2026 EDAYS

06
07
MAY

Doctoral School
of Life Health Sciences
– University of Strasbourg

Doctoral School Days

ABSTRACT BOOK

Sponsorisé par

More informations :

Contact the organizing committee
Gur Lauriane, Pouenat Clementine, Bala Krishnan Rakhshana, Battaglia Filippo, Sieber Matthieu, Heimberger Kyllian
Or send a message on our LinkedIn (Doctoral School Days of Strasbourg)

PROGRAM

2026 ED DAYS
Event Timetable

8:45
Arrival & registration
Badge pick-up, participant check-in, speaker welcome

9:00
Opening adresses
Caroline Habold (Director, ED414)
Frédéric Gros (Deputy Director, ED414)
ED Days 2026 organizing committee

9:20
Inspiring talk 1
Stéphanie Blandin, PhD – "At the crossroads of immunology and mosquitoes"
Immune responses in vector mosquitoes — IBMC

10:10
Coffee break

10:30
Long Talk session 1
7 talks (10 min + 5 min Q&A)

10:30 – 10:45 · LT1 – "Unraveling leukemogenesis and drug resistance mechanism of CDX2/ UBTF B-Acute Lymphoblastic Leukemia (B-ALL)" – Guillaume Goncalves - CRBS

10:45 – 11:00 · LT2 – "Ikaros and RORγt : It takes two to control the cell fate decisions of Th17 cells" – Camille Hollinger – IGBMC

11:00 – 11:15 · LT3 – "Insights into the altered expression of the co-inhibitory receptor BTLA in lupus cell subsets" – Melanie Sayah – IBMC

11:15 – 11:30 · LT4 – "Too Hot for Penguins ? Risks and Consequences of Heat Stress in Two Penguin Species" – Leo Marcouillier - EPE

11:30 – 11:45 · LT5 – "Regulation of the Relish-dependent transcriptional output of STING vs IMD signaling in Drosophila" – Taima Lorentzen – M3i

11:45 – 12:00 · LT6– "Decoding the Function of the Enigmatic Proteins Nazo and C19ORF12 in Virus Control and Organelle Biology" – Ananya Aravind – IGBMC

12:00 – 12:15 · LT7– Submicroscopic malaria infections and inadequate IPTp-SP coverage in pregnant women in rural Burkina Faso: a prospective cohort study" – Yssimini Nadege Guillene Tibiri – PHAVI

12:15-14:00
Lunch Posters - Lunch break + Poster Session

14:00
Blitz Talk
6 talks (3 min + 2 min Q&A)

14:00 – 14:05 · BT1 – "Discovery of HBV cccDNA Host Factors as Antiviral Targets for a Cure" – Christos Satratzemis – ITM

14:05 – 14:10 · BT2 – "Characterization of the HIV-1 Vif protein interactome: identification of cellular RNA targets" – Layla Tajer – IBMC

14:10 – 14:15 · BT3 – "Olfactory impairment at prodromal stage predicts cognitive decline in dementia with Lewy bodies and Alzheimer's disease: a clinical and neuroanatomical study" – Maria Fiori – IMIS

14:15 – 14:20 · BT4 – "Tumor cell mechanics modulate neutrophil activation without altering interaction during dissemination" – Elodie Marchal – CRBS

14:20 – 14:25 · BT5 – "DNA's great escape: how damaged ribosomal genes leave home for repair" – Priyanka Pundir – IGBMC

14:25 – 14:30 · BT6 – "Deciphering the target selection mechanisms of the Arabidopsis endoribonuclease DNE1" – Paulo Eberhardt – IBMP

14:30
Long Talk session 2
4 talks (10 min + 5 min Q&A)

14:30 – 14:45 · LT8 – "Combined intensive resistance and endurance exercise in low-disease activity inflammatory myopathies : A Hospital vs. Community -Based prospective study" – Quentin Toumazeau – CRBS

14:45 – 15:00 · LT9 – "Functional characterization of mS77 in plant mitochondrial translation initiation" – Sana Afifah Wigati – IBMP

15:00 – 15:15 · LT10 – "Post-translational regulation of AGO1, a key effector of RNA silencing in Arabidopsis thaliana" – Agathe Vercelletto – IBMP

15:15 – 15:30 · LT11– "Impact of the tVTA on motor and non-motor symptoms in a murine model of Parkinson's disease" – Lucie Mazé – INCI

15:30
Extended coffee break + Poster Session

16:30
Long talks - Blitz Talks
Mixed session

16:30 – 16:45 · LT12 "Development of protease inhibitory aptamers using ultrahigh-throughput screening" – Eleonore Moittie – IBMC

16:45 – 17:00 · LT13 "Aptamer-mediated selective and modulable siRNA delivery" – Lea Denechere – LBP

17:00 – 17:15 · LT14 "A story of a frog, a fungus and some bacteria: Siderophore mediated colonisation resistance against chytridiomycosis" – Christos Paschalidis – ESBS

17:15 – 17:20 · BT7"Microbiota alterations induced by intermittent vs continuous sucrose intake in mice" – Jeanne Tyrode – LNCA

17:20
Social
Drinks & quiz game - Aperitif · coffee · snacks

18:00
Social Evening
Departure for Croque Bedaine — social dinner

20 26 EDAYS

Event Timetable

9:00

Inspiring Talk 2

Jill Pilet, PhD – "Between Passion and Doubt: The Journey of a Researcher in the Making"
Childhood Oncology Research Unit –Childhood Oncology Research Unit — Institut Curie, Paris

9:55

Industry Talk

Antinéa Babarit, PhD
Technical sales engineer — Macherey-Nagel

10:30

Coffee break

11:00

Round table
Gender-based and sexual violence in academia
With doctoral students, researchers and Femmes & Sciences association

12:00-14:00

Award ceremony, attendance sign-off & lunch
Best long talk · best blitz talk · best poster · closing remarks

Thanks !

LONG TALK

Long Talk

Session 1

10:30 – 10:45 · LT1 – "Unraveling leukemogenesis and drug resistance mechanism of CDX2/UBTF B-Acute Lymphoblastic Leukemia (B-ALL)" – Guillaume Goncalves - CRBS

10:45 – 11:00 · LT2 – "Ikaros and RORγt : It takes two to control the cell fate decisions of Th17 cells" – Camille Hollinger – IGBMC

11:00 – 11:15 · LT3 – "Insights into the altered expression of the co-inhibitory receptor BTLA in lupus cell subsets" – Melanie Sayah – IBMC

11:15 – 11:30 · LT4 – "Too Hot for Penguins ? Risks and Consequences of Heat Stress in Two Penguin Species" – Leo Marcouillier - EPE

11:30 – 11:45 · LT5 – "Regulation of the Relish-dependent transcriptional output of STING vs IMD signaling in Drosophila" – Taima Lorentzen – M3i

11:45 – 12:00 · LT6– "Decoding the Function of the Enigmatic Proteins Nazo and C19ORF12 in Virus Control and Organelle Biology" – Ananya Aravind – IGBMC

12:00 – 12:15 · LT7– Submicroscopic malaria infections and inadequate IPTp-SP coverage in pregnant women in rural Burkina Faso: a prospective cohort study" – Yssimini Nadege Guillene Tibiri – PHAVI

Session 2

14:30 – 14:45 · LT8 – "Combined intensive resistance and endurance exercise in low-disease activity inflammatory myopathies : A Hospital vs. Community -Based prospective study" – Quentin Toumazeau – CRBS

14:45 – 15:00 · LT9 – "Functional characterization of mS77 in plant mitochondrial translation initiation" – Sana Afifah Wigati – IBMP

15:00 – 15:15 · LT10 – "Post-translational regulation of AGO1, a key effector of RNA silencing in Arabidopsis thaliana" – Agathe Vercelletto – IBMP

15:15 – 15:30 · LT11– "Impact of the tVTA on motor and non-motor symptoms in a murine model of Parkinson's disease" – Lucie Mazé – INCI

Mixed session

16:30 – 16:45 · LT12 "Development of protease inhibitory aptamers using ultrahigh-throughput screening" – Eleonore Moittie – IBMC

16:45 – 17:00 · LT13 "Aptamer-mediated selective and modulable siRNA delivery" – Lea Denechere – LBP

17:00 – 17:15 · LT14 "A story of a frog, a fungus and some bacteria: Siderophore mediated colonisation resistance against chytridiomycosis" – Christos Paschalidis – ESBS

Long Talk n°1: Unraveling leukemogenesis and drug resistance mechanism of CDX2/UBTF B-Acute Lymphoblastic Leukemia (B-ALL)

Guillaume Goncalves[1], Alicia Perrin[2], Lauriane Gur[1], Hugo Bergugnat[3], Isabelle Duluc[1], Elisabeth Martin, Claudine Ebel, Laetitia Paulen[1], Emmanuelle Clappier[3], Jacky Goetz[1], Jean-Noel Freund, and Claire Domon-Dell[1]

[1]Immuno-Rhumatologie Moléculaire, INSERM U1109, Tumor Biomechanics Lab, Université de Strasbourg, Institut National de la Santé et de la Recherche Médicale, Université de Strasbourg, France
[2]IGBMC, Illkirch, CNRS UMR 7104, INSERM U1258, Université de Strasbourg, Illkirch, France
[3]Institut de Recherche Saint Louis, Institut National de la Santé et de la Recherche Médicale, Université Paris Cité, France

Abstract

Whereas B Acute Lymphoblastic Leukemia (B-ALL) prognosis has been improved for children, it remains poor for young adults due to a high relapse rate. We described the CDX2/UBTF B-ALL subgroup characterized by a specific transcriptomic profile, the massive ectopic expression of the homeotic transcription factor CDX2 and the expression of a newly identified fusion protein named UBTF::ATXN7L3. This unique and systematic coincidence of the two proteins suggests their involvement in the oncogenesis processes. UBTF and ATXN7L3 are both regulating transcription of rRNA and mRNA. CDX2 exerts a tumor suppressor function in intestine but transforms murine hematopoietic stem cells (HSC) toward Acute Myeloid Leukemia. Its oncogenic function in lymphoid lineage remains unknown. We aim to determine the consequences of CDX2 expression in B progenitors and to understand UBTF::ATXN7L3 function. Thanks to original conditional cre/lox mouse models, co-expression of CDX2 and KRASG12D in pre-pro B progenitors led to premature death and blocked lymphoid determination toward immature myeloid phenotype. When CDX2 and KRASG12D were expressed in pro-B cells, a B-ALL-like disorder arose with blockage of immature transformed pro-B cells and a reduced survival. In parallel, we revealed a punctiform nuclear localization of UBTF::ATXN7L3, its interaction with the deubiquitinase USP22 and in consequence, the global decrease of H2B deubiquitination. Overall, our results identify a CDX2 oncogenic function in B cell lineage in mutated KRAS context and suggest an epigenetic reprogramming orchestrated by UBTF::ATXN7L3. Whether and how these two proteins cooperate remains to be explored by modeling their co-expression in pro-B progenitors in mice.

Keywords: B Acute Lymphoblastic Leukemia, CDX2, UBTF::ATXN7L3, Animal model

Long Talk n°2: Ikaros and RORγt: It takes two to control the cell fate decisions of Th17 cells

Camille Hollinger[1]

[1]Institut de Génétique et de Biologie Moléculaire et Cellulaire, Institut National de la Santé et de la Recherche Médicale, Centre National de la Recherche Scientifique, U964, UMR7104, U1258, Université de Strasbourg

Abstract

During a response to bacterial and fungal infections, conventional Th17 cells (cTh17) secrete IL-17, a pro-inflammatory cytokine essential to maintain tissue homeostasis. However, in autoimmune diseases, some subset of Th17 cells acquire a pathogenic (pTh17) profile characterized by lower secretion of IL-17 along with enhanced production of GM-CSF that drives an intense inflammation. Surprisingly, the lineage-defining transcription factor RORγt, which is essential for IL-17 expression is well expressed in both Th17 cells subsets, which suggest that additional factor control Th17 polarization. Our lab recently showed that the transcription factor Ikaros represses the pTh17 signature thereby supporting cTh17 identity (Bernardi et al. 2021). However the mechanism underlying this cell fate decision remains unclear. Using a tamoxifen inducible deletion of Ikaros mouse model, we show that both Ikaros and RORγt bind promoters of cTh17-related genes, suggesting that they functionally cooperate. Strikingly, we show that Ikaros physically interacts with RORγt and that, in the absence of Ikaros, RORγt loses access to cTh17 targets while binding novel sites, thereby reshaping its transcriptional program. Our findings uncover a previously unrecognized mechanism whereby Ikaros stabilizes RORγt activity to ensure effective cTh17 polarization, providing new insights into immune regulation and autoimmune pathology.

Keywords: Th17 cells, Inflammation, Ikaros, RORγt, IL, 17, gene regulation

Long Talk n°3: Insights into the altered expression of the co-inhibitory receptor BTLA in lupus cell subsets

Mélanie Sayah[1], Fanny Monneaux[1]

[1]Immunologie, Immunopathologie et Chimie Thérapeutique, Institut de biologie moléculaire et cellulaire, Université de Strasbourg, Centre National de la Recherche Scientifique

Abstract

Co-inhibitory receptors are essential for maintaining immune homeostasis and preventing autoimmune diseases such as systemic lupus erythematosus (SLE). SLE is characterized by the production of autoantibodies leading to immune complex deposition in vital organs and severe complications. B and T lymphocyte attenuator (BTLA) is a co-inhibitory receptor expressed on various immune cells. BTLA-deficient lupus mice exhibit exacerbated disease, revealing its protective role in SLE. Our team previously reported altered BTLA expression in activated regulatory T cells and in double negative B cells from lupus patients. We also showed that administration of an agonistic anti-BTLA antibody in lupus-prone mice had therapeutic effects, highlighting BTLA as a promising target for similar strategies in humans. However, its impaired expression in SLE remains a challenge. Restoring BTLA expression prior to targeting is therefore crucial. My project aims to elucidate the mechanisms regulating BTLA surface expression. Cytokines are of particular interest due to their central role in SLE and elevated levels in patient sera. My results show that in $CD4^+$ T cells, IL-6 and TNF-α seem to enhance BTLA expression, whereas IFN-α reduces it. In B cells, IL-6 and IL-21 tend to increase BTLA expression, while IFN-α and TNF-α decrease it. Furthermore, sera from SLE patients significantly increase BTLA expression in B and $CD4^+$ T cells, an effect reversed by neutralizing anti-cytokine antibodies. These findings demonstrate that cytokines elevated in SLE modulate BTLA expression and suggest that therapeutically targeting these cytokines could help normalize BTLA expression prior to its targeting.

Keywords: Autoimmunity, Systemic lupus erythematosus, inhibitory receptors, B and T lymphocyte attenuator, cytokines

Long Talk n°4: Too hot for Penguins? Risks and Consequences of Heat Stress in Two Penguin Species.

Léo Marcouillier[1], Aude Noiret[1], Elsa Marçon[2,3], Norith Eckbo, Zohria-Lys Guillerm, Samuel Laporte, Colline Richard[4,5], Thierry Raclot[6], Etienne Chalet, Frederic Angelier[7], Akiko Kato[8], Yan Ropert-Coudert[9], Agnès Lewden[10,11,12], Antoine Stier[13,14]

[1,2]Département Ecologie, Physiologie et Ethologie, Institut Pluridisciplinaire Hubert Curien, Université de Strasbourg, CNRS
[3]Bureau d'études Akongo Bureau d'études Akongo
[4]Département Ecologie, Physiologie et Ethologie, Université de Strasbourg, CNRS, IPHC UMR 7178, 67000 Strasbourg, France
[5]Master de Biologie, École Normale Supérieure de Lyon, Université Claude Bernard Lyon 1, Université de Lyon, 69342 Lyon Cedex 07, France
[6]Institut Pluridisciplinaire Hubert Curien, UMR7178, CNRS, Université de Strasbourg, CNRS
[7]CEBC CNRS UMR 7372, CNRS UMR 7372, Centre d'Études Biologiques de Chizé, Université de La Rochelle, 79360 Villiers-en-Bois, France
[8]Centre d'Etudes Biologiques de Chize (CEBC UMR7372), Centre National de la Recherche Scientifique 79360 Villiers-en-Bois, France
[9]Centre d'Etudes Biologiques de Chizé, UMR 7372 CNRS, 79170, Villiers en Bois, France
Institut Universitaire Européen de la Mer (IUEM), Institut de Recherche pour le
[10]Développement, Institut National des Sciences de l'Univers, Université de Brest, Centre National de la Recherche Scientifique
[11]ISblue, ISblue
[12]Université de Bretagne Occidentale - UFR Sciences et Techniques (UBO UFR ST), Université de Brest
[13]Institut Pluridisciplinaire Hubert Curien, Centre National de la Recherche Scientifique, UMR7178, université de Strasbourg
[14]University of Turku, FI-20014 Turun yliopisto, Finlande

Abstract

Polar and subpolar endothermic species, adapted to cold climates, may be particularly sensitive to heat stress, especially in the context of accelerated global warming at these latitudes. Penguins, for example, possess particularly effective thermal insulation that may limit their ability to dissipate excess heat. During the breeding season, access to water for king penguins (Aptenodytes patagonicus) and Adélie penguins (Pygoscelis adeliae) is limited because they must remain on land to incubate their eggs or protect their chicks. This situation effectively reduces their ability to regulate their body temperature through behavioral responses (seeking shade, entering the water, exposing the brood pouch) for periods that can last several weeks. Despite these various risk factors, few studies have focused on the physiological and reproductive effects of heat on penguins during their breeding season on land. Our objectives therefore focus on 1) detecting behavioral responses related to heat stress using accelerometry and direct observation, and 2) modeling body temperature as a function of climatic parameters (air temperature, wind speed, solar radiation, humidity) and intrinsic factors (species, circadian cycle, fasting stage, reproductive stage). This will allow us to better understand the environmental conditions that induce heat stress in two species living in contrasting climates, their ability to thermoregulate, and the consequences of a potential inability to thermoregulate effectively on their physiology and reproduction.

Keywords: Heat stress, Ecophysiology, Polar ecology, Penguin

Long Talk n°5: Regulation of the Relish-dependent transcriptional output of STING vs IMD signaling in Drosophila

Taima Lorentzen[1]

[1]Université de Strasbourg, Modèles Insectes Immunité Inée (M3i), CNRS/UPR9022

Abstract

A constant arms race between host and virus has shaped the innate immunity system of all living organisms, resulting in a diverse range of anti-viral responses. Some pathways are conserved across evolution, such as the STING pathway, which regulates the NF-kB response and IFN responses in humans (Ablasser and Chen, 2019). The STING pathway also exists in Drosophila (Cai et al., 2020, 2023) (Goto et al., 2018). My lab has recently shown that the STING pathway actually shares a lot of common factors with an anti-bacterial innate immune pathway, the Immunodeficency (IMD) pathway. Indeed, one pathway is activated by viral RNA and the other by Gram- bacteria, but both converge towards activation and cleavage of a common transcription factor, Relish. Relish translocates to the nucleus and initiates either the transcription of the STING regulated genes, or antimicrobial peptides. I hope the uncover what element is key to differentiate both transcriptional responses. I started to work on the Charon protein, as its expression is triggered by the STING pathway. Furthermore, Charon has been reported to interact with Relish in two papers that have opposing conclusions concerning Charon's regulatory role within the IMD pathway. On another hand. Relish is known to be phosphorylated in the IMD pathway, and the same kinase responsible for this is necessary for the STING pathway. However, we have not yet shown if Relish is subject to phosphorylation in the STING pathway as well. Both aspects could be key to understand the difference between both pathways.

Keywords: STING, anti, viral, IMD, immunology, gene regulation

Long Talk n°6: Decoding the Function of the Enigmatic Proteins Nazo and C19ORF12 in Virus Control and Organelle Biology

Ananya Aravind[1,2], Fabien Alpy[1], Carine Meignin[2], Jean-Luc Imler[2], Catherine Tomasetto[1]

[1]Institut de Génétique et de Biologie Moléculaire et Cellulaire, université de Strasbourg, Institut National de la Santé et de la Recherche Médicale, U964, UMR7104, U1258
[2]Modèles Insectes de l'Immunité Innée, Institut de biologie moléculaire et cellulaire, université de Strasbourg, Centre National de la Recherche Scientifique, Institut National de la Santé et de la Recherche Médicale

Abstract

Nazo and C19ORF12 are evolutionarily conserved proteins independently identified for their involvement in antiviral immunity in Drosophila melanogaster and neurodegenerative disease in humans, respectively. Nazo functions as an antiviral effector in Drosophila melanogaster, as its inhibition leads to increased Drosophila C virus viral levels. In humans, loss-of-function mutations in the Nazo homolog C19ORF12 gene causes neurodegeneration with brain iron accumulation (NBIA) characterized by iron deposition in the basal ganglia. Despite these distinct physiological contexts, studies in both Drosophila and mammalian systems suggest that Nazo and C19ORF12 converge on a shared role in lipid metabolism and organelle homeostasis. The goal of my project is to bridge findings from flies and humans by defining a unifying molecular framework that links Nazo and C19ORF12 to antiviral immunity and cellular organelle biology. To this end, I have generated C19ORF12 knockout MRC5 lung fibroblast cell lines to investigate its function in innate immune signalling and lipid metabolic pathways. Preliminary data indicate that, in contrast to Nazo in Drosophila, C19ORF12 is not transcriptionally induced by canonical innate immune pathways in MRC5 cells. To investigate whether C19ORF12 contributes to antiviral defense, C19ORF12 knockout cell line are being complemented with wild-type and disease-associated mutant C19ORF12, as well as crossspecies complementation using Drosophila Nazo. These approaches are being used to assess functional conservation and to determine whether Nazo and C19ORF12 modulate antiviral responses through their roles in organelle biology and lipid metabolic pathways.

Keywords: Viral linfection, Innate Immunity, Antiviral Response, Drosophila, Evolution

Long Talk n°7: Submicroscopic malaria infections and inadequate IPTp-SP coverage in pregnant women in rural Burkina Faso: a prospective cohort study

Yssimini Nadège Guillène Tibiri[1]

[1]Pathogens Host Arthropod Vectors Interfaces, université de Strasbourg

Abstract

Malaria in pregnancy remains a major cause of maternal and neonatal morbidity in sub-Saharan Africa. Intermittent preventive treatment with sulfadoxine-pyrimethamine (IPTp-SP), delivered presumptively at antenatal care (ANC) visits reduces malaria-associated adverse outcomes including low birthweight (LBW). However, routine diagnostics miss low-density infections and IPTp-SP coverage remains suboptimal in many endemic settings. We conducted a prospective cohort study at Koubri health center, Burkina Faso. Women were enrolled at their first ANC visit, where Plasmodium infection was assessed by real-time-PCR, microscopy, and RDT. We used Poisson regression to identify predictors of PCR-confirmed infection at first ANC visit and factors associated with LBW. In the subset of 183 women with all three tests available, prevalence was 23.5% by PCR versus 8.2% by microscopy and 8.7% by RDT, with approximately 2/3 of PCR-confirmed infections undetected by routine diagnostics. Primigravidae had twice the infection risk of multigravidae, and women living more than 5 km from the facility had higher risk. Each additional IPTp-SP dose was associated with an 11% reduction in PCR-confirmed infection prevalence during pregnancy. PCR-confirmed infection at first ANC was associated with a 52% higher risk of LBW. Pfdhfr/Pfdhps genotyping shows the 540E and 581G mutations, associated with SP treatment failure, were each detected in only 3.4% of isolates, suggesting that SP resistance is not yet a primary driver of prophylaxis failure in this setting. Routine IPTp-SP retained a measurable protective effect under routine conditions. The residual burden of malaria in pregnancy is driven more by implementation failures than by drug failure.

Keywords: Malaria, TPIg, SP, CPN, PCR, low birth weight (LBW)

Long Talk n°8: Combined intensive resistance and endurance exercise in low-disease activity inflammatory myopathies: A Hospital vs. Community-Based prospective study

Quentin Toumazeau[1], Lea Debrut[1,2], Pauline Asael, Alizée Heit, Anne-Laure Charles, Bernard Geny, Margherita Giannini[3], Alain Meyer[3]

[1]UR3072, Centre de Recherche en Biomédecine de Strasbourg (CRBS), Fédération de Médecine Translationnelle, université de Strasbourg
[2]Faculty of Sport Sciences, European Centre for Education, Research and Innovation in Exercise Physiology (CEERIPE), University of Strasbourg, France
[3]Physiology and Muscle Function Explorations Department, Referral Centre for Systemic Rare Autoimmune Diseases, Strasbourg University Hospitals, Strasbourg, France, Les Hôpitaux Universitaires de Strasbourg (HUS)

Abstract

Inflammatory myopathies (IM) are autoimmune diseases characterized by chronic skeletal muscle inflammation and weakness. Many IM patients feature persistent impairment in exercise capacity despite treatments wich is associated with fatigue, disability and mortality. Combined resistance and endurance exercise (REE) has been reported to improve exercise capacity. However, i) these results have not been replicated, ii) was conducted in a hospital setting limiting patients access to the care, iii) the mechanism underlying the efficacy of exercise in IM has been scarcely investigated. This study aimed to validate the efficacy of REE in patients with low disease activity IM, to compare hospital-based and community-based programs and explore the underlying mechanisms. Eighty patients with IM were prospectively included for a 36-session REE. The program consisted of 30min of aerobic exercise and 20min of resistance exercise, 3 sessions per week for 12 weeks with evaluations conducted before and after the intervention. Participants <50km from the hospital trained onsite (HB); others used local physiotherapists (CB). Twenty-five patients completed the protocol. VO_2max increased after both the HB and CB REE. Strength, 6-min walk distance, were similarly improved in both groups. Perceived fatigue decreased in the HB as well as in the CB. PBMCs mitochondrial respiration improved as shown by complex IV activity. In both settings, REE is feasible, safe and effective for low disease activity IM patients. Improvement of exercise capacity, muscle strength, disability, and fatigue were associated with an improvement in mitochondrial respiratory capacity that may play a role in the efficacy of this non-pharmacological treatment.

Keywords: Physical therapy, Physiotherapy and Physical Activity, Non-pharmacological interventions

Long Talk n°9: Functional characterization of mS77 in plant mitochondrial translation initiation

Sana Afifah Wigati[1], Vasileios Skaltsogiannis[1], Mathilde Arrivé[1], Benoît Castandet[1], Hakim Mireau[2], Philippe Giegé[1]

[1]Institut de biologie moléculaire des plantes, université de Strasbourg, Centre National de la Recherche Scientifique, UPR2357, Centre National de la Recherche Scientifique
[2]Institut Jean-Pierre Bourgin, Sciences du végétal, AgroParisTech, Université Paris-Saclay, Institut National de Recherche pour l'Agriculture, l'Alimentation et l'Environnement

Abstract

Mitochondria were acquired by eukaryotes through endosymbiosis of a bacterium related to alphaproteobacteria. Despite their bacterial origin, they have evolved into semi-autonomous organelles hosting ATP production and various metabolic pathways, while having their own genome and gene expression machineries that contain eukaryote-specific traits. Mitochondrial ribosomes (mitoribosomes) have highly diverged compared to those of bacteria and evolved differently across different eukaryotic groups. Previous biochemical and structural studies of plant mitoribosome revealed its increase in both RNA and protein content, compared to those of bacteria. A novel mechanism of translation initiation was hypothesized using mS77, a ribosomal pentatricopeptide repeat (PPR) protein located in the mRNA exit channel, by binding an A-rich motif found in the 5'UTRs of plant mitochondrial mRNAs, both located at a similar distance from the decoding center and start codon, respectively, as a means to position the start codon in the A-site. Hereby, we aim to characterize the involvement of mS77 in this process through two main approaches: (i) reverse genetic approaches through depletion or modification of the protein to disrupt the putative interaction and observe their effect on translation and (ii) in vitro binding assays between mS77 and mRNAs to assess the affinity. Preliminary results suggest that mS77 may enhance the interaction of mRNAs with the ribosome in the presence of the A-rich motif, although other protein factors are likely involved especially for the mRNA recruitment. In the long term, this study will contribute to our understanding of the diversity of translation initiation processes in mitochondria.

Keywords: Mitochondrial translation, translation initiation, PPR proteins, mitochondrial gene expression, organellar gene expression

Long Talk n°10: post-translational regulation of AGO1, a key effector of RNA silencing in Arabidopsis thaliana.

Agathe Vercelletto[1], Esther Lechner[1], Pascal Genschik[2]

[1]Institut de Biologie Moléculaire des Plantes, CNRS, université de Strasbourg
[2]Institut de Biologie Moléculaire des Plantes (IBMP), université de Strasbourg, Centre National de la Recherche Scientifique CNRS 12 Rue du général Zimmer 67084 STRASBOURG CEDEX, France

Abstract

In plants, the homeostasis of AGO1, a key RNA silencing protein, is crucial for development. Previous findings in the laboratory showed that AGO1, and more specifically its unloaded form, is regulated at the post-translational level by the ubiquitin ligase SCF-FBW2 (Hacquard et al. 2022). However, the degradation pathway involved still needs to be characterized. A pharmacological approach suggests that autophagy is involved in this process. The aim of my project is to confirm this role and identify the specific autophagy receptor (SAR) involved. The role of autophagy will be investigated using a genetic approach, studying the degradation of AGO1 following FBW2 induction in autophagy-deficient lines. Additionally, AGO1 delivery to the vacuole will be investigated using microscopy with Arabidopsis transgenic lines expressing pAGO1:GFP-AGO1 and XVE:FBW2 under a beta-estradiol-inducible promoter, as well as mCherry ATG8, a marker for the autophagosome. Two SARs that could be involved in FBW2-mediated AGO1 degradation have been identified in the FBW2 interactome. Each candidate is being studied using genetic approaches and interaction assays to determine their involvement in the process. Preliminary results suggest that autophagy is involved in FBW2-mediated AGO1 degradation. The receptors involved are still being characterized.

Keywords: Mi Arabidopsis thaliana, RNA silencing, AGO1, FBW2, Autophagy

Long Talk n°11: Impact of the tVTA on motor and non-motor symptoms in a murine model of Parkinson's disease

Lucie Mazé[1], Benjamin Muller[1], Eva Clerc[1], Stéphane Doridot[1], Laura Harsan[2], Jennifer Kaufling[1], Dominique Ciocca[1], Michel Barrot[1]

[1]Institut des Neurosciences Cellulaires et Intégratives, université de Strasbourg, Centre National de la Recherche Scientifique
[2]ICube, UMR7357, ICube UMR7357, Strasbourg, France

Abstract

Parkinson's disease, characterized by the loss of dopaminergic neurons leading to motor symptoms, also induces deleterious non-motor consequences including pain, mood disorders, and cognitive impairment. In a preclinical hemi-parkinsonian model, co-lesion of a brain region encompassing the tail of the ventral tegmental area (tVTA), a GABAergic structure that notably regulates dopaminergic neurons, alleviates mechanical hypersensitivity and motor deficits. The recent identification of Sox14 as a selective molecular marker of tVTA neurons now enables a more precise investigation of the effects of their pharmacogenetic manipulation in the 6-OHDA Parkinsonian model. The results obtained are promising: acute DREADD-mediated inhibition of the tVTA in Sox14-Cre mice rescues mechanical and thermal hypersensitivity (both heat and cold), as well as cognitive, anxiodepressive, and motor symptoms. These findings demonstrate that acute inhibition of the tVTA is associated with a simultaneous improvement across multiple symptomatic dimensions in this model. Chronic manipulation experiments are currently underway to assess the durability of these effects. In addition, magnetic resonance imaging (MRI) analyses will provide a comprehensive overview of the alterations in brain circuits and interactions between structures underlying the broad beneficial effects observed following tVTA inhibition.

Keywords: Parkinson, 6, hydroxydopamine, tVTA, Pain, Therapeutic strategies

Long Talk n°12: Development of protease inhibitory aptamers using ultrahigh-throughput screening

Eléonore Moittié[1]

[1]Michaël Ryckelynck, STRASBOURG ED414

Abstract

Proteases are enzymes playing key roles in various infectious and pathologic processes such as tissue invasion by pathogens, cancer cells dissemination or inflammatory diseases. Therefore, specific protease inhibitors hold great therapeutic promises. Our lab explores the potential of aptamers (single-stranded oligonucleotides) to be used as a new class of enzyme inhibitory drugs, following the path pioneered by the development of Macugen, the first therapeutic aptamer used to treat Macular degeneration. Indeed, nucleic acid aptamers are promising candidates for the production of therapeutics owing to their high affinity and specificity, comparable to those of antibodies, as well as their low immunogenicity. Aptamers are usually developed using SELEX (Systematic Evolution of Ligand by Exponential enrichment), which is an efficient method for enriching a nucleic acid library in molecules with high affinity for a defined target (i.e. binders). In addition to the SELEX, we use microfluidic-assisted in vitro compartmentalization (µIVC) to functionally screen enriched libraries for the aptamers endowed with good inhibitory capacity. This ultrahigh-throughput method allows to screen larger libraries than microplate screening, in a cost-effective manner. My PhD project is to use this optimized pipeline to select an inhibitory RNA aptamer against the macrophage metalloelastase, also known as MMP12, which plays many roles within the organism (tissue remodeling, elastin degradation, antiviral response...). Importantly, its dysregulation often leads to various diseases, such as Chronic obstructive pulmonary disease (COPD) and emphysema, or Atherosclerosis and cardiovascular disease, which makes it a relevant target for therapy development.

Keywords: RNA, aptamer, protease inhibitor, therapeutics

Long Talk n°13: Aptamer-mediated selective and modulable siRNA delivery

Léa Denechere[1], Charlène D'ancona[1,2], Béni Twendimbadi[1], Sandrine Pelet[1], Sylvie Friant[2], Séverine Bär[2], Mayeul Collot[3], Pierre Fechter[4], Laurence Choulier[1]

[1]Laboratoire de Bioimagerie et Pathologies, université de Strasbourg, Centre National de la Recherche Scientifique
[2]Laboratoire de Génétique Médicale, université de Strasbourg, Institut National de la Santé et de la Recherche Médicale
[3]Chémo-Biologie Synthétique et Thérapeutique, université de Strasbourg, Institut de Chimie, CNRS Chimie, Centre National de la Recherche Scientifique
[4]Biotechnologie et Signalisation Cellulaire, CNRS : UMR7242

Abstract

Small interfering RNAs (siRNAs) are specific and effective molecules for gene silencing. However, their use is limited by poor cell penetration due to their negative charge and their size. To overcome these barriers, the conjugation of siRNAs to ligands targeting cell-surface receptors is a promising approach. Aptamers appear to be interesting candidates since they are single-stranded DNA or RNA with a high affinity and selectivity to a specific target, such as cell-surface receptors. Thus, we aim to develop selective and modulable vehicles associating a siRNA with one or more aptamers called mono- or multivalent aptamer-siRNA chimeras (AsiCs). First, we would like to create a monovalent AsiC using either an RNA or DNA aptamer. We compared two AsiCs formed by an RNA-RNA or RNA-DNA sticky bridge. Our preliminary results show a similar assembly. Second, we designed an innovative multivalent AsiC. Our vehicle combines one siRNA with one to three homo- or heterovalent aptamers. The various elements contain hybridization sequences that will enable controlled self-assembly. Thanks to their versatile nature, the number, position and type of aptamers (DNA or RNA) could be easily changed. So far, we have designed RNA sequences, predicted their 2D structure and checked the vehicle assembly. Our preliminary results of RNA-RNA and RNA-DNA AsiC are encouraging and confirm the feasibility to combine DNA and RNA aptamers in multivalent AsiC, new siRNA active delivery tool with great potential. As perspectives, we wish to deepen characterization of AsiCs (stability and functional cell assays) and study their intracellular traffic by bioimaging.

Keywords: siRNA, aptamer, active targeting delivery, innovative delivery tool, multivalent conjugate, cell surface receptor

Long Talk n°14: A story of a frog, a fungus and some bacteria: Siderophore mediated colonisation resistance against chytridiomycosis

Christos Paschalidis[1], Elliot Murphy[2], Emily Read[3], Fritz Ka-Ho Ho[2] , Marc-Emmanuel Dumas[4,2], Kieran Bates[3], Olivier Cunrath[1]

[1]Ecole Supérieure de Biotechnologie de Strasbourg, Université de Strasbourg, CNRS
[2]Division of Systems Medicine, Department of Metabolism, Digestion and Reproduction, Imperial College London, London, W12 0NN
[3]Queen Mary University of London
[4]Institut Européen de Génomique du Diabète, European Genomic Institute for Diabetes, FR 3508 Institut Pasteur de Lille, Institut National de la Santé et de la Recherche Médicale, Université de Lille

Abstract

Batrachochytrium dendrobatidis (Bd) is a devastating fungal pathogen that has led to catastrophic declines in amphibian populations worldwide, leading to the single biggest extinction crisis in vertebrates. Bd zoospores infect amphibian's skin, disrupting its function and ultimately leading to cardiac arrest and death. One of the main host defence mechanisms is nutritional immunity, wherein the host restricts essential nutrients, such as metals, to impede pathogen growth. In parallel, the skin microbiome plays a pivotal role in shaping the outcome of infection. The skin of amphibians harbours a diverse microbiome, which have been shown to also protect against Bd, notably by producing antifungal peptides. Our research suggests that metal limiting conditions - similar to those encountered during infection - triggers skin bacteria to produce siderophores, specialized iron-chelating molecules. These secondary metabolites, show potent anti-fungal properties, suggesting that they may act in synchrony with host- induced nutritional immunity, providing colonisation resistance against Bd.

Keywords: Batrachochytrium dendrobatidis, colonisation resistance, amphibian skin microbiome, siderophores, metabolomics

BLITZ TALK

BlitzTalk

Session 1

14:00 – 14:05 · BT1 – "Discovery of HBV cccDNA Host Factors as Antiviral Targets for a Cure" – Christos Satratzemis – ITM

14:05 – 14:10 · BT2 – "Characterization of the HIV-1 Vif protein interactome: identification of cellular RNA targets" – Layla Tajer – IBMC

14:10 – 14:15 · BT3 – "Olfactory impairment at prodromal stage predicts cognitive decline in dementia with Lewy bodies and Alzheimer's disease: a clinical and neuroanatomical study" – Maria Fiori – IMIS

14:15 – 14:20 · BT4 – "Tumor cell mechanics modulate neutrophil activation without altering interaction during dissemination" – Elodie Marchal – CRBS

14:20 – 14:25 · BT5 – "DNA's great escape: how damaged ribosomal genes leave home for repair" – Priyanka Pundir – IGBMC

14:25 – 14:30 · BT6 – "Deciphering the target selection mechanisms of the Arabidopsis endoribonuclease DNE1" – Paulo Eberhardt – IBMP

Mixed session

17:15 – 17:20 · BT7 "Microbiota alterations induced by intermittent vs continuous sucrose intake in mice" – Jeanne Tyrode – LNCA

Blitz Talk n°1: Discovery of HBV cccDNA Host Factors as Antiviral Targets for a Cure

Christos Satratzemis[1], Laura Meiss-Heydmann[1], Aurélie Batt[1], Florine Zettl[1], Charlotte Bach[1], Pauline Casetta[1], Eloi Verrier[1,2], and Thomas Baumert[1,3]

[1]Institute for Translational Medicine and Liver Disease, Université de Strasbourg, Institut National de la Santé et de la Recherche Médicale, France
[2]Centre International de Recherche en Infectiologie, Université Claude Bernard Lyon 1, Centre National de la Recherche Scientifique, France
[3]L'Institut hospitalo-universitaire de Strasbourg – Hôpitaux Universitaires de Strasbourg, France

Abstract

Infection with the hepatitis B virus (HBV) causes chronic hepatitis B, affecting approximately 300 million people worldwide and carrying up to a 45% risk of disease progression. Over one million deaths each year are attributed to HBV-related complications, primarily to hepatocellular carcinoma. During infection, HBV hijacks host cell proteins to produce cccDNA (covalently closed circular DNA). Owing to its chromatin-like organization, cccDNA persists in infected hepatocytes and serves as a viral reservoir, driving the development of chronic HBV infection and hepatitis. cccDNA thus represents the main barrier to achieving a cure for chronic hepatitis B. Current therapies suppress viral replication and disease progression but fail to eliminate cccDNA. Consequently, treatment must be administered lifelong and does not prevent severe complications. This PhD project aims to identify host factors involved in cccDNA formation that could be therapeutically targeted. A high-throughput screening strategy based on a cccDNA reporter cell line was used to perform two siRNA screenings targeting cellular kinases and membrane proteins, followed by a secondary screening in a bona fide HBV infection model with a cccDNA-specific readout. This approach enabled the identification of 21 putative cccDNA host factors. Preliminary validation using complementary loss-of-function approaches in relevant cell-based models supports the involvement of two candidates in cccDNA biology, and ongoing work seeks to elucidate their molecular mechanisms. Understanding these virus-host interactions will enable the development of novel therapeutic strategies targeting cccDNA via its host factors and contribute to efforts toward a cure for chronic hepatitis B.

Keywords: HBV, cccDNA, host factors, chronic hepatitis B, novel therapeutic strategies

Blitz Talk n°2: Characterization of the HIV-1 Vif protein interactome: identification of cellular RNA targets

Layla Tajer[1,2], Jason Decotter[1], Benjamin Stupfler[1], Ziad Fajloun[2,3], Serena Bernacchi[1], Roland Marquet[1], Sarah Gallois-Montbrun[4], and Jean-Christophe Paillart[1]

[1]Université de Strasbourg, CNRS, Architecture et Réactivité de l'ARN, UPR 9002, IBMC, 2 Allée Konrad Roentgen, F-67000 Strasbourg, France, Centre National de la Recherche Scientifique, CNRS, France
[2]Laboratory of Applied Biotechnology (LBA3B), Azm Center for Research in Biotechnology and its Application, EDST, Lebanese University, 1300, Tripoli, Lebanon, Liban
[3]Department of Biology, Faculty of Sciences 3, Campus Michel Slayman Ras Maska, Lebanese University, 1352, Tripoli, Lebanon, Liban
[4]Université Paris Cité, INSERM U1016, CNRS UMR8104, Institut Cochin, 27 rue Faubourg Saint-Jacques, F-75014 Paris, France, Centre National de la Recherche Scientifique, CNRS, Institut National de la Santé et de la Recherche Médicale, INSERM, France

Abstract

To successfully infect humans, HIV-1 must overcome several physical and immunological barriers. A key viral strategy involves Vif protein, which plays a critical role in counteracting these host defenses. The interaction between Vif and the cytidine deaminase APOBEC3G (A3G), a potent restriction factor, is a key mechanism by which HIV-1 evades the host's intrinsic immunity. In non-permissive cells, Vif counteracts A3G mainly by promoting its degradation via the ubiquitin–proteasome pathway. In addition, Vif plays a critical role in the inhibition of A3G translation. This repression involves direct interactions between Vif and the 5′ untranslated region of A3G mRNA. Furthermore, Vif has been shown to interact with the viral gRNA both in vitro and ex vivo. These results suggest that Vif may target other cellular mRNAs to modulate their expression and recruit protein partners involved in translational regulation. Based on this hypothesis, this study aims to characterize the RNA interactome of HIV-1 Vif and to identify novel cellular RNA targets potentially involved in Vif-dependent regulatory mechanisms. A transcriptome-wide analysis was previously performed in our laboratory using PAR-CLIP, leading to the identification of multiple RNA candidates. From this dataset, high-priority candidates were selected based on multiple criteria. To validate these candidates, UV cross-linking RNA immunoprecipitation assays were performed. Preliminary results indicate that several cellular RNAs specifically associate with Vif. Notably, HSPA8, SRRM2, and HMGB2 mRNAs were found to be enriched in Vif-containing ribonucleoprotein complexes, supporting their interaction with Vif. These preliminary findings support the existence of a broader Vif-associated RNA interactome.

Keywords: HIV1, Vif, RNA binding protein, UV Crosslinking

Blitz Talk n°3: Olfactory impairment at prodromal stage predicts cognitive decline in dementia with Lewy bodies and Alzheimer's disease: a clinical and neuroanatomical study

Maria Fiori[1], Cécile Pernossi[1], Laura David[1], Olivier Bousiges[1,2], Léa Sanna[3], Candice Muller[3], Alix Ravier[3], Catherin Demuynck[3], Frédéric Blanc[1,3], and Luc Marlier[1]

[1]Imagerie Multimodale Intégrative en Santé, Laboratoire des sciences de l'ingénieur, de l'informatique et de l'imagerie, France
[2]Hôpital de Hautepierre, Hôpitaux Universitaires de Strasbourg, France
[3]Centre Mémoire Ressources et Recherche, Centre Hospitalier Universitaire, France

Abstract

Olfactory dysfunction is a hallmark of dementia with Lewy bodies (DLB) and Alzheimer's disease (AD). However, modality-specific cognitive and subjective profiles, their prognostic value, and neuroanatomical substrates remain poorly characterized. Objective: To investigate higher-order olfactory signatures, their predictive value for longitudinal cognitive decline, and their structural neural bases in early DLB and AD. Methods: Participants with early DLB, early AD, and cognitively unimpaired (CU) controls underwent standardized olfactory assessments (detection thresholds, identification, familiarity, and hedonicity). Longitudinal decline was tracked via the annualized change in Mini-Mental State Examination (ΔMMSE) score. Structural correlates were explored in a neuroimaging sub-sample (n = 62) using whole-brain voxel-based morphometry (VBM). Results: The final cohort included 72 participants (37 DLB, 17 AD, 18 CU). Odor identification was severely impaired in DLB and AD compared to CU. Specifically, DLB patients exhibited a significant reduction in subjective odor familiarity. Preserved baseline identification predicted a slower rate of cognitive decline across both disease cohorts (DLB: $\rho = 0.398$, $p = 0.015$; AD: $\rho = 0.667$, $p = 0.009$). VBM analyses ($p < 0.001$, $k \geq 50$) revealed that in DLB, decreased hedonicity correlated with the right parietal operculum, altered familiarity with right fronto-striatal white matter (WM) degradation, and poorer detection thresholds with reduced WM volume within bilateral cerebellar pathways. Conclusion: Preserved odor identification serves as a prognostic marker for slower global cognitive decline in early DLB and AD. Structural correlates underscore a profound, multi-level central disconnection spanning affective, implicit semantic, and attentional-predictive brain regions in DLB.

Keywords: Alzheimer's disease, Dementia with Lewy bodies, Magnetic resonance imaging, Olfaction

Blitz Talk n°4: Tumor cell mechanics modulate neutrophil activation without altering interaction during dissemination

Elodie Marchal[1]

[1]Immuno-Rhumatologie Moléculaire, Université de Strasbourg, Institut National de la Santé et de la Recherche Médicale, France

Abstract

The mechanical properties of tumor cells influence their spread and the formation of metastases. We have shown that low-viscosity cells behave a bit like honey and deform much more easily than high-viscosity cells (which are stiffer). These mechanical properties enable tumor cells to arrest in small blood vessels such as capillaries. In the secondary organ, low-viscosity tumor cells form larger metastases than high-viscosity tumor cells. However, these low-viscosity cells do not proliferate any more than high-viscosity cells. To explain this greater metastatic potential of low-viscosity tumor cells, our hypothesis is that they evade the immune system more effectively. Studies have shown that tumor cells interact extensively with neutrophils in the bloodstream, which gives them a greater potential for metastasis, but we do not know whether neutrophils can detect the mechanical state of tumor cells and whether this influences their immune responses. I have demonstrated that neutrophils interact as much with cells exhibiting reduced elasticity and viscosity as with control tumor cells, both in vitro and in vivo using the zebrafish model. However, neutrophil activation by netosis is reduced, even though they interact to the same extent.

Keywords: Immunology, Biomechanics, Oncology, Neutrophils

Blitz Talk n°5: DNA's great escape: how damaged ribosomal genes leave home for repair

Priyanka Pundir[1]

[1]IGBMC, University of Strasbourg, France

Abstract

DNA is constantly under threat, but some DNA types are more fragile than others. Ribosomal DNA is one of them; being most transcribed and easily broken is not the most ideal combination. But here's the catch: when it breaks, it cannot be repaired where it resides and leaves the nucleolus. Using live-cell microscopy in yeast, I tracked these damaged sites in real time, and what I found changes how we think about this escape.

Keywords: DNA repair

Blitz Talk n°6: Deciphering the target selection mechanisms of the Arabidopsis endoribonuclease DNE1

Paulo Eberhardt[1]

[1]IBMP – Institut de Biologie Moléculaire des Plantes du CNRS, Université de Strasbourg, 67084 Strasbourg, France

Abstract

Cytoplasmic mRNA decay is central to post-transcriptional gene regulation in plants. DNE1 (DCP1-ASSOCIATED NYN ENDORIBONUCLEASE 1) is an Arabidopsis thaliana MARF1-family endoribonuclease harboring an N-terminal NYN catalytic domain and C-terminal OST-HTH/LOTUS RNA-binding domains. DNE1 interacts with the decapping cofactor DCP1 and the NMD effector UPF1, positioning between multiple mRNA decay pathways. Its cleavage products are 5' monophosphorylated RNA fragments subsequently degraded by XRN4. Loss of DNE1 in a decapping mutant background causes defective phyllotaxy, highlighting its developmental relevance. Despite this importance, the rules governing target mRNA selection by DNE1 remain poorly understood. Transcriptome-wide studies identified over 200 DNE1-dependent cleavage sites, predominantly in coding sequences, with a subset of targets containing upstream open reading frames (uORFs) and RNA G-quadruplex (rG4). Our work aims to elucidate the molecular basis of DNE1 target recognition. We use structure-guided mutagenesis of the DNE1-DCP1 interaction interface to assess the functional relevance of this interaction. In parallel, we map DNE1 cleavage sites at single-nucleotide resolution using NanoMUCT, a targeted Nanopore sequencing workflow based on 5' adapter ligation, reverse transcription, and nested PCR, enabling high-confidence identification of cleavage events. Preliminary NanoMUCT results on candidate targets are presented, together with the genetic and biochemical framework used to dissect DNE1 functional domains. These approaches are building toward a mechanistic model of substrate selection by a plant cytoplasmic endoribonuclease.

Keywords: mRNA decay, endoribonuclease, DNE1, Arabidopsis thaliana, Nanopore sequencing

Blitz Talk n°7: Microbiota alterations induced by intermittent vs continuous sucrose intake in mice

Jeanne Tyrode[1]

[1]Laboratoire de neurosciences cognitives et adaptatives, Université de Strasbourg, CNRS – France

Abstract

Binge eating disorder (BED) is characterized by the consumption of large amounts of palatable food (sugar/fat) in a short period of time. It is associated with a feeling of loss of control, which is also observed in addiction-like behaviors. In our modern societies, this disorder is the most prevalent eating disorder with 2-3% of the adult population affected. However, despite this high prevalence, the biological mechanisms underlying BED remain poorly understood. Among others, palatable food items rich in sugar represent a key vulnerability factor in the development and maintenance of BED. Notably, sucrose is largely consumed through sugary drinks and ultra-processed foods. Recent studies have shown that gut microbiota is altered in individuals with maladaptive addictive behaviors, and also play a key role in behavioral responses to palatable food. However, few studies have evaluated this peripheral aspect in BED. Therefore, it is essential to better understand how binge eating influences the gut microbiota composition. Here, we used a mouse model of BED with intermittent free-choice access to sucrose that mimics uncontrolled consumption. We then compared the impact of sucrose bingeing in male and female mice with that of continuous free-choice sucrose access on gut microbiota composition. Using 16S rRNA sequencing, we observed distinct alterations in gut microbiota composition in both access conditions, as well as notable sex-dependent differences. These findings highlight the need to determine whether such changes may, in turn, influence behavior and contribute to eating disorders.

Keywords: Binge eating disorder, Gut microbiota, Sucrose

POSTERS

Poster n°1: Unraveling leukemogenesis and drug resistance mechanism of CDX2/UBTF B-Acute Lymphoblastic Leukemia (B-ALL)

Guillaume Goncalves[1], Alicia Perrin[2], Lauriane Gur[1], Hugo Bergugnat[3], Isabelle Duluc[1], Elisabeth Martin, Claudine Ebel, Laetitia Paulen[1], Emmanuelle Clappier[3], Jacky Goetz[1], Jean-Noel Freund, and Claire Domon-Dell[1]

[1]Immuno-Rhumatologie Moléculaire, INSERM U1109, Tumor Biomechanics Lab, Université de Strasbourg, Institut National de la Santé et de la Recherche Médicale, Université de Strasbourg, France
[2]IGBMC, Illkirch, CNRS UMR 7104, INSERM U1258, Université de Strasbourg, Illkirch, France
[3]Institut de Recherche Saint Louis, Institut National de la Santé et de la Recherche Médicale, Université Paris Cité, France

Abstract

Whereas B Acute Lymphoblastic Leukemia (B-ALL) prognosis has been improved for children, it remains poor for young adults due to a high relapse rate. We described the CDX2/UBTF B-ALL subgroup characterized by a specific transcriptomic profile, the massive ectopic expression of the homeotic transcription factor CDX2 and the expression of a newly identified fusion protein named UBTF::ATXN7L3. This unique and systematic coincidence of the two proteins suggests their involvement in the oncogenesis processes. UBTF and ATXN7L3 are both regulating transcription of rRNA and mRNA. CDX2 exerts a tumor suppressor function in intestine but transforms murine hematopoietic stem cells (HSC) toward Acute Myeloid Leukemia. Its oncogenic function in lymphoid lineage remains unknown. We aim to determine the consequences of CDX2 expression in B progenitors and to understand UBTF::ATXN7L3 function. Thanks to original conditional cre/lox mouse models, co-expression of CDX2 and KRASG12D in pre-pro B progenitors led to premature death and blocked lymphoid determination toward immature myeloid phenotype. When CDX2 and KRASG12D were expressed in pro-B cells, a B-ALL-like disorder arose with blockage of immature transformed pro-B cells and a reduced survival. In parallel, we revealed a punctiform nuclear localization of UBTF::ATXN7L3, its interaction with the deubiquitinase USP22 and in consequence, the global decrease of H2B deubiquitination. Overall, our results identify a CDX2 oncogenic function in B cell lineage in mutated KRAS context and suggest an epigenetic reprogramming orchestrated by UBTF::ATXN7L3. Whether and how these two proteins cooperate remains to be explored by modeling their co-expression in pro-B progenitors in mice.

Keywords: B Acute Lymphoblastic Leukemia, CDX2, UBTF::ATXN7L3, Animal model

Poster n°2: The Role of CD180 in B Cell Activation

Wenxin Ou[1], Mathieu Vogt, Clémentine Pouenat, Ludivine Robin, Romain Vauchelles, Jean-Daniel Fauny, Jacques-Eric Gottenberg, Hélène Dumortier, Laurent Mauvieux, Thomas Lavaux, and Laurent Miguet

[1]Institut de biologie moléculaire et cellulaire, Université de Strasbourg, Centre National de la Recherche Scientifique, France

Abstract

Background: Lipopolysaccharide (LPS) typically induces immune responses through the TLR4 receptor complex. While TLR4 is not expressed on human B cells, these cells exhibit robust responses to LPS stimulation. CD180, an orphan member of the TLR family, shares structural homology with TLR4 and is highly expressed on B cells; however, it lacks a classic Toll/Interleukin-1 receptor (TIR) domain. This study aims to elucidate the mechanism by which human B cells sense LPS and to define the functional role of CD180 in this process. Methods: We performed RNA-seq analysis on LPS-stimulated versus unstimulated primary human B cells to assess transcriptional responses. We utilized flow cytometry to evaluate the physical interaction between LPS and B cells. Furthermore, we employed the Proximity Ligation Assay (PLA) to quantify nanoscale co-localization between CD180 and surface molecules, including CD19, CD20, and CD40, to investigate potential receptor cooperation. Results: Transcriptomic analysis revealed significant gene expression changes in primary human B cells following LPS stimulation, involving key immune signaling pathways. PLA experiments demonstrated that LPS stimulation significantly enhances the nanoscale association between CD180 and CD40. Conclusion: Our findings indicate that human B cells possess the capacity to sense LPS via non-TLR4-dependent mechanisms. The increased proximity of CD180 to CD40 upon LPS stimulation suggests that CD180 may contribute to LPS-induced B-cell activation through receptor cooperation, potentially compensating for its lack of a TIR domain to facilitate NF-κB signaling and cellular activation.

Keywords: CD180, LPS, B cells, Innate immunity

Poster n°3: Tumor cell mechanics modulate neutrophil activation without altering interaction during dissemination

Elodie Marchal[1]

[1]Immuno-Rhumatologie Moléculaire, Université de Strasbourg, Institut National de la Santé et de la Recherche Médicale, France

Abstract

The mechanical properties of tumor cells influence their spread and the formation of metastases. We have shown that low-viscosity cells behave a bit like honey and deform much more easily than high-viscosity cells (which are stiffer). These mechanical properties enable tumor cells to arrest in small blood vessels such as capillaries. In the secondary organ, low-viscosity tumor cells form larger metastases than high-viscosity tumor cells. However, these low-viscosity cells do not proliferate any more than high-viscosity cells. To explain this greater metastatic potential of low-viscosity tumor cells, our hypothesis is that they evade the immune system more effectively. Studies have shown that tumor cells interact extensively with neutrophils in the bloodstream, which gives them a greater potential for metastasis, but we do not know whether neutrophils can detect the mechanical state of tumor cells and whether this influences their immune responses. I have demonstrated that neutrophils interact as much with cells exhibiting reduced elasticity and viscosity as with control tumor cells, both in vitro and in vivo using the zebrafish model. However, neutrophil activation by netosis is reduced, even though they interact to the same extent.

Keywords: Immunology, Biomechanics, Oncology, Neutrophils

Poster n°4: Proteoglycofilli: a new antimicrobial fiber containing glycosaminoglycans secreted by neutrophils against Shigella

Jules Tritschler[1]

[1]Institut de biologie moléculaire et cellulaire, université de Strasbourg, Centre National de la Recherche Scientifique, France

Abstract

Neutrophils are first immune cells recruited to an infectious site and possess several antimicrobial functions to kill bacteria. Belongs these functions, NET (Neutrophil Extracellular Traps) are characterized by the permeabilization of the plasma membrane and the release of DNA fibers and antimicrobial proteins to eliminate microbes, but are accompagnied by neutrophils cell death. In the context of the Shigella infection, a bacteria infecting human colon, my host team discovered the release of a new type of fibers by neutrophils without neutrophil death, rising the question of functional and content differences between this new fiber and NET. This new fiber was named Proteoglycofilli (PGF) as composed of proteins and glycosaminoglycans (GAG) and a major difference with NET is the absence of DNA. PGF release is induced after a Shigella challenge and displays an antimicrobial activity against multiple Shigella species. This activity is dependent on the presence of GAG and these GAG are also found in NET and mandatory for its antimicrobial activity. 2 main questions arise from these results : what are the secretion's mechanisms as storage location and trafficking pathway and how GAG exert an antimicrobial activity. For long terms, thanks to understanding how neutrophils switch between NET and PGF, PGF release could be induced against sensitive bacteria to this secretion to reinforce the immune response.

Keywords: Neutrophil, Shigella, bacteria, infection, glycosaminoglycan, extracellular vesicles, hypoxia

Poster n°5: Macrophages promote human sensory neuron progenitor proliferation in a developing dorsal root ganglia model

Nathan Garnier[1,2], Coralie Mollard[1,2], Jean-Daniel Fauny[1], Romain Vauchelles[1], Alina Nicolae[3], Manuela Tavian[4], Christopher Mueller[1], Vincent Flacher[1], and Benjamin Voisin[1,2]

[1]CNRS UPR3572/I2CT Immunologie, Immunopathologie et Chimie Thérapeutique, Institut de Biologie Moléculaire et Cellulaire ; Institut du Médicament de Strasbourg ; Strasbourg, France, Centre National de la Recherche Scientifique, CNRS, France
[2]Universit´e de Strasbourg, université de Strasbourg, France
[3]Hôpital de Hautepierre, Hôpitaux Universitaires de Strasbourg, France
[4]Centre de Recherche en Biomédecine de Strasbourg, université de Strasbourg, Institut National de la Santé et de la Recherche Médicale – France

Abstract

Myeloid immune cells play a key role during development. Macrophages are involved in brain development and are also closely associated with the peripheral nervous system. This system consists of nerve fibers that extend from dorsal root ganglia (DRG), in which the cell bodies (soma) of sensory neurons reside. In humans, several macrophage subsets are found in both embryonic and adult DRGs, but their functions remain unknown. We hypothesized that one of these populations may promote neuronal proliferation, as observed in mouse models. To investigate this, we set up an in vitro coculture model involving iPSC-derived sensory progenitors and monocyte-derived macrophages. Using different readouts, we noticed that cell divisions of neuronal progenitors were increased in the presence of macrophages. Moreover, physical separation of macrophages from differentiating neurons did not decrease the extent of proliferation, suggesting that macrophages may exert their influence through a soluble factor. In parallel, we initiated in situ immunofluorescence analysis of human embryos and found different patterns of proliferating cells in DRG across peripheral nervous system development. Altogether, our data support the hypothesis that macrophages modulate the proliferation of sensory neuron progenitors through the secretion of a soluble factor.

Keywords: Macrophages, sensory neurons, development

Poster n°6: Overcoming bacterial resistances using a macrolide - antimicrobial peptide chimera

Théo D'andrea[1,2], Louise Tirole[1], Sandeep Mummadi[4], Dominique Burnouf[1], Jérôme, Wagner[1], Guillaume Compain[3], Gilles Guichard[4], Emmanuel Giudice[2], Reynald Gillet[1], and Eric Ennifar[1]

[1]Architecture et réactivité de l'ARN, Université de Strasbourg, CNRS, France
[2]Institut de Génétique et Développement de Rennes (IGDR) UMR 6290, Université de Rennes 1, CNRS, France
[3]CBMN, IECB UMR 5248, Univ. Bordeaux, CNRS, Bordeaux INP, CBMN, France
[4]Institut de Chimie et Biologie des Membranes et des Nano-objets – Univ. Bordeaux, CNRS, Bordeaux INP, CBMN, UMR 5248 – France

Abstract

In 2019, antimicrobial resistance (AMR) was associated with an estimated 4.95 million deaths worldwide. Projections also show that antimicrobial resistance could kill up to 10 million people by 2050 while also threatening animal health and the agricultural sector. To address this growing health crisis, the World Health Organization has designated a group of pathogens, known as "ESKAPE(E)" (E.faecium, S.aureus, K.pneumoniae, A.baumannii, P.aeruginosa, Enterobacter spp., and E.coli), as critical targets for the development of new antibiotics. In this context, we are developing a strategy to avoid the emergence and dissemination of resistance by designing conjugates in which macrolides are covalently linked to small proline-rich antimicrobial peptides (PrAMPs). Macrolides are bacteriostatic antibiotics commonly used against Gram-positive pathogens, while PrAMPs, naturally produced by arthropods and mammals, exhibit bactericidal activity against Gram-negative bacteria. Both of these classes of molecules interact with the peptide exit tunnel (PET) of the ribosome. Our group had previously shown that, unexpectedly, both macrolides and PrAMPs can coexist within the PET. By conjugating a macrolide with a PrAMP, we aim to combine their activities, potentially overcoming macrolide resistance, extending the antimicrobial spectrum of macrolides and limiting the emergence of resistances. Biophysical experiments, such as ITC assays were conducted as well as microbiological manipulations as MIC. Taking into account the results of both physical and microbiological parts, we resolved the structures of the ribosomes in complex with the compounds by cryo-electron microscopy. These approaches open promising avenues for the development of next-generation antibiotics.

Keywords: Ribosome, Antibiotics, Antimicrobial peptides, Antimicrobial resistance

Poster n°7: Aptamer-mediated selective and modulable siRNA delivery

Léa Denechere[1], Charlène D'ancona[1,2], Béni Twendimbadi[1], Sandrine Pelet[1], Sylvie Friant[2], Séverine Bär[2], Mayeul Collot[3], Pierre Fechter[4], Laurence Choulier[1]

[1]Laboratoire de Bioimagerie et Pathologies, université de Strasbourg, Centre National de la Recherche Scientifique
[2]Laboratoire de Génétique Médicale, université de Strasbourg, Institut National de la Santé et de la Recherche Médicale
[3]Chémo-Biologie Synthétique et Thérapeutique, université de Strasbourg, Institut de Chimie, CNRS Chimie, Centre National de la Recherche Scientifique
[4]Biotechnologie et Signalisation Cellulaire, CNRS : UMR7242

Abstract

Small interfering RNAs (siRNAs) are specific and effective molecules for gene silencing. However, their use is limited by poor cell penetration due to their negative charge and their size. To overcome these barriers, the conjugation of siRNAs to ligands targeting cell-surface receptors is a promising approach. Aptamers appear to be interesting candidates since they are single-stranded DNA or RNA with a high affinity and selectivity to a specific target, such as cell-surface receptors. Thus, we aim to develop selective and modulable vehicles associating a siRNA with one or more aptamers called mono- or multivalent aptamer-siRNA chimeras (AsiCs). First, we would like to create a monovalent AsiC using either an RNA or DNA aptamer. We compared two AsiCs formed by an RNA-RNA or RNA-DNA sticky bridge. Our preliminary results show a similar assembly. Second, we designed an innovative multivalent AsiC. Our vehicle combines one siRNA with one to three homo- or heterovalent aptamers. The various elements contain hybridization sequences that will enable controlled self-assembly. Thanks to their versatile nature, the number, position and type of aptamers (DNA or RNA) could be easily changed. So far, we have designed RNA sequences, predicted their 2D structure and checked the vehicle assembly. Our preliminary results of RNA-RNA and RNA-DNA AsiC are encouraging and confirm the feasibility to combine DNA and RNA aptamers in multivalent AsiC, new siRNA active delivery tool with great potential. As perspectives, we wish to deepen characterization of AsiCs (stability and functional cell assays) and study their intracellular traffic by bioimaging.

Keywords: siRNA, aptamer, active targeting delivery, innovative delivery tool, multivalent conjugate, cell surface receptor

Poster n°8: Deciphering the role of two essential chloroplast PPR RNA-binding proteins in Arabidopsis thaliana

Hélane Larue[1]

[1]Institut de biologie moléculaire des plantes, université de Strasbourg, Centre National de la Recherche Scientifique : UPR2357, Centre National de la Recherche Scientifique – France

Abstract

Chloroplasts are essential plant organelles that drive photosynthesis and support key metabolic processes. Although they retain a reduced prokaryote-derived genome, the expression of chloroplast genes is largely regulated by nuclear-encoded RBP imported into the organelle. Among them, PPR proteins form a large plant-specific family that plays pivotal roles in organellar RNA metabolism. We previously identified MDA1 as a transcription factor that binds the psbE promoter, enhancing transcription and stabilizing its nascent 5′ end. Co-immunoprecipitation experiments revealed that MDA1 is associated in vivo with a previously uncharacterized RBP from the PPR family, MIP1 (MDA1-Interacting-Partner-1), suggesting it may contribute to protecting the 5′ end of the psbE transcript. Arabidopsis encodes a paralog of MIP1, MIP2, which shares 42% sequence identity. While mip1 or mip2 single mutants do not display visible phenotypes, the mip1/mip2 double mutant exhibits severe dwarfism, chlorosis, and lethality after six weeks of growth. TEM revealed highly disorganized chloroplast ultrastructure with poorly developed thylakoid membranes, demonstrating that MIP1 and MIP2 are essential for chloroplast biogenesis. Transcript profiling by RT-qPCR showed a marked reduction of PEP-dependent transcripts, while NEP-dependent mRNAs remained largely unaffected. Northern blot analyses confirmed specific defects in early PEP-transcribed genes. To elucidate the molecular function of MIP1 and MIP2, complemented mip1/mip2 lines expressing epitope-tagged proteins were generated for co-IP and proximity labeling approaches to identify their protein partners and in vivo RNA targets. Together, our work aims to clarify how these two PPR RNA-binding proteins act at the intersection of transcription and RNA stabilization during early chloroplast development.

Keywords: Arabidopsis thaliana, chloroplasts, PPR RNA, binding proteins, RNA metabolism, proximity labeling, gene expression

Poster n°9: RNA structures regulating the packaging and translation of HIV-1 RNA isoforms

Ulyana Myerkina[1], Layla El Kamali[1], Anne-Sophie Gribling-Burrer[2], and Redmond Smyth[2]

[1]Institut de biologie moléculaire et cellulaire, université de Strasbourg, Centre National de la Recherche Scientifique, France
[2]CNRS, université de Strasbourg, France

Abstract

Human Immunodeficiency Virus type 1 (HIV-1) remains a major global health challenge. Its 9 kb integrated genome produces over 50 RNA isoforms, which must be tightly regulated to ensure efficient viral replication. These transcripts are classified into three groups: unspliced (US), partially spliced (PS), and fully spliced (FS) RNAs. US RNA serves both as the genomic RNA packaged into virions and as a template for structural and enzymatic proteins, whereas PS and FS RNAs encode regulatory and accessory proteins and are excluded from packaging.HIV-1 RNAs also display heterogeneity at their 5′ ends, including transcription start site variation (e.g., G or GGG) and distinct cap structures such as m^7G and m^3G. However, how these features contribute to isoform-specific regulation remains poorly understood. Studying HIV-1 RNAs is challenging, as they represent 1–2% of total cellular mRNAs.To overcome this limitation, we developed a targeted pulldown strategy using oligonucleotides against the 5′UTR, enabling enrichment of ~80% of both unspliced and spliced HIV-1 RNAs. Direct RNA sequencing using Nanopore revealed m^6A modifications predominantly enriched in the 3′ regions of spliced RNAs.We further examined the impact of 5′ transcription start site heterogeneity on RNA structure using isoform-resolved long-read structural probing (Nano-DMS-MaP). Strikingly, mutants expressing exclusively G- or GGG-initiated RNAs exhibited distinct 5′UTR structures under translational arrest, particularly in spliced isoforms. These findings suggest that isoform-specific RNA structures may play a role in regulating translation. Ongoing work aims to elucidate these mechanisms and may inform the development of RNA-based antiviral strategies.

Keywords: HIV, 1, Coiffe, Capture, Transcription, isoform, Nanopore

Poster n°10: Characterization of the HIV-1 Vif protein interactome: identification of cellular RNA targets

Layla Tajer[1,2], Jason Decotter[1], Benjamin Stupfler[1], Ziad Fajloun[2,3], Serena Bernacchi[1], Roland Marquet[1], Sarah Gallois-Montbrun[4], and Jean-Christophe Paillart[1]

[1]Université de Strasbourg, CNRS, Architecture et Réactivité de l'ARN, UPR 9002, IBMC, 2 Allée Konrad Roentgen, F-67000 Strasbourg, France, Centre National de la Recherche Scientifique, CNRS, France
[2]Laboratory of Applied Biotechnology (LBA3B), Azm Center for Research in Biotechnology and its Application, EDST, Lebanese University, 1300, Tripoli, Lebanon, Liban
[3]Department of Biology, Faculty of Sciences 3, Campus Michel Slayman Ras Maska, Lebanese University, 1352, Tripoli, Lebanon, Liban
[4]Université Paris Cité, INSERM U1016, CNRS UMR8104, Institut Cochin, 27 rue Faubourg Saint-Jacques, F-75014 Paris, France, Centre National de la Recherche Scientifique, CNRS, Institut National de la Santé et de la Recherche Médicale, INSERM, France

Abstract

To successfully infect humans, HIV-1 must overcome several physical and immunological barriers. A key viral strategy involves Vif protein, which plays a critical role in counteracting these host defenses. The interaction between Vif and the cytidine deaminase APOBEC3G (A3G), a potent restriction factor, is a key mechanism by which HIV-1 evades the host's intrinsic immunity. In non-permissive cells, Vif counteracts A3G mainly by promoting its degradation via the ubiquitin–proteasome pathway. In addition, Vif plays a critical role in the inhibition of A3G translation. This repression involves direct interactions between Vif and the 5′ untranslated region of A3G mRNA. Furthermore, Vif has been shown to interact with the viral gRNA both in vitro and ex vivo. These results suggest that Vif may target other cellular mRNAs to modulate their expression and recruit protein partners involved in translational regulation. Based on this hypothesis, this study aims to characterize the RNA interactome of HIV-1 Vif and to identify novel cellular RNA targets potentially involved in Vif-dependent regulatory mechanisms. A transcriptome-wide analysis was previously performed in our laboratory using PAR-CLIP, leading to the identification of multiple RNA candidates. From this dataset, high-priority candidates were selected based on multiple criteria. To validate these candidates, UV cross-linking RNA immunoprecipitation assays were performed. Preliminary results indicate that several cellular RNAs specifically associate with Vif. Notably, HSPA8, SRRM2, and HMGB2 mRNAs were found to be enriched in Vif-containing ribonucleoprotein complexes, supporting their interaction with Vif. These preliminary findings support the existence of a broader Vif-associated RNA interactome.

Keywords: HIV1, Vif, RNA binding protein, UV Crosslinking

Poster n°11: Discovery of HBV cccDNA Host Factors as Antiviral Targets for a Cure

Christos Satratzemis[1], Laura Meiss-Heydmann[1], Aurélie Batt[1], Florine Zettl[1], Charlotte Bach[1], Pauline Casetta[1], Eloi Verrier[1,2], and Thomas Baumert[1,3]

[1]Institute for Translational Medicine and Liver Disease, Université de Strasbourg, Institut National de la Santé et de la Recherche Médicale, France
[2]Centre International de Recherche en Infectiologie, Université Claude Bernard Lyon 1, Centre National de la Recherche Scientifique, France
[3]L'Institut hospitalo-universitaire de Strasbourg – Hôpitaux Universitaires de Strasbourg, France

Abstract

Infection with the hepatitis B virus (HBV) causes chronic hepatitis B, affecting approximately 300 million people worldwide and carrying up to a 45% risk of disease progression. Over one million deaths each year are attributed to HBV-related complications, primarily to hepatocellular carcinoma. During infection, HBV hijacks host cell proteins to produce cccDNA (covalently closed circular DNA). Owing to its chromatin-like organization, cccDNA persists in infected hepatocytes and serves as a viral reservoir, driving the development of chronic HBV infection and hepatitis. cccDNA thus represents the main barrier to achieving a cure for chronic hepatitis B. Current therapies suppress viral replication and disease progression but fail to eliminate cccDNA. Consequently, treatment must be administered lifelong and does not prevent severe complications. This PhD project aims to identify host factors involved in cccDNA formation that could be therapeutically targeted. A high-throughput screening strategy based on a cccDNA reporter cell line was used to perform two siRNA screenings targeting cellular kinases and membrane proteins, followed by a secondary screening in a bona fide HBV infection model with a cccDNA-specific readout. This approach enabled the identification of 21 putative cccDNA host factors. Preliminary validation using complementary loss-of-function approaches in relevant cell-based models supports the involvement of two candidates in cccDNA biology, and ongoing work seeks to elucidate their molecular mechanisms. Understanding these virus-host interactions will enable the development of novel therapeutic strategies targeting cccDNA via its host factors and contribute to efforts toward a cure for chronic hepatitis B.

Keywords: HBV, cccDNA, host factors, chronic hepatitis B, novel therapeutic strategies

Poster n°12: Olfactory impairment at prodromal stage predicts cognitive decline in dementia with Lewy bodies and Alzheimer's disease: a clinical and neuroanatomical study

Maria Fiori[1], Cécile Pernossi[1], Laura David[1], Olivier Bousiges[1,2], Léa Sanna[3], Candice Muller[3], Alix Ravier[3], Catherin Demuynck[3], Frédéric Blanc[1,3], and Luc Marlier[1]

[1]Imagerie Multimodale Intégrative en Santé, Laboratoire des sciences de l'ingénieur, de l'informatique et de l'imagerie, France
[2]Hôpital de Hautepierre, Hôpitaux Universitaires de Strasbourg, France
[3]Centre Mémoire Ressources et Recherche, Centre Hospitalier Universitaire, France

Abstract

Olfactory dysfunction is a hallmark of dementia with Lewy bodies (DLB) and Alzheimer's disease (AD). However, modality-specific cognitive and subjective profiles, their prognostic value, and neuroanatomical substrates remain poorly characterized. Objective: To investigate higher-order olfactory signatures, their predictive value for longitudinal cognitive decline, and their structural neural bases in early DLB and AD. Methods: Participants with early DLB, early AD, and cognitively unimpaired (CU) controls underwent standardized olfactory assessments (detection thresholds, identification, familiarity, and hedonicity). Longitudinal decline was tracked via the annualized change in Mini-Mental State Examination (ΔMMSE) score. Structural correlates were explored in a neuroimaging sub-sample (n = 62) using whole-brain voxel-based morphometry (VBM). Results: The final cohort included 72 participants (37 DLB, 17 AD, 18 CU). Odor identification was severely impaired in DLB and AD compared to CU. Specifically, DLB patients exhibited a significant reduction in subjective odor familiarity. Preserved baseline identification predicted a slower rate of cognitive decline across both disease cohorts (DLB: $\rho = 0.398$, $p = 0.015$; AD: $\rho = 0.667$, $p = 0.009$). VBM analyses ($p < 0.001$, $k \geq 50$) revealed that in DLB, decreased hedonicity correlated with the right parietal operculum, altered familiarity with right fronto-striatal white matter (WM) degradation, and poorer detection thresholds with reduced WM volume within bilateral cerebellar pathways. Conclusion: Preserved odor identification serves as a prognostic marker for slower global cognitive decline in early DLB and AD. Structural correlates underscore a profound, multi-level central disconnection spanning affective, implicit semantic, and attentional-predictive brain regions in DLB.

Keywords: Alzheimer's disease, Dementia with Lewy bodies, Magnetic resonance imaging, Olfaction

Poster n°13: Reconstitution of Human Elongation Complexes to Study their Structure by Cryo-EM

Rakhshana Bala Krishnan[1], Roberto Bahena Ceron[1], Antony Lechner[2], Philippe Wolff[2], Isabelle Hazemann[1], Ottilie Von Loeffelholz[1], and Bruno Klaholz[1]

[1]CBI-IGBMC, CNRS UMR7104, Inserm U964, Université Louis Pasteur, Strasbourg, France
[2]Institut de biologie moléculaire et cellulaire, université de Strasbourg, Centre National de la Recherche Scientifique, France

Abstract

The ribosome is a large ribonucleoprotein complex that translates proteins from mRNA templates. The fundamental process of peptide bond formation is conserved across all domains of life. The central processes in translation are carried out in distinct stages: initiation, elongation, termination and recycling during which translation factors bind and dissociate from the ribosome and modulate its functional specificity during protein synthesis. Transfer RNAs play an important role in decoding the genetic code. It forms a link between the mRNA at the decoding center and the amino acids attached at the CCA end of the tRNA in the peptidyl transferase center. The project aims to better understand the fidelity mechanisms during translation elongation. For this, we aim to reconstitute human translation elongation complexes and study their structure by cryo electron microscopy. The established protocol to purify 80S ribosome is adapted for the purification of 40S and 60S ribosomal subunits and the quality of the sample was analysed by negative staining. Two aminoacyl tRNA synthetases were also expressed and purified. Future works include the purification of elongation factors, aminoacylation of tRNA, complex reconstitution and cryo-EM for structure analysis. Through this research work we'll be able to contribute towards better understanding the molecular mechanisms of translation elongation and how translation fidelity is ensured. Faithful translation of mRNA to protein is necessary to produce functional proteins and maintain cellular homeostasis. Errors like misincorporation events and frameshifting can produce non-functional proteins and disrupt cellular pathways resulting in cancer & other neurodegenerative diseases.

Keywords: Eukaryotic translation, ribosome, transfer RNA

Poster n°14: Combined intensive resistance and endurance exercise in low-disease activity inflammatory myopathies : A Hospital vs. Community-Based prospective study

Quentin Toumazeau[1], Lea Debrut[1,2], Pauline Asael, Alizée Heit, Anne-Laure Charles, Bernard Geny, Margherita Giannini[3], Alain Meyer[3]

[1]UR3072, Centre de Recherche en Biomédecine de Strasbourg (CRBS), Fédération de Médecine Translationnelle, université de Strasbourg
[2]Faculty of Sport Sciences, European Centre for Education, Research and Innovation in Exercise Physiology (CEERIPE), University of Strasbourg, France
[3]Physiology and Muscle Function Explorations Department, Referral Centre for Systemic Rare Autoimmune Diseases, Strasbourg University Hospitals, Strasbourg, France, Les Hôpitaux Universitaires de Strasbourg (HUS)

Abstract

Inflammatory myopathies (IM) are autoimmune diseases characterized by chronic skeletal muscle inflammation and weakness. Many IM patients feature persistent impairment in exercise capacity despite treatments wich is associated with fatigue, disability and mortality. Combined resistance and endurance exercise (REE) has been reported to improve exercise capacity. However, i) these results have not been replicated, ii) was conducted in a hospital setting limiting patients access to the care, iii) the mechanism underlying the efficacy of exercise in IM has been scarcely investigated. This study aimed to validate the efficacy of REE in patients with low disease activity IM, to compare hospital-based and community-based programs and explore the underlying mechanisms. Eighty patients with IM were prospectively included for a 36-session REE. The program consisted of 30min of aerobic exercise and 20min of resistance exercise, 3 sessions per week for 12 weeks with evaluations conducted before and after the intervention. Participants <50km from the hospital trained onsite (HB); others used local physiotherapists (CB). Twenty-five patients completed the protocol. VO_2max increased after both the HB and CB REE. Strength, 6-min walk distance, were similarly improved in both groups. Perceived fatigue decreased in the HB as well as in the CB. PBMCs mitochondrial respiration improved as shown by complex IV activity. In both settings, REE is feasible, safe and effective for low disease activity IM patients. Improvement of exercise capacity, muscle strength, disability, and fatigue were associated with an improvement in mitochondrial respiratory capacity that may play a role in the efficacy of this non-pharmacological treatment.

Keywords: Physical therapy, Physiotherapy and Physical Activity, Non-pharmacological interventions

INSPIRING TALK

Inspiring Talk

Session 1

Inspiring talk 1 : Stéphanie Blandin, PhD – "At the crossroads of immunology and mosquitoes"- Immune responses in vector mosquitoes — IBMC

Session 2

Inspiring talk 2 Jill Pilet, PhD – "Between Passion and Doubt: The Journey of a Researcher in the Making"Childhood Oncology Research Unit –Childhood Oncology Research Unit — Institut Curie, Paris

Doctoral School of Life Health Sciences
– University of Strasbourg
06 & 07 May 2026

Committee :
Gur Lauriane, Pouenat Clementine, Bala Krishnan Rakhshana, Battaglia Filippo, Sieber Matthieu, Heimberger Kyllian

Graphic Designer :
Imbs Océane

2026 EDAYS

THANKS

University of Strasbourg