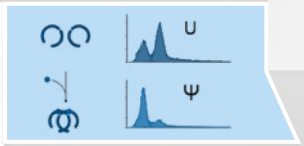
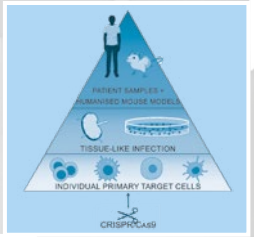
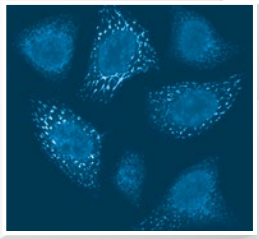
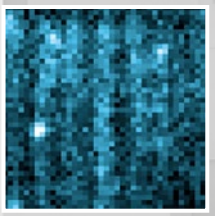
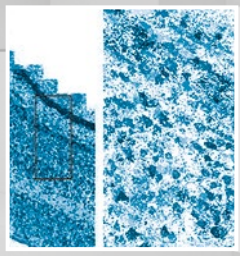
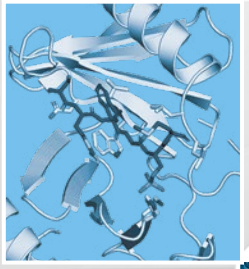
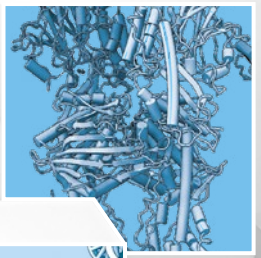
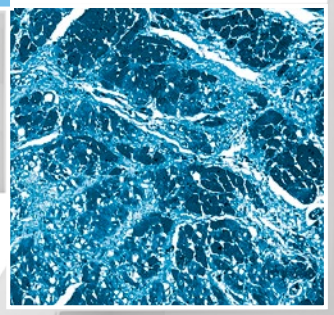
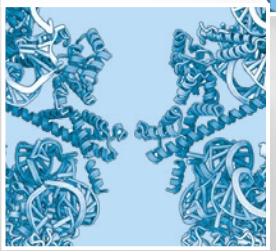
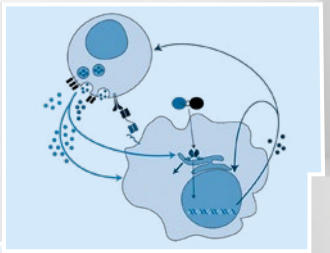



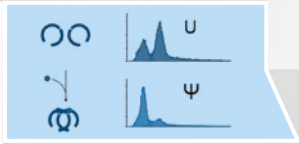

GENE CENTER MUNICH

Report 2020-2025

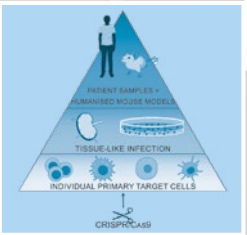
LMU LUDWIG-MAXIMILIANS-UNIVERSITÄT MÜNCHEN

Some of the Latest Research Highlights at the Gene Center

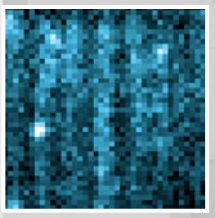
Pseudouridine RNA avoids immune detection through impaired endolysosomal processing and TLR engagement
Cell. 2025, S0092-8674(25)00619-1.



Rapid, efficient and activation-neutral gene editing of polyclonal primary human resting CD4+ T cells allows complex functional analyses
Nat Methods. 2022, 19(1):81-89.

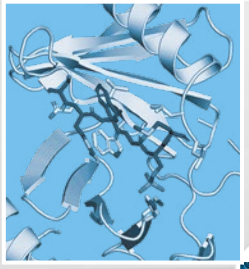


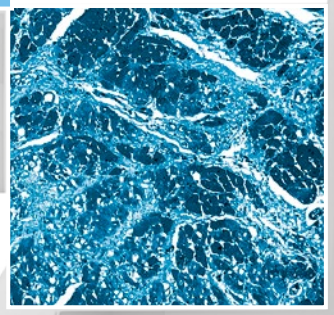
DELE1 tracks perturbed protein import and processing in human mitochondria.
Nat Commun. 2022, 13(1):1853.



LINE-1 ribonucleoprotein condensates bind DNA to enable nuclear entry during mitosis
Sci Adv. 2025, 11(18):eadt9318.

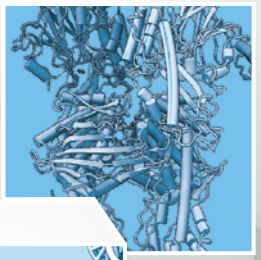
PLK1-Mediated Phosphorylation Cascade Activates Mis18 Complex to Ensure Centromere Inheritance.
Science. 2024, 285 (6713): 1098-1104.





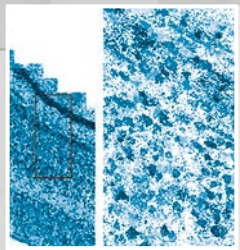
Systemic deletion of DMD exon 51 rescues clinically severe Duchenne muscular dystrophy in a pig model lacking DMD exon 52
PNAS 2023, 120(29):e2301250120.

Cryo-EM structure of the Mre11-Rad50-Nbs1 complex reveals the molecular mechanism of scaffolding functions
Mol Cell. 2023, 83(2):167-185.e9.

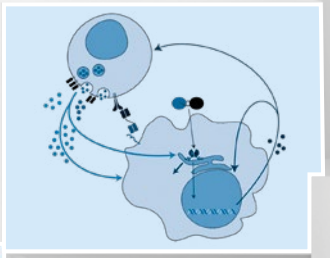


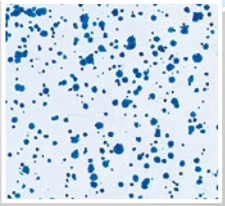
UFMylation orchestrates spatiotemporal coordination of RQC at the ER.
Sci Adv. 2025, 11(18):eadv0435.

TACCO unifies annotation transfer and decomposition of cell identities for single-cell and spatial omics
Nat Biotechnol.
2023, 41(10):1465-1473.



STING activation improves T-cell-engaging immunotherapy for acute myeloid leukemia.
Blood. 2025, 145(19):2149-2160.





RNF14-dependent atypical ubiquitylation promotes translation-coupled resolution of RNA-protein crosslinks
Mol Cell. 2023, 83(23):4290-4303.e9.

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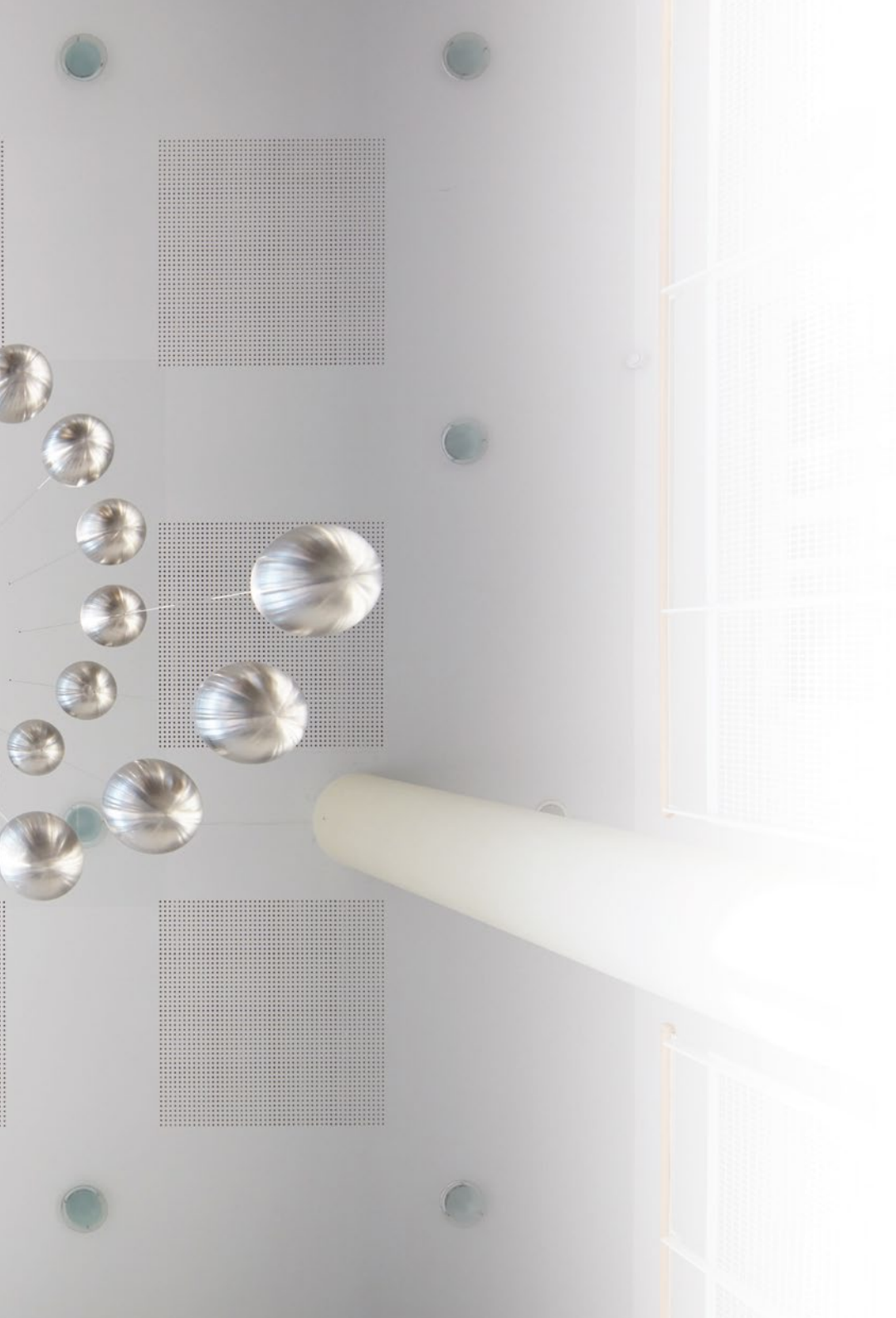
Current Groups

Max Planck-Humboldt Research Award

Alumni



In Memoriam

Facilities and Services

Administration and Infrastructure

Teaching and Training

Networking

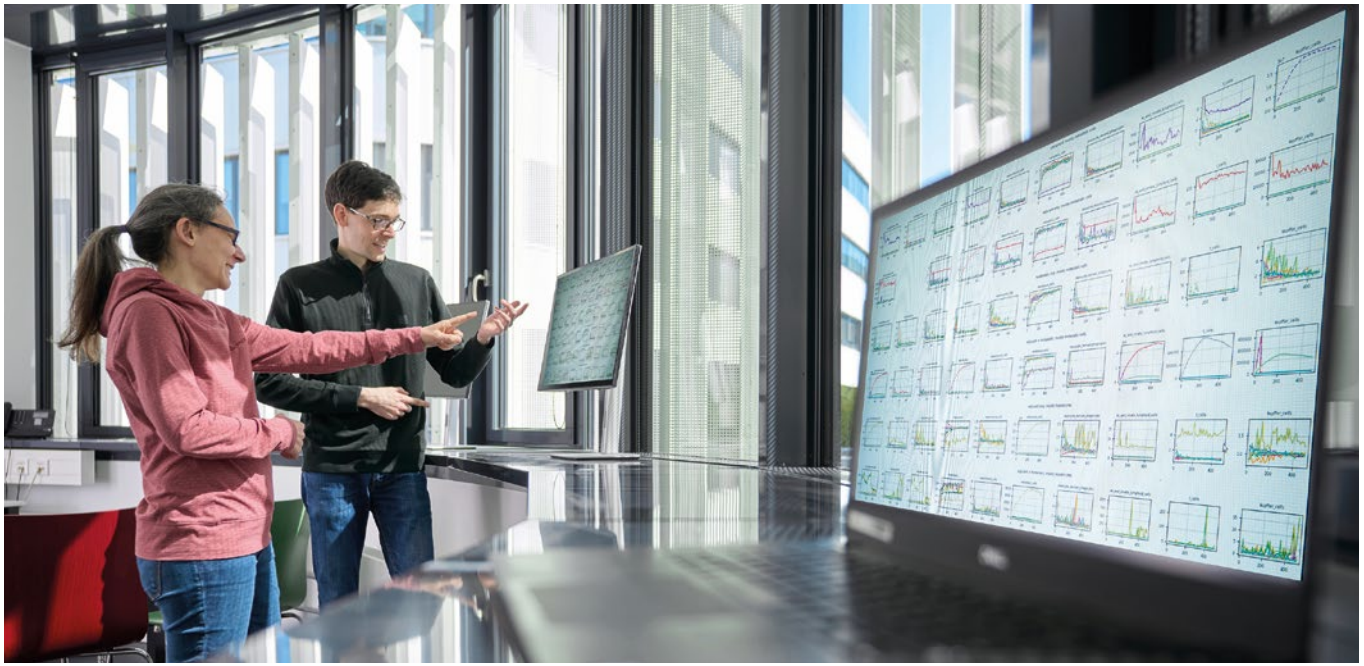
Appendices

Directors Report

2020–2025

The Gene Center is a central scientific institution of the Ludwig-Maximilians-Universität München. We take a highly interdisciplinary approach to answering fundamental scientific questions in basic and translational molecular life sciences, with a focus on genome biology, immunology, and biomedicine.

2020–2025 has been a dynamic, productive, and highly successful period—though not without its challenges. The first half was strongly affected by the COVID-19 pandemic. Gene Center scientists contributed in many ways to SARS-CoV-2 research and various services. Our researchers worked on the molecular basis of the disease and its clinical manifestations and developed reagents, vaccine technology, and diagnostic tools, including wastewater monitoring technology for viral RNA. During the reporting period, nine new junior and senior faculty members started their research groups at the Gene Center and brought new scientific topics and techniques. Numerous scientific breakthroughs, large research clusters, ERC funding, and major awards marked a successful period. We continued to build on our core strengths in the science of nucleic acids and their protein complexes, which culminated in a leading role in the new cross-institutional NUCLEATE Excellence Cluster, starting in 2026.



New Faculty

In the past five years, the Gene Center has welcomed several outstanding new group leaders and professors, particularly strengthening the areas of translational biomedical research, computational biology, and artificial intelligence. Marcia Ferraz joined in 2021 as a W2 Professor of Reproductive Biology and Medicine through a Sofja Kovalevskaja Award, while Maximilian Münchhoff launched his junior research group in 2022, focusing on host-virus interactions of pandemic pathogens. In 2023, Daniel Reichart joined the Gene Center as an Emmy Noether Research Group Leader with a focus on translational cardiovascular medicine.

In 2024, Jonathan Bohlen established a research group investigating how dysregulated mRNA translation impacts human physiology and disease. Computational biology and AI emerged as further areas of strategic expansion. Johanna Klughammer joined in 2021 as a tenure-track W2 Professor for Systems Immunology, strengthening both computational biology and spatial omics, as well as immunology, at the institute. Simon Mages established an innovative junior research group exploring the physics of high-dimensional biological data, applying tools from quantum field theory to analyse complex biological systems. Finally, Lukas Milles began a tenure-track W2 position at the Gene Center in 2024 in conjunction with an Emmy Noether Group at the Max Planck Institute of Biochemistry in the emerging field of protein design using deep learning methods.

We are also privileged to host two internationally renowned scientists as guest or visiting professors: Jeyaprakash Arulanandam (ERC Advanced Grant recipient) and Rotem Sorek (Max Planck-Humboldt Award laureate). Jeyaprakash Arulanandam investigates the complex molecular and structural mechanisms of chromosome segregation, while Rotem Sorek explores the emerging field of prokaryotic immunity and anti-phage defence systems, elucidating their molecular foundations. Franz Herzog (Structural Mass Spectrometry) and Stefan Canzar (Computational Biology) left the Gene Center to take on new positions, and we are very grateful for their numerous outstanding contributions over many years.

Research Highlights

During 2020–2025, Gene Center scientists reported numerous breakthrough findings across molecular biology, immunology, computational biology, and biomedical research. Our researchers secured more than 57 M€ in third party funding, often from highly interdisciplinary and collaborative research grants and cluster, such as Transregio 237 "Nucleic Acid Immunity" that is currently coordinated at the Gene Center (Speaker Veit Hornung).

In basic science, the Beckmann lab revealed how a cellular quality-control complex, Ccr4-Not, monitors protein synthesis for "hard-to-read" genetic code. They showed that this complex attaches to ribosomes stalled on non-optimal mRNA sequences and triggers targeted degradation of those mRNA molecules, linking slow translation to mRNA decay and maintaining fidelity in gene expression (Buschauer et al. (2020), Science 368:eaay6912). The Jae lab previously discovered a mitochondrial "stress sensor" pathway centred on the protein DELE1 and analysed the underlying mechanism further (Fessler et al. (2022), Nat. Commun. 3:1853). Their findings explain how human cells detect and respond to internal mitochondrial damage, which may inform new treatments for diseases involving mitochondrial dysfunction. The Hopfner lab helped uncover how a chromatin remodeller deals with nucleosomes missing a pair of histones, adding a new layer to our understanding of gene regulation (Zhang et al. (2023), Science 381:313–319). Julian Stingele and his team discovered a repair pathway that can deal with protein–RNA crosslinks (Zhao et al. (2023), Mol. Cell 83:4290–4303.e9), leading to the emergence of a new field that the team featured in an invited review (Cordes et al. (2025), Cell 188:885–900). The Stigler lab uncovered how the LINE-1 retrotransposon protein ORF1p forms RNA–protein condensates that bind DNA during mitosis—a key step in LINE-1 insertion into the genome (Zernia et al. (2025), Sci. Adv. 11:eadt9318). The Förstemann lab discovered that, during antiviral defence in fruit flies, key RNA-processing proteins form liquid-like droplets inside cells. This compartmentalisation helps the cell efficiently produce small interfering RNAs and fine-tune its antiviral immune response (Hipp et al. (2025), Nucleic Acids Res. 53:gkaf664). Jeyaprakash Arulanandam's lab clarified the conundrum how centromeres, a key feature of our chromosomes and chromatin, are inherited in cell division processes (Parashara et al., (2024), Science 385: 1098-1104). The Hornung lab elucidated the activation mechanism of the endosomal receptor TLR7: they showed that RNase T2 and phospholipases D3/D4 digest viral RNA into fragments that activate TLR7 (Bérouti et al. (2024), Immunity 57:1482–1496). In follow-up work, the team showed that mRNAs containing methyl-pseudouridine are not cleaved by these enzymes—explaining why such modified mRNA vaccines are immune-tolerant and highly effective (Bérouti et al. (2025), Cell S0092-8674(25)00619-1).

In computational biology, the Canzar group introduced a new approach to analysing RNA transcripts and improving transcriptome reconstruction (Ringeling et al. (2022), Nat. Biotechnol. 40:741–750). Simon Mages, Johanna Klughammer, and co-authors developed TACCO, a computational framework for the analysis of multi-omics data (Mages et al. (2023), Nat. Biotechnol. 41:1465–1473). This framework was also used by the Klughammer lab and collaborators to produce a detailed atlas of metastatic breast cancer using single-cell and spatial genomics with multi-modal data integration. Their work showcases how such comprehensive maps can uncover clinically relevant tumour-immune interactions in metastases (Klughammer et al. (2024), Nat. Med. 30:3236–3249).

In translational biomedical research, many important results were achieved. The Keppler lab and collaborators developed a rapid CRISPR gene-editing method for human T cells, enabling detailed studies of HIV biology and immune functions in previously hard-to-manipulate cells (Albanese et al. (2022), Nat. Methods 19:81–89). The Klein lab developed miniature human

bone marrow organoids from induced pluripotent stem cells—a powerful new platform to study human blood development and disorders ex vivo (Frenz-Wiessner et al. (2024), Nat. Methods 21:868–881). The Münchhoff lab investigated the role of cytotoxic T cells in SARS-CoV-2 infections, uncovering how CD8 T cells influenced viral mutations during the first wave of the pandemic (Khatamzas et al. (2022), Nat. Commun. 13:5586). The Wolf lab advanced xenotransplantation technology toward clinical application and investigated therapeutic strategies for Duchenne muscular dystrophy in large animal models (Stirm et al. (2023), PNAS 120:e2301250120). Marion Subklewe's team discovered that the endogenous signalling nucleotide cGAMP enhances T-cell-engaging immunotherapy for acute myeloid leukaemia (Linder et al. (2025), Blood 145:2149–2160).

These are only a few selected highlights; a comprehensive list of outstanding contributions can be found on the individual group pages and in the appendix.

Awards

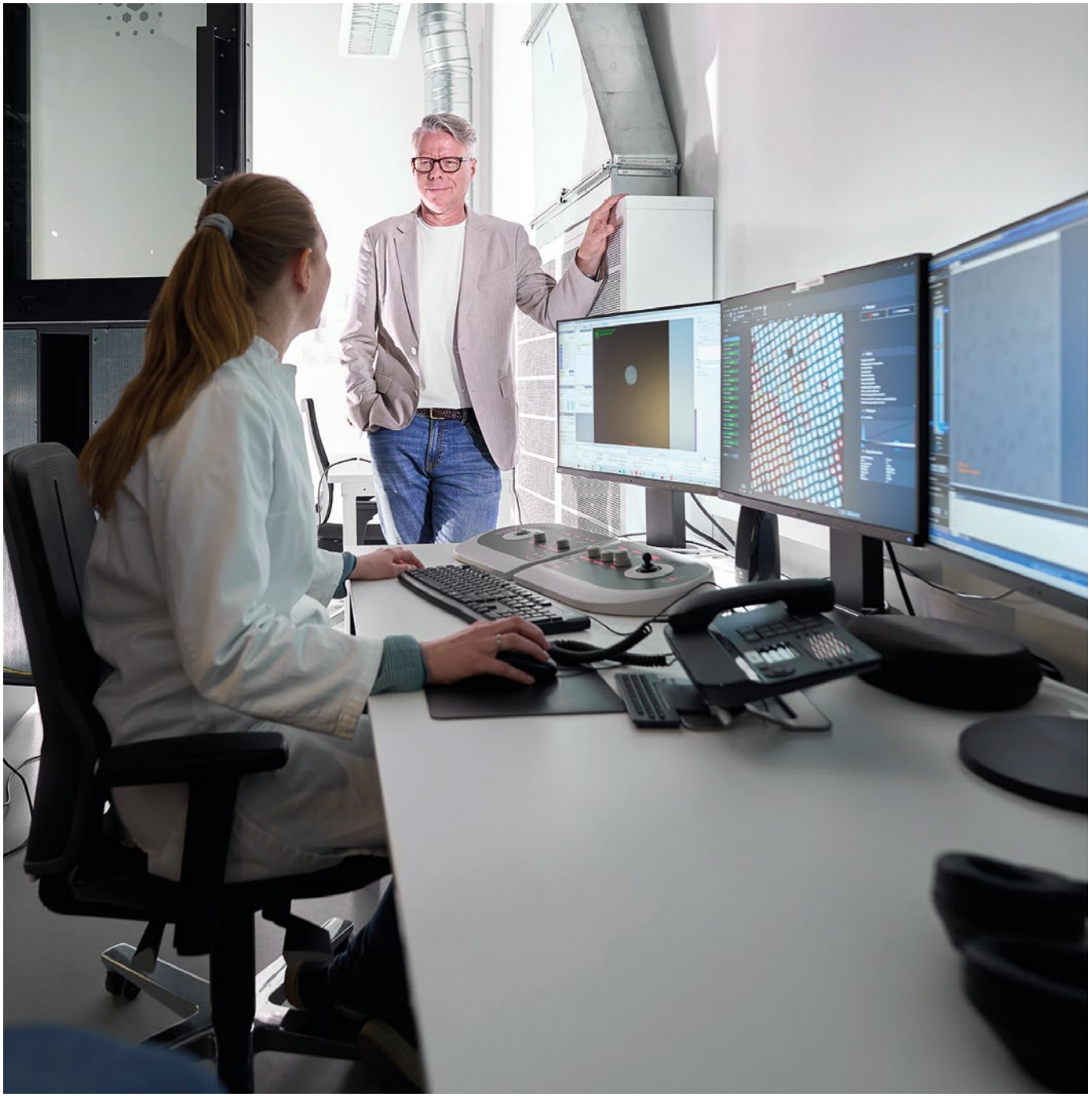
From early-career accolades to major international prizes, our scientists received significant recognition for their outstanding contributions to biomedical research.

Veit Hornung was co-recipient of the William B. Coley Award for Distinguished Research in Basic and Tumor Immunology (2020) and received the prestigious Collen-Jeantet Award for Translational Medicine for his groundbreaking work in innate immune signalling pathways (2025). Marcia Ferraz was awarded the Sofja Kovalevskaja Award for her pioneering contributions to assisted reproduction. Julian Stingele was selected as an EMBO Young Investigator and honoured with the Vallee Scholar Award (2021) for his pioneering work on DNA–protein crosslink repair. Lucas Jae received the Life Sciences Bridge Award, the Alfried Krupp Prize, and the Vallee Scholar Award for his work on mitochondrial stress signalling. Oliver T. Keppler was awarded the Bavarian Order of Merit for his leadership and scientific contributions during the COVID-19 pandemic. In 2023, Rotem Sorek received the Max Planck–Humboldt Research Award for his work on bacterial and human innate immunity. ERC grants were awarded to Veit Hornung, Julian Stingele, and Lukas Milles. Among our emerging scientists, Michael Ameismeier received the Bayer Pharmaceuticals PhD Award (2021) for his dissertation on ribosome maturation. Additional awards from the Römer Foundation honoured early-career researchers across the departments, including Shubo Zhao, who also received the PhD Award of the Munich University Society.

I congratulate all awardees on their exceptional work and recognitions.

Training

Scientific training at the Gene Center is one of our core missions and spans the full academic range–from undergraduate education to graduate training and mentoring early-career investigators. Undergraduate teaching is carried out in several programs in close cooperation with colleagues from the Department of Chemistry and the Faculties of Informatics and Biology. Our international Master's program in Biochemistry and our PhD-level programs consistently attract 30–40% international students. Doctoral training is highly research-oriented and complemented by internal seminar series led by PhD students and postdocs. Many students enrol in the QMB or IMPRS-LS graduate schools. A biannual retreat further fosters dialogue between early-career and senior researchers. A cornerstone of our academic culture is early independence for young group leaders. With flat hierarchies and a cooperative work environment, junior researchers are encouraged to take on responsibility early and grow into leadership roles–as exemplified by the promotion of Julian Stingele to full professor. We are proud that many of our students and postdocs become successful PIs and that our young investigators secure prestigious grants and positions.



Outlook

While five years may seem brief for science, the period 2020–25 has witnessed technological breakthroughs fundamentally reshaping research and education. In 2021, mRNA vaccines revolutionized medicine, and DeepMind's AlphaFold2 resolved the longstanding protein folding problem, transforming protein science. Since late 2022, large-scale AI models such as ChatGPT have begun redefining the information age.

With the imminent launch of the NUCLEATE Excellence Cluster and the recruitment of faculty advancing computational and experimental research, the Gene Center is poised to contribute significantly to these transformations in biomedical science. Our future priorities include achieving the ambitious goals of NUCLEATE, further integrating experimental and computational disciplines, and initiating collaborative networks spanning nucleic acid biology, macromolecular complexity, and AI-driven protein design.

The Gene Center's standing as a leading institution in life and biomedical sciences is the result of the dedication of its entire community—students, postdocs, staff scientists, facility managers, and principal investigators—alongside indispensable support from our administrative and technical teams, including administration, IT, finance, workshops, lab services, outreach, and the research network office. I sincerely thank all members of the Gene Center for their exceptional commitment to fostering a collaborative and thriving environment.

Finally, I thank the LMU leadership, particularly President Prof. Bernd Huber, for their crucial support and partnership in our achievements. We look forward to collaborating with the incoming leadership team under Prof. Matthias Tschöp to shape the future of our life science campus.

The coming years will be challenging, driven by the transformative impacts of AI on science and society, as well as economic constraints and shifting financial priorities in a complex geopolitical environment. Recent trends in the US, linking research funding increasingly to ideological rather than merit- or evidence-based criteria, pose a threat to scientific freedom, integrity and innovation. Protecting and reinforcing open and free scientific research remains essential—not only for groundbreaking discoveries but also to safeguard democratic values, economic prosperity, international collaboration and increasing resiliencies of societies.

In this dynamic environment, the Gene Center and its vibrant international and interdisciplinary community are well positioned to drive innovation in both computational and experimental life sciences and to train the next generation of scientists who will help shape the future of biomedical research.

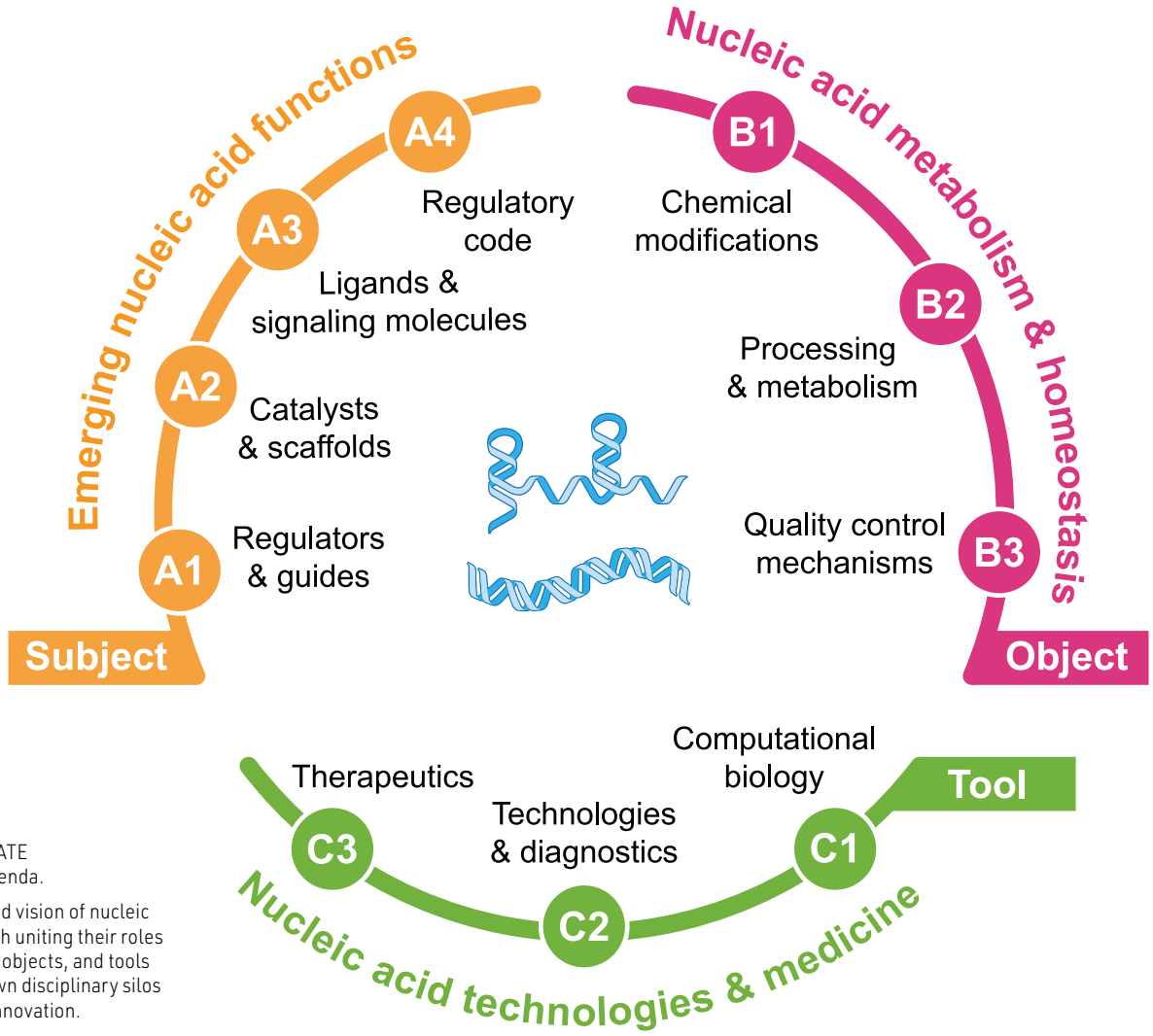
Cluster for Nucleic Acid Sciences and Technologies – NUCLEATE

NUCLEATE
Cluster for Nucleic Acid Sciences and Technologies

In 2025, NUCLEATE was successfully approved as one of the newly funded Clusters of Excellence under Germany's Excellence Strategy. The cluster is jointly coordinated by three universities: Ludwig Maximilian University of Munich (LMU), Technical University of Munich (TUM), and Julius Maximilian University of Würzburg (JMU), with LMU serving as the coordinating lead institution. Veit Hornung, from the Gene Center at LMU, acts as the LMU spokesperson within the cluster's speaker team. NUCLEATE was selected through a highly competitive, international, peer-reviewed process. It is one of 70 research clusters that will receive funding within the Excellence Strategy, a federal and state initiative in Germany that strengthens top-level research at universities. Funding will begin on January 1, 2026, for an initial period of seven years.

Research focus and scientific approach

NUCLEATE pursues a systematic and innovative approach to nucleic acid research. The aim is to study RNA and DNA molecules not only in their classic role as information storage devices, but also as active regulators and tools. The focus is on three perspectives: nucleic acids as subjects (active molecular actors), as objects (targets of biological regulation), and as tools (basis for therapeutic or technological applications). This conceptual tripartite division allows for a novel structuring of research approaches within the cluster. A particular focus is on elucidating previously unexplored RNA functions, characterizing regulatory networks, and developing new nucleic acid-based technologies, including genome editing, RNA inhibitors, synthetic therapeutics, and diagnostic tools.



The NUCLEATE research agenda.

An integrated vision of nucleic acid research uniting their roles as subjects, objects, and tools to break down disciplinary silos and foster innovation.

Core competence of the LMU Gene Center

The Gene Center plays a key role in the cluster. Several of the cluster's investigators are based at the Gene Center, including Veit Hornung, Karl-Peter Hopfner, Roland Beckmann, and Julian Stingele. Their work covers central aspects of nucleic acid biology - from genome integrity and RNA processing to innate immunity and the structural mechanisms of gene regulation. The Gene Center combines long-standing expertise with a broad methodological spectrum, including high-resolution structural biology techniques (such as cryo-electron microscopy), biophysical approaches, cell biology, and advanced sequencing technologies. This concentration of scientific and technical competence provides a strong foundation for NUCLEATE's overarching goal: to gain fundamental insights into the molecular control of cellular processes and to pave the way for new biomedical and technological applications.

Interdisciplinary collaboration and translational focus

A defining strength of NUCLEATE is its highly interdisciplinary structure. The cluster brings together expertise from across the participating institutions, integrating disciplines such as organic chemistry, molecular and cell biology, bioinformatics, immunology, pharmacology, and clinical medicine. This close collaboration across scientific fields and institutional boundaries enables complex biological questions to be tackled from multiple complementary perspectives. NUCLEATE addresses both fundamental research questions and the strategic development of innovative applications. These include new classes of RNA- and DNA-based therapeutics, novel treatment strategies for genetic diseases, next-generation vaccines, and precise in vivo gene regulation technologies. A particular focus lies on overcoming major translational challenges – e.g., the targeted delivery of nucleic acid-based drugs within the body to specific cells and tissues.

Structure formation, promotion of young talent, and future prospects

NUCLEATE is more than a scientific initiative - it is a long-term investment in the research ecosystem. Through the establishment of new professorships, junior research groups, and a joint, inter-university master's program, the cluster will build a sustainable academic framework that connects the three partner institutions. Early-career researchers will be immersed in a collaborative, interdisciplinary research environment and also gain access to cutting-edge technologies. Over the coming years, NUCLEATE aims to establish itself as one of Europe's leading centers for nucleic acid research. By promoting scientific excellence, training the next generation of researchers, and driving innovation, the cluster will enhance the international visibility of the field and strengthen the biomedical research landscape in Germany and beyond.

Veit Hornung is one of the spokespersons for the new NUCLEATE Cluster of Excellence.
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Celebrating 40 Years of the Gene Center Munich

In 2024, the Gene Center Munich proudly celebrated its 40th anniversary, marking four decades of excellence in molecular life sciences, interdisciplinary collaboration, and pioneering research. To honor this milestone, a series of events were organized in the first half of the year, bringing together leading international scientists, alumni, and current members of the Gene Center.

As part of the anniversary celebrations, the Horizons 20XX lecture series invited outstanding researchers to share their visions for the future of life sciences. These forward-looking talks provided insights into emerging scientific fields:

- Karsten Borgwardt (Max Planck Institute of Biochemistry) discussed "Machine Learning in Systems Biology: Now and Next," highlighting how artificial intelligence is poised to transform biological research.
- Rotem Sorek (Weizmann Institute of Science) presented "The Immune System of Bacteria: Beyond CRISPR," offering fascinating glimpses into bacterial defense mechanisms that extend far beyond the well-known CRISPR system.
- Julia Mahamid (EMBL Heidelberg) spoke on "Enabling Discovery by In-Cell Structural Biology," demonstrating how cutting-edge microscopy techniques allow researchers to visualize molecular processes within intact cells.

The anniversary celebrations culminated on June 21, 2024, in a half-day symposium titled "Celebrating 40 Years of Discovery and Envisioning Tomorrow's Science." The event began with welcoming remarks from distinguished speakers, including Karl-Peter Hopfner (Director of the Gene Center), Bernd Huber (President of LMU Munich), Markus Blume

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(Bavarian State Minister of Science and the Arts), and deans from both the Faculty of Chemistry and Pharmacy and the Faculty of Medicine. Patrick Cramer, President of the Max Planck Society (and former Director of the Gene Center), contributed a video message, emphasizing the Gene Center's importance within Germany's research landscape.

The symposium provided an opportunity to reflect on the Gene Center's journey. Founding Director Ernst-Ludwig Winnacker opened the program with "The Ideas Behind Its Existence," followed by Karl-Peter Hopfner outlining "Science Beyond Disciplines," which emphasized the center's longstanding commitment to interdisciplinary research.

The scientific program highlighted contributions from researchers who have taken important career steps in close connection with the Gene Center and its scientists. Clemens Plaschka, former PhD student and postdoctoral researcher with Patrick Cramer, presented "Mechanisms of Messenger RNA Maturation," while Johanna Klughammer, now a W2 professor at the Gene Center, shared her work on "A Single-Cell and Spatial Expression Map of Metastatic Breast Cancer Biopsies."

Further talks were given by Moritz Gaidt, former PhD student with Veit Hornung, on "Virulence Factor-Triggered Innate Immune Sensing," and Carina Baer de Oliveira Mann, former PhD student with Karl-Peter Hopfner, who discussed "Regulatory Mechanisms of Nucleotidyltransferase Activation." Early-career researchers contributed posters during the coffee break, before the day concluded with Daniel Wilson's entertaining talk "Beer, Bretzen and Blobology." Wilson, former group leader at the Gene Center, closely collaborated with Roland Beckmann during his time in Munich.

The 40th anniversary events underscored the Gene Center Munich's dynamic role in advancing life sciences research and training future scientific leaders. True to its mission, the Gene Center continues to foster curiosity-driven research and interdisciplinary collaboration, preparing for the discoveries of the next 40 years.

LMU LUDWIG-MAXIMILIANS-UNIVERSITÄT MÜNCHEN

40 years of research –
selected key publication
of every Gene Center group leader
(1984–2024)

Nature. 1989
341(6241):415-21.

Novel putative receptor tyrosine kinase encoded by the melanoma-inducing *Tu* locus in *Xiphophorus*

Schartl* lab

Nature. 1990
347(6288):90-2.

Megabase inversions in the human genome as physiological events

Zachau* lab

Nature. 1989
341(6240):337-40.

Krüppel requirement for *knirps* enhancement reflects overlapping gap gene activities in the *Drosophila* embryo

Jäckle* lab

Curr Biol. 2006
16(15):1538-43.

Coordination of endoplasmic reticulum and mRNA localization to the yeast bud

Jansen* lab

Mech Dev. 2005
122(7-8):914-27.

The *Drosophila* caspase DRONC is required for metamorphosis and cell death in response to irradiation and developmental signals

Petritsch* lab

J Exp Med. 1991 173(1):209-19.

Polymorphic structure of the tumor necrosis factor (TNF) locus: an NcoI polymorphism in the first intron of the human TNF-β gene correlates with a variant amino acid in position 26 and a reduced level of TNF-β production

Riethmüller/E Weiss* lab

Nature. 1989
342(6248):453-6.

Identification of a novel translation factor necessary for the incorporation of selenocysteine into protein

Böck* lab

RNA. 2009
15(11):2002-12.

Formation of She2p tetramers is required for mRNA binding, mRNP assembly, and localization

Jansen and Niessing* lab

Nucleic Acids Res. 1991
19(16):4515-21.

Further sequence requirements for male germ cell-specific expression under the control of the 14bp promoter element (β2UE1) of the *Drosophila* β2 tubulin gene

Renkawitz-Pohl* lab

FEBS Lett. 1988
236(1):27-32.

Isolation and characterization of the porcine nuclear factor I (NFI) gene

Winnacker* lab

Nat Methods. 2011
9(2):173-5.

HHblits: lightning-fast iterative protein sequence searching by HMM-HMM alignment

Söding* lab

Cell. 2003
114(3):347-57.

Architecture of the RNA polymerase II-TFIIS complex and implications for mRNA cleavage

Cramer* lab

Circulation. 1993
87(2):454-63.

Altered expression of β-adrenergic receptor kinase and β_1-adrenergic receptors in the failing human heart

Lohse* lab

Nature. 1988
332(6159):87-90.

Cooperativity of the glucocorticoid receptor and the CACCC-box binding factor

Renkawitz* lab

Mol Syst Biol. 2011
7:458.

Dynamic transcriptome analysis measures rates of mRNA synthesis and decay in yeast

Tresch* and Cramer* labs

Science. 2002
297(5582):854-7.

Cytomegalovirus recruitment of cellular kinases to dissolve the nuclear lamina

Koszinowski* lab

Cell. 1994
77(2):249-59.

Mdj1p, a novel chaperone of the DnaJ family, is involved in mitochondrial biogenesis and protein folding

Neupert* lab

Science. 1988
240(4855):1038-41.

Assembly of a functional immunoglobulin F_v fragment in *Escherichia coli*

Plückthun* lab

Mol Cell. 2013
52(2):173-83.

Argonaute and Triman generate dicer-independent priRNAs and mature siRNAs to initiate heterochromatin formation

Halic* lab

EMBO J. 2001
20(21):5863-75.

The 37-kDa/67k-Da laminin receptor acts as the cell surface receptor for the cellular prion protein

S Weiss* lab

Plant J. 1994
6(2):187-99.

Water channels in the plant plasma membrane cloned by immunoselection from a mammalian expression system

Schäffner* lab

Nature. 2020
579(433-437).

A pathway coordinated by DELE1 relays mitochondrial stress to the cytosol.

Jae* lab

PLoS Genet. 2013
9(11):e1003914.

Recruitment of TREX to the Transcription Machinery by Its Direct Binding to the Phospho-CTD of RNA Polymerase II

Cramer, Söding and Strässer* lab

Science. 2001
294(5548):1929-32.

Real-Time Single-Molecule Imaging of the Infection Pathway of an Adeno-Associated Virus

Hallek and Bräuchle* (CUP) labs

Science. 1996
274(5292):1517-20.

Dependence of yeast pre-mRNA 3′-end processing on CFT1: a sequence homolog of the mammalian AAUAAA binding factor

Domdey* lab

*corresponding lab

Science. 2021
371(6528):eabd0811.
Human NLRP1 is a sensor for double-stranded RNA.
Hornung* lab

Science. 2020
368(6488):eaay6912.
The Ccr4-Not complex monitors the translating ribosome for codon optimality.
Beckmann* lab

Mol Syst Biol. 2015
11(1):785.
Negative feedback buffers effects of regulatory variants
Gagneur* lab

Genes Dev. 2001
15(23):3088-103.
PIASy, a nuclear matrix-associated SUMO E3 ligase, represses LEF1 activity by sequestration into nuclear bodies
Grosschedl* lab

Science. 1996
272(5266):1343-7.
Structural Basis of Ligand Discrimination by Two Related RNA Aptamers Resolved by NMR Spectroscopy
Famulok* lab

Cell Rep. 2021
34(6):108742.
The glucocorticoid receptor recruits the COMPASS complex to regulate inflammatory transcription at macrophage enhancers
Uhlenhaut* lab

Nature. 2018
564(7736):430-433.
Consistent success in life-supporting porcine cardiac xenotransplantation.
Wolf* lab

Nat Commun. 2015
6:6123.
Proteasome assembly from 15S precursors involves major conformational changes and recycling of the Pba1-Pba2 chaperone
Herzog and Wendler* lab

EMBO J. 2000
19(11):2525-36.
Cytohesin-1 regulates β-2 integrin-mediated adhesion through both ARF-GEF function and interaction with LFA-1.
Kolanus* lab

Protein Sci. 1999
8(11):2245-50.
Intrabody construction and expression III: Engineering hyperstable V_H domains
Steipe* lab

Nat Biotechnol. 2022
40(5):741-750.
Partitioning RNAs by length improves transcriptome reconstruction from short-read RNA-seq data.
Gagneur (TUM) and Canzar* labs

Nat Genet. 2018
50(3):344-348.
Human TGF-β1 deficiency causes severe inflammatory bowel disease and encephalopathy
Hopfner and Klein* labs

Science. 2015
349(6243):70-4.
Single-cell-initiated monosynaptic tracing reveals layer-specific cortical network modules.
Conzelmann* lab

Nature. 2000
406(6793):306-9.
Presenilin is required for proper morphology and function of neurons in *C. elegans*
Baumeister* lab

Mol Syst Biol. 2022
18(2):e9816.
Large-scale analysis of *Drosophila* core promoter function using synthetic promoters
Söding and Gaul* labs

Nature. 2018
556(7701):386-390.
Structural basis for ATP-dependent chromatin remodelling by the INO80 complex.
Hopfner* lab

Mol Syst Biol. 2015
11(1):785.
Negative feedback buffers effects of regulatory variants
Gagneur* lab

Cell Rep. 2022
41(10):111778.
Stabilization of DNA fork junctions by Smc5/6 complexes revealed by single-molecule imaging
Stigler* lab

Mol Cell. 2017
67(4):711-723.e7.
Systematic Identification of MCU Modulators by Orthogonal Interspecies Chemical Screening
Perocchi* lab

Nat Commun. 2015
6:6123.
Proteasome assembly from 15S precursors involves major conformational changes and recycling of the Pba1-Pba2 chaperone
Herzog and Wendler* lab

Nat Cell Biol. 2024
26(5):797-810.
Transcription-coupled repair of DNA-protein cross-links depends on CSA and CSB
Stingele* lab

Nat Methods. 2022
19(1):81-89.
Rapid, efficient and activation-neutral gene editing of polyclonal primary human resting $CD4^+$ T cells allows complex functional analyses.
Conzelmann, Hornung and Keppler* labs

Nature. 2017
541(7638):546-549.
Structural basis for ArfA-RF2-mediated translation termination on mRNAs lacking stop codons
Beckmann and Wilson* labs

PLoS Pathog. 2017
13(4):e1006281.
Human cytomegalovirus glycoprotein complex gH/gL/gO uses PDGFR-α as a key for entry
Adler* lab

Nat Biotechnol. 2023
41(10):1465-1473.
TACCO: Unified annotation transfer and decomposition of cell identities for single-cell and spatial omics.
Mages and Klughammer* labs

Haematologica. 2022
107(9):2096-2107.
Increased visceral fat distribution and body composition impact cytokine release syndrome onset and severity after CD19 chimeric antigen receptor T-cell therapy in advanced B-cell malignancies
Theurich* and Subklewe* labs

Blood. 2022
140(10):1104-1118.
T-cell exhaustion induced by continuous bispecific molecule exposure is ameliorated by treatment-free intervals.
Theurich and Subklewe* labs

Nucleic Acids Res. 2017
45(W1):W276-W284.
compleXView: a server for the interpretation of protein abundance and connectivity information to identify protein complexes
Herzog* lab

PLoS Genet. 2017
13(6):e1006861
Splicing stimulates siRNA formation at *Drosophila* DNA double-strand breaks
LAFUGA and Förstemann* lab

Symposium

in Honor of Ernst-Ludwig Winnacker's 80th Birthday

On September 9 and 10, 2021, the Gene Center Munich hosted a two-day scientific symposium to celebrate the 80th birthday of its founding director, Prof. Dr. Ernst-Ludwig Winnacker. Under the theme "Life between Science and the Arts," the event brought together distinguished scientists, colleagues, friends, and guests to reflect on Winnacker's exceptional scientific career and his profound influence on research, education, and science policy in Germany and beyond.

The symposium opened with welcoming addresses by representatives from academia and politics, including Karl-Peter Hopfner (Director of the Gene Center), Hans van Ess (Vice-President of LMU Munich), Bernd Sibler (Bavarian State Minister of Science and the Arts), Angelika Vollmar (Dean of the Faculty of Chemistry and Pharmacy), and Gerald Haug (President of the German National Academy of Sciences Leopoldina). Their speeches highlighted Winnacker's pivotal role in establishing the Gene Center Munich and shaping the scientific landscape of Germany.

The scientific program reflected the diversity of modern biosciences, featuring presentations from leading researchers such as Emmanuelle Charpentier, Nobel Laureate and pioneer of CRISPR-Cas9 genome editing, who spoke about the transformative potential of CRISPR technology. Other highlights included talks by Michael Hallek on targeted cancer therapies, Ulrich Hartl on protein folding, Eckhard Wolf on xenotransplantation, and Christiane Nüsslein-Volhard, Nobel Laureate, on the evolution of biological aesthetics.

A standout moment of the program was the keynote lecture by Patrick Cramer, who at the time served as Director at the Max Planck Institute for Multidisciplinary Sciences in Göttingen and has since become President of the Max Planck Society. In his talk "Ernst-Ludwig Winnacker and the thread of life," he paid tribute to Winnacker's lifelong contributions to molecular biology and his visionary leadership in German science policy.

Beyond the scientific discourse, the symposium embraced the connection between science and the arts. The program included a concert featuring Michael Wehrmeyer (violoncello) and Clara Isabella Siegle (grand piano), creating a unique atmosphere that reflected Winnacker's passion for both scientific discovery and cultural enrichment.

On the occasion of Ernst-Ludwig Winnacker's 80th birthday, the "Ernst-Ludwig Winnacker Award for Enhancing the Impact of Science for the Benefit of Society" was established by the Bayer Foundation. As a special birthday gift, the award was presented to him during the symposium to honor his exceptional contributions to strengthening the dialogue between science and society – an effort that has characterized his career far beyond the laboratory. Since then, the award has been conferred on Antje Boetius (2023) and Alena Buyx (2025).

The symposium was a fitting tribute to a scientist whose influence has shaped generations of researchers and whose legacy continues to inspire.

On Academic Freedom

Some Thoughts by Ernst-Ludwig Winnacker

A cornerstone of any functioning democracy is academic freedom. In Germany, it is therefore guaranteed by the Basic Law, specifically in Article 5, Paragraph 3–albeit ranked behind human dignity, the right to life, and gender equality, but still enshrined. The consequences of losing academic freedom can currently be observed in the conflict between the present U.S. administration and Harvard University. In accordance with a dictum by the Vice President, stating that „professors are our enemies," the administration is pulling every lever it believes could harm the university. So far, Harvard continues to defend itself.

Other universities, such as Columbia University in New York, seem to have resigned themselves–mainly due to the severe financial losses threatened by the administration. Yet this cultural war is no longer just about individual universities, but about the entire system of science, research, and education, as evidenced by threats to revoke student visas for international students. In reality, science knows no borders. These newly imposed or anticipated barriers will have serious consequences for the American education system. They will also damage the reputation of the United States and the prosperity of its citizens–perhaps even of us all.

One is involuntarily reminded of conditions in Germany in 1933. Back then–in April 1933, to be precise–the Law for the Restoration of the Professional Civil Service served to radically purge institutions of higher education of people of Jewish descent. At that time, 20% of the faculty, including a large part of the intellectual elite, were dismissed. In truth, we have never fully recovered from this loss. Before 1933, scientific publications in the natural sciences were written in German–afterward, no more.

Even in the U.S., such events are not without precedent. In the late 1960s, there were major protests at American universities–especially in Berkeley, though not only there. These centered around the Vietnam War. When tensions escalated in the spring of 1969, then-Governor and later President Ronald Reagan sent in the National Guard to Berkeley. The campus was surrounded for weeks; regular lab work was only possible in the early morning hours. The unrest–known as the "People's Park" incident–only truly ended with the conclusion of the Vietnam War. Afterward, the Berkeley campus returned to the productive and admirable state we associate with successful research and teaching–until recently, when the authoritarian force of the MAGA movement brought that to an end.

So, what can we learn from these experiences today? The freedom of research, guaranteed already in the Weimar Constitution (Article 42) and today's Basic Law, is a fragile plant that can only flourish in a state governed by the rule of law. But even there, it must be nurtured. This "nurturing" includes appropriate framework conditions, such as sufficient funding, opportunities for international exchange, coherent support for early-career researchers, effective mechanisms for communication between science and society, and finally, incentives and institutional structures. These must be continually reviewed and adjusted.

What does that mean specifically? When the Excellence Initiative was launched 25 years ago, there were few funding opportunities for interdisciplinary collaboration. Apart from the Collaborative Research Centers–which often lacked critical mass–funding focused mainly on individual researchers' projects. Meanwhile, the pendulum has swung in the other direction, toward the creation of clusters. However, after three rounds of selection, the system has become so complex that individual applicants often receive only very modest support compared to the effort required to apply.

The current interest from top researchers abroad–especially from the U.S.–in our system presents an opportunity to shift the focus slightly back toward individual funding. The coalition agreement of the Merz government even provides for this to some extent.

It must not be overlooked, however, that it's not just about positions but also about infrastructure. In this respect, the Excellence Clusters–which include program allowances–are of great value, particularly for our universities. These institutions remain severely underfunded and yet bear almost the entire burden of education. At this point, I would like to sincerely congratulate the principal investigators of the NUCLEATE Excellence Cluster, Karl-Peter Hopfner and Veit Hornung, for not only launching this cluster together with colleagues from TUM and the University of Würzburg but also leading it to success.

What happens next? According to political statements, there will be major strategic focus areas in artificial intelligence, quantum computing, and space travel. That makes sense–at least at first glance, and maybe even at second. One is reminded of how Kennedy's call to land on the moon unleashed enormous energy. New technologies were needed and were developed at great speed. It quickly became clear that it wasn't just about rockets and payloads, but also about computing power and data processing. I had the chance to witness the launch of the Gemini rockets in Cape Canaveral in October 1965. At that time, the first of

A truly international team:
A total of 236 individuals are currently employed at the Gene Center, 78 of whom (33%) are international researchers representing 32 nationalities (as of August 7, 2025).

the two rockets was "lost," so the planned "rendezvous" couldn't happen. This type of maneuver had to be practiced again in later missions to ultimately enable the moon landing.

Artificial intelligence is currently on everyone's lips, often mentioned alongside quantum computing. The U.S. lead in this field seems insurmountable—if President Trump's numbers are even remotely accurate. OpenAI, the company founded by Sam Altman, recently opened an office in Munich (on May 22, 2025)—interestingly, on the same day we celebrated the 30th anniversary of the IZB in Martinsried. Back in 1995, we were globally competitive in biotechnology. What concerns me now are not only the considerable efforts—particularly by our federal and state governments—to keep pace in these areas, but also the lack of emphasis on basic research. While colleagues such as Geoffrey Hinton—whom I met in early December 2024 during the awarding of his Nobel Prize—believe that human-level intelligence has already been reached, further fundamental steps will still be needed to come even closer to this goal.

That is why I am—and remain—a proponent of basic research: research that does not pursue a specific goal, and whose breakthroughs emerge from the concept of serendipity. In this regard, I hope that amidst all the hype—understandable though it may be—about these transformative topics, the necessary humility is not lost. I'm very curious to see what we will observe and report in the next Gene Center report five years from now.

Barbara Adler

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Since 2005	Group leader, Max von Pettenkofer-Institute & Gene Center LMU
1997-2005	Research Assistant, Helmholtz Center Munich and Max von Pettenkofer-Institute, LMU
1995 – 1997	Postdoc, Dana Farber Cancer Institute and Harvard Medical School, Boston, USA
1992 - 1995	Postdoc, Institute of Veterinary Virology, Bern, Switzerland
1992	PhD, Albert-Ludwigs-University, Freiburg

Biology of Cytomegaloviruses

Goals and Impacts for Society

Human cytomegalovirus (HCMV) infections are a major health threat for immunocompromised humans like transplant and tumor patients. Furthermore, intrauterine HCMV infections of the developing fetus are a frequent cause of severe birth defects. As current HCMV treatments often cause severe side effects or induce viral resistance, vaccines protecting from HCMV disease or intrauterine infection are urgently needed. Our research focuses on the roles of CMV envelope glycoproteins in entry and spread. At the same time, we investigate the potential of CMV glycoproteins as vaccine antigens in preclinical models.

Research Highlights

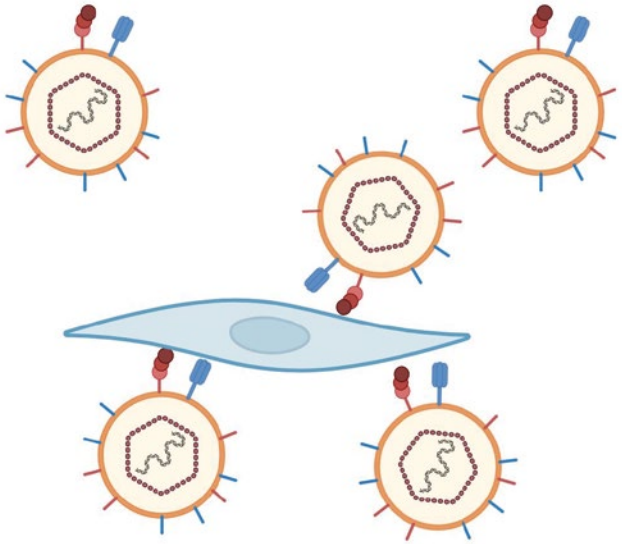
Viral envelope glycoproteins are mainly considered as keys to enter host cells. Beyond that, binding of these glycoproteins to cellular receptors or coreceptors can induce cellular signaling pathways and thus modulate the antiviral immune response. During the last five years, our research focused on the HCMV gHgL glycoprotein complexes, gHgLgO and gHgLpUL(128,130,131A), and the functionally homologues gHgL complexes of murine cytomegalovirus (MCMV).

After having identified PDGFRα as the cellular receptor for HCMV gHgLgO in 2017, we could very recently identify an essential second role for gHgLgO in virion tethering to host cell surface heparan sulfate proteoglycans. This was enabled through a Gene Center cooperation with the Fröhlich proteomics lab. Our finding challenges the previous text book knowledge on HCMV tethering. Additionally, the dual role of the gHgLgO complex renders the gHgLgO complex a highly interesting vaccine target.

The gHgLpUL(128,130,131A) complex of HCMV is special as its constituent pUL128 is a functional CC chemokine, which also has a dual role: It can attract monocytes to the site of infection and activate them and, as part of the gHgLpUL(128,130,131A) complex, it promotes entry receptor binding. How these functions contribute to a CMV infection in vivo has since years been a matter of speculation. By studying different MCMV chemokine mutants either lacking the chemokine function or the ability to integrate into the gHgLchemokine complex, we could clearly show that the chemokine activity and the gHgLchemokine interaction with its receptors are independent functions of the chemokine. The chemokine modulates the antiviral innate and adaptive immune responses and the gHgLchemokine complex secures horizontal virus spread through infection of salivary glands.

Future Directions

Our future focus will be to evaluate the potential of the gHgLgO complex as a vaccine antigen by dissecting the potential of antibodies specific for HCMV gHgLgO and by comparing anti-MCMV gHgLgO immune responses in the infection and after vaccination of mice. Additionally, we will evaluate how the CMV CC chemokine shapes the vaccination outcome when evaluating CMV glycoproteins as vaccine targets.



Glycoprotein-driven virus - cell interactions

Selected Publications

(1) Thiessen L, Garuti R, Kubic L, Kösters M, Amarambedu Selvakumar D, Krey T, Görzer I, Fröhlich T, **Adler B**. (2025) **Identification of the human cytomegalovirus gHgLgO trimer as the central player in virion infectivity. PLoS Pathog.** 21(7):e1013341.

(2) Rožmanić C, Lisnić B, Pribanić Matešić M, Mihalić A, Hiršl L, Park E, Lesac Brizić A, Indenbirken D, Viduka I, Šantić M, **Adler B**, Yokoyama WM, Krmpotić A, Juranić Lisnić V, Jonjić S, Brizić I (2023). **Perinatal murine cytomegalovirus infection reshapes the transcriptional profile and functionality of NK cells**. **Nat Commun**. 14(1):6412.

(3) Xu M, Ito-Kureha T, Kang HS, Chernev A, Raj T, Hoefig KP, Hohn C, Giesert F, Wang Y, Pan W, Ziętara N, Straub T, Feederle R, Daniel C, **Adler B**, König J, Feske S, Tsokos GC, Wurst W, Urlaub H, Sattler M, Kisielow J, Wulczyn FG, Łyszkiewicz M, Heissmeyer V (2024). **The thymocyte-specific RNA-binding protein Arpp21 provides TCR repertoire diversity by binding to the 3'-UTR and promoting Rag1 mRNA expression**. **Nat Commun.** 15(1):2194.

(4) **Adler B**, Adler H (2022). **Type I interferon signaling and macrophages: a double-edged sword? Cell Mol Immunol.** 19(9):967-968.

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Since 2022	Professor of Structural Cell Biology and Head of the Institute, Institute of Cell Biology, University of Edinburgh
2012-2022	Wellcome Research Fellow/Research Group Leader, Wellcome Centre for Cell Biology, University of Edinburgh
2004-2011	Postdoc - Alexander von Humboldt Fellow (2004-2005) and Marie-Curie Incoming International Fellow (2005-2007), EMBL-Heidelberg & Postdoc Fellow (2007-2011), MPI for Biochemistry, Martinsried
2004	PhD Indian Institute of Science, Bangalore

Mechanistic Biology of Chromosome Segregation

Goals and Impacts for Society

The accurate transfer of genetic information during cell divisions is essential for the survival of organisms and requires faithful segregation of chromosomes to the daughter cells. Errors in this process will result in aneuploidy, abnormal chromosome numbers or structures implicated in miscarriages, infertility, birth defects and several human cancers. Understanding the mechanisms behind chromosome segregation is crucial not just for basic biology but also for addressing significant health issues. Our research utilizes integrative structure-function approaches, combining biochemical, structural, and cell biological methods, to uncover detailed mechanisms that safeguard accurate cell division.

Research Highlights

Error-free chromosome segregation relies on the selective stabilization of chromosome-microtubule attachments and the maintenance of sister chromatid cohesion until all chromosomes are correctly bi-oriented. Two key chromosomal regions are central: the centromere, marked by CENP-A nucleosomes, and the inner centromere, which regulates kinetochore-microtubule interactions and the timing of chromatid separation. CENP-A nucleosomes are diluted during DNA replication, requiring replenishment during each cell cycle to preserve centromere identity and ensure proper microtubule attachment. The inner centromere acts as a regulatory hub, recruiting factors essential for accurate segregation.

Our research addresses three critical questions: (1) How is the inner centromere regulatory platform established? (2) How does it ensure accurate chromosome segregation? (3) How is centromere identity maintained through cell generations? We leverage advanced structural and functional techniques, such as X-ray crystallography, cryo-electron microscopy, crosslinking/mass spectrometry, and cell-based assays, to dissect these processes. The Chromosomal Passenger Complex (CPC) is a major regulator of cell division. CPC centromere association is critical for its essential role in ensuring error-free chromosome segregation. Our recent work provided a high-resolution structural basis for CPC centromere association and discovered a novel chromatin protection mechanism of the CPC, demonstrating that perturbing the CPC-chromatin interaction weakens the chromatin association of the CPC, causing mitotic defects (1).

Our recent work on CENP-32, a putative RNA methyltransferase initially identified as a novel mitotic chromosome association protein, revealed that it is an active RNA methyltransferase (2) and its activity is critical for maintaining the structural integrity of the mitotic spindle, the chromosome segregation apparatus. We also demonstrated that CENP-32 disease variants observed in patients with a broad spectrum of neurodevelopmental disorders compromise the enzymatic activity and perturb mitotic spindle integrity, thereby revealing CENP-32 as a clinically relevant essential regulator of cell division and proliferation.

Centromere maintenance (replenishment of CENP-A levels at centromeres through active CENP-A deposition) is a tightly controlled process, and its spatio-temporal regulation is achieved by Cdk1 and PLK1 kinases. Mechanistic understanding of how Cdk1 and PLK1 act as licensing factors for the timely CENP-A deposition remained a critical unanswered question for more than a decade. Our work on the core CENP-A deposition machinery, the Mis18 complex, and its interaction with the PLK1 kinase revealed essential roles of a PLK1-mediated phosphorylation cascade in activating the Mis18 complex. We demonstrated that PLK1-mediated conformational activation of the Mis18 complex is critical for maintaining the correct CENP-A levels at the centromere (3, 4).

Future Directions

Our work is paving the way for addressing long-standing questions on centromere inheritance and centromere-mediated control of sister-chromatid cohesion, error-correction, and timely chromosome segregation. Using this information, we will provide a comprehensive mechanistic understanding of how cells achieve error-free chromosome segregation. We are also keen to discover novel non-canonical chromosome segregation mechanisms in the evolutionarily divergent, yet clinically highly relevant, human fungal pathogens, including Cryptococcus neoformans.

Selected Publications

(1) Gireesh A*, Abad M*, Nozawa R, Sotelo-Parrilla P, Dury L, Likhodeeva M, Spanos C, Peralta CC, Rappsilber J, Hopfner K, Wilson M, Vanderlinden W, Hirota T, Jeyaprakash AA§ (2025). **Chromatin Protection by the Chromosomal Passenger Complex. bioRxiv** (2025).
(*equal contribution; § Corresponding author)

(2) Dharmadhikari AV, Abad MA et al., Davis EE§, Jeyaprakash AA§, Liao J§ (2025). **RNA Methyltransferase SPOUT1/CENP-32 Links Mitotic Spindle Organisation with the Neurodevelopmental Disorder SpADMiSS. Nat Commun**. 16:1073.

(3) Parashara P*, Medina-Pritchard B*, Abad MA*, Sotelo-Parrilla P*, et al., **Jeyaprakash AA§** (2024). **PLK1-Mediated Phosphorylation Cascade Activates Mis18 Complex to Ensure Centromere Inheritance. Science** 285 (6713): 1098-1104.

(4) Thamkachy R*, Medina-Pritchard B*, Park SO*, et al., **Jeyaprakash AA§** (2024). **Structural Basis for Mis18 Complex Assembly and its Implications for Centromere Maintenance. EMBO Rep** 25(8):3348-3372.

Roland Beckmann

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2023-2025	Dean of the Faculty of Chemistry and Pharmacy, LMU
Since 2006	Professor and Chair, Gene Center and Department of Biochemistry, LMU
2001-2006	Group leader Volkswagenstiftung, Charité, Humboldt University, Berlin
1995-2000	Postdoc, Rockefeller University, New York, USA
1995	PhD (Dr. rer. nat.), Free University Berlin

Structural Ribosome Biochemistry

Goals and Impacts for Society

Our research is driven by a fundamental curiosity to understand how ribosomes–the core machinery of protein synthesis–are assembled, regulated, and function within cells with essential roles beyond their protein synthesis activity. By using structural and biochemical approaches, we aim to uncover the molecular principles that facilitate and govern these processes. While our work is rooted in basic science, these insights have the potential for future medical advances, as they lay the groundwork for understanding and eventually treating diseases caused by perturbed ribosome assembly, failed quality control or translation defects, such as ribosomopathies, cancer and neurodegeneration.

Research Highlights

A core area has been the structural analysis of eukaryotic ribo–some biogenesis, a complex assembly pathway, involving hundreds of proteins and rRNA. In close collaboration with the lab of Ed Hurt, we have captured and characterized key assembly intermediates of both the 40S and 60S ribosomal subunits. These studies revealed the coordinated actions of assembly factors, helicases, and quality control enzymes that facilitate stepwise maturation and chec point control of pre-ribosomal particles. Among our findings were new insights into 5S RNP recruitment, release and stress sensing via the p53 pathway, as well as mechanisms governing the late stages of 40S subunit maturation (3). Moreover, we solved the structure of the essential snR30 H/ACA snoRNP and discovered its unexpected function in 18S rRNA folding (1). Together with the lab of Ed Hurt, University of Heidelberg, we have summarized in two snapshot articles in Cell the current state of understanding of ribosome assembly.

Beyond ribosome assembly, we have explored how the translation machinery responds to stress, translational errors, and perturbations in mRNA decoding. We elucidated how the Ccr4-Not complex recognizes elongation slowdowns, especially at ribosomes translating non-optimal codons, and how it links these to mRNA decay (4), thereby understanding one of the major determinants of eukaryotic mRNA half-live. We also discovered that ribosomal collisions are an evolutionary conserved proxy for quality control and cellular stress by determination of the architecture of bacterial collided ribosomes together with their sensing and clearing factors SmrB and HrpA. Similarly in eukaryotes, we found how the RQT complex, a conserved helicase assembly, identifies and resolves ribosome collisions. We further investigated how during RQC, stalled incomplete nascent polypeptide chains are marked for degradation through CAT tailing, a non-canonical elongation event on the 60S subunits. In addition, we identified and structurally characterized at the ER a 60S recycling pathway dependent on the UFM1 modification of the ribosome, in which UFM1 acts as a wedge to disengage stalled ribosomes from the translocon. Further studies explained how upon ribosomal stalling the RQC machinery for CAT tailing can work together with the UFMylation machinery to ensure decay of incomplete nascent polypeptides at the ER (2), thereby maintaining cellular protein homeostasis.

Together, our recent work has advanced the molecular and functional understanding of how ribosomes operate not only as protein factories, but as sensory and regulatory hubs, vital to cellular health.

Future Directions

Building on our recent discoveries, we aim to further dissect the molecular mechanisms of translational regulation and quality control, with a focus on the role of the ribosome in the cell's sensing and responding to regulatory and to stress signals. We will expand our studies on ribosome collisions, rRNA- and mRNA-triggered quality control, and on the newly recognized non-canonical roles of snoRNPs in ribosome biogenesis. Integrating structural, biochemical, and cellular approaches, we strive to uncover new principles with relevance to human health.

Selected Publications

(1) Fischer P, Thoms M, Lau B, Denk T, Kuvshinova M, Berninghausen O, Flemming D, Hurt E, **Beckmann R** (2025). **H/ACA snR30 snoRNP guides independent 18S rRNA subdomain formation. Nat Commun.** 16(1): 4720.

(2) Penchev I, Gumbin S, Scavone F, Berninghausen O, Becker T, Kopito R, **Beckmann R (2025). UFMylation orchestrates spatiotemporal coordination of RQC at the ER. Sci Adv.** 11(18):eadv0435.

(3) Ameismeier M, Zemp I, van den Heuvel J, Thoms M, Berninghausen O, Kutay U, **Beckmann R** (2020). **Structural basis for the final steps of human 40S ribosome maturation. Nature.** 587(7835):683-687.

(4) Buschauer R, Matsuo Y, Sugiyama T, Chen YH, Alhusaini N, Sweet T, Ikeuchi K, Cheng J, Matsuki Y, Nobuta R, Gilmozzi A, Berninghausen O, Tesina P, Becker T, Coller J, Inada T, **Beckmann R** (2020). **The Ccr4-Not complex monitors the translating ribosome for codon optimality. Science.** 368(6488):eaay6912.

Selected Awards and Honors

2020 ERC Advanced Grant 'Human-Ribogenesis'

Jonathan Bohlen

Web	www.genzentrum.uni-muenchen.de research-groups/bohlen/index.html
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Since 2024	Independent group leader Gene Center, LMU, Munich, Germany
2021- 2024	Postdoc, Institut Imagine, INSERM, Paris, France, Human genetics of Infectious Disease, Prof. Dr. Jean-Laurent Casanova
2017-2020	PhD, DKFZ Heidelberg, Karls-Ruprecht Universität Heidelberg, Prof. Dr. Aurelie Teleman

mRNA Translation in Human Immunity

Goals and Impacts for Society

Our primary aim is to decode how dysregulated mRNA translation contributes to inborn errors of immunity (IEI)—the monogenic defects that leave patients vulnerable to specific infections or autoimmune manifestations. By rigorously mapping how mutations in translation factors, tRNA synthetases, and RNA-binding proteins derail protein synthesis in immune cells, we intend to pinpoint causative nodes for precision diagnostics and tailored therapies. Society will benefit from improved genetic screening panels that incorporate translational regulators, enabling earlier diagnosis of IEI. Moreover, interventions designed to correct or bypass these translational defects—such as allele-specific mRNA editing or small molecules that stabilize stalled ribosomes—could transform care for patients with otherwise refractory congenital immunodeficiencies.

Research Highlights

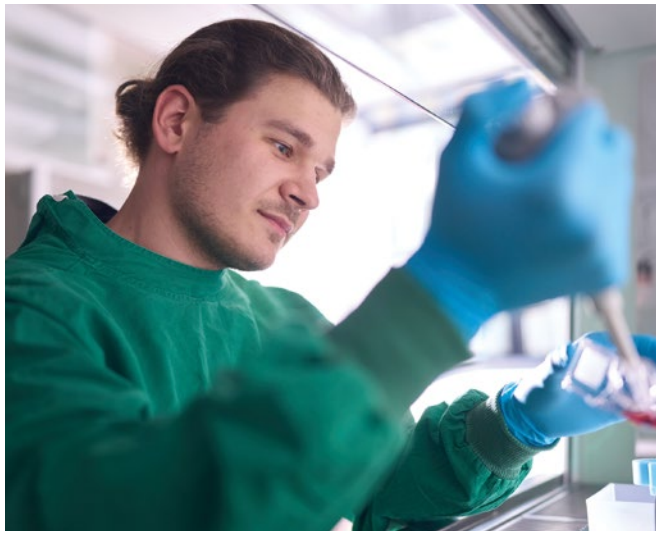
MCTS1–JAK2 Axis in Mycobacterial Susceptibility
We showed that loss of MCTS1 selectively impairs ribosomal loading on JAK2 mRNA in innate-like T cells, crippling IL-23-driven IFN-γ responses and underlying X-linked mycobacterial disease.

CBL Variants Drive Monocyte-Mediated Autoinflammation
Germline CBL loss-of-function, compounded by somatic loss of heterozygosity, provokes constitutive ERK activation in monocytes, mirroring monogenic autoinflammatory signatures and revealing CBL's key role in restraining innate activation.

UNC93B1 Mutations in TLR7-Linked Lupus
Heterozygous UNC93B1 missense changes enhance TLR7/TLR8 trafficking and signaling in patient cells and knock-in mice, predisposing to early-onset systemic lupus through hyperactive endosomal sensing.

Future Directions

We are implementing Ribo-seq and tRNA-seq workflows tailored to primary human immune cells to capture translational heterogeneity across leukocyte subsets. Concurrently, we aim to expand the catalog of inborn errors of immunity (IEI) by systematically screening cohorts with unexplained immunodeficiencies for variants in translational regulators. Integrating our Ribo-seq and tRNA-seq approaches with exome and genome data—following recent IEI classification updates—will highlight candidate genes whose disruption skews translation. Functional validation in patient-derived cells will uncover new monogenic IEI affecting factors such as aminoacyl-tRNA synthetases or ribosome-associated chaperones. Finally, we will reexamine known ribosomopathies (e.g., Diamond-Blackfan anemia, Shwachman–Diamond syndrome) within immune cell contexts: leveraging insights from translation-selectivity studies and the spectrum of ribosomopathy phenotypes, we will characterize how partial ribosomal insufficiency perturbs cytokine synthesis and immune development. Together, these efforts position us to decode translational dysregulation as a root cause of both rare and common immune disorders.



Selected Publications

(1) **Bohlen J@**, Bagaric I#, Vatovec T#, Ogishi M#, ..., Casanova JL, Bustamante J@ (2024). **Autoinflammation in patients with leukocytic CBL loss-of-heterozygosity is caused by constitutive ERK-mediated monocyte activation. J Clin Invest.** 134(20):e181604.

(2) **Bohlen J@**, Zhang Q, Philippot Q, ..., Bustamante J@, Zhang Q, Casanova JL@ (2023). **Human MCTS1-dependent translation of JAK2 is essential for IFN-γ immunity to mycobacteria. Cell.** 186(23):5114-5134.e27

(3) **Bohlen J@,*,** Roiuk M*, Neff M, Teleman AA@ (2023). **PRRC2 proteins impact translation initiation by promoting leaky scanning. Nucleic Acids** Res. 51(7):3391-3409.

(4) **Bohlen J**, Fenzl K, Kramer G, Bukau B and Teleman AA@ (2020). **Selective 40S footprinting reveals cap-tethered ribosome scanning in human cells. Mol Cell.** 79(4):561-574.e5.

Selected Awards and Honors

2025 Else-Kröner Fresenius Erstförderung

2020 RNA Society/Scaringe Graduate Student Award

Marcia Ferraz

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Since 2021	Professor (W2) for Clinical-Experimental Reproductive Medicine, Faculty of Veterinary Medicine, LMU
Since 2020	Affiliate Professor, Dept. of Biology, UBC Kelowna, British Columbia, Canada
2017-2020	Postdoc at Smithsonian National Zoo and Conservation Biology Institute, Front Royal, Virginia
2013-2018	PhD thesis, Faculty of Veterinary Medicine, Utrecht University
2011-2013	Master in Animal Genetics and Biotechnology of Reproduction Autonomous University of Barcelona and Polytechnic University of Valencia
2005-2010	Doctor Veterinary Medicine, Faculty of Veterinary Medicine, University of São Paulo

Bioengineering Reproduction for Health and Conservation

Goals and Impacts for Society

Reproduction is central to species survival, biodiversity conservation, and human and animal health. Yet, the physiological processes that govern gamete competence, fertilisation, and early embryo development remain poorly replicated in vitro. Our lab tackles this challenge by developing biomimetic models of the female reproductive system, using cutting-edge tools such as 3D culture, decellularized extracellular matrix (dECM) hydrogels, and microfluidic systems. These models help us study cellular communication, mechanotransduction, and environmental effects on fertility. Our mission is to advance assisted reproductive technologies across species, ranging from endangered wildlife to livestock and humans, while also creating platforms to test environmental toxicants and model reproductive diseases. Beyond scientific impact, we champion sustainability in research: our lab was the second German university-based lab to receive the My Green Lab certification.

Research Highlights

We focus on reconstructing reproductive environments in vitro that closely mimic in vivo conditions–both structurally and functionally. Our lab was the first to develop decellularized extracellular matrix (dECM) hydrogels derived from ovary, oviduct, and uterus, which retain tissue-specific mechanical properties and biochemical signals. These hydrogels support folliculogenesis, hormone secretion, and embryo development, and serve as powerful platforms to study ovarian aging, implantation, and tissue regeneration. To guide their design, we mapped the biomechanical profiles of both native and decellularized reproductive tissues, providing insight into how stiffness and architecture influence cellular behaviour.

Building on this, we engineered a field-deployable fluidic device –OoTrap–that enables oocyte collection and in vitro maturation outside standard laboratory infrastructure. Originally conceived for wildlife and livestock conservation, OoTrap exemplifies our translational approach: combining biological understanding with practical engineering to expand access to assisted reproductive technologies. At the same time, we are refining dynamic microfluidic systems that recreate physiological flow and shear forces, restoring mechanotransduction pathways essential for embryo competence–features lost in traditional static culture.

We also investigate how environmental pollutants affect reproductive health using our in vitro models. Our group provided the first evidence of microplastics in human and bovine follicular fluid, and showed that polystyrene microparticles compromise sperm function, reduce oocyte quality, and impair embryo development. These findings highlight the broader applications of our systems, not only for fertility enhancement but also for toxicological screening and environmental risk assessment.

Future Directions

Looking forward, we aim to reconstruct a functional mini-ovary in vitro using hiPSC-derived granulosa and germ-like cells embedded in bioactive ECM-inspired hydrogels. This model will enable the study of ovarian aging, hormone regulation, and folliculogenesis. We are also developing integrated dynamic platforms that simulate biomechanical forces during gamete and embryo development, bridging the gap between static culture systems and the in vivo reproductive tract. Another ongoing effort involves mapping how emerging contaminants–particularly microplastics–disrupt reproductive processes across species, with an emphasis on translating these findings to improve public and environmental health policies. Ultimately, our lab is committed to redefining reproductive modelling by merging developmental biology, materials science, and translational technologies to benefit medicine, agriculture, and conservation.

Embryo implantation within a 3D endometrial model.

Selected Publications

(1) Ribes Martinez E, Franko Y, Franko R, Ferronato GA, Viana AES, Windenbach E, Stoeckl JB, Fröhlich T, **Ferraz MAMM** (2025). **Developing and characterising decellularized extracellular matrix hydrogels to biofabricate female reproductive tissues. Acta Biomater.** 196:152-170.

(2) Franko R and **Ferraz MAMM** (2024). OoTrap: **Enhancing Oocyte Collection and Maturation with a Field Deployable Fluidic Device. Lab Chip.** 25(2):187-200.

(3) Franko R, Franko Y, Ribes Martinez E, Ferronato GA, Heinzelmann I, Grechi N, Devkota S, Fontes PK, Coeti R, Oshiro TSI, **Ferraz MAMM** (2024). **Mechanical Properties of Native and Decellularized Reproductive Tissues: Insights for Tissue Engineering Strategies. Sci Rep.** 14(1):7347.

(4) Grechi N, Franko R, Rajamaran R, Stöckl JB, Traphoff T, Dieterle SJ, Frölich T, Noonan MJ, **Ferraz MAMM.** (2023). **Microplastics are present in women's and cows' follicular fluid and polystyrene microplastics compromise bovine oocyte function in vitro. eLife.** 12:RP86791.

Selected Awards and Honors

2020 Sofja Kovalevskaja Award

Klaus Förstemann

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Since 2006	Professor, LMU München, Germany
2003 - 2006	Postdoc University of Massachusetts Medical School, Worcester, USA
2002 - 2003	Postdoc Swiss Institute of Cancer Research, Lausanne, Switzerland
1998 - 2002	PhD Swiss Institute of Cancer Research, Lausanne, Switzerland

Biology of Non-Coding RNAs

Goals and Impacts for Society

Non-coding RNAs are major players in the regulation of gene expression and defense against external as well as internal pathogens. We study the biogenesis and function of short interfering RNAs (siRNAs) and microRNAs (miRNAs) to gain a fundamental understanding of when, why and how a small RNA response is triggered to neutralize an invader or to compensate a change in the environment. One focus is the somatic surveillance of transposable elements, a parasitic form of DNA that can be found in the genomes of all organisms. This response is mechanistically and evolutionarily related to the anti-viral action of siRNAs, which we can study in part through experimental mimics of infection. Insects rely heavily on the resilience that small RNAs provide and the corresponding biochemical pathways are highly active, thus facilitating experimental access. We therefore use the fruit fly Drosophila *melanogaster* as a model system.

The choice of our model organism is, however, not only due to its advantages for our experiments: Many insects are disease vectors. For example, the malaria mosquito Anopheles *gambiae* is the deadliest animal in Africa. Many of the transmitted diseases are still limited by the habitat boundaries of their animal vectors. But due to climate change, some vector species have by now established resident populations in the temperate zones of Europe. This may eventually lead to a new infectious disease burden to our society. A profound understanding of arthropod biology and immunity will then become a pressing medical need.

Research Highlights

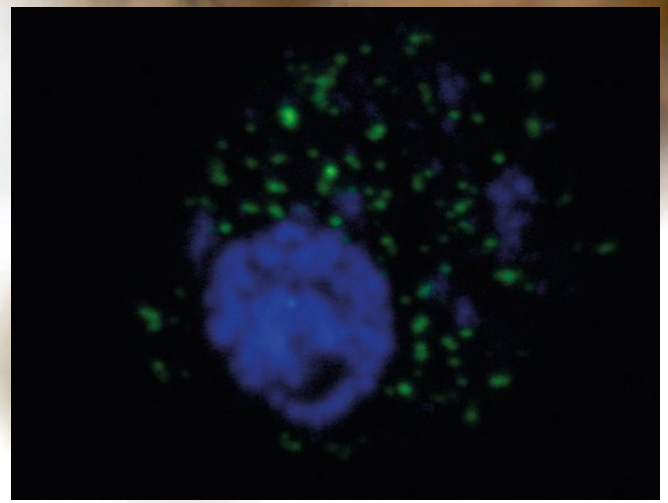
We discovered that a DNA double-strand break can trigger a small RNA response in *Drosophila*. According to the enzymes involved in their biogenesis and the repressive activity they convey the DNA break fortuitously triggers the response that we constitutively observe for transposable elements. While small RNAs play no local role in DNA repair, the phenomenon is an extraordinary tool for mechanistic studies of genome surveillance and the establishment of a persistent equilibrium between pathogen virulence and host defense. Combined with the CRISPR-cas "toolshed", we have temporal control and the necessary genomic precision to elucidate cis-acting elements that mark a transcript as foreign and trigger an siRNA response. For example, small RNA generation is substantially stimulated by stalled spliceosomes on the transcript that is affected by a DNA break.

Double-stranded RNA (dsRNA) of exogenous or endogenous origin is an essential intermediate during siRNA biogenesis and we have a long-standing interest in how this molecular species is generated, transported and processed by the biogenesis machinery. In particular, the dynamic interaction of proteins with dsRNA can be studied with a combination of biochemical, biophysical and genetic tools. Interaction of RNA with proteins can lead to spontaneous formation of sub-cellular compartments with high concentration. These membraneless organelles can be described with the physicochemical principles of phase-separation and they support efficient and specific processing among their constituents. To understand their role in siRNA biogenesis, we combined evolutionary analysis and *in vitro* biophysical measurements with observations in living cells to characterize an enigmatic disordered domain in *Drosophila Ago*2.

Future Directions

We are building the mechanistic link between stalled splicing and antisense transcription triggered by a DNA break. This is the key event that triggers dsRNA formation and thus siRNA biogenesis. Furthermore, the role of membraneless organelles in siRNA biogenesis and, ultimately, viral defense are important questions to address. For this, we will also employ homologous proteins from disease vectors such as Anopheles *gambiae* or *Aedes aegypti* and examine their properties and functionality in the convenient and safe context of our fruit flies.

Different shades of the eye pigmentation are harnessed as reporters for the efficiency of gene silencing as a function of genetic and genomic modifications.



RNA silencing condensates in the cytoplasm of cultured Drosophila cells

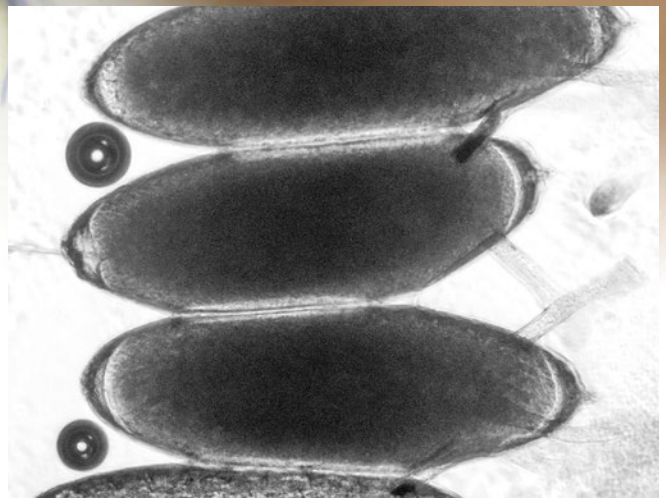
Selected Publications

(1) Hipp C, Mussgnug S, Choudhary P, Kang H-S, Asami S, Sastre J, Donau C, Böttcher R, Gemmecker G, Boekhoven J, **Förstemann K***, Sattler M* (2025). **Molecular mechanisms of biomolecular condensate formation in Drosophila melanogaster siRNA biogenesis. Nucleic Acids** Res. 2025 (in press)

(2) Böttcher R, Schmidts I, Nitschko V, Duric P, **Förstemann K** (2022). **RNA polymerase II is recruited to DNA double-strand breaks for dilncRNA transcription in Drosophila. RNA Biol.** 2022;19(1):68-77.

(3) Merk K, Breinig M, Böttcher R, Krebs S, Blum H, Boutros M, **Förstemann K** (2017). **Splicing stimulates siRNA formation at Drosophila DNA double-strand breaks. PLoS Genet.** 13(6):e1006861.

(4) Michalik KM, Böttcher R, **Förstemann K** (2012). **A small RNA response at DNA ends in Drosophila. Nucleic Acids Res.** 40(19):9596-603.



Microinjection for genome editing of Drosophial embryos

Karl-Peter Hopfner

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Since 2015	Director of the Gene Center
Since 2007	Professor and Chair, Gene Center and Department of Biochemistry, LMU
2001-2007	Tenure-Track Professor, Gene Center, LMU
1998-2001	Postdoc at The Scripps Research Institute, USA
1997	PhD Max-Planck-Institute for Biochemistry and Technical University Munich

Structural Genome Biology

Goal and Impacts for Society

The maintenance of the genetic information is a fundamental process in all of life. Genome instability due to DNA damage or pathogenic nucleic acids are major causes for cancer and immune related diseases. Using structural biology and protein science, we aim at revealing how cells shape, defend and repair their genomic information. We combine basic science with translational research to understand the molecular basis of human disease and contribute to innovative approaches for cancer immunotherapy.

Research Highlights

Many human diseases originate from DNA damage and all cells must maintain the integrity of the genome to remain healthy and respond to threats caused by damaged or pathogenic nucleic acids. Cells possess powerful protein machineries that sense and signal the presence of damaged chromosomes, or foreign nucleic acids, and trigger host responses such as DNA damage response and the antiviral interferon response.

To reveal the underlying mechanisms and molecular principles of sensing, thresholding, amplification and response to genomic stress we use cryo-electron microscopy combined with biochemistry and molecular biology. We determined the structures and mechanisms of central macromolecular complexes involved in DNA double-strand break sensing, chromatin remodelling and innate immune DNA sensing. These efforts have provided multiple important conceptual advances and key insights in the past years.

The Schlafen (SLFN) family of proteins is an emerging family of nucleic acid sensors that link various, still largely unknown, stress signals to induce ribosome mediated apoptosis by cleaving tRNAs. Among these, SLFN11 detects single-stranded DNA. We played a pioneering role in elucidated the structural analysis of Schlafen proteins, including SFLN11, using cryo-EM and biochemical analysis. Our results provide key insights into the mode of activation, DNA sensing, regulation and tRNA cleavage activity by SFLN11 (2).

In a major advance in the chromatin remodeling field, we revealed how chromatin remodellers such as INO80 and related enzymes such as Mot1 mobilize nucleosomes and other protein:DNA complexes. In an collaborative effort, we extenced remodelling mechanism to non-canonical nucleosomal particls such as hexasomes (3). Our findings define how ATP-driven chromatin remodeling by INO80 can adapt to noncanonical nucleosome conformations, offering insights into the role of this complex in DNA repair, replication, and transcription.

To understand the early steps of DNA double-strand break repair, which involve DNA break detection and tethering, we determined the cryo-EM structure of the Mre11-Rad50-Nbs1 (MRN) complex (4). These findings provided a basis for the evolutionary conserved sensor of DNA double-strand breaks and elucidate how MRN facilitates DNA end tethering activities through fromation of condensate-like assemblies.

We also could reveal how the innate immune DNA sensor is prevented from auto-activation by chromatin, despite its localization in the nucleus. We found that cGAS binds tightly to the nucleosome acid patch, which sterically prevents the formation of active DNA-bound dimers. Our results provides a detailed structural mechanism for how self-DNA is tolerated in the nucleus and how auto-immune activation is prevented. Finally, we developed new formats for multispecific antibodies, with the goal of combining specific tumor targeting with tumor-redirected immune checkpoint blockade and activation of anti-tumor immune cells.

Future Directions

Looking ahead, we will investigate how DNA break sensors, chromatin remodelers, and innate immune effectors function within chromatin-like contexts. We will employ cryo-electron microscopy and cryo-electron tomography, integrated with advanced molecular biology and biochemical reconstitution, to reconstitute and structurally characterize dynamic genome surveillance complexes. These studies will require combinations of complex biochemical reconstitution of molecular assemblies and advanced image analysis.

Selected Publications

(1) Fan Y, Kuybu F, Cui H, Lammens K, Chen J-X, Kugler M, Jung C, **Hopfner KP** (2025). **Structural basis for DNA double-strand break sensing by human MRE11-RAD50-NBS1 and its TRF2 complex BioRXiv, Nat Commun.** accepted. doi: 10.1101/2025.03.14.643254.

(2) Kugler M, Metzner FJ, Witte G, **Hopfner KP,** Lammens K. **Phosphorylation-mediated conformational change regulates human SLFN11**. Nat Commun. 3;15(1):10500. doi: 10.1038/s41467-024-54833-7, (2024)

(3) Zhang M, Jungblut A, Kunert F, Hauptmann L, Hoffmann T, Kolesnikova O, Metzner F, Moldt M, Weis F, DiMaio F, **Hopfner KP**, Eustermann S (2023). **Hexasome-INO80 complex reveals structural basis of noncanonical nucleosome remodeling.** Science 381(6655):313-319. doi: 10.1126/science.adf6287.

(4) Rotheneder M, Stakyte K, van de Logt E, Bartho JD, Lammens K, Fan Y, Alt A, Kessler B, Jung C, Roos WP, Steigenberger B, **Hopfner KP** (2023). **Cryo-EM structure of the Mre11-Rad50-Nbs1 complex reveals the molecular mechanism of scaffolding functions.** Mol Cell. S1097-2765(22)01138-8. doi: 10.1016/j.molcel.2022.12.003.

Selected Awards and Honors

2022 Visiting Scholar University of California, San Diego

Veit Hornung

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Since 2015	Professor and Chair, Gene Center and Department of Biochemistry, LMU
2014-2015	Director, Institute of Molecular Medicine, University Hospital, University of Bonn
2008-2013	Professor of Clinical Biochemistry (W2), Institute for Clinical Chemistry and Clinical Pharmacology, University Hospital, University of Bonn
2006-2008	Postdoc at the University of Massachusetts Medical School in Worcester, USA
2003-2006	Research fellow / Postdoctoral research fellow in the Division of Clinical Pharmacology at the University, Hospital Munich, LMU
2004	Doctorate (Dr. med.), University of Munich, LMU

Innate Immunity

Goals and Impacts for Society

The ability of the immune system to detect invading microbes while avoiding responses to self is essential for maintaining health. Failures in this discrimination can lead to infections, chronic inflammation, or autoimmunity. Our research focuses on uncovering how the innate immune system senses and responds to foreign entities. By combining molecular immunology with genetics, biochemistry, and structural approaches, we aim to define fundamental principles of immune recognition. Our goal is to provide mechanistic insights into host–pathogen interactions and contribute to the development of novel strategies for infection control and immune-based therapies.

Research Highlights

Cells of the innate immune system detect foreign or altered-self nucleic acids to initiate antiviral and antibacterial responses. A key challenge is how cells distinguish pathogenic nucleic acids from self-derived molecules to activate immunity without triggering autoimmunity. We aim to uncover the molecular mechanisms by which the innate immune system detects and distinguishes pathogenic nucleic acids and bacterial components from self, focusing on how immune recognition is initiated and controlled at critical intracellular sensing sites.

Toll-like receptors (TLRs), residing in the endolysosomal compartment, are critical for detecting pathogen-derived nucleic acids. However, they cannot detect full-length RNA directly; ligands must be enzymatically processed. We recently discovered how endolysosomal nucleases generate RNA ligands for TLR7. TLR7 recognizes two classes of RNA degradation products via distinct binding pockets. We found that RNase T2 and the 5′ exonucleases PLD3 and PLD4 sequentially process RNA to generate these fragments. PLD enzymes release 2′,3′-cyclic guanosine monophosphate to engage pocket 1 and generate pyrimidine-rich fragments for pocket 2. Structural studies showed PLD3/4 form homodimers with key ligand-binding interfaces, and disease-associated mutations disrupt this configuration and function.

We further explored how post-transcriptional RNA modifications influence immune sensing. Using biochemical and cellular assays, we showed that pseudouridine-containing RNA–including medically used N1-methylpseudouridine-modified RNA–evades processing by RNase T2 and PLDs. As a result, TLR7 and TLR8 fail to recognize such RNAs, explaining immune tolerance to self-RNA and the success of modified mRNA in therapy. Notably, N1-methylpseudouridine resists enzymatic degradation but can still activate TLR8 directly.

We also explored how the detection of cytosolic RNA activates the inflammasome cascade, triggering inflammation and cell death. In screening viruses, we found that those generating dsRNA robustly activate the NLRP1 inflammasome. Defined ligands showed that long dsRNA triggers NLRP1 activation. Biochemical analyses revealed NLRP1 binds dsRNA, causing a conformational shift and inflammasome assembly–identifying NLRP1 as a direct sensor of viral dsRNA.

Extending non-self recognition beyond nucleic acids, we also studied bacterial sensing. Peptidoglycan contains muramyl dipeptide (MDP), recognized by cytosolic receptor NOD2. A forward genetic screen identified N-acetylglucosamine kinase (NAGK) as essential for phosphorylating MDP to 6-O-phospho-MDP. Macrophages lacking NAGK fail to respond to MDP, revealing a novel metabolic step in microbial sensing.

Together, our work defines molecular principles governing recognition, enzymatic processing, and immune activation by foreign nucleic acids and microbial ligands, offering insights into infection, inflammation, and immunotherapy.

Future Directions

We aim to further understand how the innate immune system distinguishes self from non-self nucleic acids. We will investigate how modifications, metabolic processing, and compartmentalization contribute to immune tolerance. Integrating structural biology, biochemistry, and genetics, we will reveal how nucleic acid sensors are specifically activated by foreign ligands. These studies will provide mechanistic insight into innate immune recognition and inform targeted immunotherapies and vaccine strategies.

Selected Publications

(1) Bérouti M, Lammens K, Heiss M, Hansbauer L, Bauernfried S, Stöckl J, Pinci F, Piseddu I, Greulich W, Wang M, Jung C, Fröhlich T, Carell T, Hopfner KP, **Hornung V** (2024). **Lysosomal endonuclease RNase T2 and PLD exonucleases cooperatively generate RNA ligands for TLR7 activation. Immunity.** 57(7):1482-1496.e8.

(2) Bérouti M, Wagner M, Greulich W, Piseddu I, Gärtig J, Hansbauer L, Mueller-Hermes C, Heiss M, Pichler A, Tölke AJ, Witte G, Hopfner KP, Anz D, Sattler M, Carell T, **Hornung V** (2025). **Pseudouridine RNA avoids immune detection through impaired endolysosomal processing and TLR engagement. Cell.** 188:10.1016/j.cell.2025.05.032.

(3) Bauernfried S, Scherr MJ, Pichlmair A, Duderstadt KE, **Hornung V** (2021). **Human NLRP1 is a sensor for double-stranded RNA. Science.** 371(6528):eabd0811.

(4) Stafford CA, Gassauer AM, de Oliveira Mann CC, Tanzer MC, Fessler E, Wefers B, Nagl D, Kuut G, Sulek K, Vasilopoulou C, Schwojer SJ, Wiest A, Pfautsch MK, Wurst W, Yabal M, Fröhlich T, Mann M, Gisch N, Jae LT, **Hornung V** (2022). **Phosphorylation of muramyl peptides by NAGK is required for NOD2 activation. Nature.** 609(7927):590-596.

Selected Awards and Honors

2025 Louis-Jeantet Prize 2024

2021 ERC Advanced Grant

Lucas Jae

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Since 2023	Professor (W2), Gene Center and Department of Biochemistry, LMU
2019-2023	Tenure-Track Professor, Gene Center and Department of Biochemistry, LMU
2017	Independent Group Leader, Gene Center, LMU
2016	Postdoc at The Netherlands Cancer Institute, Amsterdam
2015	PhD, The Netherlands Cancer Institute, Amsterdam and Utrecht University

Genetic Dissection of Human Cellular Biology

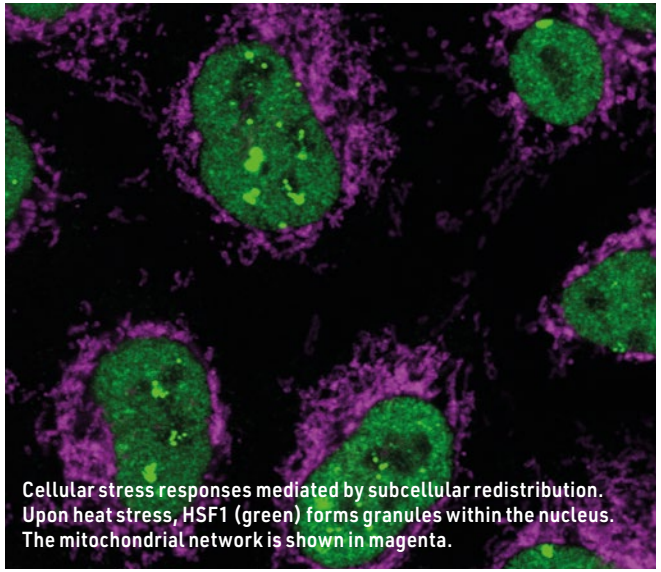
Goals and Impacts for Society

Age-related diseases–ranging from neurodegeneration to cardiovascular dysfunction–will be a defining biomedical challenge of the 21st century. A growing gap between lifespan and health span underscores the urgent need to understand the molecular basis of cellular decline. Mitochondrial dysfunction has emerged as a hallmark of aging and numerous prevalent age-related diseases. Understanding how cells monitor mitochondrial function and respond to its deterioration is essential for identifying therapeutic targets and for designing interventions that restore cellular homeostasis. The Jae lab aims to uncover the molecular circuitry that connects mitochondrial dysfunction to broader cellular stress responses, with a particular focus on the recently uncovered OMA1-DELE1-HRI pathway and its biomedical implications.

Research Highlights

In recent years, we have focused on deciphering how human cells recognize and respond to mitochondrial dysfunction. Through genome-wide genetic screening, synthetic biology, and high-resolution phenotyping, we identified the OMA1-DELE1-HRI axis as a central mitochondrial surveillance pathway in humans. This pathway connects stress in the mitochondrial compartment to the cytosolic integrated stress response (ISR), which rewires gene expression to promote adaptation. Our discovery that the protein DELE1 is cleaved by the stress-activated protease OMA1 and subsequently activates the kinase HRI established the first mechanistic link between mitochondrial damage and translational control in human cells (Nature, 2020) and serves as conceptual foundation for a new area of stress signaling research.

We subsequently unraveled that DELE1 acts as a sensor of mitochondrial protein import fidelity through its perpetual de novo synthesis and destruction inside the organelle, thereby effectively integrating a wide range of biomedically relevant mitochondrial perturbations (Nat Commun, 2022; Cell Mol Life Sci, 2021). Notably, these span processes as diverse as membrane polarization, mitochondrial energetics, protein misfolding and iron homeostasis (J Clin Invest, 2022; Mol Cell, 2023). These insights suggest that the OMA1-DELE1-HRI circuit functions as a general-purpose surveillance mechanism for mitochondrial integrity in the human system.

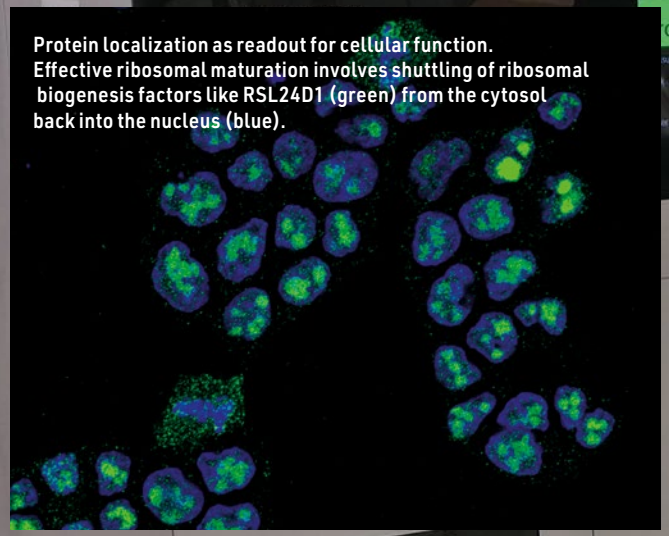


Cellular stress responses mediated by subcellular redistribution. Upon heat stress, HSF1 (green) forms granules within the nucleus. The mitochondrial network is shown in magenta.

Many of these discoveries were fueled by our unique platform for functional genomics established at the Gene Center. In a collaborative study, we harnessed its power to identify a novel enzymatic reaction essential for the cellular inflammatory response to bacterial cell wall structures (Nature, 2022).

Future Directions

Our current efforts are aimed at understanding how mitochondrial surveillance pathways intersect with other cellular stress responses and how they can be leveraged therapeutically. We are particularly interested in mapping the genetic dependencies that govern cell fate in the context of organelle dysfunction and how stress signals propagate between cells. Ultimately, we seek to define intervention points that may allow the selective reprogramming of stress responses in human disease contexts.



Protein localization as readout for cellular function.
Effective ribosomal maturation involves shuttling of ribosomal biogenesis factors like RSL24D1 (green) from the cytosol back into the nucleus (blue).

Selected Publications

(1) Sekine Y, Houston R, Eckl EM, Fessler E, Narendra DP, **Jae LT,** Sekine S (2023). **A mitochondrial iron-responsive pathway regulated by DELE1. Mol Cell.** 83(12):2059-2076.e6.

(2) Fessler E†, Krumwiede L, **Jae LT†** (2022). **DELE1 tracks perturbed protein import and processing in human mitochondria. Nat Commun.** 13(1):1853.

(3) Eckl EM, Ziegemann O, Krumwiede L, Fessler E, **Jae LT†** (2021). **Sensing, signaling and surviving mitochondrial stress. Commissioned review. Cell Mol Life Sci.** 78(16):5925-5951.

(4) Fessler E, Eckl EM, Schmitt S, Mancilla IA, Meyer-Bender MF, Hanf M, Philippou-Massier J, Krebs S, Zischka H, **Jae LT†** (2020). **A pathway coordinated by DELE1 relays mitochondrial stress to the cytosol. Nature.** 579(7799):433-437.

Selected Awards and Honors

2023 Vallee Scholar Award of the Vallee Foundation

2022 Alfried Krupp Award of the Alfried Krupp von Bohlen und Halbach Foundation

2021 Life Sciences Bridge Award of the Aventis Foundation

Oliver T. Keppler

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Since 2015	Director of the Max von Pettenkofer Institute and Chair of Virology, LMU Munich
2012-2015	Director of the Institute of Medical Virology, Chair of Virology, Johann-Wolfgang-Goethe University Frankfurt am Main
2005	Habilitation in "Experimental Virology", University of Heidelberg
1997-2002	Postdoctoral Fellow, Department of Applied Tumor Virology at the German Cancer Research Center Heidelberg and Gladstone Institute of Virology and Immunology, University of California, San Francisco
1995	Medical Degree, University of Heidelberg

HIV, SARS-CoV-2,
and Chemoresistance Mechanisms

Goals and Impacts for Society

The Keppler laboratory investigates fundamental principles of virus-host interactions, in particular for the pandemic pathogens HIV and SARS-CoV-2, as well as specific mechanisms of resistance to chemotherapy in different malignancies. Using state-of-the-art technology and primary model systems, we seek to unravel the immunodestruction induced by HIV and the ability of the virus to infect and establish latency in resting CD4 T cells. In the fight against SARS-CoV-2, we are testing novel antiviral approaches and aiming to identify drugable cellular pathways by genetic and high-throughput screening approaches. At the interface of virology and oncology we are studying the SAMHD1-dependent resistance to nucleoside analog-based chemotherapy and developing SAMHD1-targeted strategies to expand therapeutic options for hard-to-cure hematological and solid tumors. We are also interested in the surveillance of wastewater and patient material to monitor the emergence and dynamics of virus infections on a population level.

Research Highlights

HIV reservoir and immunodestruction
HIV/AIDS is one of the most devastating pandemics in recorded history and, even nowadays, the fourth-biggest global killer. Currently available pharmacotherapies can only partly control but not cure this immunodestructive viral infection. Our laboratory seeks to better understand the pathological interplay of HIV with the host's immune system and its target cells, including resting CD4 T cells, with the goal of providing new approaches for prophylaxis and therapy.

SARS-CoV-2: Host cell factors and antiviral drugs
In the fight against COVID-19 we are testing novel antiviral approaches to reduce viral replication and disease burden in ex vivo models and in vivo, including peptide-linked siRNAs. Furthermore, we are aiming to identify cellular pathways involved in SARS-CoV-2 replication and virus-induced cell death. Using newly developed cell models, we are employing unbiased genetic survival screening to determine important host cell factors, and high-throughput screening to identify small molecules as potential candidates for drug development.

Epidemiology of viral infections and surveillance
Understanding the epidemiology of viral infections and surveilling their prevalence is key to uncovering outbreaks, estimating their impact on the population, and designing strategies to protect vulnerable individuals. In larger cohort studies, our laboratory is researching risk factors for viral infections and potential benefits of countermeasures, including vaccination. Furthermore, we are developing means for the surveillance of a multitude of viral infections such as high-throughput PCR testing, and wastewater surveillance.

Overcoming SAMHD1-dependent resistance to chemotherapeutics
Nucleoside analogs are crucial for the chemotherapy of malignant diseases. Treatment success, however, is variable and often unpredictable, in part due to resistance against chemotherapeutics. Thus, the identification of biomarkers to guide treatment decisions and personalized therapeutic interventions to overcome chemoresistance are urgently needed. Our laboratory discovered the protein SAMHD1 to be a major mediator of chemoresistance, especially in acute myeloid leukemia. Currently, we are (i) deepening our understanding of the physiological and pathophysiological functions of SAMHD1, (ii) exploring the impact of this protein in mediating chemoresistance in other hematological and solid tumors, and (iii) investigating different strategies to modulate SAMHD1 expression and activity.

Future Directions

Finding strategies to combat epidemics and pandemics, including HIV/AIDS and COVID-19, is more relevant than ever, and may, indeed become even more pressing due to climate change and other global circumstances. Similarly, there is an unmet need for new treatment strategies against cancer, especially since the prevalence of malignant diseases is increasing worldwide. On a large scale, by surveilling virus infections and researching their epidemiology and, on a small scale, by investigating virus-host interactions and developing approaches to overcome chemoresistance in tumor cells, our research may contribute to the development of solutions to these high-priority demands.

Selected Publications

(1) Albanese M, Chen HR, Gapp M, Muenchhoff M, Yang HH, Peterhoff D, Hoffmann K, Xiao Q, Ruhle A, Ambiel I, Schneider S, Mejías-Pérez E, Stern M, Wratil PR, Hofmann K, Amann L, Jocham L, Fuchs T, Ulivi AF, Besson-Girard S, Weidlich S, Schneider J, Spinner CD, Sutter K, Dittmer U, Humpe A, Baumeister P, Wieser A, Rothenfusser S, Bogner J, Roider J, Knolle P, Hengel H, Wagner R, Laketa V, Fackler OT, **Keppler OT** (2024). **Receptor transfer between immune cells by autoantibody-enhanced, CD32-driven trogocytosis is hijacked by HIV-1 to infect resting CD4 T cells. Cell Rep Med**. 5(4):101483.

(2) Keppler-Hafkemeyer A, Greil C, Wratil PR, Shoumariyeh K, Stern M, Hafkemeyer A, Ashok D, Hollaus A, Lupoli G, Priller A, Bischof ML, Ihorst G, Engelhardt M, Marks R, Finke J, Bertrand H, Dächert C, Muenchhoff M, Badell I, Emmerich F, Halder H, Spaeth PM, Knolle PA, Protzer U, von Bergwelt-Baildon M, Duyster J, Hartmann TN, Moosmann A, **Keppler** OT (2023). **Potent high-avidity neutralizing antibodies and T cell responses after COVID-19 vaccination in individuals with B cell lymphoma and multiple myeloma. Nat Cancer** 4(1):81-95.

(3) Wratil PR, Stern M, Priller A, Willmann A, Almanzar G, Vogel E, Feuerherd M, Cheng CC, Yazici S, Christa C, Jeske S, Lupoli G, Vogt T, Albanese M, Mejías-Pérez E, Bauernfried S, Graf N, Mijocevic H, Vu M, Tinnefeld K, Wettengel J, Hoffmann D, Muenchhoff M, Daechert C, Mairhofer H, Krebs S, Fingerle V, Graf A, Steininger P, Blum H, Hornung V, Liebl B, Überla K, Prelog M, Knolle P, **Keppler OT**, Protzer U (2022). **Three exposures to the spike protein of SARS-CoV-2 by either infection or vaccination elicit superior neutralizing immunity to all variants of concern. Nat Med**. 28(3):496-503.

(4) Albanese M, Ruhle A, Mittermaier J, Mejías-Pérez E, Gapp M, Linder A, Schmacke NA, Hofmann K, Hennrich AA, Levy DN, Humpe A, Conzelmann KK, Hornung V, Fackler OT, **Keppler OT** (2022). **Rapid, efficient and activation-neutral gene editing of polyclonal primary human resting CD4⁺ T cells allows complex functional analyses. Nat Methods.** 19(1):81-89.

Selected Awards and Honors

2024 Adrian Ruhle: LMU Research Award for Excellent Students

2022 Paul R. Song Wratil, Marcel Stern, Alina Priller, Oliver T. Keppler: Rolf-Becker-Preis

2022 Oliver T. Keppler: Bavarian Order of Merit

Christoph Klein

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Since 2011	Director, Department of Pediatrics, LMU Munich of Biochemistry, LMU
2008-2011	Professor and Chair, Department of Pediatric Hematology/Oncology, Hannover Medical School
2000-2008	Tenure-Track Professor, Department of Pediatric Hematology/Oncology, Hannover Medical School
1995-2000	Clinical Fellow and Instructor, Boston Children's Hospital and Dana Farber Cancer Institute, Harvard Medical School

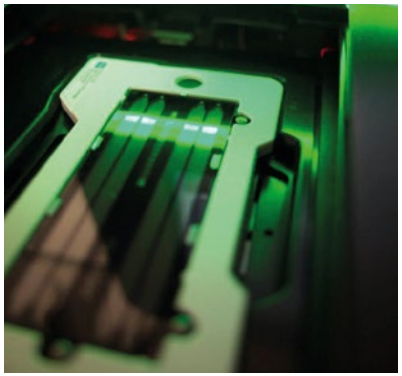
Precision Medicine – from Genes to Therapies

Goals and Impacts for Society

Precision medicine aims to understand diseases at the molecular and genetic levels to design a new era of molecular diagnosis, treatment, and prevention. By uncovering disease mechanisms, it enables the development of targeted therapies and personalized medicine based on an individual's genetic profile. This research can improve health outcomes, reduce side effects, and lower healthcare costs through early detection and precise intervention. Societal impacts include longer life expectancy, better quality of life, and a more efficient healthcare system, positioning molecular medicine as a key driver of future medical innovation and public health.

Research Highlights

Our mission is to uncover the molecular principles of blood and immune system disorders by studying the genetic diseases in children and adolescents. We use in vitro and in vivo models to validate novel genetic causes and develop cutting-edge treatments, with a strong focus on gene and cell therapy. Children with Inborn Errors of Immunity (IEI) may present with infec–tions, autoimmune or autoinflammatory conditions, allergies or cancer. Our group has elucidated several dozens of novel IEI. We (co-)coordinate several global networks to study IEI (e.g. VEO-IBD consortium on very-early onset inflammatory bowel diseases). In parallel to our focus to improve precision diagnostics, we also develop new strategies for precision therapies. Recently, we devised a novel proprietary technology to grow human bone marrow organoids, a valuable tool to test gene therapies for hematological and immune disorders.



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Future Directions

Molecular medicine is reshaping how we study and treat disease. Future research will integrate multi-omics data, single-cell technologies, and computational models to discover new biomarkers and therapies. Advances in gene editing, RNA-based treatments, and personalized cellular approaches will drive precision medicine. We currently assess a new generation of combinatorial recombinant antibodies as well as novel small molecules for immune interventions in cancer and inflammatory disorders. Global collaboration and data sharing will accelerate clinical translation. The result will be more accurate diagnostics, effective therapies, and improved outcomes across diverse diseases.

Selected Publications

(1) Frenz-Wiessner S, Fairley SD, Buser M, Goek I, Salewskij K, Jonsson G, Illig D, Zu Putlitz B, Petersheim D, Li Y, Chen PH, Kalauz M, Conca R, Sterr M, Geuder J, Mizoguchi Y, Megens RTA, Linder MI, Kotlarz D, Rudelius M, Penninger JM, Marr C, **Klein C**. (2024). **Generation of complex bone marrow organoids from human induced pluripotent stem cells. Nat Methods.** 21(5):868-881.

(2) Li Y, Yu Z, Schenk M, Lagovsky I, Illig D, Walz C, Rohlfs M, Conca R, Muise AM, Snapper SB, Uhlig HH, Garty BZ, **Klein C**, Kotlarz D. (2023). **Human MD2 deficiency-an inborn error of immunity with pleiotropic features. J Allergy Clin Immunol**. 151(3):791-796.e7.

(3) Linder MI, Mizoguchi Y, Hesse S, Csaba G, Tatematsu M, Łyszkiewicz M, Zietara N, Jeske T, Hastreiter M, Rohlfs M, Liu Y, Grabowski P, Ahomaa K, Maier-Begandt D, Schwestka M, Pazhakh V, Isiaku AI, Briones Miranda B, Blombery P, Saito MK, Rusha E, Alizadeh Z, Pourpak Z, Kobayashi M, Rezaei N, Unal E, Hauck F, Drukker M, Walzog B, Rappsilber J, Zimmer R, Lieschke GJ, **Klein C.** (2023). **Human genetic defects in SRP19 and SRPRA cause severe congenital neutropenia with distinctive proteome changes. Blood**. 141(6):645-658.

(4) Fan Y, Murgia M, Linder MI, Mizoguchi Y, Wang C, Łyszkiewicz M, Zietara N, Liu Y, Frenz S, Sciuccati G, Partida-Gaytan A, Alizadeh Z, Rezaei N, Rehling P, Dennerlein S, Mann M, **Klein C**. (2022). **HAX1-dependent control of mitochondrial proteostasis governs neutrophil granulocyte differentiation. J Clin Invest.** 132(9):e153153

Selected Awards and Honors

2023 Committee Member Pediatric Hematology (American Society of Hematology)

2021 Speaker Munich Site, German Center for Child and Adolescent Health (DZKJ), BMBF

Johanna Klughammer

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Since 2021	Tenure-track professor, Gene Center and Department of Biochemistry, LMU
2018-2021	Postdoc, Broad Institute of MIT and Harvard, USA
2017-2018	Postdoc, CeMM & Medical University Vienna, Austria
2012–2017	PhD, CeMM & Medical University Vienna, Austria

Systems Immunology

Goals and Impacts for Society

At the intersection of computational genomics, systems biology, and biomedicine we use high-dimensional data and computational methods including machine learning to explore how genes and their regulation influence biomedically relevant processes. To understand cellular systems in health and disease it is crucial to study how individual cells function and how they interact within tissues, enabled in particular through single-cell and spatial omics. These technologies allow us to analyze gene activity at the level of individual cells while considering their spatial and cellular context in tissues - essential for understanding complex multicellular processes like cancer progression, tissue development, and immune responses. With a focus on immunological processes which are ubiquitously relevant for multicellular homeostasis, we work towards deeper insights into medically relevant biological systems, more effective treatments, and generally advances in personalized medicine.

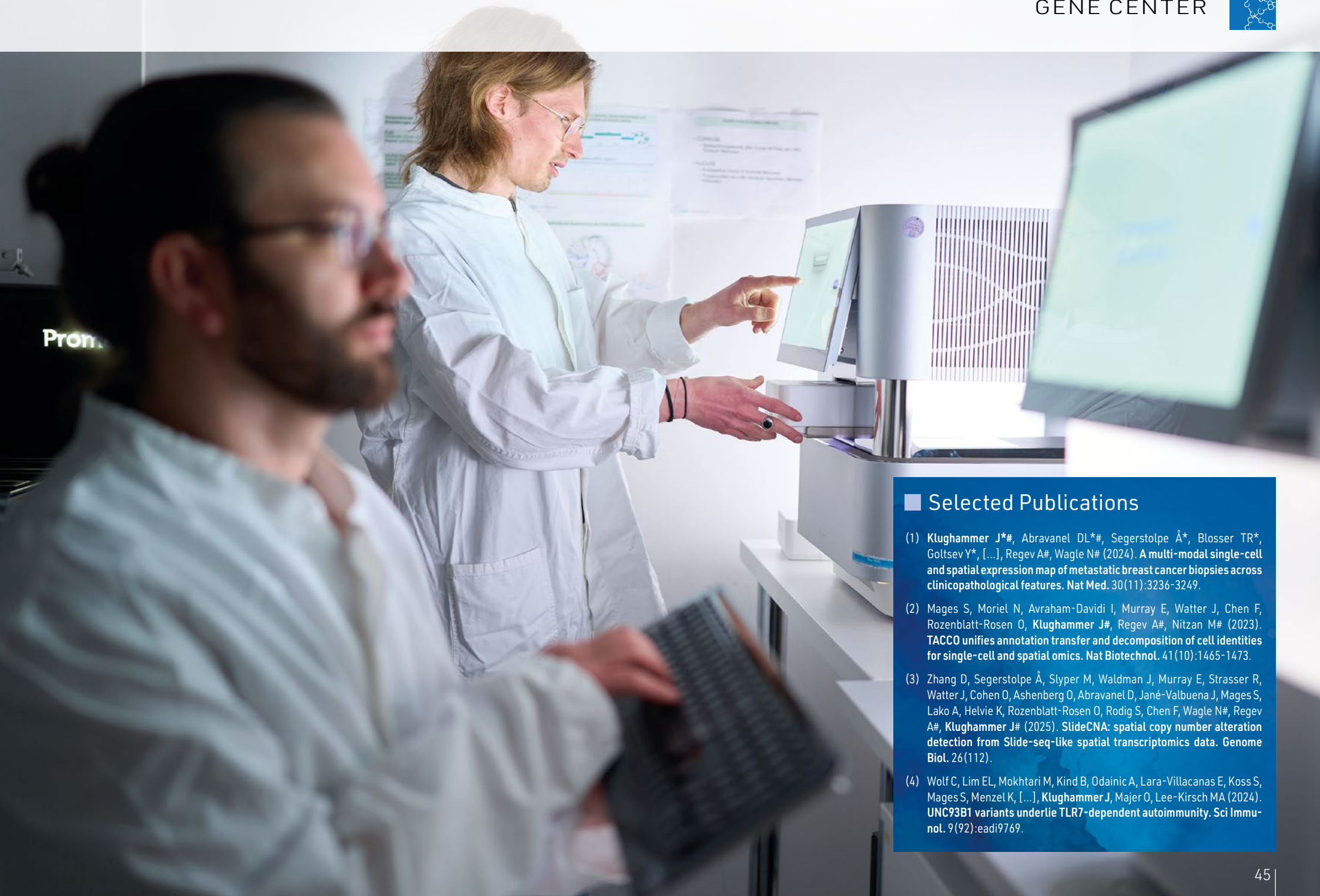
Research Highlights

Information-rich high-dimensional data as generated through single-cell and spatial omics hold immense potential to comprehensively characterize and understand complex cellular systems, but turning these data into insights requires sophisticated computational tools and analysis.

In our work, the development of computational tools often goes hand-in-hand with projects that are primarily driven by biomedical questions. For example, our multi-modal single-cell and spatial transcriptomic analysis of Metastatic Breast Cancer (MBC) biopsies (1) was boosted by co-developing a computational framework (2) which allowed us to systematically integrate and compare different spatial expression profiling approaches across modalities. In this study, we created detailed maps of tumor composition and interactions which highlighted the importance of spatial context in understanding tumor behavior, revealing how metastatic cells adapt to and shape their surroundings. However, as we delved deeper into the spatial data, we encountered a critical gap: existing methods were insufficient for detecting copy number alterations (CNAs) in highly spatially resolved but sparse data such as SlideSeq. This realization prompted us to develop SlideCNA (3), a computational approach that infers spatial CNAs from sparse transcriptomics data at near single-cell resolution. Applying SlideCNA to MBC samples, we identified spatial subclones that were associated with different expression phenotypes. While our research has primarily focused on cancer, our interest in cellular phenotypes extends beyond tumor biology. In a study investigating UNC93B1 variants and their role in TLR7-driven autoimmunity (4), we used scRNAseq and custom analysis to contribute to the characterization of aberrant (immune) pathways across all major immune cell types. This work demonstrated the value of high-dimensional transcriptional characterization for understanding monogenetic immune diseases and shows its potential for the stratification and treatment of patients.

Future Directions

Looking ahead, we aim to further address current challenges around clinical translation of insights gained from high-dimensional 'omics data, with the goal of realizing their enormous potential for the benefit of patients. Specifically, we want to use our experience in stratifying cancer patients into clinically relevant subgroups, to aid the diagnostic process for rare, presumably monogenetic diseases, where too often genetic screens fail to identify the mutated gene. In addition, we will continue to develop computational approaches that help leverage the full potential of single-cell and spatial omics data – in particular addressing new challenges and opportunities around single-molecule resolved data and cross-modality integration.



Selected Publications

(1) **Klughammer J*#**, Abravanel DL*#, Segerstolpe Å*, Blosser TR*, Goltsev Y*, [...], Regev A#, Wagle N# (2024). **A multi-modal single-cell and spatial expression map of metastatic breast cancer biopsies across clinicopathological features. Nat Med.** 30(11):3236-3249.

(2) Mages S, Moriel N, Avraham-Davidi I, Murray E, Watter J, Chen F, Rozenblatt-Rosen O, **Klughammer J#**, Regev A#, Nitzan M# (2023). **TACCO unifies annotation transfer and decomposition of cell identities for single-cell and spatial omics. Nat Biotechnol.** 41(10):1465-1473.

(3) Zhang D, Segerstolpe Å, Slyper M, Waldman J, Murray E, Strasser R, Watter J, Cohen O, Ashenberg O, Abravanel D, Jané-Valbuena J, Mages S, Lako A, Helvie K, Rozenblatt-Rosen O, Rodig S, Chen F, Wagle N#, Regev A#, **Klughammer J#** (2025). **SlideCNA: spatial copy number alteration detection from Slide-seq-like spatial transcriptomics data. Genome Biol.** 26(112).

(4) Wolf C, Lim EL, Mokhtari M, Kind B, Odainic A, Lara-Villacanas E, Koss S, Mages S, Menzel K, [...], **Klughammer J**, Majer O, Lee-Kirsch MA (2024). **UNC93B1 variants underlie TLR7-dependent autoimmunity. Sci Immunol.** 9(92):eadi9769.

Simon Mages

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E-mail	Simon.Mages@lmu.de

Since 2022	Group Leader at the Gene Center, LMU
2020-2022	Postdoc at the Broad Institute of MIT and Harvard, USA
2017-2019	Research scientist at Siemens, Germany / USA
2015-2017	Postdoc at the Jülich Supercomputing Centre, Germany
2015	PhD in Theoretical Physics, University of Regensburg"

Physics of High-Dimensional Biological Data

Goals and Impacts for Society

Data is the petroleum of the information age, in research as well as in industry. To use it we have to come up with ways to refine and transform it into actionable insights. In modern biology the data often lives in a high-dimensional product space spanned by observations like samples or cells and features like genes or proteins. Structures in this data are the key for its understanding. To find, understand, and utilize these structures we develop and apply new data analysis methods combining strategies and inspiration from bioinformatics, high-performance computing, artificial intelligence, and theoretical physics.

Research Highlights

One particularly important type of data in our research is transcriptomics data, i.e. information about the expression of individual genes as defined by the amount of mRNA transcribed from the genes. This can be measured by sequencing-based approaches or in-situ hybridization. The result are mRNA count matrices which give the number of mRNA molecules measured per gene for every observation. An observation can be a bulk tissue sample, a spatially defined location in a tissue, or a single isolated cell from a tissue giving average, spatially resolved, or single cell resolved information about gene expression. In general, all of them only provide partial views about what happens in the tissue as e.g. cell type and state diversity is averaged out, cells are captured in a biased way, or technical constraints place restrictions on the number of genes measured per sample. Yet, all views provide important complementary information about the same underlying system such that the integration of data from those views is necessary to get a more holistic and unbiased understanding of tissues. Additionally, there is a huge and growing corpus of publicly available and well analyzed data, which can be used to accelerate the process from performing a new single cell or spatial transcriptomics experiment to learning about the captured biology. We created a versatile, modular, and efficient computational framework, TACCO (1), to facilitate these tasks. With TACCO researchers can transfer annotations between datasets of different types, e.g. using the annotation in a reference single cell RNA-seq dataset they can get the cell type composition for every pixel of a spatial transcriptomics measurement which often contain contributions of multiple cells per pixel. It can also be used to analyze the spatial correlations in datasets shedding light on tissue structure and interactions between cell types, to stratify samples into transcriptionally defined spatial regions, to perform (spatially informed) enrichment analyses, for data visualization, for transcriptionally informed segmentation and annotation of single-molecule resolved spatial transcriptomics measurements, and to infer developmental trajectories of cells. TACCO has been applied by us and others e.g. for transcriptomic data in the context of mouse models for colorectal cancer (2), for transcriptomics and proteomics data in human metastatic breast cancer (3), but also to investigate more fundamental questions like the division of labor in tissues (4). We are continuously developing this framework and include new methods, models, applications, and modalities.

Future Directions

Count matrices are a very convenient "standard format" for omics data. But there are many preprocessing steps necessary which convert the real raw data into count matrices. In those steps many assumptions are made and information is lost, which diminishes and biases the potential insights to be gained from the data. We will extend our framework towards earlier steps in the data analysis to have access to quantitatively and qualitatively better information. The more we work towards raw data, the more important the optimization of analysis strategies, algorithms and implementations becomes. But in the end, it will enable a more efficient conversion of omics data to biomedically relevant insights.

Selected Publications

(1) Avraham-Davidi I, **Mages S**, Klughammer J, Moriel N, Imada S, Hofree M, Murray E, Chen J, Pelka K, Mehta A, Boland GM, Delorey T, Caplan L, Dionne D, Strasser R, Lalakova J, Niesnerova A, Xu H, Rouault M, Tirosh I, Hacohen N, Chen F, Yilmaz O, Roper J, Rozenblatt-Rosen O, Nitzan M, Regev A. (2025) **Spatially defined multicellular functional units in colorectal cancer revealed from single cell and spatial transcriptomics eLife** 14:RP104815.

(2) Klughammer J, Abravanel DL, Segerstolpe Å, Blosser TR, Goltsev Y, Yi C, Goodwin DR, Sinha A, Ashenberg O, Slyper M, Vigneau S, Jané-Valbuena J, Alon S, Caraccio C, Chen J, Cohen O, Cullen N, DelloStritto LK, Dionne D, Files J, Frangieh A, Helvie K, Hughes ME, Inga S, Kanodia A, Lako A, MacKichan C, **Mages S**, [...] Regev A, Wagle N. (2024). **A multi-modal single-cell and spatial expression map of metastatic breast cancer biopsies across clinicopathological features. Nat Med** 30(11):3236-3249.

(3) **Mages S**, Moriel N, Avraham-Davidi I, Murray E, Watter J, Chen F, Rozenblatt-Rosen O, KlughammerJ , Regev A, Nitzan M. (2023). **TACCO unifies annotation transfer and decomposition of cell identities for single-cell and spatial omics. Nat Biotechnol.** 41(10):1465-1473.

(4) Adler M, Moriel N, Goeva A, Avraham-Davidi I, **Mages S**, Adams TS, Kaminski N, Macosko EZ, Regev A, Medzhitov R, Nitzan M. (2023). **Emergence of division of labor in tissues through cell interactions and spatial cues. Cell Rep**, 30;42(5):112412.

Lukas Milles

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Since 2024	Professor (W2) Gene Center, LMU Munich
Since 2024	Emmy Noether Group Leader, MPI of Biochemistry, Martinsried
2019-2024	Postdoctoral Researcher with David Baker, Institute for Protein Design, University of Washington, Seattle
2014-2018	PhD in Physics/Biophysics with Hermann Gaub, Ludwig-Maximilians-Universität, München

De Novo Protein Design

Goals and Impacts for Society

Our recently established research group (October 2024) works at the intersection of de novo protein design and fundamental biophysics of protein function. We aim to deconstruct biological function by reconstructing such function de novo. Using deep learning-based protein design, we construct new-to-nature proteins that incorporate desired functions to better understand their biological mechanism, in particular those found at the host-pathogen interface. We can rapidly take proteins from an idea to a blueprint on the computer and finally to the wet lab (and back) to study the differences between native and computationally designed protein sequences. Our designs can find applications as biorthogonal, bespoke tools in areas such as biomaterials, immunity and the precise targeting of cellular surface receptors – allowing control over functions beyond what is achievable with natural proteins.

Research Highlights

De novo protein design has experienced a machine learning-fueled revolution, vastly expanding our ability to design complex and functional proteins beyond those found in nature. Among other areas, we are interested in mechanisms at the host-pathogen interface:

Creating synthetic "harpoons" that can covalently target selected epitopes by reconstructing an autocatalytic mechanism from gram-positive pathogens, a potential tool for irreversible, covalent opsonization. Here we aim to rebuild and understand thioester attachment chemistry found both in the human complement system of innate immune response and also (in a case of convergent evolution) gram positive bacteria's tip adhesins.

Designing self-strengthening catch-bonds, counterintuitive protein-protein interactions that bind more tightly under mechanical stress. These de novo catch bonds will be minimal models to build an understanding of their mechanism and could become the basis for novel biomaterials. Catch bonds are hallmarks of bacterial attachment to host proteins, but are also found in T Cell adhesion. Essentially, they offer a means to probe an interaction's specificity not through kinetics (that are limited by an interactions off-rate or mean lifetime) but directly through mechanical force to sample interactions more rapidly.

Additional projects include stabilizing natural proteins for improved stability and manufacturability, designing enzymes with desired functions, and creating protein binders for therapeutically relevant targetssuch as S. aureus pathogen adhesin tip domains, with promising lead peptides already emerging. We leverage our recently developed medium/high throughput screening technology to test designs (96-192 proteins per day), including biophysical characterization and Atomic Force Microscopy-based single-molecule protein folding studies. This pipeline allows rapid progress, transforming ideas from the computer into wet lab-validated designs – which remains the ultimate test for protein function. The minimal delay between design creation and experimental testing ensures rapid iterations cycles and prompt course corrections when designs are unsuccessful. The de novo proteins created and experience gained from the deconstruction through de novo reconstruction approach proposed here, could be extended to a multitude of other molecular mechanisms across the protein universe.

Future Directions

We are setting up the lab establishing methods to screen thousands of proteins in a few experiments, vastly expanding the size of the datasets used to probe protein function with de novo design. Protein design methods have matured and are now limited by experiments, in other words: we can design more proteins than we can test. There is a dearth of experimental data on de novo designed proteins – especially comparative studies contrasting them to their naturally evolved counterparts. Thus, we are now pushing techniques to assay hundreds of proteins per day and characterize their function, in the process building consistent, large datasets with thousands of designs characterized, to inform and train the next generation of protein design models.

Selected Publications

(1) Watson JL*, Juergens D*, Bennett NR*, Trippe BL*, Yim J*, Eisenach HE*, Ahern W*, Borst AJ, Ragotte RJ, **Milles LF**, Wicky BIM, Hanikel N, Pellock SJ, Courbet A, Sheffler W, Wang J, Venkatesh P, Sappington I, Torres SV, Lauko A, De Bortoli V, Mathieu E, Ovchinnikov S, Barzilay R, Jaakkola TS, DiMaio F, Baek M, Baker D (2023). **De novo design of protein structure and function with RFdiffusion. Nature.** 620(7976):1089-1100.

(2) Wicky BIM*, **Milles LF***, Courbet A*, Ragotte RJ, Dauparas J, Kinfu E, Tipps S, Kibler RD, Baek M, DiMaio F, Li X, Carter L, Kang A, Nguyen H, Bera AK, Baker D (2022). **Hallucinating symmetric protein assemblies. Science.** 378(6615):56-61.

(3) Dauparas J, Anishchenko I, Bennett N, Bai H, Ragotte RJ, **Milles LF**, Wicky BIM, Courbet A, de Haas RJ, Bethel N, Leung PJY, Huddy TF, Pellock S, Tischer D, Chan F, Koepnick B, Nguyen H, Kang A, Sankaran B, Bera AK, King NP, Baker D (2022). **Robust deep learning–based protein sequence design using ProteinMPNN. Science.** 378(6615):49-56.

(4) **Milles LF**, Schulten K, Gaub HE, Bernardi RC (2018). **Molecular mechanism of extreme mechanostability in a pathogen adhesin. Science.** 359(6383):1527-1533.

Selected Awards and Honors

2025 ERC Starting Grant

Maximilian Muenchhoff

Web	www.genzentrum.uni-muenchen.de/research-groups/muenchhoff/
E-mail	Max.Muenchhoff@lmu.de

Since 2022	Independent group leader, Max von Pettenkofer-Institute and Gene Center, LMU Munich
Since 2016	Medical doctor, clinical virology and microbiology, Max von Pettenkofer-Institute, LMU Munich, Germany
2012-2016	Postdoc, University of Oxford, United Kingdom
2011-2012	Medical doctor, Infectious Diseases, Klinikum der Universität München, LMU Munich, Germany
2011	Doctorate (Dr. med.), Technical University Munich, Germany and University of New South Wales, Sydney, Australia

Virus Interactions of Pandemic Pathogens

Goals and Impacts for Society

My research focuses on the molecular understanding of HIV and SARS-CoV-2 infections with the goal of translating scientific findings into improved diagnostic and therapeutic strategies. By investigating viral reservoirs, immune responses, and viral evolution, we aim to uncover mechanisms of viral persistence and pathogenesis. This knowledge contributes to the development of better treatment strategies for people living with HIV and informs pandemic preparedness for emerging viruses. Our translational approach bridges basic research and clinical application, ensuring that scientific progress benefits patients directly. Ultimately, our work supports public health by advancing personalized medicine and strengthening responses to global infectious disease threats.

Research Highlights

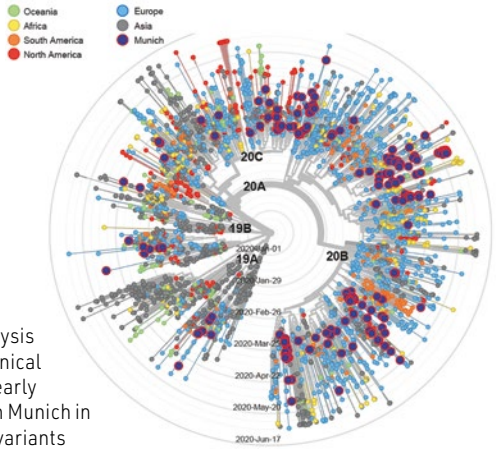
The emergence of SARS-CoV-2 has demonstrated the critical importance of rapid scientific assessment in responding effectively to novel pathogens. In collaboration with the Gene Center's LAFUGA laboratory and a network of Bavarian virology departments and public health institutions, we quickly established a genomic surveillance platform. This infrastructure enables real-time tracking of viral evolution and supports disease surveillance for SARS-CoV-2 and, more recently, other emerging pathogens.

At LMU Hospital, the early setup of a clinical COVID-19 cohort facilitated a wide range of studies that provided valuable insights into disease pathogenesis, host-virus interactions, and clinical heterogeneity. Our research group focused on characterizing immune escape mutations, particularly in immunocompromised patients with persistent infections. We identified both antibody and T-cell escape mutations, highlighting the virus's remarkable capacity to evolve under immune pressure. In addition, we investigated immunological signatures across the disease spectrum, identifying dysregulated pathways of inflammation in severe COVID cases.

Building on my longstanding commitment to HIV research and my clinical role at the National Reference Center for Retroviruses at the Pettenkofer Institute, my group investigates HIV persistence and viral reservoirs. We have developed advanced tools to quantify and genetically characterize these reservoirs, supporting global efforts towards a functional HIV cure. Specifically, we implement digital PCR assays combined with full-length sequencing to quantify and characterize HIV proviruses. We also developed ultra-sensitive tools to quantify intermediates of HIV transcription to assess the activity of the HIV reservoir in clinical samples. These methods are now being applied to studies exploring immune correlates of viral control, including the potential role of CXCR5-positive CD8+ T cells as therapeutic targets in HIV cure strategies.

Future Directions

In the next years we will intensify our studies of the HIV reservoir on a molecular level and translate our assays to the clinic. Our work will be consolidated as infrastructure and applied to various cohort studies and clinical trials within the DZIF network. In parallel, we will harness cutting-edge molecular technologies to analyze patient samples and advance genomic surveillance. These efforts aim to strengthen clinical care and elevate pandemic preparedness through integrated, data-driven approaches.



Phylogenetic Analysis of SARS-CoV-2 clinical isolates from the early pandemic phase in Munich in relation to global variants

Selected Publications

(1) Khatamzas E*, Antwerpen MH*, Rehn A, Graf A, Hellmuth JC, Hollaus A, Mohr AW, Gaitzsch E, Weiglein T, Georgi E, Scherer C, Stecher SS, Gruetzner S, Blum H, Krebs S, Reischer A, Leutbecher A, Subklewe M, Dick A, Zange S, Girl P, Müller K, Weigert O, Hopfner KP, Stemmler HJ, von Bergwelt-Baildon M, Keppler OT, Wölfel R, **Muenchhoff M***, Moosmann A* (2022). **Accumulation of mutations in antibody and CD8 T cell epitopes in a B cell depleted lymphoma patient with chronic SARS-CoV-2 infection. Nat Commun.** 13(1):5586.

(2) **Muenchhoff M**, Graf A, Krebs S, Quartucci C, Hasmann S, Hellmuth JC, Scherer C, Osterman A, Boehm S, Mandel C, Becker-Pennrich AS, Zoller M, Stubbe HC, Munker S, Munker D, Milger K, Gapp M, Schneider S, Ruhle A, Jocham L, Nicolai L, Pekayvaz K, Weinberger T, Mairhofer H, Khatamzas E, Hofmann K, Spaeth PM, Bender S, Kääb S, Zwissler B, Mayerle J, Behr J, von Bergwelt-Baildon M, Reincke M, Grabein B, Hinske CL, Blum H, Keppler OT (2021). **Genomic epidemiology reveals multiple introductions of SARS-CoV-2 followed by community and nosocomial spread, Germany, February to May 2020 Euro Surveill.** 26(43):2002066.

(3) **Muenchhoff M**, Adland E, Karimanzira O, Crowther C, Pace M, Csala A, Leitman E, Moonsamy A, McGregor C, Hurst J, Groll A, Mori M, Sinmyee S, Thobakgale C, Tudor-Williams G, Prendergast AJ, Kloverpris H, Roider J, Leslie A, Shingadia D, Brits T, Daniels S, Frater J, Willberg CB, Walker BD, Ndung'u T, Jooste P, Moore PL, Morris L, Goulder P (2016). **Nonprogressing HIV-infected children share fundamental immunological features of nonpathogenic SIV infection. Sci Transl Med.**8(358):358ra125.

(4) Payne R*, **Muenchhoff M***, Mann J, Roberts HE, Matthews P, Adland E, Hempenstall A, Huang KH, Brockman M, Brumme Z, Sinclair M, Miura T, Frater J, Essex M, Shapiro R, Walker BD, Ndung'u T, McLean AR, Carlson JM, Goulder PJ (2014) **Impact of HLA-driven HIV adaptation on virulence in populations of high HIV seroprevalence. Proc Natl Acad Sci.** 111(50):E5393-400.

Selected Awards and Honors

2025 DZIF HIV reservoir characterization infrastructure

2022 For-COVID research grant

Daniel Reichart

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Since 2025	Board Certification: Cardiology/Internal Medicine and Intensive Care Medicine
Since 2023	DFG Emmy Noether Junior Group Leader at the Gene Center, LMU Munich
Since 2021	Physician Scientist, Department of Cardiology, Medical Faculty, LMU Munich
2018-2021	Postdoctoral Fellow at the Harvard Medical School, USA (Lab of Profs. Christine and Jonathan Seidman)
2014-2018	Physician Scientist, University Heart Center Hamburg

Translational Cardiovascular Medicine

Goals and Impacts for Society

Cardiovascular diseases remain the leading cause of death worldwide, with a growing share of the burden linked to aging populations and end-stage heart failure. Our projects seek to transform cardiovascular diagnostics and therapies by applying single nucleus RNA sequencing (snRNA-seq) and cell-free RNA (cfRNA) profiling to key challenges in aging-related heart disease and transplantation. By mapping the cellular and molecular landscape of the heart at unprecedented resolution – including hard-to-access cardiomyocytes – snRNA-seq enables deep insights into disease progression and immune cell dynamics. These findings are further complemented by cfRNA, which offers a non-invasive, blood-based readout of tissue-specific gene expression. Together, these technologies pave the way for earlier diagnosis, precision immunomonitoring, and personalized treatment. In parallel, genome editing tools such as CRISPR-based base and prime editors hold the potential to correct inherited cardiovascular disorders at DNA level. Collectively, these innovations aim to reduce the societal and economic impact of heart disease through targeted diagnostics and curative approaches.

Research Highlights

Current methods for assessing immune activation and tissue injury in cardiovascular medicine – such as biopsies and serum biomarkers – are invasive, nonspecific, or limited in their sensitivity to early pathological changes. SnRNA-seq addresses these limitations by capturing high-resolution transcriptional data from individual nuclei, including those of large, post-mitotic cardiomyocytes, even from frozen or very small tissue samples.

Our published snRNA-seq atlas of the healthy and failing human heart led to insights into all major cardiac cell types, delineating chamber-specific programs and disease-associated remodeling. This foundational work now informs ongoing efforts to apply snRNA-seq to transplantation, where optimized protocols allow profiling from routine biopsy material. These studies aim to detect immune activation and uncover mechanisms of rejection and adaptation in transplanted hearts, including those in xenogeneic settings.

Transplantation research is paralleled by a strong focus on aging-related cardiovascular conditions. Atrial fibrillation, increasingly prevalent with age, is being studied at single cell level to understand its cellular drivers, particularly changes in atrial cardiomyocytes and conduction system cells. Similarly, age-related amyloidosis is being explored to dissect the interplay between protein deposition, inflammation, and fibrosis at single-cell resolution. These disease models share common hallmarks such as immune infiltration and tissue remodeling, making them ideally suited for comparative analysis using snRNA-seq.

To extend tissue-based insights into clinical monitoring, we are also advancing cfRNA profiling from plasma. cfRNA reflects real-time transcriptional activity in tissues, capturing dynamic responses to stress, inflammation, and immune activation. The combination of snRNA-seq and cfRNA creates a powerful paired diagnostic framework that holds promise for the development of blood-based biomarkers reflecting tissue-level events. On the therapeutic side, genome editing technologies are becoming increasingly viable. CRISPR-based base and prime editors allow for the correction of monogenic mutations even in terminally differentiated cardiomyocytes. These approaches are being developed in parallel with molecular diagnostics, laying the groundwork for targeted, long-term treatment of inherited cardiac diseases.

Future Directions

With our efforts, we will further integrate snRNA-seq and cfRNA profiling into clinical research workflows for immune monitoring in heart transplantation and aging-associated cardiac diseases. Emphasis will be placed on spatial transcriptomics to contextualize gene expression within tissue architecture. Ongoing development of bioinformatics tools – especially segmentation algorithms and cell-state annotation pipelines – will support scalable, clinically meaningful analyses. This systems-level approach aims to enable real-time, cell-type-resolved diagnostics and guide precision therapies in transplant and age-related heart disease.

Selected Publications

(1) **Reichart D***, Newby GA*, Wakimoto H*, Lun M, Gorham JM, Curran JJ, Raguram A, DeLaughter DM, Conner DA, Marsiglia JDC, Kohli S, Chmatal L, Page DC, Zabaleta N, Vandenberghe L, Liu DR, Seidman JG, Seidman C (2023). **Efficient in vivo genome editing prevents hypertrophic cardiomyopathy in mice. Nat Med.** 29(2):412-421.

(2) **Reichart D***, Lindberg EL*, Maatz H*, Miranda AMA, Viveiros A, Shvetsov N, Gärtner A, Nadelmann ER, Lee M, Kanemaru K, Ruiz-Orera J, Strohmenger V, DeLaughter DM, Patone G, Zhang H, Woehler A, Lippert C, Kim Y Adami E, Gorham JG, Barnett SN, Brown K, Buchan RJ, Chowdhury R, Constantinou C, Cranley J, Felkin LE, Fox H, Ghauri A, Gummert J, Kanda M, Li R, Mach L, McDonough B, Samari S, Shahriaran F, Stanasiuk C, Theotokis PI, Theis FJ, van den Bogaerdt A, Wakimoto H, Ware JS, Worth CS, Barton PJR, Lee YL, Teichmann SA, Milting H, Noseda M, Oudit GY, Heinig M, Seidman JG, Hubner N, Seidman CES (2022). **Pathogenic variants damage cell compositions and single cell transcription in cardiomyopathies. Science.** 377(6606): eabo1984.

(3) Litviňuková M*, Talavera-López C*, Maatz H*, **Reichart D***, Worth CL, Lindberg EL, Kanda M, Polanski K, Fasouli ES, Samari S, Roberts K, Tuck L, Heinig M, DeLaughter DM, McDonough B, Wakimoto H, Gorham JM, Nadelmann ER, Mahbubani K, Saeb-Parsy K, Patone G, Boyle JJ, Zhang H, Zhang H, Viveiros A, Oudit GY, Bayraktar O, Seidman JG, Seidman CE, Noseda M, Hübner N, Teichmann SA (2020). **Cells of the adult human heart. Nature.** 588:466-472.

(4) Sungnak W, Huang N, Bécavin C, Berg M, Queen R, Litvinukova M, Talavera-López C, Maatz H, **Reichart D**, Sampaziotis F, Worlock KB, Yoshida M, Barnes JL; HCA Lung Biological Network (2020). **SARS-CoV-2 entry factors are highly expressed in nasal epithelial cells together with innate immune genes. Nat Med**.26(5):681-687.

Selected Awards and Honors

2023 DFG Emmy Noether Grant

2020 Thomas W. Smith Award for Advancing Basic and Clinical Science des Brigham & Women's Hospital, Harvard Medical School

Johannes Stigler

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Since 2023	Professor (W2), Gene Center and Department of Biochemistry, LMU
2018-2023	Tenure-Track Professor, Gene Center and Department of Biochemistry, LMU
Since 2017	Emmy Noether Group leader, Gene Center, LMU
2017	Postdoc at Technical University Munich
2013–2016	Postdoc at Columbia University, New York
2012	PhD at Technical University Munich

Biophysics of Genome Stability

Goals and Impacts for Society

Chromosomes provide the scaffold for the maintenance and transmission of genetic information and for the expression and regulation of genes. Our laboratory studies the question of how molecular machines ensure the stability of chromosomes. We further investigate how the chromosomal physical environment the function of these molecular machines. For this, we combine bulk biochemistry with single molecule biophysics and computational modeling.

Research Highlights

Topological domains on interphase chromosomes of higher eukaryotes are established by SMC complexes and delineated by the transcription factor and genomic insulator CTCF.

In previous work, we found, using high-throughput single-molecule fluorescence microscopy by DNA curtains on microfluidics chips, that the SMC complex Smc5/6 is preferentially recruited to single-stranded sections on DNA. These sections are rare overall along chromosomes but specifically appear at telomeres and at sites of stalled replication forks during replication. With the help of fluorescence imaging and single-molecule optical tweezers, we could show that Smc5/6 recognizes the interface between single-stranded and double-stranded DNA and mechanically stabilizes fork-like structures. Our results provide key insights into a possible activation mechanism of Smc5/6 in DNA repair [1].

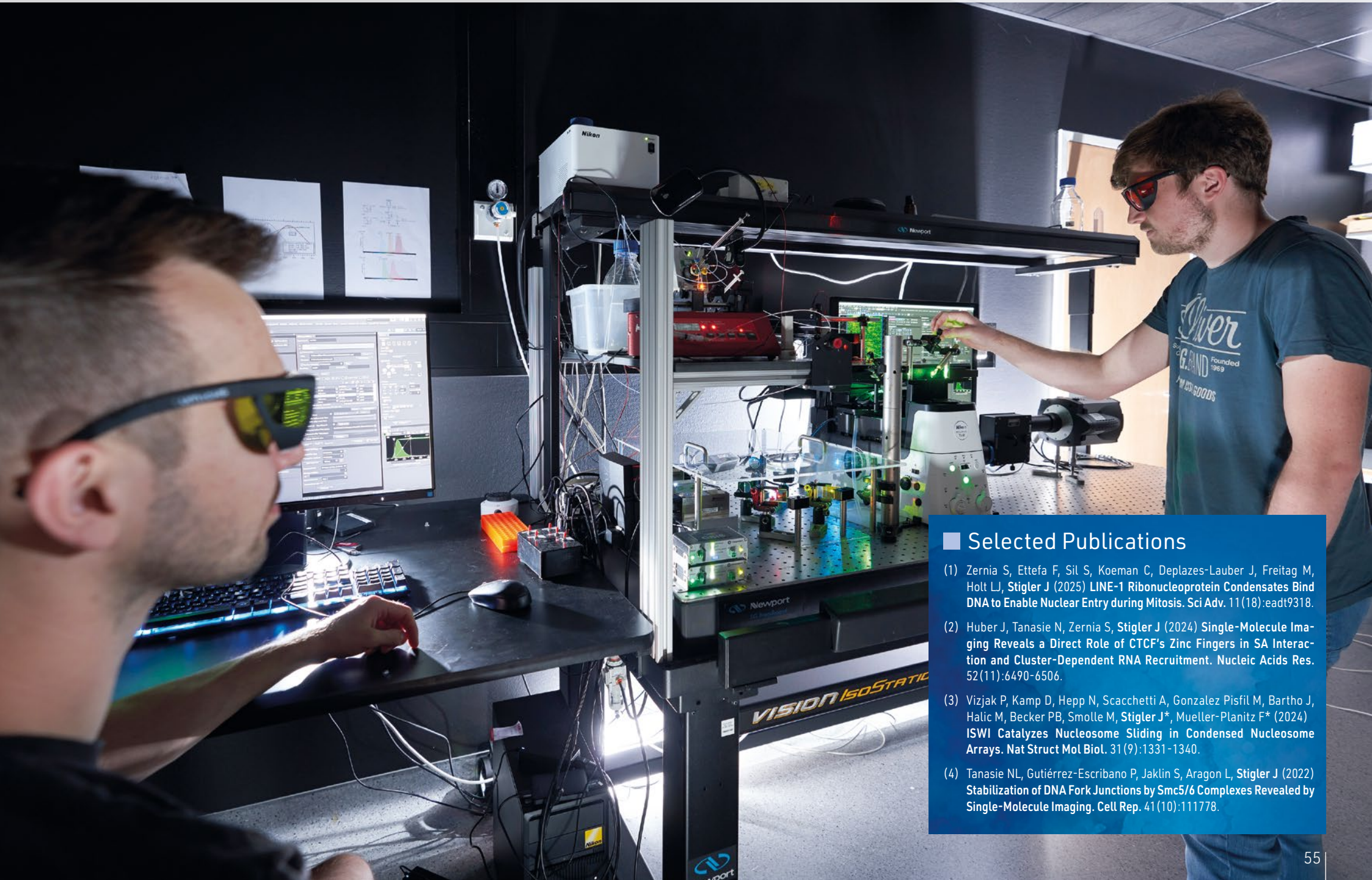
In another study, we were also able to visualize the interactions of the transcription factor and genomic insulator CTCF with domain boundary motifs on DNA and with subdomains of the SMC complex cohesin. Using live microscopy, we could show that despite its insulating activity in vivo, CTCF in vitro could be readily displaced by transcribing RNA polymerases. Moreover, we demonstrated that CTCF zinc finger domains interact with RNA and R-loops to form interaction hubs. The results thus provide context for transcription and RNA interactions are connected to chromatin domain formation through CTCF-mediated insulation [2].

At an even deeper level, the mechanical properties of interphase chromosomes are defined by interactions between individual nucleosomes. We studied the fluidity of reconstituted chromatin at highly elevated concentrations, similar to those found in the cell nucleus, with the goal of investigating the effect of nucleosome remodeling on the mechanics. Using the remodeler ISWI as an example, we showed that ISWI is only minimally inhibited in a very crowded chromatin environment and is very mobile throughout the chromatin scaffold. Intriguingly, its mobility is tightly connected to ATP hydrolysis. Failure to hydrolyze ATP renders ISWI in an inactive state, which crosslinks individual nucleosomes and leads to solidified chromatin. This work has provided a new paradigm of how ATP hydrolysis is required not only for fueling enzyme activity, but also for mobilizing certain enzymes in the nucleus and for preventing detrimental chromatin solidification [3].

Other previous work from our lab investigated genomic stresses from within: A substantial fraction of the human genome consists of remnants of LINE-1 transposable elements, which can insert themselves into chromosomes using a copy-and-paste mechanism. We found that ribonucleoprotein condensates of the LINE-1 protein ORF1p and RNA require a specific stoichiometry between protein and nucleic acid in order to bind to DNA before insertion. This study further showed that these condensates directly bind to mitotic chromosomes and enter the nucleus during nuclear envelope breakdown, where they insert and act as possibly carcinogenic genomic stressors [4].

Future Directions

In the future, we aim to continue our endeavor of reconstituting the nuclear environment one component at a time and studying the interplay between chromatin and chromosomal enzymes from the mesoscale to the single molecule level. For this, we will continue to develop bulk biochemical, single molecule biophysical and computational methods to elucidate the connection between the physical and biochemical world at the nanoscale.



Selected Publications

(1) Zernia S, Ettefa F, Sil S, Koeman C, Deplazes-Lauber J, Freitag M, Holt LJ, **Stigler J** (2025) **LINE-1 Ribonucleoprotein Condensates Bind DNA to Enable Nuclear Entry during Mitosis. Sci Adv.** 11(18):eadt9318.

(2) Huber J, Tanasie N, Zernia S, **Stigler J** (2024) **Single-Molecule Imaging Reveals a Direct Role of CTCF's Zinc Fingers in SA Interaction and Cluster-Dependent RNA Recruitment. Nucleic Acids Res.** 52(11):6490-6506.

(3) Vizjak P, Kamp D, Hepp N, Scacchetti A, Gonzalez Pisfil M, Bartho J, Halic M, Becker PB, Smolle M, **Stigler J***, Mueller-Planitz F* (2024) **ISWI Catalyzes Nucleosome Sliding in Condensed Nucleosome Arrays. Nat Struct Mol Biol.** 31(9):1331-1340.

(4) Tanasie NL, Gutiérrez-Escribano P, Jaklin S, Aragon L, **Stigler J** (2022) **Stabilization of DNA Fork Junctions by Smc5/6 Complexes Revealed by Single-Molecule Imaging. Cell Rep.** 41(10):111778.

Julian Stingele

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Since 2025	Professor (W3), Gene Center and Department of Biochemistry, LMU
2022-2025	Professor (W2), Gene Center and Department of Biochemistry, LMU
2017-2022	Tenure-Track Professor, Gene Center and Department of Biochemistry, LMU
2014-2017	Postdoctoral research fellow at The Francis Crick Institute, UK
2015	PhD, Max-Planck-Institute of Biochemistry and LMU Munich

Cellular Responses to DNA and RNA Damage

Goals and Impacts for Society

Cells are constantly exposed to reactive substances that cause complex damage to cellular macromolecules. These damages stem from both endogenous reactive metabolites and a wide range of exogenous factors, including various forms of radiation, chemotherapeutic agents, and tobacco smoke. Almost all reactive substances act pleiotropically, meaning they cause diverse cellular lesions, particularly in DNA and RNA. If these damages are not detected and resolved, they can lead to premature aging, cancer, and other diseases.

A key goal of our research is therefore to understand how damage to DNA and RNA is sensed and eventually resolved. By uncovering the underlying molecular mechanisms, we aim to lay the groundwork for new therapeutic strategies to prevent or treat cancer and degenerative diseases.

Research Highlights

A particularly relevant source of endogenous cellular damage are reactive aldehydes, which are byproducts of various metabolic processes. For example, acetaldehyde is released during alcohol breakdown in the liver and is the primary cause of ethanol toxicity. Formaldehyde arises during one-carbon metabolism and enzymatic demethylation reactions. These aldehydes induce covalent DNA-protein crosslinks (DPCs), which are highly toxic, because they block transcription and replication. Over the last years, we have discovered several pathways and regulatory mechanisms enabling the repair of DPCs.

The specialized DPC protease SPRTN degrades the protein component of DPCs, thereby promoting replication of DPC-containing DNA. The DPC protease SPRTN is highly promiscuous, which is useful, given that virtually every chromatin protein can become crosslinked to DNA. However, how cleavage is restricted to crosslinked proteins had remained unclear. We found that SPRTN achieves specificity by recognizing specific DNA structures which trigger activation of the protease (Ref 1). Additionally, we discovered that SPRTN cannot process folded protein adducts by itself, which requires in addition the activity of the FANCJ helicase. Using in vitro reconstitution, we showed that FANCJ binds next to the DPC and uses its ATPase activity to unfold the protein adduct, exposing the underlying DNA and enabling cleavage of the crosslinked protein by SPRTN (Ref 2).

Moreover, we discovered that the proteins CSB and CSA provide resistance to agents that induce DPCs. Mutations in CSB and CSA cause Cockayne syndrome, a severe growth and neurological disorder. We found that CSB and CSA respond if RNA polymerases stall at DPCs, promoting DPC repair and restart of transcription. Our findings suggest that defective transcription-coupled DPC repair contributes to the unique features of Cockayne syndrome (Ref 3).

In addition to damaging DNA, aldehydes also induce crosslinking between proteins and RNA. We therefore hypothesized that cells must harbor pathways to detect and eliminate such RNA lesions. However, the complexity of aldehyde-induced damage complicates the analysis of the responsible quality control mechanisms. To investigate how cells respond to RNA crosslinking damage, my group developed a protocol to specifically induce RNA damage, which led to the discovery of a cellular pathway that resolves RNA-protein crosslinks (Ref 4).

Future Directions

Most DNA-damaging agents also damage RNA, a fact overlooked by most researchers for the last decades. The central objective of our future research is therefore to develop a comprehensive understanding of how cells preserve the integrity of both, DNA and RNA. By identifying and characterizing the cellular mechanisms responsible for resolving DNA and RNA damage, we aim to uncover new biological principles and to provide a foundation for innovative therapeutic strategies in cancer treatment.

Selected Publications

(1) Reinking HK, Kang HS, Götz MJ, Li HY, Kieser A, Zhao S, Acampora AC, Weickert P, Fessler E, Jae LT, Sattler M, **Stingele J** (2020). **DNA Structure-Specific Cleavage of DNA-Protein Crosslinks by the SPRTN Protease. Mol Cell.** 80(1):102-113.e6. (2) Yaneva D, Sparks JL, Donsbach M, Zhao S, Weickert P, Bezalel-Buch R, **Stingele J***, Walter JC* (2023). T**he FANCJ helicase unfolds DNA-protein crosslinks to promote their repair. Mol Cell.** 83(1):43-56.e10.

(2) Yaneva D, Sparks JL, Donsbach M, Zhao S, Weickert P, Bezalel-Buch R, **Stingele J***, Walter JC* (2023). **The FANCJ helicase unfolds DNA-protein crosslinks to promote their repair**. **Mol Cell.** 83(1):43-56.e10.

(3) Carnie CJ, Acampora AC, Bader AS, Erdenebat C, Zhao S, Bitensky E, van den Heuvel D, Parnas A, Gupta V, D'Alessandro G, Sczaniecka-Clift M, Weickert P, Aygenli F, Götz MJ, Cordes J, Esain-Garcia I, Melidis L, Wondergem AP, Lam S, Robles MS, Balasubramanian S, Adar S, Luijsterburg MS, Jackson SP, **Stingele J** (2024). T**ranscription-coupled repair of DNA-protein cross-links depends on CSA and CSB. Nat Cell Biol.** 26(5):797-810.

(4) Zhao S, Cordes J, Caban KM, Götz MJ, Mackens-Kiani T, Veltri AJ, Sinha NK, Weickert P, Kaya S, Hewitt G, Nedialkova DD, Fröhlich T, Beckmann R, Buskirk AR, Green R, **Stingele J** (2023). **RNF14-dependent atypical ubiquitylation promotes translation-coupled resolution of RNA-protein crosslinks. Mol Cell.** 83(23):4290-4303.e9.

Selected Awards and Honors

2023 ERC Consolidator Grant „Deconstruct"

2021 Vallee Scholar Award

2020 EMBO Young Investigator

Marion Subklewe

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Since 2017	Head of the Lab for Translational Cancer Immunology, Gene Center, LMU
Since 2014	Professor & Consultant for Internal Medicine with focus on Cellular Therapy, University Hospital, LMU
Since 2009	Professor for Hematology/Oncology, University Hospital, LMU
2007	Board Certificate in Hematology & Oncology, Habilitation in Internal Medicine, Charité, Berlin
2000 – 2008	Board Certificate in Internal Medicine and Fellowship in Hematology & Oncology at Charité, Berlin
1997 – 2000	Postdoc at Rockefeller University, NYC, USA
1995	Medical Dissertation, Department of Hematology & Oncology, University of Cologne

Cellular Cancer Immunotherapy

Goals and Impacts for Society

Immunotherapies have fundamentally changed the treatment landscape of cancer, enabling long-term remission and even cures for patients with otherwise fatal disease. Our goal is to further expand the reach of these therapies by understanding why some patients respond while others do not. We study resistance mechanisms, immune escape, and T cell dysfunction using patient samples and preclinical models. Our translational research bridges laboratory insights with early-phase clinical trials. In doing so, we aim to improve therapeutic efficacy, safety, and patient selection for cellular immunotherapy in cancer and beyond.

Research Highlights

One major focus of our research is the dynamic interaction between T cells and tumor cells during immunotherapy. We study this across multiple therapeutic platforms, including checkpoint blockade, T cell-redirecting antibodies, and CAR-T cells. In a recent study, we demonstrated that continuous stimulation by CD3-engaging bispecifics induces T cell exhaustion – a key resistance mechanism. Importantly, we showed that treatment-free intervals can restore T cell function and improve efficacy, providing a rationale for intermittent dosing in clinical protocols (1). In complementary work, we investigated the use of STING agonists to modulate the tumor microenvironment and cross-talk between T cells and Acute Myeloid Leukemia (AML) cells. STING activation triggered a proinflammatory response and enhanced the activity of T cell engagers in preclinical models. These findings support the potential of innate immune modulation to boost the efficacy of T cell-based immunotherapies (2).

To overcome the challenges of CAR-T cell therapy in AML, we developed an adapter CAR-T platform (AdCAR) that enables flexible, switchable targeting and fine-tuned control over T cell activation. Our work showed that treatment-free intervals also reduce CAR-T exhaustion and that sequential targeting of different AML-associated antigens is feasible in vitro using primary AML samples (3).

Together with European collaborators, we co-led the definition of "immune effector cell-associated hematotoxicity" (ICAHT), the most common toxicity after CAR-T therapy. We developed a novel grading system and clinical management algorithm that accounts for both depth and duration of cytopenia. These tools are now integrated in multiple ongoing trials and international registries (4).

In a parallel line of work, we investigated the biological mechanisms underlying severe hematotoxicity after CD19 CAR-T therapy. Using proteomics and immune profiling, we showed that patients with prolonged cytopenia exhibit systemic immune dysregulation even before treatment, including elevated levels of myelosuppressive cytokines and checkpoint molecules. These insights provide a rationale for pre-treatment risk stratification and combinatorial immunomodulation.

Future Directions

Our future research will focus on optimizing the timing, intensity, and modularity of immune effector therapies in cancer but also autoimmune diseases. We aim to better understand how inflammation, immune contexture, and target antigen density modulate cell-cell crosstalk and shape treatment response and resistance. We will use high-dimensional immune profiling, spatial proteomics, and functional T-cell assays to define biomarkers for patient stratification. In addition, we are developing universal, switchable CAR platforms and evaluating adaptive dosing strategies to reduce toxicity and preserve T cell fitness. Through these efforts, we hope to move toward more precise and durable immunotherapy for patients with hematologic malignancies and solid tumors alike, driving broader applications across cancer types.

Selected Publications

(1) Philipp N, [...], **Subklewe M** (2022). **T-cell exhaustion induced by continuous bispecific molecule exposure is ameliorated by treatment-free intervals. Blood.** 140(10):1104-1118.

(2) Linder A, Nixdorf D, Kuhl N, Piseddu I, Xu T, Holtermann AV, Kuut G, Endres R, Philipp N, Bücklein V, de Graaff J, Carell T, Kobold S, Kischel R, Hornung V, **Subklewe M** (2025). **STING activation improves T-cell-engaging immunotherapy for acute myeloid leukemia. Blood.** 145(19):2149-2160.

(3) Nixdorf D, [....], **Subklewe M** (2023). **Adapter CAR T cells to counteract T-cell exhaustion and enable flexible targeting in AML. Leukemia**. 37(6):1298-1310.

(4) Rejeski K*, **Subklewe M***, et al. (2023). **Immune effector cell-associated hematotoxicity: EHA/EBMT consensus grading and best practice recommendations, Blood** 142(10):865-877. *These authors contributed equally.

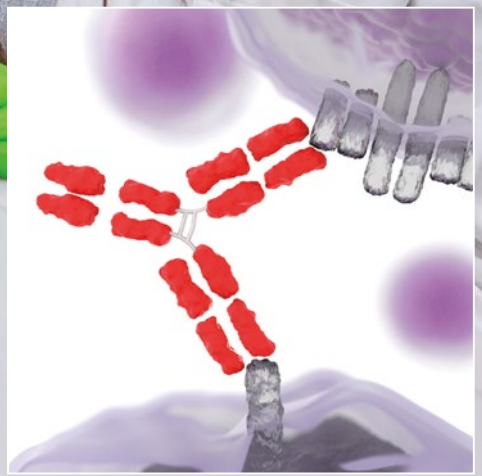
Selected Awards and Honors

2025 HemaSphere Award – Top Cited Clinical Article

2024 Highly Cited Researcher

2024 Pfleger Preis

2024 ASH Educational (AML)



Sebastian Theurich

Web	www.genzentrum.uni-muenchen.de/research-groups/theurich
E-mail	S.Theurich@lmu.de

2020	Professor (W2) of Cancer- and Immunometabolism, LMU University Hospital
2018	Interim Professor of Internal Medicine, LMU University Hospital
2015	Habilitation (venia legendi, internal medicine), Medical Faculty, University of Cologne
2013-16	Postdoctoral Research Fellow, Max-Planck-Institute for Metabolism Research, Cologne
2012	Board Certification for Internal Medicine, Hematology and Oncology
2006	Dissertation (M.D.), Max-Delbrück-Center for Molecular Medicine and Charité, Humboldt-University Berlin

Cancer- and Immunometabolism Research

Goals and Impacts for Society

Sebastian Theurich, trained as clinical hematologists and oncologist, is a physician scientist and holds a professorship in the emerging field of cancer- and immunometabolism. Here and at the crossroads of metabolism and immunity his group investigates, how systemic and environmental metabolic factors shape immune cell activity and cancer progression, and on the other hand, how one can understand and utilize metabolic programs in immune cells for cancer immunotherapy.

Research Highlights

In particular his group is active in the following areas:

1. Metabolism and Metabolic Engineering of Cytotoxic Lymphocytes:

- How do T cells and Natural Killer (NK) cells adjust their metabolic programming to function effectively–especially within metabolically stressful tumor microenvironment? The research group aims to understand key metabolic reprogramming and how this can be exploited by artificial metabolic engineering of immune cells, including CAR-T cells, in order to improve cancer immunotherapy. Current projects are conducted within the Collaborative Research Center (SFB/TRR338 LETSimmun) funded by the German Research Foundation (DFG)

2. Obesity and Inflammation ("Metaflammation"):

- The Theurich lab investigates how obesity-driven chronic inflammation (i.e., metaflammation) alters immune cell metabolism and, in particular, NK cell biology. While his previous work has pioneered the understanding of the crucial role of NK cell subsets for systemic glucose metabolism and inflammation, his current focus is on the role of obesity-associated NK cells in cancer biology.
- Such findings offer paths toward metabolic targeting–like caloric or pharmacological interventions–to improve immune responses in metabolic disorders and cancer.

3. Immunometabolic Therapeutics and Translational Metabolic Interventions

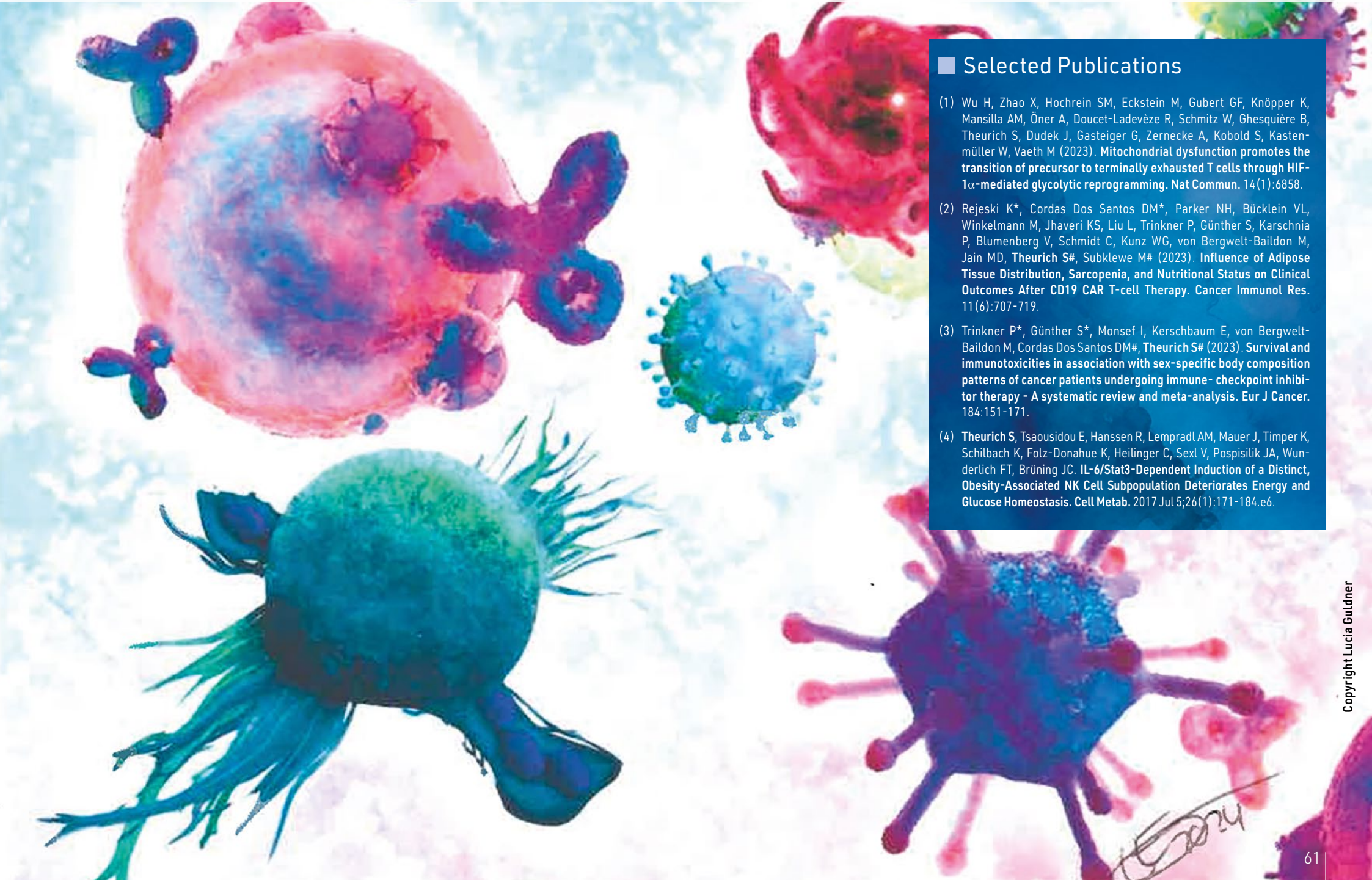
- In close collaboration between his group at the Gene Center and the Department of Hematology and Oncology at the LMU University Hospital, Sebastian Theurich´s lab examines how clinical metabolic interventions (such as defined, personalized exercise and nutrition interventions) can alter systemic immune responses and immunotherapy outcomes. The clinical branch of the group has launched a number of translational clinical studies, such as the multicenter randomized controlled INTEGRATION trial, combining lifestyle, diet, and exercise interventions with first-line oncology treatments to enhance patient outcomes, as well as prehabilitation interventions in patients before CAR-T cell therapy (OPTIMISE).

Future Directions

To understand the immunologic and metabolic heterogeneity of cancer tissues better and to exploit this for improving cancer immunotherapy, we will extend our current analyses by advanced spatial omics technologies. We will also focus on cancer-derived metabolites and their local impact on cancer immune responses. In this regard, the Gene Center offers excellent collaboration opportunities and experts, and we will furthermore continue to strengthen our translational research branch.

Selected Awards and Honors

2025	Co-spokes person of the „Center for Inflammation and Metabolism (CIM)", LMU Munich
2024	Co-Spokes Person, Working Group for Nutrition, Metabolism and Exercise (German Society of Hematology & Oncology, DGHO)
2022	Deputy chair of the Department of Medicine III; Hematology and Oncology, LMU University Hospital
2020	Scientist of the German Translational Cancer Research Consortium (DKTK)



Selected Publications

(1) Wu H, Zhao X, Hochrein SM, Eckstein M, Gubert GF, Knöpper K, Mansilla AM, Öner A, Doucet-Ladevèze R, Schmitz W, Ghesquière B, Theurich S, Dudek J, Gasteiger G, Zernecke A, Kobold S, Kastenmüller W, Vaeth M (2023). **Mitochondrial dysfunction promotes the transition of precursor to terminally exhausted T cells through HIF-1α-mediated glycolytic reprogramming. Nat Commun.** 14(1):6858.

(2) Rejeski K*, Cordas Dos Santos DM*, Parker NH, Bücklein VL, Winkelmann M, Jhaveri KS, Liu L, Trinkner P, Günther S, Karschnia P, Blumenberg V, Schmidt C, Kunz WG, von Bergwelt-Baildon M, Jain MD, **Theurich S#**, Subklewe M# (2023). **Influence of Adipose Tissue Distribution, Sarcopenia, and Nutritional Status on Clinical Outcomes After CD19 CAR T-cell Therapy. Cancer Immunol Res.** 11(6):707-719.

(3) Trinkner P*, Günther S*, Monsef I, Kerschbaum E, von Bergwelt-Baildon M, Cordas Dos Santos DM#, **Theurich S#** (2023). **Survival and immunotoxicities in association with sex-specific body composition patterns of cancer patients undergoing immune- checkpoint inhibitor therapy - A systematic review and meta-analysis. Eur J Cancer.** 184:151-171.

(4) **Theurich S**, Tsaousidou E, Hanssen R, Lempradl AM, Mauer J, Timper K, Schilbach K, Folz-Donahue K, Heilinger C, Sexl V, Pospisilik JA, Wunderlich FT, Brüning JC. **IL-6/Stat3-Dependent Induction of a Distinct, Obesity-Associated NK Cell Subpopulation Deteriorates Energy and Glucose Homeostasis. Cell Metab.** 2017 Jul 5;26(1):171-184.e6.

Eckhard Wolf

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Since 2015	Director of the Center for Innovative Medical Models (CiMM), LMU Munich
Since 2005	Director of the Laboratory for Functional Genome Analysis (LAFUGA), Gene Center, LMU Munich
Since 1995	Professor and Chair, Gene Center and Department of Veterinary Sciences, LMU Munich
1994	Group leader, University of Veterinary Sciences Vienna
1991-1993	Postdoc, LMU Munich
1990	Dr. med. vet., LMU Munich

Engineering Innovative Medical Models

Goals and Impacts for Society

The translation of new discoveries in basic medical research into clinical applications is a long, costly, and often inefficient process. To accelerate progress in translational medicine, we develop genetically tailored pigs as models for metabolic research (diabetes mellitus, obesity, atherosclerosis) and for rare monogenic diseases. Another key focus is the creation of genetically multi-modified pigs as sources of cells, tissues, and organs for xenotransplantation. Our porcine disease models help overcome the limitations of current model systems in predicting the efficacy and safety of new therapies in humans. Genetically modified pigs for organ donation offer a realistic solution to the current shortage of human organs available for transplantation.

Research Highlights

As the large animal platform of the German Center for Diabetes Research (DZD) we developed genetically (pre)diabetic pig models and pigs expressing specific reporter genes in the pancreatic islets. Transgenic pigs expressing SNAP-tagged insulin allow for the first time pulse labeling of insulin secretory granules in the beta cells and studies of their turnover in vivo (1). Pigs expressing near-infrared protein facilitate the monitoring of transplanted islets using optical imaging or multispectral optoacoustic tomography (MSOT).

Our most successful model for a rare monogenic disease is a pig resembling a frequent form of Duchenne muscular dystrophy (DMD), caused by the loss of *DMD* exon 52 (*DMD*Δ52). In contrast to mouse models for DMD, the porcine model develops a severe disease phenotype in an accelerated mode. DMD pigs have been used for testing novel therapies such as gene editing to restore an intact DMD reading frame and for validating MSOT as an imaging modality to monitor disease progression and the efficacy of therapeutic interventions (reviewed in 2). To predict the best possible outcome of gene editing to delete *DMD* exon 51 and reframe the transcript in *DMD*Δ52 pigs, we generated pigs lacking both exon 51 and 52, thereby resembling a model for the milder Becker muscular dystrophy.

(BMD) (3). These pigs express a shortened but functional dystrophin and appear healthy in most of the clinical parameters investigated, indicating that somatic gene editing is a promising strategy to ameliorate DMD pathology, if sufficient efficiencies are achieved.

For the development of source pigs for xenotransplantation, we established a colony of Auckland Island (AI) pigs, which fit the size of humans and are free of porcine endogenous retrovirus type C (PERV-C). Moreover, they are genetically homogeneous (e.g. MHC uniform) without signs of inbreeding depression and have an excellent heart function (4). AI pigs have been genetically modified to eliminate the major carbohydrate xenoantigens αGal, Neu5Gc, and Sda (knockouts of *GGTA1*, *CMAH*, *B4GALNT2/B4GALNT2L*) and to express the human complement pathway regulator CD46 and human thrombomodulin. This is considered the minimal set of genetic modifications required for clinical cardiac xenotransplantation. Within the Leducq-Network XenExoCor we are testing strategies to replace entire compartments (e.g. the endothelium) of pig hearts by human cells. This is achieved by complementing organogenesis-impaired porcine embryos with human stem cells.

Future Directions

We will establish arterio-venous metabolomics and metabolic flux studies in diabetic and obese pig models to unravel organ cross-talk in these metabolic disturbances. *MSTN* knockout pigs will serve as a model for studying the role of muscle hypertrophy in normalizing metabolic disbalances. Pig models for monogenic diseases such as DMD will be used for testing the efficacy and safety of new therapies. The newly established Interfaculty Center for Endocrine and Cardiovascular Disease Network Modelling and Clinical Transfer (ICONLMU) will serve as an ideal platform for these studies. Hearts from genetically multi-modified AI pigs will be tested in orthotopic xenotransplantation experiments in baboons, before initiating a clinical pilot study.

Genetically multi-modified Auckland Island pigs as donors for organ xenotransplantation

Selected Publications

(1) Kemter E, Müller A, Neukam M, Ivanova A, Klymiuk N, Renner S, Yang K, Broichhagen J, Kurome M, Zakhartchenko V, Kessler B, Knoch KP, Bickle M, Ludwig B, Johnsson K, Lickert H, Kurth T, **Wolf E***, Solimena M* (2021). **Sequential in vivo labeling of insulin secretory granule pools in INS-SNAP transgenic pigs. Proc Natl Acad Sci U S A**. 118(37):e2107665118.

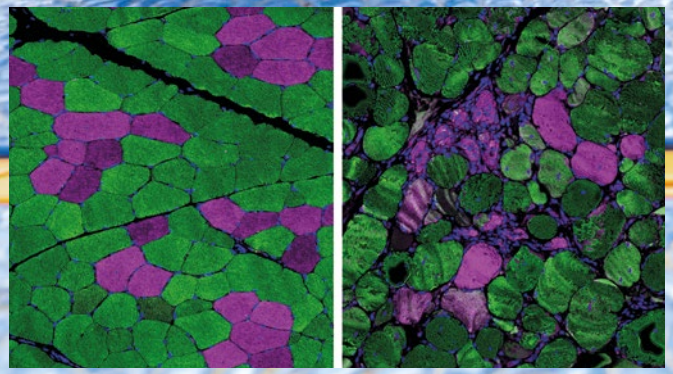
(2) Stirm M, Klymiuk N, Nagashima H, Kupatt C, **Wolf E** (2024). **Pig models for translational Duchenne muscular dystrophy research. Trends Mol Med**. 30(10):950-964.

(3) Stirm M, Shashikadze B, Blutke A, Kemter E, Lange A, Stöckl JB, Jaudas F, Laane L, Kurome M, Keßler B, Zakhartchenko V, Bähr A, Klymiuk N, Nagashima H, Walter MC, Wurst W, Kupatt C, Fröhlich T, **Wolf E** (2023). **Systemic deletion of DMD exon 51 rescues clinically severe Duchenne muscular dystrophy in a pig model lacking DMD exon 52. Proc Natl Acad Sci U S A**. 120(29):e2301250120.

(4) Lange A, Medugorac I, Ali A, Kessler B, Kurome M, Zakhartchenko V, Hammer SE, Hauser A, Denner J, Dobenecker B, Wess G, Tan PLJ, Garkavenko O, Reichart B, **Wolf E***, Kemter E* (2024). **Genetic diversity, growth and heart function of Auckland Island pigs, a potential source for organ xenotransplantation. Xenotransplantation** 31(2):e12858.

Selected Awards and Honors

2025 Elected Member, Bavarian Academy of Science and Humanities



Skeletal muscle sections of a wild-type and a Duchenne muscular dystrophy pig

Microscope stage for embryo micromanipulation

Rotem Sorek

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Since 2024	Visiting Professor within the framework of the Max Planck-Humboldt Research Award, Gene Center, LMU
Since 2018	Full Professor, Department of Molecular Genetics, Weizmann Institute of Science, Rehovot, Israel
2014 - 2018	Associate Professor, Weizmann Institute of Science, Rehovot, Israel
2008 - 2014	Senior Scientist, Weizmann Institute of Science, Rehovot, Israel
2007	Ph.D. Human Genetics, Tel Aviv University, Tel Aviv, Israel
2006 - 2008	Post-doctoral Fellow, Lawrence Berkeley National Lab, Berkeley, USA

In 2023, microbial genomics expert Professor Rotem Sorek from the Weizmann Institute of Science was honored with the Max Planck-Humboldt Research Award, endowed with 1.5 million euros. The award supports his collaborative project with Veit Hornung (Gene Center Munich) and Jörg Vogel (Helmholtz Institute for RNA-based Infection Research, Würzburg), which explores the ancient roots of innate immunity.

Sorek's research has shown that bacteria possess sophisticated antiviral defense systems–many of which represent evolutionary precursors of human immune pathways. By investigating these mechanisms, the collaboration aims to uncover new antiviral strategies that could eventually inform innovative therapies.

Immunologist Veit Hornung emphasizes the transformative potential of Sorek's findings: "Thanks to Sorek's work, we now know that the bacterial immune system is the evolutionary origin of important components of the human innate immune system – a discovery that can also provide deeper insights into the complexity of the human immune system."

To further strengthen this collaboration, Rotem Sorek joined the Gene Center as a visiting scientist for one year starting in July 2024, which has significantly deepened the scientific exchange and joint research efforts with Veit Hornung's group.

The Immune System of Bacteria

Goals and Impacts for Society

Our lab of microbial genomics and systems biology studies the immune system of bacteria. We are interested in deciphering the molecular mechanisms providing bacteria with protection against viruses that infect them (phages). Our studies describe the function of ancient innate immunity, and reveal how modern immune systems of humans and plants evolved from genetic systems that protect bacteria against phages.

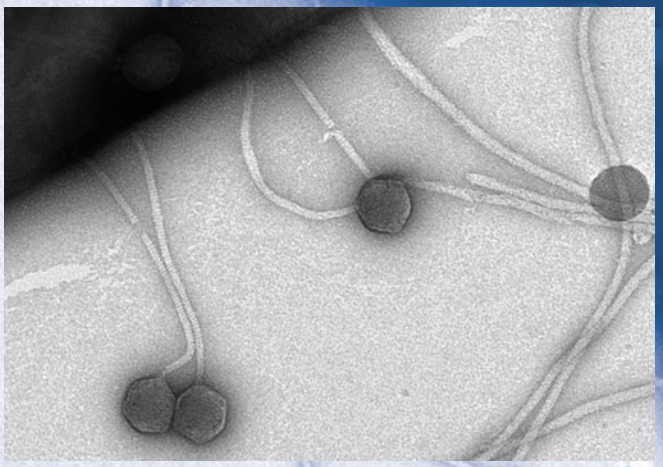
Research Highlights

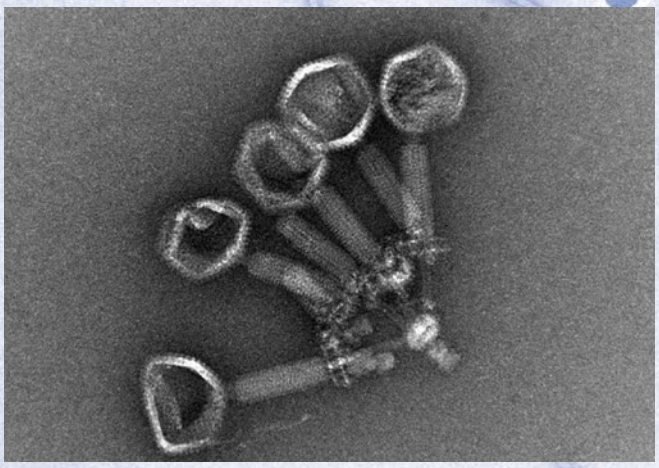
We discovered that many components of the human cell-autonomous innate immune system evolved from defense systems that protect bacteria from phage infection (1). For example, we showed that the cGAS-STING antiviral pathway, originally discovered in animals, is also widespread in bacteria and protects them against phage infection. In addition, we found that genes with Toll-interleukin receptor (TIR) domains are involved in bacterial defense against phages, providing evidence for a common, ancient ancestry of innate immunity components shared between animals, plants, and bacteria (2). In other studies we showed that viperins, enzymes producing antiviral molecules in humans, evolved from bacterial enzymes that have similar functions, and that a human inflammatory process called Pyroptosis also originated in bacteria. Defense systems employing a eukaryotic-like ubiquitin conjugation machinery were also described by our lab (3), as well as defense systems that use caspase-like proteins to execute immune-activated cell death in bacteria (1).

Our discoveries explain the evolution of the human cell-autonomous innate immune system. In addition to explaining how our immune system evolved, we also showed that understanding mechanisms in bacterial immune systems solves new mechanisms in the animal and plant immune systems (4).

Future Directions

Looking ahead, we are now applying the knowledge we gained on the immune system of bacteria to discover new concepts in human and plant immunity. We draw inspiration from the conservation between bacterial and human immunity, and explore parallels that can teach us more about how our immune system recognizes and mitigates infection.





Selected Publications

(1) Wein T, **Sorek R** (2022). **Bacterial origins of human cell-autonomous innate immune mechanisms. Nature Reviews in Immunology**, 22(10):629-638.

(2) Rousset F, Osterman I, Scherf T, Falkovich AH, Leavitt A, Amitai G, Shir S, Malitsky S, Itkin M, Savidor A, **Sorek R** (2025). **TIR signaling activates caspase-like immunity in bacteria. Science**, 387(6733):510-516..

(3) Hör J, Wolf SG, **Sorek R** (2024). **Bacteria conjugate ubiquitin-like proteins to interfere with phage assembly. Nature**, 631:850-856.

(4) Rousset F, Yirmiya E, Nesher S, Brandis A, Mehlman T, Itkin M, Malitsky S, Millman A, Melamed S, **Sorek R** (2023). **A conserved family of immune effectors cleaves cellular ATP upon viral infection. Cell**, 186(17), 3619-3631.

Selected Awards and Honors

2025 Robert Koch Prize

2025 Gruber Prize in Genetics

2023 Max Planck-Humboldt Research Award

2023 HFSP Nakasone Award

Stefan Canzar

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Since 2024	Professor, University of Regensburg
2023	Associate Professor, Penn State University, USA
2016-2022	Group Leader, Gene Center, LMU
2014-2016	Research Assistant Professor, TTIC, Chicago, USA
2009-2014	Postdoctoral positions at CWI Amsterdam, The Netherlands, and Johns Hopkins University, Baltimore, USA
2008	PhD MPI for Informatics, Saarbrücken, and LORIA, Nancy, France

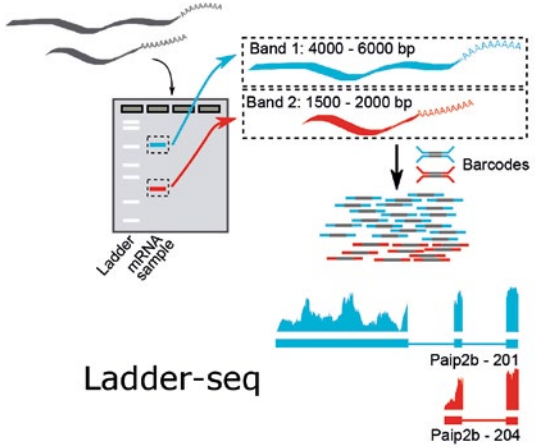
Computational Genomics

Goals

The lab's goal is the development of accurate mathematical models, efficient algorithms, and usable software for the efficient and effective interpretation of vast and complex life sciences data. The link between advances in algorithmic theory and new insights into fundamental problems in biology and human disease constitutes our driving force for research.

Research Highlights

For the development of accurate and efficient computational methods we combine techniques from combinatorial optimization and machine learning. For instance, we have developed exact and approximate algorithms for a graph coloring problem to increase the resolution of experimental protein structure data, used neural networks to project high-dimensional cellular measurements to an interpretable low-dimensional space, and extended dynamic time warping to the comparison of complex trajectories of, e.g., differentiating immune cells.



In traditional sequencing, mRNA is fragmented, sequenced in parallel, and reassembled computationally–though some information is lost. To improve this, we introduced Ladder-seq: before fragmentation, RNA is separated by length via electrophoresis, producing a visible gel ladder. This size information enables more accurate sequence reconstruction. Modified from publication (1).

Selected Publications

(1) Ringeling FR, Chakraborty S, Vissers C, Reiman D, Patel AM, Lee KH, Hong A, Park CW, Reska T, Gagneur J, Chang H, Spletter ML, Yoon KJ, Ming GL, Song H, **Canzar S** (2022). **Partitioning RNAs by length improves transcriptome reconstruction from short-read RNA-seq data. Nat Biotechnol.** 40(5), 741–750.

(2) Monteagudo-Mesas P, Brönner C, Kohvaei P, Amedi H, **Canzar S**, Halic M (2022). **Ccr4-Not complex reduces transcription efficiency in heterochromatin. Nucleic Acids Res.** 50(10), 5565–5576.

(3) Do VH, **Canzar S** (2021). **A generalization of t-SNE and UMAP to single-cell multimodal omics. Genome Biol.** 22(1), 130.

(4) Do VH, Rojas Ringeling F, **Canzar S** (2021). **Linear-time cluster ensembles of large-scale single-cell RNA-seq and multimodal data. Genome Res.** 31(4), 677–688.

At University of Regensburg: As part of an international effort to construct "complete" T2T reference genomes of seven ape species – including humans–we demonstrated how using a T2T assembly can enhance RNA read mapping and transcript reconstruction from long-read RNA sequencing technologies. Additionally, using RNA long reads, we estimated copy numbers for multi-copy gene families and compared the number of genes per family in the previous assembly versus the T2T assembly. We found that nearly all gene families showed increased copy numbers across all the great ape species studied, with gains ranging from modest to truly striking. Yoo et al, Nature, 2025.

At Gene Center: The experimental fragmentation of RNA in popular short-read sequencing assays results in a loss of information that cannot be fully restored by computational methods alone. Our team therefore developed Ladder-seq, which combines modifications to the RNA-seq protocol with tailored algorithms to identify transcripts that remain invisible to conventional RNA-seq methods. Figuratively speaking, an additional experimental step adds color information to the genetic puzzle pieces, allowing our algorithms to assemble the puzzle more precisely than before. Using Ladder-seq, we were able to decode the function of regulatory units in neural stem cells in the brains of mice. Ringeling et al, Nature Biotechnology, 2022.

Biological Mass Spectrometry

Goals

A focus of our research effort is on the development of mass spectrometric technologies for the de novo sequencing of antibodies and on the characterization of epitopes and affinities of the antigen-antibody interaction. In contrast to established standard methods for recombinant antibodies in the pharmaceutical industry, these mass spectrometric approaches should enable the analysis of endogenous antibodies from blood samples. Characterizing the patient-specific antibody spectrum will provide the molecular basis for the development of efficient tailored therapies.

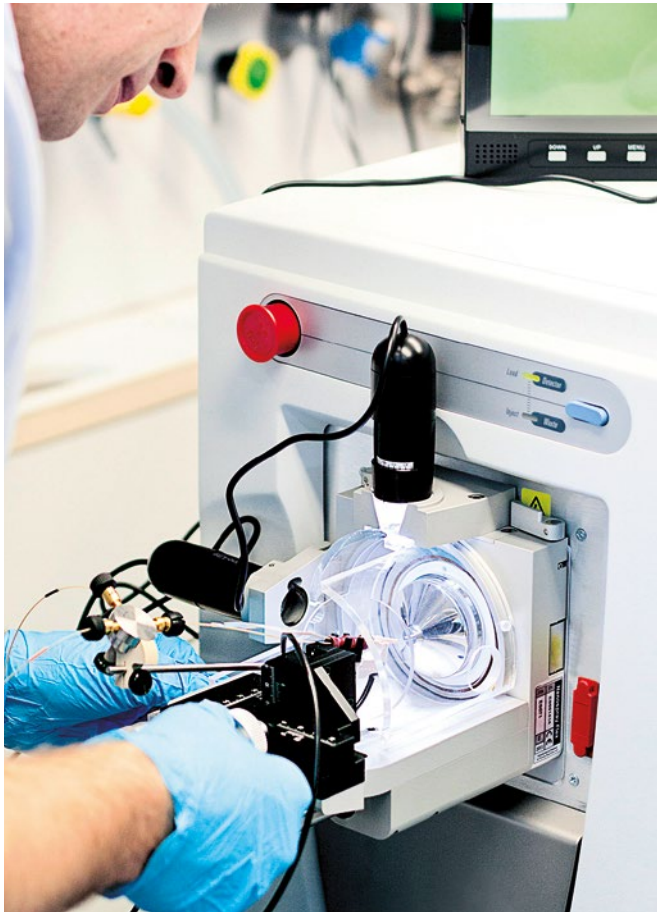
Selected Publications

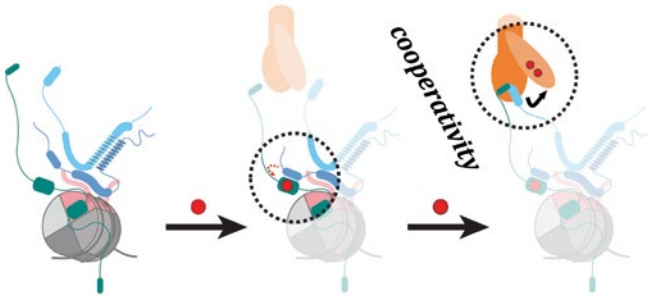
(1) Woike S, Eustermann S, Jung J, Wenzl SJ, Hagemann G, Bartho J, Lammens K, Butryn A, **Herzog F**, Hopfner KP (2023). **Structural basis for TBP displacement from TATA box DNA by the Swi2/Snf2 ATPase Mot1. Nat Struct Mol Biol.** 30(5):640-649.

(2) Köhnke T, Liu X, Haubner S, Bücklein V, Hänel G, Krupka C, Solis-Mezarino V, **Herzog F**, Subklewe M (2022). **Integrated multiomic approach for identification of novel immunotherapeutic targets in AML. Biomark Res.** 10(1):43.

(3) Kratzat H, Mackens-Kiani T, Ameismeier M, Potocnjak M, Cheng J, Dacheux E, Namane A, Berninghausen O, **Herzog F**, Fromont-Racine M, Becker T, Beckmann R (2021). **A structural inventory of native ribosomal ABCE1-43S pre-initiation complexes. EMBO** J. 40(1):e105179.

(4) Ghodgaonkar-Steger M, Potocnjak M, Zimniak T, Fischböck-Halwachs J, Solis-Mezarino V, Singh S, Speljko T, Hagemann G, Drexler DJ, Witte G, **Herzog F** (2020). **C-Terminal Motifs of the MTW1 Complex Cooperatively Stabilize Outer Kinetochore Assembly in Budding Yeast. Cell Rep.** 32(13):108190.





Franz Herzog

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Since 2021	Endowed Professor, IMC University of Applied Sciences, Austria
2012-2021	Group Leader, Gene Center, LMU
2008-2012	Postdoc at the Swiss Federal Institute of Technology, Zurich, Switzerland
2007	Postdoc at the Research Institute of Molecular Pathology, Vienna
2003-2006	Associate Member, St. Jude Children's Research Hospital, Memphis, USA

In Memoriam:

Karl-Klaus Conzelmann

It is with great sadness that we received the news of the passing of our colleague and good friend, Professor Karl-Klaus Conzelmann. Klaus was a long-standing member of the Gene Center Munich and a former faculty member at the Max von Pettenkofer Institute. He was one of the pillars who shaped the Gene Center's international reputation in molecular virology.

Klaus was not only a visionary scientist but also a dedicated mentor and a sought-after collaborator. He embodied a rare combination of intellectual rigor and deep understanding of his field of expertise, coupled with cross-disciplinary curiosity. His pioneering contributions to reverse genetics using rabies virus cDNA transformed the molecular manipulation of non-segmented RNA viruses, laid the groundwork for an entire generation of viral vector technologies, enabled synaptic tracing in neurobiology, and culminated in a promising vaccine design developed by his lab during the SARS-CoV-2 pandemic.

Karl-Klaus Conzelmann began his research career in virology at the Federal Research Center for Virus Diseases of Animals in Tübingen (now the Friedrich-Loeffler-Institute), where he studied viruses of veterinary and public health relevance. In parallel, Klaus pioneered reverse genetics for negative-strand RNA viruses. In a landmark study in 1994 (EMBO J), his lab reported the first recovery of infectious rabies virus from a full-length cDNA clone–a technique that initiated a whole new field of research. Today, every artificially created negative-strand RNA virus, including measles and influenza viruses, is based on the principles of Klaus's technique. His deep interest in fundamental molecular mechanisms led him to investigate the assembly of rabies virus, demonstrating that virion budding could occur with core structural components alone and independently of the glycoprotein–a key insight published in Cell (1996). These findings informed the rational design of attenuated viral strains and vaccine vectors.

After joining the LMU Munich faculty in 1999, Klaus's research expanded into the molecular mechanisms underlying virus–host interactions. A defining moment in innate immunity research came in 2006, when Klaus co-authored a Science paper together with one of us (VH), demonstrating how cells discriminate viral RNA from self. My (VH) first contact with Klaus was as a young research associate, when I reached out to him for his virology expertise. I had a question about a reagent, and since it was the summer holiday season, I wasn't expecting much of a response–especially after receiving an out-of-office reply. But it didn't take long before Klaus got in touch–while still on vacation, I believe in Italy–and immediately offered his help. That generous gesture marked the beginning of what became a rewarding collaboration. We worked together to understand how viruses are sensed by innate immune cells, a field still emerging in the mid-2000s. At the time, there was only a hunch that cytosolic RNA sensing pathways existed, but the molecular players remained elusive. With the discovery of RIG-I by the Fujita lab in 2004, we finally had a clearer idea of where to look. Following our discovery that triphosphate RNA is a highly potent agonist for RIG-I, we were eager to connect this finding to the recognition of viruses in the cytosol–and Klaus immediately supported us by providing RNA from virus-infected cells to test our hypothesis. The resulting study resonated widely in the field and quickly became a foundational reference for understanding viral RNA sensing–something we could not have achieved without Klaus's early and enthusiastic support.

Prior to this publication, I (KPH) vividly remember how Klaus, all secretive, came to my office just as we were beginning work on RIG-I structures. He told me that he and his collaborators had figured out how RIG-I can discriminate self from non-self. But, with a boyish smile, he continued that "unfortunately, he can't really tell me any details". He kindly agreed to a little guessing game–the outcome of which I'll leave open here. In any case, the mechanism was officially revealed shortly thereafter in Science (2006), where Klaus and we (VH and the team around Gunther Hartmann and Stefan Endres) showed that uncapped 5'-triphosphate RNA is the natural ligand for the cytosolic receptor RIG-I to discriminate self from non-self RNA. Klaus's expertise in virology was instrumental in generating the defined viral RNA species and engineered viruses used in these studies.

Following this line of research, Klaus's lab revealed viral immune evasion strategies across multiple pathogens, including rabies virus, measles virus, and respiratory syncytial virus–helping to explain how these viruses establish infection in humans. His favorite protein of the rabies virus was the P protein, now recognized as one of the strongest viral antagonists of human interferon responses. Another favorite of his was the V protein, which antagonizes MDA5-based RNA sensing. With his great enthusiasm, he incited us (KPH) and then collaborated to reveal its structural mechanism (Science, 2013).

On a clear day in early 2020, in the wake of the COVID-19 pandemic, I (KS) visited Klaus in an almost deserted Gene Center. Though physically distanced, we intensely discussed a novel emerging virus–SARS-CoV-2–and how we, as virologists, could help to combat this threat. These discussions sparked a series of interdisciplinary studies that clarified immune evasion strategies mediated by individual SARS-CoV-2 proteins (Science, 2020; Cell Reports, 2021). Klaus's lab also applied his viral engineering expertise to vaccine development against SARS-CoV-2. His group created replication-deficient rhabdovirus replicons expressing heterologous antigens–a platform that enabled the development of a chimeric VSV/rabies vector presenting a SARS-CoV-2 minispike, which elicited robust neutralizing antibody responses in preclinical models (PLOS Pathogens, 2021). These works highlight Klaus's engagement with pressing biomedical challenges and his astonishing ability to flexibly apply his expertise to emerging threats.

Among Klaus's most transformative contributions was the engineering of glycoprotein-deleted rabies virus vectors for transsynaptic tracing of neuronal circuits. In a stroke of genius, he and his collaborating neuroscientists recognized the potential of the exclusively synaptic spread of rabies virus for mapping the connectome of neurons in the brain. Together with his team and collaborators, Klaus developed recombinant rabies viru-

ses capable of monosynaptic spread, enabling cell-type-specific mapping of neuronal inputs. These systems, described in Neuron and Nature Methods (2007), became cornerstone tools in systems neuroscience. This work highlights the power of interdisciplinary basic science to enable transformative technologies that reshape entire fields. Klaus's lab continued to refine these vectors, expanding their application to studies of pain, emotion, and behavior in animal models. His innovations provided unprecedented access to the architecture of neural circuits and remain essential in contemporary neurobiological research. Klaus Conzelmann's trans-synaptic tracing of neuronal circuits through engineered rabies viruses stands among the Gene Center's most significant contributions to technology.

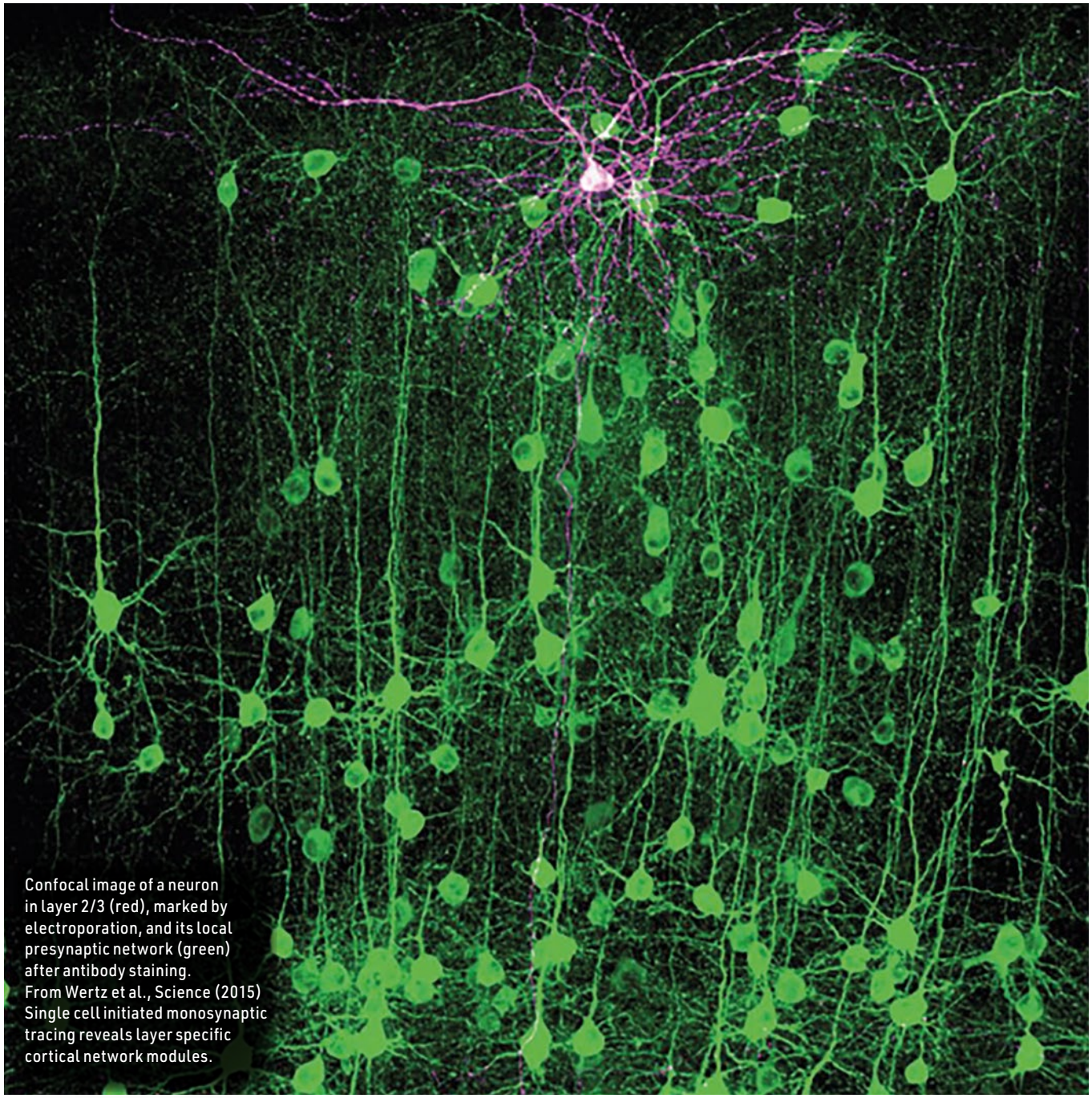
Klaus was a highly sought-after and active participant in many collaborative research initiatives spanning virology, innate immunity, and neurobiology. He contributed extensively to the scientific community through editorial service, peer mentorship, and interdisciplinary collaboration. He was an excellent teacher and supervisor, and I (KS) heard many times that his lectures at the Faculty of Chemistry and Pharmacy even transformed biology-skeptical chemistry students into passionate molecular virologists. Klaus guided many students to their PhDs and remained a friend and mentor to them thereafter.

We will remember Klaus as an extraordinary scientist, rigorous discussion partner, and inspiring mentor. He remained deeply curious about developments in virology, innate immunity, neurobiology and beyond, and his enthusiasm for science never waned. We will always remember the spark in his eyes whenever we exchanged ideas—he had a way of engaging with science that was both rigorous and joyful. Klaus also had a wonderful sense of perspective. There was often a twinkle in his eye and an easy smile—a lightness that made conversations with him a pleasure. In a world that often takes itself too seriously, Klaus brought light, laughter, and sincerity. His spirit left a mark on all of us—and his memory will stay with us always.

He will be deeply missed.

Karl-Peter Hopfner
Veit Hornung
Konstantin Sparrer

Munich, April 4th, 2025



Confocal image of a neuron in layer 2/3 (red), marked by electroporation, and its local presynaptic network (green) after antibody staining.
From Wertz et al., Science (2015)
Single cell initiated monosynaptic tracing reveals layer specific cortical network modules.

LAFUGA

Goals and Impacts for Society

The Laboratory for Functional Genome Analysis (LAFUGA) is an integrated technology platform with the research units Genomics, Proteomics and Model Organisms. It combines state-of-the-art molecular profiling techniques with clinically relevant, tailored animal models to address import questions in medical research and environmental health. The LAFUGA-Units are thus key components of multiple national and international research consortia.

Research Highlights

LAFUGA-Genomics

Helmut Blum, Stefan Krebs
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High-throughput sequencing (NGS) has become an indispensable tool for biomedical research by allowing the detailed analyses of transcriptomes and genomes and by providing readouts for biological processes on the molecular, cellular and organismal level. In addition to the Illumina technology, LAFUGA-Genomics also performs long-read sequencing with nanopores and offers the innovative AVITI technology for most cost-efficient short-read production and direct in-situ single-cell mRNA sequencing and multiome imaging. With this variety of sequencing techniques we support research projects in a multitude of disciplines, including translational biomedical research, biochemistry, immunology, virology, archeology among others. We analyze genetically modified pigs as xenograft organ donors and disease models by characterizing their genome, transcriptome and single-cell gene expression.

LAFUGA-Genomics carried out projects in the fields of mammalian reproduction, early embryonic development and evolution of hematopoietic malignancies as part of international and national research consortia (e.g. DFG SFB 1243 "Cancer Evolution"). In response to the SARS-CoV2 pandemic we developed platforms for viral genome surveillance, waste-water monitoring and viral metagenome sequencing, funded by the multicenter project BayVOC (Bavarian State Ministry of Health, Care and Prevention).

LAFUGA-Proteomics

Thomas Fröhlich
Email: Thomas.Froehlich@lmu.de

Thanks to advances in modern mass spectrometry-based proteomics in terms of sensitivity and throughput, proteomics has become an integral part of translational biomedical research. For proteomic analysis, we use latest high-performance mass spectrometry instruments (e.g. timsTOF HT, Q Exactive HF-X) facilitating the precise quantification of thousands of proteins out of complex proteomes like cell lysates. For targeted approaches, dedicated methods (e.g. Selected/Parallel Reaction Monitoring) are applied, to precisely quantify low-abundant proteins in the attomole range. The research focus of LAFUGA-Proteomics is on biomedicine, including diabetes, Duchenne muscular dystrophy, fertility disorders and ecotoxicology. For instance, recent proteomic analyses of tissues from neonatal offspring of transgenic pig models for diabetes discovered molecular pathways relevant for the increased health risks of children of diabetic mothers. Furthermore, proteomic studies in a transgenic porcine model of Duchenne muscular dystrophy have recently demonstrated to be a powerful proof-of-concept tool for innovative gene therapy approaches. Moreover, in light of increasing environmental pollution, we have joined forces with scientists from diverse disciplines in the SFB 1357-Mikroplastics (coordinated by Prof. Laforsch, University Bayreuth). The aim of this SFB is to develop a fundamental understanding of the effects of microplastics on biological, physical and chemical processes.

All LAFUGA-Proteomics research projects are funded by either the German Research Foundation (DFG), the Else Kröner-Fresenius Foundation (EKFS) or the European Union's Horizon Europe research and innovation programme.

LAFUGA-Animal Models

Eckhard Wolf
Email: EWolf@lmu.de

This unit is specialized in the development, characterization and implementation of genetically tailored pigs as disease models and as donors of cells, tissues and organs for xenotransplantation. In addition to the Center for Innovative Medical Models (CiMM; https://www.lmu.de/cimm/), the Interfaculty Center for Endocrine and Cardiovascular Disease Network Modeling and Clinical Transfer (ICON LMU; https://www.med.lmu.de/icon/en/) has recently been opened as a unique infrastructure for large animal research in translational medicine. We are/were thus the core facility for large animal models in a number of national and international research consortia, including the German Center for Diabetes Research (DZD), the DFG SFB/TR 127 "Biology of xenogeneic cell, tissue and organ transplantation – from bench to bedside", the EU project iNanoBIT "Integration of Nano- and Biotechnology for Beta-Cell and Islet Transplantation", and the recently established EU Innovative Health Initiative NHPig (https://www.nhpig.eu/). For highlights of our research, please see the report of the Wolf group.

Future Perspectives

LAFUGA aims to continue its general strategy but improve the depth and spatial resolution of its molecular analyses and refine its large animal models to allow tissue-specific and/or developmental stage-specific analyses of disease mechanisms. With the establishment of CiMM and ICON LMU, treatment trials of clinically relevant large animal models will – in concert with the LAFUGA-Genomics and -Proteomics Units – enable the discovery of molecular biomarkers for safety and efficacy of novel therapies.

Selected Publications

(1) **Multi-omics analysis of diabetic pig lungs reveals molecular derangements underlying pulmonary complications of diabetes mellitus.** Shashikadze B, Flenkenthaler F, Kemter E, Franzmeier S, Stöckl JB, Haid M, Riols F, Rothe M, Pichl L, Renner S, Blutke A, **Wolf E**, **Fröhlich T**. **Dis Model Mech.** 2024 Jun 20:dmm.050650. doi: 10.1242/dmm.050650. PMID: 38900131

(2) **Systemic deletion of DMD exon 51 rescues clinically severe Duchenne muscular dystrophy in a pig model lacking DMD exon 52.** Stirm M, Shashikadze B, Blutke A, Kemter E, Lange A, Stöckl JB, Jaudas F, Laane L, Kurome M, Keßler B, Zakhartchenko V, Bähr A, Klymiuk N, Nagashima H, Walter MC, Wurst W, Kupatt C, **Fröhlich T**, **Wolf E**. **Proc Natl Acad Sci** U S A. 2023 Jul 18;120(29):e2301250120. doi: 10.1073/pnas.2301250120. Epub 2023 Jul 10. PMID: 37428903

(3) **Maternal hyperglycemia induces alterations in hepatic amino acid, glucose and lipid metabolism of neonatal offspring: Multi-omics insights from a diabetic pig model.** Shashikadze B, Valla L, Lombardo SD, Prehn C, Haid M, Riols F, Stöckl JB, Elkhateib R, Renner S, Rathkolb B, Menche J, Hrabě de Angelis M, **Wolf E**, Kemter E, **Fröhlich T**. **Mol Metab.** 2023 Jul 4:101768. doi: 10.1016/j.molmet.2023.101768PMID: 37414142

(4) **A pathway coordinated by DELE1 relays mitochondrial stress to the cytosol.** Fessler E, Eckl EM, Schmitt S, Mancilla IA, Meyer-Bender MF, Hanf M, Philippou-Massier J, **Krebs S**, Zischka H, Jae LT. **Nature**. 2020 Mar;579(7799):433-437. doi: 10.1038/s41586-020-2076-4. PMID: 32132706.

(5) **Perinatal dysfunction of innate immunity in cystic fibrosis.** Jaudas F, Bartenschlager F, Shashikadze B, Santamaria G, Reichart D, Schnell A, Stöckl JB, Degroote RL, Cambra JM, Graeber SY, Bähr A, Kartmann H, Stefanska M, Liu H, Naumann-Bartsch N, Bruns H, Berges J, Hanselmann L, Stirm M, **Krebs S**, Deeg CA, **Blum H**, Schulz C, Zawada D, Janda M, Caballero-Posadas I, Kunzelmann K, Moretti A, Laugwitz KL, Kupatt C, Saalmüller A, **Fröhlich T**, **Wolf E**, Mall MA, Mundhenk L, Gerner W, Klymiuk N. **Sci Transl Med**. 2025 Jan 22;17(782):eadk9145. doi: 10.1126/scitranslmed.adk9145. PMID: 39841805.

(6) **Three exposures to the spike protein of SARS-CoV-2 by either infection or vaccination elicit superior neutralizing immunity to all variants of concern.** Wratil PR, Stern M, Priller A, Willmann A, Almanzar G, Vogel E, Feuerherd M, Cheng CC, Yazici S, Christa C, Jeske S, Lupoli G, Vogt T, Albanese M, Mejías-Pérez E, Bauernfried S, Graf N, Mijocevic H, Vu M, Tinnefeld K, Wettengel J, Hoffmann D, Muenchhoff M, Daechert C, Mairhofer H, **Krebs S**, Fingerle V, Graf A, Steininger P, **Blum H**, Hornung V, Liebl B, Überla K, Prelog M, Knolle P, Keppler OT, Protzer U. **Nat Med.** 2022 Mar;28(3):496-503. doi: 10.1038/s41591-022-01715-4. PMID: 35090165.

Other Research Facilities

A hallmark and key component of the Gene Center's research infrastructure is its array of shared research facilities. These facilities enable the Gene Center's research groups to conduct world-class research using a wide range of state-of-the-art technologies and methodologies.

The **cryo-electron microscopy (cryo-EM) facility** enables large-scale data collection for single-particle reconstruction, allowing the determination of biomolecular structures at near-atomic resolution. Established in 2006 and significantly expanded in 2018 with the addition of a second high-end microscope, the facility further advanced in 2022 with the integration of a new direct electron detector and energy filter, ensuring state-of-the-art capabilities for structural biology research.

For high-resolution cryo-EM single-particle analysis and cryo-electron tomography, the facility operates two advanced 300 kV Titan Krios electron microscopes. The first (Krios G1) is equipped with a Gatan K2 direct electron detector and BioQuantum energy filter, while the second (Krios G3) features a Falcon 4i direct electron detector, and Selectris X energy filter, providing flexibility and precision across diverse specimen requirements.

Additionally, a 120 kV Tecnai Spirit and a 100 kV Morgagni transmission electron microscope are available for initial cryo-EM sample screening and negative-stain data collection, respectively, supporting efficient project workflows from sample optimization to high-resolution structure determination.

Our **crystallization lab** supports high-throughput macromolecular crystallization using a broad range of experimental approaches. Liquid handling systems assist in the efficient and reproducible preparation of crystallization screens. The facility includes low-vibration, temperature-controlled incubators specifically designed for sensitive crystallization trials. Dedicated workspaces and specialized tools for crystal harvesting, manipulation, and cryo-preservation in liquid nitrogen ensure optimal sample quality for downstream structural analysis.

The **biophysics lab** is outfitted with an extensive instrumentation suite for the quantitative analysis of biomolecular properties and interactions. Capabilities include surface plasmon resonance (SPR), microscale thermophoresis (MST), dynamic light scattering (DLS), and size-exclusion chromatography coupled with static light scattering (SEC-RALS). The lab also offers Mass Photometry, real-time PCR-based differential scanning fluorimetry (RT-PCR DSF), a nanoDSF device, and precision plate readers with monochromators. Additional equipment includes isothermal titration calorimetry (ITC), a high-resolution spectrophotometer and fluorimeter, and advanced chromatographic systems. For custom design and rapid prototyping of lab components and experimental apparatus, a high-resolution FDM 3D printer is available in-house.

A **robotic high-throughput facility** has been set up to provide state-of-the-art instrumentation and expertise to support industrial research in the academic field. The facility is equipped with high-end robots in a volume range from 2.5nl to 1ml. These are flexible liquid handling workstations that integrate additional instruments for sample management, handling and various types of assays. In addition, the facility has detection and analysis systems that enable assay read-out and quality control. Thanks to this integrated concept, high-throughput projects can be carried out from assay development to assay transfer, automation, validation and finally screening. The facility, which has long been the preserve of industry, enables a wide range of high-throughput genome-scale projects to be carried out in an academic environment.

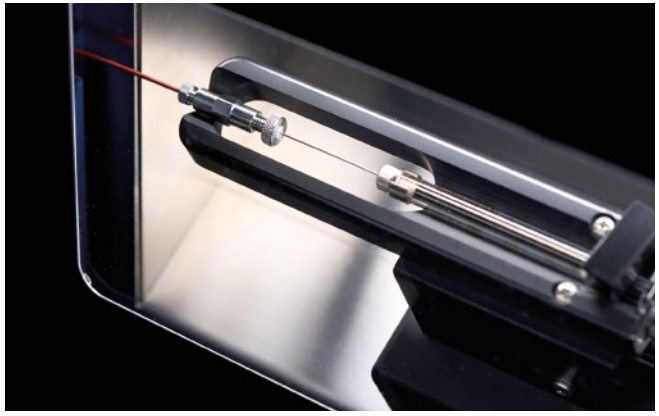
Our **high throughput sequencing platform** includes technologies for short read sequencing (Illumina NextSeq2000&1000; Element Biosciences AVITI) and long read sequencing (Oxford Nanopore, PromethION P24, MinION). For short read sequencing, a variety of protocols are established, like e.g. exome sequencing, mRNAseq, nonrRNAseq or targeted re-sequencing. The AVITI sequencer can perform single-cell multiome analysis including in-situ mRNA sequencing directly on the instrument without prior library preparation. Long read sequencing enables sequencing of genomes, epigenomes, full-length cDNA or native DNA and RNA. Data analysis is performed on a Galaxy platform hosted on a cooperative data processing infrastructure.

Flow Cytometry: A state-of-the-art flow cytometry facility that is equipped with one high end analytical (BD LSRFortessa) and two preparative flow cytometers (BD FACSAria Fusion and BD FACSMelody) has been established in the BioSysM building. The facility provides a wide range of flow cytometry-based services such as project planning, panel design and optimization, instrument operation and sorting and data analysis. Analytical and preparative flow cytometry can be conducted under BSL1 and BSL2 conditions.

In the BioSysM building, a range of advanced light microscopes are designed for a wide variety of **imaging and biophysical studies**.

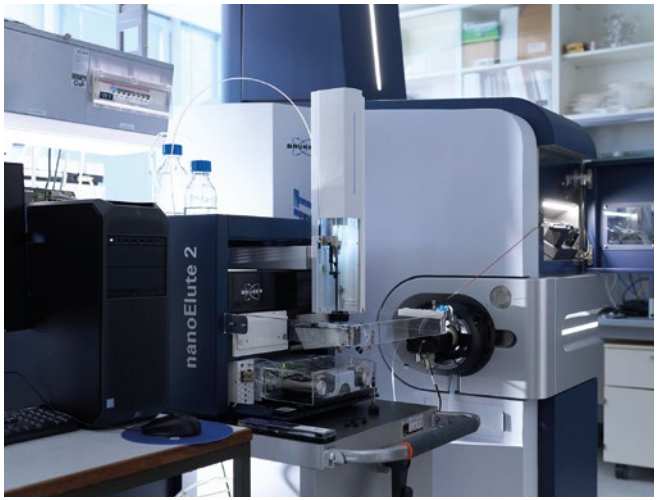
One of the main instruments is a Leica SP8 confocal microscope, a state-of-the-art system designed for high-resolution 3D fluorescence imaging. It features a white light laser (WLL) for flexible excitation across a wide spectrum, and hybrid detectors with single-photon sensitivity, enabling the detection of even weak signals. In addition, an integrated deconvolution software is available, allowing for improved image contrast and resolution. A live-cell incubation chamber allows for time-lapse imaging under physiological conditions.

A unique addition is the HiP-FA setup (High Performance Fluorescence Anisotropy), a custom-built microscope optimized for precise measurements of protein-protein and protein-DNA/RNA interactions. It uses automated fluorescence anisotropy microscopy to record binding curves with very high sensitivity, and is especially robust against issues like protein aggregation, making it ideal for sensitive fluorescence anisotropy measurements with minimal artefacts. The method requires only a small amount of protein, can capture hundreds of data points per titration and supports high-throughput measurements using multi-well formats.



We have also expanded our **mass spectrometry facilities**. LAFUGA proteomics has recently installed a new, very fast scanning mass spectrometer with improved sensitivity for deep proteome analysis which is available within the framework of collaborations. Furthermore, LAFUGA proteomics provides additional instrumentation for protein identification using nano-liquid chromatography and high-resolution FT mass spectrometers including a Q Exactive HF-X. Additionally, an Orbitrap Elite mass spectrometer is dedicated to the identification of cross-linked peptides which determine distance restraints for the structural analysis of macromolecular complexes.

Recognizing the growing demands of data-intensive research, our **scientific computing infrastructure** is continuously expanded and upgraded. The facility supports a wide spectrum of computational needs, ranging from high-performance 3D workstations and dedicated analytical servers to scalable GPU- and CPU-based computing clusters. This infrastructure ensures robust capabilities for data processing, modeling, simulation, and large-scale storage, enabling cutting-edge computational research across diverse scientific disciplines.



Administration and Support

The Gene Center's administration and infrastructure is run by a team whose philosophy is to assist all of our scientists – from principal investigators to students – in the most efficient and supportive way. We enable our scientists to focus on their research by reducing the time they spend on administrational matters to an unavoidable minimum. Thus, we contribute to the creation of a stimulating atmosphere for scientists, which is one of the keys to the Gene Center's success.

Sabine Bergelt, **Stefanie Wolf**, **Gudrun Römer**, **Annika Büscher**, **Yanet Abera Bauer**, **Emma Weber** and **Sylvia Hornig** have supported and/or currently support our PIs and groups with personnel administration, traveling and events - apart from many other things. During its current funding period, Sabine also helps to run the TRR 237 Nucleic Acid Immunity with its three sites Bonn, Munich and Dresden.

Andrea Schwane-Pieloth, **Thomas Stein** and **Yamei Li** take care of accounting and offer extensive support in the administration of third-party funded projects. Andrea advises our PIs on procurement procedures and utilization of funds and she also keeps track of the institute's central funds.

Leslie Heinz and **Angela Dietzmann** maintain the computational infrastructure of the Gene Center and offer IT support. They are working closely with computational experts from different groups, foremost **Gregor Witte** as head of the IT team.

Michael Englschall, supported by **Gabriela Bittner**, is in charge of the Gene Center's general supply with chemicals, gases, consumables and basic lab equipment. Together with **Michael Till** and **Dieter Zech** from the Gene Center's precision mechanics and electronic workshops they help to run the labs smoothly and to get new labs started within a short time. **Gabriela Bittner** is also running our guest rooms and **Michael Till** acts as our in-house photographer whenever we need one – many pictures in this report have been taken by him. The research labs are also supported by **Natasa Boskovski**, **Nevena Gmitrovic Balta**, **Homa Popal**, and **Dorchanai Schams**, who handle laboratory dishwashing services, and by **Olga Fetscher** who manages the research reagent and media service.

Carola Vogt manages the institute's resources and university / academic affairs together with the director (Gene Center/ Department Biochemistry) and the department steering committee and she heads the science support team. **Beate Hafner** organizes our public outreach activities: website, social media, events and this report.

Since 2024, Beate also coordinates our graduate training program Quantitative and Molecular Biosciences (formerly Quantitative Biosciences Munich). Undergraduate teaching at the Department of Biochemistry is supported by centrally funded positions as well: **Johanna Turck** coordinates our Master Biochemistry program and **Louiza Papatheodorou** organizes practical courses for our students.

Last but not least, **Helmut Blum**, **Thomas Fröhlich**, **Sabine Bergelt**, **Kristin Leike**, and **Michael Englschall** provide the Gene Center groups with expertise in workplace, biological, fire and radiation safety.

Undergraduate Teaching

Education of students is a key part and central mission of our work at the Gene Center and Department of Biochemistry at the LMU. Gene Center scientists are actively involved in conducting undergraduate courses, ranging from basic training in biochemistry to more advanced methods courses. Young students enrolling each year in a Bachelor program Chemistry and Biochemistry receive a sound basic education in both fields and can decide during the first years in which field they want to specialize and continue their studies in advanced Master courses.

Over the last five years 150 students have been selected for the Master program in Biochemistry. This program optimally prepares young scientists for a future career in academia and industry. All courses are taught in English, which improves students' language skills, enables international Professors to contribute to teaching and makes the program very attractive for foreign students. In fact, currently about 40% of our master students are from abroad. In 2016 new study rules were implemented and the choice of eighteen subjects in various combinations leaves students with a high flexibility and many options to pursue their interests.

The Gene Center is situated on the Campus Grosshadern/Martinsried and embedded in a community of institutes in the life and medical sciences ranging from different Faculties of the LMU, the Helmholtz- and Max-von-Pettenkofer Institutes and two Max-Planck Institutes. The close proximity of education and research provides a state-of-the-art environment and guarantees students to be taught at a high standard. Well-equipped labs allow for hands-on teaching of modern methods in practical courses. In addition, students are encouraged to obtain research experience in laboratories both on campus and abroad by doing internships or carrying out their master thesis projects nationally or internationally.

The high standard of education and training owes much to the commitment of the coordinator of our Master Program, **Johanna Turck**, and the coordinator of the biochemistry practical courses, **Louiza Papatheodorou**. Together with our professors and lecturers they organize teaching and support students with advice. **Birgitta Beatrix** should be mentioned here as a representative of the institute's staff scientists who are very committed to teaching and very active in adapting our courses to changing teaching needs.

Graduate Training

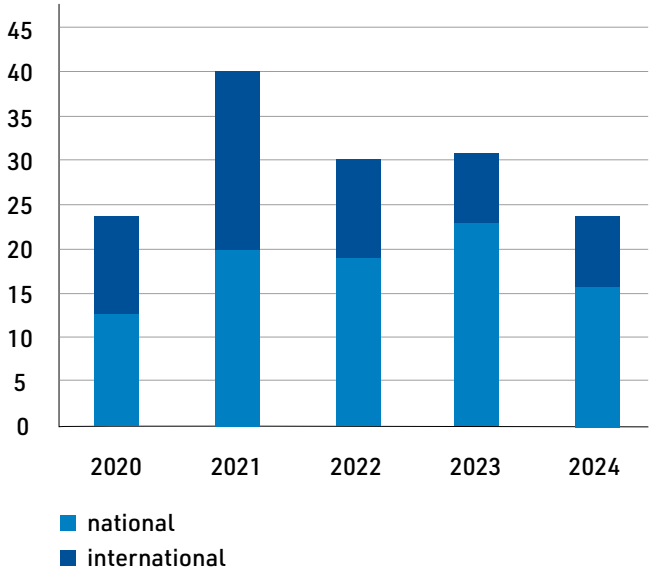
Our commitment to fostering young scientific talent continues through dedicated support for PhD students at the Gene Center. As the driving force behind much of the institute's research, PhD students are at the heart of our scientific community. Between 2020 and 2024, 149 students completed their PhDs in Biochemistry at LMU, with 49% identifying as female and 39% coming from international backgrounds. Graduate students are enrolled in the Gene Center's structured graduate program, which includes a biannual retreat and a weekly research seminar series. This series features both internationally renowned speakers and presentations by Gene Center PhD students and postdoctoral researchers, providing a platform for scientific exchange and visibility. Their training is further enriched by transferable skills courses offered by the GraduateCenterLMU, focusing on scientific writing and oral presentation. Since the 2024 retreat, a PhD student-led initiative has introduced a monthly happy hour to promote informal exchange and networking among early-career researchers.

Many PhD students at the Gene Center also take part in specialized graduate schools and training programs. These include, among others, the International Max Planck Research School for Molecules of Life (IMPRS-ML), the successor to IMPRS-LS, based at the neighboring Max Planck Institute of Biochemistry.

Since 2012, two graduate schools funded by the German Research Foundation (DFG) have been located at the Gene Center. The Research Training Group on ‚Integrated Analysis of Macromolecular Complexes and Hybrid Methods in Genome Biology', coordinated by Karl-Peter Hopfner, concluded in 2021 after successfully completing its second and designated final funding period. Also established in 2012 under the German Excellence Initiative, the Graduate School of Quantitative and Molecular Biosciences Munich (QMB)–originally named Quantitative Biosciences Munich–expanded its thematic focus in 2024. This broadening of scope led to the current name, reflecting its wider scientific reach. The school is coordinated by **Roland Beckmann** and **Erwin Frey**, and brings together investigators from LMU, the Technical University of Munich (TUM), the Max Planck Institute of Biochemistry, and Helmholtz Center Munich. Administrative coordination is led by **Beate Hafner**, in collaboration with lecturer **Christophe Jung**, who supports the program in mathematics, physics, and programming.

In addition, Gene Center PhD students are actively involved in six different Collaborative Research Centers, further enhancing interdisciplinary training and scientific exchange.

PhD theses in Biochemistry



Networks That Drive Innovation and Impact

Since its establishment in 1984, the Gene Center has been a leading research institution, renowned for its scientific excellence, interdisciplinary collaboration and pioneering innovation in biochemistry and medical research.

Connecting Minds, Creating Value

It is therefore no surprise that the network programs initiated by the Bavarian State Ministry for Science and the Arts since 2003–bringing together the expertise of Bavaria's top scientists–are anchored at this innovative hub.

The coordinating office, which is responsible for these programs, benefits greatly from the Gene Center´s inspiring, innovation-driven environment. Located in the BioSysM building, the office offers an ideal workspace and facilities for hosting guests and organizing events in an international and dynamic setting.

The basic idea for this kind of integrated research networks was conceptualized by Prof. Ernst-Ludwig Winnacker, the founder and intellectual father of the Gene Center, who remains in close and regular contact with the coordinating office staff up to this day.

These network programs include:

- **BayGene** (Functional Genomics Research, (2004-2012))
- **BioSysNet** (Molecular Biosystems Biology Research, (2012-2018)
- **bayklif** (Climate Research, (2018-2023)
- **bayresq.net** (Research on Multidrug-Resistant Pathogens, (2019-2025)

This year, the Bavarian Climate Research Network was extended for another five-year funding period and expanded to include new cross-cutting themes. This reinforces interdisciplinary research on the challenges of climate change in Bavaria in a sustainable manner.

Results That Shape the Future

The current Network concerning life science, bayresq.net, is in it's final stage . For six years, intensive research was conducted on innovative approaches to combat multidrug-resistant pathogens. The network's focus was on basic research aimed at developing new strategies to fight antibiotic resistant infections.

In the **DynamicKit** project, scientists made significant progress researching dormant tuberculosis bacteria. They developed new methods to analyze the intact proteins of these resilient bacteria and to study their response to antibiotics. They also established a new technique to specifically detect changes in proteins, which is an important step toward a better understanding of resistance mechanisms. Additionaly, MALDI-TOF mass spectrometry was employed to swiftly detect protein alterations and to develop novel combination therapies against tuberculosis. This project contributes significantly to the fight against antibiotic-resistant infections and promotes medical progress in Bavaria.

In the **Helicopredict** project, a collection of over 500 Helicobacter pylori isolates was established and tested extensively for resistance to three clinically important antibiotics. Using whole-genome sequencing and advanced statistical methods, models were developed that can predict resistance based solely

on genetic data. This dataset provides the foundation for new diagnostic tools that enable targeted, resistance-based therapy in clinical settings.

As part of the **Metabodefense** project, metabolic and gene expression analyses of infected cells were conducted and evaluated using computational methods. These analyses led to the identification of new approaches for detecting metabolic signals for diagnostics and for strengthening the body's own defense mechanisms. Since bacteria can develop resistance to many antimicrobial agents, supporting the immune response is considered a promising complement to antibiotic therapy. This opens up new possibilities for the precise detection and effective treatment of infections.

In the **IRIS** project, a comprehensive collection of multidrug-resistant Staphylococcus epidermidis isolates from clinical samples was first established and characterized genomically. Subsequently, strains that specifically activate dendritic cells, particularly DC2, were identified. These strains contribute to the activation of antigen-specific memory T cells. S. epidermidis-mediated immunomodulatory effects were discovered that can dampen certain immune responses – an approach relevant

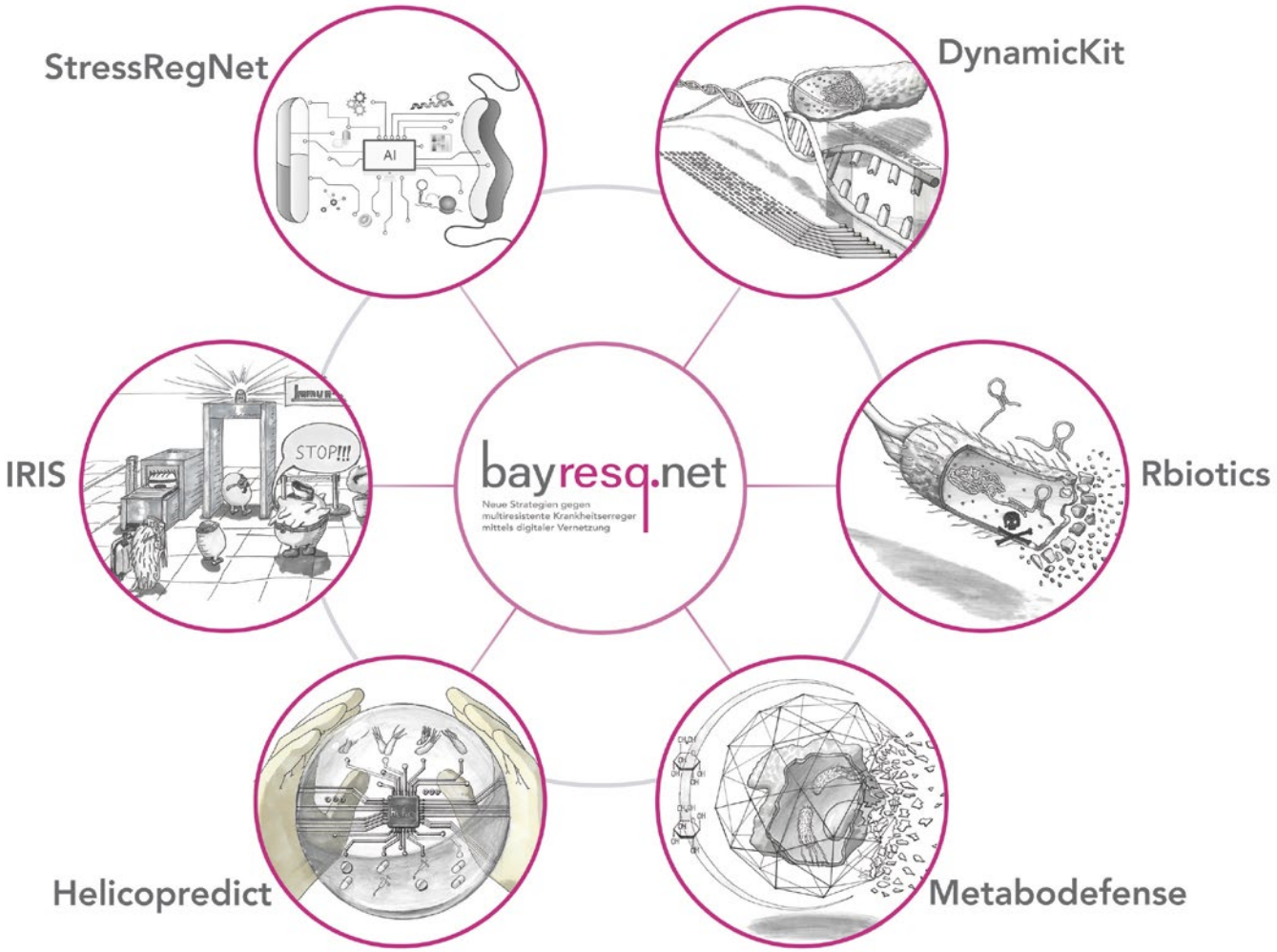
to developing new immunotherapies. Thus, the project opens up new perspectives for tackling antibiotic resistance and demonstrates how interactions between immune cells and the microbiome can be harnessed to modulate immune reactions deliberately.

The **Rbiotic** project developed an experimental pipeline to evaluate the effectiveness of antisense oligonucleotides (ASOs) against various gut bacteria and pathogens. The goal was to identify suitable target genes whose inhibition would impair bacterial survival or virulence. Additionally, criteria were established for ASO design and predicting potential off-target effects. With MASON, an interactive web interface, custom ASOs can be designed. RNA sequencing is used to better understand the impact of ASO treatment. This method captures immediate changes in bacterial gene expression and provides valuable insights into the compound´s efficacy.

The **StressRegNet** project took an innovative approach to combating antibiotic-resistant bacterial pathogens. It focused on two of the most common causes of foodborne illnesses worldwide: Salmonella Typhimurium and Campylobacter jejuni. The goal was to identify substances that can weaken or kill these bacteria. Due to the large number of potential effective compounds, researchers used artificial intelligence and data science to systematically analyze their effects. These insights support the development of new therapeutic strategies using host-specific metabolites, food additives, and non-antibiotic drugs and enable the targeted prediction of antimicrobial properties in previously untested substances.

Planning Beyond Tomorrow

Since all past networks have demonstrated that cooperation and competition – at the same time – form an excellent basis for successful research, we are convinced that similar networks, but on different topics, will continue to be an important element of the Bavarian scientific ecosystem.



Barbara Adler

2025

Identification of the human cytomegalovirus gHgLgO trimer as the central player in virion infectivity.
Thiessen L, Garuti R, Kubic L, Kösters M, Amarambedu Selvakumar D, Krey T, Görzer I, Fröhlich T, **Adler B**.
PLoS Pathog. 2025 Jul 24;21(7):e1013341.
doi: 10.1371/journal.ppat.1013341

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Johanna Klughammer

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LAFUGA

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Speaker	Institution	Title	Date
2025			
Oliver Ladendorf and Hannah Fischer	Law firm Kraus & Lederer	From Innovation to IP: Navigating Patents in Biotech and Pharma	23.07.25
Daphne Selvaggia Cabianca	Helmholtz Munich	Shaping the genome with diet: dietary control of chromatin organization and function in a whole organism	07.07.25
Oleh Khalimonchuk	University of Nebraska-Lincoln, USA	Regulation of mitochondrial shape and function: From basics to disease	06.02.25
Evangelos Karousis	University of Bern, CH	Decoding viral secrets using cell-free translation	12.05.25
Marieke Oudelaar	MPI for Multidisciplinary Sciences, Göttingen	Molecular drivers of 3D genome folding and their function in gene regulation	08.04.25
2024			
Alessandro Scacchetti	University of Pennsylvania, USA	Biochemical and genetic dissection of extracellular RNA biogenesis	09.12.24
Nadine Schwierz	University of Augsburg	Ions, nucleic acids and ionizable lipids: Highly charged problems for biomolecular simulations	04.11.24
Shravan Kumar Mishra	Indian Institute of Science Education and Research Mohali, India	Origin and Function of the Ubiquitin Fold in Sde2	21.10.24
Thomas Tuschl	Rockefeller University, USA	Developing inhibitors of viral and metazoan mRNA cap N7-G methyl transferases	23.09.24
The Munich Gene Center Symposium –	Gene Center Munich	Celebrating 40 Years of Discovery and Envisioning Tomorrows Science	24.06.24
Guifen Wu	Aarhus University, Denmark	The crossroad of gene expression: Nuclear export or decay?	17.06.24
Stephanie Fesser	BioNTech SE, Mainz	Design and production of mRNA therapeutics	10.06.24
Graeme Hewitt	King's College London, UK	Using CRISPR screens to map genetic determinants of radiation response	21.05.24
Marina Chekulaeva	Berlin Institute for Medical Systems Biology	RNA regulation in neurons and neurodegeneration	13.05.24
Jan Rehwinkel	University of Oxford, UK	MDA5 guards against infection by surveying cellular RNA homeostasis	06.05.24
Horizons 20XX: Julia Mahamid	EMBL Heidelberg	Enabling Discovery by In-Cell Structural Biology	15.04.24
Horizons 20XX: Rotem Sorek	Weizmann Institute of Science, Israel	The immune system of bacteria: Beyond CRISPR	18.03.24
Katharina Höfer	MPI Marburg	The best of two worlds: RNAylation covalently links RNA to proteins	19.02.24
Marvin Tanenbaum	Hubrecht Institute, Utrecht, The Netherlands	Dissecting post-transcriptional gene expression regulation in humans and viruses	08.02.24
Pierre Savatier	University of Lyon, France	The challenges of pluripotent stem cell-based systemic chimeras in rabbits and primates	05.02.24
Horizons 20XX: Karsten Borgwardt	MPI of Biochemistry, Martinsried	Machine Learning in Systems Biology: Now and Next	29.01.24
Felix Randow	MRC Laboratory of Molecular Biology, Cambridge, UK	How cells defend their cytosol against bacteria: LPS ubiquitylation and other tricks	22.01.24
2023			
Matthias Geyer	University of Bonn	Cryo-EM structures of the NLRP3 inflammasome	11.12.23
Shixin Liu	Rockefeller University, USA	Machines on Genes: A Single-Molecule Perspective	20.11.23
Bennett van Houten	UPMC-Hillman Cancer Center, Pittsburgh, USA	A new single molecule approach to study DNA repair protein dynamics: seeing is believing.	15.11.23

Speaker	Institution	Title	Date
Antonina Roll-Mecak	National Institutes of Health, USA	How cells read and write the tubulin code	19.10.23
Suckjoon Jun	UCSD, USA	Bacterial replication initiation as precision control by protein counting	09.10.23
Susi Bantele	University of Copenhagen, Denmark	Repair of DNA double strand breaks leaves heritable scars to chromatin function	03.07.23
Jacob Corn	ETH Zürich, Switzerland	Better genome editing by listening to the cells	26.06.23
Eiji Kobayashi	Keio University, Japan	The Pig as an In vivo Bioreactor for Human Organs	21.06.23
Helle Ulrich	IMB Mainz	Table Manners for Cells: Knife & Fork Etiquette during DNA Replication	19.06.23
Laura Czech	University of Marburg	Bacterial stress management: from membrane protein biogenesis to interactions with bacteriophages	12.06.23
Yukihide Tomari	University of Tokyo, Japan	The mechanism of RNA silencing and beyond	22.05.23
Markus Höpfler	MRC Laboratory of Molecular Biology, Cambridge, UK	Mechanism of regulated tubulin mRNA degradation	17.04.23
Alena Khmelinskaia	LiMES, Bonn	Expanding the repertoire of de novo protein assemblies	27.03.23
Johannes Walter	Harvard Medical School, USA	Discovering Mechanism with AlphaFold: A Case Study from Vertebrate DNA Replication	20.03.23
Mohammad Lotfollahi	Helmoltz-Zentrum Muenchen	Deep learning for multi-scale analysis of single-cell data to understand health and disease	27.02.23
Alexander Stark	IMP Vienna, Austria	Decoding transcriptional regulation	06.02.23
2022			
Rune Hartmann	Aarhus University, Denmark	Two cGAS-like receptors induce Sting-dependent antiviral immunity in Drosophila melanogaster via the NF-κB pathway	12.12.22
Simon Boulton	Francis Crick Institute, UK	Chromosome end protection	05.12.22
Daniel Durocher	University of Toronto, Canada	Charting genome maintenance networks: new biology and therapeutic opportunities	28.11.22
Cecilia Dominguez-Conde	Human Technopole, Italy	Human immune cells across tissues and life stages	21.11.22
Alberto Ciccia	Columbia University Irving Medical Center, USA	Replication-coupled repair: from gap suppression to immune evasion	14.11.22
Stefan Oehlers	Institute for Infectious Diseases, Sydney, Australia	Insights into mycobacterial pathogenesis from zebrafish infection models	12.09.22
Daniel Reichart	LMU Hospital	Single cell analyses of healthy and diseased human hearts	25.07.22
Toshifumi Inada	University of Tokyo, Japan	Mechanisms and physiological roles of quality controls that ensure translation quality	18.07.22
Maria Hondele	University of Basel, Switzerland	DEAD-box ATPases are global regulators of phase-separated organelles	04.07.22
Gaëlle Legube	Centre de Biologie Intégrative, Toulouse	Chromatin and chromosome dynamics at DNA double strand breaks	13.06.22
Leif Ludwig	Max Delbrück Center for Molecular Medicine, Berlin	Single Cell 'Mitochondrial' Genomics – from Lineage Tracing to Human Phenotypes	23.05.22
Marianne Bauer	Princeton University, USA	Sensing transcription factors in the fly embryo	25.04.22
Ziga Avsec	TUM, DeepMind	Learning the sequence grammar of gene expression using AI	14.03.22
Gabriel Núñez	University of Michigan, USA	Host-Microbiota Interactions in Health and Disease	07.03.22
Kikue Tachibana	MPI Biochemistry, Martinsried	The establishment of 3D genome architecture at the start of life	07.02.22
Greg Newby	Broad Institute of MIT and Harvard, USA	Precision Genome Editing to Correct Genetic Disease	11.01.22

Speaker	Institution	Title	Date
2021			
Simon Ausländer	Roche GmbH, Penzberg	Addressing manufacturing challenges of complex antibody formats	08.11.21
Heiko Lickert	Helmholtz Zentrum München	Beta cell development, heterogeneity and regeneration	25.10.21
Francesca Mattiroli	Hubrecht Institute, Utrecht, The Netherlands	Mechanism of CAF-1 mediated nucleosome assembly during DNA replication	18.10.21
Camilo Perez	University of Basel, Switzerland	Mechanism of cell wall transporters and role in virulence of bacterial pathogens	11.10.21
Stephanie Panier	MPI for Biology of Ageing, Cologne	The good, the bad and the ugly: Telomere recombination in the Alternative Lengthening of Telomeres pathway	12.07.21
Mihaela Pertea	Johns Hopkins School of Medicine, USA	Towards a new comprehensive human gene catalogue	28.06.21
David Pincus	University of Chicago, USA	The Heat Shock Response in Time: Initiation Signals and Feedback Loops	07.06.21
Susanna Manrubia	CSIC, Madrid, Sapin	How the architecture of hyperastronomically large genotype spaces shapes evolutionary dynamics	28.05.21
Scott Blanchard	St. Jude Children's Research Hospital Memphis, USA	Variant Ribosomal RNA Alleles Are Conserved, Exhibit Context-Specific Expression and Regulate Gene Expression	15.03.21
Tanja Kortemme	University of California, San Francisco, USA	Sensors and new shapes: Computational design of new molecular geometries and ligand-controlled functions	02.03.21
Lori Passmore	MRC Laboratory of Molecular Biology, Cambridge, UK	Molecular insights into DNA crosslink repair	01.03.21
Rachel Green	The Johns Hopkins University School of Medicine, USA	How ribosome collisions drive cellular signaling pathways	22.02.21
Maria Tanzer	MPI for Biochemistry, Martinsried	Proteomic profiling of cell death and inflammatory pathways	15.02.21
Dan Bachovchin	Memorial Sloan Kettering Cancer Center, New York, USA	Activation of the NLRP1 and CARD8 inflammasomes	08.02.21
Michiel Vermeulen	Radboud Institute for Molecular Life Sciences, The Netherlands	Deciphering gene expression regulation in health and disease using integrative omics approaches	01.02.21
David Dulin	Interdisciplinary Center for Nanostructured Films, Erlangen	Mechanochemistry and drug targeting of the SARS-CoV-2 replicase: a single molecule perspective	25.01.21
2020			
Maren Büttner	Institute of Computational Biology, Helmholtz Zentrum München	scCODA - detecting compositional cell-type changes in single-cell data	14.12.20
Gaia Pigino	MPI of Molecular Cell Biology and Genetics, Dresden	Towards a mechanistic understanding of motile and primary cilia with cryo-electron tomography	30.11.20
Jernej Ule	The Francis Crick Institute London, UK	Studies of TDP-43 uncover new roles of phase separation in RNA regulatory networks	09.11.20
Karl Duderstadt	MPI for Biochemistry, Martinsried	Sliding, Pushing, Crashing: the treacherous pathways of replication origin selection in a crowded world	02.11.20
Achim Tresch	University of Cologne	Quantification of RNA synthesis, nuclear export and degradation and functional consequences	19.10.20

Date	Title	Investigator	Media
30.06.25	Innate immune system: How modified RNA tricks the immune system	Veit Hornung	LMU press release
30.06.25	Concentrated expertise for medical research transfer	Eckhard Wolf	LMU interview
06.03.25	Was passiert im Körper bei einer Chemotherapie?	Sebastian Theurich	Podcast CCC Munich
22.05.25	NUCLEATE Cluster of Excellence: beacon of nucleic acid research	Veit Hornung	LMU press release
15.04.25	Sport und Bewegung bei Krebs	Sebastian Theurich	Podcast CCC Munich
10.04.25	Veit Hornung, winner of the 2025 Collen Jeantet Prize for Translational Medicine	Veit Hornung	YouTube: Foundation Louis-Jeantet
27.02.25	"Let us not forget the most vulnerable": A Conversation with Prof. Christoph Klein	Christoph Klein	WXPress
20.02.25	Overlooked alarm signal: RNA damage moves into the spotlight	Julian Stingele	LMU interview
04.02.25	Gamechanger in der Krebsmedizin	Marion Subklewe	Frankfurter Allgemeine Zeitung / Verlagsspezial
29.01.25	Christoph Klein: Seltene Erkrankungen, Gentherapien, Kinderwürde, Ethik	Christoph Klein	YouTube Lehmann Podcast
15.01.25	Polio im Münchner Abwasser: Wer jetzt schnell nachimpfen sollte	Oliver T. Keppler	Süddeutsche Zeitung
22.12.24	Lage bei Corona und Grippe vor Weihnachten relativ entspannt	Oliver T. Keppler	Süddeutsche Zeitung
22.12.24	Risiko von Corona und Grippe bleibt zu Weihnachten überschaubar	Oliver T. Keppler	Süddeutsche Zeitung
19.12.24	Patientenfragestunde: „Sport und Bewegung bei Krebs"	Sebastian Theurich	BRCA-Netzwerk e.V.
10.12.24	Prof. Algorithmus - So revolutioniert Künstliche Intelligenz die Forschung	Karl-Peter Hopfner	Bayern 2 Podcast IQ - Wissenschaft und Forschung
28.11.24	Animal welfare: reducing lab experiments on primates	Eckhard Wolf	LMU press release
19.11.24	18 LMU-Forschende unter den „Highly Cited Researchers"	Veit Hornung	LMU press release
17.09.24	Leichtes Spiel für Viren auf dem Oktoberfest	Oliver T. Keppler	Süddeutsche Zeitung
09.09.24	Neue Chancen bei Krebs	Marion Subklewe	tz
09.09.24	Immuntherapie rettet Krebspatienten	Marion Subklewe	Münchner Merkur
05.09.24	The key to centromere's eternal life unravelled	Jeyaprakash Arulanandam	LMU press release
16.07.24	Abwasser zeigt „moderate" Corona-Welle in Bayern	Oliver T. Keppler	Zeit Online

Date	Title	Investigator	Media
16.07.24	Abwasser zeigt „moderate" Corona-Welle in Bayern	Oliver T. Keppler	Süddeutsche Zeitung
24.06.24	Pacesetter for the life sciences	Karl-Peter Hopfner	LMU interview
21.06.24	Löst Mikroplastik Erektionsstörungen aus?	Marcia Ferraz	Spielgel Wissenschaft
18.06.24	Das Genzentrum der LMU wird 40	Karl-Peter Hopfner	LMU press release
05.06.24	Im Kurzinterview: Wie kommen Innovationen bei den Patienten an?	Marion Subklewe	Frankfurter Allgemeine Zeitung / Verlagsspezial
28.05.24	Xenotransplantationen - Wie weit ist die Forschung?	Eckhard Wolf	3Sat NANO
08.05.24	Snapshots of the architecture of life	Roland Beckmann	LMU press release
02.05.24	Activation of innate immunity: Important piece of the puzzle identified	Veit Hornung	LMU press release
25.04.24	Gene regulation: modification in the nucleosome jungle	Johannes Stigler	LMU press release
22.04.25	LMU immunologist Veit Hornung wins ERC Advanced Grant	Veit Hornung	LMU press release
10.04.24	CAR-T-Zellen - Nutzen und Risiken	Marion Subklewe	ZDF, Volle Kanne
10.04.24	Cockayne syndrome: new insights into cellular DNA repair mechanism	Julian Stingele	LMU press release
07.01.24	Xenotransplantation: Schweineherzen als Lebensretter	Eckhard Wolf	ZDF heute
07.12.23	Structure of a central component of the human immune system revealed	Veit Hornung	LMU press release
29.11.23	Hearts and minds	Eckhard Wolf	LMU press release
10.11.23	Colliding ribosomes activate RNA repair	Julian Stingele	LMU press release
06.11.23	Five ways in which rookie lab leaders can get up to speed	Marcia Ferraz	LMU press release
11.10.23	Prof. Dr. Eckhard Wolf: Xeno-Transplantation	Eckhard Wolf	BR IQ-Wissenschaft und Forschung
06.10.23	Lucas Jae receives Vallee Scholarship	Lucas Jae	LMU press release
12.09.23	Distinction for research project: how bacteria fend off viruses	Veit Hornung	LMU press release
01.09.23	Advancing stem cell-based therapy for type 1 diabetes	Eckhard Wolf	LMU press release
10.07.23	Animal mode: prediction of therapeutic potential in Duchenne muscular dystrophy	Eckhard Wolf	LMU press release

Date	Title	Investigator	Media
27.04.23	Structural insight into process of gene regulation	Karl-Peter Hopfner	LMU press release
11.04.23	Waisen der Medizin - Wenn Kinder schwer erkranken	Christoph Klein	BR IQ-Wissenschaft und Forschung
29.03.23	WHO ändert Impfempfehlung: Covid-Booster-Impfung nur noh für Risikogruppen	Oliver T. Keppler	Merkur
01.03.23	LMU-Professor über Vogelgrippe-Ausbrüche: „Wahrscheinlichkeit hat sich erhöht“	Oliver T. Keppler	Abendzeitung
06.02.23	Science Talks an der LMU - Forschung zu Risiken künftiger Pandemien	Oliver T. Keppler	YouTube: LMU
02.02.23	The Impact of Alphafold on Protein Research	Karl-Peter Hopfner	Center of Advanced Studies: Panel Discussion
23.01.23	Viren: Unsichtbare Gefahr aus der Wildnis	Oliver T. Keppler	LMU News
15.01.23	Keppler empfiehlt Frühwarnsystem für neue Corona-Varianten	Oliver T. Keppler	Süddeutsche Zeitung
15.01.23	Virologe empfiehlt Covid-Frühwarnsystem für neue Varianten	Oliver T. Keppler	BR24
13.01.23	Virologe Oliver Keppler: Grippewelle flacht ab	Oliver T. Keppler	Süddeutsche Zeitung
13.01.23	Münchner Virologe Keppler: Corona verschiebt Grippewelle	Oliver T. Keppler	BR24
31.12.22	Reisende aus China: Virologe Keppler gegen schärfere Kontrollen	Oliver T. Keppler	BR24
22.12.22	Was ist bloß mit den Viren los?	Oliver T. Keppler	Süddeutsche Zeitung
23.11.22	Das Ende der Maskenpflicht naht	Oliver T. Keppler	Süddeutsche Zeitung
16.11.22	Fifteen LMU scientists and academics make Highly Cited Researchers list	Veit Hornung	LMU press release
12.11.22	Ein Überblick über die wichtigsten Fragen zum Ende der Isolationspflicht	Oliver T. Keppler	Süddeutsche Zeitung
17.10.22	Corona-Schnelltests	Oliver T. Keppler	Deutschlandfunk
24.08.22	Innate immunity: the final touch for antimicrobial defence	Lucas Jae, Veit Hornung	LMU press release
23.08.22	„Die Wiesn wird einen enormen Einfluss auf das Infektionsgeschehen haben“	Oliver T. Keppler	Süddeutsche Zeitung
23.08.22	Die Wiesn naht – mit ihr die Corona-Welle?	Oliver T. Keppler	Süddeutsche Zeitung
18.08.22	Falsches Sicherheitsgefühl: das große Missverständnis um die Corona-Schnelltests	Oliver T. Keppler	Welt+
18.08.22	Corona und die Wiesn 2022	Oliver T. Keppler	BR24, Merkur

Date	Title	Investigator	Media
18.08.22	Virologe zur Wiesn: „Das ist synchronisiertes Superspreading“	Oliver T. Keppler	BR24
28.07.22	Die Martinsried-Story	Karl-Peter Hopfner	Süddeutsche Zeitung
22.07.22	Corona-Vorsorge für den Herbst	Oliver T. Keppler	LMU press release
20.06.22	Alfried Krupp Prize awarded to Lucas T. Jae	Lucas Jae	LMU press release
06.04.22	Cell biology: How mitochondria report stress	Lucas Jae	LMU press release
01.04.22	„Viele Schnelltests erkennen Omikron schlechter als andere Varianten“	Oliver T. Keppler	Süddeutsche Zeitung
01.04.22	Selbsttest und Maskenpflicht	Oliver T. Keppler	BR3
18.03.22	„Deutschland ist ein großer Hotspot“	Oliver T. Keppler	BR24 TV
16.03.22	Corona Lockerungen und steigende Infektionszahlen	Oliver T. Keppler	Zeit Online
16.03.22	„Wird Zahlen stark befeuern“: Münchner Virologe sieht Sommer-Probleme auf uns zukommen	Oliver T. Keppler	Merkur
16.03.22	Virologe Keppler: Durchseuchung darf nicht unser Ziel sein	Oliver T. Keppler	BR24
10.03.22	Bacterial ribosomes: molecular collision activates protection mechanism	Roland Beckmann	LMU press release
28.02.22	Können Corona-Schnelltests die dominierende Omikron-Variante erkennen?	Oliver T. Keppler	Frankfurter Allgemeine Zeitung
24.02.22	Corona-Tests „Das sind alles elementare Mängel“	Oliver T. Keppler	Süddeutsche Zeitung
23.02.22	Schnelltst-Zentren: Null Prozent sind null plausibel	Oliver T. Keppler	Tagesschau
23.02.22	Labore befürchten Untererfassung von Corona-Fällen	Oliver T. Keppler	Rhein-Neckar Zeitung
22.02.22	Antigen-Schnelltests	Oliver T. Keppler	BR Gesundheit
22.02.22	Forschungspionier Eckhard Wolf: „Klonen von Menschen sollte absolut tabu bleiben“	Eckhard Wolf	Deutschlandfunk
16.02.22	Omikron	Oliver T. Keppler	Süddeutsche Zeitung
16.02.22	Virologe nennt neue Schnelltest-Daten im BR „ernüchternd“	Oliver T. Keppler	Münchner Merkur
12.02.22	„Acht von neun Antigen-Schnelltests erkennen Omikron schlechter als Delta“	Oliver T. Keppler	Welt/Welt am Sonntag
12.02.22	Schnelltests mit Omikron-Schwächen	Oliver T. Keppler	tagesschau

Date	Title	Investigator	Media
12.02.22	Este Lockerungen in Bayern, unzuverlässige Schnelltests bei Omikron	Oliver T. Keppler	BR Rundschau
12.02.22	Corona Schnelltests: Die große Schnelltest-Misere	Oliver T. Keppler	Zeit Online
03.02.22	Schweineherzen: Rettung für schwerkranke Patienten?	Eckhard Wolf	Stern
01.02.22	Covid-19: Drei Kontakte mit Spike-Protein helfen, Immunität aufzubauen	Oliver T. Keppler	LMU press release
24.01.22	Virologe Keppler: Omikron ist „kein milder Erreger“	Oliver T. Keppler	BR24, ARD
22.01.22	Virologe: Omikron nicht „mild“ - Kliniken vor neuer Welle	Oliver T. Keppler	Süddeutsche Zeitung
16.01.22	Das Test-Dilemma	Oliver T. Keppler	Welt am Sonntag
10.01.22	Corona Tests brauchen einen Test	Oliver T. Keppler	Handelsblatt
10.01.22	Unsichere Schnelltests	Oliver T. Keppler	Deutschlandfunk
09.01.22	Schnell- und Selbsttests	Oliver T. Keppler	Bericht aus Berlin
23.12.21	HIV infection: Better understanding the reservoir of virus in the body	Oliver T. Keppler	LMU press release
01.12.21	Mit CAR-T-Zell-Therapie erfolgreich gegen Krebs	Marion Subklewe	BioM Podcast
30.11.21	Gerät die 4. Welle außer Kontrolle?	Oliver T. Keppler	report München
28.11.21	Weiterer Omikron-Verdachtsfall in Bayern bestätigt	Oliver T. Keppler	BR24
27.11.21	Vierte Welle und nun Omikron	Oliver T. Keppler	BR24
27.11.21	Omikron-Variante: Zwei Fälle in Bayern bestätigt	Oliver T. Keppler	BR24
26.11.21	Fragen und Antworten zur Corona-Impfung	Oliver T. Keppler	YouTube: BR24live
26.11.21	Virologe Keppler: „Kontakte reduzieren, Maske tragen, impfen“	Oliver T. Keppler	BR24
16.11.21	Fifteen LMU scientists and academics make Highly Cited Researchers list	Veit Hornung	LMU press release
15.11.21	Aktuell: Corona-Lage	Oliver T. Keppler	3sat Wissen Nano
14.11.21	Vierte Corona-Welle: Wie kommen wir durch den Winter?	Oliver T. Keppler	BR24
11.11.21	Bayern ruft den Corona-Katastrophenfall aus: Brauchen wir eine Impfpflicht für alle?	Oliver T. Keppler	Bayern 2

Date	Title	Investigator	Media
08.11.21	Vierte Corona-Welle	Oliver T. Keppler	BR24 extra
22.10.21	Schweine als Organspender – das Immunsystem ist die große Hürde	Eckhard Wolf	Die Welt
15.10.21	„Dieses Virus wird uns erhalten bleiben“	Oliver T. Keppler	AZ
14.10.21	Corona-Lage	Oliver T. Keppler	Main-Echo
11.10.21	Qualitätskontrolle bei Bürgertests	Oliver T. Keppler	Deutschlandfunk
28.09.21	Structural biology: Mechanisms of novel anti-cancer drugs elucidated	Karl-Peter Hopfner	LMU press release
22.09.21	New research center for Grosshadern/Martinsried	Eckhard Wolf	LMU press release
10.09.21	Diabetes research: A new model for studies of beta-cell function	Eckhard Wolf	LMU press release
30.08.21	Julian Stingele receives generously endowed scholarship	Julian Stingele	LMU press release
21.07.21	Krebsimmuntherapie: Erste ambulante CAR-T-Zell-Therapie - Eine Studie zeigt neue Wege auf	Marion Subklewe	LMU Klinikum Pressemitteilung
19.07.21	Studie Lymphdrüsenkrebs: ambulante Behandlung	Marion Subklewe	BR Fernsehen
05.07.21	Pandemie: Ruhiger Sommer, gefährlicher Herbst	Oliver T. Keppler	Süddeutsche Zeitung
21.05.21	Antigen-Schnelltests	Oliver T. Keppler	Straubinger Tagblatt/Landshuter Zeitung
10.05.21	Tests in der Pandemie – Wie zuverlässig sind Antigen-Schnelltests?	Oliver T. Keppler	tagesschau
06.05.21	Interview mit Herrn Prof. Dr. phil. h.c. mult. Lejeune	Oliver T. Keppler	YouTube „The Lejeune Academy“
26.04.21	Schnelltests: trügerische Sicherheit?	Oliver T. Keppler	WISO
20.04.21	Gefährlicher Boom? Unsichere Corona-Schnelltests für jedermann	Oliver T. Keppler	Report Mainz
17.04.21	Die Test-Illusion und Defekte Notbremse	Oliver T. Keppler	Der Spiegel
13.04.21	Interview: Falsche Sicherheit	Oliver T. Keppler	BR
06.04.21	„Brauchen sofort harten Lockdown“: Interview zum Brücken-Lockdown	Oliver T. Keppler	Tagesschau
24.03.21	„Wo stecken Menschen sich an?“	Oliver T. Keppler	Bayern 2
02.02.21	Die Corona-Impfung: Fakten, Fakten, Fakten	Oliver T. Keppler	YouTube: Wirtschaftsbeirat Bayern

Date	Title	Investigator	Media
27.01.21	Verbreitung der Corona-Mutationen	Oliver T. Keppler	Berliner Zeitung
19.01.21	Bedingt aussagekräftig	Oliver T. Keppler	Berliner Zeitung
18.01.21	Virenschutz: Seit heute FFP2-Maskenpflicht	Oliver T. Keppler	BR Fernsehen
15.01.21	Sequenzierung von SARS-CoV-2 zur Untersuchung des aktuellen Ausbreitungsgeschehens	Max Muenchhoff	Galileo ProSieben
05.01.21	Lockdown	Oliver T. Keppler	BR Fernsehen
27.12.20	Die Corona-Impfung hat begonnen	Oliver T. Keppler	BR Fernsehen
07.12.20	Kein Freifahrtschein	Oliver T. Keppler	Süddeutsche Zeitung
07.12.20	EMBO Young Investigators	Julian Stingele	LMU press release
29.11.20	Der bittere Mediziner	Oliver T. Keppler	FAZ
27.11.20	How epithelial cells ward off viruses	Veit Hornung	LMU press release
20.11.20	LMU academics among most highly cited researchers	Veit Hornung	LMU press release
20.11.20	Lockdown & Infektionszahlen	Oliver T. Keppler	BR Fernsehen
09.11.20	Corona Impfstoff	Oliver T. Keppler	BR Fernsehen
09.10.20	Corona-Infektionszahlen steigen rasant: Sind wir zu unvorsichtig geworden?	Oliver T. Keppler	Bayern 2, ARD Alpha
10.09.20	Veit Hornung mit Coley-Award ausgezeichnet	Veit Hornung	LMU press release
10.09.20	How cGAS is kept bottled up	Karl-Peter Hopfner, Veit Hornung	LMU press release
03.09.20	Sofja Kovalevskaja Awardee at LMU	Marcia Ferraz	LMU press release

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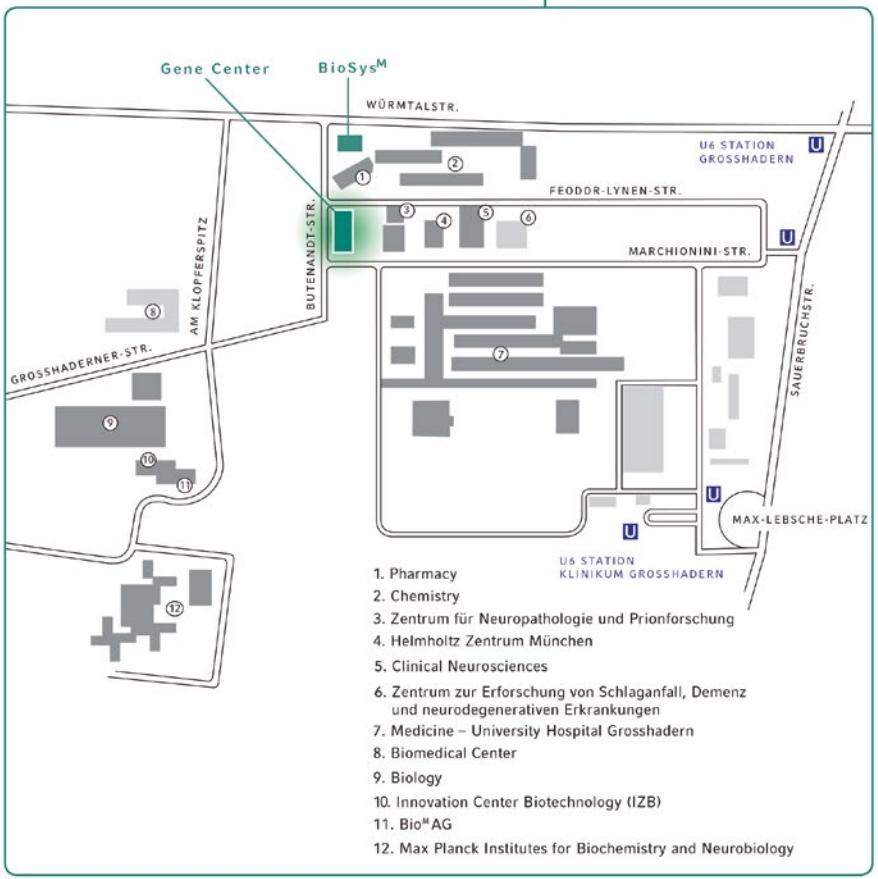

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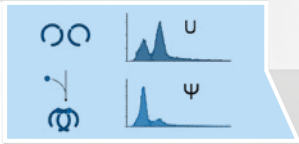
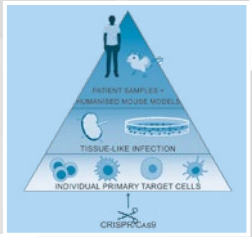
Some of the Latest Research Highlights at the Gene Center

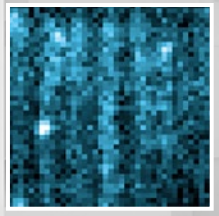
Pseudouridine RNA avoids immune detection through impaired endolysosomal processing and TLR engagement
Cell. 2025, S0092-8674(25)00619-1.



Rapid, efficient and activation-neutral gene editing of polyclonal primary human resting CD4+ T cells allows complex functional analyses
Nat Methods. 2022, 19(1):81-89.

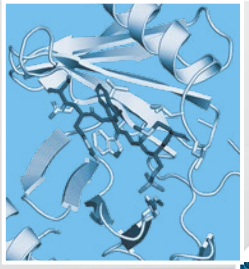


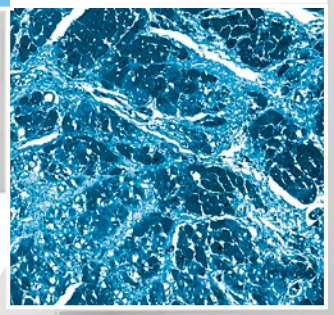
DELE1 tracks perturbed protein import and processing in human mitochondria.
Nat Commun. 2022, 13(1):1853.



LINE-1 ribonucleoprotein condensates bind DNA to enable nuclear entry during mitosis
Sci Adv. 2025, 11(18):eadt9318.

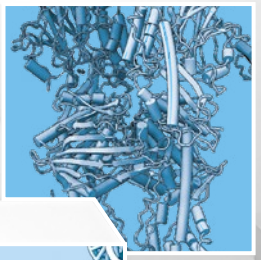
PLK1-Mediated Phosphorylation Cascade Activates Mis18 Complex to Ensure Centromere Inheritance.
Science. 2024, 285 (6713): 1098-1104.





Systemic deletion of DMD exon 51 rescues clinically severe Duchenne muscular dystrophy in a pig model lacking DMD exon 52
PNAS 2023, 120(29):e2301250120.

Cryo-EM structure of the Mre11-Rad50-Nbs1 complex reveals the molecular mechanism of scaffolding functions
Mol Cell. 2023, 83(2):167-185.e9.



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TACCO unifies annotation transfer and decomposition of cell identities for single-cell and spatial omics
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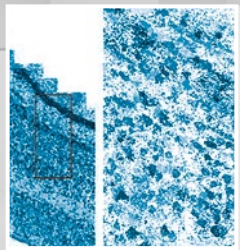
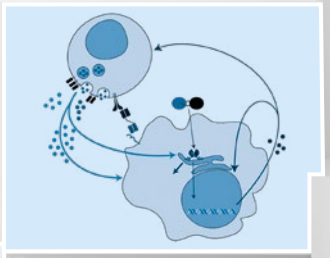
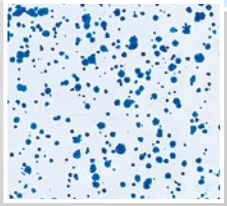


STING activation improves T-cell-engaging immunotherapy for acute myeloid leukemia.
Blood. 2025, 145(19):2149-2160.





RNF14-dependent atypical ubiquitylation promotes translation-coupled resolution of RNA-protein crosslinks
Mol Cell. 2023, 83(23):4290-4303.e9.