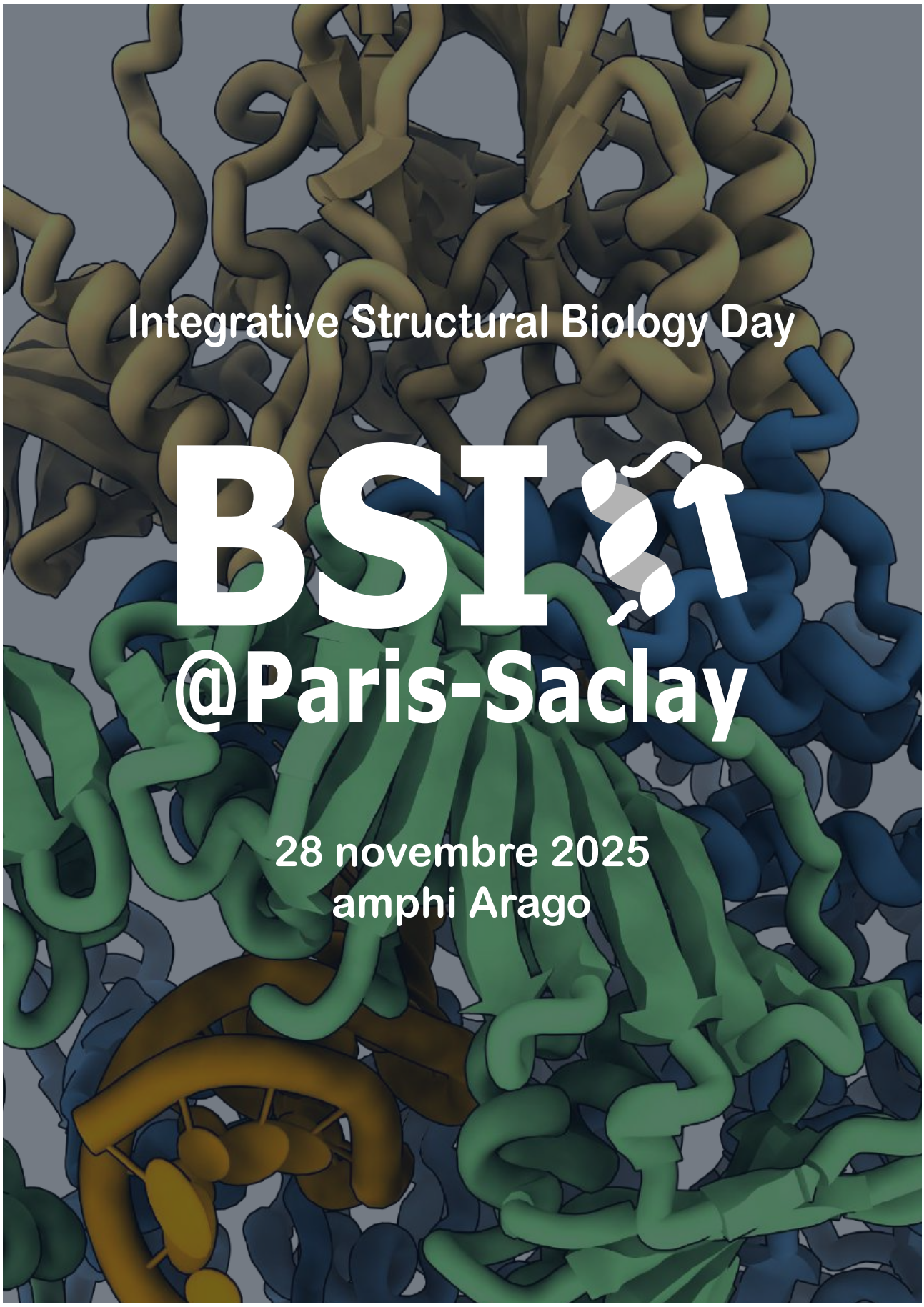
Integrative Structural Biology Day
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Integrative Structural Biology Day

November 28, 2025

Amphi Arago - École polytechnique

8h30-9h00	**WELCOME**	
9h00-9h10	**INTRODUCTION**	
9h10-10h30	**SESSION 1**	*Chairwoman: Françoise Ochsenbein*

Yawa Dossou - I2BC - Gif/Yvette
Structures of functional oligomers of protein NS5A from highly replicating strains of hepatitis C virus

Aanchal Mishra - CBM - Orléans
SUMO interacting motif of CBX4 adopts a unique conformation on binding to SUMO1

Audrey Labarde - I2BC - Gif/Yvette
Imaging of bacteriophage particle assembly in vivo using cryoFIB-SEM combined with cryoET

Zichen Feng - BioCis - Orsay
Modeling conformational ensembles of TDP43-RNA complexes using SAXS data and MD simulations

10h30-11h00	**COFFEE BREAK AND POSTERS**	
11h00-12h00	**RECENT UPDATES ON LOCAL PLATFORMS**	*Chairwoman: Sophie Combet*

Pierre Mahou - Mosphoscope - École polytechnique

Julie Menetrey - FRISBI

Ewen Lescop - RMNHC@UPSAY

12h00-14h00	**LUNCH AND POSTERS**	
14h00-15h00	**KEYNOTE**	*Chairman: Yves Mechulam*

Juliette Fédry - LMB-MRC - Cambridge, UK
Cryo-ET insights into mammalian proteostasis

15h00-16h20	**SESSION 2**	*Chairman: Thomas Bertrand*

Emma Maillard - I2BC - Gif/Yvette
In silico design of binder targeting the chromatin

Laura Pieri - I2BC - Gif/Yvette
Visualization of counterions adsorbed on self-assembled peptide nanotubes: An explanation of diameter modulation ?

Haoyang Zheng - I2BC - Gif/Yvette
Targeting biomolecular condensates of TopBP1 to inactivate ATR signaling pathway

Florentin Allemand - BIOC - Palaiseau
Drug–membrane interactions modulate GPCR binding: the example of P2Y12 and antiplatelet drugs"

16h20-16h50	**COFFEE BREAK AND POSTERS**	
16h50-17h30	**TEACHING ONTEGRATIVE STRUCTURAL BIOLOGY ON THE SACLAY AREA: MASTER'S DEGREE PROGRAMMES**	*Chairman: Ewen Lescop*

Yves Mechulam - Masters de l'Ecole polytechnique

Agathe Urvoas - Marielle Valerio-Lepiniec - Guillaume Lenoir: Masters de l'Univ. Paris-Saclay

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Abstracts for oral presentations

Structures of functional oligomers of protein NS5A from highly replicating strains of hepatitis C virus

Yawa Dossou **1**, Thibault Tubiana **1**, Gabriel Vanegas Arias **1**, Marion Babot **1**, Stéphane Bressanelli **1**, Virginie Gervais **1**

1 : Institute for Integrative Biology of the Cell (I2BC)*;* Université Paris-Saclay

Hepatitis C virus (HCV) is a major global health issue. A hyperreplicative strain of HCV was recently identified in a liver transplant patient (Heuss, *PLoS Pathog* 2022). This genotype 1b strain, referred to as GLT1 (German Liver Transplant 1) causes dramatic rearrangements of host cell membranes. This is associated with mutations in NS5A, a non-structural protein of the virus, specifically within a region (aa 237-342), termed the Replication Enhancing Domain (ReED). A unique substitution (P237L) was found as necessary and sufficient to confer the hyperreplicative phenotype in a consensus genotype 1b (gt1b) background and increase the dimerization of NS5A in huh7_lunet cells (P. Rothhaar et al., *in review (2025)*). Cyclophilin A (CypA) is a host peptidyl prolyl cis-trans isomerase, recruited by HCV to interact with NS5A, particularly with multiple proline-rich motifs within D2 (Hanoulle*, J. Biol. Chem.* 2009).

NS5A is essential for both HCV replication and virion assembly. It consists of three domains (AH-D1, D2, D3), connected by short low complexity sequences (LCS). NS5A is anchored to the membrane via its N-terminal amphipathic helix (AH) and is known to self-associate through D1. Structural predictions using AlphaFold (AF) suggest that an additional dimerization interface may be present, involving the ReED region in the N-terminus of D2. Given that CypA engages multiple sites in D2, we hypothesized that it might also interact with this region, stabilizing the peptide and potentially promoting its putative dimerization.

We used NMR to characterize this novel dimerization property, considering peptides of various lengths in ReED. Our data indicate the presence of an α-helix within the region predicted to be helical in AF model. However, no evidence of dimerization was found in solution. NMR titration experiments with CypA revealed chemical shift changes indicative of an interaction between this region and CypA, which were confirmed by Isothermal Titration Calorimetry (ITC) studies. Additional experiments are now required to determine how this interaction may stabilize the peptide conformation and potentially influence its dimerization. We also focus on a longer peptide, comparing variants with and without P237 to evaluate its role in dimerization and its link to the hyperreplicative phenotype.

SUMO interacting motif of CBX4 adopts a unique conformation on binding to SUMO1

Aanchal Mishra 1,2, Barbara Michalec-Wawiórka **3**, Stéphane Goffinont **1**, Franck Coste **1**, Leszek Wawiórka, **3**,Bertrand Castaing **1**, Marek Tchórzewski **3**, Marcin J. Suskiewicz **1**
1 Centre de Biophysique Moléculaire (CBM), UPR 4301, CNRS, Orléans, France
2 École Doctorale "Santé, Science Biologique & Chimie du Vivant" (ED549), Université d'Orléans, France
3 Department of Molecular Biology, Institute of Biological Sciences, Maria Curie-Skłodowska University, Lublin, Poland

SUMOylation, is a type of post-translational modification that involves covalent conjugation of small ubiquitin-like modifier (SUMO) proteins to target substrates. Most functions of SUMO depend on the establishment of non-covalent protein-protein interactions between SUMOylated substrates and their binding partners. The vast majority of these interactions involve a conserved surface in the SUMO protein and a SUMO interacting motif (SIM), a short stretch of hydrophobic amino acids and an acidic region, in the interactor protein. CBX4, a chromobox protein, has been proposed to possess two SIMs and function as a SUMO E3 ligase, an enzyme that accelerates the SUMOylation reaction. To understand the structural insights of this interaction, we crystallized SUMO1 alone using a fusion-assisted crystallization method, revealing a structure with an accessible pocket for SIM binding. Co-crystallization of this construct with a peptide comprising a SIM from CBX4 revealed an unusual binding mode in which the SIM is wedged between two SUMO molecules. The isothermal titration calorimetry (ITC) analysis of the interaction between the wild-type or mutant CBX4 SIM and SUMO1 reveals differential effects on SUMO1- SIM binding affinity, highlighting specific residues critical for this interaction. Cellular assays further confirm the effects of SIM mutations on SUMO colocalization, with the wild-type and mutant forms showing distinct localization patterns. Overall, this work will provide structural and biochemical insights into SUMO:SIM interactions, specifically elucidating the non-covalent interface between SUMO1 and the SIM of CBX4.

Keywords: SUMO1:SUMO-interacting motifs (SIM) interaction; fusion protein-assisted protein crystallisation; isothermal titration calorimetry (ITC); cell biology

Imaging of bacteriophage particle assembly *in vivo* using cryoFIB-SEM combined with cryoET

Audrey Labarde 1

1 Département de Virologie, Institut de Biologie Intégrative de la Cellule (I2BC) – CNRS : UMR9198 – Bât. 14B, 1 avenue de la terrasse 91198 Gif-sur-Yvette cedex, France

Viruses are obligate intracellular parasites that require massive host resources to multiply efficiently. These manipulations lead to major cell remodelling including formation of viral-induced compartments for viral genome replication and/or assembly of viral particles. While viruses of eukaryotic cells can rely upon existing cellular structures, bacteriophages, the viruses that infect bacteria, have to multiply in an initial non-compartmentalized cytoplasm.

SPP1 is a strictly lytic phage of the Gram-positive bacterium Bacillus subtilis. The molecular mechanisms underlying the different steps of its viral cycle were well characterized in vitro but their integration in the crowded bacterial cytoplasm remains unclear. We showed that more than 300 copies of the viral genome are synthetized within the first 30 minutes after phage entry. These DNA molecules occupy an important space in the cytoplasm where pro-capsids proceed to SPP1 genome encapsidation before segregating in warehouses (Labarde et al, 2021). We then used cryo-FIB milling and cryo-electron microscopy (cryoET) to analyze the extensive spatial reorganization of SPP1-infected cells at single viral particle resolution in near-native conditions. The most prominent feature is the formation of a large viral DNA compartment from which ribosomes are excluded. This also allowed to image sequential steps of the viral particle assembly pathway that are confined to specific locations in the cell. In particular, we showed that procapsid formation initiates at the cellular membrane in a process that requires the presence of the portal protein. Open procapsid precursors remain associated to the membrane until procapsid completion. Procapsids then relocalize to the bacterial DNA compartment for DNA packaging. Finally, DNA-filled capsids leave this compartment for tail binding and clustering in warehouses until cell lysis. Altogether, we provide comprehensive mechanistic insights into the complete assembly pathway from membrane-associated precursors of procapsids to DNA-filled head-and-tail capsids and to their trajectory in the infected bacterium.

Keywords: Viral-induced compartments, initiation of procapsid formation, portal protein, viral genome encapsidation, assembly pathway

Modeling conformational ensembles of TDP43-RNA complexes using SAXS data and MD simulations

Zichen Feng 1, Tap Ha-Duong **1** , and Liuba Mazzanti **1**
1 Université Paris-Saclay, CNRS, BioCIS, Orsay – Université Paris-Sud - France

TAR DNA binding protein 43 (TDP43) is involved in RNA-related metabolism (1). TDP43 is composed of 3 folded domains, the N-terminal domain (NTD) and two RNA-recognition motifs (RRM1 and RRM2), connected by unstructured linkers, and an intrinsically disordered C-terminal low-complexity domain (LCD) (2). Previous experimental studies showed the affinity of the two RRMs to GU-rich RNA sequences (3), and their blocking effect on the TDP43 aggregation (4). Unfortunately, the molecular details of the aggregation-preventing effect of TDP43 binding to RNA remains undiscovered, since traditional experimental methods are unsuccessful to investigate such intrinsically disordered protein-RNA complexes. To fill this gap, we employed molecular dynamics (MD) simulations to study the conformational ensembles of TDP43-RNA assemblies in combination with experimental SAXS data.
We will present the results of MD simulation analyses of TDP43 alone (NTD-RRM1-RRM2), monomeric TDP43/(GU)6, and dimeric TDP43/(GU)12 complexes, using AMBER03ws (5) and the AMBERchiol3 (6) force fields for protein and RNA, respectively. We then used CRYSOL 3.0 (7) to compute the theoretical SAXS intensities provided by simulations and compared them with available experimental SAXS data. Conformational ensembles extracted from MD trajectories were then refined using GAJOE (8, 9) to ensure agreement with the experimental results.

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In silico design of binder targeting the chromatin

Emma Maillard 1, Françoise Ochsenbein **1,2**, Raphaël Guérois **1**, and Baptiste Roelens **1**
1Institut de Biologie Intégrative de la Cellule (I2BC) – Commissariat à l'énergie atomique et aux énergies alternatives, Université Paris-Saclay, Centre National de la Recherche Scientifique – France
2 CEA Saclay / Institut de Biologie Intégrative de la Cellule – Institute for Integrative Biology of the Cell (I2BC), Universitey Paris-Saclay, Université Paris-Sud, CNRS, CEA, 91400 Orsay, France – Service de Bioénergétique, Biologie Structurale et Mécanismes (SB2SM) CEA Saclay, Institut Joliot, bat 144 Biochimie Biophysique et Biologie Structurale (B3S) I2BC, UMR 9198 91191 Gif-sur-Yvette, France

In eukaryotic cells, the genetic information is stored in DNA, organized into chromatin, which elementary unit is the nucleosome, a wrap of 147 base pair around an octamer of histones. Histones chaperone are paramount for DNA replication, both for preserving genome integrity and contributing to nucleosome dynamic. The histone chaperone Chromatin Assembly Factor 1 (CAF-1) is a three subunits complex that orchestrate the assembly of histone H3-H4 coupled to DNA synthesis during DNA replication and repair. WHD is a conserved 8.5kDa globular domain at the C-terminal extremity of its large subunit. Previous studies showed that WHD is necessary for nucleosome assembly in vitro and in vivo but its role remains to be unraveled.
To further study the role of WHD, we undertook the development of specific binders for this domain. Since 2020, the newly developed artificial intelligence tools such as AlphaFold, RosettaFold Diffusion and ProteinMPNN have deeply modified the protein design methodologies. For this project, I chose to use the RosettaFold Diffusion (1), as well as the Bindcraft pipeline (2) to generate de novo binders for WHD. Binders were first discriminated using visual and rational criteria such as hydrophobicity, packing of the interface and polar network. Then, the interaction with WHD was tested using a quantitative bacterial double hybrid system. This method allowed the selection of four binders, two of them were selected for further bio-chemical characterization. The binders were expressed, purified and characterized using DLS for their size and denaturation temperature measurement. The binders' interaction with WHD was then mostly studied using NMR on N15-labeled WHD, showing the binder interact with WHD through the predicted region. The affinity and kinetics of the interaction will be measured using BioLayer Interferometry.

(1) Watson et al, Nature, 2023
(2) Pacesa et al, Nature, 2025

Visualization of counterions adsorbed on self-assembled peptide nanotubes: An explanation of diameter modulation?

Laura Pieri 1, Rémi Ruedas **2**, Stéphane Bressanelli **1**, and Maïté Paternostre **1**
1Institut de Biologie Intégrative de la Cellule, Gif-sur-Yvette (I2BC) – CNRS – France
2Basel University, Biozentrum, Basel – Suisse

Lanreotide is a dicationic octapeptide that spontaneously assembles into hollow nanotubes of homogeneous diameter. The nanotube diameter can be slightly modulated either by counterion exchange[1] or by specific residue substitution[2]. In 2022, we solved the atomic structure of lanreotide–acetate nanotubes at 2.4 Å resolution using high-resolution cryo-electron microscopy (cryo-EM)[3]. Interestingly, some faint density peaks in the cryo-EM map could not be attributed to the lanreotide atomic model. Suspecting that these peaks might correspond to counterions embedded within the nanotube wall, we first improved the resolution of the lanreotide–acetate map to 2.2 Å. We then replaced acetate with counterions of increasing electron density, i.e. chloride, bromide, and iodide. Cryo-EM maps and atomic structures were obtained at resolutions ranging from 2.0 to 2.4 Å for nanotubes formed in the presence of these different counterions, as well as for nanotubes formed by the phenyl-acetate derivative of lanreotide, in which the D-Trp residue at position 4 was replaced by D-Phe. Extra densities that could not be explained by the lanreotide atomic model were observed at the same positions in all maps. For some of these sites, the peak heights were similar across counterions, whereas for others, the peak intensity increased with the electron density of the counterion.
Here, I will illustrate and compare the cryo-EM structures of lanreotide nanotubes obtained by counterion exchange or residue substitution, and discuss the possible assignment of these extra densities to counterions or water molecules. Resolving this question may shed light on the respective roles of counterions and hydration in modulating nanotube wall curvature.

1- Gobeaux et al. JACS, 2012, 134, 723-733
2- Tarabout et al Proc Natl Acad Sci U S A. 2011 108,7679–7684
3- Pieri et al. Proc Natl Acad Sci U S A. 2022, 119 (4).

Targeting biomolecular condensates of TopBP1 to inactivate ATR signaling pathway

Haoyang Zheng 1, Simona Miron **1**, Leonardo Bartkevihi-Di-Piero **1**, Atifa Badar-Majeed **1**, Stephane Plancqueel **1**, Elia Moarbess **2**, Tom Egger **2**, Sophie Zinn-Justin **1**, and Jihane Basbous **2**

1 Institute for Integrative Biology of the Cell (I2BC), University of Paris-saclay – France
2 Institut de génétique humaine (IGH) – Centre National de la Recherche Scientifique, Université de Montpellier – France

Cancer cells exhibit an increased dependence on the serine/threonine kinase ataxia-telangiectasia and Rad3-related (ATR) pathway to tolerate DNA damage(Heyza et al., NAR Cancer, 2023). Once activated, ATR phosphorylates multiple substrates, including the checkpoint kinase Chk1, to mediate cell cycle arrest, replication fork stabilization, and DNA damage repair (Qiu et al., Radiother. Oncol., 2018). Mechanistically, ATR activation is regulated by the formation of TopBP1 biomolecular condensates, which act as a molecular switch. TopBP1 self-assemblies into condensates upon phosphorylation by ATR, thereby enhancing ATR kinase function (Frattini et al., Mol. Cell, 2021). This highlights TopBP1 condensates as an attractive alternative therapeutic target.

Here, we elucidated the structural mechanism underlying TopBP1 condensation. TopBP1 comprises nine BRCA1 C-terminal (BRCT) domains and an intrinsically disordered ATR Activation Domain (AAD). The AAD is indispensable for ATR activation but insufficient to drive condensation on its own (Frattini et al., Mol. Cell, 2021). Moreover, deletion of BRCT5 abolishes TopBP1 condensation during interphase (De Marco Zompit et al., Nat. Commun., 2022), suggesting that condensation may depend on inter- or intramolecular interactions between AAD and BRCT5. Using nuclear magnetic resonance (NMR) spectroscopy, we established that AAD physically interacts with the BRCT4/5 domains. Notably, AAD phosphorylation at Ser1138 increased its binding affinity, which could provide a mechanistic basis for TopBP1 condensation.

Finally, we explored a nanobody-based approach to disrupt this interaction and modulate TopBP1 condensates. Nanobodies, single-domain antibodies derived from camelids, are valued for their small size and high affinity. We successfully generated three nanobodies (A03, G07, and F08), all of which exhibit nanomolar affinity for the AAD in vitro. Interestingly, these nanobodies recognize distinct epitopes within the AAD, enabling functional dissection of local regions. In cells, they colocalize with TopBP1 condensates. Further in cell characterizations are ongoing to assess the ability of these nanobodies to inactivate ATR. Together, our strategy could open avenues in cancer therapy and overcome chemo-/radiotherapy resistance.

Drug–membrane interactions modulate GPCR binding: the example of P2Y12 and antiplatelet drugs

Florentin Allemand 1, Jennifer Lagoutte-Renosi **2,3**, Siamak Davani **2,3**, and Christophe Ramseyer **4**

1 Laboratoire de Biologie Structurale de la Cellule – Ecole Polytechnique, CNRS – France

2 SINERGIES – Université Marie et Louis Pasteur – France

3 Service de Pharmacologie Clinique et Toxicologie – CHU Besançon – France

4 Laboratoire Chrono-environnement – Centre National de la Recherche Scientifique, Université Marie et Louis Pasteur – France

Most drugs target membrane proteins, over a third being G protein-coupled receptors (GPCRs). The P2Y12 receptor, which is a target for antiplatelet drugs, is a GPCR embedded in the platelet plasma membrane. This complex membrane contains lipids with different heads and tails, showing vertical and lateral asymmetry. Lipid domains form, such as rigid lipid rafts, while regions rich in PolyUnsaturated Fatty Acids (PUFA) are disordered. The platelet plasma membrane contains high levels of the PUFA ARachidonic Acid (ARA). Coarse-grained molecular dynamics (MD) has shown that lipids containing these ARA chains form nanodomains in which P2Y12 receptors oligomerize. Lipids containing ARA or cholesterol molecules localize and remain stable at the interface between the different receptors forming these oligomers. Antiplatelet drugs targeting P2Y12, ticagrelor and Prasugrel Active Metabolite (PAM), can insert into the lipid bilayer due to their physico-chemical properties. Calculations of free energy landscapes have shown that the insertion of these molecules is possible at ARA-enriched domains but not at lipid rafts. MD simulations of ARA-enriched domain models have shown that the insertion of ticagrelor and PAM rigidifies these domains. In addition, in vitro experiments demonstrated that ticagrelor rigidifies the platelet plasma membrane. This affects the conformation of the P2Y12 receptors embedded within them. Indeed, ensemble docking calculations have revealed that ticagrelor binds more favorably to P2Y12 in a rigid, lipid raft-like environment, as well as after its insertion into an ARA-enriched environment. PAM is an irreversible P2Y12 antagonist which forms a disulfide bridge with a cysteine residue of the receptor. A key parameter for this binding is therefore the distance between the sulfur atom of the ligand and that of the cysteine residue of P2Y12. Results from ensemble docking showed that inserting PAM into ARA-enriched nanodomains modifies its binding mode to P2Y12, distancing the sulfur atom from that of the cysteine residue. The insertion of ticagrelor and PAM into ARA-rich nanodomains has opposite effects on their binding to P2Y12, promoting and inhibiting it, respectively. Thus, the lipid environment influences ligand binding, and ligands in turn modify this environment.

Abstracts for poster presentations

Poster 1

Exploration of the stability of the HSP90 N-terminal domain at various pressures and temperatures by NMR spectroscopy

Diane D'esquermes 1,2, Faustine Henot **1**, Elisa Rioual **1**, Eric Jacquet **2**, François Giraud **1**, Guillaume Bouvignies **3**, Jérôme Boisbouvier **1**, and Ewen Lescop **2**

1 Institut de biologie structurale (IBS - NMRLA) – Centre National de la Recherche Scientifique, Institut de Recherche Interdisciplinaire de Grenoble, Université Grenoble Alpes – France

2 Institut de Chimie des Substances Naturelles (ICSN) – Université Paris-Saclay, Centre National de la Recherche Scientifique, Institut de Chimie du CNRS – France

3 Chimie Physique et Chimie du Vivant (CPCV) – Département de Chimie - ENS-PSL, Institut National de la Santé et de la Recherche Médicale, Institut de Chimie - CNRS Chimie, Sorbonne Universite, Centre National de la Recherche Scientifique – France

HSP90 is a chaperone responsible for the correct folding of client proteins. When this protein dysfunctions it can cause various illnesses, therefore, it is a therapeutic target for possible treatments. This protein is highly flexible and undergoes large-scale structural rearrangements to perform its functional cycle. It is composed of three domains, and it is the ATP binding N-terminal domain (NTD, 25 kDa) that is the most flexible due to the presence of a long loop named ATP-lid that covers the nucleotide binding site. It was previously demonstrated that the HSP90-NTD possesses one major fundamental state in exchange with one minor excited state (1) characterized respectively by the ATP-lid in an open and closed conformation. The behavior of this flexible loop has been extensively described at various temperatures but never at higher pressures. However, pressure, like temperature, is a fundamental thermodynamic variable that can be used to modulate the free energy landscape of biomolecules, thereby providing a tool to study conformational fluctuations. Here, we investigated and optimized by NMR and thermal shift assays the pressure/ temperature conditions ensuring protein stability. We notably revealed at high pressure a new HSP90-NTD largely disordered state that is largely reversible. This preliminary study paves the way for the complete structural comprehension and analysis of HSP90-NTD.

(1) : Henot et al.;Visualizing the transiently populated closed-state of human HSP90 ATP binding domain. Nature Communications, 2022

Poster 2

Structural observations of *Vibrio cholerae* XerCD complex on DNA

Mael Balanec 1, Francois-Xavier Barre , James Iain Provan , Arthur Vitard **2**, Pierre Legrand , Magda Teixeira-Nunes , and Françoise Ochsenbein **3**

1 Institut de Biologie Intégrative de la Cellule (I2BC) – Commissariat à l'énergie atomique et aux énergies alternatives, Université Paris-Saclay, Centre National de la Recherche Scientifique – France

2 Institut de Biologie Intégrative de la Cellule – Commissariat à l'énergie atomique et aux énergies alternatives, Université Paris-Saclay, Centre National de la Recherche Scientifique, Commissariat à l'énergie atomique et aux énergies alternatives : DRF/I2BC, Centre National de la Recherche Scientifique : UMR9198 – France

3 Laboratoire de Biologie Structurale et Radiobiologie (LBSR) – CEA – France

Chromosome dimers form in around 10 % of the cells per generation in bacteria. If left unresolved, they remain trapped between the two daughter cells during division, which leads to chromosome shearing and eventually cell death. Chromosome dimers are separated by the addition of a crossover at a specific site, dif, by two highly conserved tyrosine recombinases, XerC and XerD. Numerous bacterial viruses exploit their host's Xer machinery to integrate in their host's genome. This includes viruses that provided the cholera toxin genes to pandemic strains of *Vibrio cholerae*.

XerC and XerD form heterotetrameric nucleoprotein complexes, composed of two target sites and a pair of each of the two recombinases. XerC and XerD act successively to exchange a specific pair of strands. Two recombination pathways can be defined depending on whether XerC or XerD initiates the process. Chromosome dimer resolution is initiated by XerD. This requires a direct contact with the extreme C-terminal domain of a cell division protein, FtsK. Bacteriophages can bypass the FtsK requirement by utilising the basal FtsK-independent activity of XerC and/or by producing their own Xer activation factor. Thirty years of genetic and molecular research has yielded a deep understanding of Xer recombination. However, structural understanding of the process is still lacking. This is mainly due to the poor solubility and tendency to aggregate of XerC and XerD. Our recent work enabled homogeneous purification of *Vibrio cholerae* XerCD-dif nucleoprotein complexes at a scale suitable for structural characterization by X-ray crystallography and Cryo-electron microscopy studies. Building on previous genetic and molecular biology knowledge on the Xer recombinases, we assembled XerCD-dif nucleoprotein complexes trapped at various stages of the recombination reaction. We obtained CryoEM models of the synaptic complex using wild-type XerC and XerD proteins, the XerD-cleaved complex using XerC and a hyperactive XerD-FtsK fusion on a nicked dif site and the XerC-cleaved complex at resolutions between 2.3 Å and 2.7 Å. We also obtained the first model of wild-type XerCD-dif Holliday-junction intermediate at 3 Å by Xray-crystallography.

Poster 3

Exploring the link between RNA-binding of TDP-43 and its aggregation

Jiawei Dong 1, Yitian Feng **1**, Marie-Jeanne Clément **1**, Célia Rabhi **1,2**, Vandana Joshi **1**, Sirhii Pankivskiy **1**, Juan Rengifo Gonzalez **1**, Aurélien Thureau **3**, David Pastré **1**, and Ahmed Bouhss **1**

1 Structure et activité des biomolécules normales et pathologiques (SABNP) – Université d'Evry-Val-d'Essonne, Institut National de la Santé et de la Recherche Médicale, Université Paris-Saclay – France

2 4P-PHARMA – Institut Pasteur – Institut Pasteur Lille – France

3 Synchrotron SOLEIL (SOLEIL) – Centre National de la Recherche Scientifique, Centre National de la Recherche Scientifique : UR1 – France

TDP-43 is a nuclear RNA-binding protein found in cytoplasmic inclusions of neurons in two major neurodegenerative diseases, ALS and FTLD (1). So far, there is no efficient drug for curing these diseases. Recently, we demonstrated that tandem RNA recognition motifs (RRM) of TDP-43 bind to its RNA target in a cooperative manner through intermolecular interactions (2). This cooperative binding of TDP- 43 to mRNA is critical to maintain the solubility of TDP- 43 in the nucleus and the miscibility of TDP- 43 in cytoplasmic stress granules (2). More recently, we successfully developed an automated detection pipeline to assess TDP-43 self-assembly combined to functional screen in living cells. We showed that the post-translational modifications affect nuclear TDP-43 localization and consequently its functions. Currently, we explore the structural effect of a pathological mutation on residues belonging to RRM domains. This fundamental knowledge will pave the way to elucidate the mechanism of TDP-43 proteinopathy and to design small molecules interfering with TDP-43 aggregation.

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Poster 4

Translation initiation of leaderless mRNAs in *Deinococcus deserti*

Valentina Varriale 1, Clément Madru **1**, Gabrielle Bourgeois **1**, Christine Lazennec-Schurdevin **1**, Laurence Blanchard **2**, Arjan De Groot **2**, Yves Mechulam **1**, and Emmanuelle Schmitt **1**

1 Laboratoire de Biologie Structurale de la Cellule – Ecole Polytechnique, Centre National de la Recherche Scientifique – France

2 CEA-DRF-BIAM-MEM – Centre de recherche du Commissariat à l'Energie Atomique - CEA Cadarache (Saint Paul-lez-Durance, France) – UMR 7265 CEA-CNRS-AMU CEA Cadarache 13108 Saint Paul Lez Durance, France

Translation initiation kickstarts protein synthesis thanks to the formation of a stable initiation complex (IC). While canonical leadered mRNAs (SD-mRNAs) rely on Shine–Dalgarno (SD)–anti-SD pairing for ribosome recruitment, leaderless mRNAs (lmRNAs) do not carry a typical SD and initiate translation through alternative, less-defined mechanisms. Importantly, lmRNAs are widely used by both archaea and bacteria1. We applied structural and molecular biology techniques to the study of the initial phases of translation in *Deinococcus deserti*, a bacterial species in which SD-mRNAs and lmRNAs coexist in almost equal proportions2. We established an in vitro reconstitution system for *D. deserti* translation initiation and obtained the first cryo-EM structure of a 30S translation initiation complex from this organism. In this reconstruction, no stable SD-anti-SD interaction was observed, despite the use of a SD-mRNA during *in vitro* reconstitution. Instead, we detected a codon-anticodon interaction involving a 5' start codon preceded by a triphosphate group, typical features of lmRNAs, suggesting that our isolated 30S subunits are bound to lmRNAs in this species. Using toeprinting experiments, we optimized experimental conditions to observe robust SD-dependent and lmRNA IC formation. Interestingly, ribosomal protein uS2 was consistently absent in our 30S reconstitutions. To investigate this further, we prepared a new batch of ribosomes using a recently developed method, RNA affinity purification using poly-lysine beads (RAPPL)3. Cryo-EM reconstruction of these ribosomes revealed that P-site tRNA-bound 70S ribosomes either contain uS2 (67%) or lack uS2 (33%), highlighting potential structural heterogeneity among *D. deserti* ribosomes. Furthermore, toeprinting experiments revealed that the stability of 30S initiation complexes with SD-mRNAs – but not with lmRNAs - is enhanced if an excess of uS2 protein is present. Together, our results suggest that uS2 may function as a molecular switch during translation initiation in *D. deserti*, contributing to the regulation of ribosomal activity during this critical phase of protein synthesis.

Poster 5

Artificial protein scaffolds to facilitate the Cryo-EM structure determination of tricky proteins

Nathan Epalle 1

1 Institut de Biologie Intégrative de la Cellule (I2BC) – Commissariat à l'énergie atomique et aux énergies alternatives, Université Paris-Saclay, Centre National de la Recherche Scientifique – France

The Protein Engineering and Modeling (MIP) team has designed a family of artificial proteins composed of alpha helices adapted from HEAT repeats (αReps). The highly-variable internal face of these αReps can be used to select specific binders for proteins of interest. This work is using these binders, fused with artificial protein trimers in order to attach the proteins of interest to a large protein scaffold. Such complexes would be more easily detected with Cryo-Electron Microscopy, which struggles to accurately detect smaller proteins (below 50 kDa). Once a suitable workflow is established, these scaffolds could be adapted for any protein of interest provided that an appropriate αRep binder has been selected against it.

Poster 6

Exploration of lipid phase transitions and coupling with scaffold protein in nanodiscs using high-pressure NMR.

Clarisse Fourel 1,2, Alexandre Pozza **1**, François Giraud **2**, Elodie Point **1**, Christel Le Bon **1**, Karine Moncoq **1**, Laurent Catoire **1**, Ewen Lescop **2**

1 Institut de biologie physico-chimique (IBPC (FR550))− −Sorbonne Universite, Centre National de la Recherche Scientifique− 13 Rue Pierre et Marie Curie 75005 PARIS, France

2 Institut de Chimie des Substances Naturelles (ICSN) – Institut de Chimie du CNRS, Université Paris-Saclay, Centre National de la Recherche Scientifique – CNRS, Bâtiment 27, Avenue de la Terrasse, 91198, Gif-sur-Yvette cedex, France

Biological membranes play a fundamental role in cell function, controlling the passage of molecules and orchestrating processes such as signalling. Nanodiscs (NDs) are a widely used model for mimicking biological membranes and studying membrane proteins in a controlled lipid environment. These nanoparticles consist of hundreds of phospholipids surrounded by two lipoproteins. However, the dynamic interactions between lipids and lipoproteins in these systems are still poorly understood.

In this work, we use liquid-state 1H, 13C NMR combined with hydrostatic pressure and temperature variations to probe the fluid-to-gel phase transitions of lipids in NDs at the atomic scale, and to simultaneously reveal atomic-resolution dynamic details for both the lipids and the lipoprotein. We compared NDs assembled with MSP1D1 and two different lipid compositions, DMPC and D9-cis-PC. We show that only DMPC undergoes a pressure-sensitive phase transition at 35◦C. A detailed analysis of the 13C NMR chemical shifts along the phospholipid chains in the same samples provides insights into the conformational behavior of the carbon atoms. Carbons located in the hydrophobic core of the bilayer, except for the terminal C14, appear to be more sensitive to pressure than those in the polar headgroup region. This may reflect pressure-induced rearrangements of the gauche/trans dihedral angles along the acyl chains. We prepared DMPC-based nanodiscs using three different lipoproteins: D1 (reference, ∽10nm), E3 (larger, ∽12nm), and DH5 (smaller, ∽8nm). Temperature-dependent 1H NMR data, supported by DSC measurements, confirm the presence of a phase transition in all three nanodisc systems. The 13C NMR spectra show highly similar profiles across the three nanodiscs. To assess the behavior of the lipoprotein during the lipid phase transition, we also monitored a 15N, 13C and partially 2H labelled lipoprotein D1 within a DMPC ND by NMR. The 15N and 13C HSQC spectra revealed significant side chain orientation for a subset of tryptophan and asparagine/glutamine residues, most likely associated with the partial exclusion of the lipoprotein from the gel phase. These results, which are already of significant interest, establish a solid foundation for incorporating membrane proteins into these nanodisc systems in future studies.

Poster 7

Histone assembly mechanism coupled to DNA synthesis

Eleni Litsardaki 1, Fouad Ouasti **1**, Maxime Audin**1**, Karine Fréon **2**, Jean-Pierre Quivy **3**, Mehdi Tachekort **1**, Aurélien Thureau **4**, Gwenaelle Moal **1**, Raphaël Guérois **1**, Sarah Lambert **2**, Geneviève Almouzni **3**, and Françoise Ochsenbein **1**

1 Institut de Biologie Intégrative de la Cellule (I2BC) – Commissariat à l'énergie atomique et aux énergies alternatives, Université Paris-Saclay, Centre National de la Recherche Scientifique – France

2 Institut Curie, Orsay – Institut Curie, PSL Research University, CNRS, Equipe Labellisée Ligue Nationale Contre le Cancer, INSERM U1278, University Paris-Saclay – France

3 Institut Curie, Paris – Institut Curie, PSL Research University, CNRS, Sorbonne University, Nuclear Dynamics Unit – France

4 Synchrotron SOLEIL (SOLEIL) – Centre National de la Recherche Scientifique : UR1, Centre National de la Recherche Scientifique – France

Most cancers or cellular aging processes result not only from the alteration of gene sequences but also from the loss of epigenetic information that regulates gene expression. Essential epigenetic marks carried by histones may be lost during DNA replication or DNA repair, processes that require dissociation and reassembly of nucleosomes. Chromatin Assembly Factor 1 (CAF-1) is a three subunits complex, conserved in all eukaryotes, that orchestrates the assembly of histones H3-H4 coupled to DNA synthesis in the context of DNA replication and repair (1). This histone chaperone is thus particularly important for the maintenance of cell identity, but its action mechanism remains poorly understood. It has been established that its association with the DNA polymerase processivity factor, PCNA, is required for its functions during DNA replication, heterochromatin maintenance, and genome stability (2-3). A model for the histone deposition mechanism has been proposed from studies of a truncated S. cerevisiae CAF-1 complex (4), but we lack a framework to demonstrate its generality and how histone deposition is coupled to PCNA interaction.

We have undertaken structural and functional studies of the CAF-1 complex from yeast *S. pombe*, using an integrative approach combining functional studies, biochemistry and structural biology methods and bioinformatics modeling. In particular, we have investigated how this complex (composed of Pcf1, Pcf2 and Pcf3) and its individual domains interact with different partners to deliver histones onto DNA. We focused on CAF-1 interactions with DNA, histones H3-H4 and PCNA. We show that the ED domain of Pcf1 mediates histone binding and promotes conformational changes in CAF-1 (5). We then explored how additional interactions of histones H3-H4 with the two other subunits, Pcf2 and Pcf3 may trigger this conformational change. We also investigated the interactions of the full CAF-1 complex with both DNA and PCNA, and here we present results revealing new binding interfaces and conformational adaptations within CAF-1 during histone assembly.

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Poster 8

Characterization of m6A 18S rRNA methyltransferase METTL5 mutants responsible for neurodevelopmental disorders

Laurianne Li En Tay 1, 2, Reza Maroofian **3**, Domizia Pasquetti **4**, Nathalie Ulryck **1**, David Touboul **2** , Denis L.J. Lafontaine **5**, Marc Graille **1**

1 Laboratoire de Biologie Structurale de la Cellule (BIOC) Ecole Polytechnique, Centre National de la Recherche Scientifique

2 Laboratoire de Chimie Moléculaire (LCM) Ecole Polytechnique, Centre National de la Recherche Scientifique

3 Department of Neuromuscular Diseases, UCL Institute of Neurology

4 Università Cattolica del Sacro Cuore, Milano - Italie

5 Université libre de Bruxelles (ULB), Charleroi

Modified nucleotides in non-coding RNAs, such as rRNAs, tRNAs and snRNAs, represent an important layer of gene expression regulation through their ability to fine-tune mRNA maturation and translation. Dysregulation of such modifications and the enzymes installing them have been linked to various human pathologies including neurodevelopmental disorders and cancers.

Several methyltransferases (MTases) are regulated allosterically by human TRMT112. All the TRMT112-MTase complexes modify factors (snRNAs, tRNAs, rRNAs and proteins) involved in the mRNA maturation and translation processes. Indeed, the TRMT112-BUD23 and TRMT112-METTL5 complexes catalyze the formation of N7-methylguanosine (m7G) and N6-methyladenosine (m6A) on the 18S rRNA and participate in 40S ribosomal subunit biogenesis pathway. The TRMT112-HEMK2 complex modifies the translation termination factor eRF1, which is a tRNA mimicry critical for the release of the newly synthesized proteins. The TRMT112-ALKBH8, TRMT112-TRMT11 and TRMT112-THUMPD3 complexes contribute to translation elongation by modifying tRNAs. Finally, the TRMT112-THUMPD2 complex methylates U6 snRNA, the core component of the major catalytic spliceosome and hence is important for optimal splicing of weak splice sites.

Recent results obtained on the functional characterization of eight homozygous METTL5 missense mutants identified in patients suffering from intellectual disability and microcephaly with varying degrees of penetrance will be presented. This work brings decisive information on the importance of this 18S rRNA m6A methylation, in normal brain development. Considering that the modification is adjacent to the ribosomal decoding site, it suggests that its absence rewires translation with important consequences in maturing neurons.

Poster 9

Functional and structural studies of *S. cerevisiae* and *S. pombe* NHEJ pathways

Claire Tettaravou 1, Sophia Missoury **2**, Sara Castelli **3**, Selma Dyca **4**, Vincent Morin **1**, Pierre Legrand **5**, Mauro Modesti **6**, Sarah Lambert **4**, Stephane Marcand **3**, Marc Delarue **2**, Virginie Ropars **1**, Jean-Baptiste Charbonnier **1**

1 Institute for Integrative Biology of the Cell (I2BC), UMR9198, Gif-sur-Yvette

2 Institut Pasteur, UMR3528, Paris

3 Institut de Biologie François Jacob, CEA, UMRE008, IRCM,Fontenay-aux-Rose

4 Institut Curie, UMR3348, Orsay

5 Synchrotron SOLEIL, L'Orme des Merisiers, Gif-sur-Yvette

6 Cancer Research Center of Marseille, UMR7258, Marseille

KU70-KU80 (called KU hereafter) is a central actor of genome stability through its multifaceted role in DNA metabolism. Its first seminal role is to recognize DNA ends at double-strand breaks (DSBs) through its ring-shaped structure and coordinate the recruitment and assembly of most factors of the NHEJ (Non-homologous end joining) DNA repair pathway. The main factors in human are XLF, XRCC4, Ligase 4, APLF,PAXX and DNA-PKcs. Ku recruits factors to DSBs through a motif called Ku binding motif (KBM). There are three types of KBM : A-KBM, X-KBM,P-KBM. In contrast to human, yeast lacks DNA-PKcs, which enable DNA end joining ; another protein, MRX (Mre11-Rad50-Nbs1/Xrs2) may replace this function, although this remains uncertain.

Furthermore, during NHEJ repair, KU interacts with several factors and form two types of complexes in human, whose structure are known : long-range complex, which involves the presence of DNA-PKcs, and the short-range complex, formed during DNA ligation. In yeast, *Saccharomyces cerevisiae* and *Schizosaccharomyces pombe*, however, we do not yet know how the factors interact with each other to form the complex. Therefore, Ku is involved in NHEJ, but in addition to this, it's also implicated during DNA replication and telomere maintenace.

In this poster, I will present the first structure analysis of the short range complex in *Saccharomyces cerevisiae*.

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Poster 10

Identification and characterization of filament-forming transcription factors

Astride Matéo 1,2, Franck Coste **1**, Rafeal Cepa **1**, Stéphane Goffinont**1**, Lucija Mance **1,2**, Max Staller **3,4,5**, Antoni Wrobel **6**, Bertrand Castaing **1**, and Marcin Suskiewicz **1**

1 Centre de biophysique moléculaire (CBM - UPR 4301) – Centre national de la recherche scientifique - CNRS (France) – France
2 Ecole doctorale Santé, Sciences Biologiques et Chimie du Vivant (ED 549 - SSBCV) – Université d'Orléans – France
3 Department of Molecular and Cell Biology, University of California –Etats-Unis
4 Chan Zuckerberg BioHub [San Francisco, CA] –Etats-Unis
5 Center for Computational Biology, University of California –Etats-Unis
6 Department of Biochemistry, University of Oxford – Royaume-Uni

Transcription factors (TFs) - key proteins that control gene expression by binding to specific DNA sequences - have recently been found to form filamentous self-assemblies driven by folded domains, such as the BTB domains in human ZBTB proteins. This newly discovered function leads to the formation of homomultimers, also known as filaments. However, the phenomenon of TF filamentation remains under-explored.
This poster presents two approaches for studying human TF polymerisation: *in silico* and *in vitro*. As we are currently developing a new AlphaFold pipeline to identify potential filamentous human TF candidates, our aim is to study their properties in vitro using structural techniques (crystallography and cryo-EM), as well as biophysical techniques (ITC, SEC-MALS, etc.). This combined approach could help us understand the functions of TF filamentation in regulating gene expression and might offer new insights into novel inhibitor targets.

Poster 11

Interplay between mRNA translation and decay in Archaea

Charles CIRIO 1, Christine LAZENNEC-SCHURDEVIN **1**, Gabrielle BOURGEOIS **1**, Clément MADRU **1**, Régine CAPEYROU **2**, Marta KWAPISZ **2**, Marie BOUVIER **2**, Béatrice CLOUET-DORVAL **2**, Yves MECHULAM **1**, Emmanuelle SCHMITT **1**

1 Laboratoire de Biologie Structurale de la Cellule (BIOC), CNRS, Ecole polytechnique, Institut Polytechnique de Paris, Palaiseau, 91120, France
2 MCD, Centre de Biologie Intégrative (CBI), CNRS, Université de Toulouse, UT3, Toulouse, 31000, France

Archaea are critical research models for gaining insights into the origin of the eukaryotic cell and the evolution of life, as well as for studying biogeochemical processes (1). Post-transcriptional regulation of gene expression requires accurate and timely RNA processing and decay to ensure coordinated cellular behaviors and fate decisions. Therefore, understanding RNA metabolic pathways and identifying RNA processing machineries are major challenges in RNA biology. Currently, the best-understood RNA-dedicated pathways at the molecular level are those of Bacteria and Eukarya. In contrast, in Archaea, these molecular processes have been overlooked and are far from being understood.

Proteomic landscapes, direct protein–protein interaction analyses and phylogenomic data support that aRNase J, a 5'-3' exoribonuclease of the β CASP family conserved in Euryarchaeota, engages specifically with a Ski2-like helicase and the RNA exosome to potentially exert control over RNA surveillance, at the vicinity of the ribosome (2). In Eukarya and Bacteria, a general common feature is that mRNA decay and translation are closely coordinated. (3, 4). While aRNase J homologs are found in bacteria, the RNA exosome and the Ski2-like RNA helicase have eukaryotic homologs, underlining the mosaic aspect of archaeal RNA machines.

Our current work aims to study the aRNaseJ/Ski2-like helicase/exosome/ribosome complex by cryo-EM after *in vitro* reconstitution.

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Keywords: Archaea, RNA, Decay, Translation, Cryo-EM

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Participants

Last Name	First Name	Email	Affiliation
AFENSISS	Sana	sana.afensiss@polytechnique.edu	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
AKCAN	Enver Kamil	enverkamilakcan@gmail.com	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
ALLEAUME-BUTAUX	Aurélie	aurelie.alleaume-butaux@polytechnique.edu	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
ALLEMAND	Florentin	florentin.allemand@hotmail.fr	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
ANDREANI	Jessica	jessica.andreani@cea.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
ARTENI	Ana Andreea	ana-andreea.arteni@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
BACONNAIS	Sonia	sonia.baconnais@gustaveroussy.fr	Institut Gustave Roussy - Villejuif
BALANEC	Mael	mael.balanec@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
BARRETEAU	Hélène	helene.barreteau@universite-paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
BEKKAR	Carole	carole.bekkar@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
BERTIN	Aurélie	aurelie.bertin@curie.fr	Institut Curie/Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
BERTRAND	Thomas	thomas.bertrand@sanofi.com	Sanofi - Vitry sur Seine
BESWICK	Veronica	veronica.beswick@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
BONVOISIN	Alice	alice.bonvoisin@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
BOURAND-PLANTEFOL	Alexa	alexa.bourand-plantefol@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
BOURGEOIS	Gabrielle	gabrielle.bourgeois@polytechnique.edu	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
BOUSMAH	Yasmina	yasmina.bousmah@universite-paris-saclay.fr	Institut de Chimie Physique (ICP) - CNRS/Univ. Paris-Saclay - Orsay
BRESSANELLI	Stéphane	stephane.bressanelli@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
BRUNET-PREVOT	Heloise	heloise.brunet-prevot@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
BRUXELLES	Tatiana	tatiana.bruxelles@polytechnique.edu	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
CAI	Hoang Hung	hoanghung.cai@univ-evry.fr	Laboratoire de Structure et activité des biomolecules normales et pathologiques (SABNP) - Univ. Evry Val d'Essonne - Evry
CARRE MITMA	Lucia	lucia.carre-mitma@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
CHANVIN	Marine	marine.chanvin@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette

Last Name	First Name	Email	Affiliation
CHARBONNIER	Jean-Baptise	jb.charbonnier@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/ Yvette
CIRIO	Charles	charlescirio@gmail.com	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
CLOAREC	Camille	camille.cloarec@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
COLLINET	Bruno	bruno.collinet@sorbonne-universite.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/ Yvette
COMBET	Sophie	sophie.combet@cea.fr	Laboratoire Léon-Brillouin (LLB) - CEA - Saclay
COSTE	Franck	franck.coste@cnrs-orleans.fr	Centre de Biophysique Moléculaire (CBM) - CNRS - Orléans
COUREUX	Pierre-Damien	pierre-damien.coureux@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/ Yvette
D'ESQUERMES	Diane	diane.desquermes@ibs.fr	Institut de Chimie des Substances Naturelles (ICSN) - CNRS/Univ. Paris-Saclay - Gif/ Yvette
DESLIGNIÈRE	Evolène	evolene.desligniere@cnrs.fr	Institut de Chimie des Substances Naturelles (ICSN) - CNRS/Univ. Paris-Saclay - Gif/ Yvette
DIEUDONNÉ	Thibaud	dieudonne.thibaud@gmail.com	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
DIGRAZIA	Timeo	timeo.digrazia@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
DONG	Jiawei	dongjiawei1994@gmail.com	Laboratoire de Structure et activité des biomolecules normales et pathologiques (SABNP) - Univ. Evry Val d'Essonne - Evry
DOSSOU	Yawa	yawa.dossou@universite-paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/ Yvette
DOUMALIN	Lou-Ann	lou-ann.doumalin@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
EPALLE	Nathan	nathan.epalle@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/ Yvette
FAGART	Jerome	jerome.fagart@polytechnique.edu	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
FENG	Zichen	zichen.feng@universite-paris-saclay.fr	Biomolécules : Conception, Isolement, Synthèse (BioCis) - Univ. Paris-Saclay - Orsay
FERNANDEZ VARELA	Paloma	pfvarela@gmail.com	CNRS
FERRANDEZ	Yann	yann.ferrandez@ens-cachan.fr	LBPA/ENS Paris Saclay
FIEULAINE	Sonia	sonia.fieulaine@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/ Yvette
FLAQUIERE	Nael	nael.flaquiere@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
FOUREL	Clarisse	clarisse.fourel@cnrs.fr	Institut de Chimie des Substances Naturelles (ICSN) - CNRS/Univ. Paris-Saclay - Gif/ Yvette
FOURMY	Dominique	dominique.fourmy@ens-paris-saclay.fr	LBPA ENS-Paris-Saclay
GAILLARD	Thomas	thomas.gaillard@polytechnique.edu	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
GAYEN	Aranyadip	aranyadip.gayen@cnrs-orleans.fr	Centre de Biophysique Moléculaire (CBM) - CNRS - Orléans

Last Name	First Name	Email	Affiliation
GERHALTER	Magdalena	magdalena.gerhalter@polytechnique.edu	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
GIRLEANU	Maria	mari_girleanu@yahoo.com	IRBA
GRAILLE	Marc	marc.graille@polytechnique.edu	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
GUIANVARCH	Dominique	dominique.guianvarch@universite-paris-saclay.fr	Institut de Chimie Moléculaire et des Matériaux d'Orsay (ICMMO) - CNRS/Univ. Paris-Saclay - Orsay
GURA	Sinan	sinan.gura@etu-upsaclay.fr	Etudiant M2 Ingénierie et Chimie des Biomolécules - Univ. Paris-Saclay
HEDDI	Brahim	brahim.heddi@ens-cachan.fr	LBPA ENS-Paris-Saclay
HENRI	Julien	julien.henri@upmc.fr	Biologie Computationnelle Quantitative et Synthétique - Sorbonne Univ. - Paris
HERDERING	Eva	eherdering@ifam.uni-kiel.de	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
HU	Wanwan	wanwan.hu@polytechnique.edu	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
JEHL	François	francois.jehl@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
LABARDE	Audrey	Audrey.Labarde@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
LALUET	Nicolas	nicolas.laluet@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
LE CHEVALIER	Malo	malo.lechevalier@sanofi.com	Sanofi - Vitry sur Seine
LEGENDRE	Maeline	maeline.legendre@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
LEGRAND	Pierre	pierre.legrand@synchrotron-soleil.fr	Synchrotron SOLEIL - Gif/Yvette
LELONG BLAN	Louise	louise.lelong-blan@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
LENOIR	Guillaume	guillaume.lenoir@universite-paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
LESCOP	Ewen	ewen.lescop@cnrs.fr	Institut de Chimie des Substances Naturelles (ICSN) - CNRS/Univ. Paris-Saclay - Gif/Yvette
LESIEUR	Maeli	maeli.lesieur@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
LITSARDAKI	Eleni	eleanalits@gmail.com	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
LLINAS GARCIA	Paola	paola.llinas@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
MADRU	Clément	clement.madru@polytechnique.edu	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
MAHOU	Pierre	pierre.mahou@polytechnique.edu	Laboratoire d'Optique et de Biosciences (LOB) - CNRS/Ecole Polytechnique - Palaiseau
MAILLARD	Emma	emma.maillard@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
MANIÈRE	Xavier	xavier.maniere@exapto.tech	EXAPTO
MARSIN	Stéphanie	stephanie.marsin@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette

Last Name	First Name	Email	Affiliation
MARTINEZ MOREAU	Louise	louise.martinez-moreau@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
MATEO	Astride	astride.mateo@cnrs-orleans.fr	Centre de Biophysique Moléculaire (CBM) - CNRS - Orléans
MAUCLAIRE	Maia	maia.mauclaire@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
MAUFFRET	Olivier	olivier.mauffret@ens-paris-saclay.fr	LBPA ENS-Paris-Saclay
MECHULAM	Yves	yves.mechulam@polytechnique.edu	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
MENETREY	Julie	julie.menetrey@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
MIRON	Simona	simona.miron@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
MISHRA	Aanchal	aanchal.mishra@cnrs-orleans.fr	Centre de Biophysique Moléculaire (CBM) - CNRS - Orléans
MOAL	Gwenaelle	gwenaelle.moal@cea.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
MONTIGNY	Cédric	cedric.montigny@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
MORERA	Solange	solange.morera@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
NAWROTEK	Agata	agata.maalouf@ens-paris-saclay.fr	LBPA ENS-Paris-Saclay
NESSLER	Sylvie	sylvie.nessler@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
NOIRAY	Magali	magali.noiray@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
OCHSENBEIN	Francoise	francoise.ochsenbein@cea.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
OULDALI	Malika	malika.ouldali@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
OYMAKAS	Lara	lara.oymakas@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
PAGÈS	Cyril	pagescyril7@gmail.com	LBPA ENS-Paris-Saclay
PAUMIER	Clémence	clemence.paumier@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
PIERI	Laura	laura.pieri@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
PLANCQUEEL	Stéphane	stephane.plancqueel@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
PONCET	Laetitia	laetitia.gargowitsch@laposte.net	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
PROCACCIA	Jehanne	jehanne.procaccia@gmail.com	Cibles Thérapeutiques et Conception de Médicaments (CiTCoM) - Univ. Paris Cité - Paris
PROVAN	James	james.provan@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
PYLYPENKO	Olena	pylypenk@curie.fr	Institut Curie - Paris

Last Name	First Name	Email	Affiliation
QUEVILLON-CHERUEL	Sophie	sophie.quevillon-cheruel@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
QUIGNOT	Chloé	chloe.quignot@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
RAPISARDA	Chiara	chiara.rapisarda@sanofi.com	Sanofi - Vitry sur Seine
REIS THEVENOT	Rosana	rosana.inaciodosreisthevenot@servier.com	Servier
RENAULT	Louis	louis.renault@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
RIBEIRO	Mathilde	mathilde.ribeiro1@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
RIGAUDIE	Laura	laura.rigaudie@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
ROELENS	Baptiste	baptiste.roelens@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
ROEVES	Wilbur	wilbur.roeves@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
SAINT-GEORGES	Valerie	valerie.saint-georges@sanofi.com	Sanofi - Vitry sur Seine
SALVI	Nicola	nicola.salvi@sanofi.com	Sanofi - Vitry sur Seine
SAMSON	Camille	camille.samson@sanofi.com	Sanofi - Vitry sur Seine
SAUVANET	Cécile	cecile.sauvanet@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
SIZUN	Christina	christina.sizun@cnrs.fr	Institut de Chimie des Substances Naturelles (ICSN) - CNRS/Univ. Paris-Saclay - Gif/Yvette
SOMME	Eliott	eliott.somme@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
SOUFARI	Heddy	heddy.soufari@synchrotron-soleil.fr	Synchrotron SOLEIL - Gif/Yvette
SUSKIEWICZ	Marcin	marcin.suskiewicz@cnrs-orleans.fr	Centre de Biophysique Moléculaire (CBM) - CNRS - Orléans
TABBA	Emessa	emessa.tabba@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
TAY	Laurianne Li En	laurianne-li-en.tay@polytechnique.edu	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
TETTARAVOU	Claire	claire.tettaravou@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
ULRYCK	Nathalie	nathalie.ulryck@polytechnique.edu	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
URVOAS	Agathe	agathe.urvoas@universite-paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
VALERIO-LEPINIEC	Marie	marielle.valerio@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
VAN BRUTZEL	Sophie	sophie.van-brutzel@universite-paris-saclay.fr	Etudiant LDD1 - Univ. Paris-Saclay
VANEGAS ARIAS	Gabriel	gabriel.vanegas-arias@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette
VARRIALE	Valentina	Valentina.varriale@polytechnique.edu	Laboratoire de Biologie Structurale de la Cellule (BIOC) - CNRS/Ecole Polytechnique - Palaiseau
VIGOUROUX	Armelle	armelle.vigouroux@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/Yvette

Last Name	First Name	Email	Affiliation
WALBOTT	Hélène	helene.walbott@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/ Yvette
WEI	Yuexian	yuexian.wei@univ-evry.fr	Laboratoire de Structure et activité des biomolecules normales et pathologiques (SABNP) - Univ. Evry Val d'Essonne - Evry
YIN	Wenlu	WENLUYIN@OUTLOOK.COM	Laboratoire d'Optique et de Biosciences (LOB) - CNRS/Ecole Polytechnique - Palaiseau
YOSHIZAWA	Satoko	satoko.yoshizawa@ens-paris-saclay.fr	LBPA ENS-Paris-Saclay
ZARGARIAN	Loussiné	loussine.zargarian@ens-paris-saclay.fr	LBPA ENS-Paris-Saclay
ZEA	Diego Javier	diegozea@gmail.com	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/ Yvette
ZHENG	Haoyang	haoyang.zheng@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/ Yvette
ZIMMECK	Marcel	marcel.zimmeck@i2bc.paris-saclay.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/ Yvette
ZINN-JUSTIN	Sophie	sophie.zinn@cea.fr	Institut de Biologie Intégrative de la Cellule (I2BC) - CNRS/Univ. Paris-Saclay - Gif/ Yvette