Cell News

Newsletter of the German Society for Cell Biology

full electronic version

01/2025

International Meeting of the German Society for Cell Biology

50 years of Cell Biology: the heart of life sciences – past, present and future

October 26 – 29, 2025 | Heidelberg, Germany

Topics

- Cilia and Centrosomes
- Membrane Trafficking and EVs
- Cytoskeleton and Molecular Motors
- Cell Polarity and Cell Migration
- Cell Adhesion and Mechanobiology
- Membrane Organization and Contact Sites
- Cell Biology of the Immune System
- Functional Organization of the Nucleus
- Imaging for Cell Biology
- Mitosis and Meiosis
- Cellular and Organismal Proteostasis
- Physics of the Cell

Speakers

Alba Diz-Munoz Germany
Ana Garcia Saez Germany
Andrea Pauli Austria
Anne Bertolotti UK
Benoit Ladoux Germany
Bernd Bukau Germany
Christel Verollet France
Elias Barriga Germany
Gaia Pigino Italy
Hesso Farhan Austria
Jay Gopalakrishnan Germany
Joao Matos Austria
Laura Aradilla Zapata Germany
Maria Leptin Germany
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Nicoletta Petridou Germany
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Robin Klemm UK
Sara Cuylen-Häring Germany
Stefan Diez Germany
Stefan Hell Germany
Tony Hyman Germany
Ulrich Hartl Germany
Volker Haucke Germany

Organizers
Gislene Pereira, Heidelberg
Elmar Schiebel, Heidelberg
Oliver Gruss, Bonn
Zuzana Storchova, Kaiserlautern
Sandra Iden, Homburg
Roland Wedlich-Söldner, Münster

Contact: Frederike Seibold
Christiane Edelmann-Mohr

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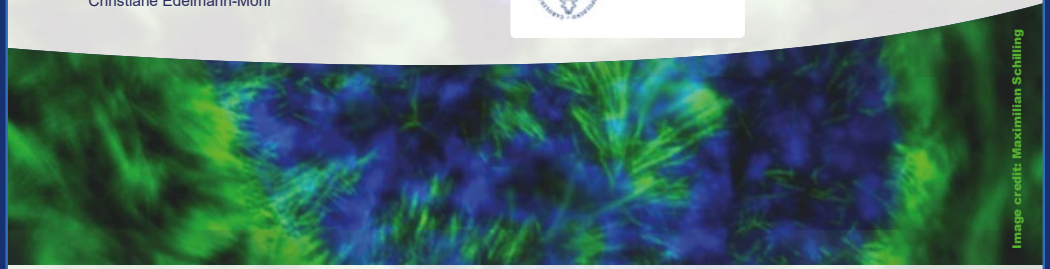
Early bird registration until May 31, 2025

Abstract submission until June 15, 2025



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Seeing beyond

CONTENT

DGZ Office

Sabine Reichel-Klingmann
c/o Deutsches Krebsforschungs-zentrum (DKFZ)
Im Neuenheimer Feld 280
69120 Heidelberg
Tel.: 0 62 21 / 42-34 51
Fax: 0 62 21 / 42-34 52
E-Mail: dgz@dkfz.de
Internet: www.zellbiologie.de

Cover Image: International Meeting of the German Society for Cell Biology: 50 years of Cell Biology: the Heart of Life Sciences – Past, Present and Future. October 26-29, 2025 in Heidelberg, venue: Conference Center, German Cancer Research Center (DKFZ). Information and registration: https://www.zellbiologie.de/dgz-international-meeting-2025/

Dear members of the DGZ,

2025 has shaped up to be a challenging year for scientists worldwide. With shocking speed, the Trump administration is threatening the U.S. science system with funding freezes and cuts, the layoff of federal employes from national agencies that serve public health, the termination of research programs that address climate change, vaccine development, and sex and gender differences, and the cancellation of policies and initiatives in diversity, equity and inclusion. The U.S. science ecosystem that drove scientific innovation and fostered intellectual freedom for decades is at risk of collapse. The consequences of this will be global and go far beyond the fate of individual scientists and research programs; they affect the progress of health for all. In Germany, scientific and academic freedom are fundamental rights and enshrined in Article 5 of the constitution. European countries have developed shared definitions of freedom of scientific research such as in the Bonn declaration 2020. Germany also benefits from a functioning scientific funding system that enables every one of us to participate. Many DGZ members are members of DFG review boards or other DFG committees or are involved in science policy. Although funding programs at the DFG have also been cut to an unprecedented extent (e.g. no new calls for priority programs in 2025), these measures do not force a bias in certain lines of research.

Nevertheless, the recent elections in Germany and other European countries have shown the continued rise of right-wing parties and the spread of "alternative truths". Not only in solidarity with our colleagues and friends in the U.S., but also to protect scientific freedom and progress worldwide, we must stand up for science and emphasize the value of scientific progress to society and public health. We encourage everyone to do this, even if it is only in small steps and in local circles. Discuss with your peers and lab members, families, politicians, and importantly engage in your scientific societies such as the DGZ or GBM (DGZ/GBM dual membership agreement). If you are a PI and have open positions that are suited for scientists that (planned to) work in the U.S., consider sharing the job posts at the DGZ webpage (e-mail to the DGZ office). At larger scale, European research institutions indeed are openly inviting U.S. based scientists that wish to leave today's US sciences. Related to the next federal government, we also refer to the DFG's 10 impulses for the new legislative term that address key areas for action and policy recommendations.

Perhaps in view of, rather than despite these difficult times, we are happy to celebrate that 2025 marks the **50th anniversary of the DGZ**, founded on April 26, 1975, in Heidelberg by German and Austrian cell biologists. The DGZ aims to be an inclusive cell biology society that provides a stage for both main and emerging cell biological topics, and a place for early career scientists and junior PIs to expand their networks and to connect to senior scientists. As we celebrate this important year, we also reflect on the progress the cell biology community in Germany has made over the past five decades. The DGZ and its members have fostered collaboration, innovation, and knowledge-sharing, paving the way for significant discoveries that have shaped the landscape of cell biological science in Germany, Europe and globally. In this spirit, we hope to see many of you in person at our **50-year anniversary meeting in Heidelberg** (October 26-29,2025) to celebrate the past, present and future of cell biology. The meeting includes keynote lectures by Tony Hyman, Stefan Hell, Maria Leptin and Bernd Bukau. There will be short-talks selected from abstracts, and networking opportunities throughout the event (page 5). Please also encourage your peers to join us and enrich their networks. Mark your calendar: early-bird registration ends May 31, regular registration September 15. For any questions related to the Heidelberg meeting please contact the local organizer Gislene Pereira.

We would also like to briefly remind our members how we can all support the cell biology community and in particular the next generation of cell biologists through various DGZ activities: Become a member of our DGZ working groups and join their online **DGZ Focus Workshops**, nominate emerging talent for the **DGZ Awards**, or apply on your own (pages 24-25). Please encourage colleagues and lab members who are not yet members to join the DGZ. We would like to thank everybody who submitted contributions to the recent **photo competition**, which revealed beautiful microscopy and cell biology predominantly from early career scientists! Further highlighting cell biology in our anniversary year, Leonhard Moeckl will guest-edit a **special issue on glycobiology** in the European Journal of Cell Biology; one focus will be on immunology, but also innovative methods for the detection of glycans, glycans in membrane organization, and else. Contact Leonhard (leonhard.moeckl@fau.de) in case you are interested to contribute.

As always, we welcome your feedback and suggestions on how we can continue to improve our cell biology society.

Best regards, and see you in Heidelberg,

The DGZ Board –
Julia Groß, Jörg Höhfeld, Maria Bohnert & Sandra Iden

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Ralf Jungmann Germany
Robin Klemm UK
Sara Cuylen-Häring Germany
Stefan Diez Germany
Stefan Hell Germany
Tony Hyman Germany
Ulrich Hartl Germany
Volker Haucke Germany

Organizers
Gislene Pereira, Heidelberg
Elmar Schiebel, Heidelberg
Oliver Gruss, Bonn
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Image credit: Maximilian Schilling

Walther Flemming Award 2024
Caveolae – specialized plasma membrane domains

Claudia Matthäus

Original Publications: Matthaeus et al., PLOS One 2019; Matthaeus et al., PNAS 2020, Matthaeus et al., Nature Comms 2022, Puchkov et al., Front Dev Cell 2023

Introduction

The uptake of nutrients in eukaryotic cells is highly regulated by various cellular processes. In my research I aim to understand how these uptake routes work, which molecules are involved and how cells regulate the uptake in response to their metabolic needs. Thereby, I primarily focus on a specific plasma membrane domain called caveolae. Caveolae are 50-80 nm sized membrane invaginations found in adipocytes, endothelial or muscle cells (Fig. 1)[1–3]. They are involved in lipid uptake and endocytosis, adapting membrane tension and regulating signaling processes at the plasma membrane[2,4]. Structurally, caveolae are formed by caveolin and cavin that localize to the plasma membrane and form the characteristic bulb-shape invagination5. In mammals, caveolin-1 and cavin-1 are essential to form caveolae, however the caveolin protein family also includes caveolin-2 and the muscle-specific caveolin-3[5]. The cavin protein family comprises in addition to cavin-1 cavin-2 and -3 (and the muscle-specific cavin-4) that form elongated oligomers with cavin-1 and thereby shaping the specific caveolar protein coat observed in metal replica electron microscopy[2,5] (EM) (Fig. 1).

Besides the caveolar coat forming proteins also membrane-bending proteins localize to caveolae domains at the plasma membrane. Here, the ATPase EHD2 specifically localizes to the neck region of caveolae and thereby stabilizing caveolae at the plasma membrane[6,7]. Interestingly, structural studies revealed that EHD2 forms ring-like oligomers inducing tube formation in liposomes[8]. It is therefore assumed that EHD2 also forms a ring-like oligomer at the caveolar neck that helps to stabilize caveolae at the plasma membrane[9,10]. Loss of EHD2 or its membrane-binding function destabilizes caveolae resulting in increased caveolae mobility and endocytosis[9,11–13]. The BAR-domain containing protein syndapin2 / pacsin2 also localizes to caveolae[14]. It was shown previously that pacsin2 is needed to form the caveolar neck. In the absence of pacsin2 caveolae appear less invaginated – flatter – at the plasma membrane[14]. Several actin binding proteins were also associated to caveolae like EHBP1 or filamin A[15].

Platinum replica electron microscopy

As caveolae are mainly found at the plasma membrane, the ideal analyzing technique combines highest resolution with the possibility to inspect large cellular membrane areas throughout many cells per sample. Transmission electron microscopy (TEM) or super-resolution fluorescence microscopy like stimulated emission depletion microscopy (STED) achieve the necessary resolution to detect and visualize single caveola including their outer boundary and neck[9,10,13]. However, TEM requires ultra-thin sectioning of samples resulting only in small plasma membrane areas that can be investigated. The preparation of metal replicas of large membrane areas overcomes this problem[16]. Thereby, cells seeded on a glass coverslip are 'unroofed': by using high shear force or ultrasonication all cellular components (e.g.: cytosol, nucleus, organelles) are removed and the adherent plasma membrane remains on the coverslip[16] (Fig. 2). After fixation and drying of the membrane sheet a 3 nm platinum coat is applied forming the replica[10,16]. Platinum replicas can be inspected by TEM and allow a detailed view of the plasma membrane (Fig. 1A).

Caveolae coat formation and curvature

When investigating the cytosolic side of the plasma membrane with high resolution platinum replica EM we are able to visualize, detect and analyze caveolae and their specific coat[10,16]. As illustrated in Fig. 1A caveolae differ in their size, coat assembly and curvature as well as actin association. Initially, caveolin-1 assembles into round-shaped 11-mer oligomers that form a disc with a flat lipophilic surface (membrane-facing side) and hydrophilic side (cytosol-facing) with a central beta-barrel that spans both sides[17]. When the caveolin-1 discs localize to the plasma membrane the lipophilic surface intercalates into the phospholipid bilayer and thereby moving several phospholipid molecules from the cytosolic membrane leaflet[17–19]. Based on size measurements from the cryo-EM structure of the caveolin1 discs it is assumed that 13-18 caveolin-1 oligomers are associated into a single caveola[17–19]. The characteristic caveolar coat is then formed by cavin oligomers (cavin-1/cavin-2 or cavin-1/cavin-3) that assemble in elongated stripes[18,20,21] (Fig.1B arrows).

In previous research we showed that caveolae can be classified in low-curved (flat-like), medium-curved (bulb-shaped) and highly curved (vesicle-like) membrane invaginations[10] (Fig. 1B). Electron tomography revealed that the accumulation of the caveolar coat increases with increasing curvature and therefore further stabilizing the membrane invagination[10]. The deeply curved caveolae form vesicle-like invaginations with a small neck size that most likely can undergo endocytosis (Fig. 1B, C). However, in contrast to clathrin-mediated endocytosis the molecular mechanism of the fission of caveolae from the plasma membrane is currently not understood. In our previous research we observed that caveolae can shift their curvature depending on prevalent membrane tension. With increasing membrane tension caveolae adapt to less curved invaginations without the complete disassembly of the caveolar coat[10]. This suggest that caveolae are flexible plasma membrane domains that adapt to local changes in membrane properties and during signaling processes.

Caveolae and lipid uptake

The absence of caveolin-1 or cavin-1 in mice results in resistance to diet-induced overweight and obesity, accompanied by a lipodystrophic phenotype[4]. These findings underscore the critical role of caveolae in cellular lipid uptake and metabolism. Recent investigations have further elucidated that caveolin-1 and caveolae are integral to the uptake of various lipid species[18,19]. How caveolae facilitate the lipid uptake and trafficking? Caveolae contain a distinct lipid composition, suggesting that extracellular lipids may preferentially accumulate within these membrane invaginations. Additionally, the fatty acid-binding receptor CD36, which localizes to caveolae, facilitates the translocation of fatty acids across the plasma membrane[9]. Following a currently unknown trigger, caveolae undergo endocytosis, leading to the detachment of caveolar vesicles from the plasma membrane and subsequent intracellular lipid transport. Several studies and our own research indicate that caveolin-1 accumulates at lipid droplets in response to fatty acid stimulation, such as exposure to oleic acid[7]. However, the mechanisms underlying the migration of caveolae from the plasma membrane to lipid droplets and the transfer of lipids from caveolar vesicles to lipid droplets remain poorly understood. Alternative pathways may also contribute to caveolae mediated lipid transport, including the classical endocytic pathway, where lipid trafficking may involve lysosomal degradation of caveolar vesicles and the subsequent accumulation and processing of fatty acids in the endoplasmic reticulum.

Caveolae-mediated lipid uptake can be modulated through alterations in either the number of caveolae present at the plasma membrane or the rate of their endocytosis[7,9]. When we investigated the effect of an increased caveolae uptake by deletion of EHD2 in vivo we observed an increased fatty acid accumulation leading to significantly enlarged lipid droplets[9]. Additionally, in mice fed a high fat diet, EHD2 expression was down regulated resulting in detachment of caveolae from the plasma membrane[9]. These finding suggests that cells can dynamically regulate caveolae-mediated lipid uptake in response to their metabolic needs or environmental conditions. Notably, adipocytes, which are specialized for the uptake, transport, and storage of substantial quantities of lipids, exhibit the highest density of caveolae at their plasma membranes[10].

Conclusion

Recent studies showed the involvement of caveolae in lipid uptake, metabolism, and intracellular trafficking and therefore underscores their importance in maintaining lipid homeostasis and cellular energy balance. The regulation of caveolae through factors such as caveolae number or endocytosis rate, and environmental stimuli like high-fat diets, highlights the adaptability of cells to metabolic demands. However, significant gaps remain in understanding the precise mechanisms by which caveolae migrate, transfer lipids, and interact with lipid droplets or other organelles. In addition, the accumulation of lipid species (which lipids?) in caveolae and how caveolae curvature and its coat adapts to changing lipid composition is currently not understood. Future research should focus on elucidating these pathways, offering insights for new therapeutic strategies targeting metabolic disorders such as obesity and lipodystrophy.

Acknowledgement

I would like to thank my postdoc supervisors Oliver Daumke (MDC) and Justin Taraska (NIH) that supported me throughout my postdoc projects. Their guidance and mentoring taught me highly appreciated skills and encouraged my own scientific independence. My great postdoc time set the foundation for my own research group. I also thank all my collaborators and co-authors that supported us in the past and present. In particular, I thank Martin Lehmann and Volker Haucke (FMP) for their support and critically discussion about science and life. I want to thank my research team at the University of Potsdam, their enthusiasm, curiosity and courage to join me in the fascinating world of caveolae. I am thankful to all funding agencies that support our research including Chan Zuckerberg Initiative, DFG, Else Kröner-Fresenius foundation, and DGE. Lastly, I thank DGZ and ibidi for honouring me the Walther-Flemming award 2024.

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Dr. Claudia Matthaeus

EDUCATION

11/2015	PhD in Biology, Free University Berlin, Germany (Date: 26 Nov 2015)
12/2011	Chemistry, Master of Science, Technical University Dresden, Germany
09/2008	Chemistry, Bachelor of Science, Technical University Dresden, Germany

CURRENT POSITION

From 03/2023	Assistant Professor (W1, Tenure-Track), Cellular Physiology, University Potsdam, Institute for Nutritional Science, Germany

POSITIONS

11/2019 – 10/2022	Postdoc Fellow with Dr. Justin Taraska, Molecular and Cellular Imaging Laboratory, National Heart, Lung and Blood Institute, NIH, US
07/2016 – 10/2019	Postdoc in Laboratory of Prof. Oliver Daumke, Structural Biology of Membrane-associated Processes, Max-Delbrück-Center of Molecular Medicine, Berlin, Germany
02/2012 – 06/2016	PhD researcher in Laboratory for Prof. Fritz Rathjen DevelopmentalNeurobiology, Max-Delbrück-Center of Molecular Medicine, Berlin

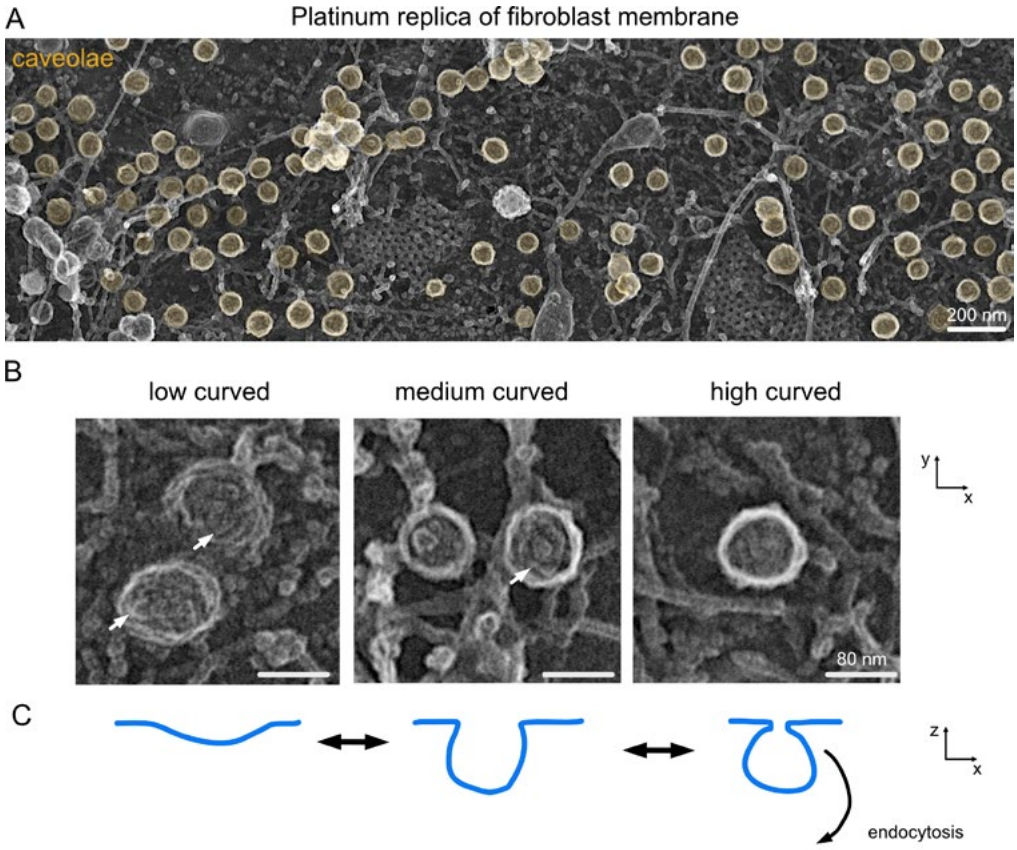

Figure 1: Caveolae at the plasma membrane. *(A) Representative EM image of a platinum replica of a fibroblast membrane sheet, caveolae are manually color-coded in yellow. Scale bar is 200 nm. (B) Example images of low, medium and high curved caveolae in PREM images, arrow indicates to cavin protein coat (stripes). Scale bar is 80 nm. (C) Scheme shows zx-view of low, medium and high curved caveolae in B.*

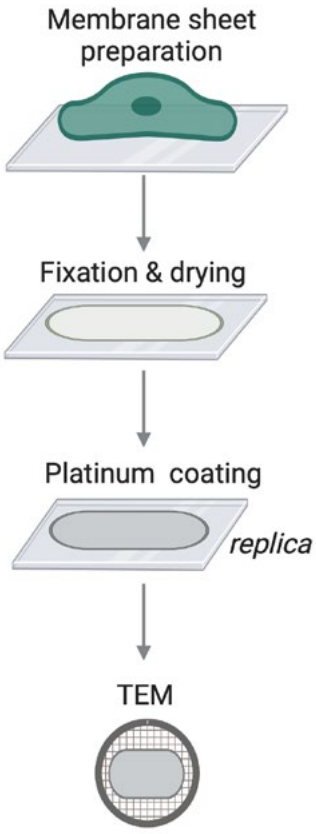

Figure 2: Platinum replica electron microscopy workflow. *Cells are seeded on glass coverslips, and membrane sheets are prepared by removing all cellular parts except the plasma membrane adherent to the glass. After fixation and drying a 3 nm sized platinum coat is added by a metal coater (e-beam). Afterwards the coverslip is removed, and the replica are placed on TEM grids for inspection. Scheme is created in BioRender. Matthaeus, C. (2025), https://BioRender.com/j59g727.*

Nikon Young Scientist Award 2024
Microtubule Nucleation by the γ-TuRC and partners: From structure to function

Martin Würtz

Original publications: Würtz et al., Open Biology 2021; Würtz et al., Nature commun 2022; Zupa et al., Nature commun 2022.

Introduction

Microtubules are essential components of the eukaryotic cytoskeleton, orchestrating processes like cell division, intracellular transport, and overall cellular organization. These dynamic polymers form hollow tubes with a diameter of 25 nm and are central to the remodeling of the cytoskeleton, enabling cells to respond to developmental cues, environmental signals, and mechanical stress. Microtubules are assembled de novo from α/β-tubulin dimers, with the γ-tubulin ring complex (γ-TuRC) serving as the primary nucleator that starts the assembly process [1]. Over 30 years of research into γ-tubulin complexes has established the "template model", suggesting that γ-TuRC acts as a scaffold with exposed γ-tubulins that bind α/β-tubulin dimers [2]. This interaction establishes the fundamental architecture of a microtubule, guiding the formation of its characteristic 13-protofilament cylindrical structure (Figure 1A). In cells γ-TuRC localizes to microtubule organizing centers (MTOCs), such as centrosomes, and nucleates microtubules at various locations throughout the cell cycle. During cell division, when thousands of microtubules are rapidly assembled, microtubule branching is a central pathway for fast and efficient spindle assembly to ensure chromosome segregation [3]. This process is mediated by the augmin complex, which recruits γ-TuRC to pre-existing microtubules and initiates new microtubule assembly with defined orientation (Figure 2A). The microtubule branching reaction during mitosis exemplifies a fundamental question in the field of microtubule research: how is microtubule nucleation regulated to occur at the right time and location?

Reconstituting the γ-TuRC

When I began my PhD at the end of 2019, the field of microtubule nucleation was entering an exciting era. At that time, the manuscript describing the cryo-electron microscopy (cryo-EM) structure of the *Xenopus laevis* γ-TuRC was under revision [4]. The vertebrate γ-TuRC is a complex assembly of over 30 proteins arranged in a left-handed spiral, with 14 γ-tubulin subunits bound to 14 γ-tubulin complex proteins (GCPs) and a centrally embedded actin molecule (Figure 1B) [5]. This work, alongside other cryo-EM structures of the human γ-TuRC [6,7], set the stage for the start of a systematic dissection of the complex and offered me a unique opportunity to contribute to understanding the assembly and function of this key molecular machine. While these advances addressed longstanding questions, they also raised new ones, such as how γ-TuRC assembles to this specific organization and the roles of individual components like the single actin molecule that is embedded at a specific position in the γ-TuRC.

To address these questions, I established a recombinant expression strategy in insect cells to reconstitute both human γ-TuRC subcomplexes and the entire complex (Figure 1B,C) [8]. Our recombinant γ-TuRC system provided a versatile platform for dissecting the roles of individual γ-TuRC components. Cryo-EM analysis allowed us to map the modular assembly pathway, showing how individual subcomplexes assemble into the full ring. Notably, this revealed that the γ-tubulin small complex (γ-TuSC), a subcomplex identified decades ago in yeast and composed of the γ-tubulin complex protein 2 (GCP2) and GCP3, each binding one γ-tubulin molecule via the conserved GRIP2 domain (Figure 1A,C), is successively connected to the starting core of GCP4-GCP5-GCP4-GCP6 subunits, with each component also binding one γ-tubulin molecule [9]. This recombinant system in combination with cryo-EM and other methods also enabled functional studies. For example, by mutating the actin-binding interface of GCP6, we generated γ-TuRC complexes that could not stably integrate actin (Figure 1D). Remarkably, these complexes still assembled into full 14-spoked rings and nucleated microtubules. However, cells lacking the N-terminus of GCP6 (ΔN-GCP6), and thus unable to integrate actin into γ-TuRC, exhibited an increased frequency of defects, such as prolonged mitosis. This demonstrates that while actin is not strictly required for γ-TuRC assembly, it influences its function (Figure 1E).

The recombinant reconstitution, guided by the structural understanding of γ-TuRC and complemented by similar recombinant systems developed in the field [10,11], has expanded our understanding of γ-TuRC and provided a foundation for further functional dissection of the complex. For me, the most interesting aspect, exemplified by our ΔN-GCP6 mutants, was the integration of methods—combining recombinant reconstitution, cryo-EM, and translating these findings into specific manipulations to study their impacts in cells.

Deciphering the augmin complex architecture

While γ-TuRC is the principal microtubule nucleator, it relies on recruitment factors for precise spatial and temporal activity. One such factor is the octameric augmin complex, first discovered in *Drosophila melanogaster* [12] as a key player in increasing microtubule spindle density. Augmin binds to microtubules and recruits γ-TuRC, facilitating microtubule branching (Figure 2A). Despite its critical role, the structural basis of augmins function has remained elusive since its discovery. Initial characterizations identified augmin as an eight-subunit complex forming an h-shaped rod, presenting significant challenges for structural studies [13,14].

Recognizing the importance of γ-TuRC interaction partners in understanding its function, I extended my focus to include the characterization of the augmin complex and employed once more the recombinant expression in insect cells to reconstitute *Xenopus laevis* augmin complexes. These efforts coincided with the release of AlphaFold2. Shortly after publication, we applied AlphaFold2 to investigate our "augmin complex problem", making it a compelling test case (Figure 2B,C). By integrating the protein structure predictions with biochemical experiments, EM, and crosslinking mass spectrometry, we determined the architecture of the augmin complex (Figure 2C,D) [15]. Our work, complemented by similar approaches with very similar findings [16,17] exemplifies how advancements in protein structure prediction are transforming structural biology and broader life sciences research, which is further underscored by the awarding of the 2024 Nobel Prize in Chemistry to Demis Hassabis, John Jumper, and David Baker.

The structural characterization of augmin provided important insights, including the identification of a microtubule-binding unit essential for its role in recruiting γ-TuRC to microtubules to initiate the microtubule branching reaction. We found that the binding region consists of previously unidentified calponin homology (CH) domains in the N-termini of HAUS6 and HAUS7, which, together with the unstructured N-terminus of HAUS8 (formerly HICE1), form a composite binding domain (Figure 2C-F). Interestingly, this domain is compositionally very similar to that of the Ndc80 complex, which binds microtubules at the kinetochore [18]. This discovery now paves the way for a systematic dissection of augmin complexes across different species.

For me, the augmin complex exemplifies both the elegance and complexity of molecular systems, representing a conserved interplay of eight distinct proteins that assemble into a single, interconnected functional unit with distinct roles distributed along its elongated structure. Consistently, all HAUS proteins are encoded by essential genes [19], underscoring augmin's critical role especially in cell division. With the advanced tools now available, we have an unprecedented opportunity to study the functional properties of this important complex and explore its structural and functional conservation across evolution. By reconstituting minimal functional units (Figure 2G), for instance, we can conduct more targeted studies of augmin's role in cellular processes and unravel the underlying principles that govern its conserved functionality.

Conclusion and Outlook

The term "From structure to function", which I used in my title, is commonly used in structural biology, yet it reflects an important strategy in life science research. Recent advances in methodologies such as cryo-EM, recombinant protein expression, and AI-driven structure prediction now facilitate the study of complex molecular systems and their mechanisms in molecular detail. For example, the description of the γ-TuRC cryo-EM structure has initiated a series of groundbreaking studies on microtubule nucleation. In the future, combining detailed structural insights with functional studies of individual components, as well as their integration into the cellular context, will be pivotal for unraveling the intricate functional relationships underlying microtubule nucleation—a process fundamental to cellular architecture and tightly linked to the regulation of the cell cycle.

Acknowledgments

Firstly, I would like to thank Elmar Schiebel and Stefan Pfeffer for their guidance and for fostering an open and supportive work environment. I am also grateful to all lab members, facility staff, and students who collaborated on various projects. Many thanks to collaborators and partners over the years, to Sebastian Eustermann for his support and valuable discussions, and to close co-workers such as Erik Zupa and Bram Vermeulen. I would also like to acknowledge previous collaborators and mentors, including Katalin Toth and Jörg Langowski. Lastly, I want to thank Nikon and DGZ for their support, the award, and their efforts in fostering a collaborative and dynamic scientific community.

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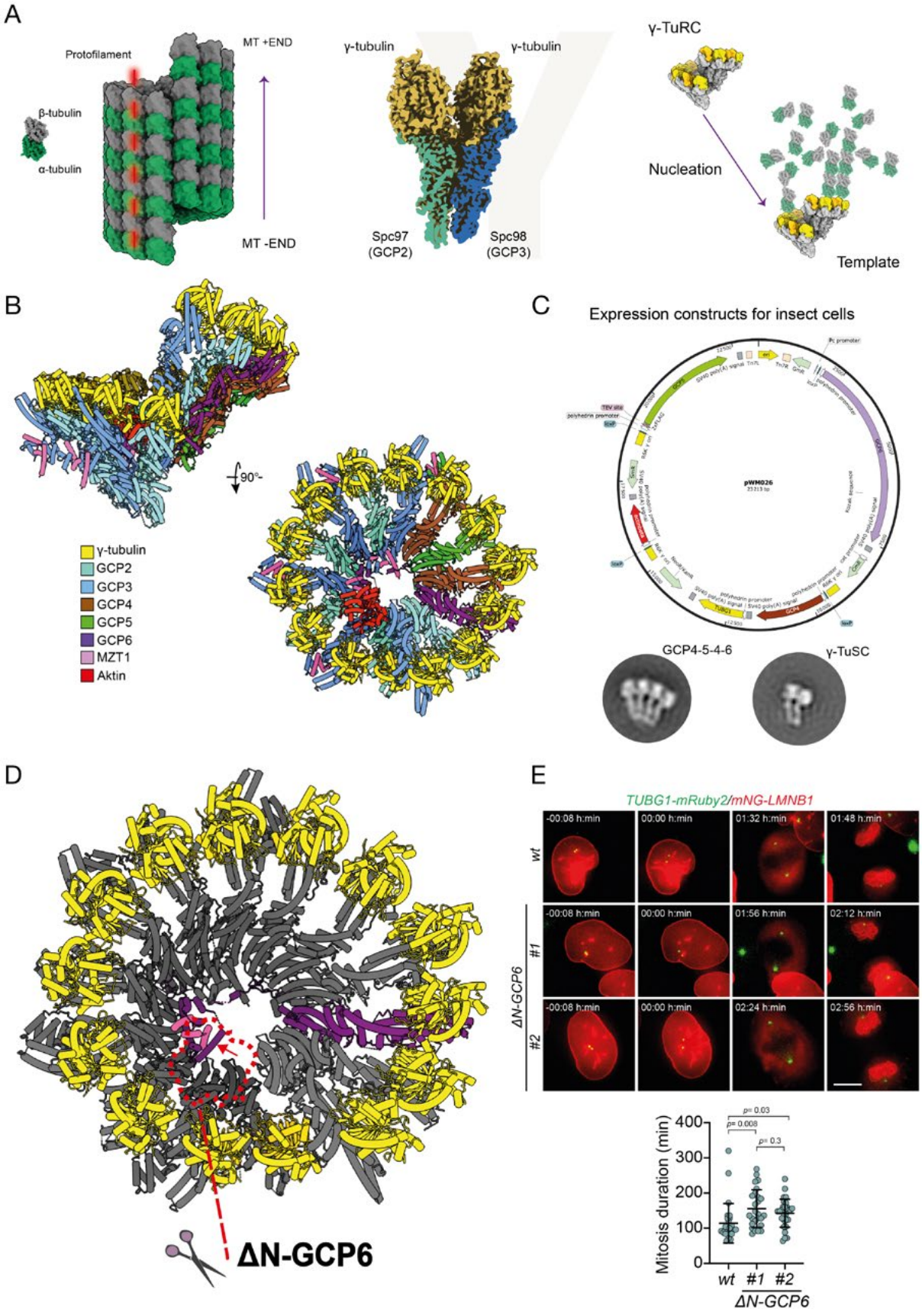
Martin Würtz earned his bachelor's and master's degrees in Molecular Biotechnology from the University of Heidelberg, where he focused on the biophysics of nucleosomes in the laboratory of Jörg Langowski at the DKFZ in Heidelberg. For his PhD, he joined the research group of Elmar Schiebel at the ZMBH, working closely with Stefan Pfeffer's group. His PhD research bridged cell and structural biology, with a focus on microtubule nucleation. Currently, he is a postdoctoral researcher in the group of Sebastian Eustermann at EMBL Heidelberg, supported by an Interinstitutional Postdoctoral Fellowship from the Heidelberg Mannheim Life Science Alliance.

Contact:

Mail: martin.wuertz@embl.de
m.wuertz@zmbh.uni-heidelberg.de
ORCID iD: 0000-0002-4504-4723

Figure 1 *γ-TuRC, a multi-subunit complex as the principal template for microtubule nucleation:* **A)** *Left: Microtubules are dynamic polymers built by α/β-tubulin dimers. They assemble into hollow cylinders where the α-tubulin side is the so-called "–END", and the β-tubulin side is the "+END". In cells, microtubules consist of 13 protofilaments that are longitudinal stretches of α/β-tubulin dimers. Middle: The basic unit of γ-tubulin complexes that regulate microtubule formation is the γ-tubulin small complex, which forms a Y-shaped structure (Figure adapted from [20]). Right: The γ-tubulin small complex like subunits assemble into a spiral that serves as a template for the microtubule formation (nucleation) and binds the microtubule –END.* **B)** *Organization of the recombinant human γ-TuRC. Proteins are indicated (PDB:7qjc).* **C)** *Top: Exemplary MultiBac construct for the expression of human γ-TuRC in insect cells, containing the coding sequences for GCP5, GCP4, GCP6, γ-tubulin, and β-actin. Bottom: Negative stain 2D class averages of two different recombinantly reconstituted γ-TuRC sub-complexes. Left: GCP4-5-4-6 with γ-tubulin. Right: γ-tubulin small complex (see A).* **D)** *Visualization of γ-TuRC highlighting GCP6 (purple), which spans through the complex. The N-terminus of GCP6, highlighted with an asterisk, binds actin and is deleted in ΔN-GCP6 constructs. The actin position is indicated with a red dashed line.* **E)** *Representative section and quantification of Live-cell imaging of ΔN-GCP6 cell lines that cannot integrate actin into γ-TuRC. ΔN-GCP6 cells can nucleate microtubules but show mitotic phenotypes, such as elongated duration of mitosis. Scale bar 10 µm (adapted from [9]).*

Figure 1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GCP2
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GCP4
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GCP6
MZT1
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Mitosis duration (min)
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Figure 2

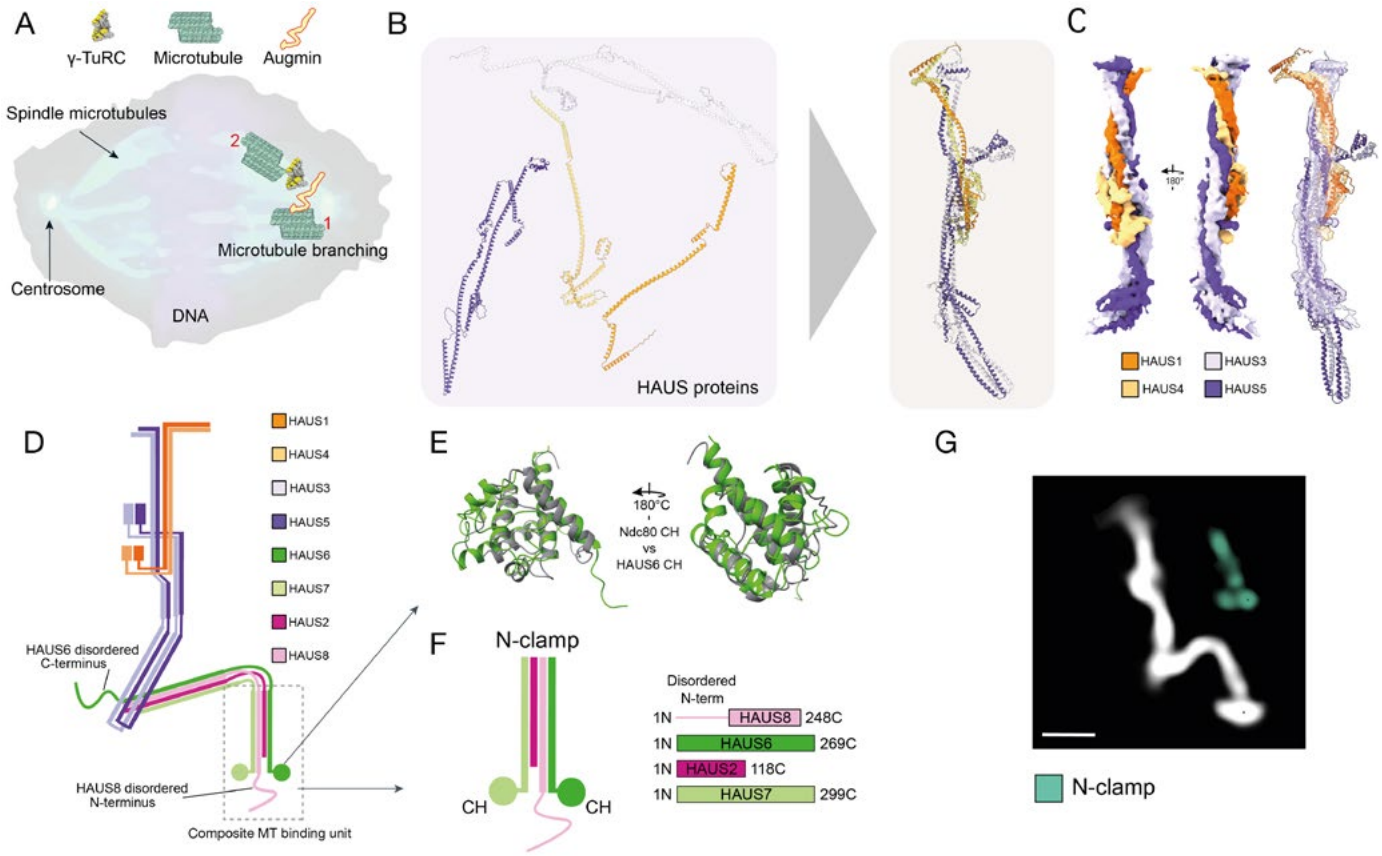

Figure 2 *Architecture of the augmin complex:* **A)** *Schematic representation of a mitotic spindle. The augmin complex binds microtubules and recruits γ-TuRC for microtubule branching, a pathway for the formation of a new microtubule (2) based on an existing one (1).* **B)** *Left: AlphaFold2 predictions of individual HAUS proteins. Right: Prediction of four HAUS proteins together showing a coiled-coil rod-like complex as a result.* **C)** *AlphaFold2-predicted structures fitted into the cryo-EM density of the corresponding augmin subcomplex, showing a good fit between the predicted structure and the density (adapted from [15]).* **D)** *Organization map of the augmin complex, highlighting structural units. Coloring as indicated.* **E)** *Overlay of the CH domain of HAUS6 (green) and the CH domain of Ndc80 (grey, PDB-3IZ0), showing a similar fold (adapted from [15]).* **F)** *The composite microtubule-binding unit of the augmin complex consists of the N-termini of HAUS2, HAUS6, HAUS7, and HAUS8.* **G)** *Negative stain EM 2D class averages of the augmin octamer, highlighting a separately reconstructed part of the minimal microtubule-binding unit, the augmin N-clamp (magenta). This demonstrates that with the structural information, specific functional units can be constructed to characterize augmin's functions. Scale bar 10 nm.*

BINDER Innovation Prize
Cells have feelings too

Alba Diz-Muñoz

Summary/ Introduction

Functional compartmentalization is a defining feature of life. The interior of cells is divided into numerous specialized compartments, and, on a larger scale, the cell as a whole is separated from the external environment by means of a thin surface. Membranes surrounding cells and organelles (in eukaryotic cells) facilitate the formation and maintenance of chemically distinct environments by providing an active diffusion barrier, selectively allowing for exchange of nutrients and expulsion of waste. However, the role of surfaces goes beyond this chemical control function: they must also maintain a mechanical and topological integrity. Membrane-bound compartments are exposed frequently to forces- they are pulled, compressed and deformed. In the case of the cell, active deformations that occur during processes such as migration, differentiation, growth, and division add to this complex challenge. We study the surface of the cell, the boundary surrounding the fundamental compartment of life, with an interdisciplinary perspective informed by physics and engineering.

The cell surface is an immensely complex system **(Figure 1)**. It comprises a fluid lipid bilayer, the cell membrane, coupled to an underlying polymer meshwork, the actomyosin cortex. The specialized proteins connecting these two layers are called membrane-to-cortex attachment (MCA) proteins. Borrowing the terminology from materials science, such a system is a 'composite interface': two materials with distinct physical properties are coupled, giving rise to emergent properties which could not be predicted from the individual characteristics of each material alone. Composite materials are frequently exploited in both natural and artificial constructions, from the reinforced concrete in skyscrapers to the mother-of-pearl in mollusc shells. In contrast to composites in static systems, however, the cell surface is additionally an active material. That is, intrinsic forces generated by the conversion of chemical to mechanical energy hold the system in a delicate out-of-(thermodynamic)-equilibrium state. The entire system is also highly dynamic: the membrane is constantly turned over by the addition or removal of lipids; actin is polymerized, depolymerized, crosslinked, and contracted; MCA proteins are activated, deactivated, and trafficked.

The interplay between the different components of this interface is still poorly understood. The dynamism, fast timescales, and small spatial scales of activity at the cell surface have made it difficult to isolate individual properties or to examine individual components. However, tools and concepts from physics and bioengineering are now enabling us to glimpse into the functioning of this remarkable system. In my lab we are developing and applying tools to dissect the complex biophysics at the cell surface. Our work is revealing that the coupling of polymer physics to a fluid boundary gives rise to unexpected emergent phenomena and encodes regulatory dials and switches which we believe are critical to understanding cellular function.

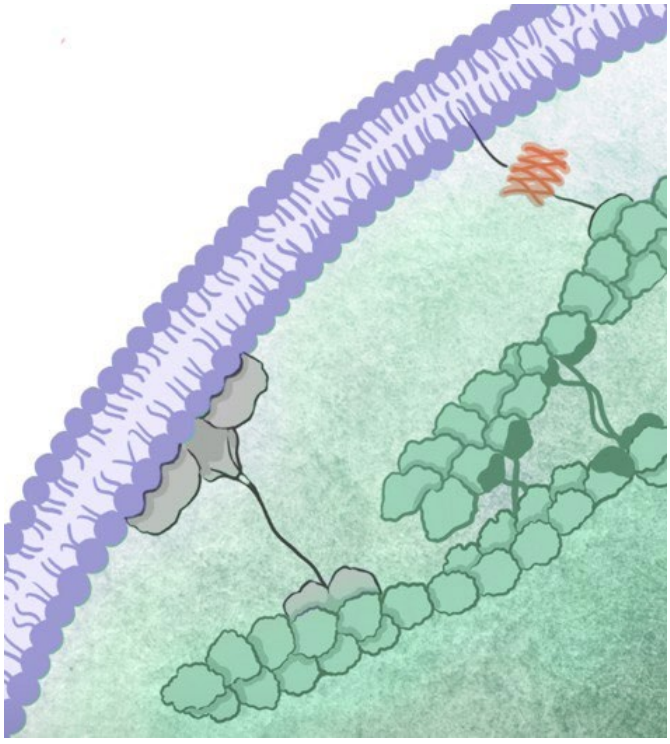

Figure 1: *The cell surface composite*

In this simplified view, the membrane (purple) is connected to cortical actin filaments (green) by specialized linker proteins (grey). Credit: Dr. Anusha Gopalan

Here I will focus on one piece of work from my lab that showcases our interdisciplinary approach and provides one example of the exciting insights which can arise from a mechanistic understanding of the cell surface. In Sitarska et al., we looked at how immune-like cells migrate and use the topography of their membrane as a sensor to help them navigate complex environments[1]. We identified a mechanism by which the curvature-sensing BAR domain protein Snx33 inhibits actin polym-

erization and therefore leading-edge expansion at regions of low inward plasma membrane curvature. Our results show how cells can read out their surface topography and utilize actin and plasma membrane biophysics to interpret their environment, allowing them to adaptively decide if they should move ahead or turn away.

The world of a migrating cell

Cells of the immune system provide an impressive demonstration of morphological plasticity and adaptability. Immune cells patrol throughout the body as they seek out and destroy infected cells and pathogens. This requires motion through highly heterogeneous 3D environments- cells must respond to a wide variety of different signals, including chemotactic cues indicating the location of pathogens or other immune cells, and mechanical barriers such as fluctuations in tissue density[2,3]. The cell surface is the first point of contact for the recognition of and response to these diverse inputs.

Propulsion in immune cells depends on actin polymerization at the leading edge of the cell[4,5]. Thus, signals which affect navigation generally converge on cytoskeletal regulators to promote or inhibit actin dynamics. The cell faces the challenge of integrating and reconciling this cacophony of signals- including chemotactic cues, biomechanical inputs, or ligands on the surface of surrounding cells- all of which influence the cytoskeletal network. While chemotaxis has been more extensively studied, much less is understood about how cells navigate their physical surroundings. In particular, how do cells understand and respond to the information encoded in the topography of the environment, and how is this information integrated by cells to aid in decision-making processes during migration?

We chose to address this question using the immune-like HL-60 cells. Fully differentiated HL- 60 cells have a neutrophil-like morphology and behavior: they are highly motile and display an actin- rich lamellipodia as well as extensive membrane ruffles at the leading edge. Using a range of techniques to visualize and model the behaviour of these cells at multiple scales of space and time, we describe a mechanism by which detection of topographical changes by the cell surface is translated into changes in actin dynamics to guide cell steering.

Sensing the cell surface: Snx33

As a cell moves through a complex environment, its surface is deformed repeatedly, locally changing membrane curvature. We reasoned that curvature-sensing proteins might thus play an important role in sensing environmental topography.

The BAR (Bin, Amphiphysin, and Rvs) domain-containing proteins are a family of proteins known to bind the membrane in a curvature-dependent manner: the BAR domain is shaped like a banana, and thus has a structural affinity for membrane structures that match its shape. BAR domain- containing proteins are known to have diverse functions in the regulation and modulation of the actin cytoskeleton[6]. We therefore transcriptionally profiled these proteins before and after differentiation of HL-60 cells, a process in which these cells transform from a rounded to a complex, neutrophil-like morphology. Strikingly, we found that the expression of the BAR-domain-containing protein Snx33 was 16-fold greater in fully differentiated HL-60 cells than in the precursor cells.

To further assess the localization and dynamics of Snx33, we began by performing a course- grained molecular dynamics (MD) simulation[7], which indicated that Snx33 should bind stably to regions of local inward curvature. This is expected from the structure of Snx33, as the membrane- binding domain of Snx-33 is on the concave surface of the protein. To place this in the context of our system, we then studied the cell surface more closely using three complementary microscopy techniques. We employed scanning electron microscopy (SEM) to structurally characterize the cell membrane in high resolution in a fixed cell; polarized total internal reflection fluorescence microscopy (p-TIRFM) to observe membrane-curvature dynamics in live cells; and finally lattice light- sheet microscopy to image the subcellular localization of Snx33. We found that the apical surface of migrating HL-60 has many regions of high curvature (SEM), that this surface is immensely dynamic (p-TIRFM), and, consistent with the MD simulations, that Snx33 seemed to be excluded from areas of high outward membrane curvature (lattice light-sheet microscopy).

To test the functional roll of Snx33 in cell motility, we generated a knockout cell line (Snx33-/-) and used total internal reflection microscopy (TIRF) to capture movies of wild-type and Snx33-/- cells. Knock-out and wild-type cells differed across a broad spectrum of migration-relevant parameters: the leading edge was bigger in the knock-outs, and the cells were more spread and elongated. We also used an atomic force microscope to measure the force required to pull tethers from the plasma membrane, giving us a metric of membrane tension[8]. Snx33 knockdown led to a strong increase in apparent membrane tension, an expected result of the morphological changes described above[9,10]. This constellation of characteristics all point to the maintenance of a more stable leading edge.

These morphological changes strongly suggested that Snx33 was affecting actin polymerization dynamics. As a first test of this hypothesis, we stimulated HL-60 cells with a chemoattractant and measured the change in actin content. We observed that Snx33-/- cells showed a greater increase in actin content than wild-type controls, consistent with an inhibitory role of Snx33 on actin polymerization. To hone in on how Snx33 interacts with the machinery regulating actin dynamics we co-immunoprecipitated Snx33 with binding partners and characterized the results using mass spectrometry. We found a direct interaction of Snx33 with the Actin Related Complex 2/3 (Arp2/3) and various actin Nucleation

Promoting Factors (NPFs). Arp2/3 activity generates branched actin networks and is responsible for the formation of lamellipodia and membrane ruffles[11]. NPFs help in this process by potentiating Arp2/3 activity[12]. We then focused on the NPF WAVE2, the upstream activator of Arp2/3 in neutrophils. Propagating waves of WAVE2 have been shown to be critical for immune cell motility[4]. We observed a strong effect of Snx33 on WAVE2 patterns: WAVE2 patches were bigger in the Snx33 knockout cells, though expression levels were unchanged. Altogether, these results suggest that cells lacking Snx33 are primed for persistent rather than exploratory, migration; and that Snx33 affects actin polymerization dynamics via changes in WAVE2 activity.

The cell surface is key to navigation in complex environments

To directly assess how these changes in the dynamics of the actin control machinery influence cell motility, we challenged Snx33-/- and wild-type cells to choose the correct path in microfluidic devices. Here, the cells migrated through channels and encountered obstacles in the form of differently-sized pores. Snx33-/- cells required almost 20% more time to navigate these decision points and to find the path of least resistance than wild-type cells. On the other hand, in decision- free channels, Snx33-/- cells migrated 80% faster than their wild-type counterparts. The loss of Snx33 therefore renders cells less prone to spontaneous turning and less effective in circumnavigating an object while displaying more persistent migration in decision-free environments.

We next tested the potential role of Snx33 in contact- inhibited locomotion: we seeded a high density of cells to make collisions inevitable, and then imaged how two cells interacted. We again turned to electron microscopy to enable us to image the changes in curvature at this interface with precision. We observed that upon collisions, the distribution of surface curvature changed from predominantly outward curvature, to a flatter curvature distribution. We found that fluorescently- tagged Snx33 localizes to the contact site upon collisions, and that this is accompanied by a reduction in WAVE2 at the contact site **(Figure 2A-B)**. Thus, Snx33 activity at contact sites redistributes WAVE2 activity away from the collision site, and thus reorients cell movement **(Figure 2C)**.

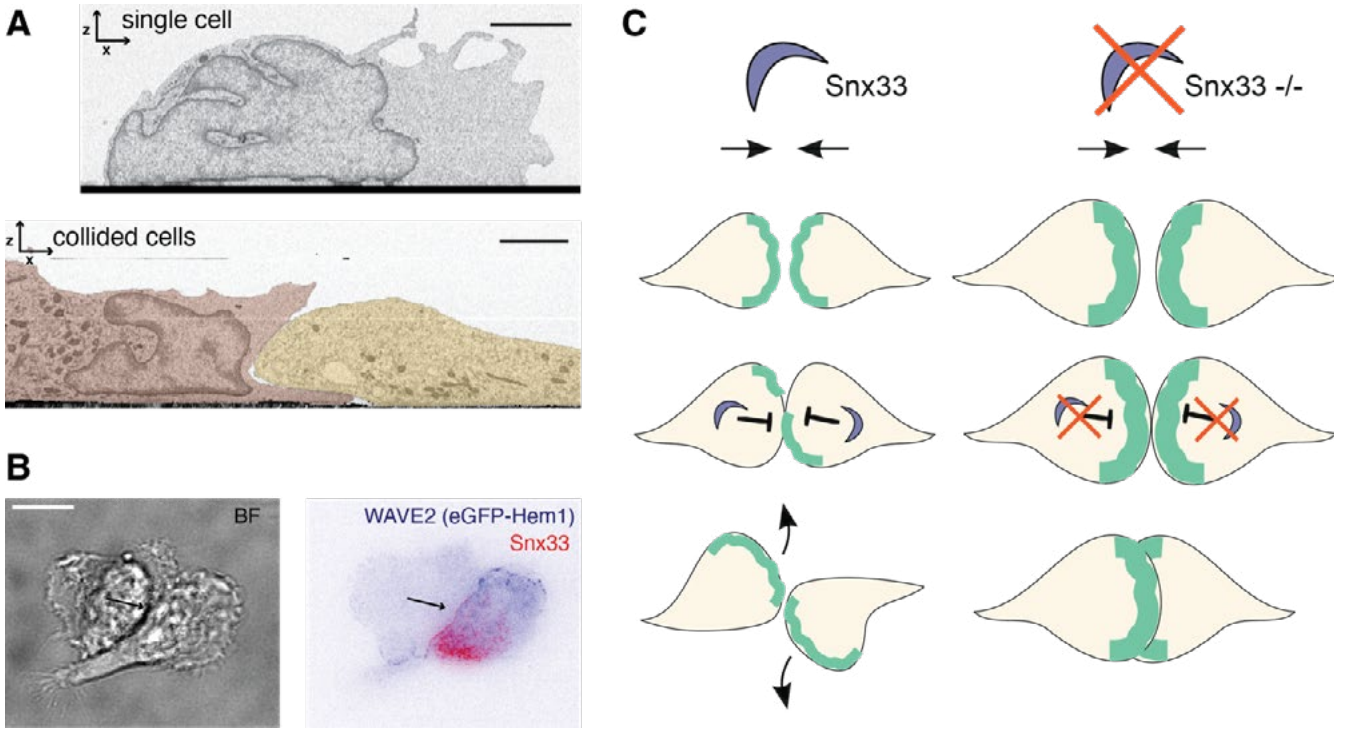

Figure 2: *Snx33 directs actin polymerization in response to surface deformations*

(A) Cross-section of a single (top) and collided (bottom) migrating dHL-60 cell by serial block face scanning electron microscopy. B) Bright-field (left) and polarized total internal reflection microscopy (right) images of cell–cell contact in wild-type dHL-60 cells. Snx33 and the WAVE2- complex component Hem1 are fluorescently labelled in red and purple, respectively. C) Graphical abstract: In wild-type cells (left) Snx33 inhibits actin polymerization at the leading edge upon contacts with other cells, inducing cells to turn away from the contact point. In Snx33 knockout cells (right), this mechanism does not occur, cells do not navigate obstacles as effectively.

The cell surface as a decision-making hub for motile cells

The cell surface is decorated by a diversity of receptor complexes. Particularly in the case of motile cells, these receptors receive, process, filter, combine, and integrate a continuous flow of stimuli which together inform cellular 'decisions'. The cross-talk between different chemotactic cues and signals received from surface-bound receptors has been well studied. Here, we add another layer of complexity, showing that membrane curvature is also integrated at the cell surface to inform cytoskeletal behavior and ultimately cell motility. Altogether, our results contribute to a picture of cell migration in which all external signals, both chemical and mechanical, are processed through the filter of the cell surface to determine their impact on the subjacent machinery.

Outlook

In Sitarska et al, we showed how cells use their surface to read out environmental topography and avoid obstacles during navigation. The mechanism we propose relies on an intimate connection between the cell membrane and the actin polymerization machinery, mediated via the curvature- sensing BAR-domain protein Snx33. Object evasion is key not only for the migration of immune and cancer cells in complex tissue environments, but is also fundamental during embryogenesis and collective migration; thus, we expect that this mechanism may be employed across many biological functions which require shape change.

Apart from this specific mechanism, my lab's research is building up a picture wherein the elegant biophysics at the cell surface, and particularly this connection between membrane and cytoskeleton, encodes fundamental regulatory mechanisms which underlie basic cellular functions. We have shown, for example, that releasing the attachment of the membrane to the underlying actin cortex is required to allow the exit from naïve pluripotency[13]. And we have revealed a novel mechanism by which membrane-to-cortex distance acts as a 'nanogate' to control the activity of another critical part of the actin machinery, the formin mDia[14]. Most recently, we have shown that symmetry breaking can be initiated at the cell surface via a mechanism we call 'linker caging' in which membrane-to-cortex attachment proteins become entangled and immobilized in the underlying actin meshwork[15].

These distinct mechanisms are all physically localized in a minute space between the membrane and the cortex and are thus likely all regulated by an overlapping multiparametric space which includes the biophysical properties of the membrane, cortex, and linker proteins. Moreover, while individual proteins and other components differ, the basic structure of the cell surface, comprising a lipid membrane linked to a polymer network, is highly conserved across the tree of life. I believe that the universality and persistence of this structure is due to highly tunable biophysical mechanisms, which, due to their reliance on geometrical constraints rather than specific biochemical interactions are robust to genetic instability.

Cells across the tree of life exhibit a dazzling variety of shapes as well as an enormous morphological plasticity, adapting their shape as they move, grow, and divide. Observing basic physical principles and mechanisms which underlie the existence of this zoo of forms and functions, provides us with a glimpse past the chaos and into the elegance, borrowing the meaning from physics, of biology. We are excited to see what other molecular switches are encoded in the biophysics of the cell surface composite.

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I thank all the past and current members of the Diz-Muñoz lab for their hard work, dedication, and for creating and amazing and supportive community. Specially, Dr Martin Bergert for joining me in this journey. For the project described here, I particularly thank Dr. Ewa Sitarska, the first author. This work is a reflection of her perseverance, bravery and curiosity, as well as eagerness to learn new techniques. I also want to thank Dr. Sarah Foster and Dr. Eva Pillai, ever-inspiring forces of writing- inspiration and invaluable contributors to this piece. I also am grateful to members of the EMBL core facilities for support and advice with imaging and data analysis, and our wonderful collaborators both within and outside of EMBL for their hard work and numerous scientific discussions.

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Finally, I thank my mentors for their unwavering support and nomination to this award, and the Binder Committee and the DGZ for recognizing my lab's work.

Please note that some of the text was taken directly or slightly modified from Sitarska et al (2023).

About the Author

Alba Diz-Muñoz received her PhD at the Max Planck Institute for Cell Biology and Genetics in Dresden, Germany. She then worked for her postdoctoral research in the US between UC Berkeley and UCSF.

Since 2016, Alba leads a laboratory at the EMBL in Heidelberg focusing on how composite interfaces govern cell morphogenesis, immune migration, and stem cell fate.

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Alba Diz-Muñoz

Group Leader and Deputy Head of Cell Biology and Biophysics

European Molecular Biology Laboratory

Meyerhofstraße 1 69117, Heidelberg, Germany
alba.dizmunoz@embl.de

EDUCATION

MPI-CBG and Technical University of Dresden, Dresden, DE	2008 - 2011
PhD, Biophysics	
Universitat Pompeu Fabra, Barcelona, ES	2002 - 2007
BA/MS, Human Biology	
Universitat Autonoma de Barcelona, Barcelona, ES	2002 - 2007
Medical school (years 1-3)	

RESEARCH

EMBL, Heidelberg, DE	2016 - present
Group leader	
University of California San Francisco and UC Berkeley, San Francisco, USA	2012 - 2016
Damon Runyon Postdoctoral Fellow	
MPI-CBG, Dresden, DE	2011 - 2012
Bridging Postdoctoral Fellow	

SELECTED AWARDS

Binder Innovation Prize	2024
Allen Distinguished Investigator Award	2024
ERC Consolidator Grant	2024
CZI Ben Barres Acceleration Award	2023
BPS Early Career Mechanobiology Award	2023

WALTHER FLEMMING AWARD 2025

The German Society for Cell Biology (DGZ) and ibidi GmbH offer the "Walther Flemming Award" for excellent research in cell biology. The award consists of a financial contribution of EUR 3000 and is given to senior postdoctoral researchers and early career group leaders for recent work that defines their emerging independent research profile.

Candidates need to be members of the DGZ and can either be nominated or apply directly for the prize.

Applications should be submitted **in a single pdf file (max. 10 MB)** and consist of cover letter, CV and copies of 1-3 publications that document the relevant work of the applicant. Applications will be reviewed by a dedicated award committee of the DGZ.

Please send your application by e-mail to the DGZ office:
dgz@dkfz.de

Deadline: **July 31, 2025**

NIKON YOUNG SCIENTIST AWARD 2025

The German Society for Cell Biology (DGZ) and Nikon Deutschland (Business Unit: Healthcare) annually offer the "Nikon Young Scientist Award" for excellent research in cell biology by PhD students or young postdoctoral researchers within 3 years after graduating (an extension of up to 2 years will be granted for periods of parental leave). The awardee will receive a financial contribution of EUR 1500.

Candidates need to be members of the DGZ and can either be nominated or apply directly for the prize.

Applications should be submitted **in a single pdf file (max. 10 MB)** and consist of cover letter, CV and copies of publications that document the work of the applicant. Applications will be reviewed by a dedicated award committee of the DGZ.

Please send your application by e-mail to the DGZ office:
dgz@dkfz.de

Deadline: **July 31, 2025**

DGZ MASTER THESIS AWARDS 2025

Call for Nominations: DGZ Master Thesis Awards

Since last year the DGZ offers the DGZ Master Thesis Awards, an exciting opportunity to support and recognize excellent work by emerging cell biologists in Germany.

Overview
Awards: We will present two awards each year, with DGZ Workgroup speakers playing a key role in selecting the awardees.
Eligibility: PIs are invited to nominate Master theses from their current or former students. The thesis must have been completed within the past 12 months, and the nominee must be a DGZ member.
Workgroup Alignment: The application should specify which DGZ Workgroup aligns with the nominee's work.
Awards Details: Each awardee will receive EUR 500 and the opportunity to present their work in a 10-minute slot during the DGZ Award Session in autumn.

This initiative is designed to acknowledge early academic achievement in cell biology and provide a platform for the awardees to showcase their research.

We encourage you to participate in this initiative and look forward to your nominations from your lab. Please send your applications (cover letter, student CV and thesis) **as a single pdf file (max. 10 MB)** by e-mail to the DGZ office: dgz@dkfz.de

Your input will be invaluable in promoting the next generation of cell biologists in Germany.

Deadline: **July 31, 2025**

INNOVATION PRIZE 2025

The Innovation Award 2025 will be given for outstanding contributions to cell biology and is aimed at junior investigators that have already established and developed their own research profile. The awardee will receive a financial contribution of EUR 3.500.

Candidates need to be members of the DGZ and can either be nominated or apply directly for the prize.

Applications should be submitted in a single PDF file (max. 10 MB) and consist of cover letter, CV, a research profile and copies of three selected first/last author publications. Applications will be reviewed by a dedicated award committee of the DGZ.

Please send your application by e-mail to the DGZ office: dgz@dkfz.de

Deadline: **July 31, 2025**

The 15th Physics of Cancer Symposium

The conference was held September 30th – October 2nd 2024 at the Center for Biotechnology and Biomedicine (BBZ), Leipzig

Metastasis is the process by which cancer cells spread from the primary tumor site to the entire body and affect the whole human as a systemic disease. It is responsible for 90 % of cancer-related deaths. Understanding metastasis, interactions between tumor cells and their microenvironment, and the mechanisms behind successful metastasis are crucial for developing effective treatments for patients with metastatic disease. The metastatic cascade is a complex and dynamic process influenced by various factors, including interactions with the microenvironment, genetic changes, and immune responses. In order to understand cancer progression, metastatic potential of different tumor types and the impact of physical properties such mechanics and dynamics of cells and tissues on various time and length scales, the 15th annual symposium "Physics of Cancer" aimed to address these issues. By bringing together world's leading scientists from medicine, biology, physics and engineering, the three-days symposium was not only focusing on latest research and developments in this field, but was also highlighting the work of the newly established DFG research group "Multiscale MRE: in vivo physics of cancer" (FOR5628). In fact, the first of five different sessions was conducted by members of the FOR5628 in order to present latest research results and to present the newly established DFG research group to a broad international audience. The other four sessions included the topics: (Session I) Cell and tissue mechanics, (Session II) Intermediate filaments, (Session III) Innovative ex-vivo cancer models, and (Session IV) Mechanical immune escape mechanisms.

The conference was organized by Josef Käs (Leipzig University), Christoph Mark (FAU Erlangen), Ben Fabry (FAU Erlangen), Anna Taubenberger (TU Dresden) Mareike Zink (Leipzig University) and Jörg Schnauß (Leipzig University) and was held in a hybrid format (all talked were presented in person and an online audience could participate by asking questions in an online chat). In total, we welcomed up to 100 participants from around the world. There were 21 invited and 11 contributed talks selected from the submitted abstracts. Of all 32 speakers, 10 were female. 46 posters were submitted and presented at this year's meeting – the largest numbers of poster presentations in the history of the POC symposium.

Monday

The first conference day started with a focus session of the DFG research group FOR5628 during which four junior scientists presented their research results to an international community of cancer scientists. After a lunch break, Karl H. Hillebrand (Charité Berlin) opened the first POC session presenting new insights into biomechanics of cancer from a surgical perspective. Unfortunately, the next presentation, the keynote talk from Pramod Pullarkat (Bangalore, India) had to be cancelled on short notice due to illness of the speaker. Nevertheless, a vital and exciting discussion on experimental data ranging from the impact of ECM structure (Hanna Engelke, Graz) to theoretical simulations of glassy dynamics and cancer formation (Takeshi Kawasaki) showed the exciting and successful start of the symposium.

After the coffee break, Viola Vogel from ETH Zürich presented her work on fibronectin as an important mechano-chemical switch that represents a key regulator for the remodeling of the extracellular matrix that we see in various types of cancer. Using bacterial peptide adhesins, her group showed that invasive breast carcinomas are enriched in untensed (unstretched) fibronectin fibers, while fibronectin fibers are highly stretched in different healthy organs. Interestingly, the Vogel group found that immune effector cells appear to accumulate very close to unstretched fibronectin fibers, indicating that the mechanical tension of the fibronectin network in the ECM guides immune cell retention in cancer, possibly in turn regulated by a change in binding affinity of fibronectin to interleukin 7.

In a contributed talk, Clayton Molter from McGill showed his recent work on understanding how androgen-inhibition treatment not only helps to prevent short-term tumor growth, but in the long-term facilitates the tumor to develop an androgen-independent, highly invasive phenotype. He described how his work indicates that the emergence of this androgen- independent phenotype is likely due to androgen-deprivation induced EMT in the cancer cells. He could recapitulate this EMT induction in vitro by quantifying cell motility and contractility.

Debora Monego from the MPI for polymer research presented on her work at the interface between biophysics and mechano-chemistry, namely on the often overlooked property of collagen fibers to form radicals under mechanical load. This effect has far-reaching implications for the tumor microenvironment, as it gives the extracellular matrix a way to self- report on damage by the release of reactive oxygen species, which cells that inhabit the tumor microenvironment can in turn react to.

Alexander Dunn from Stanford presented recent insights from his lab on how molecular linkers between cell-cell junction protein complexes and the cytoskeleton behave under load. Specifically, he studied PDZ domains and showed that these domains can selectively behave a catch-bonds or sliding anchors de-

pending on their binding partners. This differential force sensitivity likely plays an important role in the vertical organizations of cell-cell junctions and the role that this organization plays in pathological cases such as cancer.

In the last talk of Session I (cell and tissue mechanics), Wei Guo from UPenn introduced his findings on exosome secretion, in particular on how tumor cells grown on stiff substrates show a higher degree of exosome secretion, regulated by the Rab8-GTPase. Interestingly, not just the abundancy of exosomes is increased by growing the cells on stiff substrates, but this culture conditions also renders these exosomes into tumor promoting exosomes, regulated by Jagged-1, a ligand for the Notch signaling pathway.

The first symposium day closed with a conference dinner at the historic Aula of the Physics building (Leipzig University), serving a dinner buffet accompanied by live music (special guest: Hot Club d'Allemagne).

Tuesday

Sandrine Etienne-Manneville started the second symposium day (Session II: Intermediate filaments) presenting new insight into the impact of intermediate filaments in the progression of glioblastoma. She showed that intermediate filament depletion in glioblastoma cells results in a reduced invasiveness due to hindered force transmission to the nucleus.

Additionally, on the example of colorectal cancer, Linda Decker presented in a contributed talk, that invasiveness and liver metastasis are strongly influenced by the stiffness of the surrounding tissue and the tumor itself. In fact, patients with soft tumor tissue exhibit a higher survival rate and she asked the question if tumor softening can improve the patients' outcome. Together with a newly developed method combining stiffness mapping on microscopic scale with micrometer-resolution gene expression analysis, she proposed that a mechanical analysis of colorectal cancers can be used for novel therapeutic approaches reducing the risk of liver metastasis.

Acting forces during cell-matrix interactions were in focus of David Böhringer's oral presentation. His group studied collective force oscillations of 3D cell spheroids in a matrix material. They found regular force oscillations taking place every 30 minutes in 3D networks with fibroblast cells which origin was attributed to actin protrusions and the impact of the actomyosin engine. Force transmission between the cells was expected to take place via gap junctions, while calcium signaling coordinates the force oscillations in a 3D environment.

After the lunch break, two highlights of the symposium took place: First, we presented a video "HELA ET AL." by the video artist Tess Marschner. Her work and the video production took place during her artistic research stay at Institute for Bioengineering of Catalonia (IBEC, Spain). The video serves as a reflection on female and reproductive bodies within the technosciences. Subsequently, the poster session took place on the second day - Tuesday, October 1, 2024. 46 posters were presented by mainly junior scientists (undergraduate and graduate students).

Wednesday

The third and last day of the Physics of Cancer symposium was divided into two sessions: Session III: Innovative ex-vivo cancer models and Session IV: Mechanical immune escape mechanisms.

Two talks by Maté Biro and Eleni Dalaka focused on the role of cancer-associated fibroblasts (CAFs) and matrix remodeling on immune cell infiltration of the tumor stroma. Showing stunning time-lapse videos obtained by a bessel-beam lightsheet microscope, Maté Biro illustrated how CAFs build a tunnel system within the tumor stroma that circumvents to tumor mass, and how immune cells follow this tunnel system, effectively diverting them from infiltrating the tumor. Eleni Dalaka nicely complemented this study, by showing that the TGF- beta signalling pathway controls CAF contractility, offering a treatment vector for indirectly enhancing immune infiltration of the tumor stroma, which is in line with an increased survival of mice under TGF-beta inhibition therapy.

Emmanuel Donnadieu shifted the focus from basic research to the study of clinically used CAR T cells, and how novel ex-vivo assays can help to evaluate the efficacy of novel engineered immune effector cells before application to animals and humans. His ex-vivo, tissue-slice culturing method allows to evaluate immune cell function in physiologically relevant tissues (including tumor samples, but also healthy tissues to identify off-target effects) with respect to possible obstacles to immune infiltration, namely suppressing macrophages, dense ECM, and lack of adhesion molecules. In a surprising study, he found that using low-affinity CARs may work better than high-affinity CARs against certain types of tumor, as the low-affinity CAR T cells engage high-antigen expressing targets more specifically, minimizing off-target effects.

Angela Riedel and Trung Phan reported on novel insights into advanced cancer therapy approaches. First, Angela Riedel showed a surprising finding, namely that Anti-PD-L1 checkpoint inhibition treatment works regardless of the PD-L1 expression by the patients' tumor cells. This curious finding led to the investigation of possible off-target effects that can explain the efficacy of the treatment. Her works suggests that the off-target effect in question happens in the tumor-draining lymph node and can be traced to an interaction between fibroblasts and monocytes. Second, Trung Phan from Princeton showed how mathematical elements of game theory can be applied to chemotherapy cancer treatment, specifically to devise an optimal timing of treatment doses to keep the growth of different subpopulations of resis-

tant tumor cells under control and avoid run-away growth that leads to metastasis. Such an optimization process is complicated by the fact that one cannot know how many cells of each resistant subpopulation is present in the patient, one only knows the total cancer burden. His algorithms therefore aim to infer these details from noisy biomarker data to enhance chemotherapy, with first treatments already under way in the clinic.

Finally, Christoph Mark closed the last symposium day with his talk on the role of traction forces during immune cell migration. He showed how immune cells switch between different migration modes in mechanically challenging environments, always reconciling the need to move fast with the need to overcome steric hindrance. The fact that immune cells can exert strong pulling forces via short contractile bursts extends our understanding of immune cell migration and may offer a novel vector for enhancing tumor infiltration by immune cells.

After the final oral presentation, the young scientist award (funded by the DGZ) was announced and awarded to the following junior scientists (all first prices with 250 € each): Linda Decker (Leipzig University): Decoding the biomechanome of colorectal cancer. Mathilde Lettinga (TU Dresden): Plectin mediates cell mechanics across time and length scales. Vaibhav Mahajan (TU Dresden): Growing tumor spheroids from single cells is associated with changes in cell volume and mechanical properties. Sergi Olivé (University of Barcelona, Spain): Stiff environments promote an aberrant adhesion reinforcement.

Author: Mareike Zink, Leipzig University

Christoph Mark during his oral presentation

Acknowledgements

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Meeting report of the 22nd workshop "Cell Biology of Viral Infections" of the German Society for Virology (GfV) in Schöntal

Gabrielle Vieyres[1] and Christian Sieben[2]

1 Institute of Virology and Cell Biology, University of Lübeck, Lübeck, Germany

2 Nanoscale Infection Biology Group, Helmholtz Centre for Infection Research, Braunschweig, Germany and Institute of Genetics, Technische Universität Braunschweig, Braunschweig, Germany

The „Cell Biology of Viral Infections" Workshop of the German Society of Virology (GfV) was held from November 4-6th for its 22nd edition. It is already 10 years ago that the meeting moved to the scenic Schöntal and its monastery, in Baden-Württemberg. This year´s meeting attracted 36 participants, including 21 undergraduate or PhD students, 6 postdocs or senior scientists and 9 PIs. For the second time, the meeting was organized by Gabrielle Vieyres (University of Lübeck) and Christian Sieben (Helmholtz-Centre for Infection Research).

The spotlight of this year´s meeting was set on "membranes" with four invited keynote speakers from Germany, Switzerland, Sweden and the Netherlands and a full session devoted to this topic. The program included 3 additional sessions, following viral infections through entry, viral replication and morphogenesis as well as host-virus interactions.

Prof. Montserrat Bárcena opened the workshop with the first keynote lecture on "Intracellular makeover: viral replication organelles". Dr. Bárcena is an assistant professor at the Leiden University Medical Center where she uses classic and novel electron microscopy approaches to study the replication of positive-strand RNA viruses. She gave an overview of the intracellular membrane reshuffling occurring during viral infections, presenting similarities and differences between her main working horses, including poliovirus and coronaviruses. She also guided us through a deep dive into the structure of SARS-CoV-2 replication organelles, with the structural elucidation and molecular identification of the crown-shaped pores connecting the virus-induced double-membrane vesicles to the cytosol. The following session of the meeting was then devoted to early events in virus infection and included presentations on virus receptor interactions and their inhibition, on the routes of virus internalization and the barrier function of mucus in influenza A virus (IAV) infection.

Prof. Franscesca Bottanelli from the Free University of Berlin kicked off the second day of the meeting with a lecture on "Unravelling the inner secrets of cells with gene editing and live-cell super-resolution microscopy". Prof. Bottanelli illuminated the intracellular vesicular trafficking along the endocytic and secretory pathways using live cell microscopy and a collection of CRISPR-Cas9-based fluorescent knock-in cell lines. Her presentation underscored the organization of cellular membranes in microdomains and shook up textbook knowledge of vesicular transport by presenting clathrin vesicles carried on larger tubular structures. Finally, Prof. Bottanelli shared her newest findings on the membrane organization of the immunological synapse during T-cell activation.

The following session on virus-host Interactions gave the stage for talks assessing the coinfection between emerging flaviviruses, the antiviral activity of cyclosporin A against Nipah virus, or the relevance of the phosphorylation of the arenavirus matrix protein. A presentation on a host-virus binding screening platform set the stage for the use of reductionist synthetic membrane models in studying infection. Another contribution highlighted the potential of confocal interferometric scattering microscopy (C-iSCAT) to track viral infections in label-free conditions.

The third keynote lecture was given by Dr. Markku Hakala, from the University of Geneva. His presentation tackled "the interplay between protein scaffolds and membrane domains at the plasma membrane and endosomes". Dr. Hakala presented his recent work on the ESCRT protein HRS during the formation of intraluminal vesicles in multivesicular bodies. By presenting intriguing biophysical in vitro and cell culture data, Dr. Hakala showed that HRS can form dense multilayer condensates, which supported by clathrin can cluster cargo molecules and aid vesicle formation. Dr. Hakala furthermore highlighted his role is a recently published study showing the organization of membrane domains retained in isolated yeast eisosome filaments.

The following session focused on membranes and further brought them into the context of viral infection with new insights on the role of ceramides in the formation of the coronaviral replication organelle or of lipid mediators in flavivirus replication. Strikingly, the IAV hemagglutinin was shown in cryo-electron microscopy to remodel and zipper intracellular membranes into a so far unrecognized compartment that likely participates in the bundling of the viral ribonucleoproteins for accurate genome packaging.

To round off this exciting day, Prof. Erdinc Sezgin, from the SciLifeLab / Karolinska Institute in Sweden, reported on "Synthetic biology tools to study host-pathogen interactions".

Prof Sezgin´s lab engineers biomimetic systems with reduced complexity such as giant unilamellar vesicles or functionalized bead-supported membrane bilayers. He presented the usefulness of these models to unravel membrane properties but also to study virus-host interactions, for instance by incorporating the SARS-CoV-2 receptor ACE2 in the system and screening for inhibitors of virus binding. The final session was devoted to virus replication and morphogenesis. Unbiased proteomic approaches showed promise in giving new insights into the replication machinery of pestiviruses or in preventing the formation of ebolavirus replication organelle. A presented cryo-electron tomography study also shed light onto the peculiar poxvirus envelopment process.

As usual, the workshop featured several hot topics and cutting-edge techniques in the field of cell biology that currently drive forward our understanding of host-virus interactions. This included the fields of synthetic membrane models, the topic of membrane microdomains and membrane remodeling but also of molecular condensates. The latter was underscored by several speakers including Yannick Jensen, PhD student from the Leibniz Institute of Virology in Hamburg, who revealed how the phosphoprotein pp150 drives liquid-liquid phase separation and tegument formation during human cytomegalovirus assembly. In recognition of this insightful talk, the attendees awarded Yannick Jensen the prize for the best oral presentation (see photo). In terms of methods, the meeting highlighted the usefulness of proximity labeling to unravel the protein machinery involved in viral replication and morphogenesis, with examples on ebolavirus, bovine viral diarrhea virus and Lassa virus. It stressed the potential of gene editing to fluorescently tag endogenous proteins with simplified protocols. It also featured striking examples of cryo-electron microscopy studies and their impact on our understanding of multiple aspects of the viral replication cycles, from the entry and morphogenesis of influenza A virus to the replication organelles of plus-strand RNA viruses or the assembly of the large poxviruses. Finally, the iSCAT technology opened new perspectives to investigate virus infections with minimal perturbations.

We are grateful to all participants of this 22nd edition for sharing their exciting research, participating in the lively discussions and contributing to the friendly atmosphere of the meeting. Our gratitude also goes to our four guest speakers and to our faithful sponsors, the German Society for Virology (GfV), the German Society for Cell Biology (DGZ), and the company ReBlikon GmbH, whose continued financial and administrative support over the years make this meeting durable. The 22nd workshop "Cell Biology of Viral Infection" of the German Society of Virology (GfV) will take place from October 8th to 10th 2025, again at Schöntal Monastery. For updates, please visit our website at https://cellviro.g-f-v.org/.

The 22nd edition of the "Cell Virology of Viral Infections" Workshop of the GfV took place from November 4th to November 6th at the Kloster Schöntal.

Yannick Jensen, PhD student at the Leibniz Institute of Virology (Hamburg), surrounded by the organizers Christian Sieben and Gabrielle Vieyres. Yannick Jensen received the prize for his presentation on the role of liquid-liquid phase separation in HCMV tegument formation.

27th Meeting on Signal Transduction 2024 by the Signal Transduction Society (STS)

The 27th STS Meeting on Signal Transduction took place from November 4-6, 2024, at the Leonardo Hotel in Weimar. This Meeting was corporately organized by the STS and the DGZ study group 'Membrane Organization & Contact Sites (headed by Julia Groß)' together with the German Societies for Immunology (DGfI), for Biochemistry and Molecular Biology (GBM), for Pharmacology (DGP) as well as the Collaborative Research Center "Structural Dynamics of GPCR Activation and Signaling" (SFB1423, Leipzig, Berlin, Halle). 121 participants including the industrial partners attended this years' STS meeting. We are grateful to our many industrial sponsors for their ongoing support and the DFG for supporting this meeting.

Each workshop started with keynote presentations by well-known experts in the specific research field. In the 'Immune Cell Signaling' workshop, Bernard Malissen (Marseille, France) and Jonathan Jantsch (Köln) opened the meeting on Monday at noon. Bernard Malissen presented new data on T cell receptor (TCR)-mediated signaling. Using a quantitative "interactomics" approach and genetically engineered mouse strains lacking molecules of the TCR pathway, his group could show that the linker for activation of T cells (LAT) is a central molecule in inflammatory and autoimmune diseases similar to human IgG4-related diseases. Jonathan Jantsch highlighted that sodium ion levels influence the process of inflammatory reactions during bacterial infections by osmotic upregulation of cytokine synthesis. Three short talks selected from the submitted contributions completed the workshop with highly interesting aspects on neutrophil kinase signaling (Mareile Schlotfeldt), on cytokine expression (Christian Ehlting) and cytokine receptor signaling (Doreen Floss). 'Hot topics in Signal Transduction' were presented in the evening workshop on after dinner. Barbara Di Ventura (Freiburg) gave fascinating insights into the dynamics of transcription factors and gene expression using synthetic transcription factors based on p65 and p53 as models together with live cell microscopy and mathematical modeling. Markus Feuerer (Regensburg) introduced the transposable elements (TE) landscape of CD45+ immune cells as well as synthetic immunology tools, such as Artificial Immune Receptors (AIRs). Bioinformatics and Artificial Intelligence were the topic of the short talk on Computational pipelines based on artificial intelligence in which Julian Borbeck (Mainz/Tübingen) introduced the Xyna.bio platform and suggested that such pipelines can be used as flexible and low-threshold tools for biomedical research. Lukas Althoff (Freiburg) closed the session by explaining OptoMitoImport, an optogentic tool for controlling protein import into the mitochondrial compartment. This inspiring session was followed by an informal get-together in front of the Belvedere exhibition hall creating a perfect opportunity to enjoy scientific discussions, reconnect with old colleagues and make new connections with a drink or a snack in hand.

Vivid conversations at the STS evening reception. © Tina Hagedorn

The second day of the meeting started with a session on 'Growth Factors, Cytokines and their Receptors' and keynote talks by Jürgen Scheller (Düsseldorf) and Harald Wajant (Würzburg). Jürgen Scheller (Düsseldorf) gave a highly interesting talk about deciphering Interleukin 6 signaling by designing synthetic cytokine receptors (SyCyRs) and nanobodies as extracellular sensors for homo-and heteromeric synthetic cytokine modules which induce receptor dimerization. Harald Wajant's talk focused on the family of tumor necrosis factor receptors (TNFRs) which can be subdivided into two classes depending on their ability to get activated by soluble (type I) or membrane bound (type II) trimeric ligands. The short talks by Yin Xiao and Katharina Kubatzky highlighted further aspects of TNFR family members and the importance of protein succinylation in RANK-induced signaling. Doan Duy Hai Tran explained in his short talk the effect of the germinal center-associated nuclear protein (GANP) for the transcription of immediate early genes in cytokine-induced signaling cascades.

In the Workshop, 'From Basics to Translation', Mohamed Lamkanfi (Ghent) suggested that signaling events of the inflammasome can be used to address inflammatory diseases and highlighted the role of Gasdermin D. Daniel Krappmann (Neuherberg) showed how a combination of precision oncology and immunotherapy can help in the treatment of BCR-driven lymphomas by targeting the MALT1 protease. These talks was followed by short talks on the use of a multi-lineage 3D culture system to model tumor cells (Alexandra Koch), the role of MID1 in the aberrant production of the neurotoxic mutant HTT protein in Huntingdon's disease (Sybille Krauss) and a presentation by Annika Reisbitzer on a newly developed protein-protein interaction system.

STS medal award ceremony. Ursula Klingmüller, Detlef Neumann, Katharina Kubatzky, Lewis Cantley, Klaudia Giehl, Jonathan Lindquist (left to right). © Tina Hagedorn

The SFB1423 'Structural Dynamics of GPCR Activation and Signaling' (Leipzig, Berlin, Halle) represented by Claudia Stäubert, organized the Workshop 'G Protein Mediated Signaling'. Alexander Hauser (Copenhagen) explained the concept of genomic medicine using the glucose-dependent insulinotropic polypeptide (GIP) receptor (GIPR) in combination with glucagon-like peptide-1 (GLP-1) receptor (GLP-1R) and demonstrated the impact of β-arrestins in regulating GIPR signaling. In a second keynote talk, Julian Hanson (Liege) explained how orphan GPCRs contribute to the control of cellular energy homeostasis thereby focusing on orphan receptors in the hypothalamus and their possible functions. Short talks were presented by Tim Pelczyk (Leipzig) on the neuropeptide Y Y4 receptor agonists, Sarah Bühler (Jena) talking about the interaction of SPRED1 and Ras, and Claudia Stäubert (Leipzig) presenting the Hydroxycarboxylic acid receptor 2.

The evening was dedicated to the STS Honorary Medal Award ceremony for Lewis Cantley. The STS Medal Award was established in 2010 to honor outstanding scientists from cell biology, biomedicine or bioinformatics, who made landmark contributions in the fields of basic and/or applied signal transduction research. The identification and characterization of PI3 kinase signal transduction by Lewis Cantley was a milestone in the field of growth factor signal transduction, which had greatly increased the understanding of cancer cell biology. The award ceremony was opened by a very personal laudation for the awardee by Ursula Klingmüller (Heidelberg), a former postdoc in the Cantley lab. The medallist then gave the 'Honorary Medal Lecture', in which he presented an overview of his research with a focus on prediction methods of protein phosphorylation sites by protein kinases.

The last day started with the DGZ sponsored workshop on 'Tumor Cell Biology' where Sandrine Etienne-Manneville (Paris) highlighted how mechanotransduction affects glioblastoma, a highly invasive although not metastasizing brain tumor, thereby concentrating on the role of intermediate filament system. A second keynote talk was given by Manfred Jücker (Hamburg) on the Akt/mTOR pathway and its effect on the metastatic potential of circulating tumor cells in a colorectal carcinoma xenotransplantation model. Short talks were given by Maike Buchner (München) talking about the dual-specificity phosphatases DUSP1 and DUSP6 and Setenay Gupse Özcan (Jena) presenting new aspects of the RFX transcription factor family.

Katja Simon (Berlin) addressed in her vivid keynote presentation, in the workshop 'Differentiation, Stress, and Death', the role of autophagy in immune metabolism, differentiation and aging. Katja Simon's group combines mouse models, human blood cells and data from clinical trials for her autophagy studies. Lina Herhaus (Frankfurt) gave a short talk on the role of the MHC-I quality control in tumor cell immune evasion and Igor Kovacevic (Halle) how ER stress inhibition enhances formation of triacylglcerols and protects endothelial cells from lipotoxicity. Anna Burova (Halle) explained the role of the Myocardin-Relat-

ed Transcription Factors (MRTF) by using transgenic mice, which have a defect in feeding their offspring.

The last workshop of the meeting addressed aspects of 'Infection and Inflammation'. Roland Lang (Erlangen) opened the workshop and addressed the role of the metabolite itaconate in immune-regulation and anti-bacterial defense mechanisms. His presentation was followed by a keynote talk by Serge Mostowy (London), who focused on the role of the cytoskeleton in the control of intracellular Shigella infections and presented new aspects for the role of septins in host defence. In the short talks Elisabeth Seebach highlighted the role of the cGAS/STING pathway in S. aureus infections, Patricia Korn (Aachen) talked about the role of Mono-ADP-ribosylation in the context of viral infections and the session was closed by Franziska Greulich (Weihenstephan) with her talk on the role of BRG1 in the activation of glucocorticoid induced gene transcription.

As in the previous years, posters were presented on two occasions in the plenum in the 'My Poster in a Nutshell' sessions as one-minute short talks before meeting interested colleagues in the poster exhibition area. The workshop chairpeople selected five award-winning posters out of the many contributions, and awarded prizes to a total value of 750 € to Steffen Harms and Mia Mönnig (both Osnabrück), Mareike Sieler and Anna Hagemann (both Witten-Herdecke) and Xiaolo Lou (Würzburg).

Since 2005, the STS Science Award has been offered as a reward for excellent research by an early career researcher of the STS. This year, the STS Science Award went to Lina Herhaus (Braunschweig) for her excellent work on 'Tumor immune evasion through IRGQ-directed autophagy'. Lina is a junior group leader of the MICROSTAR project at the HZI Braunschweig. Moreover, Annika Reisbitzer (Siegen) was awarded with the GBM Innovation award (500 €). In addition, seven students, namely Yue Gao, Heidelberg, Franziska Bischof, Leipzig, Annika Reisbitzer, Siegen, Marieke Krüger, Gießen, Dora Gjirlic, Halle, and Tianjao Luo from Würzburg received travel grants of 300 € each to allow their meeting attendance.

Of course, preparations for the 28th STS Meeting (3.-5.11.2025 at the Leonardo Hotel in Weimar) have already started, and regular updates on the schedule and contents of the meeting can be found at https://www.sigtrans.de. Additionally, news regarding the work of the STS can be accessed via LinkedIn and BlueSky (@sigtrans.bsky.social).

Best wishes,
Klaudia Giehl (on behalf of the STS council)

Contact:
Prof. Dr. Klaudia Giehl
Justus-Liebig-Universität Gießen
Signaltransduktion zellulärer Motilität
Medizinisches Forschungszentrum Seltersberg
klaudia.giehl@innere.med.uni-giessen.de

Winners of the poster prizes 2025 handed over by Jonathan Lindquist: Xiaoli Lou, Mareike Sieler, Mia Mönnig, Steffen Harms . © Tina Hagedorn

In Memoriam – Angelika A. Noegel

Angelika Noegel, a renowned cell biologist and "role model female scientist", sadly passed away on January 19, 2025. She was born on February 14, 1952 in the beautiful town of Bamberg in Upper Franconia, Bavaria. She studied Biology in Würzburg with a scholarship of the prestigious *Studienstiftung des deutschen Volkes,* moved then to the lab of Prof. Dr. Werner Goebel and received her PhD in 1979 from the University Würzburg. From 1979 until 1983 she worked as postdoctoral researcher at the Rockefeller University in New York City. From there, she went to the Max Planck Institute (MPI) of Biochemistry, Department of Cell Biology (Director: Günther Gerisch) in Martinsried near Munich, where she stayed until 1997 as researcher and Principal Investigator (PI). In 1997 she was appointed Full Professor (C4) of the Institute for Biochemistry I at the Medical Faculty of the University of Cologne, where she stayed until her retirement in 2017.

For more than 30 years, Angelika has devoted most of her research to the unicellular amoeba *Dictyostelium discoideum* and has made major contributions to the fields of the cytoskeleton, signal transduction and genomics. As PI at the MPI of Biochemistry, she and colleagues published analyses of several single mutants of evolutionarily highly conserved actin-interacting proteins (Andre et al., 1989; Brink et al., 1990; Noegel and Witke, 1988). Surprisingly, these mutants showed no detectable phenotype under laboratory conditions, leading to the hypothesis of redundancy of actin-interacting proteins (Bray and Vasiliev, 1989). In the following years, the team around her and her husband Michael Schleicher were able to generate double knock-out mutants of actin-interacting proteins by homologous recombination that showed clear phenotypes, supporting the concept of functional redundancy in the microfilament system (Haugwitz et al., 1994; Noegel and Schleicher, 2000; Witke et al., 1992). Angelika was also a key player in the international consortium to sequence the *D. discoideum* genome. This project was a major challenge at that time because of the high AT-content of the genome and her contributions were instrumental for its successful completion (Eichinger and Noegel, 2003; Eichinger et al., 2005; Glockner et al., 2002).

Angelika was not only extremely dedicated to her work in *D. discoideum*. In her years at the University of Cologne, she very successfully extended her activities to research with *Mus musculus*, focusing on the composition and function of the protein

Angelika Noegel and her husband Michael Schleicher at the International Dictyostelium Conference 2005 in Autrans, France (picture provided by Dr. Yogikala Prabhu, National Institute of Allergy and Infectious Diseases, USA)

complexes involved in the connection between the nucleus and the cytoskeleton (Noegel and Neumann, 2011; Zhen et al., 2002). She also investigated the genetic basis of microcephaly with Peter Nürnberg's group at the Cologne Center for Genomics (CCG) (Martin et al., 2014; Moawia et al., 2017).

Moreover, Angelika was a very active and highly respected member of the scientific community. In the year 2000, she was elected as member of the *European Molecular Biology Organization* (EMBO). From 2002 until 2005, she acted as vice president, from 2005 until 2006 as president of the *German Society for Cell Biology* (DGZ), and from 2007 until 2009 as vice president of the *Verband Biologie, Biowissenschaften und Biomedizin in Deutschland e.V.* (VBIO). In addition to these roles, she has served as a reviewer for prominent funding organizations such as the *European Research Council* (ERC), EMBO, the *Deutsche Forschungsgemeinschaft* (DFG), and as member of the Editorial Board of the Journal of Cell Science. In her various science policy functions and as head of the Institute for Biochemistry I, she was always a strong advocate of basic research and for women in science. In her career, she supervised numerous bachelor and master students and about 100 PhD students, several of which are now professors themselves in Germany, the USA and China.

A former PhD student of her characterized her aptly in a letter of mourning to her husband: "Frau Noegel was a woman of few words but she changed the lives of many people in a very meaningful way. Actions speak louder than words. She was not only a great mentor but also a very important part of my journey. She was humble, polite, elegant, warm, and just a wonderful soul. She was kind and treated everyone with respect, no matter who we were and where we came from. I have very fond memories with her, and I am going to cherish them and will miss her deeply" (personal communication).

We have both known Angelika for nearly forty years and we have always admired her passion for research. Despite her many duties, she worked almost every day also at the bench until her retirement. In her leisure time, Angelika loved to cook and she was an enthusiastic visitor of operas, in particular those by Richard Wagner. It is worth to mention, that her love for music was not just passive, she actively played the piano herself. Furthermore, she also enjoyed reading classical literature and city trips, in particular she was fascinated by Rome.

With Angelika Noegel we have lost an outstanding researcher, an excellent mentor of young scientists, and a dear friend and colleague. Finally, a short text from her death notice, which in our opinion characterizes her very well: "I only ever saw her move forward. I never saw her stop when she had achieved something" (similar to an old saw from Confucius).

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Affiliations

Annette Müller-Taubenberger, Biomedical Center – Cell Biology (Anatomy III), LMU Munich

Ludwig Eichinger, Center for Biochemistry, Medical Faculty, University of Cologne

Protokoll der Mitgliederversammlung 2024 der Deutschen Gesellschaft für Zellbiologie e.V.

Versammlungsleiter und Protokollführer: Prof. Dr. Roland Wedlich Söldner, Präsident

Geschäftsführerin: Prof. Dr. Gislene Pereira

Die Mitgliederversammlung fand am 11.10.2024, 12.00 Uhr bis 13.00 Uhr, online über Zoom statt.

Alle Mitglieder wurden rechtzeitig durch Ankündigung in unserem Online-Mitgliederjournal „Cell News" sowie mehrmals über E-Mail eingeladen und über die Tagesordnung und Zugangsdaten zur Versammlung informiert.

Tagesordnung:

1. Bestätigung des Protokolls der letzten Sitzung
2. Jahresbericht des Präsidenten mit anschließender Diskussion
3. Geschäfts- und Kassenbericht über das abgelaufene Kalenderjahr
4. Bericht der Rechnungsprüfer:in
5. Entlastung des Vorstandes
6. Ergebnisse der DGZ Wahlen 2024-2026
7. Sonstiges

TOP 1. Bestätigung des Protokolls der letzten Sitzung

Das Protokoll der letzten Mitgliederversammlung 2023 (online über Zoom am 12.12.2023) war in unserem Online-Mitgliederjournal „Cell News", Ausgabe 1/2024 veröffentlicht worden und wird bestätigt.

TOP 2. Jahresbericht des Präsidenten

Roland Wedlich-Söldner berichtet über die Aktivitäten der DGZ in 2023 und 2024. Zu den Mitgliederzahlen informiert er, dass wir im Jahr 2023 43 neue Mitglieder gewinnen konnten und 64 Austritte verzeichnen mussten. Zum Zeitpunkt der Mitgliederversammlung hatten wir für das laufende Jahr 2024 18 Neuzugänge und 29 Austritte. Die aktuelle Mitgliederzahl beträgt 668.

Die DGZ-Preise 2024 wurden online per Zoom am 07.10.2024 verliehen. Der Werner Risau Preis wurde 2023 zum letzten Mal verliehen, stattdessen wurden 2024 zum ersten Mal zwei Preise für ausgewählte Masterarbeiten verliehen. Die Preisträger*innen für diesen „DGZ Master Thesis Award" waren Tamara Eleanore Hamann und Konrad Steinbach. Zudem erhielt Dr. Martin Würtz den Nikon Young Scientist Award, Dr. Claudia Matthäus den Walther Flemming Award und Dr. Alba Diz-Munoz den BINDER Innovationspreis. Die Carl Zeiss Lecture wird seit 2023 zweijährig jeweils im Rahmen der DGZ International Meetings verliehen, also das nächste Mal 2025 in Heidelberg. Die Preisträger:innen hielten Online-Vorträge, die von den Mitgliedern im gemeinsamen Zoom-Webinar verfolgt werden konnten.

Als Publikationsorgan der DGZ wurden in 2024 zwei Ausgaben der „Cell News" veröffentlicht. Zudem wurden alle wichtigen Informationen über E-Mail und zusätzlich via X/Twitter verbreitet. Weiterhin berichtet Roland Wedlich-Söldner über Aktivitäten der DGZ auf politischer Ebene (Wissenschaftszeitvertragsgesetz, Tierversuche).
Sandra Iden stellte den Stand der seit 2022 laufenden Focus Workshop-Serie und einige Neuerungen in der Zusammensetzung für 2024 vor.
Roland Wedlich-Söldner stellt abschließend noch die Aktivitäten der DGZ auf Twitter/X sowie den Stand der DGZ-Webseite dar.

TOP 3. Geschäfts- und Kassenbericht über das abgelaufene Kalenderjahr

Die Geschäftsführerin Gislene Pereira berichtet über die Finanzlage der DGZ im Geschäftsjahr 2023 und erläutert diese im Detail anhand der Einnahmen- und Ausgaben-Bilanzen und v.a. eingehend auf Einzelpositionen wie Einkünfte durch Mitgliedsbeiträge sowie Ausgaben für Büro- und Personalkosten, Konferenzunterstützung und Steuerberatung. Das Guthaben betrug zum 31.12.2023 EUR 73.401,38.

TOP 4. Bericht der Rechnungsprüfer*in

Die Einnahmen und Ausgaben im Geschäftsjahr 2023 waren durch die Rechnungsprüferin Prof. Dr. Maria Bohnert und den Rechnungsprüfer Prof. Dr. Ralph Gräf geprüft und für richtig befunden worden, es gab keine Beanstandungen. Die Prüfung der Unterlagen erfolgte digital, die Unterlagen wurden per E-Mail an die Rechnungsprüfer:in zur Durchsicht und Überprüfung geschickt.

TOP 5. Entlastung des Vorstandes

Der Vorstand wird über online-Abstimmung (in Zoom) einstimmig – mit Enthaltungen der Vorstandsmitglieder – entlastet.

TOP 6. Ergebnisse der DGZ Wahlen 2024–2026

Der Vorstand, drei Beiratsmitglieder und die beiden Kassenprüfer der Gesellschaft wurden neu gewählt. Die Wahl wurde vom 9. September 2024 bis 30. September 2024 anonym und

BILANZ 2023

EINNAHMEN	EUR	AUSGABEN	EUR
Mitgliedsbeiträge (abzgl. Retouren)	47.470,00	Bankkosten	815,05
Spenden, Preisgelder	15.500,00	Retoure Mitgliedsbeiträge	730,00
Zinsen	284,34	Spenden, Preisgelder	23.500,00
Cell News, Homepage (Werbeanzeigen, Firmen-Links)	5.533,50	Cell News	895,50
		Tagungen	620,00
Tagungen	0,00	Reisekosten	1.380,23
Überträge	15.846,31	Bürokosten/Gehalt Sekr.[1] Büromaterial, Homepage	30.707,28
Sonstige	1.280,00		
		Überträge	15.846,31
		Sonstige	6.631,64
Summe der Einnahmen:	85.914,15	Summe der Ausgaben:	81.126,01
Guthaben am 31.12.2022:	68.613,24	**Guthaben am 31.12.2023:**	**73.401,38**
Guthaben DGZ:	50.341,92	Guthaben DGZ:	59.239,09
Werner Risau Preis:	18.271,32	Werner Risau Preis:	14.162,29

Die Einnahmen und Ausgaben wurden von Kassenprüferin Julia Groß und Kassenprüfer Ralph Gräf geprüft und für richtig befunden.

personalisiert online durchgeführt, und die Mitglieder wurden in unserem Online-Mitgliederjournal „Cell News" und mehrmals per E-Mail mit Link zum Wahlportal darüber informiert. An der Wahl nahmen 108 Mitglieder teil. Die Wahlergebnisse werden in der Mitgliederversammlung bekanntgegeben:

Vorstand:
Sandra Iden (bisher Vizepräsidentin) wurde mit 103 Ja-Stimmen, 1 Nein-Stimme, 4 Enthaltungen zur neuen Präsidentin gewählt.
Julia Groß (bisher Vizegeschäftsführerin) wurde mit 100 Ja-Stimmen, 3 Nein-Stimmen, 5 Enthaltungen zur neuen Vizepräsidentin gewählt.
Jörg Höhfeld wurde mit 100 Ja-Stimmen, 2 Nein-Stimmen, 6 Enthaltungen zum neuen Geschäftsführer gewählt.
Maria Bohnert wurde mit 98 Ja-Stimmen, 2 Nein-Stimmen, 8 Enthaltungen zur neuen Vizegeschäftsführerin gewählt.

Als Beiratsmitglieder wurden gewählt:
M. Cristina Cardoso
mit 101 Ja-Stimmen, 4 Nein-Stimmen, 3 Enthaltungen.
Volker Gerke
mit 103 Ja-Stimmen, 0 Nein-Stimmen, 5 Enthaltungen.
Anne Straube
mit 100 Ja-Stimmen, 3 Nein-Stimmen, 5 Enthaltungen.

Als Rechnungsprüfer wurden gewählt:
Fabian Erdel
mit 102 Ja-Stimmen, 0 Nein-Stimmen, 6 Enthaltungen.
Ralph Gräf
mit 105 Ja-Stimmen, 1 Nein-Stimme, 2 Enthaltungen.

Alle Gewählten waren vorab über das Wahlergebnis informiert worden und haben auf Nachfrage ihre Wahl angenommen.

TOP 7. Sonstiges

Roland Wedlich-Söldner berichtet über die neu etablierte Kollaboration mit dem European Journal of Cell Biology. Der Sprecher der Workgroup „Physics of the Cell", Dr. Leonhard Möckl, organisiert über dieses Journal momentan ein Schwerpunkt zum Thema Glykobiologie.

Abschließend weist Gislene Pereira auf das kommende 50-Jahre-Jubiläum der DGZ hin und auf die damit verbundene DGZ-Tagung in Heidelberg vom 26.-29. Oktober 2025. Sie berichtet detailliert über das Programm und den Ablauf.

Prof. Dr. Roland Wedlich-Söldner
Präsident
Versammlungsleiter
und Protokollführer

Prof. Dr. Gislene Pereira
Geschäftsführerin

DGZ Focus Workshops 2025

Zoom, last Tuesday of a month, noon – 2pm

(for questions contact Maria Bohnert, bohnertm@uni-muenster.de)

April 29, 2025	Cell Biology of the Immune System Eva Kiermaier, Pablo Saez
May 27, 2025	Functional Organization of the Nucleus Zuzana Storchova, Cristina Cardoso
June 24, 2025	Imaging for Cell Biology Helge Ewers, Stefan Pfeffer
July 29, 2025	Cellular and Organismal Proteostasis Jörg Höhfeld, Thorsten Hoppe
September 30, 2025	Mitosis and Meiosis Simone Reber, Thomas Mayer

https://www.zellbiologie.de/workgroups/

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Editors:
Prof. Dr. Julia Groß
(HMU Health and Medical University Potsdam)
Prof. Dr. Jörg Höhfeld
(Universität Bonn)
Prof. Dr. Maria Bohnert
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Prof. Dr. Ludwig Eichinger
(Universität zu Köln)
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(Universität Münster)

Editorial Office Manuscripts/Advertisements:
Sabine Reichel-Klingmann
Office of the German Society for Cell Biology
c/o German Cancer Research Center
Im Neuenheimer Feld 280
69120 Heidelberg
Tel.: 06221/42-3451
Fax: 06221/42-3452
E-mail: dgz@dkfz.de
Internet: www.zellbiologie.de

Media Creation:
Werner Rehberger
WR Grafik-Design
68799 Reilingen
wr@wr-grafikdesign.de

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