Cell News

Newsletter of the German Society for Cell Biology

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CONTENT

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Cover Image:
Group photo of the 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 (Photo Benedikt R. Dürr)

Dear members of the DGZ,

A remarkable year comes to an end. In our last editorial, we discussed the turbulent times that scientists worldwide face. Since then, several support measures have been implemented to support some of those who wish to relocate to Europe.

In this CellNews issue, we wish to celebrate one more time that 2025 marks the 50th anniversary of the DGZ. At the end of October, around 260 participants (approximately 40 % of whom were DGZ members) came together in Heidelberg for a highly interactive and stimulating DGZ International Meeting (https://www.zellbiologie.de/dgz-international-meeting-2025/) (page 17).

We heard inspiring keynote lectures by Tony Hyman, Stefan Hell, Bernd Bukau, and Maria Leptin, who provided insights into ERC strategies. We learned about the latest data from many young scientists in short talks and intensive poster sessions. Participants enjoyed a beautiful venue and numerous opportunities for networking and the exchange with sponsors at the industry booths. We were also very pleased that this year's DGZ awards could be presented during the conference and that all awardees gave a presentation of their research (see articles on the Nikon Young Scientist Award, the Walther Flemming Award, and the Innovation Award in this issue, page 6-16).

We are grateful to everyone who made this meeting such a success! See you in Bonn in 2027 (more information will follow in the coming months).

November also saw a very constructive exchange between the DGZ Executive Board and the DGZ Advisory Board, during which several ideas were discussed that we are now working on behind the scenes. A main goal will be to further increase the attractiveness of the DGZ for young scientists. In addition, we recently held the annual DGZ General Meeting online (see page 23 for the minutes). As always, we welcome your feedback and suggestions on how we can continue to improve our cell biology society.

We wish everyone who celebrates Christmas a peaceful holiday season and all our members a happy new year.

Best regards,

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

DGZ Awards 2025

DGZ Master Thesis Awards

Bastian Hinkel
RPTU Kaiserslautern-Landau

Silja Zedlitz
MPI-MR Heidelberg,
now at MPI-CBG

Nikon Young Scientist Award

Tobias Kletter
i3S, Porto, Portugal

Walther Flemming Award

Lina Herhaus
HZI Braunschweig

Innovation Prize

Elias H. Barriga
PoL & TU Dresden

Carl Zeiss Lecture

Julia Mahamid
EMBL Heidelberg

Nikon Young Scientist Award 2025
Physical properties of the cytoplasm drive mitotic organelle size control

Tobias Kletter

Introduction

Throughout the lifetime of the animal organism, cells proliferate, undergo developmental changes, form tissues and cope with stresses. **How do organelles adapt to these changing cellular contexts to maintain function?** The mitotic spindle is a prime example to investigate organelle plasticity. It is a highly dynamic, self-organising structure, transiently assembling and disassembling within a short time to perform the critically important task of chromosome segregation. All of these characteristics emerge from countless molecular interactions of small building blocks, tubulins, polymerizing into dynamic filaments called microtubules that assemble into a spindle with the help of molecular motors and microtubule-associated proteins. Spindle assembly errors can have grave consequences for both the developing as well as the adult organism[1]. Thus, investigating subcellular construction principles, such as those exemplified by the spindle, promises insights into the fundamental biology underlying health and disease.

Proper construction and geometry are critical to spindle function: two poles – and exactly that number! – need to span an adequate distance to fully separate the sister chromatids onto the nascent daughter cells. Therefore, **the spindle needs to reach a proper size relative to the dividing cell**.

Early animal embryos have served as the perfect systems to investigate spindle plasticity across length scales[2–7]. Animal embryos begin as a single large cell that quickly divides to produce smaller and smaller cells (Figure 1a). In very large cells, spindle size is decoupled from cell size but spindles start scaling after cells reach a critical size[6] (Figure 1b). Current models propose volume-sensing[3,8] and surface-sensing[5,9] mechanisms for spindle scaling. Briefly, microtubule nucleation and dynamic instability are thought to respond to changes in building block and/or growth regulator availability. Since this availability is expected to change with cell volume and/or surface area, spindle sizes will self-correct to match the dimensions of the dividing cell[10].

While the field learned a lot by studying these early embryonic divisions, we still know little about spindle plasticity in later stages of development, when the cellular environment is expected to undergo changes as cells differentiate into various tissues (Figure 1c). Of all the embryonic tissues, the developing brain is especially sensitive to mutations in spindle and centrosome genes[11], **prompting us to ask how neurally-differentiating cells cope with spindle scaling**.

A differentiation-mediated switch in spindle size scaling

Adapting established protocols[12], we differentiated murine embryonic stem cells towards neural cell fates and confirmed the time-course of marker expressions and morphological changes until terminal differentiation into beta3-tubulin positive neurons. Throughout the time-course, using a customized and fully automated adaptive feedback microscopy workflow, we imaged thousands of differentiating cells over several generations. With the use of high-throughput image analysis tools[4,13] to measure spindle and cell geometry, we made our first surprising discovery. Yes, spindles scaled relative to cell sizes in our differentiation system. However, **when we compared cells with equal volumes, spindles in differentiating cells were consistently shorter, narrower and made from less microtubule mass than spindles in cells before differentiation**. What factor other than cell size was controlling spindle size in this system?

Spindle microtubule mass emerges from processes that regulate tubulin polymerization, such as nucleation, growth and shrinkage. Using the microtubule tip marker EB1, we unexpectantly found that spindle microtubule growth speed and the total number of growing tips was consistent between the differentiation states. However, **we found a shift in microtubule growth from the spindle bulk towards the spindle poles in differentiating cells**. This prompted us to take a closer look into centrosomes, important microtubule-organizing centers at the poles of metaphase spindles and responsible for the nucleation of astral microtubules. Indeed, centrosomes in differentiating cells were relatively enlarged and recruited more of the available gamma-tubulin, a specialized tubulin isoform involved in microtubule nucleation. Together, these findings suggested that differentiation influenced the microtubule nucleation capacity of the centrosomes to fine-tune spindle architecture.

Cytoplasmic dilution upon differentiation modulates mitotic architecture

There is a crosstalk between cellular differentiation and the biochemical, physical and mechanical properties of the cells and their environment. Already classical purification protocols made use of the differential buoyant densities of embryonic brain cells[14]. Mature neurons were found to be less dense than less-differentiated precursors. Thus, we speculated that the physical properties of the cytoplasm could influence subcellular architecture in our differentiation model.

Indeed, when we used quantitative phase imaging[15] on mitotic cells to determine their dry mass densities, we found a 10 % decrease in differentiating cells compared to undifferentiated stem cells. Consistently, when we matched mitotic dry mass densities by diluting the undifferentiated cytoplasm by osmotic treatments, we could fully recapitulate the differentiation-mediated mitotic scaling phenotype, as microtubules shifted from the spindle bulk to the asters in the diluted cytoplasm. Conversely, re-concentrating the cytoplasm of already differentiated cells restored spindle scaling to pre-differentiation levels. These results thus indicated that cytoplasmic dilution was sufficient to alter spindle scaling and architecture along differentiation trajectories.

How could cytoplasmic dilution stimulate centrosomal microtubule nucleation? It has been described that the centrosomal regulator CPAP can influence centrosomal activity by helping to recruit microtubule-nucleating factors, but is inhibited by unpolymerized tubulin[16]. We reasoned that cytoplasmic dilution could liberate CPAP from this interaction to drive centrosomal activity upon differentiation. Indeed, when we treated undifferentiated cells with a small molecule drug[17] that specifically liberated CPAP from tubulin inhibition without affecting overall tubulin concentration, the cells increased centrosomal nucleation capacities, nucleated more astral microtubules yet assembled smaller spindle bulks (Figure 2). In summary, this suggested that **centrosomes reacted to differentiation-mediated changes in cytoplasmic density to fine-tune spindle architecture**.

While complementing established organelle scaling models[3,5,18] our findings demonstrate that the physical properties of the cytoplasmic environment hold cell state-specific information for organelle size control[19] (Figure 2). Our study also suggests a possible link between cell physical properties and neurodevelopmental defects, since CPAP is a known culprit 20 connecting spindle orientation defects with aberrant brain development. Thus, it will be intriguing to further investigate the upstream causes as well as the downstream consequences of changes in cytoplasmic density along developmental trajectories.

Acknowledgements

I would like to thank the DGZ and Nikon Deutschland for the award and highlighting our work. A special thank you to Simone Reber for many years of great mentorship and unconditional support, and to all members and alumni of the lab for creating such a friendly place to work. I would like to thank all of our collaborators for their expertise and help. I am grateful for the support at the Maiato lab, my current home. Lastly, I want to express my heartfelt gratitude towards my family and friends.

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Tobias Kletter

Tobias holds a Bachelor's in Biology during which he investigated the cell biology of apicomplexan parasites. During his Master's in Molecular Life Sciences (HU Berlin), he first worked in the field of RNA biology, before falling in love with the microtubule cytoskeleton, cell division and microscopy. To study the plasticity of the microtubule cytoskeleton in dividing stem cells, he joined the group of Prof. Simone Reber (HU Berlin and MPI for infection biology, Berlin) as a PhD student, while frequently visiting the Advanced Light Microscopy Facility at the EMBL Heidelberg. Recently, for his postdoc, Tobias joint Prof. Helder Maiato's group in Porto to investigate evolutionary aspects of mitosis.

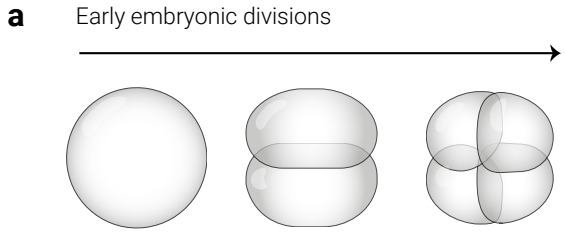

Figure 1a: *Animal development starts with the fertilized egg that produces a growing number of smaller and smaller cells, which have served as an ideal model system to study organelle size scaling.*

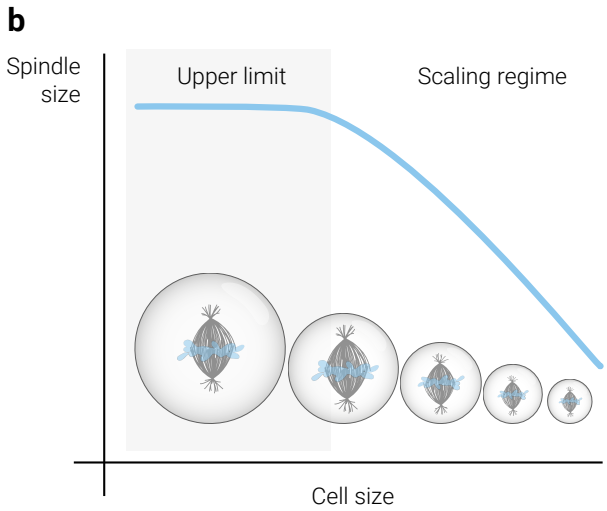

Figure 1b: *Generally, in very large cells, spindle size is decoupled from cell size and reaches the so-called "upper limit". After a certain number of reductive divisions (little cell growth in between mitoses), cell size reaches a threshold after which spindles will scaling with cell size.*

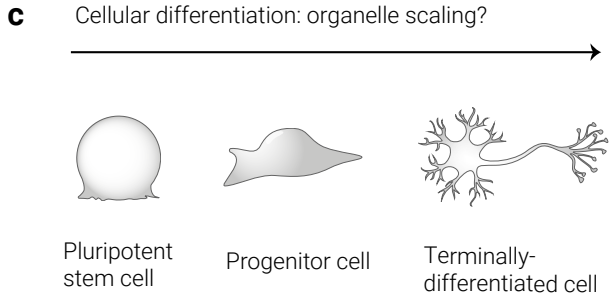

Figure 1c: *Before terminal differentiation to mature neurons that stop dividing, non-committed cells transition through multiple stages of progenitor cells. How cells undergoing functional changes - such as the ones happening during differentiation - cope with organelle scaling during mitosis is an open question.*

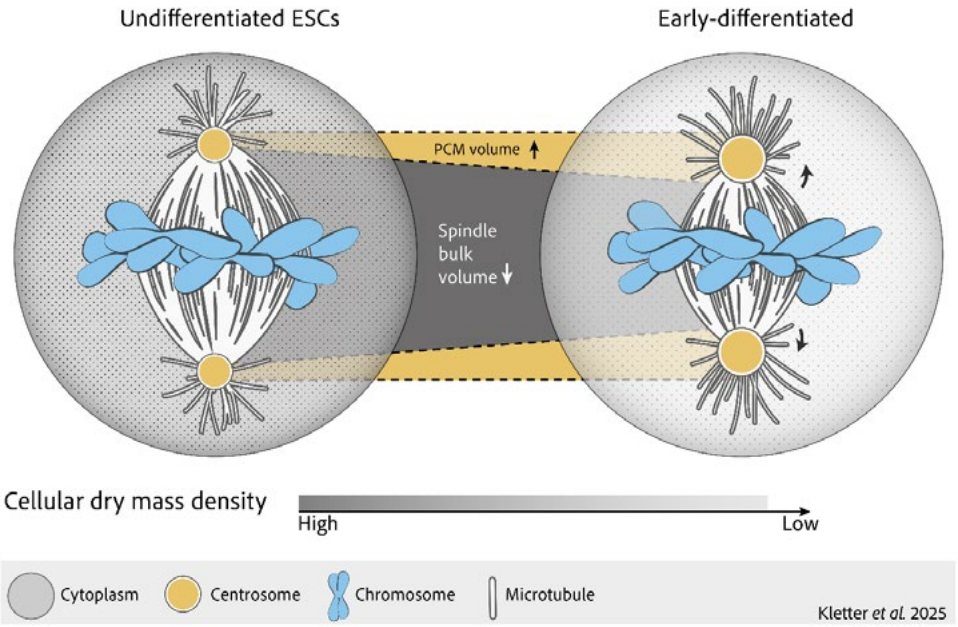

Figure 2: *A model for changes in mitotic architecture during early differentiation towards neural lineages. Dilution of the cytoplasm leads to enlarged centrosomes, redirecting microtubule growth towards the poles, while the size of the spindle bulk is decreasing.*

Walther Flemming Award 2025
IRGQ: Linking Autophagy to Immune Surveillance through MHC-I Quality Control

Lina Herhaus

Original publication: Herhaus L. et al., Cell 2024
Further reading: Gestal Mato, U., & Herhaus L., J. Cell Biochem. 2024

Introduction

Cells rely on intricate degradation systems to preserve protein homeostasis and defend against disease. Among these systems, autophagy acts as a major quality-control pathway that identifies, sequesters, and degrades misfolded or damaged cellular components via the lysosome. Beyond its canonical role in recycling cytoplasmic material, autophagy has recently emerged as a crucial modulator of the immune system, shaping antigen presentation and immune tolerance (1, 2).

In parallel, MHC class I (MHC-I) molecules are key gatekeepers of immune recognition. They present intracellular peptides to cytotoxic T lymphocytes, enabling the immune system to detect infected or transformed cells (2). While peptide loading in the endoplasmic reticulum (ER) and presentation at the plasma membrane are well described, how defective MHC-I molecules are cleared to prevent aberrant immune activation remained unknown.

In our recent Cell publication, we uncovered that Immunity-Related GTPase Q (IRGQ) serves as a previously uncharacterized autophagy receptor that directly links protein quality control to antigen presentation. This discovery reveals a novel pathway by which autophagy safeguards immune accuracy and tumor immune surveillance (3).

Discovery of IRGQ as a Selective Autophagy Receptor

Using quantitative proteomics and live-cell imaging, we identified IRGQ as a previously uncharacterized autophagy receptor that controls the turnover and surface expression of MHC-I. Further biochemical mapping revealed that IRGQ interacts with MHC-I heavy chains within the ER and recruits autophagy modifiers GABARAPL2 and LC3B. IRGQ contains two distinct LC3-interacting regions (LIRs) that enable dual engagement with the autophagy machinery, thereby promoting efficient delivery of misfolded MHC-I molecules to autophagosomes (Figure 1). This mechanism positions IRGQ as a molecular adaptor that ensures the fidelity of antigen presentation (3).

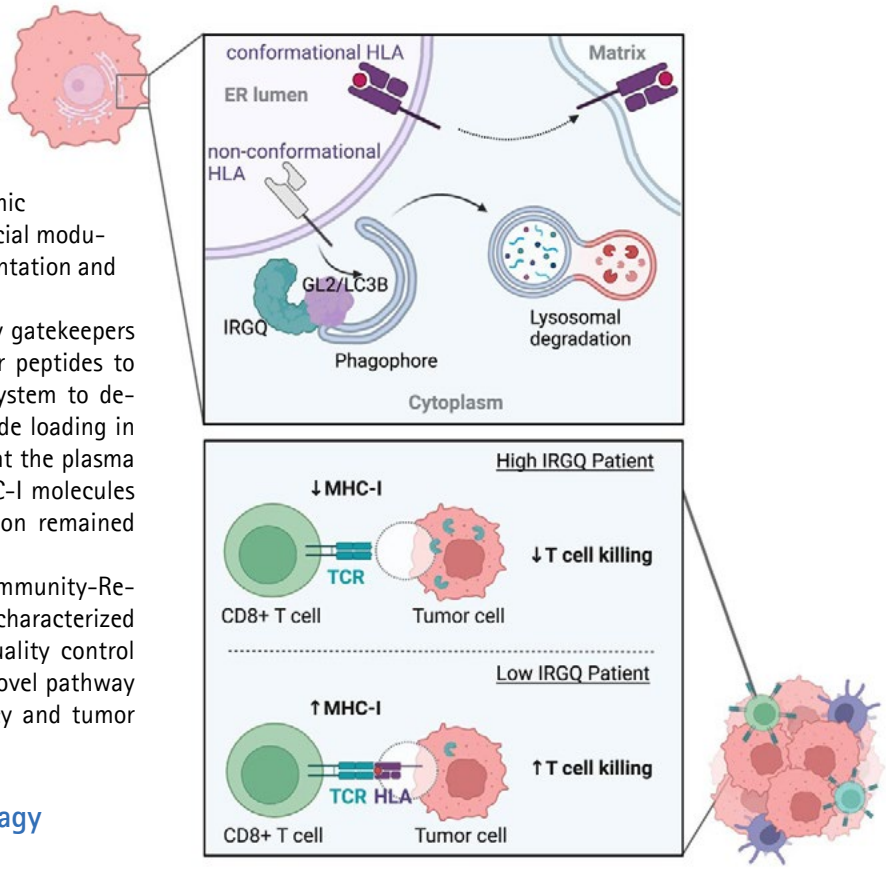

Figure 1: *IRGQ-mediated MHC-I quality control. Schematic: IRGQ recognizes misfolded MHC-I molecules in the ER, binds GABARAPL2/LC3B, and channels defective complexes into autophagic degradation, thereby maintaining surface antigen quality.*

MHC-I Quality Control and Immune Accuracy

The presentation of antigenic peptides by MHC-I molecules is a cornerstone of immune surveillance, allowing cytotoxic T lymphocytes to recognize and eliminate aberrant or infected cells. However, this system requires exceptional precision: only correctly folded and peptide-loaded MHC-I complexes should reach the cell surface. Misfolded or unstable MHC-I molecules risk presenting erroneous antigens, which could trigger inappropriate immune activation or autoimmunity.

Our work revealed that IRGQ enforces this crucial proofreading step by linking ER quality control to autophagic degradation. IRGQ recognizes misfolded or unassembled MHC-I heavy chains at the ER and directs them to lysosomal degradation through selective autophagy. By removing defective complexes, IRGQ ensures that only properly assembled MHC-I molecules are presented to $CD8^+$ T cells (3).

This quality-control mechanism positions IRGQ as an essential immune regulator, fine-tuning the balance between immune activation and tolerance. When IRGQ is absent or dysregulated, misfolded MHC-I molecules can escape elimination, modifying T-cell responses.

These discoveries highlight a previously unrecognized dimension of autophagy in adaptive immunity: not merely as a degradative process, but as a determinant of immune accuracy. By maintaining the structural integrity of MHC-I molecules, IRGQ upholds the fidelity of immune recognition and potentially prevents harmful or misdirected immune activation.

IRGQ and Immune Regulation in Hepatocellular Carcinoma

IRGQ functions as a quality-control receptor within the autophagy pathway and prevents the display of defective antigens and maintains immune fidelity. Through this selective degradation pathway, IRGQ acts as an essential immune regulator that calibrates antigen presentation and T-cell activation.

Our data reveal that IRGQ expression profoundly influences immune responses in hepatocellular carcinoma (HCC). Analyses of liver cancer patient samples and complementary mouse models demonstrated that high IRGQ levels, which enforce a rigorous MHC-I quality control, are associated with reduced T-cell activation and lower overall survival. In contrast, low IRGQ levels allow misfolded MHC-I molecules to escape degradation, thereby enhancing antigen presentation and triggering a robust cytotoxic T-cell response (3).

These findings establish IRGQ as a critical modulator of immune dynamics in cancer: its activity defines the balance between immune restraint and activation.

A Conceptual Advance in Autophagy and Immunity

The identification of IRGQ as an autophagy receptor introduces a new conceptual framework for understanding how autophagy shapes adaptive immunity. Traditionally regarded as a degradative pathway, autophagy now emerges as a precision mechanism that determines the quality of antigen presentation.

Our study bridges two previously distinct fields of cell biology: protein quality control at the endoplasmic reticulum and antigen processing for immune presentation. By functionally linking these processes, IRGQ ensures that immune recognition remains both sensitive and specific. It prevents the display of defective self-antigens while enabling robust cytotoxic responses against transformed cells.

This discovery underscores the role of selective autophagy as more than a cellular recycling system: it acts as a guardian of immune accuracy and a central regulator of antigenic integrity.

Outlook

Our laboratory continues to explore how IRGQ coordinates selective autophagy and immune regulation. Building on this discovery, my current research at the Helmholtz Centre for Infection Research (HZI) aims to extend these mechanistic insights from tumor biology to infectious disease. By leveraging our understanding of autophagy-driven quality control, we now seek to uncover how similar pathways influence immune recognition during host–pathogen interactions. This transition marks an exciting shift from cancer research to infection biology, linking fundamental cell biology to translational immunology.

Acknowledgement

I would like to express my heartfelt gratitude to Ivan Dikic, whose mentorship and scientific vision have profoundly shaped my career and approach to research. My sincere thanks go to Uxía Gestal Mato, an exceptionally dedicated PhD student whose commitment and creativity have been central to our discoveries. I am deeply grateful to all members of my former and current laboratory teams for their enthusiasm, perseverance, and collaborative spirit, as well as to our national and international collaborators for their invaluable expertise and inspiration. This work has been supported by the DFG Collaborative Research Center SFB 1177 "Molecular Characterization of Selective Autophagy" and now by the BMFTR-funded MicroStar Program. Receiving the Walther Flemming Award 2025 from the German Society for Cell Biology is a great honor and reflects our shared commitment to uncovering how fundamental cell-biological mechanisms underpin human health.

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Short CV
Education and Scientific Career

Education

02/2011 – 10/2014	PhD in Biochemistry and Cell Signalling, MRC University of Dundee, UK (Lab of Prof. Gopal Sapkota)
09/2007 – 06/2010	B.Sc. Molecular Biology and Applied Biology (Double Degree, Hochschule Bonn-Rhein-Sieg / University of Dundee)
1999 – 2006	Abitur, Gymnasium Alfeld, Germany

Scientific Positions

Since 10/2024	Group Leader, Immune Signaling – MicroStar Program, Helmholtz Centre for Infection Research (HZI)
05/2020 – 09/2024	Team Leader – Immune Signaling Group, Goethe University Frankfurt
06/2019 – 04/2020	Staff Scientist, Frankfurt Cancer Institute
12/2014 – 05/2019	Postdoctoral Researcher and EMBO Long-Term Fellow, Lab of Prof. Ivan Dikic, Frankfurt
Earlier research stays	ECOSUR (Mexico), Sappi-Alfeld GmbH (Germany)

Selected Awards

2025 Walther Flemming Award, DGZ
2024 Science Award, Signal Transduction Society (STS)
2024 Member of AIM Council of International Rising Stars
2023 Best Oral Presentation, University Cancer Conference Marburg
2014 Tim Hunt Prize for Outstanding Contributions to Cell Biology (University of Dundee)

Electric currents that shape life

Dr. Elias H Barriga

Mechanical forces generate endogenous bioelectrical fields that guide collective cell migration during development.

During embryogenesis, tissues are sculpted by the coordinated movement of cells that sense and respond to their physical and chemical surroundings. Morphogenesis depends not only on genetic programs and biochemical gradients but also on the dynamic exchange of mechanical and electrical information between cells. In our team we are interested in studying how these biophysical quantities emerge within tissues and how they are integrated into cells and translated into collective cellular processes such as migration and differentiation.

The case of endogenous electric fields. Every cell carries a transmembrane potential that generate local and long-range electric current patterns that support the formation of what has been named endogenous electric fields. These electric current patterns have therefore the potential to influence how cells grow, divide, and move within tissues. Bioelectric currents are well assumed in neurosciences, but less is known about the bioelectrics of non-neural tissues. This view is rapidly changing from the hand of technological advances which are enabling us to link bioelectrical signals to cellular and molecular frameworks of tissue morphogenesis.

Still, and despite increasing recognition of its importance, the origins and functions of endogenous electric fields in developing tissues have remained elusive. Electric currents have been observed in wounds, regenerating tissues, and cultured cells exposed to externally applied currents, but whether similar fields exist within intact embryos — and how they might interact with mechanical forces to control collective behavior — has been unclear. Theoretical work and in vitro experiments have suggested that membrane tension, ion channel activity, and tissue architecture could interact to generate spatially patterned electric current gradients. Nonetheless, direct experimental demonstrations of such intriguing mechanisms were missing. Understanding how these current patterns emerge and how cells integrate them into instructive signals is crucial for a comprehensive view of morphogenesis, where cells must integrate chemical, mechanical, and electrical information to move collectively and build complex structures (Reference 1 for more information).

In our recent study we explored how bioelectricity contributes to directed cell migration, for this we investigated the cranial neural crest of Xenopus laevis embryos, a population of highly migratory cells that move collectively to form craniofacial structures. This system offered an opportunity to observe collective migration in its natural context and to measure the electrical landscape along its native migratory path. By complementarily mapping extracellular currents and transepithelial voltages with high spatial resolution of embryonic tissues, we discovered that endogenous electric fields ranging from ~7 to 45 mV mm^{-1} are established along the migratory route of the neural crest cells.

When neural crest clusters were exposed to electric fields of matching magnitude and polarity in ex vivo assays, they migrated collectively toward the anode, mimicking their in vivo trajectory. Reversing the field reversed their direction, demonstrating that the electric field functions as a directional guidance cue. At the same time, applying physiologically relevant currents in parallel to the migratory path of neural crest cells, enhanced migration efficiency in vivo, whereas by applying the reversed currents disrupted the organization of migratory streams in frog embryos. **But are these current patterns actually relevant *in vivo*?** A key insight was that these fields are not imposed by biochemical asymmetries but are **generated by mechanical deformation** of the embryonic tissue itself. Our initial experiments suggest that these electric fields arise from patterned ionic fluxes, formed by outward currents at the neural fold and inward currents in the lateral ectoderm. In turn, these fluxes form a voltage gradient, which aligns with the migratory path of neural crest cells in vivo. Based in these initial observations, we lowered membrane tension and channel activity in the neural fold. These treatments were suffieicnt to deplete electric fields formation and led to a strong reduction of neural crest cell directionality in embryos, confirming the important instructive role of these electric fields for cell guidance *in vivo*.

Then, how can cells specifically transform these current patterns into directed cell migration? The **voltage-sensitive phosphatase Vsp1** emerged as a key molecule involved in electrosensing in the neural crest. When Vsp1 was depleted, cells retained motility but lost directional coherence. Moreover, externally applied electric fields could restore directed migration only when Vsp1 was functional, revealing that voltage-dependent signaling provides the molecular link between the physical cue and directional collective cell behaviours. This was an extremely interesting result, as there are many mechano- chemo- and electro-sensors, but their loss of function often leads to a depletion of cell motility, hence the relevance of Vsp1, which while not being required for motility was specifcally required by neural crest cells

to transform electric fields into directed migration. We are now working on up and downstream regulators and exploring its role in other tissues and biological contexts.

Our findings demonstrate that mechanical strain can give rise to endogenous electric fields that guide cell collectives through voltage-sensitive mechanisms. They establish a mechanistic continuum in which two tissues (neural fold and flanking ectoderm) mechanically interact to generate an electric field, which is then sensed and translated by molecular componenets (Vsp1) of a third tissue (the neural crest) into a collecgtive migratory response (Figure 1; Reference 2 for more details). Showing that **bioelectricity acts as both a product and a regulator of tissue morphogenesis** emphasises the idea that physical quantitites emerging from tissue intrerplay can act as pacers of the cellular events that drive tissue morphogenesis. More broadly, our work represents the first in vivo demonstration that bioelectrical cues generated by intrinsic tissue forces can organize large-scale cell movements during development, therefore bridging a long-standing conceptual gap between mechanobiology and electrophysiology during tissue morphogenesis.

In the long term, understanding the logic of these electromechanical circuits opens new avenues for guiding tissue assembly in regenerative medicine and bioengineering. If endogenous electric fields can instruct cells where to move and how to organize, then manipulating these invisible forces could one day allow us to pattern tissues or repair organs without relying solely on biochemical factors. The embryo, it seems, already knows how to use electricity to shape itself and learning its language could profoundly expand the toolkit of developmental and regenerative biology.

References

1. 2025 *Custodio Oliveira Nunes &* **Elias H Barriga***. *Bioelectricity in Morphogenesis.* **Annual Reviews in Cell and Dev Biol.** *10.1146/annurev-cellbio-101323-032747*

- Details about bioelectrics and hypothesis about electric field formationa and relevance in other contexts can be found

2. 2025 *Ferreira F, Moreira S, M Zhao,* **Elias H Barriga*.** *Stretch-induced endogenous electric fields drive directed collective cell migration in vivo.* **Nature Materials** *doi.org/10.1038/s41563-024-02060-2*

- Original article where all data is available.

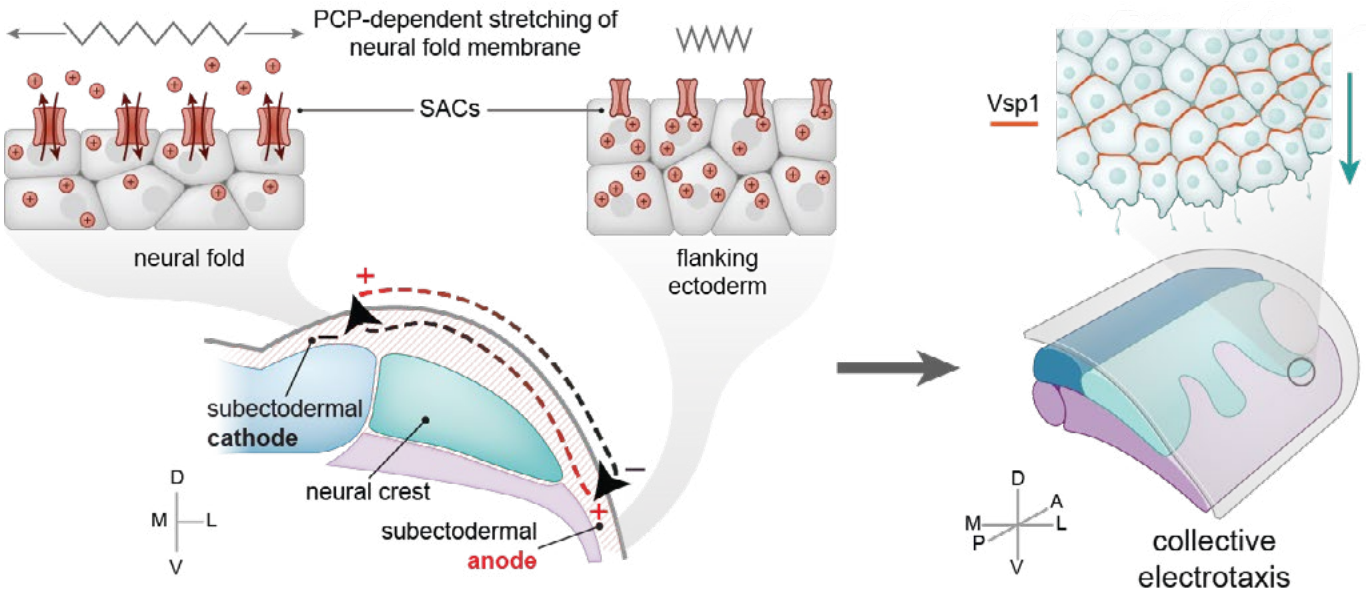

Figure 1: *Overview of stretch inducible electirc fields and electro-sensitive mechanism. Tissues and molecules are named accordingly (extracted from Ferreira et al 2025 Nature Materials).*

Dr. Elias H Barriga

Group Leader Excellence Cluster Physics of Life, Technische Universität Dresden, Germany

Identifiers/ORCID 0000-0003-2912-3613

https://physics-of-life.tu-dresden.de/team/pol-groups/barriga/people

Curriculum Vitae

Education and Career stages	Year	Details
Stages of academic/professional career	**Since Sept 2023**	Research group leader on W2 Tenure Track on Physical Measurements and Manipulation of Living Systems at the Cluster of Excellence Physics of Life, TU Dresden, Germany (**ERC Awardee** and **EMBO YIP**).
	2019–2023	Research group leader at Instituto Gulbenkian de Ciência (IGC), Portugal (**LaCaixa Fellow; EMBO Installation grantee**)
	2016–2018	Postdoctoral Researcher, University College London, United Kingdom (**Marie Curie Fellow**).
	2014–2016	Postdoctoral Researcher, University College London, United Kingdom (**EMBO Fellow**).
PhD Work	2010–2014	Molecular Biosciences (**Summa Cum laude**), Universidad Andres Bello, Santiago, Chile. (**CONICYT Fellow**).

Activities in the Research System	Year	Details
Professional Service (active only):	*Since 2025* *Since 2024* *Since 2024* *Since 2024* *Since 2021*	Head Aquatic Husbandry at PoL, Germany. *EMBO Policy Advisory Board Member, EU.* *Equal Opportunities Officer, PoL, Germany.* *Chair PoL Equal Opportunities Committee, Germany.* *Editorial board at Genesys.*

Funding and Recognition	Year	Details
Selected Awards and Prizes	*2025* *2022*	**Young Investigator Award** Xenopus International Board **Young Investigator Award**, European Molecular Biology organization.
Selected Grants (since the lab started only)	2025 2021 2021 2020 2020 2020	Wellcome Trust **(co-PI)**. ERC Starting Grant. 5-year 1.8 Mill EUR **(PI)** EMBO Installation Grant BBSRC **(Co-PI)** EUSMI Insfrastructure Grant La Caixa Foundation

Short Report | International Meeting of the DGZ, October 26-29, 2025, Heidelberg

The International Meeting of the DGZ took place from 26–29 October 2025 in Heidelberg, marking the 50th anniversary of the society's founding. Returning to its historical origins at Heidelberg, where the DGZ was established in 1975, the meeting brought together leading researchers from Germany and around the world.

Held at the German Cancer Research Center (DKFZ), the conference featured a four-day program of keynote lectures, focused scientific sessions, poster presentations, and networking opportunities. Under the anniversary motto "50 Years of Cell Biology: the heart of life sciences – past, present and future", the meeting highlighted both the legacy and the future directions of cell biology research.

The scientific program spanned a wide spectrum of modern cell biology, including cytoskeleton dynamics, membrane trafficking, nuclear organization, immunobiology, proteostasis, mechanobiology, and quantitative and biophysical approaches. Keynote speakers Stefan Hell, Tony Hyman, Maria Leptin, and Bernd Bukau delivered excellent talks related to super-resolution imaging, phase separation, European research funding, and co-translational protein folding and assemblies.

The DGZ awards underscore our strong commitment to promoting outstanding scientists at various stages of their careers. This year's award winners were honored at the conference. One of the highlights of the program was the 2025 Carl Zeiss Lecture by Julia Mahamid on the topic of "In-Cell Structural Biology." In addition, the following award winners presented their research: Bastian Hinkel and Silja F. Zedlitz (Master Thesis Awards, sponsored by DGZ), Tobias Kletter (Nikon Young Scientist Award 2025, sponsored by Nikon), Lina Herhaus (Walther Flemming Award 2025, sponsored by ibidi GmbH), and Elias H. Barriga (Innovation Prize 2025, sponsored by DGZ).

Moreover, The European Journal for Cell Biology (EJCB) sponsored two short-talk awards that went to Maximilian Schilling and Cornelia Sala. The Journal of Cell Science (JCS) sponsored three poster prizes, which were handed to Qi Gao with collaborator Florian W. Hofer, Sophia Borchert, and Jasmin Čić. Irina Levkovych and Franziska Dorn received the "Travel Green" award sponsored by The Company of Biologists to highlight attendees with the longest travel to the meeting by public transport.

Overall, the anniversary meeting showcased cutting-edge research, fostered exchange between generations of scientists, and emphasized the DGZ's role in shaping the future of cell biology in Germany and beyond. The thematic range was very broad, ranging from mechanobiology to in-cell structural analysis, reflecting the diversity of the field. By bringing together diverse subdisciplines (from imaging experts to theoretical cell biologists) the meeting laid the foundation for new collaborations and strengthened the exchange within the cell biology community.

The DGZ meeting 2025 was more than a celebration; it set the stage for the next era of cell biology in Germany and Europe, emphasizing innovation, interdisciplinarity, and strong community engagement.

We are very much looking forward to the next edition of the meeting, which is planned for 2027 at the University of Bonn.

The organizing committee (Gislene Pereira, Elmar Schiebel, Oliver Gruss, Zuzana Storchova, Sandra Iden, Roland Wedlich-Söldner, and all DGZ Workgroup Speakers).

Keynote lecturer and Nobel Laureate Stefan Hell

Keynote lecturer and ERC president Maria Leptin

Sandra Iden (left) with DGZ Master Thesis Awardees Silja Zedlitz (center) and Bastian Hinkel (right)

Julia Mahamid (center), recipient of the Carl Zeiss Lecture 2025, with Stefan Gross (CEO Carl Zeiss Microscopy Germany, left) and Sandra Iden (DGZ president, right)

Tobias Kletter (center) received the Nikon Young Scientist Award 2025 (right: Michael Glombik, Nikon Germany)

Lina Herhaus (center), recipient of the Walther Flemming Award 2025 with Sandra Iden (left) and Roman Zantl (CEO ibidi GmbH, right)

Amelia Glazier (JCS, right) and Oliver Gruss (DGZ, left) handed over the Travel Green Prize for Sustainability to Irina Levkovych and Franziska Dorn.

Short-talk awardee Cornelia Sala (left) with Klemens Rottner (EJCB, right)

Poster award winner Jasmin Čić with Oliver Gruss (left) and Amelia Glazier (right)

Short-talk awardee Maximilian Schilling (left) with Klemens Rottner (EJCB, right)

Attendees at the meeting

Poster award winner Qi Gao with collaborator Florian W. Hofer with Oliver Gruss (left) and Amelia Glazier (right)

All photos by Benedikt R. Dürr

In Memoriam: Günther Gerisch (1931-2025)

von Annette Müller-Taubenberger, LMU München

Günther Gerisch, who passed away on 26 June 2025, made a substantial impact on the field of cell biology for over six and a half decades. His research highlighted key cellular processes and set new methodological standards. He had been a member of the German Society of Cell Biology (DGZ) since 1982.

Born on 2 May 1931 in Dresden, Günther Gerisch studied biology in West Berlin, Göttingen and Tübingen. From 1958 to 1961 he conducted his doctoral thesis on Dictyostelium development at the Max Planck Institute in Tübingen, discovering conditions for precise initiation of cell development and becoming fascinated by cells moving in self-propagating waves and their mutual adhesiveness.

As assistant professor in Freiburg from 1961 onwards, he established Dictyostelium as a model system for cell aggregation, demonstrated changes in the glycoprotein composition during aggregation, developed immunochemical assays for isolating cell adhesion molecules, and produced together with the IWF spectacular movies documenting the development of Dictyostelium.

From 1969 onwards, first at the Friedrich Miescher Laboratory in Tübingen and later in Basel, he established two major research directions: immunological methods for identifying cell adhesion molecules that proved groundbreaking for higher eukaryotes, and the discovery that pulsatile cAMP secretion mediates the oscillatory relay system for cell aggregation. His innovative chemotaxis studies using micropipettes promoted Dictyostelium also as a model for mammalian cell chemotaxis.

In 1979, Günther Gerisch became director at the Max Planck Institute of Biochemistry in Martinsried, where he worked until his death. His department became an internationally recognized centre for research on cell adhesion and cytoskeletal functions. From the late 1980s, his research focused on the actin cytoskeleton. The advent of GFP technology in 1994 revolutionized the visualization of proteins and organelles in living cells and furthered numerous studies on intracellular dynamics in his research group.

After his 'official' retirement in 1999, Günther Gerisch headed the emeritus group 'Cell Dynamics' and continued working on intracellular structure visualization using TIRF microscopy, force spectroscopy and cryo-electron tomography. To utilise new technologies, he frequently collaborated with colleagues both within and outside the institute. During his last decade, he focused on chemotaxis, cytokinesis, pattern formation and actin waves.

Günther Gerisch published 300 scientific manuscripts between 1959 and 2024. His research was driven by curiosity and passion paired with precise observation and exceptional creativity. Those who met him in person recall him as quite shy, deeply passionate about science, and extremely meticulous. His scientific excellence inspired many researchers in the field of cell biology.

A more detailed tribute to Günther Gerisch's scientific achievements can be found at the following reference:

A life dedicated to science and research - a tribute to Günther Gerisch. Ecke M, Maniak M, Müller-Taubenberger A. J. Cell Sci. 2025 Nov 1;138(21):jcs264482. doi: 10.1242/jcs.264482.

Protokoll der Mitgliederversammlung 2025 der Deutschen Gesellschaft für Zellbiologie e.V.

Versammlungsleiterin und Protokollführerin: Prof. Dr. Sandra Iden, Präsidentin

Geschäftsführer: Prof. Dr. Jörg Höhfeld

Die Mitgliederversammlung fand am 26.11.2025, 12.00 Uhr bis 13.05 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 der Präsidentin mit anschließender Diskussion
3. Geschäfts- und Kassenbericht über das abgelaufene Kalenderjahr
4. Bericht der Rechnungsprüfenden
5. Entlastung des Vorstandes
6. Sonstiges

TOP 1. Bestätigung des Protokolls der letzten Sitzung

Das Protokoll der letzten Mitgliederversammlung 2024 (online über Zoom am 11.10.2024) war in unserem Online-Mitgliederjournal „Cell News", Ausgabe 1/2025, veröffentlicht worden und wird bestätigt.

TOP 2. Jahresbericht der Präsidentin

Sandra Iden berichtet über die Aktivitäten der DGZ in 2024 und 2025. In 2025 feierte die DGZ ihr 50-jähriges Bestehen (1975 in Heidelberg gegründet). Hierfür wurde ein für Medien zugänglicher Text verfasst (Publikation via Webseite). Zudem hat das Journal of Cell Science ein Interview mit Sandra Iden durchgeführt. Dies ist Teil einer Bestrebung des Journals mit verschiedenen nationalen wissenschaftlichen Gesellschaften im Bereich der Zellforschung enger zusammenzuarbeiten und diese besser international darzustellen.

Als wesentliche Veranstaltung des Jahres wurde das Internationale DGZ-Meeting Ende Oktober 2025 in Heidelberg durchgeführt. Etwa 260 Teilnehmende sowie Vertreter der Industrie, von Förderorganen und wissenschaftlichen Journalen traten für vier Tage in einen regen Austausch. In 45 Vorträgen und ca. 100 Posterbeiträgen teilten Nachwuchswissenschaftler*innen und etablierte Wissenschaftler*innen neueste Erkenntnisse aus der Zellbiologie und verwandten Disziplinen. Auch die diesjährigen DGZ-Preise wurden auf dem Internationalen DGZ-Meeting verliehen. Die Preisträger*innen für den seit 2024 verliehenen „DGZ Master Thesis Award" waren Silja Zedlitz und Bastian Hinkel. Zudem erhielten Dr. Tobias Kletter den Nikon Young Scientist Award, Dr. Lina Herhaus den Walther Flemming Award und Dr. Elias Barriga den Innovationspreis (in diesem Jahr finanziert durch die DGZ). Die Carl Zeiss Lecture wurde von Dr. Julia Mahamid gegeben. Vertreter der Sponsoren Nikon, ibidi und Carl Zeiss Microscopy waren zugegen und haben die Urkunden gemeinsam mit der Präsidentin in Person überreicht.

Als Publikationsorgan der DGZ wurde im Jahr 2025 eine Ausgabe der „Cell News" veröffentlicht, die zweite Ausgabe befindet sich zur Zeit der Mitgliederversammlung in Vorbereitung. Zudem wurden alle wichtigen Informationen über E-Mail und zusätzlich via BlueSky verbreitet.

Weiterhin berichtet Sandra Iden über den Stand der seit 2022 laufenden Focus Workshop-Serie und die DGZ-Webseite.

Zu den Mitgliederzahlen informiert sie, dass im Jahr 2023 eine Reduktion um 31 Mitglieder (33 Neuzugänge, 64 Austritte) und 2024 um 18 Mitglieder (23 Neuzugänge, 41 Austritte) verzeichnet werden musste. Zum Zeitpunkt der Mitgliederversammlung ergab sich erfreulicherweise für das laufende Jahr 2025 einen Anstieg um 26 Mitglieder (48 Neuzugänge, 22 Austritte). Die aktuelle Mitgliederzahl beträgt 680.

TOP 3. Geschäfts- und Kassenbericht über das abgelaufene Kalenderjahr

Der Geschäftsführer Jörg Höhfeld berichtet über die Finanzlage der DGZ im Geschäftsjahr 2024 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 31.12.2024 EUR 78.445,77.

BILANZ 2024

EINNAHMEN	EUR	AUSGABEN	EUR
Mitgliedsbeiträge (abzgl. Retouren)	45.640,00	Bankkosten	739,72
Spenden, Preisgelder	8.500,00	Retoure Mitgliedsbeiträge	440,00
Zinsen	190,16	Spenden, Preisgelder	13.500,00
Cell News, Homepage (Werbeanzeigen, Firmen-Links)	4.284,00	Cell News	1.035,30
		Tagungen	0,00
Tagungen	0,00	Reisekosten	0,00
Überträge	1.000,00	Bürokosten/Gehalt Sekr. Büromaterial, Homepage	33.664,05
Sonstige	647,88		
		Überträge	1.000,00
		Sonstige	4.838,38
Summe der Einnahmen:	60.216,84	Summe der Ausgaben:	55.271,45
Guthaben am 31.12.2023:	73.401,38	**Guthaben am 31.12.2024:**	**78.445,77**
Guthaben DGZ:	59.239,09	Guthaben DGZ:	65.430,53
Werner Risau Preis:	14.162,29	DGZ Master Thesis Awards:	13.015,24

Die Einnahmen und Ausgaben wurden von den beiden Rechnungsprüfern Fabian Erdel und Ralph Gräf geprüft und für richtig befunden.

TOP 4. Bericht der Rechnungsprüfer

Die Einnahmen und Ausgaben im Geschäftsjahr 2024 waren durch die Rechnungsprüfer Prof. Dr. Ralph Gräf und Dr. Fabian Erdel 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 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. Sonstiges

In offener Diskussion wurden Themen wie Mitgliedergewinnung, Adressierung des wissenschaftlichen Nachwuchses sowie Austausch mit VBIO diskutiert.

Prof. Dr. Sandra Iden
Präsidentin
Versammlungsleiterin
und Protokollführerin

Prof. Dr. Jörg Höhfeld
Geschäftsführer

DGZ Focus Workshops 2026-2027

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

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

January 27, 2026	–
February 24, 2026	Membrane Trafficking and EVs Francesca Bottanelli, Kerstin Menck
March 31, 2026	Cytoskeleton and Molecular Motors Franziska Lautenschläger, Anne Straube
April 28, 2026	Cell Polarity and Cell Migration Virginie Lecaudey, Markus Engstler
May 19, 2026	Cell Adhesion and Mechanobiology Andrew Clark, Ada Cavalcanti-Adam
June 30, 2026	Membrane Organization and Contact Sites Sabrina Büttner, Katja Zieske
July 21, 2026	Physics of the Cell Leonhard Möckl, Pierre Haas
August 2026	Summer break
September 29, 2026	Imaging for Cell Biology Helge Ewers, Stefan Pfeffer
October 27, 2026	Functional Organization of the Nucleus Zuzana Storchova, Cristina Cardoso
November 24, 2026	Cellular and Organismal Proteostasis Jörg Höhfeld, Thorsten Hoppe
December 2026	Christmas break
January 26, 2027	Cell Biology of the Immune System Eva Kiermaier, Pablo Saez
February 23, 2027	Mitosis and Meiosis Simone Reber, Thomas Mayer

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

Impressum

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