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POSTERS

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Abstracts submitted to the 49th FEBS Congress from 5th to 9th July 2025 and accepted by the Congress Organizing Committee are published in this Supplement of *FEBS Open Bio*. The abstracts are available as two PDF files: Talks (Plenary Lectures, Symposia and Special Sessions) and Posters.

About these abstracts

Abstracts submitted to the Congress are **not peer-reviewed**. In addition, abstracts are generally published as submitted and **are not fully copyedited** prior to publication. We are unable to make **corrections of any kind** to the abstracts once they are published.

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* Each poster has been given a unique number beginning with the letter P (or LB for 'late-breaking abstracts'); the next numerical part relates to the topic grouping as listed below.

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P-18-042
Nucleotide-dependent interrogation of damaged DNA during bacterial nucleotide excision repair

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Efficient elimination of DNA lesions by the nucleotide excision repair (NER) pathway is critical in all organisms. In bacteria, the early steps of NER are implemented by the successive action of three proteins, UvrA, UvrB, and UvrC via a series of large and dynamic multi-protein complexes. These complexes operate in three major stages during NER's early steps. During stage 1, a large (300-400 kDa) complex of the UvrA and UvrB proteins (AB) identifies lesion-containing DNA. This process requires rapid binding and release of DNA. Damaged DNA must be specifically recognized and distinguished from native DNA, even though the relevant lesions induce widely varying DNA structures. Lesion-containing DNA is stably bound by a dimeric form of UvrA within the AB complex (Stage 2). A major reorganization ensues wherein UvrA is lost from the ensemble, concomitantly, UvrB becomes localized at the site of damage (Stage 3). Additional events lead to excision of the damage on one strand, and repair of the resulting single-stranded gap. Here, we focus on nucleotide dependent discrimination of damaged DNA from native by UvrA, loading of UvrB at the lesion site, and the nucleotide dynamics driving these processes. Using cryogenic electron microscopy (Cryo-EM), we determined structures of UvrA bound to DNA damaged by three distinct lesions. We found that UvrA mounted a similar response against chemically distinct lesions. The dozens of structures in our study captured UvrA's proximal and distal sites filled with ADP, ATP, or ATPγS. dsDNA in our complexes was found in several configurations compared to B-form DNA; distortions in the DNA were directly linked to UvrA's nucleotide state. Our collection of structures could be grouped based on configurations of protein and DNA and arranged into a sequence of intermediates along the NER reaction coordinate.

P-18-043
Mechanistic understanding of the nucleotide excision repair: structural bases of damage detection process by UvrA and lesion hand-off to UvrB

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Nucleotide excision repair (NER) represents one of the major molecular machineries that control chromosome stability in all living species. In Eubacteria, the initial stages of the repair process are carried out by the UvrABC excinuclease complex. Despite the wealth of structural data available, some crucial details of the pathway remain elusive. In this study, we present a structural investigation of the *Mycobacterium tuberculosis* UvrAUvrB complex and of the UvrA dimer, both in complex with damaged DNA[1]. Our analyses have yielded new insights into the DNA binding mode of UvrA, showing an unexplored conformation of Insertion Domains (IDs), underlying the essential role of these domains in DNA coordination. Furthermore, we observed an interplay between the ID and the UvrB Binding Domain (UBD): after the recognition of the damage, the IDs repositions with the concomitant reorganization of UBD, allowing the formation of the complex between UvrA and UvrB. These events have been observed along the formation of the uncharacterized UvrA2-UvrB-DNA and the UvrA2-UvrB2-DNA complexes which we interpreted as hierarchical steps initiating the DNA repair cascade in NER pathway, resulting in the formation of the pre-incision complex. Thanks to the structural characterization, we were able to develop a model of DNA damage recognition, supported by biochemical data. 1) Marianna Genta, Giulia Ferrara, Riccardo Capelli et al., 04 November 2024, PREPRINT available at Research Square [https://doi.org/10.21203/rs.3.rs-5098223/v1]. Accepted by Nature Communications on 03/2025. *The authors marked with an asterisk equally contributed to the work.

P-18-044
How to be different: the structure of a non-canonical septin (Sep7) from *Magnaporthe oryzae*

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In most species, septins polymerize into hetero-oligomeric filaments involved in important cellular events which often require membrane remodelling. Over recent years we have provided crystal and cryo-EM structures of septin complexes from a wide range of different species. These have shed considerable light on how filaments are assembled and interact to form higher order structures. All reported structures are composed of "canonical" septins, classified into four different groups. These share a common fold which provides the two surfaces necessary for forming the interfaces (NC and G) required for polymerization. A "non-canonical" group of septins (group 5) has been described in some species but little is known about their role in filament assembly and subsequent downstream processes. Here we describe the structure of Sep7, a group 5 septin from *Magnaporthe oryzae*, a fungus responsible for rice blast disease which leads to a loss of 10–30% of harvests worldwide. Septin filaments form at the base of the appressorium, a structure which uses turgor pressure to invade the leaves of plants. Crystals of Sep7 could only be obtained after engineering a mutant which lacked 49 residues predicted to form a disordered internal loop. The resulting structure reveals radically different G and NC interfaces when compared to canonical septins, principally due to the insertion of a helix at the G-interface which destroys normal intersubunit contacts and generates novel ones. The interface is largely stabilized by a network of aromatic residues from a distorted 3-stranded meander at the base of the subunit. As a result, the interface is more open than normal and if a sep7 dimer were to incorporate

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into a canonical filament it would inevitably induce filament curvature. Alternatively, a compensating opening at the NC-interface could lead to homomeric linear filaments as seen in the crystal structure. Studies to investigate these possibilities are currently underway.

P-18-045
Structural biology technologies and how to access them

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Instruct-ERIC is a pan-European distributed research infrastructure making high-end technologies and methods in structural biology available to users. Instruct promotes innovation in biomedical science and operates on a non-economic basis within the scope of the ERIC Regulation. Instruct-ERIC is currently comprised of 17 Member Countries and Organisations: Belgium, Czech Republic, EMBL, Finland, France, Germany, Greece, Israel, Italy, Latvia, Lithuania, Netherlands, Portugal, Slovakia, Slovenia, Spain and United Kingdom. Researchers worldwide can apply for access to structural biology technologies and services at any of Instruct's 11 centres of excellence across Europe, whilst researchers in these 17 members can receive funded access through Instruct. Instruct-ERIC specifically supports research that uses integrated approaches and technologies. It operates with the following principles: a) scientific excellence is our priority in the services we provide and the supported research; b) transparency, equality and legality is the cornerstone of the operational model. In addition to access to high-end technologies for integrated structural biology, Instruct focuses on progressing basic research, such as sample preparation, protein production, and crystallisation for advanced structural analysis. Supporting the work of early-career researchers is a priority for Instruct; the highly successful internship programme is a central part of the organisation, providing a platform for PhD students and early-stage postdoctoral fellows to spend 3–6 months in a new laboratory in a new country to learn a new structural biology technique, to bring the expertise back to their home lab. Instruct also funds several training courses each year, focusing on different but fundamental integrated structural biology methods and techniques. Instruct also looks to provide funding for R&D, with schemes for early-career and advanced structural biology methods development.

P-18-046
Functional and structural studies of insect NavPas channel

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Voltage-gated Na^+ (Nav) channels of insects are the target of new insecticides. Despite their great importance for agriculture, there is still a lack of information on the structure and regulation of Nav channels. Eukaryotic Nav channels are formed by a core α-subunit, which is a single-polypeptide chain containing four homologous pseudo-subunits surrounding a central pore. Previously, it was reported that the function of the American cockroach NavPas channel could not be studied by electrophysiology in *Xenopus laevis* oocytes. Here, we studied the expression of this channel fused with the fluorescent protein tagRFP in eukaryotic HEK cells and observed a preferential accumulation of NavPas in intracellular compartments. Only at high expression the channel was observed on the cell membrane surface. Usage of tagRFP to visualize cells with surface expression of NavPas allowed us to study the channel activity using the whole-cell patch method. The structure of the NavPas channel was studied by cryo-EM in lipid-protein nanodiscs. The preferred orientation of the particles made it difficult to obtain a high-resolution structure. To address this issue, we collected over 23 000 images and extracted ~6 million particles. After 2D classification, 1 million "good" particles were selected, and after 3D classification, 327 000 particles were used for structure determination. Finally, a 2.9 Å structure was obtained for the monomeric form of NavPas in the nanodiscs. Compared with the previously published structure in digitonin micelles, the intracellular domains were found to be pressed against the surface of the nanodisc membrane. The obtained data provide new insights into the structure and function of the NavPas channel and are valuable for the future development of new insecticides. The work was supported by Guangdong Province Key Special Project #2023ZDZX2072. *The authors marked with an asterisk equally contributed to the work.

P-18-047
Evolution of pyridoxal kinase activity in bacterial vitamin kinase family: a kinetic, molecular and structural approach

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The biosynthesis of thiamine requires the phosphorylation of 4-amino-2-hydroxymethyl-5-methylpyrimidine (HMP) to HMP-PP with HMP-P as an intermediate. In bacteria, two homologous enzymes catalyze the phosphorylation of HMP: 1) ThiD2-ccPLK/HMPK which also phosphorylates pyridoxal (PL), and 2) ThiD-HMPPK kinases, which further phosphorylates HMP-P to produce HMP-PP. Crystal structures of an ancestral ThiD-HMPPK from Enterobacterales reveal that residues H179 and T211, essential for HMP-P phosphorylation, are missing in ThiD2-ccPLK/HMPK. Conversely, the crystal structure of *Staphylococcus aureus* ThiD2-ccPLK/HMPK shows a hemithioacetal intermediate formed between PL and the strictly conserved residue C111. Phylogenetic analyses suggest that ThiD2-ccPLK/HMPK enzymes evolved from ThiD-HMPPKs, leading to the loss of HMP-P kinase activity and the emergence of PL kinase activity as a novel function. To explore this transition, we reconstructed ancestral sequences corresponding to the last common ancestor of ThiD-HMPPK (ancThiD), an intermediate ancestor (ancC), and the last common ancestor of ThiD2-ccPLK/HMPK (ancThiD2). *In silico* analyses suggest that T211A was the first evolutionary step, followed by Q44M, A111C, and H179S, which reshaped the active site. Biochemical characterization demonstrated that ancThiD exclusively phosphorylates HMP and HMP-P, while ancC shows a wider substrate specificity, phosphorylating HMP, HMP-P, and PL. In