimized using our high throughput transfection assay. We were struck by how straightforward it was to introduce functional genes into viable cells affecting the cellular phenotype – it was stunning to realize we had this convenient and effective tool to study the function of any protein in cells. Today lipofection is routine laboratory method used around the world to introduce functional mRNA or plasmid DNA into cultured cells, enabling advances in all areas of biological science research, and biotechnology. My groundbreaking research efforts in this space also resulted in a bustling $300 million worth of lipofection reagents sold worldwide every year, providing the global community with tools leading to lifesaving treatments for infectious diseases, cancer, and autoimmune disorders.

At Syntex Research Institute in Palo Alto where this discovery research was conducted, I approached leadership with a proposal to test whether the lipoplex that works so well in cultured cells would also work in vivo. They declined saying that "Gene Therapy is for the Year 2020". This uncanny prediction resonates today since both mRNA vaccines were approved in the month of December 2020 – in the nick of time! Lacking support from Syntex, I was approached to join some UCSD faculty and started a company in San Diego called Vical. The investors agreed to support a 'back burner' gene therapy project to see whether lipofection could deliver genes in vivo. Like other things we did in this research area, it worked the first time, and my team was successful in showing mRNA and DNA delivery and expression in mice and rats.

Researchers working in gene therapy science noticed that the transgene expression measured after viral gene therapy was transient, which was eventually attributed to an immune response induced against the transgene product. While this observation was the bane of somatic gene therapy, it was a boon for nucleic acid vaccines when the intention is to induce a potent immune response against the gene product. A further advantage came out of new knowledge of two antigen processing pathways. Proteins that are transported into a cell are processed by the MHC Class 2 pathway, activate helper T cells and stimulate humoral immunity (antibodies) that can confer protection from infection. Proteins synthesized within a cell are processed by a separate MHC Class 1 pathway and stimulate CD8+ killer T cells. Thus, in addition to disease prevention, nucleic acid vaccination could potentially be used to treat preexisting chronic disease as AIDS and cancer.

My team's first demonstration of nucleic acid immunization came in a collaboration with Chiron Pharmaceuticals which was developing a recombinant protein-based vaccine against HIV. They had an HIV gp120 plasmid DNA construct that expressed high levels of secreted gp120 in transfected mammalian CHO cells. We injected animals with

this plasmid and demonstrated DNA vaccine induced antibodies against HIV gp120 Abs and cellular immune responses, and we were fortunate this breakthrough also worked the first time we tried. In partnership with Merck, we demonstrated that DNA vaccination using the sequence conserved nucleocapsid protein from influenza induces a potent cross strain protective immune response in mice. Merck's Maurice Hilleman, a leading American microbiologist who specialized in vaccinology and developed over 20 vaccines, said a major value of DNA immunization is that it can trigger immune responses against proteins without triggering one against the plasmid. Theoretically, this means the plasmid vector could repeatedly deliver DNA coding for different proteins and thus protect against a variety of diseases. "This is one of the most exciting things in modern vaccinology," said Hilleman. For the first time, we had a practical way to transduce cells and study the effect of gene on the phenotype of cells and present intracellular antigens to the immune system through the MHC Class 1 pathway.

As the next step in my research journey, two things became evident. First, we needed a way to select the best antigens to be used in vaccines, and secondly, genome sequencing methods were developing in the early 2000s that could help accomplish that. I moved from Vical to UCI to create a high throughput cloning & expression platform. In 20 years since then, my team cloned 70,000 individual genes using our high throughput PCR/recombination cloning method and express the proteins in a cell free system in vitro. We developed a protein microarray chip printing approach to print each of the individual protein from 45 bacteria, viruses, and parasites on the biodefense agents and emerging infectious disease agents list. The laboratory has printed 100,000 protein microarrays, probed 50,000 human serum specimens, discovered hundreds of immunodominant serodiagnostic vaccine antigens (Ags), and reported the results in 175 papers. This background fuels our current preclinical research on vaccines for Coxiella burnetii (Q Fever), influenza, leptospirosis, malaria and Streptococcus pneumonia.

Two years before the COVID-19 outbreak, my team constructed a protein microarray containing antigens from 6 respiratory viruses that cause common colds and flu, including a collection of common coronavirus antigens. The United States Defense Advanced Research Projects Agency, more commonly known as DARPA, supported our project to conduct serosurveillance in students residing in a large dormitory at the University of Maryland and passing respiratory infections around to each other during flu season. We were prepared when the outbreak emerged in January 2020 to add a collection of SARS-CoV-2, SARS and MERS antigens to the array. Since then, we collected and probed more than 10,000 fingerstick blood specimens from Orange County residents and followed the gradual development of naturally acquired immunity in the Orange County community. We were prepared then with data from thousands pre-vaccination specimens to observe the spectacular efficacy of the mRNA vaccines in the same community. After an aggressive vaccination campaign at the UCI Medical Center, which began in December 2020 seroprevalence jump from 13% in December to 99% vaccine induced immunity in

March 2021. After having planted the seeds and envisioning the benefits of nucleic acid immunization technology some 30 years earlier, it is thrilling to see it come to fruition now, and to participate in the analysis of its performance.

Personally, this decades-long vignette was a fascinating, and rewarding scientific journey. As an academic researcher, I encourage aspiring scientists to enter their own personal, discovery journey to benefit human society. My work supported the development of the first commercially available mRNA vaccines currently on the U.S. market, specifically the Pfizer-BioNTech and Moderna vaccines. My novel research efforts will inform the global response and management of SARS-CoV-2 and future outbreak preparation efforts. My lipofection DNA and RNA research efforts will continue informing vital treatment pathways for numerous cancers and related diseases. My vaccine antigen research efforts supported the Orange County region in the worst pandemic in a hundred years and will continue to pioneer and inform local and global clinical treatments across a multitude of disease scopes for years to come.

Drew Weissman
University of Pennsylvania

## Nucleoside-modified mRNA-LNP therapeutics.

Vaccines prevent 4-5 million deaths a year making them the principal tool of medical intervention worldwide. Nucleoside-modified mRNA was developed over 15 years ago and has become the darling of the COVID-19 pandemic with the first 2 FDA approved vaccines based on it. These vaccines show greater than 90% efficacy and outstanding safety in clinical use. The mechanism for the outstanding immune response induction are the prolonged production of antigen leading to continuous loading of germinal centers and the adjuvant effect of the LNPs, which selectively stimulate T follicular helper cells that drive germinal center responses. Vaccine against many pathogens, including HIV, HCV, HSV2, CMV, universal influenza, coronavirus variants, pancoronavirus, nipah, norovirus, malaria, TB, and many others are currently in development. Nucleoside-modified mRNA is also being developed for therapeutic protein delivery. Clinical trials with mRNA encoded monoclonal antibodies are underway and many other therapeutic or genetic deficient proteins are being developed. Finally, nucleoside-modified mRNA-LNPs are being developed and used for gene therapy. Cas9 knockout to treat transthyretin amyloidosis has shown success in phase 1 trials. We have developed the ability to target specific cells and organs, including lung, brain, heart, CD4+ cells, all T cells, and bone marrow stem cells, with LNPs allowing specific delivery of gene editing and insertion systems to treat diseases such as sickle cell anemia, Nucleoside-modified mRNA will have an enormous potential in the development of new medical therapies.

Jörg Hacker
Leopoldina –
Nationale Akademie der Wissenschaften

## Menschen, Seuchen und Mikroben – Infektionen als gesellschaftliche Herausforderung

Mitte des 20. Jahrhunderts verbreitete sich die optimistische Auffassung, dass Infektionen bedingt durch verbesserte Hygienebedingungen, den Einsatz von Antibiotika und Impfstoffen in der Zukunft keine große Rolle mehr spielen sollten. Diese Meinung hat sich nicht bestätigt. Heute, zu Beginn des 21. Jahrhunderts sind Infektionskrankheiten aktueller denn je. Der Klimawandel ändert die Ausbreitung von Vektoren bestimmter Infektionskrankheiten wie dem Westnil- oder Dengue-Virus und die Globalisierung, vor allem die wachsende Mobilität, führt zur schnelleren Ausbreitung von Erregern. Zudem gefährden zunehmend resistente Pathogene die erfolgreiche Behandlung von Infektionskrankheiten. Dies sind nur einige Faktoren, die große Herausforderungen insbesondere an die Forschung und Medizin aber auch die Gesundheitspolitik und Gesellschaft stellen. Der Vortrag stellt den Umgang mit Infektionskrankheiten in Vergangenheit und Gegenwart dar und zeigt Perspektiven auf, die sich aus den neuen Methoden der Mikrobiologie und Genomforschung für die Infektionsbekämpfung ergeben.

# Biosketches

In alphabetical order.

## Bacher, Petra

Christian-Albrechts-Universität zu Kiel

Petra Bacher holds an assistant professorship for Immunology and Immunogenetics at Kiel University (CAU), Germany. She received her PhD in Immunology from the University Jena in 2014. The Bacher lab has a strong background in human antigen-specific CD4+ T cell analyses and aims at understanding the failure of tolerance mechanisms in the human immune system during chronic inflammatory diseases. Therein, we specifically investigate the role of CD4+ T cells in immune-mediated diseases using novel direct analyses of antigen-specific T cells. Our recent work demonstrated that TCR cross-reactivity plays an important role in modulating human T-cell responses, including induction of pathogenic Th17 cells by the intestinal microbiota and modulation of the response to newly encountered antigens, which particularly affects the elderly. We also made several contributions to understanding T cell responses in infectious diseases and T cell-based diagnostics and recently analyzed the role of SARS-CoV-2 reactive T cells and preexisting immunity in COVID-19 disease and upon vaccination.

## Baldauf, Hanna-Mari

Ludwig-Maximilians-Universität München

Hanna-Mari Baldauf (née Tervo) studied Biotechnology and Biomedicine in Mannheim and Mainz. Starting with her Bachelor thesis, she became fascinated by viruses. She obtained her PhD at the former Institute of Virology in Heidelberg (now CIID) in 2010. After a short-term DAAD postdoctoral fellowship with PD Dr. Arstila at the Haartman Institute in Helsinki, she continued her postdoctoral work in the lab of Prof. Dr. Oliver T. Keppler in Heidelberg. After moving with Prof. Keppler to the Institute of Medical Virology in Frankfurt in 2012 and to the Max von Pettenkofer Institute at the LMU Munich in 2016, she started her own lab. In 2019, Hanna-Mari Baldauf received her venia legendi in "Experimental Virology".

During her postdoctoral work, she focused on several host cell factors interfering with the replication of human immunodeficiency virus (HIV). She identified the cellular deoxynucleotide triphosphohydrolase SAMHD1 as a key innate immunity factor in resting CD4 T cells and a biomarker for AML chemotherapy resistance.

The main research of her lab focuses on how innate immunity factors work in a species-specific context – identifying thereby signaling pathways / motifs responsible for their mode of action and antagonism by retroviruses. Building on the previous and current work on innate immunity factors as well as her observation that rabbits as a species display fewer blocks to HIV-1 replication than mice or rats, her lab is also making efforts to develop a fully permissive transgenic rabbit model for HIV infection.

Hanna-Mari Baldauf has received the young investigator award of the German AIDS society (DAIG) in 2009 as well as the postdoctoral fellow award of the Robert Koch foundation in 2013. She is member of the German Society for Virology (GfV), the World Lagomorph Society (WLS) and the Controlled Release Society (CRS) German Local Chapter. She is currently co-organizer of the GfV workshop "Immunobiology of viral infections" and spokesperson as well as founding board member of the junge GfV. Hanna-Mari Baldauf is also a mother of two children (3 and 8 years old).

## Bosse, Jens

Heinrich-Pette-Institut, Hamburg
Speaker

Jens Bosse studied Biotechnology and a bit of medicine at the RWTH Aachen. His fascination with virology started with reading "Outbreak." A practical at the Institute of Microbiology and Virology at the University Hospital Aachen got him intrigued by herpesviruses as some of the most fascinating and complex human viruses.

After an internship at the Divison of Virology in Cambridge UK, he did his Ph.D. at the Max von Pettenkofer Institute at LMU Munich in the Lab of Ulrich Koszinowski under the supervision of Zsolt Ruzsics where he worked on fluorescent probes to visualize virus morphogenesis. In 2011 he moved to Princeton University for a PostDoc in the lab of Lynn Enquist to illuminate how herpesviruses leave the host nucleus by developing a novel microscopy modality. In 2015 he moved back to Germany to work at the Heinrich Pette Institute, where he established live-cell microscopy technologies for correlative light- and electron microscopy of herpesvirus morphogenesis. Since May 2020, he is assistant professor for Quantitative Virology at the Center of Structural Systems Biology (CSSB) in Hamburg through the Cluster of Excellence RESIST at Hannover Medical school.

His lab develops correlative microscopy workflows, and computational analysis approaches to understand how virus particle assembly is regulated spatiotemporally at the subcellular level. Close cooperations with structural biologists and integrative data scientists aim to generate highly resolved models of viral morphogenesis.

Jens Bosse was awarded scholarships for the German Academic Scholarship Foundation during his studies and his Ph.D. He received the Robert Koch Post-doctoral Award for Virology in 2016. In 2018, his team received a Wellcome Trust Collaborative Award. His team is also part of the FOR 5200 "DEEP DV" as well as the GRK 2771 "Humans and Microbes" where they try to decipher how viruses utilize liquid-liquid phase separation for replication compartment formation.

## Brinkmann, Melanie

Technische Universität Braunschweig
Speaker

Melanie Brinkmann is Professor of Virology and Innate Immunity at the Technische Universität Braunschweig (TU-BS) and leads a research group at the Helmholtz Centre for Infection Research (HZI) in Braunschweig. She received her PhD (Dr. rer. nat.) from the Leibniz University Hannover in 2004. During her PhD at the Institute of Virology at Hannover Medical School (MHH), she focused on the oncogenic herpesvirus Kaposi's sarcoma-associated herpesvirus (KSHV) and the signaling properties of its membrane protein K15. Supported by a grant from the German Research Foundation (DFG) and the Charles King Trust (Massachusetts), she spent her postdoctoral time in the lab of Prof. Dr. Hidde L. Ploegh at the Whitehead Institute for Biomedical Research in Cambridge, USA (2006-2010). During that time, she worked on the regulation of pattern recognition receptors (PRR) which play an essential role during the innate immune response. These receptors detect viral nucleic acids in different cellular compartments and induce a signaling cascade resulting in expression of proinflammatory cytokines and type I interferons (IFN). When she received funding for her Helmholtz Young Investigator research group "Viral Immune Modulation" at the HZI in 2010, she focused on the PRR response upon herpesviral infection as well as viral immune evasion of PRR signaling. Her goal is to identify viral proteins that are directly involved in influencing the immune response and thus may (i) reveal novel insights into the host's immune response to viral infection and (ii) represent potential targets for antiviral therapies against chronic herpesvirus infections. Her work already allowed novel insights into cellular mechanisms of the innate immune response, in particular on the PRRs Toll-like receptors, the cGAS adaptor protein STING, as well as multiple interferon-stimulated gene products (ISGs) such as OASL and ZAP.

From 2012-2018 M. Brinkmann was also W1 at the MHH until she became W2 Professor of "Virus Genetics" (Institute of Genetics) at the TU-BS in 2018. In 2022, she received a "Ruf" for a W3 professorship for "Virology and Innate Immunity" at TU BS.

For her contribution to the field of virology and innate immunity, she was awarded the Robert Koch Postdoctoral Award and the Signal Transduction Society Science Award. In 2022, she was awarded with the ScienceHero Award from the Conference of Biological Faculties for her science communication during the SARS-CoV-2 pandemic.

M. Brinkmann is member of the scientific advisory board of the Helmholtz CORAERO Consortium, the German Society of Virology, the SFB900 (MHH), and the Leibniz Institute for Virology (LIV, Hamburg), and current member of the Think Tank of the president of the Helmholtz Association. She is editorial board member of EMBO Reports, EMBO Molecular Medicine, and reviewing editor for eLife. She is vice-chair of the COVID-19 Expert Council of the German Government since Dec. 2021. Since 2021 she is speaker of the DFG Research Group DEEP-DV (FOR5200) which focuses on viral and cellular transcriptional regulation in the context of DNA virus infections. She is also program committee member of the HZI and speaker of the HZI research focus “Chronic Viral Infections”.

## Brune, Wolfram

Leibniz Institute of Virology (LIV), Hamburg

Wolfram Brune is a virologist who investigates the molecular genetics and pathogenesis of cytomegalovirus, a herpesvirus with world-wide distribution.

After studying medicine at the University of Heidelberg and the University of Maryland at Baltimore, Wolfram Brune received his MD degree in 1995 for studies on papillomavirus replication at the German Cancer Research Center (advisors: Matthias Dürst and Harald zur Hausen). After an internship in pediatric neurology, he trained as a postdoc at the University of Munich with Ulrich Koszinowski and as a visiting research fellow at Princeton University with Tom Shenk. In 2002, he became an independent Junior Group Leader at the Rudolf Virchow Center for Experimental Biomedicine, University of Würzburg. In 2005, he obtained board certification (Facharzt) in microbiology, virology and infection epidemiology and his habilitation in virology. In the same year, he became Head of the Divison of Viral Infections at the Robert Koch Institute in Berlin. In 2010, he joined the Leibniz Institute of Virology (LIV) and became a professor of virology at the University of Hamburg. He was also offered professor and chair positions at the universities of Basel (2009) and Würzburg (2013), which he declined. Since 2015 he serves as the Deputy Scientific Director of the LIV. His laboratory uses forward and reverse genetics to study cytomegalovirus-host interaction with a focus on viral subversion of programmed cell death, autophagy, and innate antiviral defenses. His lab also investigates the molecular basis of the cytomegalovirus host species specificity.

Wolfram Brune received several fellowships and awards such as the Robert Koch Postdoctoral Prize (2003) and support by the Emmy Noether Program (2000-2006). He is on the editorial boards of the Journal of Virology and Apoptosis and serves as associate editor of Virology Journal and PLoS Pathogens. He has also served on the advisory board of the Society of Virology (2011-2020) and is initiator and co-organizer of the annual Mini-Herpesvirus Workshop (since 2006).

## Dobbelstein, Matthias

Universitätsmedizin Göttingen
Speaker

Trained as a medical doctor, Matthias Dobbelstein focused his scientific work on the interaction of viruses with cancer-driving mechanisms.

Matthias Dobbelstein did his M.D. thesis with Ellen Fanning at the Institute of Biochemistry at the University of Munich (LMU), Germany. He then joined the lab of Thomas Shenk at Princeton University, U.S.A., and subsequently became a group leader at the University of Marburg (Institute of Virology, director: Hans-Dieter Klenk). In 2005 he was appointed as a full professor at the University of Southern Denmark, and one year later became the Director of the Institute of Molecular Oncology at Göttingen.

Along with his coworkers, Matthias Dobbelstein is trying to understand how viruses hijack cellular pathways that are normally in place to regulate cell proliferation. Viruses provided the first access to our understanding of oncogenes and tumor suppressor genes, thereby enabling strategies to improve cancer therapy. Nowadays, this provides us with novel opportunities to counteract virus replication through interference with cellular pathways. Matthias Dobbelstein focused on the tumor suppressor p53, the Mdm2 oncoprotein, their interaction with small DNA tumor viruses and their impact on DNA replication. Currently, he is building a research line on SARS-CoV-2, evaluating the impact of nucleoside analogues and inhibitors of nucleotide synthesis on virus replication. For animal models, his group is collaborating with the Friedrich Loeffler Institute as well as the Cofoni network in Lower Saxony. In cooperation with the Max Planck Institute at Göttingen, he is also establishing camelid antibody fragments ('nanobodies') as antiviral therapeutics.

Matthias Dobbelstein is the spokesperson of three Clinician Scientist Kollegs, funded by the DFG, the Ministry of Science Lower Saxony, and the Else Kröner-Fresenius Stiftung. In 2007, he was awarded the "Lecturer of the Year" in the Molecular Medicine Master Program. He received the Postdoctoral Award for Virus Research by the Robert

Koch Foundation in 1999 and the Research Award of the Hessian Cancer Society in 1998. The postdoctoral Scholarship Infectious Biology (AIDS scholarship, DKFZ) supported him in the years 1993 – 1996, and his studies 1986 – 1992 were funded by the Studienstiftung des deutschen Volkes.

## Dobrindt, Ulrich
Institute of Hygiene, University of Münster

Ulrich Dobrindt studied biology at the University of Göttingen, Germany, and graduated at the University of Würzburg in 1999. Since 2010 he is Professor at the University of Münster, Germany. The Dobrindt group works on fundamental and applied research questions related to the geno- and phenotypic variability of bacterial pathogens, which can cause disease in humans and animals. Using Escherichia coli as a model, we want to understand (i) what distinguishes commensals from pathogens, (ii) which bacterial traits are required to cause symptomatic infection or asymptomatic colonization, and (iii) how do bacterial pathogens change during the course of an infection?

Commensal and pathogenic bacteria interact in different ways with host barriers. Successful infection or colonization of a niche requires bacterial adaptation (i) to the growth conditions encountered and (ii) in response to the cross-talk at epithelial barriers. It is our aim to identify bacterial properties and the molecular mechanisms involved in host-bacterium interaction. To correlate genomic plasticity with bacterial fitness or virulence, we investigate the (i) bacterium-host interaction at cellular barriers, (ii) genome-scale evolution and adaptation of E. coli, and (iii) E. coli factors involved in metabolic adaptation to different host niches. Another research focus is on the characterization of microbial genome plasticity and its impact on the evolution and spread of antibiotic resistances as well as on novel strategies, e.g. bacterial interference or photosensitizers, to combat bacterial infection.

Dölken, Lars
University of Würzburg

A medical doctor by trade, Lars Dölken`s research is focused on virus host interactions. He is a recognized specialist in medical microbiology, virology, and infection epidemiology.

Lars Dölken studied medicine at the Universities of Greifswald, Germany, and Otago (Dunedin) New Zealand. After obtaining his MD in 2005, he became a post-doctoral researcher at the Ludwig-Maximilians-University of Munich in the group of professor Ulrich Koszinowski. In parallel, he started his clinical training and became a registered specialist in medical microbiology, virology, and infection epidemiology in 2011. In 2008, he initiated the NGFNplus consortium researching the pathogenic role of microRNAs in herpesvirus infections in which he became a project leader from 2008-2013. After his habilitation in 2011, he moved to the University of Cambridge, UK, supported by a clinical scientist fellowship by the Medical Research Council. From 2011-2015, he also served as honorary consultant in the field of transfusion and transplantation virology for NHS blood and transplant in Cambridge. In 2015, he became full professor and director of the Institute for Virology and Immunobiology at the University of Würzburg, Germany.

His research is focused on the discovery and functional characterisation of novel mechanisms by which herpesviruses regulate their host cells and escape the immune system. A particular focus of his work is on RNA-mediated regulation and non-coding RNAs. His group employs a wide-range of systems biology methodology including single cell RNA-seq and quantitative proteomics as well as advanced virus reverse genetics systems to decipher new molecular mechanisms and the functional consequences of RNA-based regulation in different virus models.

Lars Dölken was awarded the Robert-Koch Postdoctoral Prize (2011) and a Consolidator Award of the European Research Council (2016). Since 2019, he is the spokesper-

son of the DFG Research Unit 2830 "Advanced concepts in the Cellular Immune Control of Cytomagalovirus."

**Major publications include:**

scSLAM-seq reveals core features of transcription dynamics in single cells.
Erhard F, et al. Nature. 2019 Jul;571(7765):419-423.
Improved Ribo-seq enables identification of cryptic translation events.
Erhard F, et al. Nat Methods. 2018 May;15(5):363-366.

## Drosten, Christian

Charité – Universitätsmedizin Berlin,
Institute of Virology
Speaker

Christian Drosten is a physician and virologist (board certificate in medical microbiology, virology and infection epidemiology). His research is focused on clinical, epidemiological and evolutionary aspects of RNA viruses. He has specific working experience on the diversity and animal origins of emerging coronaviruses (CoV) such as SARS- and MERS-CoV with field activities in the Middle East and Africa, working on humans as well as animals. His experimental work focuses on MERS-CoV, SARS-CoV, and SARS-CoV-2 with a particular interest on replication and evasion of interferon and antibody responses during viral adaptation. During the COVID-19 crisis his laboratory has made relevant contributions to clinical and epidemiological virology problems such as virus testing, viral load, antibodies, vaccination and antibody escape. His current interest is on functional changes during the emergence of SARS-CoV-2 variants. He is the director of the department of virology at Charité Medical Center in Berlin.

## Enninga, Jost

Institut Pasteur, Paris
Speaker

Jost Enninga heads the research unit " Dynamics of host-pathogen interactions " within the Department of Cell Biology and Infection at the Institut Pasteur in Paris. He performed his Ph.D. studies in the Laboratory of Cell Biology at the Rockefeller University, New York, under the supervision of Dr. Guenter Blobel. There, he focused on how viruses subvert host cellular trafficking processes. Afterwards, Jost moved to the Institut Pasteur in Paris in 2004 to study the early events of bacterial host cellular invasion with Dr. Philippe Sansonetti and Dr. Guy Tran Van Nhieu. Between 2008 and 2012, Jost built up his own junior team at the Institut Pasteur. He heads his own research unit since 2013, and he has been deputy director of the Cell Biology and Infection Department between 2014 and 2019. He has also been serving on many national and international committees, including INSERM, the CNRS, the DFG, and he has been on the editorial board of Cellular Microbiology and Frontiers in Immunology. His team investigates the intracellular niche formation of different bacterial pathogens, including Shigella and Salmonella. Main questions are membrane trafficking subversion by the pathogens, and how specific intracellular localizations trigger distinct immune responses. His research unit develops imaging-based technologies that allow the analysis of host-pathogen interaction dynamics with unprecedented spatiotemporal resolution. Jost's research has been acknowledged nationally and internationally, for example through the Pasteur-Vallery-Radot prize in 2018, a Schlumberger award and 2 ERC grants. He has organized workshops on advanced imaging technologies in France, Israel, South Africa and Hong Kong.

## Gerold, Gisa

Stiftung Tierärztliche Hochschule Hannover
Speaker

Gisa Gerold studied biochemistry at the Eberhard Karls University of Tübingen and performed her doctoral studies at the Max Planck Institute for Infection Biology in Berlin with Arturo Zychlinsky. She was a postdoctoral researcher at the Rockefeller University in New York, where she worked in the team of Charles M. Rice on hepatitis C virus. During her postdoctoral studies, Gisa Gerold developed her research line on high-resolution proteomics analyses of virus – host interactions. With this knowledge, she established her independent career in Hannover, Germany at the Twincore, a joint venture of Hannover Medical School and the Helmholtz Center for Infection Research. Since 2018, she is a guest professor at the Umeå University in Sweden, where she continues to have a lab outstation until today. In 2020, Gisa Gerold was appointed professor for biochemistry at the University of Veterinary Medicine Hannover and in 2022 she became head of the Institute of Biochemistry. In addition, her research group Molecular and Clinical Infection Biology is part of the Research Center for Emerging Infections and Zoonoses (RIZ).

At the heart of Gisa Gerold's research is the molecular understanding of pathogenic zoonotic virus transmission from animals to humans. With a focus on mosquito-borne viruses, arenaviruses that cause hemorrhagic fever and gastrointestinal and hepatitis viruses, the team around Gisa Gerold uses state-of-the-art proteomics approaches to discover new host factors of emerging viruses. These studies ultimately allow an assessment of host range, tissue tropism and potentially resulting pathology. Her past work identified the membrane protein complexes that coordinate virus entry of hepatitis C virus. Recently, her team delineated important protein and lipid determinants of the plasma membrane replication complex of mosquito-borne viruses. Her research aims at providing fundamental knowledge on membrane protein complexes hijacked by viruses and the biophysical requirements of virus propagation in diverse species.

Gisa Gerold is an advisory board member of the German Society for Virology (GfV) and organized GfV workshops on "Cell Biology of Viral Infection". In 2021, she initiated

the “One Health and Zoonotic Virus” workshop of the GfV and since 2020 she heads an interdisciplinary and cross-institutional consortium of computer scientists, virologists and biochemists funded by the state of Lower Saxony. She is deputy speaker of the graduate school on ‘Virus detection, pathogenesis and intervention’ (VIPER) and serves as mentor to foster careers of junior researchers.

## Goffinet, Christine

Charité – Universitätsmedizin Berlin
Speaker

After completing her doctoral and postdoctoral studies in the research group of Prof. Dr. O. Keppler, Heidelberg University, Institute for Hygiene, Christine Goffinet was recruited to an independent position as a Junior Group Leader at Ulm University Ulm, Institute of Molecular Virology. End of 2013, she accepted a joint Junior Professor position for Cell Biology of RNA Viral Infections affiliated to Hanover Medical School and a group leader position at TWINCORE. 2019 she got appointed as Associate Professor for Virology at Charité – Universitätsmedizin Berlin and BIH, Institute of Virology. Her research interests focus on the functional interplay of human-pathogenic viruses including HIV-1, alphaviruses and SARS-CoV-2, with their host cells. Specifically, her group studies the mounting and functionality of cell-intrinsic innate immune responses upon viral infection, and viral strategies of antagonism thereof. Her work has been awarded several prizes, including the AIDS Award 2017 of the German AIDS Society and the Postdoctoral Award for Virology 2012 of the Robert Koch Foundation. Her work is funded by the German Research Foundation, the Helmholtz Association, DZIF, the Hector Foundation and additional sources.

## Groß, Olaf

Universitätsklinikum Freiburg
Speaker

Olaf Groß is a German biologist investigating macrophage immunity. His undergraduate studies at the Technical University of Munich (TUM) with a focus on plant biology kindled his interest in the molecular mechanisms of stress sensing, the ensuing signaling events, and the cellular and organismal consequences. Since his doctoral thesis at the TUM University Medical Center (Klinikum rechts der Isar), he works in biomedical research. In the laboratory of Jürgen Ruland, he discovered the central role of the protein CARD9 in signal transduction upon recognition of fungal and mycobacterial pathogens by ITAM-coupled receptors. After obtaining his PhD (Dr. rer. nat.) in 2008, his interest turned to the NLRP3 inflammasome, initially studying it in the context of fungal and viral pathogens. He discovered the ability of NLRP3 to distinguish between live and dead Candida albicans, reporting this fungus as the first pathogen to engage NLRP3. As a postdoctoral research fellow in the laboratory of Jürg Tschopp in Lausanne, he reported that inflammasomes can control the unconventional release not only of caspase-1 substrates such as IL-1β, but also of other inflammatory mediators, particularly IL-1α – findings that were later explained by the discovery of gasdermin D pores mediating inflammasome-dependent release. In Lausanne, he also began working on the fascinating question of the precise molecular signals perceived by NLRP3 that are triggered by its myriad of molecularly distinct activators – a question that, irrespective of the immense clinical relevance of NLRP3 as a driver of immunopathology, is still unanswered. In 2012, Olaf Groß returned to TUM as head of an independent laboratory, equally supported by Bavarian BioSysNet junior research group funding and an ERC Starting Grant. His team discovered that NLRP3 activation – in contrast to the paradigm at the time – can occur independent of potassium efflux from macrophages. Since 2017, he is W3 professor at the Institute of Neuropathology (Director: Marco Prinz) of the University Medical Center Freiburg. Supported by multiple coordinated funding schemes of the DFG and the

EU, he and his group continue to study inflammasome biology in health and diseases, e.g., in the central nervous and cardiovascular systems. His group also develops rational approaches to target inflammasomes and metabolism for cancer therapy. Of increasing importance are the integration of macrophage signal transduction with metabolism and cell biology, and the cross-talk and cell fate decision making between distinct cell death pathways such as conventional apoptosis and inflammasome-dependent pyroptosis.

## Gruell, Henning

Uniklinik Köln
Speaker

In his work as a physician/scientist, Henning combines basic research and early-phase clinical trials to investigate the potential of neutralizing antibodies for the prevention and treatment of viral infections. While studying medicine at the University of Münster, he joined the Rockefeller University in New York as a Visiting Student. In the laboratory of Michel Nussenzweig, Henning focused on preclinical in vivo proof-of-concept studies of broadly neutralizing antibodies targeting HIV-1. After completing medical school, he started his training in Internal Medicine at the Division of Infectious Diseases at the University Hospital Cologne. Here, Henning implemented several phase I/II clinical trials evaluating HIV-1 neutralizing antibodies in first-in-human studies and investigating novel concepts for their application. To continue his clinical training in virology and microbiology, Henning subsequently joined the Institute of Virology at the University Hospital Cologne where he also works as a postdoc in the laboratory of Florian Klein. In the lab, Henning currently focuses on the characterization of antiviral neutralizing antibodies and identifying strategies for their effective clinical application. During the COVID-19 pandemic, he investigated viral antibody escape and conducted first-in-human trials of a potent SARS-CoV-2 neutralizing antibody administered by infusion or inhalation.

## Hammerschmidt, Sven

University of Greifswald

A biologist by education, Sven Hammerschmidt is focusing on the analysis of virulence strategies of Gram-positive bacteria with a special emphasis on the strategies evolved by the human specific pathogen Streptococcus pneumoniae.

Sven Hammerschmidt received his PhD in Microbiology in 1996 (Hannover, Germany) and habilitated in Microbiology in 2002. From 1996 until 2003 he was working as a post-doctoral researcher at the Helmholtz Center for Infection Research. From 2003-2007 he was a Junior Group Leader at the Research Center for Infectious Diseases at the University of Würzburg. His first appointment as Associated Professor was 2007 for Cellular Microbiology at the Max von Pettenkofer-Institute for Hygiene and Medical Microbiology at the LMU Munich. In 2008 he took over the Chair of the Department Molecular Genetics and Infection Biology and was appointed as Professor for General and Molecular Genetics at the University of Greifswald.

Sven Hammerschmidt is analyzing the host-pathogen interactions on the molecular and cellular level and research projects are especially focused on 1) the identification and molecular characterization of bacterial fitness and virulence factors of Streptococcus pneumoniae as well as their role in pathophysiological processes, 2) pneumococcal evasion strategies 3) pneumococci-induced immune responses and vaccine candidates, 4) the analyses of the biosynthesis and anchorage of pneumococcal teichoic acids, 5) the proteome signatures of pneumococci under in vivo conditions, and 6) the interplay of pathogenic bacteria with platelets.

Sven Hammerschmidt has published over 160 peer-reviewed original articles in international journals (e.g. Nature Communications, EMBO Molecular Medicine, Blood, Molecular Microbiology, FASEB J, Journal of Biological Chemistry, Journal of Infectious Disease, Scientific Reports, mBio, mSphere), several reviews, 12 book chapters, and was the editor of one book.

Sven Hammerschmidt is or was the coordinator of many collaborative projects, for example, i) Coordinator of Mecklenburg-Pomerania excellence project "KoInfekt" (2017-2021), ii) Coordinator of the BMBF Project VacoME in InfectControl 2020 "Development of vaccine against respiratory and systemic infections in humans and pigs" (2016-2020) and iii) spokesperson of the DFG-GRK1870 "Bacterial Respiratory Infections".

In 1998 Sven Hammerschmidt was awarded with the Robert-Koch postdoctoral Award and in 2007 with the Becton-Dickinson Research Award. In 2020 he was elected as member of the DFG Review Board 204 (Microbiology, Virology, Immunology) and in 2019, he was nominated Vice-President of the DGHM (German Society for Hygiene and Microbiology).

## Hauck, Christof

Universität Konstanz

Christof R. Hauck has studied biology at the University of Heidelberg. He obtained his Ph.D. from the University of Tübingen for research conducted at the Max-Planck-Institute of Biology under the direction of Thomas F. Meyer. Following postdoctoral research at the Scripps Research Institute in La Jolla, he headed a Junior Research Group at the Research Center for Infectious Diseases at the University of Würzburg. Currently, he is full professor of Cell Biology at the University of Konstanz. In 2002, he was awarded the Robert-Koch Postdoc Prize for studies at the interface between microbial pathogens and host tissues with a focus on bacterial adhesin-host receptor interaction. His research spans basic cell biology and physiology of cell adhesion molecules, including integrins and immunogobulin-related cell adhesion molecules, as well as innate immune mechanisms. His lab has been instrumental in defining epithelial exfoliation as a key defense mechanism in the urogenital tract and has spearheaded efforts to exploit this process to combat infections. Using humanized mouse models, his research team investigates the exquisite adaptations of human-restricted pathogens and the bacterial factors responsible for the molecular cross-talk with their human host. More recently, his group has become interested in understanding the evolutionary processes that shape pathogen-directed innate immune receptors in primates and the consequences of human polymorphisms in our coexistence with microbes.

## Hegazy, Ahmed

Charité – Universitätsmedizin Berlin

Ahmed N. Hegazy is a clinician-scientist and a group leader at the Department of Gastroenterology, Infectious Diseases and Rheumatology, Charité – Universitätsmedizin Berlin and German Rheumatism Research Center-Berlin. He studied medicine at Cairo University, Egypt and Hannover Medical School, Germany. He received his medical degree and did his M.D. thesis in the laboratory of Prof. Christoph Klein at Hannover Medical School, where he studied the role of CD4 T cells in antitumor immunity. He then obtained his Ph.D. in Immunology and Infection Biology from the Humboldt University of Berlin after working with Prof. Andreas Radbruch and Prof. Max Loehning at the German Rheumatism Research Center-Berlin (DRFZ). During his Ph.D., he spent two years at the laboratory of Prof. Hans Hengartner and Rolf Zinkernagel at the University of Zurich and ETH-Zurich, Switzerland. In his PhD, he investigated the T cell plasticity in viral infections. Ahmed then performed his postdoctoral training in the laboratory of Prof. Fiona Powrie at the University of Oxford. Ahmed was an EMBO postdoctoral fellow and a Marie Skłodowska-Curie Research Fellow and was awarded the Robert Koch Prize in Immunology in 2017. Since relocating to Berlin, his group is investigating the role of key cytokines in immune-epithelial interactions. Furthermore, they aim to identify new biomarkers to predict therapy response to different biological treatments in IBD.

## Hornef, Mathias

Universitätsklinikum Aachen
Speaker

Mathias Hornef studied medicine in Tübingen, Lübeck, New York and Lausanne. He worked as a research assistant and clinical fellow in Medical Microbiology at the Max von Pettenkofer Institute in Munich and the University of Freiburg and performed a postdoctoral fellowship with Staffan Normark at the Karolinska Institute in Stockholm. After an Associate Professorship for Molecular Medical Microbiology at Hannover Medical School in Hannover he became director of the Institute of Medical Microbiology at the RWTH University Hospital in Aachen. His main research interest is the interaction of pathogenic and commensal bacteria, viruses as well as parasites with the intestinal epithelium with a particular focus on the situation in the neonate host.

## Kaufer, Benedikt

Freie Universität Berlin
Speaker

Benedikt Kaufer obtained his PhD at the Department of Microbiology and Immunology at Cornell University, NY, USA in 2010. He subsequently moved to Germany and was appointed as an assistant professor (W1) at the Freie Universität Berlin in 2011. In 2016 he obtained an ERC starting grant, was awarded with a Lichtenberg Professorship in 2017 and is the director of the Institute for Virology at the Freie Universität Berlin since 2020.

Over the years, Benedikt Kaufer's laboratory investigated the molecular virology of human herpesvirus 6 (HHV-6), Marek's disease virus (MDV), varicella zoster virus (VZV) and several other herpesviruses. He developed a number of genetic systems for herpesviruses including the bacterial artificial chromosome (BAC)-based genetic system for VZV and was involved in the establishment of a mutagenesis system that facilitates the manipulation of the herpesvirus genomes in any desired way. This technology allowed the Kaufer lab to generate a plethora of recombinant herpesviruses and is commonly used in the research field.

In addition, Benedikt Kaufer demonstrate that the highly oncogenic Marek's disease virus (MDV), like human herpesvirus 6 (HHV-6), integrates its genetic material into host telomeres, a protective structure at the end of eukaryotic chromosomes. Intriguingly, MDV, HHV-6 and several other lymphotropic herpesviruses harbor telomeric repeats (TMRs) identical to host telomere sequences at either end of their linear genomes. The Kaufer lab demonstrated that these telomeric repeat sequences in MDV, HHV-6 and other viruses are essential for virus genome integration into host telomeres. They also discovered that this integration process is critical for efficient MDV-induced lymphomagenesis and reactivation from the quiescent state of infection. Beyond that, their interdisciplinary work addressed the role of the viral chemokine vIL-8 in the recruitment of the target cells of infection and the functions of the virus-encoded telomerase RNA in

MDV-induced cancer formation. Furthermore, they investigate the evolution MDV that allowed the virus to become such a deadly pathogen.

Benedikt Kaufer has received various scientific awards and honors including the Loeffler-Frosch Award (2018), the Karl-Fritzsche-Award (2018), the Koichi Yamanishi Young Investigator Award (2017), the Lichtenberg Professorship (2016), the Young Investigator Award of the German Veterinarian Association (2014) and the Robert Koch Postdoctoral Award (2014).

## Kempf, Volkhard

Universitätsklinikum Frankfurt

The research of Volkhard Kempf is focused on bacterial pathogenicity with a focus on bacterial adhesion and hypoxic reprogramming of infected host cells.

Volkhard Kempf studied medicine at the Julius Maximilians-University Würzburg and Oxford / Great Britain (final examination: 1997). He started his scientific career in Munich (Max von Pettenkofer-Institute; Director: Prof. Jürgen Heesemann) followed by a period in Tübingen (Eberhard Karls-University; Director: Prof. Ingo Autenrieth). Volkhard Kempf holds board certifications in "Medical Microbiology, Virology and Infection Epidemiology", "Hospital Infection Control", "antibiotic stewardship (expert level)" and "travel medicine". Since 2009 he is professor and head of the Institute for Medical Microbiology and Infection Control, Goethe University, Frankfurt am Main and since 2017 visiting professor (biological sciences) at the University of Leeds, Great Britain.

Volkhard Kempf´s research is focused on the interaction of bacteria with human cells, in particular on mechanisms mediating bacterial adhesion (using the human pathogenic bacterium Bartonella henselae). His research resulted in the finding that "trimeric autotransporter adhesins" are essential bacterial pathogenicity factors constructed out of conserved building blocks (driven by function). Moreover, the analysis of the infection biology of B. henselae revealed hypoxia-induced cellular reaction patterns upon infections (mediated by the hypoxia-inducible factor-1) and this demonstrates a previously unrecognized link between infection and host cell metabolism. His work is also dedicated to diagnostic medical microbiology, he invented new bacterial cultivation methods and developed serological assays for the detection of anti-Bartonella-antibodies. Since 2009 he is leading the Bartonella consiliary expert laboratory (appointed by the Robert Koch-Institute).

Volkhard Kempf has received various scientific awards (e.g., M.D. thesis award, Julius Maximilians-University Würzburg (2000), Young Investigator Award of the Deut-

sche Gesellschaft für Hygiene und Mikrobiologie (DGHM; 2005), Postdoktorandenpreis of the Robert-Koch-Foundation (2005), Felix Wankel anmial welfare award of the Felix-Wankel-Foundation (2017) and the award of State of Hesse / Germany (LOEWE: "BartoLISA"). He is co-coordinator of the DFG research Group 2251 (Acinetobacter), was president of the Medical Society Frankfurt am Main and of the section "Microbial Pathogenicity" (DGHM) and is acting president "DGHM-Stiftung". Since 2012, he is editor in chief of "Medical Microbiology and Immunology" (founded in 1886 as "Zeitschrift für Hygiene" by Robert Koch and Carl Flügge). Volkhard Kempf is rowing for the Frankfurter Rudergesellschaft Germania von 1869 and Akademischer Ruderclub Würzburg.

## Krug, Anne

Ludwig-Maximilians-Universität München
Speaker

Research in my group is focused on elucidating developmental and functional diversity of dendritic cell subpopulations, especially in the context of viral infection and vaccination. We want to find out how differentiation of plasmacytoid versus conventional dendritic cells from precursors is influenced by immune stimulation, for example in the context of viral infection. In our current work we investigate the influence of viral infection and vaccination on the functionality of dendritic cell subsets and on replenishment of DC subset from precursors. We are using mouse models as well as samples from human patients and vaccinees to address these questions.

## Lämmermann, Tim

Max Planck Institute of Immunobiology
and Epigenetics, Freiburg

Our research investigates the mechanisms that shape single cell and population dynamics of immune cells in the complexity of inflamed and infected tissues. By using a broad range of microscopy and imaging techniques, we explore the strategies that immune cells have evolved to move individually or in concert with other cells in order to achieve together an optimal immune response.

With a special focus on cells of the innate immune response, our research seeks to find a conceptual framework how these immune cells integrate the plethora of signals arising in inflammatory environments and how they coordinate their dynamic behavior with other tissue-resident cells in the context of inflammatory and infectious diseases. Moreover, we aim at understanding the remarkable plasticity that many immune cells have evolved on a single cell and population level, allowing them to adapt their dynamic responses to rapidly changing inflammatory environments. An improved understanding of the underlying mechanisms promises the tailoring of therapeutic strategies to modulate immune responses.

In our research, we are particularly interested how single cell and population processes impact (a) the sensing, detection and elimination of damaged tissues and dead cell material, (b) the specific migration patterns and strategic positioning of immune cell subsets to initiate immune responses, (c) the communication within one or between several immune cell populations for the optimal coordination of innate immune responses during wounding, inflammation, infection and anaphylaxis.

In our lab, we have several projects that fall under the following categories and address the before mentioned biological aspects: (1) Principles of self-organization in swarming neutrophils, (2) Organization principles of tissue-resident immune cell networks, and (3) Novel tools for understanding immune cell movement in tissues.

We follow an interdisciplinary research approach at the interface of immunology, tissue physiology and basic cell biology to gain the deepest possible knowledge on innate immune cell dynamics under physiologically relevant conditions. Visualization of innate immune responses in inflamed and infected tissues by intravital microscopy is our starting point for understanding leukocyte dynamics in their physiological tissue environment. However, these intravital studies often do not allow dissecting the cell biology and molecular details underlying dynamic processes. To overcome these limitations, we complement our studies with mouse genetics and innovative, often custom-built in vitro models that closely mimic the physiological situation.

## Löhning, Max

Deutsches Rheuma-Forschungszentrum Berlin,
ein Leibniz-Institut
Speaker

We fuse basic research on cellular differentiation processes and developmental programs in T lymphocytes with research on chondrocytes and other cell populations in cartilage, bone tissue, and joints, particularly in the context of osteoarthritis development and pathology.

In immunology, we mainly focus on the following topics:

- Alarmins, especially IL-33, and their signaling in antiviral T cell responses.
- T cell differentiation programs and their stability versus plasticity in immune responses to viruses.
- Memory formation and maintenance in T cells with particular focus on quantitative expression memory for cytokines and key transcription factors.

In osteoarthritis research, we mainly study the following subjects:

- Mechanical signals in angiogenesis in bone, mineralization, and osteogenesis.
- Chondrocyte metabolism and cartilage homeostasis particularly in the context of innate immunity signals.

## Maier, Lisa

Interfaculty Institute of Microbiology
and Infection Medicine Tübingen (IMIT)

Lisa Maier, a biochemist by training, studies microbiome-host interactions, in particular the impact of drugs on the microbiome and the consequences for the host.

Lisa Maier received her doctorate in 2014 from the Institute of Microbiology at ETH Zurich, Switzerland. During her PhD in the lab of Prof. Wolf-Dietrich Hardt, she investigated the role of the microbiome in Salmonella infections. As part of the interdisciplinary postdoctoral programme at EMBL in Heidelberg, she worked with Dr. Athanasios Typas and Dr. Kiran Patil on high-throughput methods for the systematic investigation of drug-microbiome interactions. In 2019, supported by the Emmy Noether Program of the German Research Foundation, she established her independent research group at the Interfaculty Institute of Microbiology and Infection Medicine Tübingen. In 2022, she accepted a call to a full professorship at the Medical Faculty of the University of Tübingen. Her lab is integrated into the Cluster of Excellence "Controlling Microbes to Fight Infections" and the interdisciplinary M3 (Malignoma-Metabolome-Microbiome) Research Institute in Tübingen.

Lisa Maier's long-term research goal is to gain a detailed and comprehensive understanding of the interactions of antibiotic and non-antibiotic drugs with the microbes of the human gut microbiome. To this end, her lab systematically maps both, the effects of drugs on growth of gut microbes, but also drug degradation and bioaccumulation by gut microbes. This includes both the identification of the responsible bacterial strains and species, the elucidation of the underlying molecular mechanisms, and the investigation of the consequences for the host, such as drug efficacy, toxicity, or side effects.

At the same time, she is capitalizing on the findings from her work to develop strategies that enable targeted modulation of the gut microbiome.

## Mayer, Christian Thomas

National Cancer Institute, National Institutes of Health, Bethesda, USA

Christian T. Mayer, a biochemist in training, has a long-standing interest in how immune responses are regulated and how self-tolerance is maintained.

Christian T. Mayer received his Ph.D. in immunology in 2012 from the Hannover Medical School, Germany, working with Tim Sparwasser. He then joined Michel Nussenzweig's laboratory as a postdoctoral fellow at The Rockefeller University, New York. In 2020 he became a Stadtman Investigator at the National Institutes of Health in Bethesda, USA.

Christian T. Mayer initially focused his work on cellular immunity and studied how regulatory T cells and dendritic cells regulate immunity and self-tolerance. He then became interested in B lymphocytes, the other arm of adaptive immunity, and developed novel apoptosis indicator mice that allowed him to study the molecular mechanisms driving B cell death during ongoing immune responses. His current research program focuses on unraveling the regulatory networks controlling cell death and determining their roles in immune cell development, differentiation and function. These studies serve to increase our fundamental understanding of immune responses and of how defects in immune regulation might contribute to diseases. The long-term goals are to identify new therapeutic strategies to (1) inhibit unwanted immune responses, (2) enhance immune responses against tumors and pathogens.

Christian T. Mayer received several early career awards including a PhD award by the Helmholtz Center for Infection research in 2013, the Fritz-and-Ursula-Melchers Postdoctoral Prize 2014 (German Society for Immunology), the Early Career Research Prize in Vaccinology 2018 (International Union of Immunological Societies), the Young Investigator Award 2018 (Scripps Center for HIV/AIDS Vaccine Immunology & Immunogen Discovery, La Jolla, USA) and the postdoctoral award in immunology 2018 by the Robert Koch Foundation.

## Muenchhoff, Maximilian

Ludwig-Maximilians-Universität München

**Personal background**

Maximilian Muenchhoff works as scientist and medical doctor at the Max von Pettenkofer Institute of Ludwig-Maximilians-Universität (LMU) in Munich, Germany. He attended Medical School in Germany, Spain and Australia. During his clinical training he did rotations in infectious diseases at the LMU Hospital, Munich, and at the Red Cross Childrens Hospital, Cape Town, South Africa. He spent postdoctoral periods at the University of Oxford with Professor Philip Goulder, at the HIV Pathogenesis Programme in Durban, South Africa, and at the Ragon Institute in Boston, USA. In 2016 he joined the Max von Pettenkofer Institute to complete his medical training in microbiology, virology and epidemiology, and to establish his own research group. His research work is devoted to understanding host-virus interactions of the two pandemic pathogens, HIV-1 and SARS-CoV-2.

**Areas of investigation**

Immunopathogenesis of viral infections

The pathogenesis of viral infections is determined by highly variable degrees of inflammation and adaptive immunity resulting in a diverse spectrum of disease phenotypes. We are studying interactions of HIV and SARS-CoV-2 with the human immune system to delineate signatures that are associated with disease severity.

We are particularly interested in humoral and cellular immune responses in specific patient populations including immunocompromised hosts.

**HIV persistence in viral reservoirs**

Despite major efforts in treating HIV infection, current antiretroviral therapy is not curative. The major obstacle to an HIV cure is persistence of HIV in pools of latently infected cells. Potential therapeutic interventions aiming at an HIV cure depend on precise meth-

ods to quantify and study these viral reservoirs. Within a DZIF project we are establishing a platform to characterize the HIV reservoir using a portfolio of different methods such as digital droplet PCR and optimized viral outgrowth assays. These assays are made available to other scientist and are used in our own studies of HIV infection in primary cell models.

**SARS-CoV-2 evolution and immune evasion**

Since its recent emergence SARS-CoV-2 is constantly evolving in adaptation to the human host. We are particularly interested in intra host evolution of SARS-CoV-2 in adaption to selection pressure mediated by the adaptive immune system in patients with prolonged infection. We aim to characterise immune escape mutations that are selected under antibody and T-cell selection pressure.

## Mühlberger, Elke

Boston University
Speaker

Dr. Elke Mühlberger is an expert in the field of highly pathogenic viruses, especially the Marburg and Ebola viruses, which belong to the filovirus family. Her research interests include filovirus replication strategies, host response mechanisms, and developing antiviral countermeasures. The Mühlberger lab uses reverse genetics technology to study filoviral replication and transcription strategies and to assess the pathogenic potential of unexplored filoviruses. The Mühlberger lab pursues a highly collaborative approach to analyzing host response mechanisms to viral infections and identifying actionable targets for antiviral countermeasures. This includes the use of human primary cells and organoids for viral infection studies to recapitulate human disease. Dr. Mühlberger has partnered with experts in stem cell research, omics studies, EM imaging, RNA biology, and innate immunity to gain deeper insights into the determinants that lead to severe disease in humans.

Dr. Mühlberger received her PhD in Virology from the Philipps University Marburg, Germany in 1993 and continued to work on filoviruses as an independent PI and group leader in Marburg. In 2008, she joined Boston University School of Medicine where she is a Professor at the Department of Microbiology and the Director of the Integrated Science Services Core at the National Emerging Infectious Diseases Laboratories.

## Münch, Jan

Ulm University Medical Center

Jan Münch (7.11.1972) studied Biology at the Friedrich-Alexander University (FAU) Erlangen-Nürnberg (1993-1998). He then joined the group of Frank Kirchhoff at the Institute of Virology (Bernhard Fleckenstein) at the FAU and received his PhD (Dr. rer. nat) in 2002. He then moved as PostDoc to the Institute of Virology in Ulm (Thomas Mertens) where he was appointed as Junior Professor in 2004. In 2010 he accepted a call on a full professorship (W3) at the newly established Institute of Molecular Virology (Frank Kirchhoff) at Ulm University and became Co-Director in 2017. Jan Münch is Deputy Speaker of the Core Facility Functional Peptidomics, Co-Founder of Ulm Competence Center for Peptide Pharmaceuticals (UPEP) and also serves as chair of the promotion committee Human Biology at the Medical Faculty. He received several honors and awards from academia and industry. His research focuses on the innate immune defense against viral infections and the development of broadly-active antiviral agents against enveloped viral pathogens, from basic research to translational approaches.

## Papenfort, Kai

Friedrich-Schiller-Universität Jena

Kai Papenfort is professor and chair of General Microbiology at the Friedrich Schiller University Jena. He studies the role of signaling molecules and regulatory RNAs in microorganisms and their significance for infectious diseases such as cholera.

Kai Papenfort studied Biology at the Phillips University of Marburg focusing on microbiology, genetics and molecular biology. After his studies, he went to the Max Planck Institute for Infection Biology in Berlin and obtained his PhD in 2010 from the Humboldt University in Berlin. During this time, he also worked as a Marie-Curie-fellow at the Institute of Food Research in Norwich (UK). Next, he moved to the Institute for Molecular Infection Biology at the University of Würzburg to continue his work on bacterial RNA regulators in the laboratory of Jörg Vogel. In 2012, he received a Long-Term post-doctoral fellowship from the Human Frontiers Science Program to study microbial communication processes with Bonnie Bassler and Ned Wingreen at Princeton University (NJ, USA). In 2015, he was appointed Professor of Microbiology at the Ludwig-Maximilians-University of Munich, Germany and in 2019 he became chair of General Microbiology at the Friedrich Schiller University Jena.

Kai Papenfort discovered that bacterial small RNAs (sRNAs), much alike their mammalian microRNA counterparts, can control the expression of multiple target mRNAs simultaneously. Further, he was able to show that sRNAs play an important role in the regulation of virulence-related genes and discovered a novel mechanism of post-transcriptional gene control in bacteria. In his work on bacterial communication, his lab made the exciting discovery of a novel interkingdom communication signal, called DPO (3,5-dimethylpyrazin-2-ol), operating between microbes and eukaryotic cells and controlling collective behaviors such as virulence and biofilm formation in the major human pathogen, Vibrio cholerae.

Kai Papenfort has received various scientific awards and honors for his research including, a PhD fellowship of the Boehringer Ingelheim Fonds (2007-2009), the VAAM dissertation award (2011), the Postdoc Award for Microbiology of the Robert Koch Foundation (2014), the Research Award of the Engelhorn Foundation (2017), the Career Development Award of the Human Frontiers Science Program (2017), a Starting Grant from the European Research Council (2017), as well as the Scholar Award from the Vallee Foundation (2019). He was also an elected member of the Young Scholars' Programme at the Bavarian Academy of Sciences and Humanities (2016-2019).

## Pfänder, Stephanie

Ruhr-University Bochum

Our research focuses on understanding the complex interplay between emerging viruses and their host. As obligate intracellular pathogens, viruses are utterly dependent on host cellular processes and exploring virus–host interactions is therefore the key to understand mechanisms regulating the viral replicative cycle and any pathological outcomes associated with infection. Specifically, during the last years my group has focused on emerging coronavirus infections, a topic which lately received significant attention due to the emergence of SARS-CoV-2. In order to understand the complex processes of viral infection, we are utilizing complex cellular models including human airway epithelial cells and three-dimensional human lung organoid systems. Using these models, we study viral immune control with an emphasis on interferon stimulated genes and cellular restriction mechanisms. Next to elucidating molecular mechanisms of virus control, we are also working on topics ranging from public health issues, encompassing studies on virus stability and disinfection to studies with clinical importance, including analysis of humoral immune responses in patients and vaccinated individuals and exploration of antiviral therapeutic approaches.

## Pöhlmann, Stefan

Deutsches Primatenzentrum, Göttingen
Speaker

I am interested in host cell interactions and pathogenesis of emerging viruses and primate herpesviruses. One focus of my lab is on viral entry into cells. We could show that SARS-CoV-2 uses the ACE2 protein as receptor and the cellular protease TMPRSS2 for spike protein activation. We would like to understand how choice of TMPRSS2 versus alternative activators impacts viral tropism, transmissibility and pathogenesis. Further, we are interested in the development of TMPRSS2 inhibitors for antiviral therapy and we are examining how SARS-CoV-2 evades neutralizing antibodies. In the context of herpesviruses, we seek to understand why Herpes B Virus (macacine alphaherpesvirus 1) but not closely related herpesviruses causes severe disease in humans. For this, we are generating recombinant viruses and new cell systems. Finally, we develop diagnostics for virus infections of non-human primates.

Salzer, Ulrich
Universitätsklinikum Freiburg

My primary research focus is on human primary immunodeficiencies, especially primary antibody deficiencies clinically summarized under the diagnosis common variable immunodeficiencies (CVIDs). Among the CVIDs, I contributed to the discoveries of ICOS, BAFFR and CD21 deficiencies and first described the occurrence of genetic variants in TACI in CVID patients.

More recent research projects involve bone marrow microenviroment studies in patients with CVIDs and bone marrow failure syndromes like GATA2 deficiency.

Since 2013, I am engaged as the head of the diagnostic laboratory at the department of Rheumatology. This gave me the opportunity to broaden my research profile to translational projects including studies of patients after B-cell depleting therapies, immune monitoring in HIV and heart failure and development of a dried blood spot test for monitoring of IgG levels at home.

**Selected recent publications (past five years)**

1. Salzer U, Müller A, Zhou Q, Nieters A, Grundmann S, Wehr C. Susceptibility to infections and adaptive immunity in adults with heart failure. ESC Heart Fail. 2022;9:1195-1205.
2. Mueller MC, Kern WV, Usadel S, Pauly MC, Cathomen T, Salzer U. Assessing the differential impact of chronic CMV and treated HIV infection on CD8+ T-cell differentiation in a matched cohort study: is CMV the key? AIDS Res Ther. 2021;18:37.
3. Kläsener K, Jellusova J, Andrieux G, Salzer U, Böhler C, Steiner SN, Albinus JB, Cavallari M, Süß B, Voll RE, Boerries M, Wollscheid B, Reth M. CD20 as a gatekeeper of the resting state of human B cells. Proc Natl Acad Sci U S A. 2021;118:e2021342118.

4. Troilo A, Wehr C, Janowska I, Venhoff N, Thiel J, Rawluk J, Frede N, Staniek J, Lorenzetti R, Schleyer MT, Herget GW, Konstantinidis L, Erlacher M, Proietti M, Camacho-Ordonez N, Voll RE, Grimbacher B, Warnatz K, Salzer U, Rizzi M. Nonpermissive bone marrow environment impairs early B-cell development in common variable immunodeficiency. Blood. 2020; 135:1452-1457.
5. Wehr C, Grotius K, Casadei S, Bleckmann D, Bode SFN, Frye BC, Seidl M, Gulsuner S, King MC, Percival MB, Pritchard CC, Walsh T, Wu D, Keel S, Salzer U. A novel disease-causing synonymous exonic mutation in GATA2 affecting RNA splicing. Blood. 2018;132:1211-1215.

## Schaufler, Katharina

Christian-Albrecht University Kiel and
University Medical Center Schleswig-Holstein &
University of Greifswald

Early in her career, Katharina Schaufler chose to integrate her interest in (veterinary) medical microbiology with questions related to the important problem of the worldwide spread of infectious agents resistant to antimicrobials. The multidrug-resistant (MDR) Gram-negative and -positive bacterial pathogens from the families of Enterobacteriaceae and Enterococcaceae that she works on belong to the microorganisms mentioned on the "WHO Pathogen Priority List 2017", which defines the pathogens that pose the world's biggest challenge in terms of antimicrobial resistance and prospective therapeutic developments.

MDR pathogens are not only present in clinical settings but also occur in locations with low antibiotic selection pressure and are – due to their pandemic occurrence in humans, animals (livestock, companion animals) and in the environment (water, soil, wildlife) – an important part of the "One Health" concept.

For her two doctoral theses at Free University of Berlin under the supervision of Prof. Lothar H. Wieler (2012-2016), she investigated mechanisms that contribute to the success of these pathogens in the One Health context. Her results indicate that plasmids render MDR Enterobacteriaceae not only more resistant to antibiotics, but also more virulent and thus highly successful. In addition, Katharina Schaufler showed that the common dogma that plasmid carriage always comes with fitness costs for the bacterial host does not apply. Her subsequent research investigations at Harvard Medical School in Boston (2017-2018), where she deepened her expertise in functional genomics and experimental evolution on vancomycin-resistant enterococci, supported her conclusions, which are as follows: a) MDR pathogens combine resistance with a high level of fitness and virulence, b) resistance plasmids contribute to the pathogen's success substantially and beyond resistance, and c) MDR pathogens develop individual, sophisticated strategies to successfully emerge.

In addition to several previous third-party projects, in fall 2020, she received funding of over two million Euros to lead a BMBF junior research group focusing on the establishment of alternative therapeutic strategies (i.e., anti-virulence approaches) to treat bacterial infections caused by MDR pathogens. Katharina Schaufler was then appointed Professor for "Medical Microbiology, Virology and Infection Epidemiology" at Kiel University in May 2021.

In summary, her main research focuses on the in-depth analysis and drivers of MDR pathogen emergence and evolution at the One Health interface, and on the establishment of innovative therapeutic strategies to fight the spread of these pathogens more effectively.

## Schindler, Michael

University Hospital Tübingen
Speaker

Michael Schindler studied Biology in Ulm and graduated in 2003. After that, he joined the group of Prof. Frank Kirchhoff for a PhD in Molecular Virology. During his PhD, he performed research on lentiviral pathogenesis with a focus on viral immune evasion mechanisms and the so-called accessory proteins.

Schindler defended his PhD in May 2006 with distinction, already starting his own group one and a half year later by the end of 2007 at the Heinrich-Pette-Institute for Experimental Virology in Hamburg, now the Leibniz Institute of Virology. There, he diversified his research, now also working on Hepatitis C virus and more specifically elaborating on methods to visualize and demonstrate protein interactions and infections in living cells.

After another group leader position at the Helmholtz Center in Munich from 2011 to 2014 Schindler was appointed as W2 Professor for Molecular Virology of Human Infectious Diseases to the University Hospital Tübingen, where he was finally tenured in 2018 as full professor (W3) for Medical Virology. In the meantime, Schindler is working with his group on immune evasion mechanisms of highly diverse viruses. Doing so, the team is able to devise novel strategies for immunotherapeutic intervention against viruses and deepens our understanding on the functioning of the human immune system. Furthermore, Schindler and his team are following up on novel innovative strategies to identify compounds with broad antiviral activity to develop the next generation of antiviral drugs.

## Schlitzer, Andreas

Medical Sciences Institute, University of Bonn

The aim of our research is to understand the complexity and function of mononuclear phagocytes during health and disease. A major focus lies on the developmental processes leading to the functional specialization of mononuclear phagocytes within complex tissue microenvironments. To investigate such processes the Schlitzer Lab utilizes high-dimensional technologies, such as single-cell OMICS, flow cytometry, and ultra high-plex imaging approaches. Taken together we decipher the interconnected molecular cues leading to the efficient and adequate immune response of mononuclear phagocytes during health and disease.

## Schneider, Tanja

Universität Bonn
Speaker

Tanja Schneider studied biology in Bonn, Germany, specialized in microbiology and obtained her PhD in 2004. Following a postdoctoral training at the Institute of Medical Microbiology, Immunology and Parasitology, University Hospital Bonn, she joined industry from 2011 until 2012. During that time she worked at the Peptide and Cell Discovery Department at Novozymes A/S, Denmark in the course of a Marie Curie Actions fellowship. Returning to Germany, she became a junior research group leader within the German Center for Infection Research (DZIF). In 2014 she completed her habilitation in Medical-Pharmaceutical Microbiology and was appointed Professor of Pharmaceutical Microbiology at the University of Bonn in 2015.

Her research area is on the bacterial cell envelope as a target for (new) antibiotics. Main focus is the elucidation of molecular mechanisms of action and resistance and the identification & characterization of novel antibacterial targets. Pivotal to these efforts is the discovery of novel antibiotics and to understand the biology beyond an antibiotic target as well as its integration into the cellular network of bacteria in support of drug discovery.

She was awarded with the Robert-Koch Post-doctoral Award for Microbiology in 2010 and the Wolfgang-Stille-Prize in 2016 for her contributions in antiinfective research.

## Schober, Kilian

Friedrich-Alexander-Universität
Erlangen-Nürnberg
Speaker

Kilian Schober is a physician scientist with a focus on antigen-specific T cell immunity. His research aims at understanding how T cell fate is determined through the T cell receptor (TCR), and to translate gained insights in technology development for immunotherapy. At the same time, he is a certified medical doctor of Microbiology, Virology and Epidemiology of Infections.

After a research stay for his medical thesis in the lab of Stephan Kissler at the Harvard Medical School, Boston/USA, Dr. Schober joined the lab of Dirk Busch at the Technical University of Munich as a postdoctoral fellow. On the one hand, he there investigated the spatiotemporal fate of antigen-specific T cells with TCRs of different binding strengths (avidities) in the context of chronic infection or cancer (e.g. Schober et al. Nature Immunology 2020). On the other hand, he developed an approach – the so-called "orthotopic TCR replacement" – that uses CRISPR/Cas9 to exchange antigen-specific TCRs at the endogenous gene locus in primary human T cells (e.g. Schober et al. Nature Biomedical Engineering 2019). With the advent of the COVID-19 pandemic and novel technological opportunities to study T cell biology on the single-cell level, Dr. Schober developed a technique ("reverse phenotyping") to decipher the TCR and phenotype of SARS-CoV-2 antigen-specific T cells in a high-throughput and unbiased manner (Fischer et al. Nature Communications 2021).

With support by the German Federal Ministry of Education and Research (BMBF), Dr. Schober in 2021 set up his own group in the field of infectious diseases research at the University Hospital of Erlangen. The Schober lab aims at understanding and engineering human T cell immunity in the context of infectious diseases, autoimmunity and cancer – with a focus on antigen-specific T cell populations and their TCRs. Recipients of SARS-CoV-2 or yellow fever virus vaccines thereby serve as one important model system to study central aspects of human T cell biology for the first time directly ex vivo.

## Schwartz, Christian

Mikrobiologisches Institut –
Klinische Mikrobiologie, Immunologie und
Hygiene, Universitätsklinikum Erlangen

Being a biologist by education, Christian Schwartz has focused his research interest on the interplay of innate and adaptive immunity during type 2 inflammation.

Christian Schwartz obtained his diploma in biology at the Eberhard-Karls-University Tübingen for which he had already joined Prof. David Vöhringer's group at the LMU Munich for his diploma thesis. After he relocated together with the research group to the University Hospital Erlangen in 2011, he finished his PhD thesis on the role of basophils during helminth infections in 2015. Afterwards, he became an EMBO long-term fellow in the group of Prof. Padraic Fallon at the Trinity College Dublin. Since 2019, he is an independent research group leader at the Institute for Clinical Microbiology at the University Hospital Erlangen.

The research interest of Christian Schwartz has always been the interaction between innate and adaptive immunity in the context of inflammation. In particular, regulation of T cells in type 2 immunity, which is found during parasitic infections and allergies, as well as adipose tissue homeostasis and tissue repair, is at the core of his research. He could show that basophils, despite their low abundance, were an important effector cell type accelerating Th2 polarization during challenge infections with gastrointestinal helminths. Furthermore, he reported that ILC2, basophils, mast cells and dendritic cells play important roles during different models of atopic dermatitis. During his postdoc period, he became interested in the cell-specific function of PD-L1 in the regulation of T cells. He could show that – in contrast to the well-studied role as an inhibitory molecule – PD-L1 on ILC2 can additionally act as an activator of Th2 cells in the context of helminth infection. During obesity, expression of PD-L1 on dendritic cells is required to maintain an anti-inflammatory immune environment and limit adipose tissue inflammation. Of clinical relevance, he also showed that PD-L1 is expressed in adipose tissue of patients with obesity. Currently, his group investigates the mechanisms behind the

impaired tissue repair during obesity, how bacterial products influence the crosstalk of antigen-presenting cells and T cells, and how macrophages limit T cell-driven lung inflammation.

Christian Schwartz has received the Robert Koch Postdoctoral Prize in Immunology for his work on the immune defense against helminths in 2018. Since 2022, he is the leader of a BMBF-funded Junior Research Group in Infection Research. He is member of the German Societies of Immunology (DGfI), Hygiene and Microbiology (DGHM) and Parasitology (DGP).

## Sharma, Cynthia

Julius-Maximilians-University of Würzburg
Speaker

Cynthia Sharma is a full professor and Chair of Molecular Infection Biology II at the Institute of Molecular Infection Biology (IMIB) at the Julius-Maximilians University of Würzburg. She studied Biology (Diplom) at the Heinrich-Heine-University of Düsseldorf and obtained her doctoral degree in bacterial RNA biology at the Max Planck Institute for Infection Biology in Berlin (MPIIB) in 2009. After short postdoctoral work at the MPIIB and the National Institutes of Health in Bethesda (USA), she joined the Research Center for Infectious Diseases (ZINF) in Würzburg as an independent junior group leader in 2010. Since 2017 she is heading the newly established Department of Molecular Infection Biology II at the IMIB in Würzburg. Moreover, she is spokesperson of the ZINF in Würzburg since 2018.

Research in her lab focuses on mechanisms of gene regulation in stress response and virulence control of bacterial pathogens as well as biology and mechanisms of prokaryotic RNA-based CRISPR-Cas immune systems. In particular, her group focuses on RNA-based regulation via small regulatory RNAs (sRNAs) and RNA-binding proteins as well as small proteins. Her lab has been employing diverse deep sequencing technologies to annotate bacterial transcriptome and translatomes as well as to identify and study sRNAs and RNA-protein complexes.

Her research was recognized with several awards, such as the 2011 Postdoc award in Microbiology of the Robert Koch Foundation and the German Association for Hygiene and Microbiology (DGHM), the 2013 DGHM Young investigator award, the Heinz-Maier-Leibnitz-Award of the Deutsche Forschungsgemeinschaft (DFG) in 2015. She was a member of the Young Academy of the Bavarian Academy of Sciences from 2012-2017. In 2021, she was selected as one of the winners in the category Life Sciences of the “Falling Walls Science Breakthroughs of the Year” and received the 2022 Pettenkofer-Award for

her work on a new CRISPR-based RNA diagnostic method. Recently, she was awarded a 2021 ERC Consolidator Grant to explore RNA binding proteins in bacteria.

More information:
www.imib-wuerzburg.de/research/sharma/group-leader
Twitter: @SharmaLab1

Stanifer, Megan
Univeristy of Florida

I have been working in the field of molecular virology and cell biology for the past 19 years. Throughout my career, I have handled a diverse array of BSL2 and BLS3 pathogens including both enveloped and non-enveloped viruses with DNA and RNA genomes. During this time, I have investigated pathogen-host interactions from viral entry to antiviral innate immune modulation. I have developed state-of-the art microscopy approaches to investigate virus entry, fusion, and immune stimulation as well as methods to evaluate host responses to viral infection at the single cell level. My PhD at Brown University in the lab of Dr. Walter Atwood focused on understanding the host/pathogen interactions of the human polyomavirus JCV. My first post-doctoral position was in the laboratory of Dr. Sean Whelan at Harvard Medical School, where I studied the entry and fusion of vesicular stomatitis virus. In 2012, I moved to Germany and joined the Department of Infectious Diseases at the University Hospital Heidelberg. There, I started a second post-doc and quickly developed a senior role in the lab where I not only ran my own project and obtained my own research funding but also trained and supervised master and PhD students. During this second post-doctoral period, I established the use of human, mouse and feline intestinal organoids to evaluate host/pathogen interactions. Using these models, I have been able to introduce methods to genetically modify these organoids and evaluate how they respond to a variety of pathogens. By establishing this system, I was uniquely poised to address questions of SARS-CoV-2 pathogenesis in primary cell cultures. Throughout the SARS-CoV-2 pandemic I have been actively working with many national and international collaborators to use our human intestinal organoids to address questions of viral pathogenesis and the development of anti-viral strategies. Additionally, due to my years working with organoids I became a member of a national collaborative network within Germany (ORGANOSTRAT) to use organoids to evaluate SARS-CoV-2 infection.

I have recently relocated to the University of Florida, College of Medicine as an Assistant Professor. My lab will continue to focus on virus infection of epithelial surfaces but will also expand our primary cell portfolio to examine host/pathogen interactions in respiratory and cervical epithelial cells.

## Stecher-Letsch, Bärbel

Ludwig-Maximilians-Universität München
Speaker

Bärbel Stecher has received her PhD in Microbiology from the ETH Zürich (Switzerland) in 2005. After a postdoctoral period at ETH, she moved as an EMBO Short-Term Fellow to McMaster University (Canada) in 2007, working on the implementation of gnotobiotic mouse models in preclinical infection research. In 2011, she was appointed assistant professor (associate professor in 2016) at the Department of Medicine, LMU Munich (Germany) where she is leading a research team consisting of scientists and students in biology and human medicine. She has made important contributions to our current understanding of the interplay between enteric pathogens and the intestinal microbiota and published 81 papers in peer-reviewed journals. In 2019, she received an ERC consolidator grant. Bärbel Stecher is deputy speaker of the DFG-funded research consortium CRC1371 and coordinator of the TTU Gastrointestinal Infections of the German Center for Infection Research (DZIF).

Her research focus is the function of the microbial ecosystem in the gut in preventing enteric infections. Her group combines classical microbiology (i.e. isolation of gut bacteria) with gnotobiotic mouse models and next generation microbiome analysis to understand microbial interactions with human enteric pathogens.

## Stenger, Steffen

Department of Medicine Microbiology
and Hygiene, University Hospital Ulm

Steffen Stenger received his medical degree from the University of Erlangen in 1994 and finished his specialty training in Medical Microbiology and Infection control in 2001. Since 2006 he is Medical Director of the Department of Medical Microbiology and Hygiene at the University Hospital in Ulm.

In 1993 he received his Dr. med for a thesis on "Cytokine interactions in experimental infection with Leishamnia major" from the University of Erlangen. From 1995-1997 he spent a postdoctoral fellowship in the laboratory of Prof. Robert Modlin at the Institute of Molecular Biology and Department of Dermatology at the University of California, Los Angeles. The fellowship was funded by the "AIDS-Stipendium" rewarded by the German Ministry of Research and Technology. Dr. Stenger returned to Germany in 1997 and established his own research team at the Insititute of Medicial Microbiology and Immunology at the University of Erlangen. In 2006 he was appointed as full Professor and Head of the Department of Medical Microbiology and Hygiene at the University of Ulm.

Since the begin of his potdoctoral training the reasearch focus is on understanding the immune response against tuberculosis in humans. The major scientific findings include the characterization of granulysin as an endogenous antimicrobial peptide, the identification a vitamin D-dependent pathway of macrophage activation and the functional characterization of lipid specific T-lymphocytes. His current interest is the development of new vaccine approaches against tuberculosis and the identification and functional characterization of antimycobacterial peptides. This research was rewarded by several scientific prizes including the Robert Koch Award for postdoctoral research and the major prize of the German Society of Hygiene and Microbiology. He served as member of the advisory board of the Community for Tuberculosis Vaccine Development (CTVD) at the Bill and Melinda Gates Foundation and as vice president of the German

Society for Hygiene and Microbiology. At present he is member of the steering committee of the Tuberculosis Vaccine Initiative (TBVI) and board member of the Paul Ehrlich Society for Chemotherapy.

## Tokoyoda, Koji

Immunology, Faculty of Medicine,
Tottori University, Japan

Koji Tokoyoda studied biology and immunology at Tokyo University of Science and Osaka University with Dr. Masato Kubo and Dr. Hiroshi Yamamoto and obtained a Ph.D. in 2002 by showing molecular mechanisms on Th cell differentiation. He then investigated B cell lymphopoiesis and maintenance in the bone marrow and found cellular niches for memory plasma cells as well as B cell precursors. Through the finding, studying immune memory attracted him. In 2005, he moved to the Deutsches Rheuma-Forschungszentrum (DRFZ) Berlin and worked as a junior scientist with Dr. Andreas Radbruch. He showed cellular niches for memory T helper cells in vivo and the finding has been honored with the Robert-Koch Postdoctoral Prize (immunology) in 2010. After working at Chiba University (Japan) for 3 years, he returned to the DRFZ Berlin and worked as a group leader in 2012-2020, studying molecular mechanisms on memory T helper cell differentiation and a Salmonella's strategy to deplete memory plasma cells. Since 2020, he has worked at Tottori University, Japan as a full professor and continues to study immune memory.

## Watzl, Carsten

Leibniz-Institut für Arbeitsforschung
an der TU Dortmund
Speaker

Carsten Watzl studied Biology in Heidelberg. During his PhD work at the German Cancer Research Centre in Heidelberg in the department of Peter Krammer, he focused on the signal transduction of the CD95 (APO-1/Fas) receptor (back then he was called Carsten Scaffidi). As a postdoctoral researcher he investigated the regulation of Natural Killer cells in the lab of Eric Long at the NIH (NIAID). In 2002 he returned to Germany as head of a junior research group at the Institute for Immunology at the University of Heidelberg where he continued to work on the topic of Natural Killer cells. In 2011, he became the scientific director of the Department of Immunology at the Leibniz Research Centre for Working Environment and Human Factors (IfADo) at TU Dortmund.

During his work on the signal transduction of the CD95 (APO-1/Fas) receptor he described two pathways of CD95-mediated apoptosis in a publication that has been cited more than 2500 times. He then focussed on the signal transduction in Natural Killer cells, where he investigated the interplay of activating and inhibitory surface receptors on the molecular level. His recent work has focussed on the molecular mechanisms of NK cell cytotoxicity, starting with the adhesion to target cells, the differential use of granule and death receptor-mediated cytotoxicity as well as the regulation of detachment from target cells and how these processes contribute to the serial killing activity of NK cells. As an additional research focus he is investigating the regulation of NK cells by neuroendocrine factors.

Carsten Watzl has received several scientific awards such as the Walther and Christine Ritzenhain Award for the best PhD thesis on cancer research in Heidelberg (1999), the Roche Molecular Biochemicals Award 2000 from the German society for Cell Biology for the best PhD thesis in cell biology in Germany (2000), the National Institutes of Health – Fellows Award for Research Excellence (FARE) (2001), the Robert Koch Postdoctoral Award from the Robert Koch Society (2003), the BioFuture Award from the Ger-

man Ministry of Education and Research (2004), and the Klaus-Georg und Sigrid Hengstberger-Award from the University Heidelberg (2006). He was the founder and speaker of the Study Group on Natural Killer Cells of the German Society for Immunology (2008 – 2015), an elected member of the DFG Review Board in Immunology (2016-2020) and since 2013 he is the elected Secretary General of the German Society for Immunology.

## Weber, Friedemann

Justus-Liebig University Gießen
Speaker

Friedemann Weber, biologist by training, received his Ph.D. in 1997 from the University of Freiburg, Germany. He was an EMBO Postdoctoral Fellow at the Institute of Virology in Glasgow, UK, and a research group leader in the Department of Virology, Freiburg, Germany. From 2010 to 2015, he held a professorship at the Institute for Virology in Marburg and is now the Director of the Institute of Virology (Veterinary Medicine) in Giessen, Germany. He is interested in the interferon responses to highly pathogenic RNA viruses (especially bunyaviruses, coronaviruses, and influenza viruses). The particular focus is on interferon-inducing viral structures (e.g. 5'-triphosphorylated RNA), their intracellular sensors (e.g. RIG-I), and the viral escape strategies (e.g. sequestration or proteasomal destruction of cellular key factors of innate immunity).

Besides being an EMBO fellow since 1998, he is a recently elected member of the Deutsche Forschungsgemeinschaft (DFG) review board ("Fachkollegium") for Microbiology, Virology and Immunology (2020-2023), in the selection panel of EVAg (European Virus Archive goes Global), and in the Editorial Boards of Journal of Biological Chemistry, Journal of General Virology, and Journal of Interferon and Cytokine Research. He had served many times as Guest Editor for PLoS Pathogens, was a member of the grants evaluation panel of the European Research Council (ERC) (2009-2015), and one of the 3 Directors of the International Society for Interferon and Cytokine Research (ISICR, now ICIS; 2010-2014). He had received several awards, including the prestigious Löffler-Frosch Award of the "Gesellschaft für Virologie" (GfV), the Heine-Medin Medal of the European Society for Clinical Virology (ESCV), the Milstein Young Investigator Award of the ISICR, and the Robert-Koch-Postdoktorandenpreis" (Robert-Koch-Stiftung, Cologne).

## Westendorf, Astrid

Universität Duisburg-Essen
Speaker

Astrid Westendorf does interdisciplinary basic research with a strong focus on immunology, infectious diseases and oncology. She received a PhD in Biology from the Helmholtz Centre for Infection Research/Technical University of Braunschweig in 2004 prior to joining the National Institute for Medical Research in London as a postdoctoral fellow. Later she became a junior professor for Mucosal Immunology at the University of Duisburg-Essen, and in 2012, she was appointed as full professor for Immunology and Infection at the University of Duisburg-Essen.

The defense against infections is one of the central tasks of the immune system. Due to its exposed position, the intestine in particular offers an enormous entry site for pathogens and has therefore developed a very complex immune system. On the one hand, pathogens must be rendered harmless by an inflammatory reaction, and on the other hand, bacteria of the natural intestinal microbiota and food must be tolerated. Astrid Westendorf studies this sensitive balance between protective immune response and tolerance. She is mainly concerned with the questions of how tolerance is induced in the intestine, how gastrointestinal infections impair intestinal homeostasis, and how intestinal inflammation promotes the development of colon cancer. Astrid Westendorf identified that pathogen-specific T cells play a decisive role in the development of autoimmunity in the mucosa and represent an important target structure for therapeutic approaches. In this context, she established nanoparticle-based approaches for the modulation of mucosal immune response. Furthermore, she is interested in how infection influences the course of autoimmunity and cancer in the intestine.

Since 2014, Astrid Westendorf acts as spokesperson of the DFG funded Research Training Group “Immune Response in Infectious Diseases” and is an elected member of the Scientific Committee of the University of Duisburg-Essen. Furthermore, she serves on the Executive Board of the German Society of Microbiome and Mucosal Immunology.

In 2017, she was honored with the "Medicine and Science Award" by the city of Essen for her contribution to interdisciplinary research in the field of nanomedicine, and in 2018, she was Visiting Professor at the University of Cordoba, Argentina. Since April 2022, Astrid Westendorf is Vice-Rector for Research and Early-Career Researchers at the University of Duisburg-Essen.

## Westermann, Alexander

University of Würzburg

Alexander Westermann's research combines RNA biology with microbiota research. As a trained molecular biologist graduating at the University of Heidelberg, Alexander Westermann worked as a visiting scholar at the University of California, Berkeley, USA. As a fellow of the Bavarian Elite Network, he joined the group of Prof. Jörg Vogel at the University of Würzburg for his PhD. He then moved to the University of California, Davis, as a visiting scientist in the group of Prof. Andreas Bäumler and, as an EMBO short-term fellow, joined the lab of Prof. David Holden at the Imperial College London. In 2018 Westermann was recruited as a junior professor to the Institute for Molecular Infection Biology where he leads the group "Dual RNA-seq". In parallel, he leads the "Host-pathogen-microbiota interactions" group at the Helmholtz Institute for RNA-based Infection Research.

The human intestinal tract offers an attractive environment for both beneficial and pathogenic bacteria. The beneficial bacteria of our microbiota feast on undigested foods and provide numerous health benefits. Enteric pathogens see this environment as an entry point for infection. Both groups influence each other, creating a tripartite interaction with us, the host. Understanding the regulatory processes that decide on the outcome of these encounters represents an emerging research area to combat infectious diseases. While the field has focused on protein-mediated processes, the Westermann lab investigates the role of RNA-centric mechanisms in controlling microbial interactions in the gut. The biological insights gained from his studies will improve our knowledge of the functions of regulatory RNA molecules and their protein partners in predominant members of the human intestinal microbiota. This will lay the groundwork needed to exploit microbiota RNA biology for diagnostics and therapy against enteric infections and microbial disorders in our gut.

The PhD thesis of Alexander Westermann was awarded with the dissertation prize of the German Center for Infection Research (DZIF) of the German Society for Hygiene and Microbiology (DGHM). In 2016, he received the Post-Doc award in Microbiology of the Robert-Koch-Foundation. In 2021, he was awarded an ERC Starting grant for his proposed project termed “GUT-CHECK”.

# Post-doctoral Awards

Since 1998, the Robert Koch Foundation, in cooperation with the German Societies for Hygiene and Microbiology, Immunology and Virology, has awarded three postdoctoral prizes annually for outstanding work to young scientists in these fields.

| Year | First name | Last name | City | Postdoctoral Award |
|---|---|---|---|---|
| 1998 | Sven | Hammerschmidt | Greifswald | Postdoctoral Award for Hygiene and Microbiology |
| 1998 | Harald | Kropshofer | Basel | Postdoctoral Award for Immunology |
| 1998 | Friedemann | Weber | Gießen | Postdoctoral Award for Virology |
| 1999 | Wolf-Dietrich | Hardt | Zürich | Postdoctoral Award for Hygiene and Microbiology |
| 1999 | Hans-Willi | Mittrücker | Hamburg | Postdoctoral Award for Immunology |
| 1999 | Matthias | Dobbelstein | Göttingen | Postdoctoral Award for Virology |
| 2000 | Christoph | Dehio | Basel | Postdoctoral Award for Hygiene and Microbiology |
| 2000 | Steffen | Stenger | Ulm | Postdoctoral Award for Immunology |
| 2000 | Henri-Jacques | Delecluse | Heidelberg | Postdoctoral Award for Virology |
| 2001 | Gundula | Schmidt | Freiburg | Postdoctoral Award for Hygiene and Microbiology |
| 2001 | Ludger | Klein | München | Postdoctoral Award for Immunology |
| 2001 | Elke | Mühlberger | Boston | Postdoctoral Award for Virology |
| 2002 | Christof | Hauck | Konstanz | Postdoctoral Award for Hygiene and Microbiology |
| 2002 | Dirk | Busch | München | Postdoctoral Award for Immunology |
| 2002 | Stefan | Pöhlmann | Göttingen | Postdoctoral Award for Virology |
| 2003 | Steffen | Backert | Erlangen | Postdoctoral Award for Hygiene and Microbiology |
| 2003 | Carsten | Watzl | Dortmund | Postdoctoral Award for Immunology |
| 2003 | Wolfram | Brune | Hamburg | Postdoctoral Award for Virology |
| 2004 | Mathias | Hornef | Aachen | Postdoctoral Award for Hygiene and Microbiology |
| 2004 | Max | Löhning | Berlin | Postdoctoral Award for Immunology |
| 2004 | Christian | Drosten | Berlin | Postdoctoral Award for Virology |
| 2005 | Volkhard | Kempf | Frankfurt/Main | Postdoctoral Award for Hygiene and Microbiology |
| 2005 | Anne | Krug | München | Postdoctoral Award for Immunology |
| 2005 | Thomas | Pietschmann | Hannover | Postdoctoral Award for Virology |
| 2006 | Jost | Enninga | Paris | Postdoctoral Award for Hygiene and Microbiology |
| 2006 | Ulrich | Salzer | Freiburg | Postdoctoral Award for Immunology |
| 2006 | Jan | Münch | Ulm | Postdoctoral Award for Virology |

| Year | First name | Last name | City | Postdoctoral Award |
|---|---|---|---|---|
| 2007 | Ulrich | Dobrindt | Münster | Postdoctoral Award for Hygiene and Microbiology |
| 2007 | Florian | Winau | Boston | Postdoctoral Award for Immunology |
| 2007 | Melanie | Brinkmann | Braunschweig | Postdoctoral Award for Virology |
| 2008 | Christoph U. | Schoen | Würzburg | Postdoctoral Award for Hygiene and Microbiology |
| 2008 | Astrid M. | Westendorf | Essen | Postdoctoral Award for Immunology |
| 2008 | Bärbel | Kaufmann | West Lafayette | Postdoctoral Award for Virology |
| 2009 | Bärbel | Stecher-Letsch | München | Postdoctoral Award for Hygiene and Microbiology |
| 2009 | Olaf | Groß | Freiburg | Postdoctoral Award for Immunology |
| 2009 | Michaela | Gack | Chicago | Postdoctoral Award for Virology |
| 2010 | Tanja | Schneider | Bonn | Postdoctoral Award for Hygiene and Microbiology |
| 2010 | Koji | Tokoyoda | Tottori | Postdoctoral Award for Immunology |
| 2010 | Michael | Schindler | Tübingen | Postdoctoral Award for Virology |
| 2011 | Cynthia | Sharma | Würzburg | Postdoctoral Award for Hygiene and Microbiology |
| 2011 | Cecilia | Chassin | Paris | Postdoctoral Award for Immunology |
| 2011 | Lars | Dölken | Würzburg | Postdoctoral Award for Virology |
| 2012 | Sandra | Schwarz | Tübingen | Postdoctoral Award for Hygiene and Microbiology |
| 2012 | Christina | Zielinski | München | Postdoctoral Award for Immunology |
| 2012 | Christine | Goffinet | Berlin | Postdoctoral Award for Virology |
| 2013 | Luisa Fernandez | Jimenez-Soto | München | Postdoctoral Award for Hygiene and Microbiology |
| 2013 | Stefanie | Eyerich | München | Postdoctoral Award for Immunology |
| 2013 | Hanna-Mari | Baldauf | München | Postdoctoral Award for Virology |
| 2014 | Kai | Papenfort | Jena | Postdoctoral Award for Hygiene and Microbiology |
| 2014 | Tim | Lämmermann | Freiburg | Postdoctoral Award for Immunology |
| 2014 | Benedikt | Kaufer | Berlin | Postdoctoral Award for Virology |
| 2015 | Nishith | Gupta | Berlin | Postdoctoral Award for Hygiene and Microbiology |
| 2015 | Christoph | Klose | Berlin | Postdoctoral Award for Immunology |
| 2015 | Maike | Dittmann | NewYork | Postdoctoral Award for Virology |

| Year | First name | Last name | City | Postdoctoral Award |
|---|---|---|---|---|
| 2016 | Alexander J. | Westermann | Würzburg | Postdoctoral Award for Hygiene and Microbiology |
| 2016 | Andreas | Schlitzer | Bonn | Postdoctoral Award for Immunology |
| 2016 | Jens | Bosse | Hamburg | Postdoctoral Award for Virology |
| 2017 | Médéric | Diard | Basel | Postdoctoral Award for Hygiene and Microbiology |
| 2017 | Ahmed | Hegazy | Berlin | Postdoctoral Award for Immunology |
| 2017 | Gisa | Gerold | Hannover | Postdoctoral Award for Virology |
| 2018 | Lisa | Maier | Tübingen | Postdoctoral Award for Hygiene and Microbiology |
| 2018 | Christian Thomas | Mayer | Bethesda | Postdoctoral Award for Immunology |
| 2018 | Christian | Schwartz | Erlangen | Postdoctoral Award for Immunology |
| 2018 | Maximilian | Münchhoff | München | Postdoctoral Award for Virology |
| 2019 | Anna | Müller | Bonn | Postdoctoral Award for Hygiene and Microbiology |
| 2019 | Daniel | Utzschneider | Melbourne | Postdoctoral Award for Immunology |
| 2019 | Henning | Grüll | Köln | Postdoctoral Award for Virology |
| 2020 | Michael | Sigal | Berlin | Postdoctoral Award for Hygiene and Microbiology |
| 2020 | Petra | Bacher | Kiel | Postdoctoral Award for Immunology |
| 2020 | Stephanie | Pfänder | Bochum | Postdoctoral Award for Virology |
| 2021 | Katharina | Schaufler | Greifswald | Postdoctoral Award for Hygiene and Microbiology |
| 2021 | Kilian | Schober | Erlangen | Postdoctoral Award for Immunology |
| 2021 | Megan | Stanifer | Tampa | Postdoctoral Award for Virology |


**Organizers**

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Mathias Hornef, Universitätsklinikum Aachen
Max Löhning, DRFZ Berlin
Andreas Radbruch, DRFZ Berlin
Katrin Moser, DRFZ Berlin
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# Alumni Symposium 2022
# Robert Koch Postdoc Prize Awardees

## 25 years of Postdoc prizes

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