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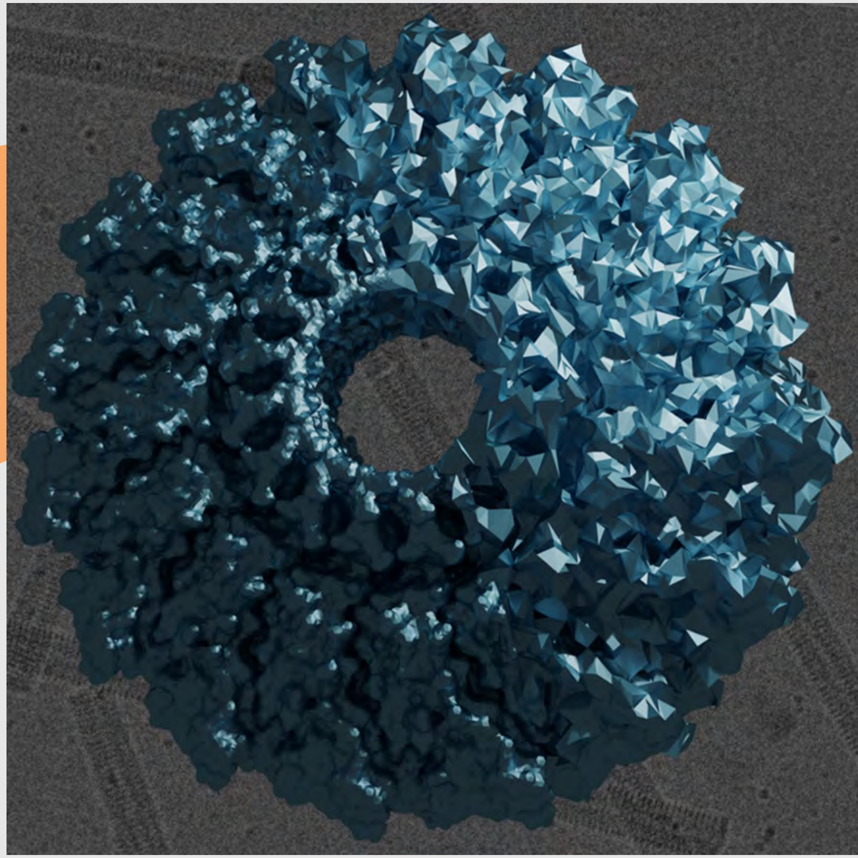

Rundschreiben 2020

Grußwort des 1. Vorsitzenden

Liebe Mitglieder der Deutschen Gesellschaft für Biophysik,

seit einem Jahr diene ich nun der Deutschen Gesellschaft für Biophysik als Vorsitzender und blicke auf eine Zeit zurück, die natürlich erheblich von der Corona-Pandemie geprägt ist. Veranstaltungen wurden zunächst verschoben, dann mussten sie schließlich vollständig abgesagt werden. Auch unsere Jahrestagung, die ursprünglich vom 20.-23. September 2020 in Konstanz stattfinden sollte, wurde auf September 2022 verlegt. Ich bedanke mich bei allen Beteiligten, die mit vollem Einsatz und großer Geduld und Frustrationstoleranz die Organisation vorangetrieben haben, insbesondere Karin Hauser und Andreas Zumbusch. Ich weiß, wie schwer ihnen die unvermeidliche Entscheidung zur Verschiebung gefallen ist. Ich freue mich schon jetzt auf die Tagung, bei der wir uns wieder persönlich und dreidimensional begegnen werden!

Noch kurz vor dem coronabedingten Lock-Down konnte sozusagen in letzter Minute das dreitägige DGfB-Membrane Biophysics Meeting mit knapp 60 Teilnehmern vom 2.-4. März 2020 in Drübeck stattfinden. Ich danke den federführenden Organisatoren Maria Hörnke und Peter Hildebrand für die ausgezeichnete Organisation und gratuliere auch den Gewinnern der ausgelobten Posterpreise nochmals sehr herzlich.

Auch den Mitgliedern des Vorstands gilt mein besonderer Dank für die Fortsetzung unserer ausgesprochen effizienten und kollegialen Zusammenarbeit in dieser besonderen Zeit, die uns so manches Mal besondere Kreativität und ein gewisses Improvisationstalent abverlangt hat.

So haben wir nach der Absage unserer Jahrestagung in Konstanz am 22. September 2020 kurzfristig ein eintägiges virtuelles Mini-Symposium mit einer Auswahl exzellenter Sprecher organisiert und durchgeführt, das recht gut virtuell besucht war.

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Grußwort des 1. Vorsitzenden

Obwohl eine solche virtuelle Veranstaltung natürlich kein persönliches Treffen ersetzen kann, haben sich doch spannende Diskussionen und Fragen ergeben. Ich denke, wir dürfen sagen: Wir haben das Beste aus der Situation gemacht. Diejenigen, die das Symposium verpasst haben, können sich die Videos der Vorträge übrigens nach wie vor über unsere Website unter „Aktuelles" herunterladen und ansehen.

Im Anschluss ans Symposium haben wir unsere virtuelle 44. Mitgliederversammlung abgehalten, die satzungsgemäß jährlich einzuberufen ist. Darin haben wir die Wahlen des Vorstandes, des Kassenführers und der Kassenprüfer durchgeführt. Ich freue mich sehr über das ausgesprochene Vertrauen, das sich in der Wiederwahl der Vorstandsmitglieder ausdrückt.

Besonders bedanken möchte ich mich bei unserem Sekretär Thomas Gutsmann und unserem Kassenführer Sandro Keller, der im Amt bestätigt wurde, wie auch bei den beiden stellvertretenden Vorständen Klaus Gerwert und Claudia Steinem, die sich beide auch weiterhin für dieses Amt zur Verfügung gestellt haben. Annette Meister und Daniel Huster danke ich dafür, dass sie die Kassenprüfung übernehmen.

Ich bedanke mich auch bei allen Mitgliedern die im Laufe des vergangenen Jahres mit Ihren Beiträgen und Anregungen dazu beigetragen haben, die Biophysik in Deutschland zu fördern und weiter auszubauen, und hoffe weiterhin auf Ihre tatkräftige Unterstützung. Schicken Sie mir gerne Ihre Ideen für Veranstaltungen, Aktionen oder Mitteilungen für die Website der DGfB und helfen Sie mit, die Sichtbarkeit unsere Gesellschaft weiter zu erhöhen!

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Grußwort des 1. Vorsitzenden

Wir freuen uns, dass die DGfB neuerdings auch im Serviceportal des ifb, dem Institut für politikwissenschaftliche Forschung und Beratung, in der Übersicht wissenschaftlicher Fachvereinigungen in Deutschland aufgeführt wird.

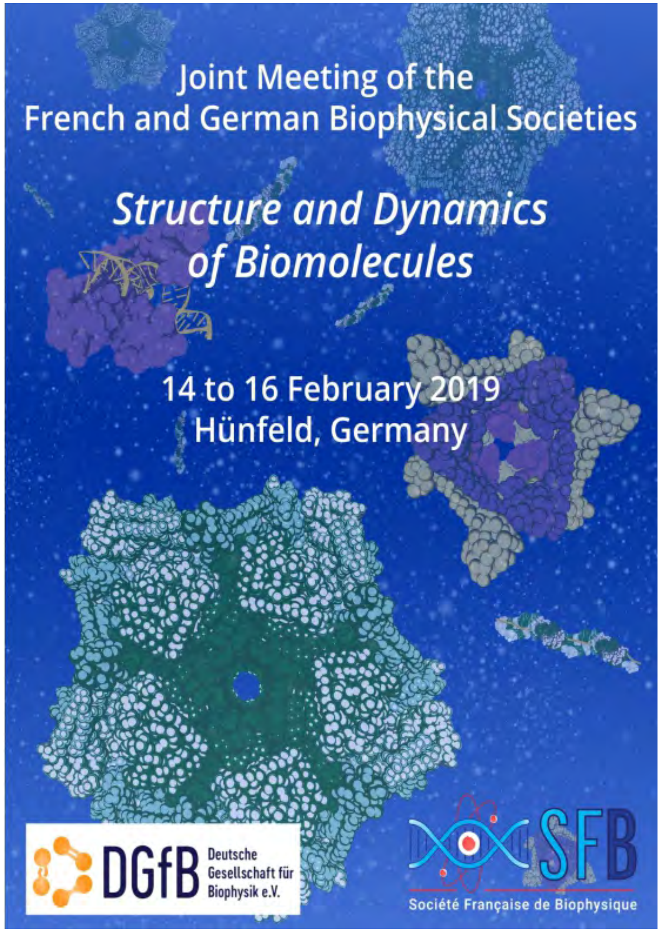
https://www.institut-politik.de/de/serviceportal/fachvereinigungen#dgfb-logo-2

Ich wünsche allen Mitgliedern ein frohes und besinnliches Weihnachtsfest, Gesundheit, und ein gutes und erfolgreiches Jahr 2021!

Helmut Grubmüller

Deutsche Gesellschaft für Biophysik e.V.

Sektion 1
Molekulare Biophysik



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Sessions
- Cell walls and membrane remodelling
- Membrane reorganization
- Nucleus und Nucleoid Organization
- Nucleic Acids
- Protein folding and IDPs
- Enzyme Mechanisms
- Membrane proteins
- New technical Developments
- Membrane Complexes and Proteins

- 21 invited speakers
- 17 selected talks
- 24 poster flash talks

Molecular Biophysics Section Meeting
Zoom, ca. 18.-20.3.2021

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Sektion 2
Membranbiophysik

DGfB - Membrane Biophysics Meeting Drübeck 2020

By our biannual tradition, in early March 2020, 70 Biophysicists from Germany and all over the world met for the Membrane Biophysics Meeting in the Monastery of Drübeck. For many of us, this was the last scientific meeting that took place in person and without keeping distances. For some of us, it also was the first meeting at all, as again, we could welcome a fair number of first-timers. Gladly, our invited speakers and participants from Europe and 3 other continents could join and return safely.

Main Sponsor:

JOACHIM HERZ STIFTUNG

Phospholipid Forschungszentrum / Research Center Heidelberg

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EBSA Biophysics in Europe

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Sektion 2 Membranbiophysik

As ever, the participants filled the meeting with life: excellent scientific talks and discussions, intense poster presentations, collaboration kick-offs at the dinner table, debates while walking in the forest, and some self-made live music.

The meeting covered a wide variety of Membrane Biophysics from simple models to biological complexity, lipid behavior to highly complex interplay of membrane proteins, covering computational, spectroscopic, thermodynamic, microscopic, and structural methods.

A round of poster-flash presentations inaugurated the lively poster session. We congratulate the winners of the jury-selected poster prizes: Jana Brehmer, Maria Sarmento, and Andra Schromm.

We gratefully acknowledge extensive administrative work by Jessika Schlögl, University of Leipzig. Support by Joachim Herz Stiftung, DGfB, Phospholipid Research Centre, and EBSA is acknowledged, too.

Let's hope that conferences with all their opportunities for interactions will soon be possible again.

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Sektion 3
Zelluläre Biophysik

Sponsored by:

IRsweep Innovation in sensing

SFB 1078 Protonation Dynamics in Protein Function

DFG Deutsche Forschungsgemeinschaft German Research Foundation

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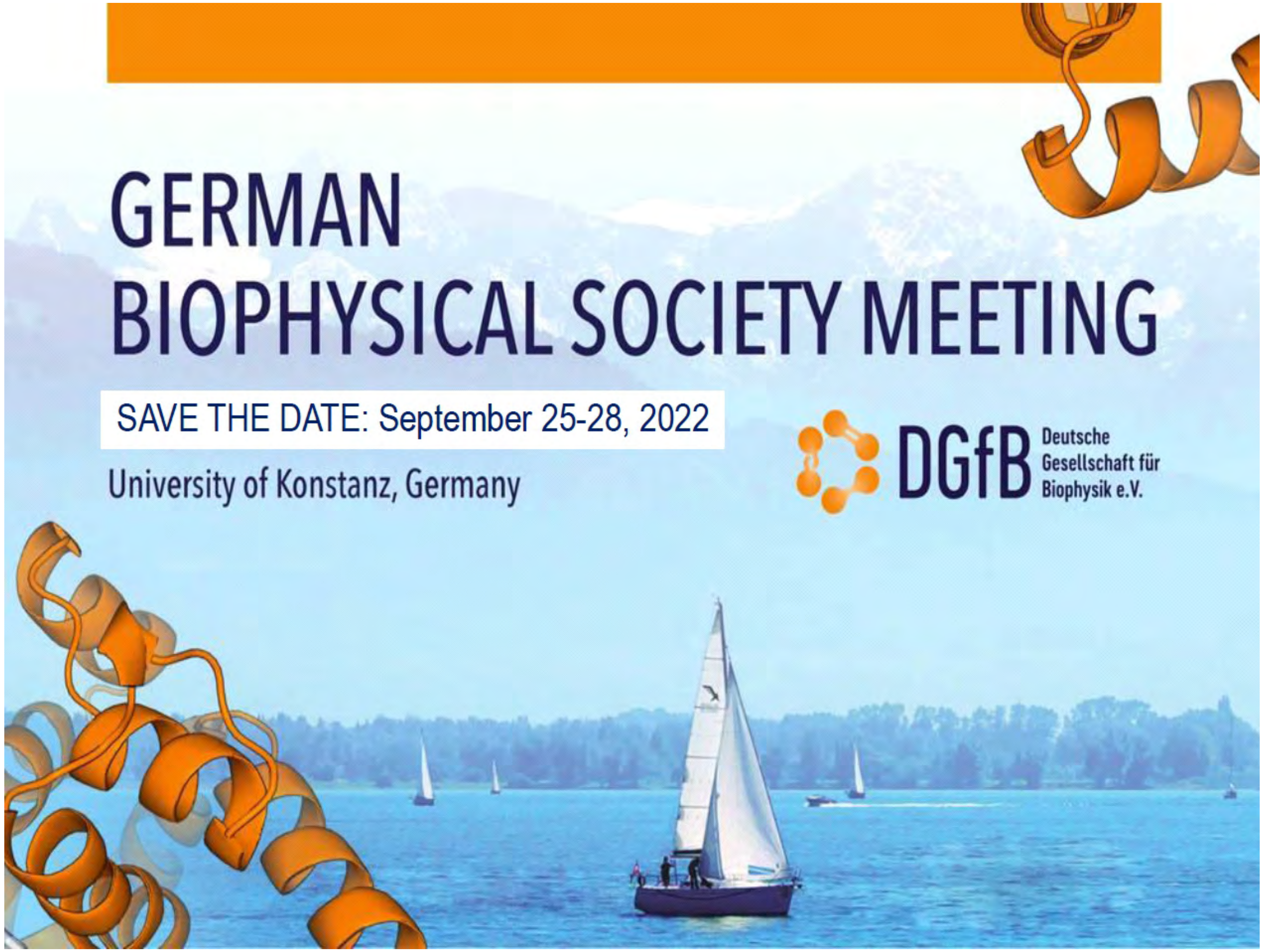
Jahrestagung der DGfB in Konstanz

SAVE-THE-DATE

September 25-28, 2022



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Kurzprofil der AG Ingrid Tessmer

www.virchow-zentrum.uni-wuerzburg.de/tessmerlab

Short profile of the Tessmer group, University of Würzburg

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   Single molecule studies of DNA lesion search and recognition in DNA repair

2. **Head of group**

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3. **Summary:**

   DNA repair is essential for the maintenance of genomic stability and cellular viability. We use single molecule imaging techniques to resolve the remarkably different strategies realised by different DNA repair systems to find and recognise their target sites in DNA, and to understand the interplay between different DNA processing systems.

The group (plus extended family) on the Christmas Market in pre-Corona 2019.

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DNA damage

DNA is damaged continuously by agents that occur naturally within our cells as well as by exogenous factors such as high-energy radiation or alkylating agents during chemotherapy. If unrepaired, the resulting DNA base modifications and strand breaks can lead to mutations in transcribed genes and tumour development as well as cell death.

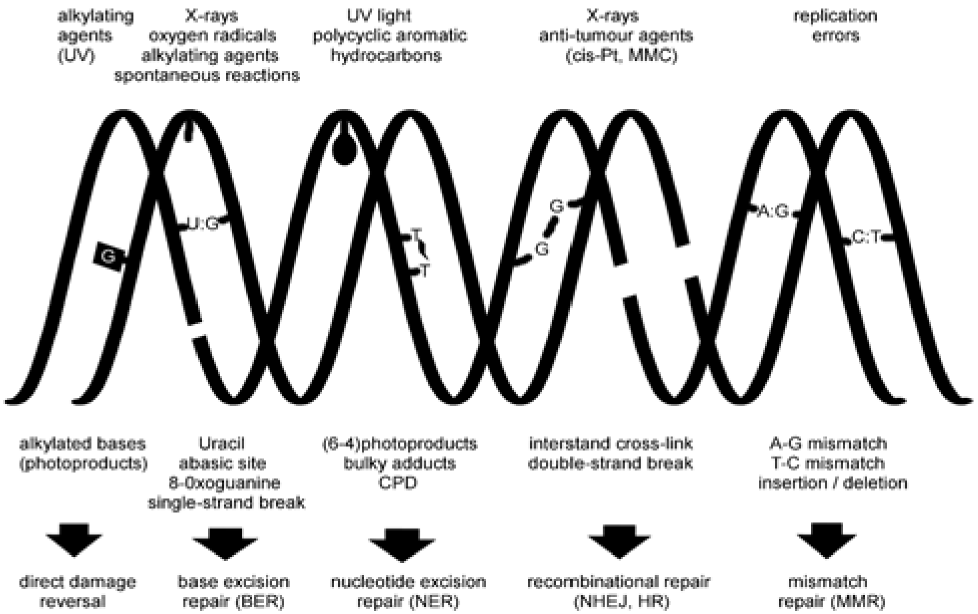

Figure 1: DNA lesion sources, types of DNA damages, and repair mechanisms.

DNA repair

Cells have hence developed a variety of DNA repair mechanisms, which are each specialised to target specific damages in the genetic code (Figure 1). The approaches to finding, identifying, and removing their target lesions differ between these mechanisms to cater for the specific needs of the lesion type.

DNA strand breaks are maximally disruptive for replication and transcription processes and are thus highly cytotoxic. These lesions are repaired by the non-homologous end joining and homologous recombination protein machineries. Subtle chemical alterations of DNA bases such as oxidation or alkylation can cause transition mutations in the DNA and thus lead to genetic instability. These lesions are typically targeted and repaired by the base excision repair (BER).

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In humans, highly mutagenic and cytotoxic base alkylation is also the only type of DNA lesion that is repaired by the most non-invasive, direct damage reversal mechanism. Bulky and strongly distorting lesions in the DNA are targets of the nucleotide excision repair (NER) pathway. We are interested in the different strategies of DNA damage search and recognition in different DNA repair pathways (Figure 2).

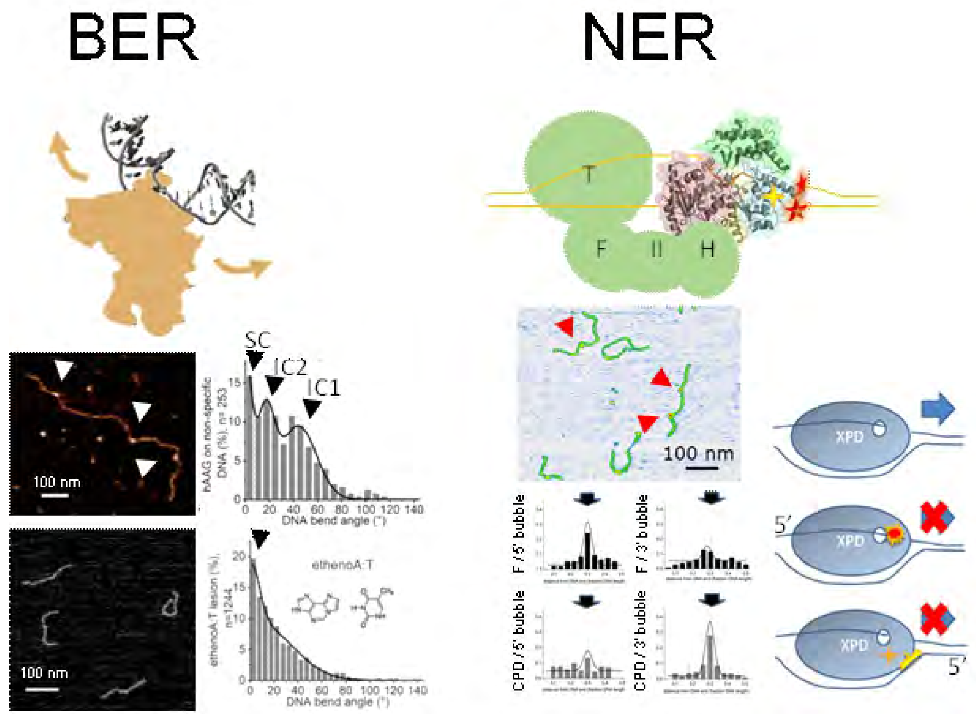

Figure 2: DNA lesion search and recognition strategies in different DNA repair mechanisms. Left: Automated AFM image analyses support an adaptation of BER glycosylases for the specific mechanical properties of their lesions (comparable DNA bending in search complex (SC) of e.g. here hAAG glycosylase (top image and plot, IC is interrogation complex conformation) and at the lesion in the absence of protein (bottom image and plot). Arrows in image indicate DNA bound proteins. Glycosylases may thus exploit the subtle energetic differences in DNA bending at a target lesion or on non-specific DNA as an initial detection criterion as they scan the DNA in their lesion search. [1,2] Right: In a bottom-up approach, we investigated lesion recognition by the isolated NER helicase XPD (in complex with its activating co-factor p44) that is part of the transcription factor IIH (TFIIH) complex. Based on the 5'-to-3' polarity of XPD and the incorporation of a loading site for XPD/p44 (a DNA bubble) either 3' or 5' to a DNA lesion, our AFM protein positional analyses allowed us to distinguish between lesion recognition by XPD on the translocated or on the opposite, non-translocated strand (arrows in plots indicate lesion position in DNA). Interestingly, the strand preference is dependent on the type of lesion, a bulky fluorescein (F) adduct or a cyclobutane pyrimidine dimer (CPD) UV-lesion. Its close proximity to the lesion in the non-translocated strand implicates an iron sulphur cluster in the enzyme (yellow star) in lesion recognition. [3,4]

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IInteractions between different DNA repair mechanisms

In vivo, many proteins function within different pathways, and different pathways are interlinked in a complex manner. Using AFM and fluorescence optical tweezers measurements we resolved one such link between different DNA repair pathways (Figure 3). Our research aims at further investigating interactions between different DNA repair pathways as well as between DNA repair and other DNA processing mechanisms such as DNA replication and transcription.

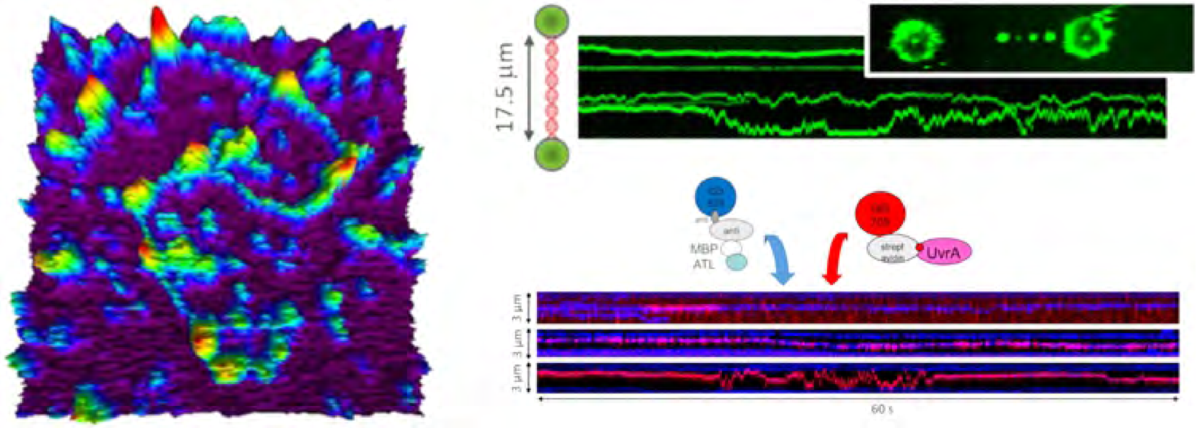

Figure 3: Alkyl-lesion repair by alkyltransferases and by alkyltransferase-like protein induced NER. The direct damage reversal DNA alkyltransferase (AGT) forms clusters on the DNA during lesion search, as visualised by single molecule AFM imaging (left). [5,6] The alkyltransferase-like protein (ATL) is structurally similar to AGT and also forms oligomeric complexes on DNA that scan the DNA rapidly during lesion search, as seen in the green fluorescent kymographs on the right. The inset shows the two optically trapped beads with the long DNA tether extended between them (non-labelled) and the individual, fluorescently labelled ATL protein clusters on the DNA. [7] Mixed samples of ATL and UvrA labelled with different colour quantum dots demonstrate that ATL can recruit UvrA, the initiating enzyme of prokaryotic NER, to DNA and transport it towards DNA lesions to trigger recognition and repair of alkyl lesions by NER. [7]

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Kurzprofil
der
AG Radek Šachl

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Fluorescence spectroscopy and microscopy in biology

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3. **Focus of the team**
The group represents a multidisciplinary team with a balanced expertise in chemistry, physics and biology that aims at understanding biologically relevant processes at the molecular level. Our primary interest is in the processes taking place on plasma or mitochondrial membranes and model systems derived from them, including vesicles and supported phospholipid bilayers. As a team with roots in the development of fluorescence methods, we mainly use fluorescence spectroscopy and microscopy methods, further develop these methods and combine them with complementary theoretical and molecular dynamics-based approaches.

3.1 The key experimental methods and instruments used by the team are:
- Time-resolved fluorescence spectroscopy and Fluorescence Lifetime Imaging (FLIM)
- MC-FRET with a nanoscopic resolution in all three dimensions; used in characterization of membrane nanodomains
- Single molecule Fluorescence Correlation and Cross-Correlation Spectroscopy (FCS and FCCS)
- imaging FCS
- Single molecule localization spectroscopy

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3.2 Current activities of the group

In-membrane oligomerization of proteins into functional units

In-membrane oligomerization is decisive for the function (or dysfunction) of many proteins. Advances in single molecule and super-resolution fluorescence microscopy opened new possibilities to accurately determine in-membrane protein oligomerization numbers. However, this often leads to a situation where these methods reveal broad distributions of oligomerization numbers for individually investigated membrane proteins where only some of the oligomers may be functional. A question arises how one can identify functionally relevant oligomerization and distinguish it from unspecific aggregation. In our group we are trying to design quantitative approaches that could distinguish between functional and nonfunctional protein oligomers.
For instance, in (Šachl et al 2020) we introduce a single molecule single vesicle statistical approach that enables to determine the average oligomer size of a protein on a vesicle and correlate it to membrane pore formation having a biological function.

Membrane nanoscale organization

Lipid membranes can spontaneously organize their components into nanodomains of different sizes and properties. The organization of membrane lipids into nanodomains may be important for various vital functions of cells and organisms. Under this umbrella of research, we are interested in:

- The role of ganglioside organization in ganglioside-protein interactions related to neurodegenerative diseases (Amaro et al 2016; Sarmento et al 2020)
- Development of new approaches that enable more efficient detection and characterization of lipid nanodomains (Koukalová et al 2017; Vinklárek et al 2019)
- The role of inter-leaflet coupling and membrane asymmetry in nanoscopic organization of the lipid bilayer (Sarmento et al 2020; Vinklárek et al 2019).

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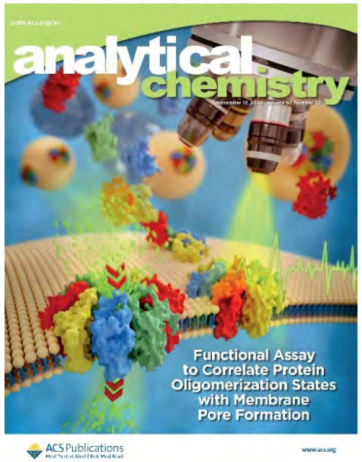

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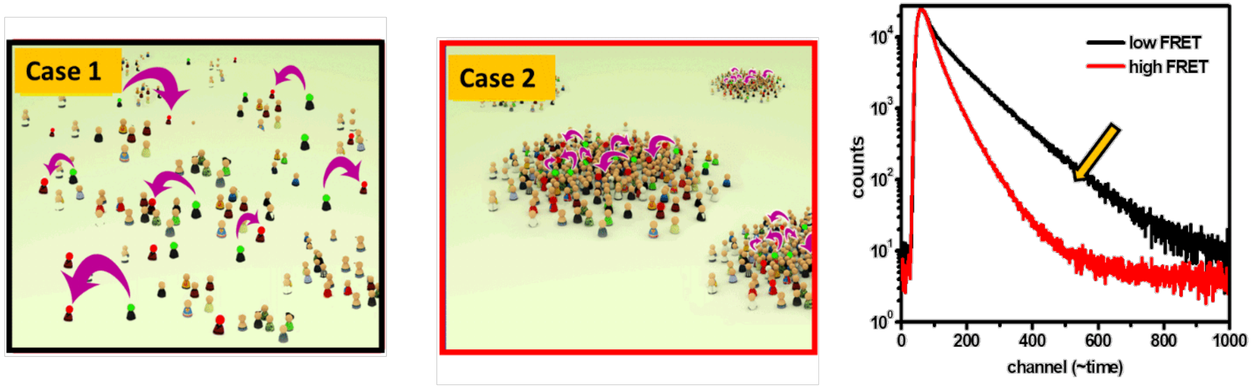
MC-FRET approach



(Case 1): Homogeneous distribution of lipids in the lipid bilayer. **FRET is intermediate. (Case 2):** Accumulation of lipids into the nanodomains. **FRET increases.** A sophisticated analysis of FRET (called MC-FRET) gives information about **the size, concentration and inter-leaflet organization of the nanodomains.**

Oxidative stress as a modulator of cellular apoptotic activity

The mitochondrial apoptotic pathway is usually initiated in response to oxidative stress which generates oxidized phospholipids as a result of irreversible oxidation of unsaturated fatty acid side chains. These modified lipids have been linked to the onset of apoptosis as well as to a wide range of diseases. In our group, we are trying to understand molecular consequences of lipid oxidation for the activity of both pro- and anti-apoptotic proteins regulating the onset of cell death.
For instance, we have shown that the presence of oxidized phospholipids is an important accelerating factor that stimulates the translocation of a pro-apoptotic Bax to the mitochondrial outer membrane and its subsequent formation of membrane-penetrating pores (Dingeldein et al 2017). A very limited understanding of the relation between the structural and functional organization of the mitochondrial membrane under oxidative stress is a clear fact in this field.

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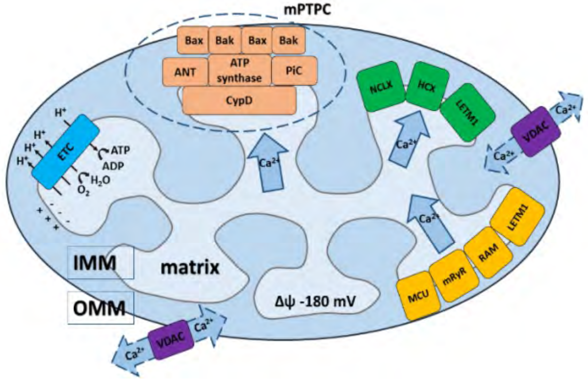

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Kurzprofil der AG Boris Musset

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Short profile of the Department of Physiology, Paracelsus Medical University in Nuremberg

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3. Summary:

Immune cells are part of nearly every defence against illnesses in the human body. We investigate the ion channels and ion transporters of these essential cells. Here, the voltage-gated proton channel HV1 is a key protein. Our research integrates electrophysiology, structure biology, and immunology.

Group picture of the Department of Physiology PMU Nuremberg:

Group members in a more relaxed environment (Fränkische Schweiz).
From left to right:
Christian Derst, Niklas Ohlwein, Boris Musset, Christophe Jardin, Gustavo Chaves, Patricia Romberg, Eva Schrader, Iryna Mahorivska and Andrea Musset.

Kurzprofil der AG Boris Musset

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3.1 Phagocytosis and respiratory burst:

The innate immune system prevents most of the infections we are confronted with before they inflict harm. We research the biophysics of immune cells and focus on ion channels and ion transporters. Ion channels play a decisive role in immune cell maturation, chemotaxis, reactive oxygen release, production of cytokines- and volume regulation, just to mention a few. One of the most important ion channels is the voltage-gated proton channel H_V1 . This channel is expressed in all phagocytes. Phagocytes are the main defence against bacterial, parasite and fungal attacks. “Phago” stands for eater and “cyt” in biology is a suffix for cell. Phagocytes are “eating cells” or “scavenger cells” devouring e.g. bacteria. The phagocyte encloses the bacterium with plasma membrane and takes the bacterium up into its cell body. This is called phagocytosis. The intracellular vesicle is called phagosome, by fusion with lysosomes a phagolysosome is created in this enclosed space (0.2-1 fl), destroying the bacterium (Figure 1).

One of the tasks of H_V1 is to prevent a high potential difference across the membrane of the phagocytes. This change in potential in positive direction is called depolarization. In phagocytes, this is due to the activity of the nicotinamide adenine dinucleotide phosphate oxidase (NADPH oxidase). This enzyme oxidises NADPH by taking electrons from NADPH and translocating them across the plasma membrane or phagosome.

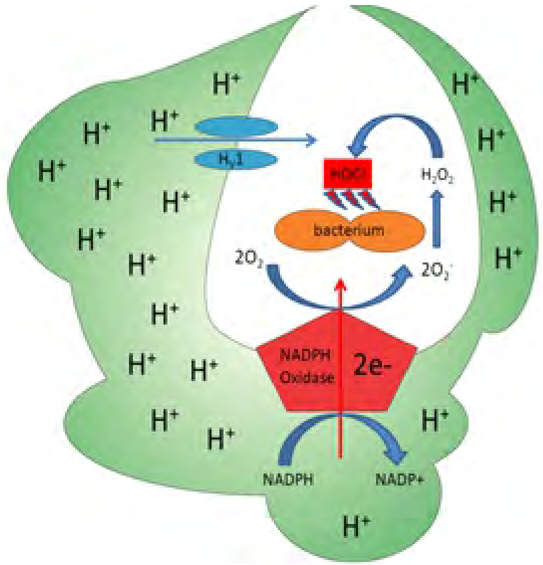

Figure 1:
Schema of the respiratory burst during phagocytosis. The phagocyte is engulfing the bacterium and the respiratory burst starts by generating electron current across the plasma membrane (NADPH oxidase, red diamond). Superoxide (O2-) is converted into hydrogen peroxide (H2O2) and enzymatically hypochlorous acid (HOCl) is generated. Charge is balanced by conducting protons (H+) via HV1 (blue ovals) to the phagosome. The internal pH drops by releasing protons from NADPH. The cytosolic pH is regulated by HV1 conducting protons out of the cell.

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Molecular oxygen (O_2) is reduced and takes up the electrons. Thus, two molecules of O_2- are formed for each molecule of NADPH. O_2^- is an oxygen-radical that will transform in the phagosome from hydrogen peroxide (H_2O_2) to hypochlorous acid (HOCl). Hypochlorous acid is a strong bacteriocide. Together with the lysosomal enzymes, HOCl is responsible for the destruction of bacteria. Oxidation/Reduction processes are connected to a redox potential. The redox potential describes the voltage that would be generated or consumed during the redox process. The redox potential of NADPH/NADP$^+$ is -320 mV. The redox potential of O_2/O_2^- is -160 mV. Resulting from these numbers, the so-called redox potential of the NADPH oxidase is around +160 mV. This value is additionally the expected quasi reversal potential of
pure electron conductance through the phagocyte membrane. The reversal potential is the value where the current changes its direction.

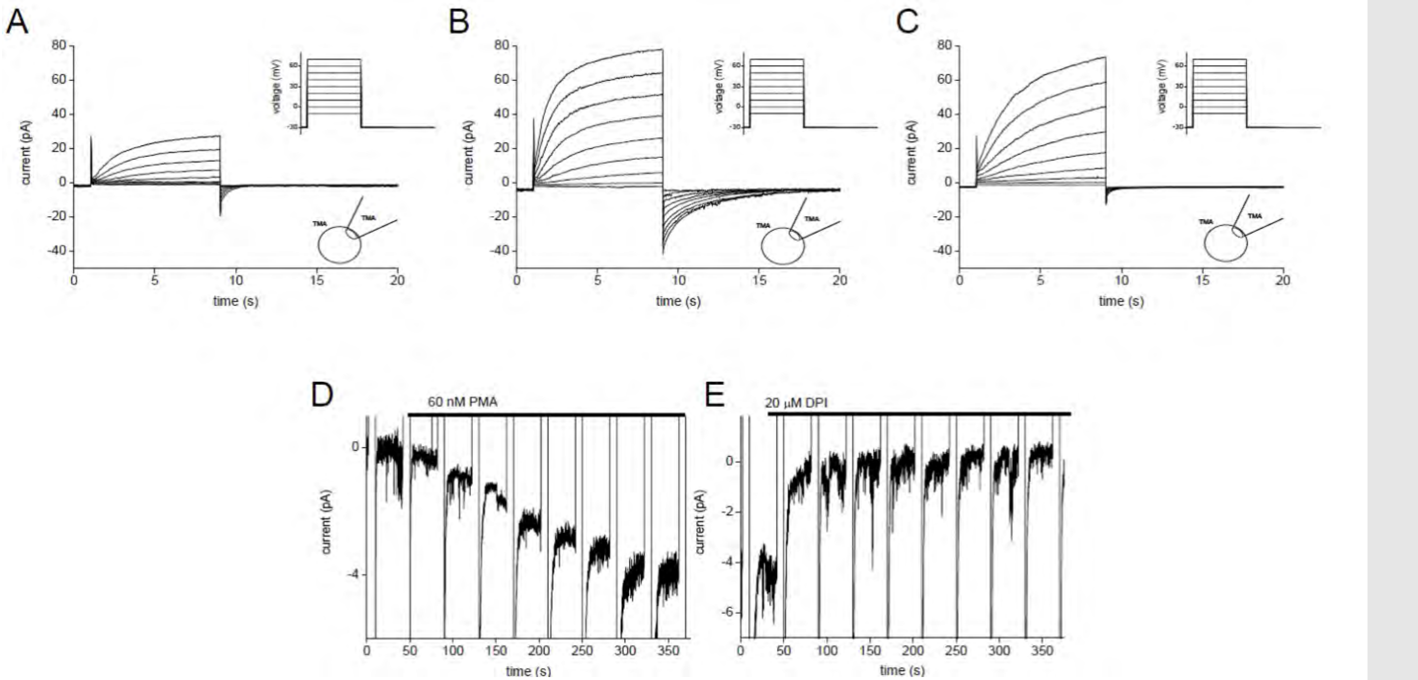

Figure 2: Proton and electron current in a human phagocyte
A) Proton current under control conditions. B) Proton currents after activation by the phorbol ester PMA. C) Proton currents after electron current inhibition by diphenyliodonium (DPI). D) Electron current activates over time. E) Electron current deactivated by DPI.

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H_V1 opens due to depolarization and selectively conducts protons (H^+) out of the cytosol. The driving force for the proton conduction is the electrochemical gradient. The electro-chemical gradient for protons is dependent on the activity of H^+ inside and outside the cell. Furthermore it depends of the electrical potential across the membrane. Under resting conditions (pH 0.1 units lower in cytosol than outside (blood)), the chemical potential will drive protons out of the cell. However, the electrical potential (cell-dependent in phagocytes around -60 mV) will hold the protons back. During the activity of the NADPH oxidase vast amounts of H^+ are generated due to the oxidation of NADPH. Additional amounts of protons are generated by the pentose phosphate pathway needed to generate and replenish NADPH. The chemical gradient would force the H^+ out of the cell. However, the electrical force is increased too. While electrons cross the focus of the electric field of the membrane, the membrane is charged, leading to a massive depolarization in the direction of +160 mV. The electrical force will drive the H^+ out of the cell too. An important detail left apart is that H_V1 is voltage-gated. H_V1 opens exclusively with depolarization and is closed as long as the electrical force is negative. Still, this voltage-dependence is dependent on the pH inside and outside the cell. The voltage-gated proton channel is able to adjust its voltage-dependent gating in a way, that it works comparable to an over pressure valve, and exclusively conducts H^+ outward. Therefore, H_V1 is the perfect partner for the NADPH oxidase, as while it is open, it conducts H^+ outside the cell preventing an acidification of the cytosol (Figure 2). While it is open, the permeation of protons will balance the charge of the moving electrons and the membrane potential will be less positive. One more detail to mention is that the electron current of a phagocyte is voltage dependent. Depolarizations that will reach the quasi reversal potential of the NADPH oxidase will cease its function. Finally, NADPH oxidase is pH dependent. The electron current decreases drastically in low pH.

3.2 Structure and Function of H_V1

We are investigating the structure and function interdependence of voltage-gated proton channels. The human H_V1 is a homodimer. Each of its monomers consists of 273 amino acids (Figure 3). Each monomer has 4 transmembrane domains, both N- and C- termini are cytosolic. Comparable to the classical voltage-gated ion channels, the fourth transmembrane domain has several positively charged arginines in an α-helix. This structure is also known as the voltage sensor.

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The mechanic of the voltage sensor is imagined as being a ratchet that moves one arginine after another through the membrane. This movement is still under scientific discussion but its goal would be to open a pore enabling ionic current to be conducted.

In contrast to the classical voltage-gated ion channels H_V1 does not have a classical pore. In potassium channels, the selectivity of the pore is achieved by a pore loop Glycine-Tyrosine-Glycine (GYG) motive that forms the selectivity filter. Dehydration of the ion, charge, and ion size are the parameters of selectivity. In sodium channels a net negative charge ensures selectivity while in calcium channels higher net charges are necessary to provide selectivity. Anyhow, size of the pore is an essential criterion of selection in all the selectivity filters. However, how is the selectivity for H^+ realized if size exclusion might be challenging?

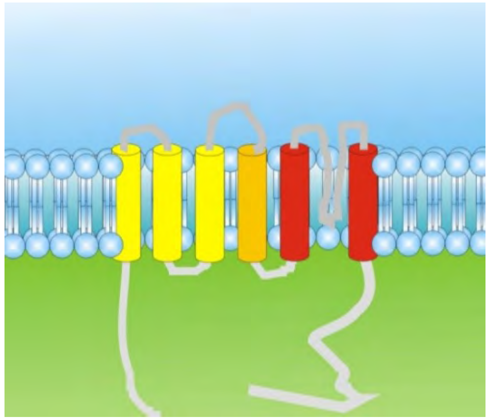
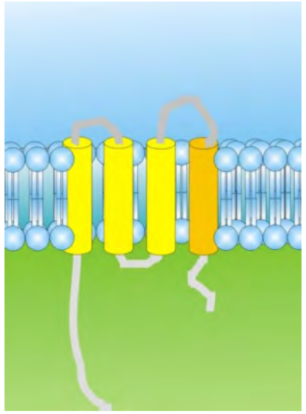


Figure 3:
Transmembrane domains of a classical voltage-gated potassium channel (left) vs. the voltage-gated proton channel HV1 (right). S1-S3 in yellow, S4 containing arginine in orange, pore domain S5-S6 in red. HV1 is missing the pore domains.



Comparison to the closest non-conducting H_V1 homolog suggested an aspartate as the selectivity filter (Figure 4) (2). Mechanistically, the aspartate is protonated and deprotonated during conduction, while without proton the opposing arginine closes the salt bridge (3,4).

We investigate the architecture of H_V1 by using zinc (Zn^{2+}) as an inhibitor of proton current (5-7). The electrophysiological data combined with molecular dynamics simulations present a funnel-like corridor in the dimer. The funnel is built by a negative electrostatic potential that guides Zn^{2+} to its binding histidines between the homo monomers.

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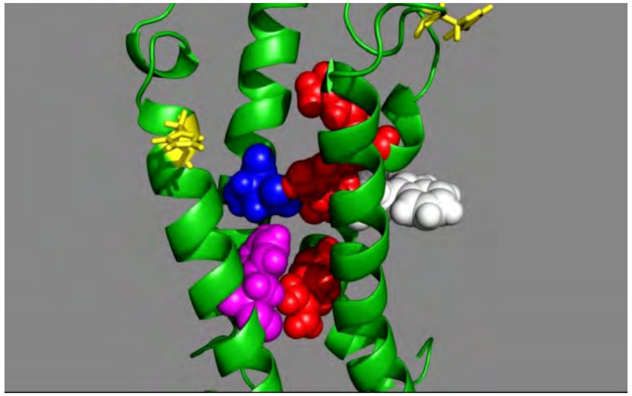

Figure 4: Snapshot from a MD simulation of a HV1 single monomer in the open state. Red: three arginines of the S4 helix, blue: aspartate D112 (selectivity filter) in S1, purple: charge transfer center F150 in S2. Yellow sticks show histidines at the external side of the channel. White: trypthophan in S4. The aspartate D112 and arginine R208 sidechains are connected via a salt bridge.

H_V1 is not exclusively expressed in phagocytes, but also in human lung cells, B-cells (8), sperm (9), muscle, and more. Furthermore, H_V1 seems to be expressed in some malignant cancers such as breast and colorectal cancer. In cancer cells, knock down of the proton channel results into less invasiveness and cell proliferation.

Several questions still unanswered.

1. Where is the conduction pathway of the H^+?
2. Is the pore water filled or dry?
3. How is the pH sensed by H_V1?
4. What is the mechanism of zinc inhibition?
5. Are there more proton channels?
6. Where is the dimerization interface?

H_V1 is not only expressed in humans but also in other eukaryotes. As far as we know today there is no H_V1 in prokaryotes. Table 1 shows proton channels which were electrophysiologically investigated. The spectrum of species ranges from protists (10) over algae and insect (11) to mouse and human (12). Several tissues and species haven't been investigated for proton channels. There is still much to research.

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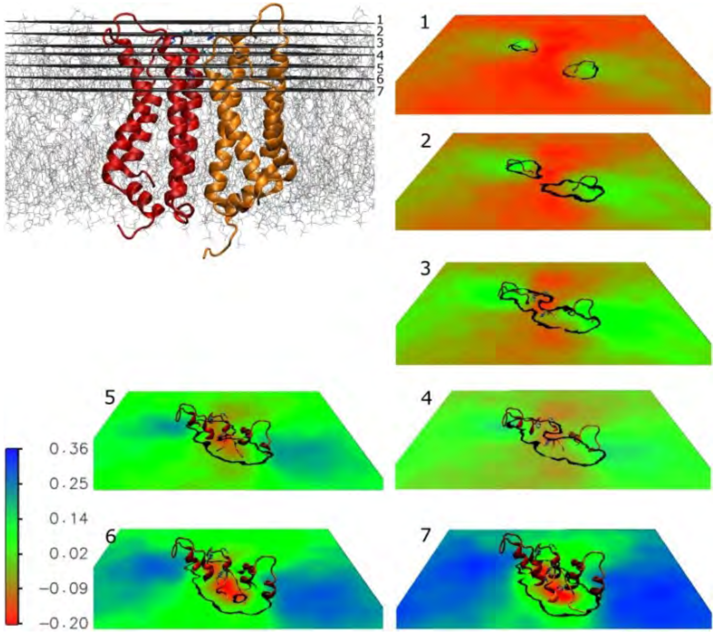

Figure 5: Electrostatic potential of the voltage-gated proton channel. Left: the dimeric proton channel embedded in the membrane (grey lines). The electrostatic is shown in 7 layers from the external bulk solution (layer 1), via the region containing the histidines H193 (layer 2), down to the region containing the two histidines H140 from each HV1 monomer (layer 7). In each layer, the black traces represent the contours of the HV1 monomers. A negative electrostatic potential is attracting Zn2+ cations from the bulk solution down to the histidine residues.

Name	Organism		molecular mass (Da)	Amino acids
hH_V1	*Homo sapiens*	(human)	31.683	273
hH_V1s	*Short Isoform*	(human)	29.415	253
mH_V1	*Mus musculus*	(mouse)	31.242	269
CiH_V1	*Ciona intestinalis*	(sea squirt)	38.501	342
SpH_V1	*Strongylocentrotus purpuratus*	(sea urchin)	37.483	328
EhH_V1	*Emiliania huxleyi*	(coccolithophore)	37.389	339
PtH_V1	*Phaeodactylum tricornutum*	(diatom)	38.582	338
CpH_V1	*Coccolithus pelagicus*	(coccolithophore)	36.281	325
kH_V1	*Karlodinium veneficum*	(dinoflagellat)	27.624	248
NpH_V1	*Nicoletia phytophila*	(insect)	27.680	239
DrH_V1	*Danio rerio*	(zebrafish)	27.110	235

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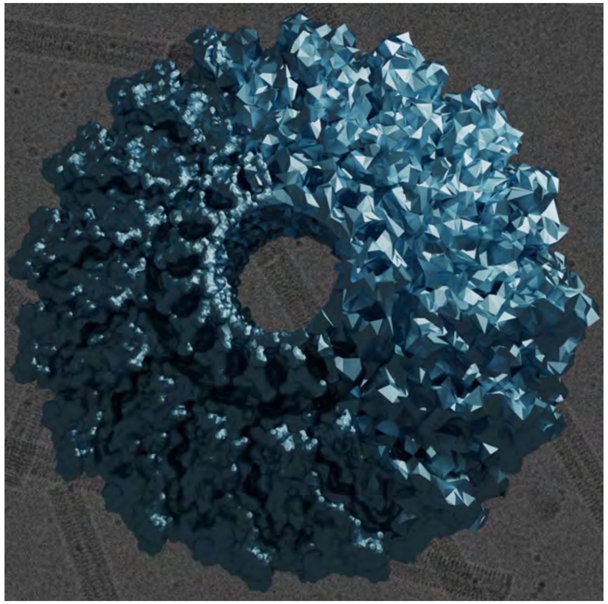
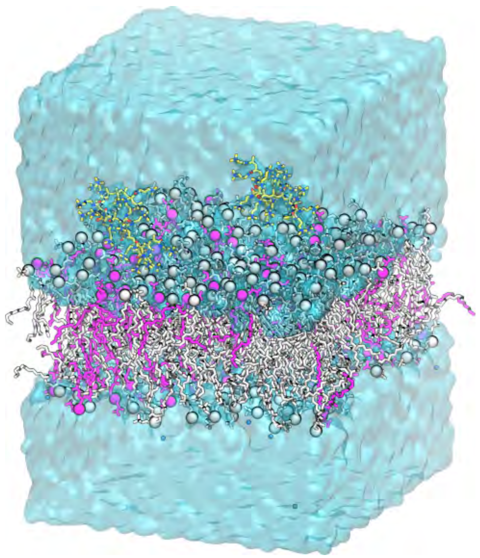


1. Preis: Daniel Mann

(Ruhr Universität Bochum)

Filaments of Tobacco mosaic virus (TMV) obtained via cryoelectron miroscopy (CryoEM). The self-assembling filaments consist of small, repetitive subunits with high information content in a small area, making TMV a great specimen for structure determination via CryoEM helical processing. Part of the near-atomic structure was heavily downsampled to obtain the low-poly look.



2. Preis: Wojciech Kopec

(Max Planck Institute for Biophysical Chemestry)

Two cationic PMAPTAC polymer molecules (yellow) adsorbed on the POPC/POPS lipid membrane (grey and magenta, respectively). Water is shown as cyan surface. Snapshot from Molecular Dynamics (MD) simulations.

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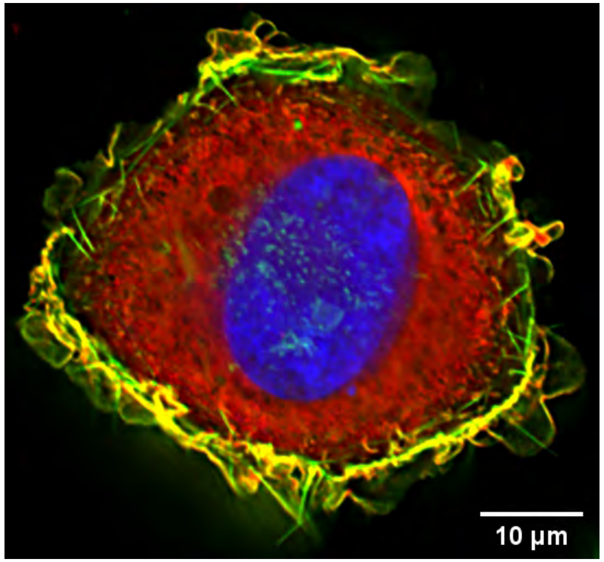

2. Preis: Philipp Dörfler

(Universität Bielefeld)

auf meinem eingereichten Bild ist eine einzelne humane embryonale Nierenzelle (HEK) zu sehen, bei der ich mit Immunofluoreszenzfärbung das Aktinzytoskelet (grün), Profilin 1 (rot) und den Zellkern (blau) gefärbt habe. In gelb erscheint Kolokalisazion von Aktin und Profilin 1. Die Zelle wurde mit einem Glutenfragment behandelt und scheint daher morphologische Veränderungen zu zeigen.
Aufgenommen wurde das Bild mit einem Deconvolution Wide-field Fluoreszenzmikroskop.

Diese einzelne Zelle erinnert mich außerdem stark an das Auge Mordors aus "der Herr der Ringe"-Verfilmung.

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Amtsträger 2019-2020

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