ADAS DDAY 2023

12 APRIL 2023
8:30-18:00

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ABSTRACT BOOK

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Faculté de biologie et de médecine
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PROGRAM

8:30

REGISTRATION AND COFFEE

9:00

WELCOME

9:10

S. GARCIA-SANTAMARIA/COMMON THERAPEUTICS INDUCE EMERGENT BEHAVIORS ON IN VITRO GUT BACTERIAL COMMUNITIES

9:40

ALISON LIN/ENHANCEMENT OF IMMUNE RESPONSE BY NANOPARTICLE VACCINATION

9:55

ELEVATOR PITCHES

10:20

COFFEE BREAK/POSTER SESSION 1

11:10

JOSEPH DUBROVSKY/A STORY OF FOUNDER CELLS

11:40

SAMUEL MOIX/CAUSES AND CONSEQUENCES OF TELOMERE SHORTENING: A MENDELIAN RANDOMIZATION STUDY

12:00

LUNCH BREAK

13:00

KEVIN FOSTER/COMPETITION AND WARFARE IN BACTERIA AND THE HUMAN MICROBIOME

13:30

CAROLINE SCHMITT KOOPMANN/SWITZERLAND'S NARCOTICS REGULATION JUNGLE

13:45

BASTIEN KRUMM/PLASMA VOLUME VARIATIONS IN ELITE ATHLETES

14:00

ANABELLE DECOTTIGNIES/ALTERNATIVE LENGTHENING OF TELOMERES IN CANCER: DIAGNOSIS AND THERAPEUTIC PERSPECTIVES

14:30

MAI THU NGUYEN/THE GENETIC ARCHITECTURE, EVOLUTION AND STABILITY OF POPULATIONS WHERE FEMALES, MALES AND HERMAPHRODITES COEXIST

14:45

ANNA GLAUS/ADAPTATION OF PLANT ARCHITECTURE DURING DOMESTICATION

15:00

COFFEE BREAK/POSTER SESSION 2

15:50

PHILIPP WALCH/DISSECTING THE IMPACT OF ENTERIC VIRAL-BACTERIAL CO-INFECTION ON THE HOST INNATE IMMUNE RESPONSE AND ITS IMPLICATIONS FOR PATHOGENICITY

16:05

HILAL LASHUEL ND BIOSCIENCE

16:35

BREAK

16:50

PRIZES AND CLOSING SPEECH

17:00

APERO

ACKNOWLEDGEMENTS

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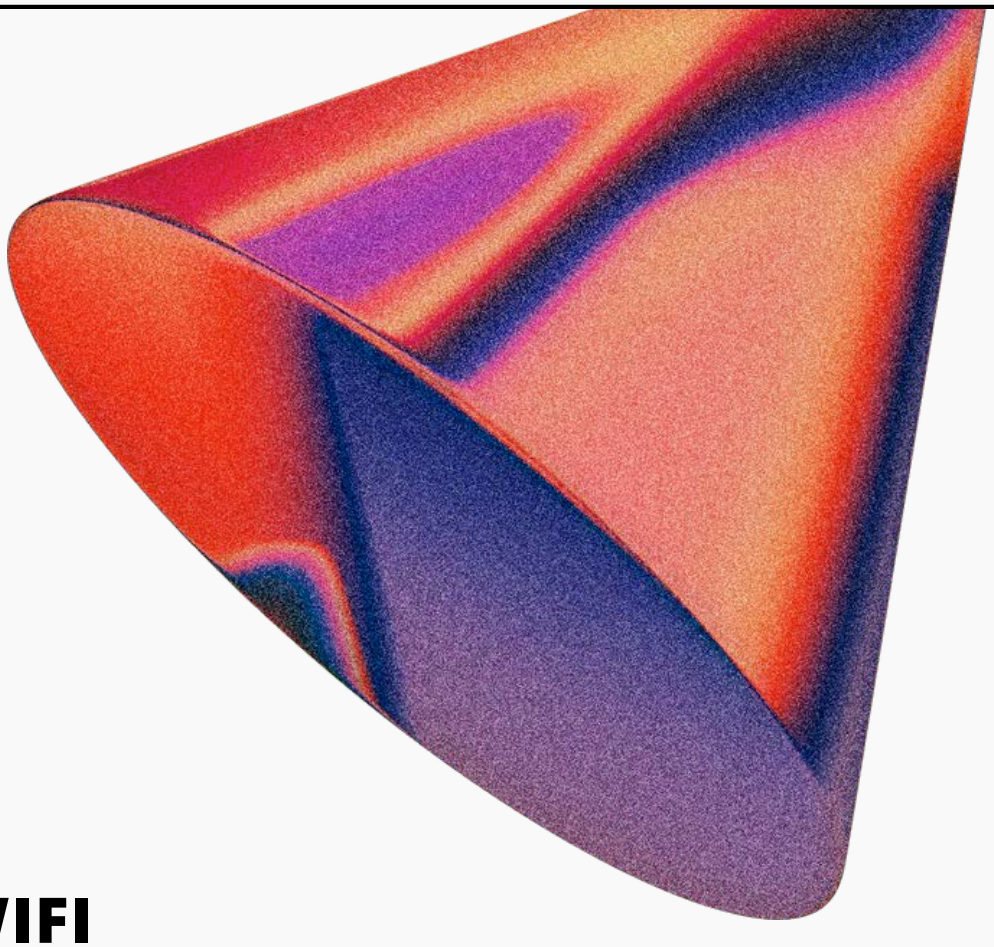

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ABSTRACT INDEX:

KEYNOTE LECTURES:

S. Garcia-Santamaria (ITQB-IGC, PORTUGAL) 12
Common therapeutics induce emergent behaviors on in vitro gut bacterial communities

J. Dubrovsky (Universidad Nacional Autónoma de México, MEXICO) 13
A story of founder cells

A. Decottignies (UCLouvain, Brussels, BELGIUM) 14
ALTernative lengthening of telomeres in cancer: diagnosis and therapeutic perspectives

K. Foster (University of Oxford, UK) 15
Competition and warfare in bacteria and the human microbiome

H. Lashuel (EPFL, Lausanne, SWITZERLAND) 16
Rising to the Challenge: ND BioSciences' innovative solutions for tackling the complexity of neurodegenerative diseases

SELECTED ORAL PRESENTATIONS:

18
SOP.01. Alison Lin
Enhancement of immune response by nanoparticle vaccination

19
SOP.02. Samuel Moix
Causes and consequences of telomere shortening: A Mendelian randomization study

SOP.03. Caroline Schmitt-Koopmann, **Switzerland's narcotics regulation jungle** 20

SOP.04. Bastien Krumm **Plasma volume variations in elite athletes** 21

SOP.05. Mai Thu Nguyen **The genetic architecture, evolution and stability of populations where females, males and hermaphrodites coexist** 22

SOP.06. Anna N. Glaus **Adaptation of plant architecture during domestication** 23

SOP.07. Philipp Walch **Dissecting the impact of enteric viral-bacterial co-infection on the host innate immune response and its implications for pathogenicity** 24

SELECTED ELEVATOR PITCHES:

SPI.01. Paul-Emmanuel Vanderriele **The glucocorticoid receptor is likely involved in the development of the salt-sensitive hypertension through the deregulation of the soluble epoxide hydrolase (sEH) in rats.** 26

SPI.02. Pauline Gut **Wideband black-blood late gadolinium enhancement imaging for patients with cardiac implantable electronic devices** 28

SPI.03. Silvia Prieto Baños **Annotation matters: the effect of gene annotation on orthology** 30

SPI.04. Arnaud Lyon **Immune-mediated improved tolerance to kidney and liver ischemia-reperfusion injury in female mice.** 31

SPI.05. Tom Citherlet 33
Is cortisol awakening response related to Acute Mountain Sickness in eumenorrheic and postmenopausal women?

SPI.06. Saray Ramos 34
The structural basis for ninjurin-1 mediated plasma membrane rupture in inflammatory cell death

SELECTED POSTERS:

SPO.01. Caroline Schmitt-Koopmann1, 37
Switzerland's narcotics regulation jungle

SPO.02. Gyda Fenn-Moltu 38
Accidental biocontrol: human-mediated dispersal of insect parasitoids and predators

SPO.03. Anna N. Glaus 39
Adaptation of plant architecture during domestication

SPO.04. Esther Landaluce-Iturriria 40
Macronutrition and signal-induced regulation of RNA splicing in adipose tissue depots

SPO.05. Arnaud Lyon 41
Immune-mediated improved tolerance to kidney and liver ischemia-reperfusion injury in female mice

SPO.06. Çağla Görkem Eroğlu 43
Presence of Neighbors Influences Buckwheat Root Exudate Composition

SPO.07. Tom Citherlet 44
Is cortisol awakening response related to Acute Mountain Sickness in eumenorrheic and postmenopausal women?

SPO.08. Axel KF Aguettaz 45
A novel PLIN3 splicing variant reveals a conserved mitochondrial targeting of the perilipin protein family.

SPO.09. Pauline Gut 46
Wideband black-blood late gadolinium enhancement imaging for patients with cardiac implantable electronic devices

SPO.10. Denho Ravi 48
Host-pathogen interactions in group B streptococcal sepsis

SPO.11. Bastien Krumm 49
Plasma volume variations in elite athletes

SPO.12. Beril Mersinoglu 50
Characterization of novel candidate factors impacting HIV life cycle

SPO.13. Saray Ramos 51
The structural basis for ninjurin-1 mediated plasma membrane rupture in inflammatory cell death

SPO.14. Mai Thu Nguyen 53
The genetic architecture, evolution and stability of populations where females, males and hermaphrodites coexist

SPO.15. Alison Lin 54
Enhancement of immune response by nanoparticle vaccination

SPO.16. Samuel Moix 55
Causes and consequences of telomere shortening: A Mendelian randomization study

SPO.17. Natalia Gonzalez Gaarslev 56
Harnessing genetic interactions driving inflorescence complexity in tomato

SPO.18. Silvia Prieto Baños 57
Annotation matters: the effect of gene annotation on orthology

SPO.19. Maria-Paraskevi Moschofidou 58
Evaluation of a new audiovisual information material for patients with surgical thyroid disease.

SPO.20. Joe Dickinson 60
Understanding the molecular mechanism of DNA translocation and loop extrusion by Smc5/6

SPO.21. Yifat Quan 61
SlABCG36 and SlABCG42 are largely redundant for tomato fruit cutin formation

SPO.22. Shannen Graf 62
The moderating role of context-encoding and -memory in the intergenerational transmission of post-traumatic stress

SPO.23. Margaux Crézé 63
Dynamics and consequences of nutrition--related microbial dysbiosis in early life

SPO.24. Simon Yersin 65
Gut microbiomes of agropastoral children from the Somali Regional State of Ethiopia show a unique bacterial composition reflecting their dietary habits

SPO.25. Bejoy Manoj 67
Mechanics of anisotropic growth

SPO.26. Paul-Emmanuel Vanderriele 68
The glucocorticoid receptor is likely involved in the development of the salt-sensitive hypertension through the deregulation of the soluble epoxide hydrolase (sEH) in rats.

SPO.27. Sarah McHugh
Causes and Consequences of Early Life Malnutrition in Later Life 70

SPO.28. Philipp Walch
Dissecting the impact of enteric viral--bacterial co-infection on the host innate immune response and its implications for pathogenicity 71

SPO.29. Brenda Rios Ochoa
Exploding sRNA crosskingdom comunication for food security 72

SPO.30. Cassandra Tabasso
Unraveling lipotoxicity, from subcellular mechanistic cues to clinical signatures 73

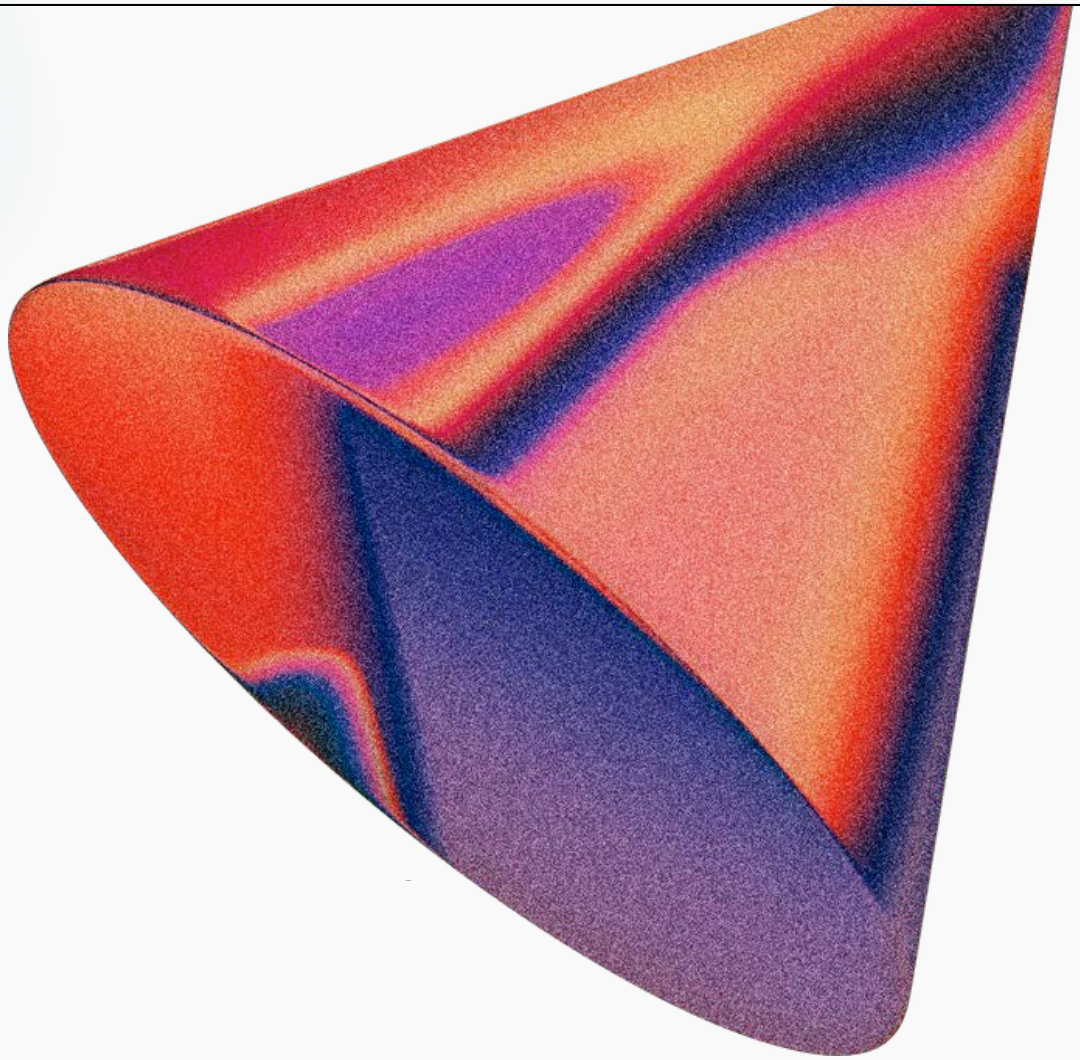
VOTING 74

FiRST AUTHORS INDEX 75

ADAS 77

ACIDUL 78

ÉCOLE DOCTORALE 80



KEYNOTE LECTURES

KEY LECTURE 1

9:10-9:40

L.1. S. GARCIA-SANTAMARIA

ITQB-IGC, PORTUGAL

Common therapeutics induce emergent behaviors on in vitro gut bacterial communities

Commonly prescribed therapeutics can change our gut microbiome composition and function. Reciprocally, the metabolic activity of the gut microbiome can alter the bioavailability, efficacy, mode of action and secondary effects of therapeutics. Understanding how drugs and gut microbes interact is crucial to design improved personalized therapies that take into account the interpersonal variability in gut microbiome composition and genetic repertoire.
High-throughput in vitro approaches have provided the first systematic views of direct gut microbiome-drug interactions. Recent reports have showcased that many non-antibiotics directly inhibited the growth of specific gut microbial species in monocultures. Yet, in our gut microbiome, bacteria are not present in isolation but co-exist in communities. Here, I will show the results of a recent screening aimed to assess how many of these drug-microbe interactions transpire in complex communities, the effects the community provides to drug responses, and mechanisms of interaction.

KEY LECTURE 2

11:10-11:40

L.2. J. DUBROVSKY

Universidad Nacional Autónoma de México, MEXICO

A story of founder cells

Rapid lateral root (LR) branching is essential for seedling establishment, the root system architecture, and plant growth. Regardless of the importance of LR development for proper function of root systems, even in a model species, Arabidopsis thaliana, our understanding of LR primordium (LRP) initiation and morphogenesis is fragmentary. How many pericycle founder cells (FCs) give rise to an LR, in what way they are involved in initiation process, whether all FCs are activated simultaneously, and whether LRP morphogenesis is somehow linked to the initiation process are all unanswered questions. To address them we first performed a clonal analysis and deduced that in most cases, just one pericycle FC starts LRP formation. This conclusion suggested (1) the existence of a stage 0 of LRP formation that precedes the first anticlinal symmetric and next asymmetric divisions leading to stage I LRP and (2) that after LRP initiation, new FCs should be gradually recruited from pre-existing pericycle cells. Next, to understand how these processes take place in vivo and confirm these possibilities, we performed time-lapse laser confocal microscopy experiments of long duration (24 and 50 h) using live root imaging. We developed “Live Plant Cell Tracking” (LiPlaCeT, https://doi.org/10.1093/plphys/kiab530) plugin coupled to the publicly available ImageJ image analysis program and generated a pipeline that allowed 4D cell tracking and lineage analysis of LRP cells. With this analysis we confirmed that in most cases the LRP initiation starts from a single FC and that after its specification, laterally and longitudinally adjacent FCs become gradually, progressively, and non-stereotypically recruited for LRP morphogenesis in an auxin-dependent manner. We also showed that the FCs are capable of gradual building a morphogenetic field contributing to LRP morphogenesis. This gradual recruitment of pericycle cells is spread longitudinally and tangentially and involves previously unrecognized phloem adjacent pericycle FCs. While laterally adjacent FCs become recruited in the LR formation, the descendants of previously involved and centrally located FCs pass through a number of cell divisions with very short, may be the shortest in plants, cell cycle. Thus, LRP morphogenesis takes place while FCs still become involved in the LR initiation. This analysis clearly showed that initiation is not a one-step process and that it is closely linked to the LRP morphogenesis.

KEY LECTURE 3

13:00-13:30

L.3. A. DECOTTIGNIES

UCLouvain, Brussels, BELGIUM

ALTernative lengthening of telomeres in cancer: diagnosis and therapeutic perspectives

Replicative immortality is currently considered one of the hallmarks of cancer cells. If this hold true for most cancer cells, recent evidence from us and others indicates that activation of a telomere maintenance mechanism (TMM) is not an absolute requirement for tumor formation and metastases. Yet we estimate that 80% of cancer cells are able to maintain the length of their telomeres. Expression of the telomerase gene is reactivated in the vast majority of tumor cells, while 5 to 10% of them set up an alternative telomerase-independent mechanism called ALT and based on homologous recombination mechanisms. However, the incidence of ALT is much higher in paediatric tumors, with approximately 30% of solid tumors with this TMM. Paediatric cancers urgently need new targeted therapies with reduced overall toxicity. Since ALT is not active in normal cells, its targeting for next generation anti-cancer therapies offers interesting perspectives. In the lab, we developed new diagnostic tools for the detection of ALT on tumor sections and have identified a putative molecular target to specifically kill ALT+ cancer cells.

KEY LECTURE 4

14:00-14:30

L.4. K. FOSTER

University of Oxford, UK

Competition and warfare in bacteria and the human microbiome

Life in a diverse microbial community, such as the human microbiome, can be highly competitive. Using a combination of theory and experiment, we study what it takes for bacteria to succeed in such communities. One way is to actively kill and inhibit competitors and I will discuss our latest work on the evolution of bacterial warfare. We are now also seeking to use our understanding of bacterial competition to manipulate gut communities for better health.

KEY LECTURE 5

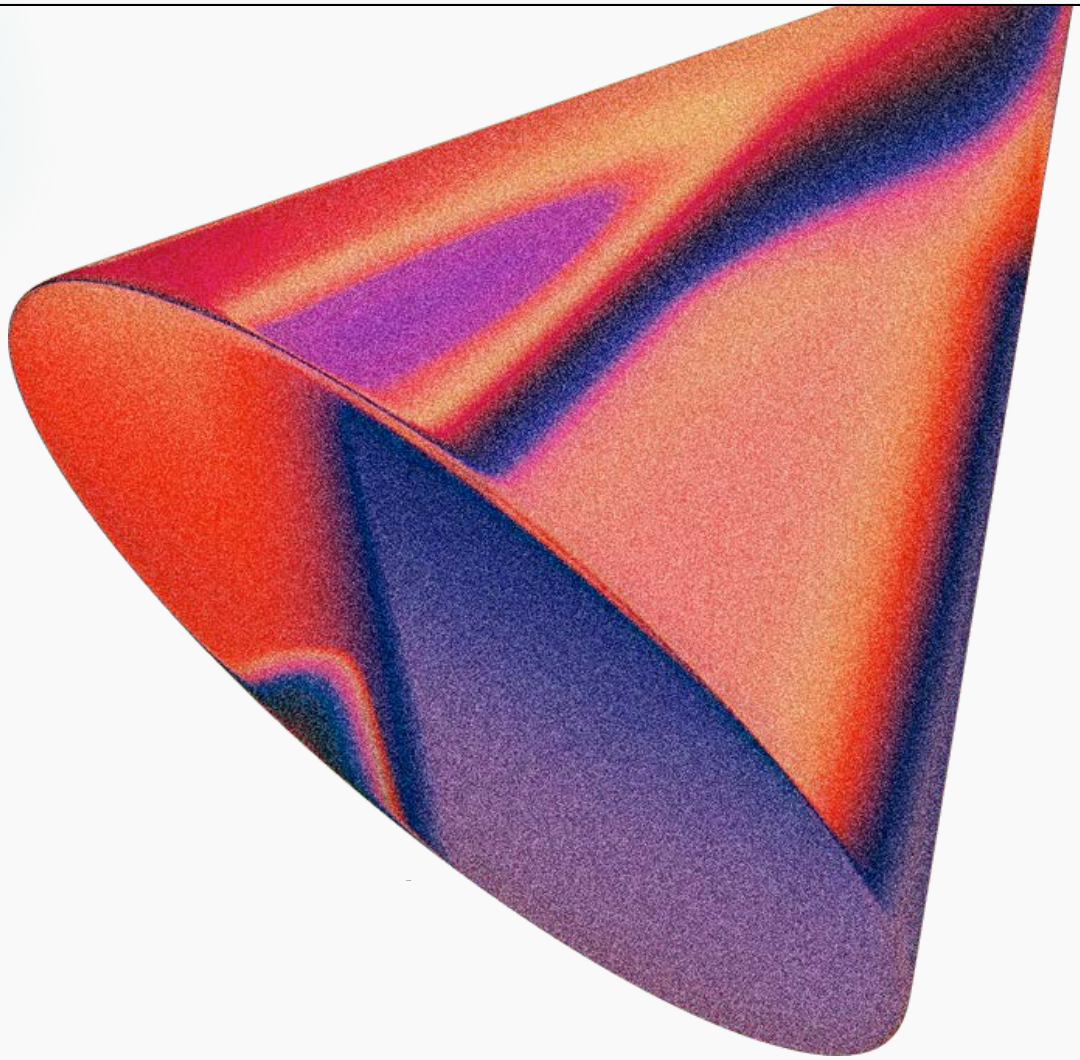
16:05-16:35

L.5. H. LASHUEL

EPFL, Lausanne, SWITZERLAND

Rising to the Challenge: ND BioSciences' innovative solutions for tackling the complexity of neurodegenerative diseases

More than a century has passed since pathological protein aggregates were first identified as defining hallmarks for several neurodegenerative diseases (NDDs), including Alzheimer's disease, and Parkinson's disease. Over the years, converging evidence pointed to a central role of protein aggregation in the initiation and progression of NDDs. This has led to the pursuit of different small molecule and antibody-based therapeutic strategies to prevent protein aggregation or stop pathology from spreading in the brain. Yet, we still do not have effective therapies to treat or slow the progression of these devastating diseases. I will reflect on some of the reasons underlying the drug development failures in NDDs, including the failure to embrace and reconstruct the neuropathological complexity and clinical heterogeneity of NDDs. Using Parkinson's disease as an example, I will present our unique technologies that allow for the first-time reproducing of the biochemical, structural, and functional properties of pathological protein aggregates observed in NDs. These unique capabilities are used by ND Biosciences to develop next-generation therapeutics and diagnostics for neurodegenerative diseases.



SELECTED ORAL PRESENTATIONS

SELECTED ORAL PRESENTATION 1

9:40-9:55

SOP.01. ALISON LIN 1,2,3, R. SCHELLING 2,3, D. VINYALS SALES1,2,3, P. NORTIER1,2,3, R. WINIGER2,3 AND L. PEREZ1,2,3

1 UNIVERSITY OF LAUSANNE (UNIL)
2 LAUSANNE UNIVERSITY HOSPITAL (CHUV), MEDICINE DEPARTMENT, SERVICE OF IMMUNOLOGY AND ALLERGY
3 CENTER FOR HUMAN IMMUNOLOGY LAUSANNE (CHIL), LAUSANNE, SWITZERLAND

Enhancement of immune response by nanoparticle vaccination

Vaccination is a cost-effective strategy in public health to limit the spread of infectious diseases. Nonetheless, conventional vaccines are not always effective in children and the elderly population. Moreover, vaccine protection requires multiple boosts to maintain efficient immunity against pathogens. Herein, we aim to develop a novel vaccination platform to increase the immune response by using the cross-linking of multiple B cell receptors and immune cell targeting. We propose a self-assembling protein nanoparticle (NP) presenting the main antigen (F protein) of the respiratory syncytial virus (RSV) and different co-activators known to be involved in B cells signaling (CD40L, IL-21 or APRIL). For this purpose, we used protein engineering, mice immunization, serological and viral neutralization assays to evaluate the different vaccine candidate's potency. We demonstrate that immunization with NP codisplaying CD40L and F-RSV generates an increase of immunoglobulin specific to RSV, whose titer was about 25-fold higher than the titer obtained with NP-F-RSV alone. This increase is also corresponding to the antibodies' potency to neutralize RSV. In contrast, other co-activators had a limited impact on immune function. Our approach presents an improvement of our previously developed nanoparticle platform that will be useful for poor vaccine responders such as immunocompromised individuals.

SELECTED ORAL PRESENTATION 2

11:40-12:00

SOP.02. SAMUEL MOIX1,3, C. AUWERX 1,2,3,4, M. SADLER1,3,4 AND Z. KUTALIK1,3,4

1 DEPARTMENT OF COMPUTATIONAL BIOLOGY, UNIL, LAUSANNE, SWITZERLAND
2 CENTER FOR INTEGRATIVE GENETICS, UNIL, LAUSANNE, SWITZERLAND
3 SWISS INSTITUTE OF BIOINFORMATICS, LAUSANNE, SWITZERLAND
4 UNIVERSITY CENTER FOR PRIMARY CARE AND PUBLIC HEALTH, LAUSANNE, SWITZERLAND

Causes and consequences of telomere shortening: A Mendelian randomization study

Telomeres are repeated DNA sequences at the ends of chromosomes that shorten with each cell division, ultimately leading to cell senescence and contributing to the aging process. Factors modulating telomere attrition and their consequences on human health remain poorly understood. We used linear regression, bidirectional univariable (MR), and multivariable Mendelian randomization (MVMR) to assess the relationships between leukocyte telomere length (TL) and 147 complex traits - including diseases, biomarkers, and lifestyle factors - in 326,363 unrelated white UK Biobank participants. Our results recapitulate telomere shortening with age ($p<2.2e-16$) and show a stronger (pdiff.=5.5e-28) decline in males (betamales=-0.026) than in females (betafemales=-0.021). Although both sex hormone-binding globulin (pdiff.=2.2e-18) and lifestyle factors such as fruit intake (pdiff.=9.6e-4) or smoking (pdiff.=3.6e-3) showed different association with TL in males versus females, accounting for them did not fully explain the difference in rate of attrition between sexes. After correcting for age, age2, and sex, 95 traits significantly associated with TL. Negative associations were found with cardiovascular, pulmonary, and renal conditions, while positive associations were observed with lipoprotein levels, female reproductive traits, and cancers. Inverse-variance weighted (IVW) MR identified 27 traits causally affected by TL, such as kidney cancer (alphaivw=0.048; p=8.1e-10), forced vital capacity (alphaivw=0.073; p=3.2e-6), and systemic lupus erythematosus (alphaivw=0.167; p=5.8e-5), and 23 traits causally influencing TL, including drinking (alphaivw=-0.086; p=1.3e-4), smoking (alphaivw=-0.142; p=1.8e-4), educational attainment (alphaivw=0.075; p=2.2e-15), and BMI (alphaivw=-0.048; p=4.9e-10), the effect of the latter on TL being potentially mediated by age at last birth and educational attainment. These results provide new insights into the biology of telomeres by distinguishing between modulators, mediators, and consequences of telomere shortening, portraying intricated relationships between TL, diseases, lifestyle, and socio-economic factors.

SELECTED ORAL PRESENTATION 3

13:30-13:45

SOP.03. C. SCHMITT-KOOPMANN1,
C. BAUD2, V. JUNOD2,3, O. SIMON1
1 SERVICE OF ADDICTION MEDICINE, LAUSANNE UNIVERSITY HOSPITAL (CHUV) AND UNIVERSITY OF LAUSANNE, LAUSANNE, SWITZERLAND
2 FACULTY OF BUSINESS AND ECONOMICS, UNIVERSITY OF LAUSANNE, LAUSANNE, SWITZERLAND.
3 FACULTY OF LAW, UNIVERSITY OF GENEVA, GENÈVE, SWITZERLAND.

Switzerland's narcotics regulation jungle

The word "narcotic" is often first associated with "illicit drugs". Yet, many "narcotic" and psychotropic substances are, in fact, medicines. Controlled medicines (CM) are products that meet the legal definition of both a "narcotic" under the Swiss Narcotics Act and of a medicine under the Therapeutic Products Act.
Our research examines how similar and how different, respectively, the implementation of CM regulations is throughout Switzerland. Based on a legal analysis of the cantonal regulations, we conducted semi-structured interviews with cantonal pharmacists and cantonal physicians to determine how they perceive and implement the federal legal requirements.
We find that some of these requirements have fallen into disuse, notably the federal duty to notify off-label use of CM. Moreover, we detect different cantonal conditions for opioid agonist treatment (OAT) authorization. In a majority of cantons, all physicians with a license to practice are allowed to prescribe OAT. In a few cantons, only so-called indication physicians, who have specific experience in OAT, can set the indication, even though another physician will treat the patient. Lastly, one canton requires physicians to follow specific cantonal training before prescribing OAT.
Our mapping of the CM regulation implementation can serve as a basis for cantons to review their implementation.

We will present intermediary results from a more extensive research project of which the pilot study was already published
(DOI: 10.3390/ijerph182413164)

SELECTED ORAL PRESENTATION 4

13:45-14:00

SOP.04. B. KRUMM1, C. LUNDBY2,3, J. HANSEN2, J. BEJDER3, T. EQUEY4, J.J. SAUGY1, F. BOTRÈ1, AND R. FAISS1

1REDS, RESEARCH & EXPERTISE IN ANTIDOPING SCIENCES, INSTITUTE OF SPORT SCIENCES, UNIVERSITY OF LAUSANNE, SWITZERLAND 2INLAND UNIVERSITY OF APPLIED SCIENCES, LILLEHAMMER, NORWAY 3DEPARTMENT OF NUTRITION, EXERCISE AND SPORT, UNIVERSITY OF COPENHAGEN, DENMARK 4WADA, WORLD ANTI-DOPING AGENCY, MONTRÉAL, CANADA

Plasma volume variations in elite athletes

INTRODUCTION: The Athlete Biological Passport (ABP) provides individual and adaptative monitoring of multiple biomarkers in anti-doping; notably hematological variables altered by various confounders such as physical exercise or extreme environments (e.g., heat or altitude exposure). Consequently, to consider plasma volume (PV) shifts inherent in athletes' daily routines, this study aimed to test the validity of a novel multi-parametric approach using serum markers to estimate PV changes, and correct concentration-based ABP parameters (e.g. [Hb]). It was hypothesized that the model validated in athletes over short periods (weeks) would remain valid for integrating PV shifts in the ABP of elite athletes over 12 months. METHODS: Full blood counts were performed monthly during one year using flow cytometry (Sysmex XN-1000) in 20 elite cyclists. Subsequently, individual profiles were generated through the official ABP software (World Anti-Doping Agency, ADAMS). Additionally, eight serum biomarkers were measured as 'volume-sensitive' variables to run a multi-parametric model providing corrected ABP thresholds through the estimates of PV shifts. Within-subject coefficients of variations were calculated to assess the variability of the serum variables. RESULTS: Eight Atypical Passport Findings (ATPF) (i.e., values beyond individual threshold limits) were generated in the study cohort. Among these 8 ATPFs, only 2 could be removed after the correction of ABP thresholds. Meanwhile, further ATPFs were generated after correction (n=14). Furthermore, a higher variability of 'volume-sensitive' markers was observed in elites ($P < 0.001$). DISCUSSION & CONCLUSION: In contrast to findings obtained in previous studies, the application of a multi-parametric model including 'volume-sensitive' serum biomarkers to estimate PV variations provided a lesser correction ability of individual ABP thresholds, and has even highlighted additional ATPFs. Overall, this study outlines the need to investigate novel approaches to better assess PV variations when interpreting individual ABP profiles.

SELECTED ORAL PRESENTATION 5

14:30-14:45

SOP.05. MAI THU NGUYEN, THOMAS MARTIGNIER, JOHN R. PANNELL

DEPARTMENT OF ECOLOGY AND EVOLUTION, UNIVERSITY OF LAUSANNE, 1015 LAUSANNE

The genetic architecture, evolution and stability of populations where females, males and hermaphrodites coexist

The co-existence of females, males and hermaphrodites, also known as trioecy, is rare and not predicted by theory. However, puzzling cases of trioecious populations do exist in nature and are still poorly understood. Here, we presented data on sex ratios and geographical distribution of 36 trioecious Mercurialis annua populations. We performed crossing experiments and simulations to infer the genetic inheritance of maleness and femaleness. We showed that maleness is determined by a Y chromosome while femaleness arises from recessive loci, with possible cytotype variation. We built a mathematical model investigating the evolution and stability of trioecy. In contrast with current models, which investigate cytoplasmic male sterility mutation (CMS) in the context of invading a population comprised of only hermaphrodites, our model investigates the invasion of CMS into a population where males and hermaphrodites already coexist (androdioecy). We compared the invasion and fixation of CMS into an androdioecious population with that into a hermaphroditic population. We show that the existence of males hinders the invasion of CMS and allows a large buffer parameter space between CMS invasion and fixation, which allow trioecy to arise.

SELECTED ORAL PRESENTATION 6

14:45-15:00

SOP.06. ANNA N. GLAUS1, MARION BRECHET1, GITI GHAZI SOLTANI1, LUDIVINE LEBEIGLE1, JOSÉ M. JIMÉNEZ-GÓMEZ2 AND SEBASTIAN SOYK1

1DEPARTMENT OF PLANT MOLECULAR BIOLOGY, UNIVERSITY OF LAUSANNE, LAUSANNE, SWITZERLAND
2CENTRO DE BIOTECNOLOGÍA Y GENÓMICA DE PLANTAS (CBGP), MADRID, SPAIN

Adaptation of plant architecture during domestication

Understanding the genes and genetic networks that regulate shoot architecture is essential for crop improvement. In flowering plants, the conserved flowering hormone, florigen, and its antagonist, anti-florigen, interact with basic leucine zipper (bZIP) transcription factors to regulate flowering and shoot architecture. However, it remains elusive to which extend mutations in the florigen pathway influenced shoot architecture during domestication. We identified a paralog of the tomato bZIP transcription factor SUPPRESSOR OF SP (SSP), which we named SSP2. Compared to ancestral accessions, SSP2 has a non-synonymous mutation in domesticated accessions . We provide evidence that this mutation affects the DNA binding capacity of SSP2 and leads to a reduction of its transcription factor activity. In order to understand the effect of this mutation on a phenotypic level, we applied CRISPR-Cas9 base-editing to repair the mutation in domesticated tomato. We found that functional SSP2 reduces the number of sympodial units and the number of flowers per inflorescence suggesting that its function not only affects shoot but also inflorescence architecture. Furthermore, we started to investigate the cis-regulatory regions that are bound by SSP2 and its paralog using DNA affinity purification sequencing (DAP-seq). Understanding the phenotypic and molecular consequences of this domestication mutation will provide us with novel insights into bZIP transcription factors networks and how mutations in bZIP transcription factors affected shoot architecture during domestication.

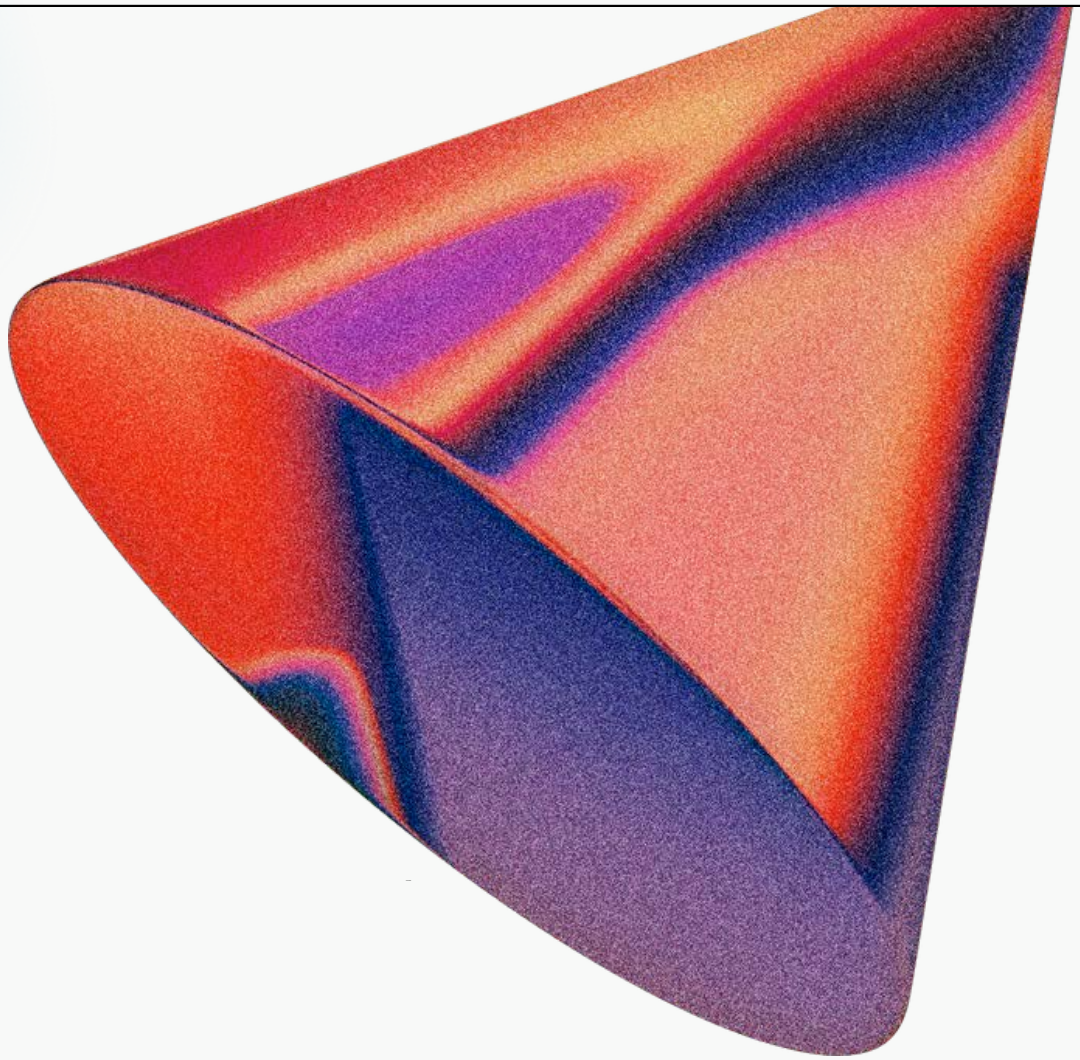
SELECTED ORAL PRESENTATION 7

15:50-16:05

SOP.07. PHILIPP WALCH, PETR BROZ
DEPARTMENT OF IMMUNOBIOLOGY, UNIVERSITY OF LAUSANNE, SWITZERLAND

Dissecting the impact of enteric viral-bacterial co-infection on the host innate immune response and its implications for pathogenicity

Understanding how pathogens cause and maintain infection is essential to develop novel therapeutics and prevent outbreaks of emerging diseases. Infectious diseases have long been treated as a threat we had successfully overcome, yet the prevalence of multi-resistant bacterial strains, the SARS-CoV-2 pandemic, and an expansion of endemic regions for tropical diseases underline the urgency of further research. While the broadening of accessible methodologies has enabled mechanistic insights, leading to the identification of new targets to disrupt infection, ongoing research heavily focuses on single pathogen infections. By contrast, little is known about the molecular mechanisms underlying co-infections, including their detection by host pattern recognition receptors and their effect on host innate immune responses. From a clinical perspective however, co-infections are highly relevant, as they occur frequently and generally exacerbate symptom severity and fatality. Here, I am describing the first systematic mapping of enteric host-pathogen-pathogen interactions, deepening our molecular understanding of the effect pathogens have on each other, on how they are recognized by the host and on the initiation of innate immune defenses, such as the activation of inflammasomes and various forms of cell death. Interestingly, the directionality of interaction, i.e. synergies versus antagonisms, is dependent on the specific viral-bacterial pair used in co-infection, infection dynamic, i.e. simultaneous versus subsequent infection, as well as host cell type and Interferongamma priming. I have generated a vast, unbiased dataset of pathogen-pathogen interactions during infection, validated a subset, and, in a subsequent step, I explored the underlying mechanisms for pathogen interaction, revealing interaction points along the course of the infection. Using a broad panel of methodologies, including proteomics, FACS, microscopy and classical biochemical assays, I am currently characterizing these interaction points to yield a mechanistic explanation for the observed interactions.



PITCHES

SELECTED PITCH 1

9:55-9:59

SPI.01. PAUL-EMMANUEL VANDERRIELE1,3
GILLES AESCHLIMANN1, SOFIA VEROUTI2,3, QING WANG4, FRÉDÉRIQUE INO1, DENISE V. KRATSCHMAR3,5, ALEX ODERMATT3,5, EDITH HUMMLER1,3

1. DEPARTMENT OF BIOMEDICAL SCIENCES, UNIVERSITY OF LAUSANNE, 1015 LAUSANNE, SWITZERLAND.
2. DEPARTMENT OF NEPHROLOGY AND HYPERTENSION, INSELSPITAL, CH-3010 BERN, SWITZERLAND
3. NATIONAL CENTER OF COMPETENCE IN RESEARCH KIDNEY.CH, 1011 LAUSANNE, SWITZERLAND.
4. DIVISION OF NEPHROLOGY AND HYPERTENSION, LAUSANNE UNIVERSITY HOSPITAL (CHUV), 1015 LAUSANNE, SWITZERLAND.
5. DIVISION OF MOLECULAR AND SYSTEMS TOXICOLOGY, DEPARTMENT OF PHARMACEUTICAL SCIENCES, UNIVERSITY OF BASEL, 4056 BASEL, SWITZERLAND.

The glucocorticoid receptor is likely involved in the development of the salt-sensitive hypertension through the deregulation of the soluble epoxide hydrolase (sEH) in rats.

Background: Salt is everywhere in food and an excess intake can lead to hypertension-development. In rats carrying a glucocorticoid receptor (GR) mutation leading to haploinsufficency (GR+/em2), a five weeks treatment of high salt diet (HSD) resulted in adrenal dysregulation of the soluble epoxide hydrolase (sEH) followed by an impairment in the degradation of the omega 3 and 6 fatty acids. GR-mutant rats developed salt-sensitive hypertension1.
Aim: To determine the role of the DNA-binding domain of the GR in the development of salt-sensitive hypertension in a transgenic rat model harboring an in frame mutation with a deletion of the exon 3 that resulted in a DNA-binding deficient receptor2.
Methods: Three salty diets are used in different groups: normal diet (0,3%), short term HSD (8% for 3 days), long term HSD (6% for 5 weeks).
ollowing these diets, blood pressure has been measured, adrenal proteins were extracted and used for Western-blot analyses, and the dilatation and/or contraction of aorta and kidney vessels were determined using pharmacological stimulants/blockers.

Results: On a normal salt diet, GR+/em4 rats presented a significant deregulation of the plasmatic corticosterone and 11-dehydrocorticosterone levels, an adrenal hyperplasia and a cardiac hypoplasia.
Following short term HSD, GR+/em4 rats exhibited a significant deregulation of the 11-dehydrocorticosterone and aldosterone. Furthermore, adrenal sEH was significantly decreased in comparison to wild-type (WT) rats.
Following a long-term HSD, GR+/em4 rats developed salt-sensitive hypertension associated with significant deregulation of adrenal and aortic sEH protein abundance.
Conclusions: Our data strongly suggest that abnormal adrenal and aortic sEH protein abundance is implicated in salt-sensitive hypertension in our rats.

SELECTED PITCH 2

9:59-10:03

SPI.02. P. GUT1,2, S. SRIDI3, F. SACHER1,4, P. JAÏS1,4, H. COCHET1,3, M. STUBER1,2, A. BUSTIN1,2,3

1IHU LIRYC, UNIVERSITÉ DE BORDEAUX, FRANCE; 2DEPARTMENT OF DIAGNOSTIC AND INTERVENTIONAL RADIOLOGY, UNIVERSITY HOSPITAL OF LAUSANNE, SWITZERLAND; 3DEPARTMENT OF CARDIOVASCULAR IMAGING, HÔPITAL CARDIOLOGIQUE DU HAUT-LÉVÊQUE, CHU DE BORDEAUX, FRANCE; 4DEPARTMENT OF CARDIAC PACING AND ELECTROPHYSIOLOGY, HÔPITAL CARDIOLOGIQUE DU HAUT-LÉVÊQUE, CHU DE BORDEAUX, FRANCE

Wideband black-blood late gadolinium enhancement imaging for patients with cardiac implantable electronic devices

Purpose: Here we propose a technology to image myocardial infarction with wideband black-blood late gadolinium enhancement (LGE) imaging in patients with implantable cardioverter defibrillator (ICD).

Methods: A 2D breath-hold single-shot ECG-triggered gradient echo wideband black-blood sequence was implemented. Black-blood images were generated by a non-selective adiabatic hyperbolic secant (HS) 180° inversion recovery (IR) pulse (duration = 10.24ms) followed by an adiabatic T2 preparation module (T2p) [1]. The adiabatic T2p consists of a 90° tip-down pulse of two adiabatic HS refocusing pulses (duration = 12.8ms each), and of a 90° tip-up pulse [2]. To eliminate the hyperintensity artefacts from the non-properly inverted myocardium signal in the presence of CIED [3], the HS IR bandwidth was increased from 0.8 to 9.2kHz with a peak B1 amplitude of 30μT and the bandwidth of the two T2p refocusing pulses were increased from 1.6 to 5.0kHz with a peak B1 amplitude of 30μT.

Reference standard bright-blood PSIR LGE [4] and wideband black-blood LGE imaging were prospectively tested on a 1.5T system (MAGNETOM Aera, Siemens) in xx patients (x female; mean age xx) with MRI-conditional ICD and with following sequence parameters: FOV=300x225mm2, resolution=1.4x1.4mm2, slice thickness=8mm, flip angle=15°, TE/TR=2.7/4.8ms, acceleration GRAPPA of factor 2 with 36 reference k-space lines, phase partial Fourier=7/8, readout bandwidth=751Hz/pixel, 5 signal averages. One expert radiologist scored the subjective image quality (1 = non-diagnostic, 2 = less than adequate, 3 = adequate, 4 = excellent) and the overall ICD-artefact severity (ripple and hyperintensity artefacts) (1 = non-diagnostic with severe ICD artefacts, 2 = less than adequate with large ICD -

-artefacts, 3 = adequate with moderate ICD artefacts, 4 = excellent with minimal ICD artefacts). Furthermore, contrast ratios between scar and healthy myocardium signals and between scar and blood signals were calculated.

Results: Reference standard bright-blood PSIR images were highly affected by hyperintensity and ripple ICD-related artefacts (score: xx), making the diagnosis unreliable or impossible. Wideband black-blood provided images with suppressed hyperintensity and ripple ICD-related artefacts (score: xx), allowing a better visualization of the heart and myocardial infarction. Image quality was better on the proposed wideband black-blood (wideband black-blood: xx, PSIR: xx). Contrast ratios on wideband black blood was xx% higher than PSIR between scar and healthy myocardium signals and xx% higher than PSIR between scar and blood signals. Wideband black-blood technique provided enhanced scar contrast and could identify xx% more hyperenhanced segments than PSIR.

Discussion: Wideband black-blood LGE provided images with suppressed hyperintensity and ripple artefacts from the ICD and with higher scar contrast than standard PSIR, allowing more reliable and more accurate myocardial scar assessment.

References: [1] Sridi S, et al., Diagn Interv Imaging 2022 Dec; 103(12):607-617. [2] Nezafat R, et al., Magn Reson Med 2006 Apr; 55(4):858-64. [3] Rashid S, et al., Radiology 2014 Jan; 270(1):269-74. [4] Kellman P, et al., Magn Reson Med 2002 Feb; 47(2):372-83.

SELECTED PITCH 3

10:03-10:07

SPI.03. SILVIA PRIETO BAÑOS1,2, YANNIS NEVERS1,2, CHRISTOPHE DESSIMOZ1,2 AND NATASHA GLOVER1,2

1DEPARTMENT OF COMPUTATIONAL BIOLOGY, UNIVERSITY OF LAUSANNE, 1015 LAUSANNE, SWITZERLAND 2SIB SWISS INSTITUTE OF BIOINFORMATICS, 1015 LAUSANNE, SWITZERLAND

Annotation matters: the effect of gene annotation on orthology

Computational protein-coding gene annotation, i.e. finding the genes present in a genome, remains a challenging task. The quality of gene models and gene repertoire lags behind the pace at which available genome assemblies are increasing. Although annotation methods are improving, there are no community standards and in practice, most published gene annotations result from ad hoc or customised pipelines. As a result, only a few non-model species have complete and accurate gene models. Annotation quality affects downstream analyses, including comparative genomics. Yet there is no focus in understanding the impact of annotation methods choice on orthology inference, a pre-requisite for comparative genomic studies. Here, we show that different annotation methods render different orthology results. We ran OMA Standalone to infer the orthology between Chordata proteomes which we obtained with four commonly used annotation pipelines (Augustus 3.4 de-novo, the NCBI Eukaryotic Pipeline, the Ensembl gene annotation system and UniProt's Reference Proteomes). Results show important differences in the orthology inferred from each of the four proteomes sets, namely in the number of orthology pairs and on the accuracy of orthology prediction reported by a standard benchmark.

SELECTED PITCH 4

10:07-10:11

SPI.04. ARNAUD LYON1,2; THOMAS AGIUS2; KEVIN KIESWORO2; SÈNAN D'ALMEIDA3; SOPHIE DE SEIGNEUX4; THOMAS VERISSIMO4; SÉBASTIEN DÉGLISE2; FLORENT ALLAGNAT2; MICHAEL MACARTHUR5, SARAH J. MITCHELL5, ALEJANDRO OCAMPO6, ALBAN LONGCHAMP2#; DÉLA GOLSHAYAN1#

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2DIVISION OF VASCULAR SURGERY, LAUSANNE UNIVERSITY HOSPITAL AND UNIL, LAUSANNE, SWITZERLAND.
3FLOW CYTOMETRY CORE FACILITY, EPFL, LAUSANNE, SWITZERLAND.
4DIVISION OF NEPHROLOGY, GENEVA UNIVERSITY HOSPITALS, GENEVA, SWITZERLAND.
5DEPARTMENT OF HEALTH SCIENCES AND TECHNOLOGY, ETH, ZURICH, SWITZERLAND.
6DEPARTMENT OF BIOMEDICAL SCIENCES, UNIL, LAUSANNE, SWITZERLAND.
# CONTRIBUTED EQUALLY

Immune-mediated improved tolerance to kidney and liver ischemia-reperfusion injury in female mice.

Background. Previous experimental and clinical studies have highlighted sex-specific susceptibility to ischemia-reperfusion injury (IRI) in multiple organs. While sex hormones seem to be important, the precise underlying mechanisms still need to be uncovered.
Methods. IRI was modelled in mouse kidneys using unilateral nephrectomy, followed by 23min ischemia and reperfusion of the controlateral kidney, and in livers by applying 35min ischemia and reperfusion. Post-operative kidney and liver function was evaluated over time by transcutaneous assessment of FITC-Sinistrin clearance and by plasma transaminases levels, respectively. Histology, immunohistochemistry and Spatial total mRNA sequencing was carried out on paraffin-embedded tissue. qPCR was performed on RNA extracted from flash-frozen organ samples. Time-course CyTOF was applied on PBMCs isolated from whole blood.

Results. Following IRI, female mice had significantly better renal and liver function. Renal histology showed increased tubulointerstitial lesions in males, together with significantly increased expression of IL-6 and TNF-alpha. Consistently, the secretion of pro-inflammatory cytokines was followed by a significantly higher increase of blood neutrophils in males at postoperative day 2. In male kidneys, this was associated with the infiltration of an immune cluster at the corticomedullary junction and downregulation of metabolism, proliferation and survival related genes in proximal tubules.
Conclusions. Our data show an increased recruitment of neutrophils at the onset of IRI, particularly in male kidneys. IL-6 and TNF-alpha seem to be sex-specific key regulators of this early immune response. Therapeutic targeting of these pathways could translate into clinical trials to improve graft outcome

SELECTED PITCH 5

10:11-10:15

SPI.05. TOM CITHERLET, ANTOINE RABERIN, GIORGIO MANFERDELLI, GRÉGOIRE P. MILLET

INSTITUTE OF SPORT SCIENCES, SYNATHLON, UNIVERSITY OF LAUSANNE, LAUSANNE, SWITZERLAND.

Is cortisol awakening response related to Acute Mountain Sickness in eumenorrheic and postmenopausal women?

Cortisol rises at altitude, but little is known about its relationship with Acute Mountain Sickness (AMS) in women, particularly in those who are menstruating or menopausal. Therefore, this study aimed to compare the cortisol awakening response and its correlation with AMS in these two groups of women.

Thirteen Eumenorrheic Women (EW) and fifteen Postmenopausal Women (PW) spent one night at altitude (3375 m) with AMS assessment. Cortisol levels were evaluated on the morning of ascent at Low Altitude (LA) and the morning after sleeping at High Altitude (HA). Saliva samples were collected at 0 min (C1), 30 min (C2), and 45 min (C3) after awakening. Three indices were calculated: First awakening sample (C1), the area under the cortisol rise curve relative to C1 (CAR1), and the area under the cortisol rise curve relative to 0 (CAR2).
The prevalence of AMS in EW and PW was 30.8% and 40.0%, respectively, 6 hours after arrival, and 15.4% and 26.7% one night after arrival. When AMS positive and AMS negative subgroups were compared, no difference in any of the cortisol parameters was found, neither for value after 6h or one night. A sub-analysis of EM and PW separately found no differences either. There was no significant difference between these two age groups.
The findings of the study suggest that there is no significant relationship between cortisol levels and AMS in EW and PW. These results are in line with Woods et al. (2012) but not with several other works that found increased cortisol levels in AMS-prone participants (Richalet et al., 1989; Estoppey et al., 2019; Gatterer et al., 2019). Different results can be due to exposure duration and altitude, and levels of physical exertion. Further research could evaluate if a sex-specific stress response explains the differences between our results and most previous investigations.

SELECTED PITCH 6

10:15-10:20

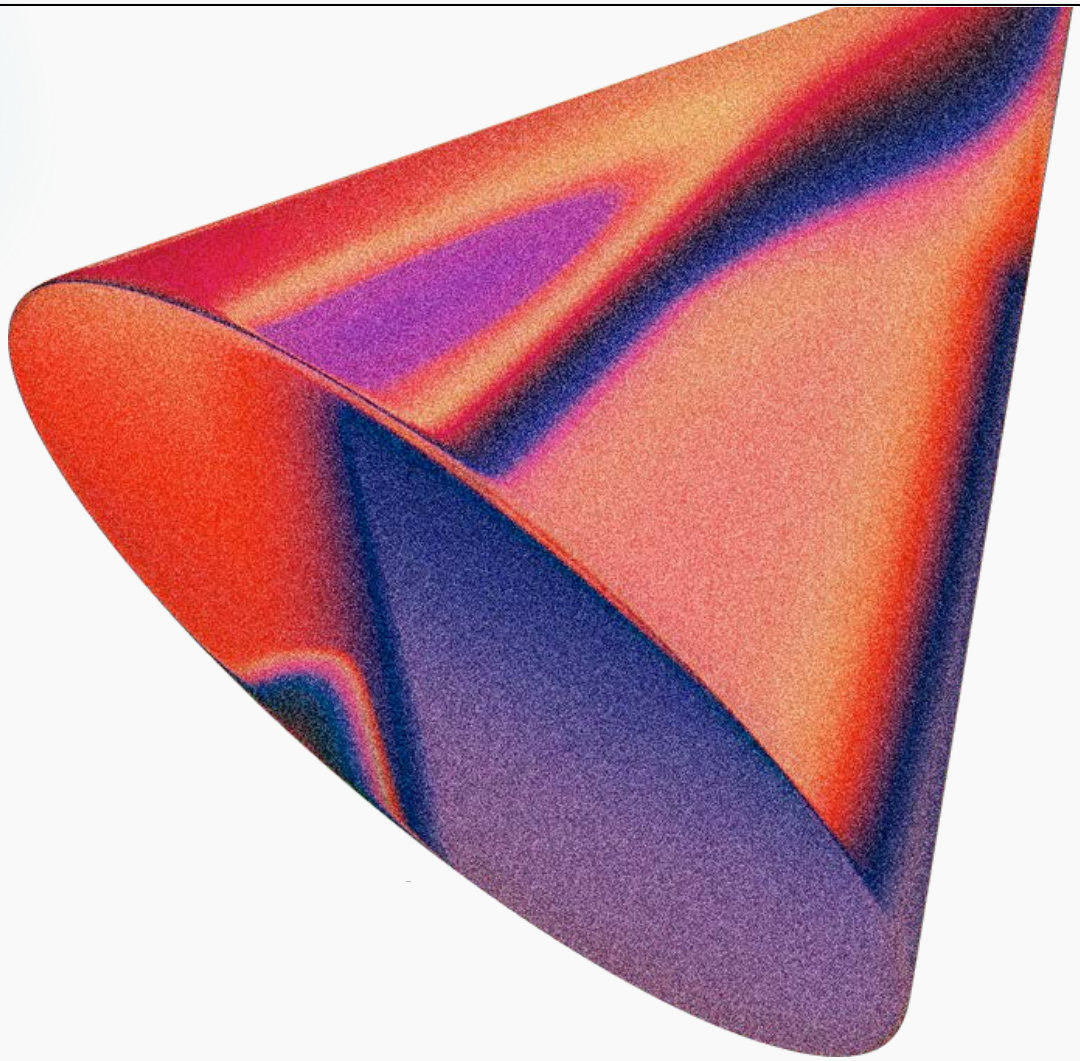
SPI.06. SARAY RAMOS2, JOSÉ CARLOS SANTOS2, PETR BROZ2, MORRIS DEGEN1, KRISTYNA PLUHACKOVA3,* GONZALO CEBRERO1, GYTIS JANKEVICIUS1, ELLA HARTENIAN2, UNDINA GUILLERM5, BASTIAN KOHL1, DANIEL J. MÜLLER4, PAUL SCHANDA5, TIMM MAIER1, CAMILO PEREZ1, CHRISTIAN SIEBEN6,7,* AND SEBASTIAN HILLER1,*

1BIOZENTRUM, UNIVERSITY OF BASEL, 4056 BASEL, SWITZERLAND
2DEPARTMENT OF IMMUNOBIOLOGY, UNIVERSITY OF LAUSANNE, 1066 EPALINGES, SWITZERLAND
3STUTTGART CENTER FOR SIMULATION SCIENCE, CLUSTER OF EXCELLENCE EXC 2075, UNIVERSITY OF STUTTGART, 70569 STUTTGART, GERMANY
4DEPARTMENT OF BIOSYSTEMS SCIENCE AND ENGINEERING, EIDGENÖSSISCHE TECHNISCHE HOCHSCHULE (ETH) ZURICH, 4058 BASEL, SWITZERLAND
5INSTITUTE OF SCIENCE AND TECHNOLOGY AUSTRIA (ISTA), 3400 KLOSTERNEUBURG, AUSTRIA
6NANOSCALE INFECTION BIOLOGY GROUP, DEPARTMENT OF CELL BIOLOGY, HELMHOLTZ CENTRE FOR INFECTION RESEARCH, 38124 BRAUNSCHWEIG, GERMANY
7INSTITUTE FOR GENETICS, TECHNISCHE UNIVERSITÄT BRAUNSCHWEIG, 38124 BRAUNSCHWEIG, GERMANY

The structural basis for ninjurin-1 mediated plasma membrane rupture in inflammatory cell death

Eukaryotic cells can undergo different forms of programmed cell death, many of which culminate in plasma membrane rupture (PMR) as the defining terminal event. PMR was long thought to be driven by osmotic pressure, until it was recently shown to be an active process, mediated by the protein ninjurin-1 (NINJ1). Here, we resolve the structure of NINJ1 and the mechanism by which it ruptures membranes. Super-resolution microscopy reveals that NINJ1 assembles structurally diverse oligomers in dying cells, including rings, arcs, and filaments.

A cryo-electron microscopy structure of NINJ1 filaments shows a tightly packed fence-like array of transmembrane α-helices. Filament directionality and stability is defined by two amphipathic α-helices that interlink adjacent filament subunits. NINJ1 filaments feature a hydrophilic and a hydrophobic side, and consequently can cap membrane edges. The resulting stable supramolecular arrangements rupture the membrane, as shown by molecular dynamics simulations and validated by site-directed mutagenesis. The membrane protein NINJ1 is thus an interactive component of the eukaryotic cell membrane that functions as an in-built breaking point in response to activation of cell death.



POSTERS

SELECTED POSTER 1

THIS WORK WAS ALSO SELECTED FOR A TALK

SPO.01. C. SCHMITT-KOOPMANN1,
C. BAUD2, V. JUNOD2,3, O. SIMON1
1 SERVICE OF ADDICTION MEDICINE, LAUSANNE UNIVERSITY HOSPITAL (CHUV) AND UNIVERSITY OF LAUSANNE, LAUSANNE, SWITZERLAND
2 FACULTY OF BUSINESS AND ECONOMICS, UNIVERSITY OF LAUSANNE, LAUSANNE, SWITZERLAND.
3 FACULTY OF LAW, UNIVERSITY OF GENEVA, GENÈVE, SWITZERLAND.

Switzerland's narcotics regulation jungle

The word "narcotic" is often first associated with "illicit drugs". Yet, many "narcotic" and psychotropic substances are, in fact, medicines. Controlled medicines (CM) are products that meet the legal definition of both a "narcotic" under the Swiss Narcotics Act and of a medicine under the Therapeutic Products Act.
Our research examines how similar and how different, respectively, the implementation of CM regulations is throughout Switzerland. Based on a legal analysis of the cantonal regulations, we conducted semi-structured interviews with cantonal pharmacists and cantonal physicians to determine how they perceive and implement the federal legal requirements.
We find that some of these requirements have fallen into disuse, notably the federal duty to notify off-label use of CM. Moreover, we detect different cantonal conditions for opioid agonist treatment (OAT) authorization. In a majority of cantons, all physicians with a license to practice are allowed to prescribe OAT. In a few cantons, only so-called indication physicians, who have specific experience in OAT, can set the indication, even though another physician will treat the patient. Lastly, one canton requires physicians to follow specific cantonal training before prescribing OAT.
Our mapping of the CM regulation implementation can serve as a basis for cantons to review their implementation.

We will present intermediary results from a more extensive research project of which the pilot study was already published
(DOI: 10.3390/ijerph182413164)

SPO.02. GYDA FENN-MOLTU, ANDREW LIEBHOLD, DONALD C. WEBER, SEBASTIEN OLLIER, CLEO BERTELSMEIER

DEPARTMENT OF ECOLOGY AND EVOLUTION, UNIVERSITY OF LAUSANNE.

Accidental biocontrol: human-mediated dispersal of insect parasitoids and predators

Globalization of human activities has increasingly facilitated the spread of alien species. Insects are among the most numerous and damaging aliens, with widespread impacts on biodiversity, agriculture, and human health. The options to manage insect invasions include classical biological control, where species 'natural enemies' are introduced from their native range to control their population. Current selection processes for such biocontrol agents are rigorous, and generally based on host-specificity. However, parasitoid, and predatory species are also increasingly being introduced accidentally through human-mediated dispersal. These random introductions may favour generalists that establish more easily due to their broad host range, with considerable impacts for both non-native and native species. We used a large dataset of border interception records in the United States of America from 1913 to 2019 to assess the human-mediated transport of parasitoid and predatory insects. 94 families of 'natural enemies' were detected, and 175 species were identified. The insects largely arrived with various plant products, but introduction pathways differed between insect groups. Most of the species detected are generalists and are likely to have suitable prey or host species available on arrival. Several species that are not currently established in the USA have hosts or prey already established there, or also being detected at the border. Furthermore, ten of the 'natural enemy' species arriving in the USA are listed as invasive –spreading widely and causing negative impacts. The extensive transport of 'accidental biocontrol' insects stands in contrast to the rigorous and protracted processes for classical biological control. The insects detected at the US border could have significant ecological impacts, both positive and negative, if they eventually become established.

SELECTED POSTER 3

THIS WORK WAS ALSO SELECTED FOR A TALK

SPO.03. ANNA N. GLAUS1, MARION BRECHET1, GITI GHAZI SOLTANI1, LUDIVINE LEBEIGLE1, JOSÉ M. JIMÉNEZ-GÓMEZ2 AND SEBASTIAN SOYK1

1DEPARTMENT OF PLANT MOLECULAR BIOLOGY, UNIVERSITY OF LAUSANNE, LAUSANNE, SWITZERLAND
2CENTRO DE BIOTECNOLOGÍA Y GENÓMICA DE PLANTAS (CBGP), MADRID, SPAIN

Adaptation of plant architecture during domestication

Understanding the genes and genetic networks that regulate shoot architecture is essential for crop improvement. In flowering plants, the conserved flowering hormone, florigen, and its antagonist, anti-florigen, interact with basic leucine zipper (bZIP) transcription factors to regulate flowering and shoot architecture. However, it remains elusive to which extend mutations in the florigen pathway influenced shoot architecture during domestication. We identified a paralog of the tomato bZIP transcription factor SUPPRESSOR OF SP (SSP), which we named SSP2. Compared to ancestral accessions, SSP2 has a non-synonymous mutation in domesticated accessions . We provide evidence that this mutation affects the DNA binding capacity of SSP2 and leads to a reduction of its transcription factor activity. In order to understand the effect of this mutation on a phenotypic level, we applied CRISPR-Cas9 base-editing to repair the mutation in domesticated tomato. We found that functional SSP2 reduces the number of sympodial units and the number of flowers per inflorescence suggesting that its function not only affects shoot but also inflorescence architecture. Furthermore, we started to investigate the cis-regulatory regions that are bound by SSP2 and its paralog using DNA affinity purification sequencing (DAP-seq). Understanding the phenotypic and molecular consequences of this domestication mutation will provide us with novel insights into bZIP transcription factors networks and how mutations in bZIP transcription factors affected shoot architecture during domestication.

SELECTED POSTER 4

SPO.04. E. LANDALUCE-ITURRIRIA1, E.A. FERNANDEZ 1, M. JAN 1, R. DREOS 1, T. CAPUTO 1, B. DESVERGNE 1, N.GUEX 1 AND I.C. LOPEZ-MEJIA1

1CENTRE DE GÉNOMIQUE INTÉGRATIVE, UNIVERSITÉ DE LAUSANNE, SWITZERLAND

Macronutrition and signal-induced regulation of RNA splicing in adipose tissue depots

Obesity and insulin resistance (IR) currently represent a public health concern due to their high global incidence (more than 1 billion people worldwide). This high incidence results mainly from the increased consumption of highly caloric and energy dense foods, containing saturated fats and high levels of sugar. RNA splicing is an important gene expression regulation mechanism in eukaryotic cells that brings proteome diversity. Alterations in alternative splicing (AS) are known to be implicated in a wide range of human diseases, but the role of macronutrient and signalingcontrolled RNA splicing in the white adipose tissue (WAT), and whether AS alterations contribute to the development of metabolic disorders remains unknown. We used RNA sequencing to measure the expression of RNA binding proteins (RBPs) and AS events in vivo in WAT from mice fed a high fat diet (HFD), replicating conditions of obesity and IR, and in WAT from mice fed a control diet (CD). We identified alterations in the expression of Lgals3, Fmr1 and Rbfox2, amongst other RBPs; as well hundreds of AS alterations, including decreased inclusion of Slc22a17 exon 4. To study RBPs-AS networks in adipocytes at a molecular level, we established an in vitro model using 3T3L1 mature adipocytes treated with different stimuli to replicate the conditions found in IR and obesity patients (inflammation, high levels of FFAs, hyperglycemia, hyperinsulinemia...). Our results show that the mRNA and protein levels of Lgals3 are increased in response to inflammation and high insulinemia, while Fmr1 and Rbfox2 showed decreased mRNA expression, suggesting our in vitro models recapitulate the alterations observed in vivo. Our in vitro model will be used to analyze RBPs-AS networks in order to understand better how macronutrient and signaling-regulated AS can be key modulators of cellular and tissue function in the context of metabolism, and investigate whether RBPs or specific AS events can be targeted pharmacologically to treat obesity.

SELECTED POSTER 5

THIS WORK WAS ALSO SELECTED FOR A TALK

SPO.05. ARNAUD LYON1,2; THOMAS AGIUS2; KEVIN KIESWORO2; SÈNAN D'ALMEIDA3; SOPHIE DE SEIGNEUX4; THOMAS VERISSIMO4; SÉBASTIEN DÉGLISE2; FLORENT ALLAGNAT2; MICHAEL MACARTHUR5, SARAH J. MITCHELL5, ALEJANDRO OCAMPO6, ALBAN LONGCHAMP2#; DÉLA GOLSHAYAN1#

1TRANSPLANTATION CENTRE, LAUSANNE UNIVERSITY HOSPITAL AND UNIL, LAUSANNE SWITZERLAND.
2DIVISION OF VASCULAR SURGERY, LAUSANNE UNIVERSITY HOSPITAL AND UNIL, LAUSANNE, SWITZERLAND.
3FLOW CYTOMETRY CORE FACILITY, EPFL, LAUSANNE, SWITZERLAND.
4DIVISION OF NEPHROLOGY, GENEVA UNIVERSITY HOSPITALS, GENEVA, SWITZERLAND.
5DEPARTMENT OF HEALTH SCIENCES AND TECHNOLOGY, ETH, ZURICH, SWITZERLAND.
6DEPARTMENT OF BIOMEDICAL SCIENCES, UNIL, LAUSANNE, SWITZERLAND.
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Methods. IRI was modelled in mouse kidneys using unilateral nephrectomy, followed by 23min ischemia and reperfusion of the controlateral kidney, and in livers by applying 35min ischemia and reperfusion. Post-operative kidney and liver function was evaluated over time by transcutaneous assessment of FITC-Sinistrin clearance and by plasma transaminases levels, respectively. Histology, immunohistochemistry and Spatial total mRNA sequencing was carried out on paraffin-embedded tissue. qPCR was performed on RNA extracted from flash-frozen organ samples. Time-course CyTOF was applied on PBMCs isolated from whole blood.

Results. Following IRI, female mice had significantly better renal and liver function. Renal histology showed increased tubulointerstitial lesions in males, together with significantly increased expression of IL-6 and TNF-alpha. Consistently, the secretion of pro-inflammatory cytokines was followed by a significantly higher increase of blood neutrophils in males at postoperative day 2. In male kidneys, this was associated with the infiltration of an immune cluster at the corticomedullary junction and downregulation of metabolism, proliferation and survival related genes in proximal tubules.
Conclusions. Our data show an increased recruitment of neutrophils at the onset of IRI, particularly in male kidneys. IL-6 and TNF-alpha seem to be sex-specific key regulators of this early immune response. Therapeutic targeting of these pathways could translate into clinical trials to improve graft outcome

SPO.06. ÇAĞLA GÖRKEM EROĞLU1,2, ALEXANDRA BENNETT3, AURELIE GFELLER1, STEPHAN HANN3, JUDITH WIRTH1

1PLANT PRODUCTION SYSTEMS, AGROSCOPE, NYON, SWITZERLAND
2FBM, UNIL, LAUSANNE, SWITZERLAND
3DEPARTMENT OF CHEMISTRY, VIENNA, AUSTRIA

Presence of Neighbors Influences Buckwheat Root Exudate Composition

Plants interact with their neighbours through various complex mechanisms directly and indirectly. Direct interactions involve the production of biochemicals that influence germination, growth, and survival neighbours. Plant roots actively and passively release various primary (amino acids, organic acids, and sugars) and secondary (phenolics, flavonoids, terpenoids, and nitrogen-containing compounds) low-molecular-weight metabolites and high-molecular-weight metabolites (mucilage and proteins). Root exudates are key players in root-root and root-microorganism interactions. In the present study a split-root system that allows differential root exudate collection is established to elucidate belowground plant-plant interactions. Since the soil is a highly complex matrix, it creates noise and complicates chromatographic analyses. Most studies collect root exudates from hydroponic systems that cause potential root damage and osmotic stress. We designed a split-root system that uses non-complex soil-free media with minimized root damage and enables fast and simultaneous collection with filtering under controlled pressure and vacuuming. The split root systems were prepared by dividing the roots of a single buckwheat (BK) plantlet into two independent compartments equally for each condition where they were isolated from each other, and one of the compartments had either homospecific, heterospecific or no neighbour. While the roots of two plants were in direct contact in the compartment having a heterospecific or homospecific neighbour, there was no direct root contact in the other. Root exudates were successfully collected, untargeted metabolomics profiling was performed and the root parameters were analysed to provide insights into the effects of root interactions on root exudation profiles and root structure. Our results showed that the BK root exudation profile is impacted by the presence and identity of its neighbour.

SELECTED POSTER 7

THIS WORK WAS ALSO SELECTED FOR A TALK

SPO.07. TOM CITHERLET, ANTOINE RABERIN, GIORGIO MANFERDELLI, GRÉGOIRE P. MILLET

INSTITUTE OF SPORT SCIENCES, SYNATHLON, UNIVERSITY OF LAUSANNE, LAUSANNE, SWITZERLAND.

Is cortisol awakening response related to Acute Mountain Sickness in eumenorrheic and postmenopausal women?

Cortisol rises at altitude, but little is known about its relationship with Acute Mountain Sickness (AMS) in women, particularly in those who are menstruating or menopausal. Therefore, this study aimed to compare the cortisol awakening response and its correlation with AMS in these two groups of women.

Thirteen Eumenorrheic Women (EW) and fifteen Postmenopausal Women (PW) spent one night at altitude (3375 m) with AMS assessment. Cortisol levels were evaluated on the morning of ascent at Low Altitude (LA) and the morning after sleeping at High Altitude (HA). Saliva samples were collected at 0 min (C1), 30 min (C2), and 45 min (C3) after awakening. Three indices were calculated: First awakening sample (C1), the area under the cortisol rise curve relative to C1 (CAR1), and the area under the cortisol rise curve relative to 0 (CAR2).
The prevalence of AMS in EW and PW was 30.8% and 40.0%, respectively, 6 hours after arrival, and 15.4% and 26.7% one night after arrival. When AMS positive and AMS negative subgroups were compared, no difference in any of the cortisol parameters was found, neither for value after 6h or one night. A sub-analysis of EM and PW separately found no differences either. There was no significant difference between these two age groups.
The findings of the study suggest that there is no significant relationship between cortisol levels and AMS in EW and PW. These results are in line with Woods et al. (2012) but not with several other works that found increased cortisol levels in AMS-prone participants (Richalet et al., 1989; Estoppey et al., 2019; Gatterer et al., 2019). Different results can be due to exposure duration and altitude, and levels of physical exertion. Further research could evaluate if a sex-specific stress response explains the differences between our results and most previous investigations.

SELECTED POSTER 8

SPO.08. AXEL KF AGUETTAZ [1], FRANCESCA AMATI [1].

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A novel PLIN3 splicing variant reveals a conserved mitochondrial targeting of the perilipin protein family.

Perilipin3 (PLIN3) is a ubiquitous member of the Perilipins lipid droplet-coating protein family. By its association with lipid droplets, PLIN3 contributes to lipid droplet growth, lipophagy, phosphatidylcholine synthesis and to the cellular protection from lipid toxicity. Perilipin3 is highly expressed in skeletal muscle, where its levels have been correlated with fatty acid oxidation and exercise training. Interested in the study of proteins at the crossroad between lipid metabolism and muscle function, we decided to focus on Perilipin3.
From human skeletal muscle samples, we cloned a previously unreported PLIN3 splicing variant, consequently named Perilipin3B. Unexpectedly, expression of PLIN3A and PLIN3B in cells highlighted a specific mitochondrial targeting of the novel isoform. Moreover, PLIN3B expression led to a reorganization of the mitochondrial network, with swollen mitochondria clustered in the perinuclear area. At the ultrastructural level, electron micrographs revealed strong alterations of the mitochondrial suborganellar organization. In accordance with the morphological phenotype, mass spectrometry analysis of PLIN3B interactors identified several mitochondrial partners, with a particular enrichment of intermembrane space proteins. To assess the validity of zebrafish as a model, we decided to compare PLIN3 isoforms with zebrafish perilipins. Astonishingly, PLIN3B mitochondrial targeting appeared to be shared by zPlin2 and zPlin3, suggesting a conserved feature of the protein family and confirming the validity of our model in the investigation of perilipins recruitment to multiple organelles.
The discovery of Perilipin3B sheds light on a splicing-dependent regulation of PLIN3 targeting, as well as on a possible ancestral mitochondrial role of the perilipins family. Anyway, additional investigations are needed to assess the biological significance of such discoveries. In the future, we will investigate PLIN3B function at mitochondria and its role in muscle adaptation, as well as the determinant of its multiple organelle recruitment. To accomplish our goal, more robust cellular and in vivo models are being developed.

SELECTED POSTER 9

THIS WORK WAS ALSO SELECTED FOR A TALK

SPO.09. P. GUT1,2, S. SRIDI3, F. SACHER1,4, P. JAÏS1,4, H. COCHET1,3, M. STUBER1,2, A. BUSTIN1,2,3

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Wideband black-blood late gadolinium enhancement imaging for patients with cardiac implantable electronic devices

Purpose: Here we propose a technology to image myocardial infarction with wideband black-blood late gadolinium enhancement (LGE) imaging in patients with implantable cardioverter defibrillator (ICD).

Methods: A 2D breath-hold single-shot ECG-triggered gradient echo wideband black-blood sequence was implemented. Black-blood images were generated by a non-selective adiabatic hyperbolic secant (HS) 180° inversion recovery (IR) pulse (duration = 10.24ms) followed by an adiabatic T2 preparation module (T2p) [1]. The adiabatic T2p consists of a 90° tip-down pulse of two adiabatic HS refocusing pulses (duration = 12.8ms each), and of a 90° tip-up pulse [2]. To eliminate the hyperintensity artefacts from the non-properly inverted myocardium signal in the presence of CIED [3], the HS IR bandwidth was increased from 0.8 to 9.2kHz with a peak B1 amplitude of 30μT and the bandwidth of the two T2p refocusing pulses were increased from 1.6 to 5.0kHz with a peak B1 amplitude of 30μT.

Reference standard bright-blood PSIR LGE [4] and wideband black-blood LGE imaging were prospectively tested on a 1.5T system (MAGNETOM Aera, Siemens) in xx patients (x female; mean age xx) with MRI-conditional ICD and with following sequence parameters: FOV=300x225mm2, resolution=1.4x1.4mm2, slice thickness=8mm, flip angle=15°, TE/TR=2.7/4.8ms, acceleration GRAPPA of factor 2 with 36 reference k-space lines, phase partial Fourier=7/8, readout bandwidth=751Hz/pixel, 5 signal averages. One expert radiologist scored the subjective image quality (1 = non-diagnostic, 2 = less than adequate, 3 = adequate, 4 = excellent) and the overall ICD-artefact severity (ripple and hyperintensity artefacts) (1 = non-diagnostic with severe ICD artefacts, 2 = less than adequate with large ICD

artefacts, 3 = adequate with moderate ICD artefacts, 4 = excellent with minimal ICD artefacts). Furthermore, contrast ratios between scar and healthy myocardium signals and between scar and blood signals were calculated.

Results: Reference standard bright-blood PSIR images were highly affected by hyperintensity and ripple ICD-related artefacts (score: xx), making the diagnosis unreliable or impossible. Wideband black-blood provided images with suppressed hyperintensity and ripple ICD-related artefacts (score: xx), allowing a better visualization of the heart and myocardial infarction. Image quality was better on the proposed wideband black-blood (wideband black-blood: xx, PSIR: xx). Contrast ratios on wideband black blood was xx% higher than PSIR between scar and healthy myocardium signals and xx% higher than PSIR between scar and blood signals. Wideband black-blood technique provided enhanced scar contrast and could identify xx% more hyperenhanced segments than PSIR.

Discussion: Wideband black-blood LGE provided images with suppressed hyperintensity and ripple artefacts from the ICD and with higher scar contrast than standard PSIR, allowing more reliable and more accurate myocardial scar assessment.

References: [1] Sridi S, et al., Diagn Interv Imaging 2022 Dec; 103(12):607-617. [2] Nezafat R, et al., Magn Reson Med 2006 Apr; 55(4):858-64. [3] Rashid S, et al., Radiology 2014 Jan; 270(1):269-74. [4] Kellman P, et al., Magn Reson Med 2002 Feb; 47(2):372-83.

SPO.10. DENHO RAVI1,2, SANDRINE GREMLICH1,2, MANUELA WEIER1,2, ERATO NTINOPOULOU1, THIERRY ROGER2, ERIC GIANNONI1,2

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Host-pathogen interactions in group B streptococcal sepsis

Background: Group B Streptococcus (GBS) is a harmless colonizing bacterium in healthy adults, but a leading pathogen of neonatal sepsis. We hypothesized that interactions between GBS and the neonatal innate immune system leads to dysregulated host responses and reduced clearance of the bacteria, contributing to the pathogenesis of sepsis.
Our aim was to compare the innate immune response of newborn and adult monocyte-derived macrophages (MDMs) to GBS.
Methods: We differentiated monocytes from umbilical cord blood of healthy term newborns and from peripheral blood of healthy adult volunteers into MDMs with M-CSF and activated them with M-CSF or IFN-γ. We exposed MDMs to 25 live clinical GBS isolates belonging to different serotypes and sequence types (ST). Phagocytosis and intracellular survival of bacteria were measured by gentamicin protection assay, and cytokine production was quantified by ELISA.
Results: Newborn MCSF- and IFNγ-MDMs exhibited a lower capacity to phagocytose serotypes Ia (1.5- and 1.7-fold), Ib (1.5- and 1.8-fold) and III (1.4- and 1.6-fold), and STs 10 (1.7- and 2.2-fold), 12 (1.7- and 2.3-fold), 19 (1.5- and 1.7-fold), 23 (1.5- and 1.7-fold) and 498 (1.8- and 2.1-fold) compared to adults. Newborn IFNγ-MDMs also showed a reduced capacity to phagocytose ST-17 (1.6-fold). Newborn exhibited a higher IL-6 (in IFNγ-MDMs) release in response to serotype III (2.2-fold), ST-19 (3.8-fold) and ST-498 (5.6-fold), and IL-10 (in MCSF-MDMs) in response to serotypes III (2-fold), V (3.2-fold), ST-19 (3.1-fold) and ST-498 (13.5-fold) compared to adult MDMs.
Conclusion: Our results show that compared to adult, newborn MDMs exhibit a dysregulated phagocytosis and cytokine production in response to GBS, and that this response is dependent on the serotype and/or sequence type of the strain. These differences probably participate in the pathogenesis of GBS-related neonatal sepsis, which occurs more often with specific GBS serotypes and STs, namely serotype III and ST-17.

SPO.11. B. KRUMM1, C. LUNDBY2,3, J. HANSEN2, J. BEJDER3, T. EQUEY4, J.J. SAUGY1, F. BOTRÈ1, AND R. FAISS1

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Plasma volume variations in elite athletes

INTRODUCTION: The Athlete Biological Passport (ABP) provides individual and adaptative monitoring of multiple biomarkers in anti-doping; notably hematological variables altered by various confounders such as physical exercise or extreme environments (e.g., heat or altitude exposure). Consequently, to consider plasma volume (PV) shifts inherent in athletes' daily routines, this study aimed to test the validity of a novel multi-parametric approach using serum markers to estimate PV changes, and correct concentration-based ABP parameters (e.g. [Hb]). It was hypothesized that the model validated in athletes over short periods (weeks) would remain valid for integrating PV shifts in the ABP of elite athletes over 12 months. METHODS: Full blood counts were performed monthly during one year using flow cytometry (Sysmex XN-1000) in 20 elite cyclists. Subsequently, individual profiles were generated through the official ABP software (World Anti-Doping Agency, ADAMS). Additionally, eight serum biomarkers were measured as 'volume-sensitive' variables to run a multi-parametric model providing corrected ABP thresholds through the estimates of PV shifts. Within-subject coefficients of variations were calculated to assess the variability of the serum variables. RESULTS: Eight Atypical Passport Findings (ATPF) (i.e., values beyond individual threshold limits) were generated in the study cohort. Among these 8 ATPFs, only 2 could be removed after the correction of ABP thresholds. Meanwhile, further ATPFs were generated after correction (n=14). Furthermore, a higher variability of 'volume-sensitive' markers was observed in elites ($P < 0.001$). DISCUSSION & CONCLUSION: In contrast to findings obtained in previous studies, the application of a multi-parametric model including 'volume-sensitive' serum biomarkers to estimate PV variations provided a lesser correction ability of individual ABP thresholds, and has even highlighted additional ATPFs. Overall, this study outlines the need to investigate novel approaches to better assess PV variations when interpreting individual ABP profiles.

SPO.12. BERIL MERSINOGLU1, RAQUEL MARTINEZ1, SARA CRISTINELLI1, AND ANGELA CIUFFI1

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Characterization of novel candidate factors impacting HIV life cycle

Throughout its life cycle, HIV interacts with host factors that can either promote (HIV dependency factors, HDF) or inhibit (HIV Inhibitory factors, HIF) viral replication. The success of HIV replication relies on the balance between HDF and HIF. Epitranscriptomics, i.e., the field of RNA modifications such as N6-methyladenosine (m6A) methylations, provides a new layer of gene regulation and an additional opportunity to uncover novel cellular players involved in HIV replication.

We performed a differential analysis on m6A epitranscriptomic landscape of CD4+ T (SupT1) cells, infected or not with an HIV-based vector over time (12h, 24h, 36h post-infection), and identified 59 m6A hypermethylated transcripts [1]. We selected the top 10 consistently m6A hypermethylated transcripts at all time points, for further investigation of their impact on HIV life cycle using a CRISPR-Cas9-mediated knock-out (KO) approach in CD4+ T (Jurkat) cells.

The top 10 hypermethylated candidates revealed an enrichment for GTPases of GIMAP protein family (known to be involved in T cell survival and T cell development), with GIMAP1, GIMAP5 and GIMAP7. We successfully generated and validated KO Jurkat cell lines for each GIMAP candidate, as well as for known HDFs as positive controls. We are currently assessing their impact on HIV life cycle upon infection with a GFP reporter HIV-vector. Preliminary data so far suggests that GIMAP1 may inhibit HIV replication.

Finding novel actors modulating HIV replication through novel opportunities, such as epitranscriptomics, may improve current understanding of HIV biology and potentially provide an array of new therapeutic targets.

1Cristinelli, S.; Angelino, P.; Janowczyk, A.; Delorenzi, M.; Ciuffi, A. HIV Modifies the m6A and m5C Epitranscriptomic Landscape of the Host Cell. Frontiers in Virology 2021, 1, doi:10.3389/fviro.2021.714475.

SELECTED POSTER 13

THIS WORK WAS ALSO SELECTED FOR A TALK

SPO.13. SARAY RAMOS2, JOSÉ CARLOS SANTOS2, PETR BROZ2, MORRIS DEGEN1, KRISTYNA PLUHACKOVA3,* GONZALO CEBRERO1, GYTIS JANKEVICIUS1, ELLA HARTENIAN2, UNDINA GUILLERM5, BASTIAN KOHL1, DANIEL J. MÜLLER4, PAUL SCHANDA5, TIMM MAIER1, CAMILO PEREZ1, CHRISTIAN SIEBEN6,7,* AND SEBASTIAN HILLER1,*

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The structural basis for ninjurin-1 mediated plasma membrane rupture in inflammatory cell death

Eukaryotic cells can undergo different forms of programmed cell death, many of which culminate in plasma membrane rupture (PMR) as the defining terminal event. PMR was long thought to be driven by osmotic pressure, until it was recently shown to be an active process, mediated by the protein ninjurin-1 (NINJ1). Here, we resolve the structure of NINJ1 and the mechanism by which it ruptures membranes. Super-resolution microscopy reveals that NINJ1 assembles structurally diverse oligomers in dying cells, including rings, arcs, and filaments.

A cryo-electron microscopy structure of NINJ1 filaments shows a tightly packed fence-like array of transmembrane α-helices. Filament directionality and stability is defined by two amphipathic α-helices that interlink adjacent filament subunits. NINJ1 filaments feature a hydrophilic and a hydrophobic side, and consequently can cap membrane edges. The resulting stable supramolecular arrangements rupture the membrane, as shown by molecular dynamics simulations and validated by site-directed mutagenesis. The membrane protein NINJ1 is thus an interactive component of the eukaryotic cell membrane that functions as an in-built breaking point in response to activation of cell death.

SELECTED POSTER 14

THIS WORK WAS ALSO SELECTED FOR A TALK

SPO.14. MAI THU NGUYEN, THOMAS MARTIGNIER, JOHN R. PANNELL

DEPARTMENT OF ECOLOGY AND EVOLUTION, UNIVERSITY OF LAUSANNE, 1015 LAUSANNE

The genetic architecture, evolution and stability of populations where females, males and hermaphrodites coexist

The co-existence of females, males and hermaphrodites, also known as trioecy, is rare and not predicted by theory. However, puzzling cases of trioecious populations do exist in nature and are still poorly understood. Here, we presented data on sex ratios and geographical distribution of 36 trioecious Mercurialis annua populations. We performed crossing experiments and simulations to infer the genetic inheritance of maleness and femaleness. We showed that maleness is determined by a Y chromosome while femaleness arises from recessive loci, with possible cytotype variation. We built a mathematical model investigating the evolution and stability of trioecy. In contrast with current models, which investigate cytoplasmic male sterility mutation (CMS) in the context of invading a population comprised of only hermaphrodites, our model investigates the invasion of CMS into a population where males and hermaphrodites already coexist (androdioecy). We compared the invasion and fixation of CMS into an androdioecious population with that into a hermaphroditic population. We show that the existence of males hinders the invasion of CMS and allows a large buffer parameter space between CMS invasion and fixation, which allow trioecy to arise.

SELECTED POSTER 15

THIS WORK WAS ALSO SELECTED FOR A TALK

SPO.15. ALISON LIN1,2,3 , R. SCHELLING 2,3, D. VINYALS SALES1,2,3, P. NORTIER1,2,3, R. WINIGER2,3 AND L. PEREZ1,2,3

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2 LAUSANNE UNIVERSITY HOSPITAL (CHUV), MEDICINE DEPARTMENT, SERVICE OF IMMUNOLOGY AND ALLERGY
3 CENTER FOR HUMAN IMMUNOLOGY LAUSANNE (CHIL), LAUSANNE, SWITZERLAND

Enhancement of immune response by nanoparticle vaccination

Vaccination is a cost-effective strategy in public health to limit the spread of infectious diseases. Nonetheless, conventional vaccines are not always effective in children and the elderly population. Moreover, vaccine protection requires multiple boosts to maintain efficient immunity against pathogens. Herein, we aim to develop a novel vaccination platform to increase the immune response by using the cross-linking of multiple B cell receptors and immune cell targeting. We propose a self-assembling protein nanoparticle (NP) presenting the main antigen (F protein) of the respiratory syncytial virus (RSV) and different co-activators known to be involved in B cells signaling (CD40L, IL-21 or APRIL). For this purpose, we used protein engineering, mice immunization, serological and viral neutralization assays to evaluate the different vaccine candidate's potency. We demonstrate that immunization with NP codisplaying CD40L and F-RSV generates an increase of immunoglobulin specific to RSV, whose titer was about 25-fold higher than the titer obtained with NP-F-RSV alone. This increase is also corresponding to the antibodies' potency to neutralize RSV. In contrast, other co-activators had a limited impact on immune function. Our approach presents an improvement of our previously developed nanoparticle platform that will be useful for poor vaccine responders such as immunocompromised individuals.

SELECTED POSTER 16

THIS WORK WAS ALSO SELECTED FOR A TALK

SPO.16. SAMUEL MOIX1,3, C. AUWERX 1,2,3,4, M. SADLER1,3,4 AND Z. KUTALIK1,3,4

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3 SWISS INSTITUTE OF BIOINFORMATICS, LAUSANNE, SWITZERLAND
4 UNIVERSITY CENTER FOR PRIMARY CARE AND PUBLIC HEALTH, LAUSANNE, SWITZERLAND

Causes and consequences of telomere shortening: A Mendelian randomization study

Telomeres are repeated DNA sequences at the ends of chromosomes that shorten with each cell division, ultimately leading to cell senescence and contributing to the aging process. Factors modulating telomere attrition and their consequences on human health remain poorly understood. We used linear regression, bidirectional univariable (MR), and multivariable Mendelian randomization (MVMR) to assess the relationships between leukocyte telomere length (TL) and 147 complex traits - including diseases, biomarkers, and lifestyle factors - in 326,363 unrelated white UK Biobank participants. Our results recapitulate telomere shortening with age ($p<2.2e-16$) and show a stronger (pdiff.=5.5e-28) decline in males (betamales=-0.026) than in females (betafemales=-0.021). Although both sex hormone-binding globulin (pdiff.=2.2e-18) and lifestyle factors such as fruit intake (pdiff.=9.6e-4) or smoking (pdiff.=3.6e-3) showed different association with TL in males versus females, accounting for them did not fully explain the difference in rate of attrition between sexes. After correcting for age, age2, and sex, 95 traits significantly associated with TL. Negative associations were found with cardiovascular, pulmonary, and renal conditions, while positive associations were observed with lipoprotein levels, female reproductive traits, and cancers. Inverse-variance weighted (IVW) MR identified 27 traits causally affected by TL, such as kidney cancer (alphaivw=0.048; p=8.1e-10), forced vital capacity (alphaivw=0.073; p=3.2e-6), and systemic lupus erythematosus (alphaivw=0.167; p=5.8e-5), and 23 traits causally influencing TL, including drinking (alphaivw=-0.086; p=1.3e-4), smoking (alphaivw=-0.142; p=1.8e-4), educational attainment (alphaivw=0.075; p=2.2e-15), and BMI (alphaivw=-0.048; p=4.9e-10), the effect of the latter on TL being potentially mediated by age at last birth and educational attainment. These results provide new insights into the biology of telomeres by distinguishing between modulators, mediators, and consequences of telomere shortening, portraying intricated relationships between TL, diseases, lifestyle, and socio-economic factors.

SPO.17. NATALIA GAARSLEV1
AND SEBASTIAN SOYK1
1DEPARTMENT OF PLANT MOLECULAR BIOLOGY, UNIVERSITY OF LAUSANNE, LAUSANNE, SWITZERLAND

Harnessing genetic interactions driving inflorescence complexity in tomato

The phenotypic diversity of inflorescences is determined by the genetic networks underlying meristem maturation. In tomato, inflorescence architecture is shaped by genetic interactions between the MADS-box transcription factor genes JOINTLESS 2 (J2), ENHANCER OF JOINTLESS 2 (EJ2), and LONG INFLORESCENCE (LIN), homologs of Arabidopsis SEPALLATA 4. It has been demonstrated that MADS-box gene dosage negatively correlates with inflorescence complexity. However, how differences in MADS-box gene dosage lead to quantitative changes in inflorescence complexity remains poorly understood at the molecular level. To tackle this challenge, we sequenced transcriptomes from a collection of single and higher-order MADS-box mutants to investigate the transcriptional networks orchestrated by J2, EJ2, and LIN during meristem maturation. Coexpression analysis on significantly deregulated genes on the mutant collection allowed associating different degrees of inflorescence complexity, including those of an additive and synergistic nature, to specific expression clusters. Moreover, we found transcription factor families previously suggested to be involved in the regulation of reproductive development, including B3-domain and MADS-box genes. Targeting two previously uncharacterized MADS-box genes by genome editing suggests additional genetic interactions and a role in inflorescence development. Our approach sheds light on gene networks and the interaction among genes regulating inflorescence complexity. Understanding the developmental pathways guiding meristem maturation in determining inflorescence architecture may allow fine-tunning inflorescences, which can be exploited for crop improvement.

SELECTED POSTER 18

THIS WORK WAS ALSO SELECTED FOR A TALK

SPO.18. SILVIA PRIETO BAÑOS1,2, YANNIS NEVERS1,2, CHRISTOPHE DESSIMOZ1,2 AND NATASHA GLOVER1,2

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Annotation matters: the effect of gene annotation on orthology

Computational protein-coding gene annotation, i.e. finding the genes present in a genome, remains a challenging task. The quality of gene models and gene repertoire lags behind the pace at which available genome assemblies are increasing. Although annotation methods are improving, there are no community standards and in practice, most published gene annotations result from ad hoc or customised pipelines. As a result, only a few non-model species have complete and accurate gene models. Annotation quality affects downstream analyses, including comparative genomics. Yet there is no focus in understanding the impact of annotation methods choice on orthology inference, a pre-requisite for comparative genomic studies. Here, we show that different annotation methods render different orthology results. We ran OMA Standalone to infer the orthology between Chordata proteomes which we obtained with four commonly used annotation pipelines (Augustus 3.4 de-novo, the NCBI Eukaryotic Pipeline, the Ensembl gene annotation system and UniProt's Reference Proteomes). Results show important differences in the orthology inferred from each of the four proteomes sets, namely in the number of orthology pairs and on the accuracy of orthology prediction reported by a standard benchmark.

SPO.19. MARIA-PARASKEVI MOSCHOFIDOU1, GERASIMOS P. SYKIOTIS1

1SERVICE OF ENDOCRINOLOGY, DIABETOLOGY AND METABOLISM, LAUSANNE UNIVERSITY HOSPITAL AND UNIVERSITY OF LAUSANNE, LAUSANNE, SWITZERLAND.

Evaluation of a new audiovisual information material for patients with surgical thyroid disease.

Background: Patient education can be useful in improving patients' health literacy and can be considered an important component of therapeutic interventions. In this context, Medronic has created a new educational material focused on patients who are about to undergo surgery for thyroid disease. The primary aim of the study is to evaluate the new information material from Project Butterfly regarding the following six aspects: content, quality, readability, understandability, actionability and usefulness. The secondary objective is to examine the potential effects of the new information material on stress, anxiety and depression levels in its patient users.
Methods: 100 eligible participants are recruited from the Lausanne hospital (CHUV) evaluate the material in 3 different phases (t1,t2,t3).The results will be collected using the following questionnaires eHIQ, (e-Health Impact Questionnaire); GAD-7, (General Anxiety Disorder-7); PEMAT-A/V, (Patient Education Materials Assessment Tool for audiovisual materials); PHQ-9, (Patient Health Questionnaire-9); PSS, (Perceived Stress Scale); et USE, (Usefulness Scale for Patient Information Material). A stratification will be made between patients operated with a diagnosis or suspicion of thyroid cancer and patients operated for a benign indication. The resultant data will subjected to statistical analysis using repeated measures ANOVA and correlations between phases and aspects.
Results: To date, n=19 patients have completed all three phases. The total score of the responses to the USE questionnaires seems to have little variability between the two phases, the lower value is 47 and the largest is 85, the median of 68 overall (mean t2=66; t3=72) indicating average to good satisfaction with the use of education material and this both before surgery and after surgery. The following significant Pearson correlations were found between the different scores: a positive correlation between the eHIQ scores at t2 and at t3 (r=0.661; p=0.0038; 95% CI: 0.26-0.86); a positive correlation between eHIQ and USE scores at t2 (r=0.5417; p=0.020; 95% CI 0.10-0.80); and a positive correlation between eHIQ and USE scores at t3 (r=0.59; p=0.011; 95% CI: 0.16-0.84).Regarding the Discern score the

majority of patient-users, seem to be satisfied with the quality of the content of the material being evaluated.
Conclusions: Based on initial data seems the new information material can be a support to improve patient management, patient experience and satisfaction, and ultimately health-related quality of life (HRQOL) although it is too early to generalize results.

SPO.20. JOE BRADLEY DICKINSON1, MICHAEL TASCHNER1, STEPHAN GRUBER1

1DEPARTEMENT DE MICROBIOLOGIE FONDAMENTALE, UNIVERSITE DE LAUSANNE

Understanding the molecular mechanism of DNA translocation and loop extrusion by Smc5/6

Structural Maintenance of Chromosome (SMC) complexes are the molecular machines that ensure chromosomes organisation. The conserved Eukaryotic Smc5/6 complex is involved in a multitude of DNA organising tasks, such as stabilising stalled replication forks, chromosome segregation and viral genome repression to name a few. All SMC complexes are DNA motors capable of hydrolysing ATP. The working hypothesis for translocation along DNA, termed segment-capture, is that the complex extrudes a DNA loop through its arms, followed by loop release at the bottom of the complex, which with repetition results in directional translocation. To gain resolution on the DNA translocation mechanism of the complex, we set up an in vitro biochemical protocol that enables us to arrest the Smc5/6 complex into a previously undescribed conformational state we have termed the Zipped-state (Z-state). Resolving the spatial distribution of 8 subunits and understanding where DNA interacts with the Z-state complex is a priority. Using protein purification, we have been able to show that the Zstate complex stays topologically entrapped on DNA in vitro. Additionally, through cysteine crosslinking we are able to trap DNA in the lower part of the complex in accordance with the segment-capture model for DNA loop extrusion. Regarding the spatial distribution of the subunits in the Z-state, we are currently performing Cryogenic Electron-Microscopy (Cryo-EM).

SPO.21. YIFAT QUAN A, AURORE GUERAULTA, DAMIEN DE BELLIS A,B AND CHRISTIANE NAWRATH A

A UNIVERSITY OF LAUSANNE, DBMV, LAUSANNE, SWITZERLAND; B UNIVERSITY OF LAUSANNE, EMF, LAUSANNE, SWITZERLAND

SlABCG36 and SlABCG42 are largely redundant for tomato fruit cutin formation

Plant cuticle formation requires the export of cutin precursors by ATP-binding cassette family G (ABCG) transporters. SlABCG36 and SlABCG42 are two homologs of Arabidopsis AtABCG32 and encode closely related full-size ABCG transporters in tomato plants. Their down-regulation by a RNAi-approach had led to reduced cutin amounts and thinner fruit cuticles. Since both genes were simultaneously and partially silenced, the exact contribution of each gene and a full knockout of these transporters remained still to be elucidated. Here, SlABCG36 and SlABCG42 knockout single and double mutants were generated by the CRISPR technology in the Micro-Tom. Despite the high expression of SlABCG36 in wildtype (WT) tomato fruits, the single slabcg36 mutant exhibited only a 20-30% reduction in cutin amount and no reduction in cuticle thickness at 20 dpa. No changes in cutin amount and cuticle thickness could be observed at 50 dpa, nor changes in epidermal cell shape. The double mutant however had a strong reduction in cuticle thickness and a flatter epidermal cell shape compared to the WT and the single mutants at least in the early developmental stage highlighting that both SlABCG36 and SlABCG42 are largely redundant for cutin formation. The absence of severe phenotypes, such as organ fusions, suggests that other export mechanisms, likely by ABCG-half transporters, also contribute to tomato fruit cuticle formation. Further investigations will be performed to shed light on potential compensatory mechanisms in slabcg36/42 double mutants. Furthermore, the relation between cutin composition/amount and cutin structure as well as biophysical properties of the cuticle will be investigated.

SPO.22. S. GRAF 1, R.J. MURRAY 1, D.A. MOSER 1, A. FREI 1, L. DETOLEDO 1, S. WOOD 1, D.S. SCHECHTER 1,2,3, S. URBEN 1

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The moderating role of context-encoding and -memory in the intergenerational transmission of post-traumatic stress

Background. This pilot study aimed to understand the moderating role of the contextualization of emotional communication and its physiological correlates in the intergenerational transmission of post-traumatic stress (PTS) related to exposure to maltreatment and/or other interpersonal violence. Methods. Thirty-one mothers (M age = 33.87 years, SD = 4.14) and their toddlers (M age = 22.66 months, SD = 7.01) participated in the study. Mothers reported adverse life events (ALE) using the Life Events Checklist for DSM-5 and their current PTS symptoms using the Post-Traumatic Stress Disorder Checklist for DSM-5. They rated regulatory problems of their toddler with the Infant-Toddler Social and Emotional Assessment. Mothers performed a context-encoding and –memory (CEM) task including angry, happy, and neutral facial expressions, embedded into contexts that had not been mentioned in the task instructions, after which they were asked to recall both the faces and contexts that they had been shown. Maternal heart rate variability (HRV) was measured during a resting phase. Results. The regression model using current stress symptoms explained a marginal part of the variance ($F(3, 27) = 2.75$, $p = .062$, $R2 = .23$), but the ones using ALE did not. Recall of contexts previously associated with angry faces moderated the link between maternal current stress symptoms and child dysregulation (beta = -.007, $p = .049$). Baseline HRV was significantly and negatively correlated with the recall of contexts previously associated with angry faces (rho = -.53, $p = .006$), and marginally with the recall of angry faces (rho = -.37, $p = .059$). Discussion. This pilot study seems to identify psychophysiological markers (i.e., CEM, HRV) that may influence the intergenerational transmission of PTS. This may open new avenues in early identification and intervention with traumatized mothers and their very young children.

SPO.23. **MARGAUX CRÉZÉ 1,** JEANNE TAMARELLE 1, VANTHANOM SAVATHDY 2, SENGRLOUN PHONEKEO 2, JORDYN WALLENBORN 3, GUENTHER FINK 3, SOMPHOU SAYASONE 2, PETER ODERMATT 3, SENGCHANH KOUNNAVONG 2, PASCALE VONAESCH 1

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Dynamics and consequences of nutrition-related microbial dysbiosis in early life

Early life under- and overnutrition (jointly termed malnutrition) is increasingly recognized as an important risk factor for adult obesity and metabolic syndrome, a diet-related cluster of conditions that occur together and increase a person's risk of heart disease, stroke, and type 2 diabetes. Nevertheless, the exact causes linking malnutrition with metabolic syndrome remain poorly characterized. We hypothesize that the microbiota plays a crucial role in this trajectory and that the pathophysiological mechanisms underlying under- and overnutrition are, to some extent, shared. The aim of this project is to investigate the pathophysiological mechanisms linking early life malnutrition to metabolic disease in later life by understanding the contribution of the mother's and child's nutritional status and microbiomes to the child's predispositions to MetS. We establish a prospective birth cohort in Laos, where the double burden of malnutrition (combining episodes of under- and overnutrition at the same or different times in life) is of special concern as obesity rates are rapidly rising, aiming to recruit 100 undernourished, 100 normally nourished, and 100 over-nourished pregnant women. Recruitment started in March 2022. Women are recruited during their third trimester of pregnancy and then followed-up with their child until he/she reaches two years of age. Five visits along this timeline allow to collect anthropometric, clinical, metabolic, and nutritional data. To assess the microbiome composition, 16S amplicon gene

sequencing will be performed on faecal and saliva samples of mothers and child and on vaginal and breastmilk samples of mothers. Shotgun metagenomic will be carried out on a subset of oral, vaginal and faecal samples. The epigenetic and lipid profile of blood will be determined in the children at two years of age. This study will bring insights into the mechanisms linking malnutrition-induced early life dysbiosis to predisposition to metabolic disease in later life and is the first study to assess the effect of early life maternal over- and under-nutrition on the child's metabolic health. The results of this study will contribute to the emerging literature linking the early life microbiota, epigenetic changes and growth/metabolic health and should help find new ways to tackle the consequences of the double burden of malnutrition on life-long health.

SPO.24. SIMON YERSIN 1, JULIAN R. GARNEAU 1, PIERRE SCHNEEBERGER 2, KADRA OSMAN ALI 3, COLIN CERCAMONDI 4, ABDIFATAH MUHUMMED 3,5, REA TSCHOPP 5,6,7, JAKOB ZINSSTAG 5, PASCALE VONAESCH 1.

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2 HELMINTH DRUG DEVELOPMENT UNIT, SWISS TPH INSTITUTE, UNIVERSITY OF BASEL
3 JIGJIGA UNIVERSITY
4 DEPARTMENT OF HEALTH SCIENCES AND TECHNOLOGY, ETHZ
5 UNIVERSITY OF BASEL, HUMAN AND ANIMAL HEALTH UNIT, SWISS TPH INSTITUTE
6 SWISS TPH INSTITUTE
7 ARMAUER HANSEN RESEARCH INSTITUTE

Gut microbiomes of agropastoral children from the Somali Regional State of Ethiopia show a unique bacterial composition reflecting their dietary habits

The composition and function of the intestinal microbiota are major determinants of human's health and are influenced by factors such as diet and lifestyle. Previously, consistent differences between pre-industrial and industrial societies were showed with a notably higher abundance of Prevotella taxa in traditional populations and Bacteroides taxa in industrialized countries. In the present study, 59 fecal samples were obtained from children aged two to five years living a traditional agropastoral lifestyle in the Somali Region State of Ethiopia where camel milk and starch-rich food are predominant components of the local diet. Samples were subjected to both 16S rRNA amplicon and shotgun metagenomic sequencing. Microbiota composition and function were described in the feces and compared to publicly available datasets from additional children living in other traditional, transitional, and industrial communities with different subsistence strategies. We find that SRS samples are low in Bacteroidaceae, Prevotellaceae and Succinivibrionaceae but high

in Akkermansiaceae, Erysipelatoclostridiaceae Bifidobacteriaceae, Lactobacillaceae and Streptococcaceae. Additionally, the degradation of lactose, D-galactose and simple carbohydrates were enriched in SRS samples compared to samples from other communities. Unlike other non-industrialized communities, the SRS samples were low in some of the main bacterial representatives of the fecal samples of children living a traditional lifestyle. The high relative abundance of other specific taxa and the enrichment of carbohydrate degradation pathways in SRS samples show that dietary choices strongly influence the microbiota composition and function even amongst traditional communities. Our study revealed that the specific and low-diversity diet likely influences the unique microbiota composition and function of agropastoral children from the SRS of Ethiopia. Their microbiota significantly differs from the microbiota of children living a similar traditional lifestyle, highlighting the need to further characterized the fecal bacterial composition of these diverse communities to better understand microbiota dynamics and associated disease-signatures to ultimately develop effective treatments.

ABSENT

SELECTED POSTER 25

SPO.25. BEJOY MANOJ AND MATEUSZ MAJDA

DEPARTMENT OF PLANT MOLECULAR BIOLOGY, UNIVERSITY OF LAUSANNE, CH-1015 LAUSANNE, SWITZERLAND

Mechanics of anisotropic growth

Growth is a complex process that involves interactions and communication between neighboring cells at multiple scales, ranging from the genome level to the cell and organ scale. In animals, cells can move freely and expand due to cellular signaling, which shapes the formation of specific organs. However, plants' growth is not solely driven by molecular interactions, but also by mechanical constraints imposed by surrounding cell walls. The cell walls limit the movement of cells and determine the shape of the cells, resulting in various cellular geometries. Previous research has demonstrated that isodiametric cells rapidly elongate and create anisotropic gradients along etiolated hypocotyls. We aim to investigate how anisotropy is regulated at the molecular level and how these cellular gradients contribute to the mechanical properties of growing hypocotyls. Our research will focus on how cell wall elasticity changes over different developmental stages and analyze mutants defective in anisotropic gradients. To assess the stiffness of the hypocotyls, we will use extensometer, micro-indentation, and atomic force microscopy methods. We will use confocal microscopy coupled with image processing to measure growth rates and other cell geometrical parameters. We will then integrate the biological data into a computational simulation using the Finite Element Method. Altogether, this will help us understand how physical forces influence development and identify the role that increasing anisotropy plays in maintaining hypocotyl posture. Overall, our study aims to shed light on how anisotropic cells support growth and structure from a mechanical perspective and how this relates to the genetic control of cell elongation.

SPO.26. **PAUL-EMMANUEL VANDERRIELE1,3** GILLES AESCHLIMANN1, SOFIA VEROUTI2,3, QING WANG4, FRÉDÉRIQUE INO1, DENISE V. KRATSCHMAR3,5, ALEX ODERMATT3,5, EDITH HUMMLER1,3

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3. NATIONAL CENTER OF COMPETENCE IN RESEARCH KIDNEY.CH, 1011 LAUSANNE, SWITZERLAND.
4. DIVISION OF NEPHROLOGY AND HYPERTENSION, LAUSANNE UNIVERSITY HOSPITAL (CHUV), 1015 LAUSANNE, SWITZERLAND.
5. DIVISION OF MOLECULAR AND SYSTEMS TOXICOLOGY, DEPARTMENT OF PHARMACEUTICAL SCIENCES, UNIVERSITY OF BASEL, 4056 BASEL, SWITZERLAND.

The glucocorticoid receptor is likely involved in the development of the salt-sensitive hypertension through the deregulation of the soluble epoxide hydrolase (sEH) in rats.

Background: Salt is everywhere in food and an excess intake can lead to hypertension-development. In rats carrying a glucocorticoid receptor (GR) mutation leading to haploinsufficency (GR+/em2), a five weeks treatment of high salt diet (HSD) resulted in adrenal dysregulation of the soluble epoxide hydrolase (sEH) followed by an impairment in the degradation of the omega 3 and 6 fatty acids. GR-mutant rats developed salt-sensitive hypertension1.
Aim: To determine the role of the DNA-binding domain of the GR in the development of salt-sensitive hypertension in a transgenic rat model harboring an in frame mutation with a deletion of the exon 3 that resulted in a DNA-binding deficient receptor2.
Methods: Three salty diets are used in different groups: normal diet (0,3%), short term HSD (8% for 3 days), long term HSD (6% for 5 weeks).
ollowing these diets, blood pressure has been measured, adrenal proteins were extracted and used for Western-blot analyses, and the dilatation and/or contraction of aorta and kidney vessels were determined using pharmacological stimulants/blockers.

Results: On a normal salt diet, GR+/em4 rats presented a significant deregulation of the plasmatic corticosterone and 11-dehydrocorticosterone levels, an adrenal hyperplasia and a cardiac hypoplasia.
Following short term HSD, GR+/em4 rats exhibited a significant deregulation of the 11-dehydrocorticosterone and aldosterone. Furthermore, adrenal sEH was significantly decreased in comparison to wild-type (WT) rats.
Following a long-term HSD, GR+/em4 rats developed salt-sensitive hypertension associated with significant deregulation of adrenal and aortic sEH protein abundance.
Conclusions: Our data strongly suggest that abnormal adrenal and aortic sEH protein abundance is implicated in salt-sensitive hypertension in our rats.

SPO.27. S. C. MCHUGH1, P. VONAESCH1

1 DEPARTMENT OF FUNDAMENTAL MICROBIOLOGY, UNIL, LAUSANNE, SWITZERLAND

Causes and Consequences of Early Life Malnutrition in Later Life

Recent years have seen a rapid global surge in deaths linked to diet-related noncommunicable diseases such as type-2 diabetes, cardiovascular disease and metabolic syndrome (MetS). Currently, 38.9 million children are overweight and over 40% of all adults are overweight or obese. Recent evidence suggests that both early life under- and overnutrition contribute to an increased risk of metabolic disease in later life. Both forms of malnutrition share common pathophysiological hallmarks and their prevalence is of particular concern in low- and middle- income countries, where both forms of malnutrition can exist within individuals concomitantly or at different stages during their lifetime ("double burden of malnutrition"). Many aspects of health are driven by the gut microbiota and dysbiosis due to early life malnutrition may contribute to the physiological responses to nutrition-related disorders. Currently, the factors linking early-life malnutrition to MetS development in later-life remain unclear. We aim to elucidate the molecular mechanisms underlying the double burden of malnutrition through a neonatal mouse model of in utero growth restriction or macrosomia. We will investigate a causal role of the gut microbiota in the trajectory from early life over- and undernutrition to MetS. Further, we focus on whether nutrition-induced maternal dysbiosis directly leads to inheritable changes in gene expression though DNA methylation patterns of relevant genes linked to nutrient absorption, metabolism, and inflammation and/or if these changes are induced by the early-life microbiota of malnourished subjects. We observed that offspring born to undernourished dams show an increased weight gain upon exposure to a high-fat diet at weaning compared to overnourished or control mice. We further report that the inheritance of the maternal gut microbiota leads to distinct changes in the microbial signatures of malnourished offspring. It is essential to understand these underlying molecular mechanisms in a bid to design targeted preventable measures and treatments.

SELECTED POSTER 28

THIS WORK WAS ALSO SELECTED FOR A TALK

SPO.28. PHILIPP WALCH, PETR BROZ
DEPARTMENT OF IMMUNOBIOLOGY, UNIVERSITY OF LAUSANNE, SWITZERLAND

Dissecting the impact of enteric viral-bacterial co-infection on the host innate immune response and its implications for pathogenicity

Understanding how pathogens cause and maintain infection is essential to develop novel therapeutics and prevent outbreaks of emerging diseases. Infectious diseases have long been treated as a threat we had successfully overcome, yet the prevalence of multi-resistant bacterial strains, the SARS-CoV-2 pandemic, and an expansion of endemic regions for tropical diseases underline the urgency of further research. While the broadening of accessible methodologies has enabled mechanistic insights, leading to the identification of new targets to disrupt infection, ongoing research heavily focuses on single pathogen infections. By contrast, little is known about the molecular mechanisms underlying co-infections, including their detection by host pattern recognition receptors and their effect on host innate immune responses. From a clinical perspective however, co-infections are highly relevant, as they occur frequently and generally exacerbate symptom severity and fatality. Here, I am describing the first systematic mapping of enteric host-pathogen-pathogen interactions, deepening our molecular understanding of the effect pathogens have on each other, on how they are recognized by the host and on the initiation of innate immune defenses, such as the activation of inflammasomes and various forms of cell death. Interestingly, the directionality of interaction, i.e. synergies versus antagonisms, is dependent on the specific viral-bacterial pair used in co-infection, infection dynamic, i.e. simultaneous versus subsequent infection, as well as host cell type and Interferongamma priming. I have generated a vast, unbiased dataset of pathogen-pathogen interactions during infection, validated a subset, and, in a subsequent step, I explored the underlying mechanisms for pathogen interaction, revealing interaction points along the course of the infection. Using a broad panel of methodologies, including proteomics, FACS, microscopy and classical biochemical assays, I am currently characterizing these interaction points to yield a mechanistic explanation for the observed interactions.

SPO.29. BRENDA RIOS OCHOA

DÉPARTEMENT D'ECOLOGIE ET D'EVOLUTION, UNIVERSITY OF LAUSANNE, SWITZERLAND

Exploding sRNA crosskingdom comunication for food security

- Cassava (Manihot esculenta) is a food security crop that forms symbiosis with Arbuscular Mycorrhizal Fungi (AMF). Inoculation with AMF has the potential to boost cassava yield through improved resistance to abiotic and biotic stress.
- small RNAs (sRNAs) are molecular elements involved in the control of plant gene expression related to the response to stress. Under the hypothesis that the host plant exchanges sRNAs with its interacting microbes and knowing that the mechanism processing sRNA is conserved and functioning in AMF, sRNAs could then be exploited for host-induced gene silencing (HIGS) and help to better understand the AM symbiosis.

SPO.30. CASSANDRA TABASSO

Unraveling lipotoxicity, from subcellular mechanistic cues to clinical signatures

Metabolic diseases like obesity and type II diabetes are increasing in prevalence and have large impacts on mortality and morbidity. Obesity is defined as pathologic lipid accumulation, in adipose tissue or ectopically, which is believed to play a role in IR. However, intramuscular lipids have been observed in chronically trained adults, leading to the idea that the lipotoxicity effect depends on the specific lipid species that are accumulated, for example DAG and ceramides. These lipids have a deleterious impact on the insulin signaling pathway, which has been demonstrated in the liver. However, their role remains inconclusive in muscle, where high contents of DAG have been found in insulin sensitive patients.
DAG have several stereoisomers that are localized in different subcellular compartments, and establishing an integrative view of their distribution and amount is the first of our aims. The effect of intramuscular lipid deposition on insulin resistance relies on the dynamic partnership between lipid droplets and mitochondria that regulates lipid storage and oxidation. Therefore, we are developing a fractionation method allowing us to study the exact lipid composition of lipid droplets and mitochondria in sedentary subjects, lean or obese, before and after a chronic exercise period and compare them to chronically trained volunteers. Our hypothesis is that exercise will improve mitochondrial function and reduce the formation of toxic metabolites, such as DAG, resulting in a decreased IR. We also aim at evaluating at characterizing the epilipidome after acute exercise. Having new insight into the mechanisms behind lipotoxicity and IR may unveil therapeutic targets for metabolic dysregulations.

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ASSOCIATION OF PHD STUDENTS AND ASSISTANTS OF THE FACULTY OF BIOLOGY AND MEDICINE

ADAS is an association that represents PhDs and Postdocs and all members of the corps intermédiaire inférieur (maître-assistant-e-s, premier-ère-s assistant-e-s, assistants diplome-e-s) from the Faculty of Biology and Medicine (FBM) at the University of Lausanne.

Initially founded in 1998 as the "Association of PhD students and assistants of the Faculty of Sciences", its main mission is to establish contact with the dean's office and other university entities to address any concerns related to work and thesis conditions. ADAS serves as a first point of contact for members who may be facing professional difficulties.

Formed by a team of 20+ members (PhDs and Postdocs), the association also plays an important role in organizing social and scientific events aimed at promoting the integration of young researchers and building a network of scientists within the faculty and beyond; conferences, career roundtables, soft workshops, get-together-barbecues, PubQuizes, and many more. Through these events, ADAS creates opportunities for knowledge sharing and fosters a sense of community among members.

Furthermore, ADAS is committed to communicate the latest scientific knowledge to the general public. Overall, ADAS serves as a vital resource for PhD students and assistants at the FBM and strives to support their academic and professional development.

KNOW MORE ABOUT "ADAS" FIND US AT POSTER 33 AND 34

ACIDUL

LET'S PUT AN END TO PRECARITY AND INEQUALITIES AMONG PHD STUDENTS AT UNIL NOW!

ACIDUL and the Public Services Union (SSP/VPOD) denounce the inequalities in status, salary and working conditions between PhD students at the University of Lausanne. To correct these inequalities, we demand that:

1. PhD students on external funding automatically receive an allowance of 1500 CHF per month to compensate for the wage gap with graduate assistants.
In 2022, the salary scale gap between first-year SNSF doctoral students and first-year graduate assistants amounted to 18'000 francs per year and reached 22'000 francs in the fourth year. UNIL already compensates for about half of this gap thanks to a monthly allowance of 750 francs that is granted to almost all PhD students on external funding. To erase this gap, we ask that the current allowance be doubled and periodically increased in accordance with the salary scale (e.g., wage indexation).

2. Graduate assistants be employed on full-time contracts, and that 70% of their time be spent on their PhD thesis, to offer equal research conditions.
In 2021, the average employment rate of graduate assistants was 84% at UNIL. Besides, graduate assistants can spend up to 50% of their working hours on other tasks than their PhD. This means that they only have two years to write their PhD out of the five years of their contract. In comparison, SNSF doctoral students, who are on full-time contracts for four years, spend 85% of their time working on their PhD. Consequently, they have an additional 11 months to complete their work. To compensate for this disparity, we ask that graduate assistant positions only be advertised as full-time, that the rectorate offer an employment rate increase to those whose contracts have already started, and that the cahier des charges of all graduate assistants specify that a minimum of 70% of their working hours be spent on their doctoral thesis.

3. The salaries of PhD students on external funding be guaranteed by UNIL, as is the case for graduate assistants.
Currently, the contracts signed between UNIL and its employees on external funding are only binding for UNIL if the funding continues. Consequently, PhD students on external funding can lose their jobs if their funding disappears for reasons external to them (e.g., their supervisor leaves UNIL). This situation, which is fortunately rare, is not acceptable for a public institution. We ask that UNIL guarantee the salaries of these employees.

4. Graduate assistants be employed on five-year contracts and SNSF doctoral students on four-year contracts, instead of multiple successive contracts.
According to the Loi sur l'Université de Lausanne (LUL), graduate assistants sign three different contracts during their five years of employment at UNIL. SNSF doctoral students also sign several contracts, with the exact number depending on the project and faculty. This system exposes PhD students to arbitrariness and pressure. We therefore demand that contracts be signed for the full duration of the PhD. A three-month probation period is already provided by law when the first contract is signed and should suffice for a contract of four or five years.

FIND MORE ABOUT "ACIDUL" AT POSTER 35

"DDAY IS POSSIBLE THANKS TO THE GENEROSITY OF THE ÉCOLE DOCTORALE FIND THEM AT POSTER 36

WELCOME TO THE DOCTORAL SCHOOL (ED) OF THE FACULTY OF BIOLOGY AND MEDICINE!THE DOCTORAL SCHOOL STRONGLY SUPPORTS THE DDAY, A SPECIAL DAY FOR THE FACULTY'S DOCTORAL STUDENTS TO MEET AND PRESENT THEIR SCIENTIFIC WORK TO THEIR PEERS AND INTERNATIONAL EXPERTS. SHE EXPRESSES HER GRATITUDE TO THE ADAS ORGANISING COMMITTEE FOR THEIR OUTSTANDING WORK AND WARMLY CONGRATULATES THEM.THE DOCTORAL SCHOOL IS LOCATED IN THE SAME PLACE AS THE DDAY THIS YEAR, I.E. IN THE AMPHIPÔLE BUILDING, AND ALSO PARTICIPATES IN THE DAY WITH A POSTER TO PRESENT HUMAN FACES TO THIS UNIT ACTIVE IN MANY IMPORTANT ASPECTS OF DOCTORAL EDUCATION.

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If you are finishing your thesis soon, don't forget to register on the ALUMNIL platform. The Doctoral School will contact you every year to invite you to the FBM Drs Alumni Night!

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