Program and abstracts book

XV INTERNATIONAL CONGRESS OF CELL BIOLOGY
CANCUN, MEXICO

ICCB 2024
INTERNATIONAL CONGRESS OF CELL BIOLOGY
CANCUN, MEXICO

November 10-14, 2024
Cancun, Quintana Roo, Mexico
Convention Center of the Grand Oasis Hotel

Symposia

--DYNAMIC ORGANIZATION OF NUCLEAR FUNCTION
--PHASE SEPARATION IN CELL BIOLOGY
--TRANSCRIPTIONAL REGULATION IN EUKARYOTES
--PHYSICS AND CELL BIOLOGY OF MITOTIC CHROMOSOME FORMATION
--CSCB-IFCB. STEM CELLS
--CELL BIOLOGY EDUCATION
--CELL BIOLOGY OF PARASITIC PROTOZOA
--CELLULAR MICROBIOLOGY
--CELL ORGANELLE DYNAMICS AND INTERACTIONS
--NEW APPLICATIONS IN CELLULAR INMMUNOLOGY
--MEMBRANE DYNAMICS
--PLURIPOTENT STEM CELLS: GENETIC, MANIPULATION, DIRECTED DIFFERENTIATION AND ORGANOIDS
--MEMBRANE ANDATPases
--CSCB-IFCB. ORGANELLE INTERACTIONS AND REGENERATION I
--TISSUE ENGENEERING
--CSCB-IFCB. ORGANELLE INTERACTIONS AND REGENERATION II
--CELL AGING
--CELL BIOLOGY OF PLANTS
--INNOVATIONS IN BIOTECHNOLOGY: CELLS AND ORGANS ON CHIP
--ADVANCES IN CRYO-ELECTRON MICROSCOPY
--SPECIAL I: STUDENTS AND POSDOCS
--SPECIAL II: STUDENTS AND POSDOCS

Plenary lectures

Bill Earnshaw , Scotland UK
Marco Igor Valencia-Sánchez, Mexico
Wanderley de Souza, Brazil
Noni Franklin-Tong, England

Chairs and Speakers

Jesús Aguirre, Rossana Arroyo -Verástegui, Griselda Ávila -Soria, Ed Banigan, Leandro Augusto Barbosa, Marlene Benchimol , Gustavo Blanco, Steven Boeynaems , Jaap Brink, Iris Brochaos, Rosa Helena Bustos Cruz, Marco Antonio Carballo -Ontiveros, Luis Cárdenas, Enrique Castaño de la Serna, Susana Castro -Obregón, Melisa Karina Chacón Lázaro, Chih-Chiang Chan, Ye -Guang Chen, Quan Chen, Ruey-Hwa Chen, Ya-Hui Chi, Fernanda Cisneros -Soberanis, Rubén G. Contreras, Alejandra Covarrubias, Felipe Cruz García, Alexander de Luna, Paul de Vos, Joao Augusto Diniz Moura, Martha Espinosa Cantellano, Xinhua Feng, Cynthia Fernández -Láinez, Brett Finlay, Tatiana Fiordelisio , Daniel Foltz, Berenice García Ponce de León, Johan Gibcus , Alicia González, Pedro Gutiérrez Castrellón, Gyorgy Hajnoczky , Arihel Hernández Muñoz, Araida Hidalgo -Bastida, Meng-Chiao Ho, Sui Huang, Lijian Hui, Parsifal Islas, Luis F. Jiménez -García, Matt Joens, Javier Andrés Juárez-Díaz, Prasanth Kumar Kannanganattu , Paul Kaufman, Diana Laird, Laurent Limozin , Yu-Chun Lin, Ya -Wen Liu, Gabriel López -Velázquez, Miriam Guadalupe Mateo Cruz, Estefanía Morales Ruiz, Ana Mora, Kalpana Nanjareddy , Fernando Navarro-García, Rosa Navarro, Jaime Ortega -López, Rosario Ortíz Hernández, Annie Pardo, Iván Nicolás Pérez Osorio, Pierre Henry Puech, José Luis Puente, Andrea Putnam, Oscar Said Quiroz Zerecero, Mauricio Rojas, Yair Romero López, Roberto Ruiz Medrano, Lizbeth Sánchez Ayala, Enrique A. Sanhueza -Carrera, María de Lourdes Segura -Valdez, Liora Shoshani , Normanda Souza Melo, Dave Thirumalai , Alfredo G. Torres, Ming -Daw Tsai , Iván Velasco, Diana Velázquez, María Cristina Velasquillo , Won-Jing Wang, Xue-Biao Yao.

https://iccb2024.fciencias.unam.mx/

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The cell at high resolution and phase separation

Welcome to Cancun, Mexico for the XV International Congress on Cell Biology. Since its first edition in 1976, Mexico is hosting for the first time this important event that brings together the international community of cell biology from many countries around the world, associated in the International Federation of Cell Biology (IFCB). I thank the Federation for choosing Mexico as the venue for this congress. The topics covered at the congress, all related to the structure, composition and function of the cell, are associated with symposia organized by scientists who are experts in their field, and include topics such as transcriptional control, aging, membrane-bound and membrane less organelles formed by liquid-liquid phase separation phenomena, plant development, physics of cell division, tissue engineering, stem cells, among many others. Four plenary conferences are presented on topics such as chromosomes and cell division, the cytoskeleton of unicellular parasites, epigenetics and self-incompatibility in plants. The event is organized by two major institutions with a tradition in cell biology, the National Autonomous University of Mexico (UNAM) and the Center for Research and Advanced Studies (CINVESTAV), with the support of IFCB. In turn, this congress takes place within the framework of the 85th anniversary of the founding of the Faculty of Sciences of the UNAM in Mexico.

The congress brings together experts in different areas of cell study, with approaches that consolidate their knowledge with very high resolution and dynamics, incorporating analysis from other branches of science such as physics.

The city of Cancun in the Mexican Caribbean allows this great academic event to take place in a relaxed atmosphere, surrounded by an environment rich in an exceptional heritage of Maya culture that develop from about 2000 b.d. to 1524 a.d. but flourished between 250 to 900 a.d. Cancún also offers great international connectivity.

Luis F. Jiménez-García
Department of Cell Biology
Faculty of Sciences, UNAM, México
Chairman

Participants from different countries and continents in this congress

Australia
Austria
Brazil
Canada
Chile
China
Colombia
Cuba
England, UK
France
Germany
Korea
Kuwait
México
Netherlands
Scotland, UK
South Korea
Taiwan
United States

Previous congresses

1976 Boston, United States
1980 Berlin, Germany
1984 Tokyo, Japan
1988 Montreal, Canada
1992 Madrid, Spain
1996 San Francisco, United States
2000 Gold Coast, Australia
2004 Nice, France
2008 Seoul, Korea
2012 Rio de Janeiro, Brazil
2014 Philadelphia, United States
2016 Prague, Czech Republic
2018 Hiderabad, India.
2022 Taipei, Taiwan
2024 Cancun, Mexico

VENUE

The Convention Center of the Grand Oasis Cancun Hotel

USTED ESTA AQUÍ / YOU ARE HERE

EXCLUSIVE THE PYRAMID

1. BENAZUZA (Sólo adultos / Adults only)
(Restaurante Gourmet tecnoemocional con toques y matices mexicanos / Gourmet techno-emotional cuisine with Mexican hints & nuances)
2. BENAZUZA BAR (Sólo adultos / Adults only)
3. COFFEE & ME PYRAMID
4. MARKET PLACE FOOD HALL
- VILLA PAOLA WINE BAR
- SCHILLABAR
5. BITES (Tentempiés americanos y mexicanos /American & Mexican snacks)
6. THE PYRAMID BEACH CLUB BAR
7. CAREYES (Parrilla y mariscos / Grill & seafood)
11. FONTAINEBLEAU (Bar) (Sólo adultos / Adults only)
12. GRAND PLUS LOUNGE
25. HEALTH BAR (Sólo adultos / Adults only)

GRAND OASIS CANCÚN

8. MAKITACO (Restaurante fusión mexicano-japonesa / Mexican & Japanese fusion restaurant)
9. GRAND PLUS BEACH CLUB (Snacks)
10. GRAND PLUS BEACH CLUB BAR
13. HAVANA CIGAR CORNER (Sólo adultos / Adults only)
14. DOS LUNAS (Restaurante italiano / Italian restaurant)
15. IL FORNO DOS LUNAS (Pizza & pasta)
16. TERRAZA DOS LUNAS BAR
17. SPORT BAR BARLOVENTO
18. COFFEE & ME (Cafés, tés & smoothies / Coffee, tea & smoothies)
19. BAHÍA STEAKHOUSE
20. TUNKUL FOOD HALL (Buffet internacional / International buffet // Restaurantes / Restaurants)
- AKERU (Cocina asiática / Asian cuisine)
- BITES (Tentempiés americanos y mexicanos / American & Mexican snacks)
21. HACIENDA SARAPE MB (Tex-Mex restaurant)
22. BAR AGAVES
23. HELADOS // ICE CREAM
24. COCOA HIPPIE CHIC (Parrilla y mariscos en la playa / Grill and seafood on the beach)
26. THE WHITE BOX (Gastrobar) (Sólo adultos / Adults only)
27. THE WHITE BOX BAR
28. MAKITACO LT (Restaurante fusión mexicano-japonesa / Mexican & Japanese fusion restaurant)
29. IL FORNO DOS LUNAS TRATTORIA (Pizza & pasta)
30. DOS LUNAS TRATTORIA (Restaurante italiano / Italian restaurant)
31. TATISH (Buffet internacional / International buffet)
32. COYOTE LOCO (Music bar) (Sólo adultos / Adults only)
33. SPORT BAR SCIROCCO
34. COFFEE & ME (Cafés, tés & smoothies / Coffee, tea & smoothies)
35. LA PLACITA (Food court & bar)
- MR. WIMPY
- HAPPY CHICKEN
- TACOS & TORTAS
- HELADERÍA / ICE CREAM
- BAR
36. HACIENDA SARAPE (Tex-Mex restaurant)
37. HAVANA NIGHT CLUB (Sólo adultos / Adults only)
38. RED KINKY NIGHT CLUB (Sólo adultos / Adults only)
39. THE O OASIS BEACH CLUB
40. RED CASINO (Sólo adultos / Adults only)
41. CANCHA DE TENIS / TENNIS COURT
42. LA ROCA / THE ROCK

Cancún, Mexico

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	SUNDAY 10	MONDAY 11	TUESDAY 12	WEDNESDAY 13	THURSDAY 14	FRIDAY 15
9:00-10:00		KEYNOTE LECTURE Bill Earnshaw UE, SCOTLAND, UK Room Cancun	PLENARY I Wanderley de Souza URJ, BRAZIL Room Cancun	PLENARY II Marco Igor Valencia-Sánchez UNAM, MEXICO Room Cancun	PLENARY III Noni Franklin-Tong UB, ENGLAND, UK Room Cancun	
10:00-10:30		BREAK			BREAK	
10:30-12:30		SYMPOSIUM 1 Room Cancun Dynamic organization of nuclear function SYMPOSIUM 2 Room Merida Phase separation in Cell Biology	SYMPOSIUM 5 Room Cancun Cell Biology of Parasitic Protozoa SYMPOSIUM 6 Room Merida Cellular Microbiology SYMPOSIUM 7 Room Queretaro Cell organelle dynamics and interactions	SYMPOSIUM 11 Room Cancun Membrane and ATPases SYMPOSIUM 12 Room Queretaro Tissue engineering	SYMPOSIUM 14 Room Cancun Cell aging SYMPOSIUM 15 Room Merida Cell biology of plants	
12:30 - 15:00		LUNCH TIME				
15:00-16:30	REGISTRATION		POSTER SESSION I	POSTER SESSION II		
16:30-18:00		SYMPOSIUM 3 Room Cancun Physics and Cell Biology of mitotic chromosome formation SYMPOSIUM 4 Room Merida CSCB-IFCB Organelle interactions and regeneration	SYMPOSIUM 8 Room Cancun New applications in Cellular Immunology SYMPOSIUM 9 Room Merida Membrane Dynamics SYMPOSIUM 10 Room Queretaro Pluripotent stem cells: genetic, manipulation, directed differentiation and organoids	SYMPOSIUM 13 Room Merida Special Cell Biology Symposium: Students and posdoc	SYMPOSIUM 16 Room Cancun Innovations in Biotechnology: cells and organs on chip SYMPOSIUM 17 Room Merida Advances in cryoelectron microscopy SYMPOSIUM 18 Room Querétaro Cell biology education	
18:00 - 18:30	OPENING CEREMONY					
18:30 - 19:30					CLOSING CEREMONY	

SCIENTIFIC PROGRAM

SUNDAY

OPENING CEREMONY

Sunday, November 10, 18:00-19:00 h

ROOM CANCUN

MONDAY

Keynote Lecture

Monday, November 11, 9:00-10:00 h

ROOM CANCUN

William C. Earnshaw, FRS

Professor and Wellcome Principal Research Fellow, Institute of Cell Biology, University of Edinburgh, SCOTLAND, UK

"Approaching mitotic chromosome structure from all directions"

Monday, November 11, 10:30-12:30 h **ROOM CANCUN**

Symposium 1 DYNAMIC ORGANIZATION OF NUCLEAR FUNCTION

Chairpersons: **Sui Huang**, Northwestern University, Chicago, USA

Thoru Pederson, University of Massachusetts Medical School, USA

10:30-10:47 **Enrique Castaño de la Serna**, Unidad de Biología Integrativa, Centro de Investigación Científica de Yucatán, Calle 43, Número 130, Chuburná de Hidalgo, Mérida, Yucatán CP 97205, México.
"Fibrillarin: evolution, function and its implications in cancer"
10:53-11:10 **Paul Kaufman,** University of Massachusetts Chan Medical School
"Triggering nuclear body formation with extracellular signals"
11:11-11:28 Justin Bodner1,2, Pranathi Vadlamani1,2, Alexander S. Lee1,2, Shashank Srivastava1,2, **Daniel R. Foltz** 1, 2, 3, 1Department of Biochemistry and Molecular

Genetics, Northwestern University Feinberg School of Medicine, 2Simpson Querrey Institute for Epigenetics, 3Robert H. Lurie Comprehensive Cancer Center
"Selective regulation of linker histone H1 at the histone locus body"
11:29-11:46 **Prasanth Kumar Kannanganattu,** Department of Cell and Developmental Biology, University of Illinois at Urbana-Champaign
"Chromatin-associated lncRNA-splicing factor condensates regulate hypoxia responsive RNA processing of genes pre-positioned near nuclear speckles"
11:46-12:13 **Sui Huang**, Northwestern University, Chicago, USA
"PNC, nucleoli, and anti-cancer therapeutics"

Monday, November 11, 10:30-12:30 h **ROOM MERIDA**

Symposium 2 PHASE SEPARATION IN CELL BIOLOGY

Chairperson: **Rosa E. Navarro**, UNAM. Mexico

10:30-10:55 **Won-Jing Wang**, National Yang-Ming University, Taiwan
"Phase separation in the control of cilia initiation"
10:55-11:20 **Steven Boeynaems**, Baylor College of Medicine, USA
"Biomolecular condensates at the nexus of innate immunity and venoms"
11:20-11:45 **Andrea Putnam**, Jieon Lee University of Wisconsin-Madison, USA
"Maternal mRNA localization in embryos"
11:45-12:10 Coral Martínez-Martínez, Teresa B. Nava-Ramírez, Marisa S. Otegui, **Alejandra Covarrubias**, Biotechnology Institute, UNAM, México
"A plant stress protein: structural flexibility and cellular organization under water deficit"
12:10-12:30 **Rosa E. Navarro**, Valeria A. Ramírez-Ramírez, Arianne M. Cristino-Miranda y Andrea V. Cervantes-Ayala
Cell Physiology Institute, UNAM, México
"Stress-Induced Biomolecular Condensates in the C. elegans Germline"

Monday, November 11, 16:30-18:30 **ROOM CANCUN**

Symposium 3 PHYSICS AND CELL BIOLOGY OF MITOTIC CHROMOSOME FORMATION

Chairperson: **William Earnshaw**, Edinburgh, Scotland, UK

16:30-17:00 **Ed Banigan**, MIT Cambridge, USA
"The hidden structure of mitotic chromosomes"
17:00-17:30 **Dave Thirumalai,** University of Texas Austin, USA
"Role of Motors in Genome Folding"
17:30-18:00 **Johan Gibcus**, University of Massachusetts, USA.

"Rules of engagement for condensins and cohesins guide mitotic chromosome formation"
18:00-18:30 **Fernanda Cisneros-Soberanis**, University of Edinburgh, Scotland.
"Chromosome formation: insights from light and electron microscopy"

Monday, November 11, 16:30-18:30 h **ROOM MERIDA**
Symposium 4 CSCB-IFCB. ORGANELLE INTERACTIONS AND REGENERATION
Chairpersons: **Ye-Guang Chen**, Tsinghua University, China
Lijian Hui, Shanghai Institute of Biochemistry and Cell Biology, China

16:30-16:55 **Ye-Guang Chen**, Tsinghua University, China
"Intestinal Epithelial Homeostasis and Regulation"
16:55-17:20 **Lijian Hui**, Shanghai Institute of Biochemistry and Cell Biology, China
"Cell Identity Conversion and Liver Regeneration"
17:20-17:45 **Quan Chen**, College of Life Sciences, Nankai University, China
"Molecular regulation of mitochondrial homeostasis and its contribution to aging associated diseases"
17:45-18:10 **Xin-Hua Feng,** *Life Sciences Institute, Zhejiang University, Hangzhou, Zhejiang 310058, China*.
"Understanding TGF-β Actions in Cancer"
18:10-18:30 **Xue-Biao Yao**, MOE Key Laboratory for Cellular Dynamics, China
"Centromere Plasticity and Cell Division Control"

TUESDAY
Plenary I
Tuesday, November 12, 9:00-10:00 h
ROOM CANCUN
Wanderley de Souza

Federal University of Rio de Janeiro (UFRJ), Brazil
"The Cytoskeleton of Parasitic Protists"

Tuesday November 12, 10:30-12:30 h **ROOM CANCUN**

Symposium 5 CELL BIOLOGY OF PARASITIC PROTOZOA

Chairperson: **Martha Espinosa Cantellano**, CINVESTAV, México

10:30-11:00 **Martha Espinosa Cantellano,** Karla Acosta-Virgen, Adolfo Martínez-Palomo, Department of Infectomics and Molecular Pathogenesis, Center for Research and Advanced Studies, Mexico City, Mexico.
"A tale of two amebas"
11:00-11:30 Juliett Anders1, Michel-Ruben Glagowski1, Antonia Müller2, David Holthaus2, Rebecca Erler1, Constantin König1, Christian Klotz2, **Iris Bruchhaus**1*
1Bernhard Nocht Institute for Tropical Medicine, Hamburg, Germany; 2Robert Koch Institute, Berlin, Germany
"How does Entamoeba histolytica interact with the human intestine?"
11:30-12:00 **Rossana Arroyo-Verástegui,** Department of Infectomics and Molecular Pathogenesis, Center for Research and Advanced Studies (Cinvestav), Mexico City, Mexico.
"Peptidases in the virulence of Trichomonas vaginalis: A deep study of two legumain-like cysteine peptidases, TvLEGU-1 and TvLEGU-2"
12:00-12:30 **Jaime Ortega-López**, Departamento de Biotecnología y Bioingeniería, CINVESTAV, Mexico
"A Therapeutic Chagas Vaccine: more than a decade of a multidisciplinary effort"

Tuesday, November 12, 10:30-12:30 H **ROOM MERIDA**

Symposium 6 CELLULAR MICROBIOLOGY

Chairperson: **Fernando Navarro-García**, CINVESTAV, Mexico

10:30-11:00 **B. Brett Finlay**, OC, OBC, FRSC, FCAHS. CIFAR Senior Fellow and Co-Director Michael Smith Laboratories. University of British Columbia, Vancouver, B.C., Canada
"Pathogenic E. coli- a master cell biologist"
11:00-11:30 **Alfredo G. Torres**, University of Texas Medical Branch, USA
"Gastrointestinal Melioidosis and the role of the T6SS in Burkholderia pseudomallei pathogenesis"
11:30-12:00 **Fernando Navarro-García**, Department of Cell Biology. Centro de Investigaciones y Estudios Avanzados del IPN (CINVESTAV). Mexico City, México
"Enteroaggregative E. coli Pic is a key factor for interacting with intestinal goblet cells"
12:00.12:30 **José Luis Puente**. Instituto de Biotecnología. Universidad Nacional Autónoma de México (UNAM). Cuernavaca, Morelos, México
"The locus of enterocyte effacement of enteropathogenic E. coli: a unique toolbox for virulence gene regulation" **(ABSTRACT, PAGE 97).**

Tuesday, November 12, 10:30-12:30 h **ROOM QUERETARO**

Symposium 7 CELL ORGANELLE DYNAMICS AND INTERACTIONS

Chairperson: **Jesús Aguirre**, UNAM, México

10:30-11:00 **Gyorgy Hajnoczky,** Benjamin Cartes Saavedra, Victor Hugo Sanchez Vazquez, Arijita Ghosh, Raghavendra Singh, David Weaver. MitoCare Center for Mitochondrial Imaging Research and Diagnostics, Thomas Jefferson University, USA**.**
"Mitochondrial Calcium Homeostasis and Fusion Dynamics"
11:00-11:30 **Jesús Aguirre.** Instituto de Fisiología Celular, Universidad Nacional Autónoma de México, México.
"Regulation of Mitochondrial Division and Actin Depolymerization by Reactive Oxygen Species"
11:30-12:00 **Alicia González.** Instituto de Fisiología Celular, Universidad Nacional Autónoma de México, México.
"A Novel Chimeric Transcriptional Modulator Maintains Mitochondrial DNA Integrity in the Yeast Saccharomyces cerevisiae"
12:00-12:30 A. Becerril-Cuevas, Hen-Ming W., A. Cheung, J. Palacios-Martínez, A. I. Chávez-Martínez, P. Jiménez-Chávez, R. Morales-Sotelo, T. J. Parra-Aguilar, J. Olivares-Grajales, O. Santana-Estrada, S. Ryken, M. Bezanilla, **Luis Cárdenas.** Instituto de Biotecnología, Universidad Nacional Autónoma de México, México.
"Plant Polar Growth Requires a Specific and Dynamic Distribution of Organelles to Coordinate Apical Growth and Responses to Biotic Interactions"

Tuesday, November 12, 16:30-18:30 h **ROOM CANCUN**

Symposium 8 NEW APPLICATIONS IN CELLULAR IMMUNOLOGY

Chairperson: **Gabriel López-Velázquez**, National Institute of Pediatrics, México

16:30-17:00 **Paul de Vos (1),** Cynthia Fernández Lainez (2), Gabriel López Velázquez (2), (1) University Medical Center Groningen, Netherlands, (2) National Institute of Pediatrics, México
"Fructan dietary fibers and immunological effects"
17:00-17:30 **Pedro Gutiérrez Castrellón**, International Scientific Council for Probiotics, México
"New applications in cellular immunology: Gut and respiratory microbiota and role of probiotics on human immunology"
17:30-18:00 **Cynthia Fernández-Lainez (1),** Paul de Vos (2), Gabriel López-Velázquez (3), (1) Laboratorio de Errores Innatos del Metabolismo y Tamiz, Instituto Nacional de Pediatría, CDMX, México. (2) Immunoendocrinology, Division of Medical Biology, Department of Pathology and Medical Biology, University of Groningen and University Medical Center Groningen, Groningen, The Netherlands. (3) Laboratorio de Biomoléculas y Salud Infantil, Instituto Nacional de Pediatría, Secretaría de Salud, CDMX, México.

"Immune intestinal rescuing effects of two Agave fructans against the Giardia virulence factor arginine deiminase"
18:00-18:30 **Enrique A. Sanhueza-Carrera**, INP, México
"Cellular immunomodulation exerted by agave fructans and probiotic exopolysaccharides through the NF-kB/AP1 pathway"

Tuesday, November 12, 16:30-18:30 h **ROOM MERIDA**

Symposium 9 MEMBRANE DYNAMICS

Chairperson: **Ruey-Hwa Chen**, Academia Sinica, Taiwan

16:30-17:00 **Ruey-Hwa Chen**, Institute of Biological Chemistry, Academia Sinica, Taipei, Taiwan
"Bro1 proteins govern multivesicular body fate switch to regulate exosome secretion and cancer progression"
17:00-17:30 **Yu-Chun Lin**, Institute of Molecular Medicine, National Tsing Hua University, HsinChu, Taiwan
"Precise control of microtubule structure and intracellular trafficking in living cells and behaving animals"
17:30-18:00 Fei-Yang Tzou, Cheng-Li Hong, Kai-Hung Chen, John P. Vaughen, Wan-Syuan Lin, Chia-Heng Hsu, Yi Hsiao, Shu-Yi Huang, **Chih-Chiang Chan***
Graduate Institute of Physiology, National Taiwan University, Taiwan
"A genetic platform for functional profiling and visualization of the sphingolipid metabolic network"
18:00-18:30 **Ya-Wen Liu**, Institute of Molecular Medicine, National Taiwan University
"Roles of cardiolipin conversion in mitochondrial dynamics and quality control"

Tuesday November 12, 16:30-18:30 h **ROOM QUERETARO**

Symposium 10 PLURIPOTENT STEM CELLS: GENETIC, MANIPULATION, DIRECTED DIFFERENTIATION AND ORGANOIDS

Chairperson: **Iván Velasco**, UNAM, Mexico

16:30-17:30 Jonathan Bayerl1, Bryan Pavlovic2, Julia Bernard1,3, Varsha Desai5, Neha Saxena5, Melissa Holmes4, Julia Brunner6, Lydia Finley6, Alex Pollen2, Siddharth Dey5, and **Diana J. Laird**1,2, 1Department of ObGyn and Eli and Edythe Broad Center for Regeneration Medicine, University of California, San Francisco (UCSF); 2Weill Institute for Neurosciences and Eli and Edythe Broad Center for Regeneration Medicine, University of California, San Francisco (UCSF); 3Bakar Aging Research Institute (BARI), University of California, San Francisco (UCSF); 4Department of Psychology, University of Toronto, Mississauga, Toronto; 5Department of Chemical

Engineering, University of California, Santa Barbara; 6Cell Biology Program, Memorial Sloan Kettering Cancer Center, New York, USA.
"States of longevity in the eusocial mammal, the Naked mole-rat"
17:30-18:30 **Iván Velasco**, César Meléndez-Ramírez, Xóchitl Flores Ponce, Fernando Becerra-Vélez, Juan Jair Santillán-Cigales, Adolfo López-Ornelas, Enrique Estudillo, Itzel Escobedo-Avila, Instituto de Fisiología Celular Neurociencias, Universidad Nacional Autónoma de México. Laboratorio de Reprogramación Celular en el Instituto Nacional de Neurología y Neurocirugía, Mexico.
"Neuronal differentiation of pluripotent stem cells in monolayer and organoids"

WEDNESDAY

Plenary II

Wednesday, November 13, 9:00-10:00 h

ROOM CANCUN

Marco Igor Valencia-Sánchez

NYU Grossman School of Medicine, NY, USA
/ Instituto de Fisiología Celular UNAM, Mexico City, Mexico

"Structural basis of the read-write and erase mechanisms of histone H2AK119 monoubiquitination by Polycomb Repressive Complexes"

Wednesday, November 13, 10:30-12:30 **ROOM CANCUN**

Symposium 11 MEMBRANE AND ATPases

Chairperson: **Rubén G. Contreras**, CINVESTAV, Mexico

10:30-11:00 **Leandro Augusto Barbosa**1, Sayonarah Carvalho Rocha1, José Augusto FP Villar1, Ruth Rincón-Heredia2, Rubén G. Contreras2, 1- Universidade Federal de São João del-Rei, Brazil; 2- CINVESTAV, Mexico.
"Effect of 21-gamma benzylidene digoxin (21-BD) on cell adhesion and cell migration"
11:00-11:30 **Liora Shoshani**, Depto de Fisiología, Biofísica y Neurociencias, CINVESTAV, Zacatenco, Mexico
"New insights on cell-cell adhesion mediated by AMOG/b2 subunit of Na,K-ATPase"

11:30-12:00 Gladis Sánchez1, Shameem Sultana Syeda2, Narsihmulu Cheryala2, Jeff McDermott1, Henry Wong2, Gunda I. Georg2, and **Gustavo Blanco1**
1Department of Cell Biology and Physiology, University of Kansas Medical Center, Kansas City, Kansas, USA. 2Department of Medicinal Chemistry, College of Pharmacy, University of Minnesota, Minneapolis, Minnesota, USA.
"The testis specific Na,K-ATPase; a "pump" specifically dedicated to sperm function"
12:00-12:30 **Rubén G. Contreras**, Catalina Flores-Maldonado, Jessica Campos-Blázquez, José Bonilla, Karen Michelle Delgado Minjares, Marcelino Cereijido, Aida Castillo, Department of Physiology, Biophysics and Neurosciences, Center for Research and Advanced Studies (Cinvestav), México City, México
"Na+/K+-ATPase: a unique multifunctional protein, serving as a sodium pump, an adhesion molecule, and a signaling receptor, crucial in controlling cell adhesion"

Wednesday, November 13, 10:30-12:30 h **ROOM QUERETARO**

Symposium 12 TISSUE ENGINEERING

Chairperson: **María Cristina Velasquillo**, Instituto Nacional de Rehabilitación Luis Guillermo Ibarra Ibarra **(**INR), Mexico

10:30-11:00 **Rosario Ortíz Hernández**. Electron Microscopy, Facultad de Ciencias, UNAM, Mexico
"Tissular Biology and Tissue Engineering"
11:00-11:30 **Rosa Helena Bustos Cruz** Universidad De La Sabana – Chía, Cundinamarca. Research in nanotechnology applied in human health, Colombia
"Application of nanotechnology in biotechnology"
11:30-12:00 **Araida Hidalgo-Bastida**, Department of Life Sc, Manchester Metropolitan University, UK
"Biomaterials for regenerative Medicine"
12:00-12:30 **María Cristina Velasquillo** Martínez, Tissue Engineering, Cell Therapy and Regenerative Medicine Unit, Instituto Nacional de Rehabilitación "Luis Guillermo Ibarra Ibarra", Facultad de Ciencias, UNAM, Mexico
"Neocartilage by tissue engineering. Potential applications for auricular reconstruction"

Wednesday, November 13, 16:30-18:30 h **ROOM MERIDA**

Symposium 13 SPECIAL CELL BIOLOGY SYMPOSIUM: STUDENTS AND POSDOCS

Chairperson: **Marlene Benchimol**, CENABIO - Centro Nacional de Biologia Estrutural e Bioimagem, Universidade Federal do Rio de Janeiro Brazil and UNIGRANRIO-Universidade da Grande Rio, Rio de Janeiro, Brasil

16:30-16:45 **João Augusto Diniz Moura**[1], Carlos Gabriel Coutinho da Silva [1], Vinícius Mengal [2]; Sonia Alves Gouvea[1, 2] .[1]Postgraduate Program in Biotechnology – UFES, Vitória, Espírito Santo, Brazil; [2]Postgraduate Program in Physiological Sciences - UFES. Vitória, Espírito Santo, Brazil

"Analysis of oxidative stress in MCF-7 cells treated with L-arginine"

16:45-17:00 **Estefanía Morales Ruiz**, Islas-Flores Tania, Villanueva Marco A. Unidad Académica de Sistemas Arrecifales, ICML-UNAM, Puerto Morelos, Quintana Roo, México.

"The Role of SBiP1 in Protein Synthesis and Nitrogen Metabolism in Symbiodinium microadriaticum"

17:00-17:15 **Griselda Ávila-Soria** 1,2, 1 Department of Biochemistry and Molecular Biology, School of Pharmacy and Molecular Sciences, James Cook University, Townsville, Qld 4814, Australia. 2 Anáhuac University, Campus Cancún, 77565, México.

"Nematocyst Venom Components of Box Jellyfish Malo kingi: Molecular Characterization, Structural in silico Analysis and Their Potential Cellular Targets"

17:15-17:30 **Melisa Karina Chacón Lázaro**, Dora Angélica Silva Olivares1 y Abigail Betanzos Fernández1,1Department of Infectomics and Molecular Pathogenesis, Center for Research and Advanced Studies (CINVESTAV) of the National Polytechnic Institute. IPN 2508 Avenue, San Pedro Zacatenco, 07360 Mexico City, Mexico.

"Association between Helicobacter pylori infection and diabetes type 2 in a murine model"

17:30-17:45 **Normanda Souza-Melo**, Giovanna Henriques de Souza, Wanderley de Souza, Laboratório de Ultraestrutura Celular Hertha Meyer, Instituto de Biofísica Carlos Chagas. Rio de Janeiro. Brazil.

"The Role of the Flagellar Adhesion Zone in Trypanosoma cruzi Infectivity: New Insights from TcFLA-1BP and TcGP72 Knockouts"

17:45-18:00 **Miriam Guadalupe Mateo Cruz**, Rossana Arroyo, Department of Infectious Disease and Molecular Pathogenesis. The Center for Research and Advanced Studies of the National Polytechnic Institute. Av. IPN 2508, Mexico City, CDMX, CP 07360, Mexico.

"Cysteine protease CP A4: study of its participation as autophagin in the autophagic process of Trichomonas vaginalis"

18:00-18:15 **Lizbeth Sánchez Ayala,** Rossana Arroyo, Department of Infectomics and Molecular Pathogenesis, Center for Research and Advanced Studies (Cinvestav), Av. IPN 2508, 07360, CDMX, Mexico.

"Trichomonas vaginalis aspartic proteinase (Tv-AP): an analysis of the protein expression, localization and enzymatic activity in different trichomonad isolates and its regulation by iron"

18:15-18:30 **Oscar Said Quiroz-Zerecero**1,2,3, Ingrid Augusto2,3, Reyna Lara-Martínez1, Claudia Geraldine León-Ramírez4, Maria de Lourdes Segura-Valdez1, Jose

Ruiz-Herrera4, Kildare Miranda2,3 Luis Felipe Jiménez-García 1*, 1 Departamento de Biología Celular, Facultad de Ciencias, UNAM, Universidad Nacional Autónoma de México, Circuito de la Investigación Científica, C.U., 04510, Mexico City, Mexico; 2 Centro Nacional de Biologia Estrutural e Bioimagem, Universidade Federal do Rio de Janeiro, Rio de Janeiro, Brazil; 3 Laboratório de Ultraestrutura Celular Hertha Meyer, Centro de Pesquisa em Medicina de Precisão, Instituto de Biofísica, Carlos Chagas Filho, Universidade Federal do Rio de Janeiro, Rio de Janeiro, Brazil; 4 Departamento de Ingeniería Genética, Unidad Irapuato, Centro de Investigación Y de Estudios Avanzados del Instituto Politécnico Nacional, Km 9.6, Libramiento Norte, Carretera Irapuato-León, 36821, Irapuato, Guanajuato, México.
"Three-dimensional reconstruction and cytochemical analysis of the nucleolus in the Ustilago maydis fungus"

THURSDAY

Plenary III

Thursday, November 14, 9:00-10:00 h

ROOM CANCUN

Noni Franklin-Tong

University of Birmingham (UB), UK

"Recognition of "self": Uncovering new components that regulate Papaver self-incompatibility using a "poppidopsis" SI-PCD model system"

Thursday, November 14, 10:30-12:30 **h ROOM CANCUN**

Symposium 14 CELL AGING

Chairpersons: **Mauricio Rojas,** Ohio State University, USA

Annie Pardo, UNAM México

10:30 **Mauricio Rojas,** Division of Pulmonary, Critical Care and Sleep Medicine, The Ohio State University, Columbus, Ohio, USA
"Introduction"

10:40- 11:00 **Alexander de Luna,** Centro de Investigación sobre el Envejecimiento, Cinvestav, Mexico
"Towards a systems-level understanding of cellular aging in yeast"
11:00-11:20 **Ya-Hui Chi,** Institute of Biotechnology and Pharmaceutical Research, National Health Research Institutes, Taiwan
"Linking Nuclear Envelope Dysfunction to Cellular Senescence"
11:20-11:45 **Ana Mora,** Division of Pulmonary, Critical Care and Sleep Medicine, The Ohio State University, Columbus, Ohio, USA
"Mitochondrial Dysfunction at the Crossroad of Aging and Lung Fibrosis"
11:45-12:05 **Susana Castro-Obregón,** Departamento de Neurodesarrollo y Fisiología, Instituto de Fisiología Celular, UNAM. Circuito Escolar SN, Ciudad Universitaria, Coyoacán 04510. CDMX. México.
"Mechanisms of neuronal senescence"
12:05–12:30 **Annie Pardo,** Facultad de Ciencias, Universidad Nacional Autónoma de México, Mexico
"Fibroaging a dismissed player regulated by extracellular matrix"

Thursday, November 14, 10:30-12:30 h **ROOM MERIDA**

Symposium 15 CELL BIOLOGY OF PLANTS

Chairperson: **Javier Andrés Juárez-Díaz**, UNAM, México

10:30-10:47 **Berenice García Ponce de León**, Instituto de Ecología, UNAM, Mexico
"XAANTAL1 and 2 in flowering transition and meristems maintenance"
10:47-11:04 **Kalpana Nanjareddy**, ENES-León, UNAM, Mexico
"Unlocking symbiosis: How SnRK1 regulates P. vulgaris partnerships"
11:04-11:21 **Roberto Ruiz Medrano**, CINVESTAV-IPN Unidad Zacatenco, Ciudad de México
"Long distance transport of RNA in plants"
11:21-11:38 **Arihel Hernández Muñoz,** Instituto de Biotecnología, UNAM, Mexico
"Marchantia polymorpha GOLDEN2-LIKE transcriptional factor is a key protein for chloroplast differentiation and plant vegetative development"
11:38.11:55 **Javier Andrés Juárez Díaz***, Andre Zaragoza Gomez, Felipe Cruz García, James Gonzalez, Víctor Hugo Anaya Muñoz, Emilio García Caffarel, Yuridia Cruz Zamora, *Facultad de Ciencias, UNAM, Mexico.
"Beyond the classical pathway: Understanding unconventional protein secretion in plant cells"
11:55-12:12 **Felipe Cruz García**, Facultad de Química, UNAM, Mexico
"Cellular insights into the S-RNase-based self-incompatibility system"
12:12-12:30 **Matt Joens**, Thermo Fisher Scientific, USA.
"Electron Microscopy and the Frontiers of Plant Research"

Thursday, November 14, 16:30-18:30 h **ROOM CANCUN**

Symposium 16 INNOVATIONS IN BIOTECHNOLOGY: CELLS AND ORGANS ON CHIP

Chairperson: **Tatiana Fiordelisio**, UNAM, Mexico

16:30-16:55 **Pierre Henry Puech,** Laboratory of adhesion and inflammation, LUMINI, Marseille, France
"Some aspects in t lymphocytes mechanobiology"
16:55-17:20 **Laurent Limozin**, Laboratory of adhesion and inflammation, LUMINI, Marseille, France
"Studying dynamic cell interactions in vitro with Celldetective"
17:20-17:45 **Iván Nicolás Pérez Osorio**, Edda Lydia Sciutto Conde, Universidad Nacional Autónoma de México, Instituto de Investigaciones Biomédicas, Departamento de Inmunología, UNAM, México
"Blood-brain barrier-on-a-chip: A novel tool to study neuroinflammation"
17:45-18:10 **Yair Romero López,** UNAM, Mexico
"Lung regeneration via 3D cultures of progenitor cells"
18:10.18:30 **Diana Velázquez,** UNAM, Mexico
"Characterization of transmigration and effect of NK cell geometry"

Thursday, November 14, 16:30-18:30 h **ROOM MERIDA**

Symposium 17 ADVANCES IN CRYO-ELECTRON MICROSCOPY

Chairperson: **Wanderley de Souza,** Rio de Janeiro, Brazil

16:30-17:00 **Ming-Daw Tsai**, Institute of Biological Chemistry, Academia Sinica, Taipei, Taiwan
"Cryo-EM in enzymology and conformational dynamics"
17:00-17:30 Jitendra Maharana, Li-An Tsai, Chun-Hsiung Wang, Yi-Ting Liao, Lourriel S. Macale, Ronelito J. Perez, Melvin C. Shen, Sunil Tewary, Todd L. Lowary**, Meng-Chiao Ho** Institute of Biological Chemistry, Academia Sinica, Taipei, Taiwan
"Cryo-EM and Cryo-ET in visualizing host interaction and genome ejection mechanism of mycobateriophage"
17:30-18:00 **Jaap Brink**, TEM Product Manager for Life Sciences, Jeol
"High throughput, high resolution imaging using the JEOL CRYO ARM platform"
18.00-18:30 **Matt Joens**, Thermo Fisher Scientific, USA
"Cellular machinery revealed by cryo electron tomography and cryo spin mill tomography"

Thursday, November 14, 16:30-18:30 **ROOM QUERETARO**

Symposium 18 CELL BIOLOGY EDUCATION

Chairpersons: **María de Lourdes Segura-Valdez**, UNAM, Mexico
Luis F. Jiménez-García, UNAM, Mexico

16:30-17:00 **Marco Antonio Carballo-Ontiveros (1)**, Marcos Nahmad (2), América Nitxin Castañeda Sortibrán (1), (1) Department of Cell Biology, Facultad de Ciencias, Universidad Nacional Autónoma de México (UNAM), Mexico, (2) Department of Physiology, Biophysics and Neurosciences, Centro de Investigación y Estudios Avanzados del IPN (Cinvestav-IPN), Mexico

"Enhancing Critical Thinking in Undergraduate Biology Students: The Effect of Gradual Active Learning Strategies"

17:00-17:30 **Parsifal Islas (1)**, Ruy Echavarría (2), Martha Rodriguez (2), Martha Romero (2), Alberto Partida (2), Lourdes Segura-Valdez (2), Yudy Tibaduiza (2), Adolfo Martinez Palomo (1), Luis F. Jiménez-García (2), UNAM, CINVESTAV, Mexico

“Leeuwenhoek and the microscopy tradition in Mexico: novel approaches and discoveries in the history of microscopy in the Americas for innovation of cell biology education”

POSTER SESSION I

Tuesday, November 12, 15:00-16:30 h **ROOM CANCUN (POSTERS AREA)**

Poster no. 1

Extranucleolar ribonucleoproteic particles ultrastructure in members of the Pinophyta group

Agredano-Moreno L.T.1, Segura-Valdez M. de L.1, Muñiz-Diaz de León M. A.2, Jiménez-García L.F.1

1Department of Cell Biology and 2Department of Comparative Biology, Faculty of Sciences, UNAM, México

Poster no. 2

Nrf2 transcriptional activation after exposure to inorganic arsenic in hepatocellular carcinoma cells

Julio A. Alcocer-Zuñiga(1); Luz del Carmen Sánchez-Peña(2); Emilio J. Córdova(3) and Araceli Hernández-Zavala(1).

(1) National Institute Polytechnique, Mexico City, Mexico. (2) Center of Studies and Advance Research, Mexico City, Mexico. (3) National Institute of Genomic Medicine, Mexico City, Mexico.

Poster no. 3

Functional Characterization of Sulfate Transporter 3 in Phaseolus vulgaris During Symbiosis with *Rhizobium*

Manoj-Kumar Arthikala, Nataly Palomino, Miguel Lara, Kalpana Nanjareddy

Ciencias Agrogenómicas, Escuela Nacional de Estudios Superiores Unidad León, Universidad Nacional Autónoma de México (UNAM), León 37689, Mexico.

Poster no. 4

Different cell death pathways induced by Quercetin and Quercetagetin in cervical cancer cells

Bahena-Salmerón D., Sánchez-Sánchez L., López-Muñoz H., Alvarado-Sansininea J., Torres- Ramírez N., Echeverría O.M., Muñoz-Velasco I., Juárez-Chavero S., Escobar M.L.
Facultad de Ciencias, UNAM.

Poster no. 5

Study of the correlation between stress granules and mitochondria in *C. elegans*

Bernal-Palacios A.E., Campos-Martínez G., Salinas L.S. and Navarro, R.E.
Departamento de Biología Celular y Desarrollo.
Instituto de Fisiología Celular, Universidad Nacional Autónoma de México. Ciudad de México, México.

Poster no. 6

Genetic alterations and expression of factors that regulate mitocondrial genes in breast cancer

Susana Brito Molina, Karla G. Calderón González Juan Pedro Luna-Arias
Cell Biology Department, Center for Research and Advanced Studies of the National Polytechnic Institute (Cinvestav).

Poster no. 7

Analysis of Pulmonary Surfactant Protein C during Aging

Uriel Camacho-Silverio, Tania Valdivia-Herrera, Mariana Río de la Loza, David A. Lizcano, Remedios Ramírez, Fernanda Toscano-Márquez, Moisés Selman, Annie Pardo, Yair Romero.
Facultad de Ciencias, UNAM, Mexico

Poster no. 8

SIAH2 is potentially related to HIF-α increase in human and rat experimental renal cell carcinoma cell lines

Copado-Romero Jorge Luis, Solano-Becerra José Dolores, Ibarra-Rubio María Elena*
Lab F-225
Department of Biology, Faculty of Chemistry, UNAM. C.U. Mexico City. 04510, Phone +52(55)56223869. jlcopado24@gmail.com, meir@unam.mx*

Poster no. 9

Viridicatin isolated from the antarctic fungus *Penicillium* sp. shows neuroprotective effect on HMC-3 microglia cells and prevents HO-1 traslocation to the nucleus under H2O2 oxidative stress

Nicole Cortez, y Cristian Paz
Laboratory of Natural Products & Drug Discovery, Universidad de La Frontera, Center CEBIM, Temuco, Chile.

Poster no. 10

Three-dimensional analysis of interchromatin granules in the reptile *Sceloporous torquatus*

Cruz-Gómez S.J., Acosta-Cárdenas J., Mendoza-von der Borch A. P., González-Ruiz K.D., Labastida-Negrete R.E., Martínez-Flores K.G., Jiménez-García, L.F., Segura-Valdez M.L.
Cell Nanobiology Laboratory, Department of Cell Biology, Faculty of Sciences, UNAM, Mexico

Poster no. 11

Evaluation of fibrillar components of extracellular matrix in the vaginal wall in women with pelvic organ prolapse

Lizandra Maia de Sousa (a), Luana Amorim Hassun (b), Juliana do Carmo Fazzolari (b), Luiz Gustavo Oliveira Brito (b), Sílvio Roberto Consonni (a)
a Department of Biochemistry and Tissue Biology, Institute of Biology (IB), State University of Campinas (Unicamp), Campinas, Brazil b Department of Tocogynecology, School of Medical Sciences, State University of Campinas (Unicamp), Campinas, Brazil.

Poster no. 12

NOX-mediated Downregulation of VEGF function by Omega-3-fatty acid in aortic smooth muscle cell cultures treated with High Glucose and Palmitic acid

Gursev Dhaunsi and Maira Alsaeed
Department of Pediatrics, College of Medicine, Kuwait University, Jabriya, Kuwait.

Poster no. 13

Annexin A1 Deficiency Exacerbates Liver Pathology in a Type 1 Diabetes Mouse Model: Insights into Hepatocyte Protection Mechanisms

Diego Dias dos Santos1; Rafael André da Silva2; Antônio Thiago Pereira Campos3; Luiz Phillipe de Souza-Ferreira1; Carlos Lenz-César3; Cristiane Damas Gil1,2*.
1 Structural and Functional Biology Graduate Program, Universidade Federal de São Paulo (UNIFESP), São Paulo, SP 04023-900, Brazil. 2 Biosciences Graduate Program, Institute of Biosciences, Letters and Exact Sciences, Universidade Estadual Paulista (UNESP), São José do Rio Preto, SP 15054-000, Brazil. 3 National Institute of Photonic Applied to Cell Biology, Universidade Estadual de Campinas (UNICAMP), Campinas, SP 13083-865, Brazil. *Corresponding Author: Department of Morphology and

Genetics, Universidade Federal de São Paulo, Rua Botucatu 740, Edifício Lemos Torres - 3° andar, São Paulo, SP 04023-900, Brazil.

Poster no. 14

Morphological aspects and immunolocalization of hormone receptors in the uterine cervix of primiparous and multiparous senescent mice during pregnancy and postpartum

Rafaela Dias Neves a,b, Lizandra Maia de Sousa b, Sílvio Roberto Consonni b
a School of Medical Sciences (FCM), State University of Campinas (Unicamp), Campinas, Brazil; b Department of Biochemistry and Tissue Biology, Institute of Biology (IB), State University of Campinas (Unicamp), Campinas, Brazil.

Poster no. 15

Differentiation into osteoblasts of Wharton's Gelatin-derived Mesenchymal cells on gelatin-hyaluronic acid scaffolds loaded with microspheres containing BMP-2 and VEGF

Gamero Buendía S.1, Castell Rodríguez A.1, Piñón Zarate G.1, Ángeles Castellanos A.2, Herrera Enríquez M.A.1, Hernández Téllez B.1, Aguilar Sandoval M.1, Jarquín Yáñez K.1.
Laboratorio de Inmunoterapia e Ingeniería de Tejidos, Departamento de Biología Celular y Tisular1, Departamento de Anatomía2, Facultad de Medicina, Universidad Nacional Autónoma de México, México, CDMX, Mexico.

Poster no. 16

Ultrastructural Stereopairs analysis of the archaea *Haloferax volcanii*

Raziel Eduardo Garza-Melchor*, Paulina Mendoza von der Borch, Sarai de Jesús Cruz-Gómez, Segura Valdez M.L., Jiménez-García L.F.*
Cell Nanobiology Laboratory, Department of Cell Biology, Faculty of Sciences, UNAM, México
Posgrado en Ciencias Biológicas, UNAM, México

Poster no. 17

Ultrastructure of the nucleolus of *Taxodium mucronatum*

Karla Daniela González-Ruiz, Saraí de Jesús Cruz-Gómez, Ana Paulina Mendoza von der Borch, María de Lourdes Segura-Valdez, Luis Felipe Jiménez-García
Facultad de Ciencias, Universidad Nacional Autónoma de México.

Poster no. 18

Development of a murine organoid model for the study of tuft cells during the colonic regenerative process

Ma. Gabriela Guaita-Gavilanes1*, Karla Acosta-Virgen1, Jonatan Castillo-Millán1, Adolfo Martínez-Palomo1, Martha Espinosa-Cantellano1

1Department of Infectomics and Molecular Pathogenesis, Center for Research and Advanced Studies, Mexico City, Mexico

Poster no. 19

Mutational analysis of genes related to the antibiotic resistance in different *Helicobacter pylori* strains

Dulce Alheli Guzmán González1, Luis Esaú López Jácome2, Angélica Silva Olivares1, Abigail Betanzos1, Melisa Karina Chacón Lázaro1
1Department of Infectomics and Molecular Pathogenesis, Center for Research and Advanced Studies of the National Polytechnic Institute, Av Politécnico Nacional 2508, San Pedro Zacatenco, Gustavo A. Madero, C.P. 07360, CDMX 2Clinical Microbiology Laboratory, Division of Infectology, National Institute of Rehabilitation, Calz. México-Xochimilco 289, Coapa, delegación Tlalpan, C.P. 14389, CDMX, Mexico.

Poster no. 20

Analysis of TFIID Transcriptional Complex Components and Their Contribution to Heat Shock Response in Breast Cancer Cells

Edgar Hernández-Martínez; María Luisa Labra-Barrios; Rodolfo Moreno-Castillo; Iván de Jesús Salgado-Ramos; Juan Pedro Luna-Arias
Department of Cell Biology, Center for Research and Advanced Studies of the National Polytechnic Institute (Cinvestav-IPN), Av. I.P.N. 2508, Col. San Pedro Zacatenco, Alcaldía Gustavo A. Madero, C.P. 07360 Mexico City, Mexico.

Poster no. 21

Role of Gp32 protein in the induction of H66 prophage to the lytic cycle

1Emanuel Mejía-Jiménez 1Jair Martínez-Martínez & 1Gabriel Guarneros
1Department of Genetics and Molecular Biology. Address: Av. Instituto Politécnico Nacional 2508, San Pedro Zacatenco, Gustavo A. Madero, 07360 Mexico City, CDMX, Mexico.

Poster no. 22

Identification of Integrins associated with metastasis in breast cancer cell extracellular vesicles by flow cytometry

Olivo-Escalante Karen Donají (1), López-Pacheco Cynthia Paola (1,2) , Vergara-Bahena Irene (1), Herrera Juan Sebastian (1), Ortega-Soto Enrique (1) and Soldevila Gloria (1,2).
1. Department of Immunology, Biomedical Research Institute, UNAM, Mexico City, Mexico. 2. National Laboratory of Flow Cytometry, Biomedical Research Institute, UNAM, Mexico City, Mexico.

Poster no. 23

SYCP3 yields accumulations in primary spermatocytes during the first spermatogenic wave in murine models (*Rattus norvegicus* and *Mus musculus*)

Ortiz-Hernández Rosario 1, Pacheco-Gutierrez Sebastian 1, Guzmán-Vargas Luis Pablo 1, Torres-Ramírez Nayeli 1, Espinoza-Simón Emilio 2, Romero-López Yair 3, Sánchez-Mejía Sandra Nicole 1, Echeverría-Martínez Olga M 1
1.- Departamento de Biología Celular, UNAM, CDMX, México. 2.- Departamento de Bioquímica y Biología Estructural, Instituto de Fisiología Celular, UNAM, CDMX, México.

POSTER SESSION II

Wednesday, November 13, 15:00-16:30 h **ROOM CANCUN (POSTERS AREA))**

Poster no. 24

Cross-Neutralizing Anti-Dengue 2 IgG Antibodies from Patients and BALB/c Mice against Chikungunya Virus

Araceli Posadas-Mondragón1; José A. Santiago-Cruz1; Angélica Pérez-Juárez1; Norma E. Herrera-González1; Yessica S. Tapia-Guerrero2; Jessamyn R. Crespo-Sandoval1; Rocio Jiménez-Jiménez1; Nadia M. Vargas-Freyre1; J. Leopoldo Aguilar-Faisal1.
1.-Instituto Politécnico Nacional, Ciudad de México, México; 2.-Instituto Nacional de Rehabilitación, Ciudad de México, México.

Poster no. 25

Evaluation of Oxidative Stress and Therapeutic Resistance in MCF-7 cells Treated with L-arginine and Doxorubicin

Lorena Souza Rittberg Mauricio[1], Carlos Gabriel Coutinho da Silva[1], Vinícius Mengal[2]; Sonia Alves Gouvea[1], [2], 1Postgraduate Program in Biotechnology – UFES, Vitória, Postgraduate Program in Physiological Sciences - UFES. Vitória, Espírito Santo, Brazil

Poster no. 26

Identification of Entamoeba histolytica and *Entamoeba invadens* Peroxin 4 and ACSL4

Rivera Reséndiz Miguel Ángel, Acosta-Virgen Karla, Lizbeth Salazar-Villatoro, Martínez-Palomo Adolfo, Espinosa-Cantellano Martha
Department of Infectomics and Molecular Pathogenesis, Center for Research and Advanced Studies, Mexico.

Poster no. 27

Evidence that the EPO receptor is necessary for the cellular protective activity of EPOrh in Neuro-2a cells exposed to damage by oxygen and glucose deprivation

Rodriguez Jaramillo Karen, Beas Zárate Carlos, Rivera Cervantes Martha Catalina
Licenciatura en Biología, estudiante de Doctorado en Ciencias en Biología Molecular en Medicina, Universidad de Guadalajara, Mexico

Poster no. 28

Analysis of virulence factors in extracellular vesicles secreted by *Naegleria fowleri*

Rodríguez-Mera Itzel Berenice, Carrasco-Yépez María Maricela, Rojas-Hernández Saúl, Rosales Cruz Érika
Universidad Nacional Autónoma de México, FES Iztacala, Laboratorio de Microbiología Ambiental, México.

Poster no. 29

Autophagy Activation in a Mouse Model of Hypersensitivity Pneumonitis induced by *Saccharopolyspora rectivirgula* exposure

Andrea Montserrat Sánchez Barajas, Miguel Gaxiola, Annie Pardo Cemo, Moises Selman Lama, Sandra Cabrera Benítez.
Laboratorio de Biopatología Pulmonar Ciencias-INER, Facultad de Ciencias, UNAM, Circuito interior S/N, Ciudad Universitaria, Coyoacán, 04510 CDMX.

Poster no. 30

Glycine effects on orphan receptors and APP expression in Wistar rat neonatal glial cells culture

Moisés Sánchez Coria
Escuela Superior de Medicina, IPN, Mexico

Poster no. 31

Antibody neutralizing capacity of anti-Chikungunya IgG against Dengue Virus type 2 from patients and BALB/c mice

José Angel Santiago-Cruz1; J. Leopoldo Aguilar Faisal1; Angelica Pérez Juárez1; Norma E. Herrera González1; Yessica S. Tapia Guerrero2; Paola Juárez Trujillo1; Jessamyn R. Crespo Sandoval1; Rocio Jiménez Jiménez1; Araceli Posadas-Mondragón1
1.-Instituto Politécnico Nacional, Ciudad de México, México; 2.-Instituto Nacional de Rehabilitación, Ciudad de México, México

Poster no. 32

Microscopic evidence of contamination in hepatocytes of *Anolis porcatus* (SQUAMATA: POLYCHROTIDAE)

Sanz-Ochotorena A.C1, I. Estrada Acosta1, Y. Rodríguez-Gómez1, R. Lara Martínez2, V. Falcón-Cama3, S. Tehuacanero-Cuapa4, ML. Segura-Valdéz2 and LF. Jiménez-García2

1Faculty of Biology. University of Havana. 2Faculty of sciences. National Autonomous University of Mexico. 3Center for Genetic Engineering and Biotechnology Havana. 4Institute of Physics, National Autonomous University of Mexico

Poster no. 33

Scanning electron microscopy of the undulating membrane in spermatozoa of amphibians from Cuba

Sanz-Ochotorena A.C1, Y. Rodríguez-Gómez1, R. Lara Martínez2, ML. Segura-Valdéz2 and LF. Jiménez-García2;
1Faculty of Biology. University of Havana. 2Faculty of Sciences. National Autonomous University of Mexico.

Poster no. 34

Parthenogenetic eggs of *Bombyx mori* L. (LEPIDOPTERA, BOMBYCIDAE)

Sanz-Ochotorena A.C1, A. Ruiz Bárcenas2, J. Pita Diaz2 D. Pérez Almazan2, ML. Segura-Valdéz3 and L.F. Jiménez-García3
1Faculty of Biology. University of Havana. 2Center for Research in Protein Plants and Bionatural Products. Havana City 3Faculty of Sciences National Autonomous University of Mexico.

Poster no. 35

Correlation between *Helicobacter pylori* Infection with Metabolic Syndrome in a population of Mexico City

Mizel Alonso Saucedo Jaime, Ma. Guadalupe de Dios Bravo2, Melisa Karina Chacón Lázaro2 , Abigail Betanzos1
1Centro de Investigación y Estudios Avanzados (CINVESTAV), IPN, Mexico,2 UACM, San Lorenzo Tezonco

Poster no. 36

An up-close look at the larval development of red harvester ants

Natalia Terpan Arenas1, Ian A. E. Butler1, Alberto Carlos Martínez2, Ingrid A. Fetter Pruneda1
1 AntLab, Instituto de Investigaciones Biomédicas, UNAM. 2 Laboratorio de Microscopia electrónica. Instituto Nacional de Rehabilitación, Mexico.

Poster no. 37

Mitochondrial dysfunction in ovary of offspring of mice with polycystic ovary syndrome

Nayeli Torres-Ramírez, Paola del Valle, Carlos García, Emilio Espinoza-Simón, David Barrera, James González, Silvia Juárez, Luisa Escobar, Olga Echeverría
Facultad de Ciencias, UNAM, Mexico.

Poster no. 38

Morphological and metabolic characteristics in the pancreas and liver of male offspring of a polycystic ovary syndrome mouse model

Nayeli Torres-Ramírez1, Erick López-Cruz1, Rosa López-Castillero1, Emilio Espinoza-Simón2, David Barrera3, Rosario Ortiz1, Luisa Escobar1, Olga Echeverría1
1Departamento de Biología Celular, UNAM, México; 2Departamento de Bioquímica y Biología Estructural, IFC, UNAM, México; 3Departamento de Biología de la Reproducción, INCMNSZ, México.

Poster no. 39

Aging related epigenetic derepression of LINE-1 retrotransposon in Idiopathic Pulmonary Fibrosis

Tania Valdivia-Herrera, Mariana Río de la Loza, Marco Espina-Ordoñez, Uriel Camacho-Silverio, Remedios Ramírez, Annie Pardo, Moisés Selman Lama, Yair Romero
Laboratorio de Biopatología Pulmonar Ciencias-INER, Mexico

Poster no. 40

Characterization of the effect of white, red and blue LED light in the growth, photosynthesis and phycocyanin content in *Arthrospira maxima*

Perales Vela Hugo Virgilio; Salcedo Álvarez Martha Ofelia, Vega de Luna Félix, Gaviria González Llaraí Carolina, Esquivel Saldaña Haide
Laboratorio de Bioquímica, Unidad de Morfología y Función. Facultad de Estudios Superiores Iztacala. UNAM, Mexico.

Additional posters at the end of this abstract book , after the Author index (page 94).

ABSTRACTS

ABSTRACTS

Keynote and plenary lectures

Keynote conference

Approaching mitotic chromosome structure from all directions

William C. Earnshaw, FRS1*, Kumiko Samejima1, Johan Gibcus2, Sameer Abraham3, Fernanda Cisneros-Soberanis1, Itaru Samejima1, James R. Paulson3, Linfeng Xie3, Job Dekker2, Leonid Mirny4, Anton Goloborodko5.

1University of Edinburgh, Scotland, UK, 2University of Massachusetts, USA, 3University of Wisconsin Oshkosh, USA, 4Massachusetts Institute of Technology, USA, 5IMBA, Vienna, Austria.

*Professor and Wellcome Principal Research Fellow, Institute of Cell Biology, University of Edinburgh, SCOTLAND, UK.

Attempts to understand how the DNA is packaged in mitotic chromosomes are confounded by the huge size of the DNA, the incredible chromatin density in mitotic chromosomes and the complexity of the machinery that does the DNA packaging. We study this problem by combining chemical genetics, Hi-C genomic analysis, polymer modelling, light microscopy and electron microscopy. In our system, the entire cell population of chicken DT40 lymphocytes enters mitosis with near perfect synchrony within 2 to 3 minutes of release of a G2 phase arrest. This allows us to perform biochemical and structural analyses with minute-by-minute resolution as we "kinetically section" the process of mitotic chromosome formation. The cells can be engineered so that chromosome formation is directed by single SMC complexes: cohesin, condensin I or condensin II. Our latest models reveal that chromosomes are a disorderly helix of loops created by the SMC complexes. Condensin II drives the formation of cylindrical chromosomes but is restrained from achieving its ideal state by residual cohesive cohesin. The speed of loop formation *in vivo* is very similar to the speed of loop formation in *in vitro* systems but the process must be more complicated in vivo. Our electron microscopy analysis in human cells reveals that nucleosomes achieve a near millimolar concentration in mitotic chromosomes. In vitro, SMC motors are fast but weak, so how do they function in such a dense environment? The data from our electron microscopy and modelling are most consistent with chromosome formation involving a combination of looping by SMC complexes and chromatin phase separation. However, the chromatin concentration in chromosomes is much higher than the concentration of nucleosomes in phase-separated droplets in vitro. Thus, despite over 140 years of study, the essential mysteries of miotic chromosome formation remain elusive.

Plenary lectures

Plenary I

The cytoskeleton of Parasitic Protists

Wanderley de Souza

Rio de Janeiro Federal University, Institute of Biophysics Carlos Chagas Filho, Rio de Janeiro, Brazil.

Parasitic protists constitute a large number of eukaryotic microorganisms, which are responsible for a large number of infectious diseases widely distributed throughout the world. Among the diseases of high prevalence in humans, we mention malaria, toxoplasmosis, sleepless disease, leishmaniasis, Chagas disease, giardiasis, trichomoniasis, and amoebiasis, among others. Also includes diseases of animals with a large economic interest, such as those caused by *Eimeria, Babesia*, etc. They are unicellular organisms containing most of the characteristic organelles found in mammalian cells, such as the nucleus, endoplasmic reticulum, Golgi complex, endosomes, lysosomes, ribosomes, and

mitochondria. Some present highly specialized organelles such as kinetoplast, hydrogenosome, apicoplast, mitosome, glycosome, acidocalcisome, and special secretory organelles. One characteristic feature of eukaryotic cells is the presence of a cytoskeleton that maintains the intracellular architecture and dynamic changes that occur during the cell cycle and cell differentiation. It comprises three structures: microtubules, actin microfilaments, and intermediate filaments. Each of these is formed by specific proteins associated with many others and forms a complex network. Although well characterized in mammalian cells, there is little biochemical information on such structures in parasitic protists, which form complex structures. Examples, which will be described in some detail here, include (a) special arrays of microtubules forming the sub-pellicular microtubules found in trypanosomatids and Apicomplexa, the cytostome found in some kinetoplastida, the adhesive disc, median body and funis of Giardia, the axostyle of trichomonadids, (b) filaments found in the paraflagellar rod of trypanosomatids, and complex structures such as the costa and the parabasal filaments in trichomonads. These structures have been analyzed using microscope techniques such as expansion, atomic force, high-resolution scanning electron (and ion), transmission electron microscopy of thin sections, and freeze-fracture replicas. Some of these structures have been isolated and characterized by proteomic analysis. More recently, Cryo-electron microscopy has added new and relevant information. Acknowledgment. CAPES, CNPq, Faperj, and Finep have supported studies carried out in the author's laboratory.

Plenary II

Structural basis of the read-write and erase mechanisms of histone H2AK119 monoubiquitination by Polycomb Repressive Complexes

Marco Igor Valencia-Sánchez, Jonathan F. Thomas, Victoria Godínez-López, Pablo De Ioannes, Rachel Lee, Stephen Abini-Agbomson, Brian A. Sosa, Simone, Tamburri, Susan L. Gloor, Samantha Rustichelli, Pablo De Ioannes, Kristjan, Gretarsson, Jonathan M. Burg, Allison R. Hickman, Lu Sun, Saarang Gopinath, Hailey, Taylor, Matthew J. Meiners, Marcus A. Cheek, William Rice, Evgeny Nudler, Chao Lu, Michael-Christopher Keogh, Diego Pasini, Jean-Paul Armache, Karim-Jean Armache
NYU Grossman School of Medicine, NY, USA/ Instituto de Fisiología Celular UNAM

The interplay between different Polycomb group complexes is essential to maintain gene expression patterns during metazoan development. Disruption of this system produces severe developmental problems or can contribute to cancer initiation and progression. Mono-ubiquitination of histone H2A lysine 119 (H2AK119Ub) is catalyzed by the noncanonical Polycomb Repressive Complex 1 (ncPRC1) E3 ligase and reversed by the Polycomb Repressive Deubiquitinase (PR-DUB) complex. The epigenetic inheritance of silent chromatin domains is crucial for cellular memory during embryogenesis, though it must overcome the dilution of repressive histone modifications during DNA replication. One key component of this inheritance is the post-replication re-establishment of H2AK119Ub by ncPRC1 to restore the silent Polycomb domain. The exact mechanism behind this restoration has remained enigmatic. By combining cryo-EM with functional approaches, we uncovered how the asymmetrical binding of ncPRC1 containing RYBP to H2AK119Ub nucleosomes enables read-write mechanisms within the same and between nucleosomes. These findings highlight the significance of ncPRC1 in H2AK119Ub restoration. In contrast, balancing the activity of ncPRC1, the PR-DUB complex cleaves mono-ubiquitin from H2AK119Ub, restricting it at Polycomb target sites and protecting active genes from aberrant silencing. However, how PR-DUB achieves specificity for H2AK119Ub to regulate Polycomb silencing is unknown. Our structural, biochemical, and cellular data reveal the molecular interactions of PR-DUB critical for establishing specificity for H2AK119Ub within the nucleosome and provide a molecular explanation for how mutations in the components of PR-DUB, BAP1, and ASXL1 found in cancer can dysregulate H2AK119Ub deubiquitination. Through these studies, we provide functional and structural evidence for a unifying model of the cornerstone roles of Polycomb complexes in maintaining transcriptional repression and cellular identity.

Plenary III

Self-Incompatibility in Papaver: A novel receptor-ligand interaction that triggers a signaling network leading to programmed cell death

Vernonica (Noni) Franklin-Tong
University of Birmingham, UK.

Self-incompatibility (SI) plays a decisive role in determining flowering plant reproductive success by preventing inbreeding through recognition and inhibition of 'self' pollen. During pollination, “self” (incompatible) pollen is discriminated from compatible pollen, and rejected. The SI system in *Papaver rhoeas* (poppy) is an outstanding model system for cell-cell recognition/signalling, involving interaction of the S-determinants, PrsS and PrpS, triggering a Ca2+ dependent signaling network culminating in programmed cell death (PCD). PrsS is a secreted protein that acts as a signalling ligand; this interacts with its cognate pollen S-determinant PrpS, a small novel transmembrane protein. This interaction triggers increases in cytosolic Ca2+ and reactive oxygen species, cytosolic acidification, a decrease in ATP and changes in actin organisation, leading to activation of caspase-like proteases and PCD. Functional expression of the Papaver S-determinants PrpS and PrsS in the self-compatible model plant, A. thaliana demonstrated that PrsS and PrpS are sufficient for a functional synthetic S-locus in vivo. Plants co-expressing cognate PrpS and PrsS exhibit robust SI, with no self-seed set achieved. This was the first trans-genera transfer of non-orthologous S-determinants into a highly divergent species (>140 m.y. apart). Combining this “poppydopsis” system with genetically encoded fluorescent probes and live-cell imaging has allowed us to study SI-induced cellular alterations using genetic tools that were previously not possible. Studies have uncovered new information about dramatic cytosolic acidification, actin alterations, ATP depletion, involvement of mitochondria in production of ROS and changes in energy metabolism. Using a suppressor screen, we identified identification of “Highlander”: HLD1/AtPGAP1, an orthologue of the human GPI-inositol deacylase PGAP1, as a critical component required for the SI response. No major developmental defects were observed in hld1/atpgap1 knockout plants, but SI was completely abolished. Our findings demonstrate that GPI-remodelling activity and GPI-APs are required for SI, implicating involvement of further interactions at the plasma membrane.

ABSTRACTS
Oral and poster presentations
In alphabetical order of the last name of the first author

Extranucleolar ribonucleoproteic particles ultrastructure in members of the Pinophyta group

Agredano-Moreno L.T.[1], Segura-Valdez M. de L.[1], Muñiz-Diaz de León M. A.[2], Jiménez-García L.F.[1]
[1]Department of Cell Biology and [2]Department of Comparative Biology, Faculty of Sciences, UNAM, México.

Lacandonia granules (LGs) are intranuclear ribonucleoprotein particles (RNPs), involved in nuclear RNA metabolism. They have been found in the plants *Lacandonia schismatica* and *Triuris brevistylis* as well as in ancient plants as *Ginkgo biloba*, *Cycas revoluta*, *Ceretozamia mexicana* and *Welwitschia mirabilis*. Here, we used standard and cytochemical transmission electron microscopy techniques to investigate the nuclear ribonucleoprotein particles ultrastructure of other taxonomic groups as *Pinus att*enuata (Pinaceae), *Sequoiadendron giganteum* (Cupressaceae) and *Podocarpus reichei* (Podocarpaceae). Nuclear particles about 32 nm in diameter were found in the three different groups. These particles are positive to the EDTA regressive technique for ribonucleoproteins and are distributed among and surrounding compact chromatin strands. Our results show that Lacandonia granules are present in these species, suggesting that are involved in essential nuclear RNA metabolism of gymnosperms.

Regulation of Mitochondrial Division and Actin Depolymerization by Reactive Oxygen Species

Jesus Aguirre
Instituto de Fisiologia Celular, Universidad Nacional Autónoma de Mexico, Mexico.

Our group has advanced the understanding of reactive oxygen species (ROS) as critical regulators of different aspects of development and cellular physiology. Our work on the p38-type SakA stress MAPK pathway in ROS signaling within the fungus *Aspergillus nidulans* has revealed diverse roles for H2O2 in mitochondrial division regulation. We found that H2O2 induces extensive mitochondrial division, a process that relies on the dynamin-like protein DnmA and its receptor FisA (Drp1 and Fis1 in animal cells). While the absence of mitochondrial division has minor effects on respiration, it significantly impacts polar growth and development, and results in increased levels of mitochondrial ROS. H2O2 triggers a generalized mitochondrial constriction response, prior to actual division. This response involves a gradual depolarization of mitochondria, the participation of Ca2+, and requires interaction between mitochondria and the endoplasmic reticulum. Furthermore, H2O2 promotes DnmA aggregation, which is indicative of higher-order oligomerization and its recruitment to mitochondria. Our findings also indicate that H2O2 induced a rapid depolymerization and reorganization of actin, implying that actin dynamics are regulated by redox mechanisms. Notably, substitutions at DnmA C450S and C776S severely impair both mitochondrial and peroxisomal division. Interestingly, these substitutions do not significantly alter DnmA's general distribution but exhibit opposing effects on DnmA oligomerization in the absence of FisA. Molecular dynamics simulations have provided further insights into how the C450S and C776S substitutions, along with C450 oxidation, affect DnmA's signaling element-stalk domain angle, solvent-accessible surface area, and salt bridge interactions. The high likelihood of C450 oxidation—and its consequential structural changes—suggests that this modification by H2O2 could influence DnmA's multimeric structure. We propose a model wherein H2O2 regulates mitochondrial division by orchestrating both the generation of mitochondrial constrictions and the oligomerization of DnmA. In this framework, C450 oxidation serves as a critical priming event that facilitates the transition to productive self-assembly necessary for mitochondrial scission. Our work was supported by grants CONACYT-DFG 277869 and PAPIIT-UNAM IN200719, IV200519 and IN215622.

Nrf2 transcriptional activation after exposure to inorganic arsenic in hepatocellular carcinoma cells

Julio A. Alcocer-Zuñiga(1); Luz del Carmen Sánchez-Peña(2); Emilio J. Córdova(3) and Araceli Hernández-Zavala(1).
(1) National Institute Polytechnique, Mexico City, Mexico. (2) Center of Studies and Advance Research, Mexico City, Mexico. (3) National Institute of Genomic Medicine, Mexico City, Mexico.

Arsenic is a well-known carcinogenic contaminant for humans. Its biotransformation occurs primarily in the liver, where the AS3MT enzyme is predominantly expressed. This enzyme generates monomethylated and dimethylated organic metabolites, which exert toxicity through mechanisms distinct from inorganic arsenic. Nrf2 is a transcription factor that plays a crucial role in the cellular response to oxidative stress by regulating the expression of various antioxidants and detoxifying proteins. This provides protection against a wide range of xenobiotics. Objective To investigate the effect of arsenic metabolites exposure on the activation of Nrf2 in hepatocarcinoma (HepG2) cells. Materials and Methods A concentration-response curve (0, 0.1, 1, 2.5, and 5 µM inorganic arsenic) was first conducted to assess transcript levels of HMOX1, a key Nrf2 target gene, in hepatocarcinoma (HepG2) cells using RT-qPCR. Then, a time-course curve (0, 3, 6, 9, 12, and 24 h) was performed using the 5 µM arsenic concentration to evaluate the transcript levels of TXL, SQSTM1, FTL, GCLM and ABCC1 (additional Nrf2 target genes) via RT-qPCR. The concentration of arsenic metabolites was determined using hydride-generation and cryotrapping atomic absorption spectrometry (HG-CT-AAS) at the same exposure intervals. Results Statistical analysis showed upregulation of target gene expression in both the concentration-response and time-course assays. For certain genes, there was an association between their expression and the generation of

methylated arsenic metabolites. Conclusion These findings underscore the role of Nrf2 as a defense mechanism against arsenic and its methylated metabolites, showing the dual nature of this transcription factor in providing cytoprotection in cancerous cells.

How does *Entamoeba histolytica* interact with the human intestine?

Juliett Anders1, Michel-Ruben Glagowski1, Antonia Müller2, David Holthaus2, Rebecca Erler1, Constantin König1, Christian Klotz2, Iris Bruchhaus1*
1Bernhard Nocht Institute for Tropical Medicine, Hamburg, Germany
2Robert Koch Institute, Berlin, Germany.

An organoid-derived monolayer (ODM) model was used to analyse the processes driving the invasion of intestinal tissue by E. histolytica. Organoids of the colon and small intestine were grown from intestinal crypts containing adult stem cells and maintained in culture. Organoid-derived single cells were seeded onto a filter membrane in a transwell system and grown in differentiation medium. The ODMs were co-incubated with two amoeba clones differing in virulence (non-pathogenic A1np clone and pathogenic B2p clone). Co-incubation experiments with ODMs derived from the small intestine showed that i) A1np and B2p trophozoites (500) are unable to destroy the epithelium of the small intestine, ii) several hundred genes are up- and downregulated after co-incubation with A1np and B2p trophozoites, iii) A1np and B2p trophozoites mainly trigger upregulation of genes involved in the immune response. Co-incubation experiments with ODMs derived from the colon showed that i) colon cell response to co-incubation with 500 B2p trophozoites starts later but is overall stronger than the response to 500 A1np trophozoites, ii) B2p trophozoites trigger upregulation of genes encoding proinflammatory/antimicrobial chemokines, FOS/NF□B pathway, T cell regulation. For E. histolytica, a faster upregulation of genes encoding antioxidant proteins and DNA repair proteins was observed in B2p trophozoites compared to A1np trophozoites after co-incubation with colon cells.
In addition, the function of extracellular vesicles (EVs) secreted by E. histolytica was analysed. No difference was observed between A1np- and B2p-EVs in size and number of secreted EVs. Furthermore, E. histolytica surface molecules were detected on the EVs, proteins involved in EV biogenesis and signalling were enriched and EVs contained virulence factors and antioxidants. It was shown that A1np-EVs and B2p-EVs can stimulate monocytes. While heat-inactivated A1np- EVs can stimulate monocytes, heat-inactivated B2p-EVs cannot.

Peptidases in the virulence of *Trichomonas vaginalis*: A deep study of two legumain-like cysteine peptidases, TvLEGU-1 and TvLEGU-2

Rossana Arroyo-Verástegui
Department of Infectomics and Molecular Pathogenesis, Center for Research and Advanced Studies (Cinvestav), Mexico City, Mexico.

Trichomonas vaginalis is the causative agent of trichomoniasis, the most prevalent non- viral neglected sexually transmitted infection worldwide. Trichomonad degradome is extensive, and cysteine peptidases (CPs) are the most abundant in T. vaginalis. Some peptidases are immunogenic virulence factors secreted during infection that play key roles in cytoadherence, cytotoxicity, hemolysis, apoptosis induction, or as hemoglobinases. Some peptidases are differentially regulated by microenvironmental factors such as iron, glucose, polyamines, and zinc. The CPs of the clan CD in T. vaginalis include 10 genes that encode legumain-like peptidases that belong to the C13 family. TvLEGU-1 and TvLEGU-2 are CPs detected in the immunoproteome using trichomoniasis patient sera. This work will show evidence that these legumains are differentially regulated by glucose and iron at the expression, localization, and in vitro secretion, and participate in different virulence properties, even though they are localized in the same type of organelles, are immunogenic, and are found in secretions of Tv(+) patients, suggesting its relevance during trichomonas infection.

Functional Characterization of Sulfate Transporter 3 in Phaseolus vulgaris During Symbiosis with *Rhizobium*

Manoj-Kumar Arthikala, Nataly Palomino, Miguel Lara, Kalpana Nanjareddy
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Sulfur is an essential element for all living organisms. Plants convert inorganic sulfur into organic sulfur compounds through complex enzymatic processes. Various types of sulfate transporters (SULTRs) have been identified in plants, each expressed and regulated in specific organs, cell types, and subcellular compartments. These transporters play key roles in several physiological functions. However, the role of SULTRs in legume-rhizobium symbiosis remains poorly understood. In Phaseolus vulgaris (common bean), the SULTR gene family consists of 15 members. Our previous RNA-Seq data revealed that, among these 15 members, SULTR3 is specifically induced during rhizobium colonization in beans. This study focuses on the molecular characterization of the SULTR-3 gene in symbiosis between Phaseolus and Rhizobium using an RNA interference (RNAi) approach. Transgenic P. vulgaris hairy roots expressing the SULTR-3/RNAi construct showed an 85% reduction in SULTR-3 transcripts compared to controls (empty vector). Upon inoculation with *Rhizobium tropici* CIAT899, we observed a significant reduction in rhizobial infection threads, as well as in the number of nodule primordia and mature nodules, compared to controls. Mature nodules in RNAi roots were small, white, and lacked leghemoglobin. Histochemical analysis revealed an absence of infected cells in the SULTR-3/RNAi roots. Additionally, RT-qPCR analysis showed that the expression of symbiotic marker genes was significantly decreased in SULTR-3/RNAi roots and nodules compared to controls. These results indicate that SULTR-3 is essential for nodule development in P. vulgaris. We acknowledge PAPIIT-UNAM for funding this research through grant no. IN208424 to A. M-.K; and IN217724 to K.N.

Nematocyst Venom Components of Box Jellyfish *Malo kingi*: Molecular Characterization, Structural *in silico* Analysis and Their Potential Cellular Targets

Griselda Ávila-Soria 1,2
1 Department of Biochemistry and Molecular Biology, School of Pharmacy and Molecular Sciences, James Cook University, Townsville, Qld 4814, Australia. 2 Anáhuac University, Campus Cancún, 77565, México.

Irukandji jellyfish, *Malo kingi*, is a rare yet lethal marine stinger with a potent venom, causing the debilitating Irukandji syndrome. Venom components are stored in nematocysts, stinging organelles produced from the Golgi apparatus as a secretory product within a specialized cell, the nematocyte. This study aimed to elucidate the molecular mechanisms underlying toxicity by identifying and characterizing novel toxins from *M. kingi*. A cDNA library was constructed from *M. kingi* and screened for toxin-related sequences. Two novel cytolytic toxins designated MkTX-A and MkTX-B, were identified. Sequence analysis revealed their membership in a novel family of pore-forming toxins within the phylum Cnidaria, the most ancestral animal group on planet Earth. Structural modelling predicted a unique action mechanism involving an N-terminal amphipathic α-helix for membrane binding and a C-terminal β-barrel domain for pore formation. Unlike other cationic pore-forming peptides in cnidarians, MkTX-A and MkTX-B, along with other cubozoan cytolytic toxins, are disulphide-bonded polypeptides with structural similarity to the ricin B-like lectin superfamily, suggesting potential lectin-like properties. In addition to MkTX-A and MkTX-B, two other novel proteins were identified: MkPR, a pathogen-like protein with homology to ShK/SXC/Tox1 toxins, and MkDTx, a dermatopondin cytotoxin-like protein a serine protease comprising the ShK/SXC/Tox1 and unknow proteins displaying in tandem ShK/SXC/Tox1 toxin like motif. These findings suggest a complex venom cocktail in M. kingi, with a diverse cellular mechanism of action likely involved in defence and predation. Identifying and characterising these toxins may provide critical insights into the molecular and cellular basis of Irukandji syndrome. Understanding the mechanisms of action of these toxins can potentially lead to the development of novel therapeutic strategies for treating

Irukandji envenomation. Palabras clave: Cnidaria, Cubozoa, Irukandji, nematocyst, lysins. Irukandji syndrome, box jellyfish; CSL antivenom, nematocyst extracts; antigenicity; human sera, human antibodies.

Different cell death pathways induced by Quercetin and Quercetagetin in cervical cancer cells

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Cancer diseases have a high impact around the world and in Mexico. Cervical cancer impacts importantly to the Mexican population. Currently, several strategies are used to treat this pathology, including chemotherapy. However, it is not always effective, and it is generally associated with significant side reactions. All of this results in constant research for new alternatives in which compounds of natural origin have been considered. Some of the compounds of natural origin that have been studied are flavonoids, to which several biological activities have been attributed, including antioxidants anti-inflammatory, antimutagenic and anticancer effects. Although there are several types of these flavonoids, exhaustive studies are required to know in depth the effect of various members of this group of compounds such as quercetin and quercetagetin. In the present study, CaSki (HPV-16) and ViBo (HPV-negative) cervical cancer cells were treated with quercetin and quercetagetina to identify the antiproliferative and necrotic effect, and ability of the compounds to induce ultrastructural changes related to cell death. The results obtained showed that both compounds exert a dose-dependent antiproliferative effect, with quercetagetin being the most effective compound on CaSki cells, since lower concentrations are required than for ViBo cells. On the other hand, the microscopy results obtained denote that CaSki cells are driven to cell death by a pathway mediated by paraptosis, due to the described characteristics of the cellular organelles, while the cells of the ViBo line are eliminated by apoptotis. Both compounds conduce cervical cancer cells to a type of controlled cell death, which shows us the efficacy of these compounds to prevent cell proliferation, highlighting the importance that this type of death is not necrosis.

Effect of 21-gamma benzylidene digoxin (21-BD) on cell adhesion and cell migration

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Cellular adhesion involves stimulant signals that regulate cellular differentiation, the cell cycle, migration, and survival. The Na,K-ATPase, an important enzyme for the maintenance of cell homeostasis, also plays a key role in regulating cell-cell adhesion, cell adhesion to substrate, polarity regulation and cell migration. Our laboratory is currently studying the effects of cardiotonic steroids on cellular junctions. 21-benzylidene digoxin (21-BD) is a digoxin derivative that affects tight and adherens junctions. 21-BD treatment of MDCK cells increased the expression and internalization of the β1-subunit of Na,K-ATPase, as well as β-catenin translocation to the nucleus. Moreover, MDCK cells resistant to cardiac steroids did not show significant modulation by 21-BD, demonstrating that Na,K-ATPase is the main receptor for 21-BD. In addition, 21-BD was able to modulate tight junction proteins as Claudin-4 and -8 and ZO1 proteins. Moreover, 21-BD facilitated the formation of new cellular junctions faster than in untreated cells. In addition, an increase in lysosomal activity and decrease in cell migration were observed. Taken together, these results indicate that 21-BD is able to strengthen the junction between cells, which is of great importance in the context of decreased migration and cell invasion. These cellular effects could make 21-BD a prototype for the development of an anticancer agent that prevents the invasion of tumor cells in other tissues, facilitating treatment and improving the prognosis of the disease.

Stem cell models for aging in the eusocial mammal, the Naked mole-rat

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The regulation of lifespan by signaling between the reproductive system and intestine was revealed by studies in C. elegans, and correlative studies suggest such a relationship in mice and humans. The life history of the naked mole-rat (NMR) provides unique inroads to deconvoluting reproduction and lifespan. Similar to honeybees, NMRs live in colonies with a single reproductive queen while other females remain prepubertal via social suppression. In this 'super-ager' mammal, queens live substantially longer than subordinates, with no age-related decline in fecundity, and exhibit hallmarks of aging only after age 25. This, together with the potential for all subordinates to undergo reproductive activation, argues for an epigenetic regulation of lifespan in NMRs that is coupled to reproductive activation. We established a collection of NMR primary dermal fibroblasts and found that queen fibroblasts had discrete phenotypes compared to age-matched subordinate females: slower growth kinetics and decreased senescence. An elevation in the number and activity of mitochondria in queen fibroblasts was supported by RNA-seq, Mitotracker/TMRE and metabolomic studies. We devised a novel reprogramming platform for NMRs and found that queen fibroblasts reprogram more slowly. Resulting queen and subordinate NMR induced pluripotent stem cells (iPSCs) retained phenotypic differences, even through subsequent differentiation. This suggests increased epigenetic buffering and resistance to cellular reprogramming in queen NMR cells. Our ongoing efforts are testing the hypothesis that increased quiescence and epigenetic resilience promote longevity in queen NMRs through the induction and maintenance of a discrete epigenetic and metabolic state. This unique cellular toolbox provides the opportunity to characterize the epigenetic and metabolic features of queen cells, identify the underlying mechanisms, induce this state switch in vitro, and carry out high throughput screens in pluripotent and differentiated NMR cells. Funding from this work comes from the Keck Foundation, Weston-Havens Foundation and EMBO.

Plant Polar Growth Requires a Specific and Dynamic Distribution of Organelles to Coordinate Apical Growth and Responses to Biotic Interactions

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The apical growth of root hairs, pollen tubes and caulonema moss cells involves the regulation of the ions flow, calcium homeostasis, exocytosis, and cytoskeleton. However, is well stablished that Reactive Oxygen Species (ROS) and calcium also play an important role in the polar growth. The production of the ROS occurs in organelles such as mitochondria, chloroplasts, and peroxisomes. ROS is also produced by the enzymatic activity of NADPH oxidase, these enzymes transfer electrons from NADPH to an acceptor, the oxygen, to form the superoxide radical from which other ROS originate as H2O2 and been transported by aquaporins. NADPH oxidases can be recruited in membrane microdomains enriched in tetraspanins or flotillin at the tip of the growing cells. On the other hand, tetraspanins are integral membrane proteins with two extracellular loops containing several highly conserved cysteine residues. These loops allow their association to microdomain forming clusters in different levels of complexity. Tetraspanins also contributes to exosome formation, which are extracellular vesicles derived from the

multivesicular body that carry DNA, mRNA, microRNA, proteins, and lipids. Thus, the key role for exosomes is intercellular and interkingdom communication. We analyzed the phenotypes associated with PvTET8 silencing or overexpression during mutualistic interactions and provides evidences that contribute to mutualistic interaction in addition to its role in pathogenic responses. We also measured the dynamics of H2O2 levels in living plant roots, root hairs from Arabidopsis, pollen tubes and Caulonemas cells, expressing a genetic ROS sensor "Hyper". This sensor can be studied by a ratiometric analysis. Since moss experiment a polar growth in caulonema cells, the dynamic and role of ROS can be studied and correlated with intracellular calcium changes by expressing two sensors during polar growth and analyze their responses to external pathogenic elicitors. Our results depict an apical distribution of H2O2 in tip growing cells such as root hairs and moss caulonema cells. In addition, the NADPH oxidase enzyme also localize at the tip of growing root hair cells. The NADPH oxidase inhibitors, or treatment with external elicitors, had a profound impact on intracellular ROS levels. This is the first time that we can depict the ROS dynamic and its correlation with mutualistic interaction, polar growth, organelles distribution and dynamics.

Study of the correlation between stress granules and mitochondria in *C. elegans*

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To manage stress, organisms have developed some responses such as the formation of stress granules (SGs) and mitochondrial aggregation. SGs are one type of membraneless organelles or biomolecular condensates that contain RNA and RNA binding proteins. SGs in mammals are formed in response to translational arrest, in which scaffold proteins like TIA1/TIAR-1, TIAR and TTP/GLA-3 promote the condensation of stalled translational arrest complexes to protect them from harmful conditions. Once the stress is over, SGs dissociated and mRNAs can return to initiate translation. SGs formation and composition have been thoroughly studied however there is still so much to learn about their function and formation. In the C. elegans, stress granules are formed in the center of the gonad or raquis. We have observed that when one-day-old adult animals are exposed to starvation (6 h of bacterial deprivation) or heat shock (3 h of 31o C) SGs are formed in the gonad core and oocytes. Our group found that the RNA binding proteins TIAR-1 and GLA-3 are essential to induce SGs formation in the gonad core during stress. However TIAR-1 is dispensable for SGs formation in oocyte. We have also observed that during stress, mitochondria change their morphology becoming elongated and associate mainly in the gonad's raquis forming mitochondrial aggregates.

There is evidence that indicates that mitochondria can serve as scaffolds for the formation of other type of RNA granules like processing bodies, nuage or the chromatoid body. We have observed that under stress conditions SGs are formed in close proximity to the mitochondrial aggregates raising the question about the mechanisms that induce mitochondrial aggregation during stress and testing if there is a connection between the morphological change of mitochondria during stress and the formation of SGs. We determined that mitochondrial aggregation is not dependant of SGs formation; however, we found that formation of mitochondrial aggregates is dependant of mitochondrial dynamics. Assembling and disassembling of mitochondrial aggregates is mediated by mitochondrial fusion and fission events respectively. We aim to determine if the formation of mitochondrial aggregates play a role in the SGs formation evaluating the impact over the SGs formation during the impairment in the formation of mitochondrial aggregates. We would like to acknowledge Grants IN210821 and IN (PAPIIT-UNAM), and Conahcyt for supporting our research.

Selective regulation of linker histone H1 at the histone locus body

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The histone locus body (HLB) is a specialized nuclear sub-compartment responsible for the biosynthesis and post-transcriptional processing of histone mRNA. Replication-dependent core histones (H2A, H2B, H3 and H4) and linker histones (H1) are encoded by a large number of genes that are positioned within 4 clusters in the human genome. While the core histones contribute to formation of the nucleosome, and require strict equal stoichiometry, the linker histone H1 is not an obligate member of the nucleosome and does not have equal stoichiometry with the core histones. How histone H1 is differentially regulated within the HLB is not known. Perturbations in histone gene expression and stoichiometry exert detrimental effects on cellular processes and fitness. The formation of HLBs and the expression of the core histone genes is dependent on a well-conserved set of factors that includes NPAT. Here we investigate additional conserved factors involved in HLB formation and histone gene expression, with a particular focus on their selective regulation of the linker histone H1. We demonstrate a functional interaction between human CRAMP1 and the histone H1 genes. Localization of CRAMP1 demonstrates that it is a component of the human HLB, and affinity purification of CRAMP1 shows that it forms a complex with GON4L/YARP, a known human HLB component. Suppression of CRAMP1 leads to a reduction in histone H1 mRNA but shows no effect on core histone gene expression. Taken together, these data suggest that CRAMP1, in conjunction with NPAT and/or GON4L/YARP, selectively regulates histone H1 expression.

Biomolecular condensates at the nexus of innate immunity and venoms

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In the last decade it has become increasingly clear that biomolecular condensates are widespread within cells. Besides membranebound compartments, these assemblies constitute a powerful and versatile way for the cell to compartmentalize and orchestrate its internal biochemistry. While the number of validated scaffold and client proteins has steadily grown, how this behavior is actually encoded in sequence remains understudied. Since transmembrane domains can be pretty accurately predicted from sequence, similar efforts have been explored for condensate proteins. Most of the current prediction tools unfortunately rely on the available in vitro data for condensation. As the field initially focused on prion-like domains, the resulting predictions are often biased towards such sequences. Many condensates do not rely on such prion-like domains though, but typically come in different flavors of their chemical space. Here we leveraged a simple paradigm of complex coacervation to probe such additional chemistries. Using ex vivo enrichment strategies coupled to mass spectrometry-based proteomics, we unbiasedly uncover a whole new set of candidate condensate proteins. Training a machine learning algorithm on this dataset allows us to predict such behavior with surprising accuracy from sequence. This approach allowed us to uncover a whole new set of uncharacterized condensate proteins with essential functions to human cells. Additionally, our algorithm predicts that condensation behavior is pervasive—not only within—but also outside of cells. We unexpectedly uncover novel and evolutionary conserved roles for condensation in animal physiology, ranging from innate immune and venom systems to biomaterials and biofluids. Several of these protein classes and their condensation-driving features are rapidly evolving, arguing that phase separation is not merely an epiphenomenon but a core functional mechanism under direct selective pressure.

High throughput, high resolution imaging using the JEOL CRYO ARM platform

Jaap Brink
TEM Product Manager for Life Sciences, Jeol.
Cryo-EM has taken an enormous flight over the past years. Progress in scope automation and camera technology have made it possible to routinely image macromolecular complexes in their frozen hydrated state and solve their structure to near, and in some cases true atomic resolution. This talk will discuss

the technological advances made on the JEOL CRYO ARM platform and highlight a few specific case studies.

Genetic alterations and expression of factors that regulate mitochondrial genes in breast cancer

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Breast cancer is the most common cancer in women and the second leading cause of cancer death in the worldwide. Mitochondria in breast cancer, modulate bioenergetic plasticity that allows tumor cells to adapt and survive the tumor environment. Mitochondrial biogenesis is regulated by transcriptional programs that coordinate the induction of genes located in the mitochondria and the nucleus, which encode mitochondrial proteins. Our laboratory has a research project related to the analysis expression of Oxidative Phosphorylation System (OXPHOS) mRNA and proteins in breast cancer cell lines, where we found that 24 OXPHOS subunits were subexpressed, while the mRNA of the genes that code for some of these subunits was upregulated in the majority of breast cancer cell lines studied. It is of our interest to know what happens at the transcriptional level. For this reason, in this work we determined the expression of the mitochondrial transcription factors TFAM, TFB2M, NRF1, GABPA and the coactivator PGC1α in different breast cancer cell lines, as well as the genetic alterations and survival analysis. Gene expression was performed by RT-qPCR and the other analyzes were by bioinformatics tools using the cBioPortal, COSMIC and GEPIA databases. Our results showed that most genes were overexpressed in breast cancer lines. Missense mutations were the most frequent in all the genes evaluated. No significant differences in gene expression and survival in breast cancer patients. We conclude that breast cancer cells deregulate the expression of factors that control the transcription process of mitochondrial genes, which could be therapeutic targets in therapy against breast cancer.

Application of Nanotechnology in Biotechnology

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Nanotechnology has emerged as a transformative tool in biotechnology, offering innovative solutions to complex biological challenges. By manipulating matter at the nanoscale, novel materials and devices with unique physical, chemical, and biological properties can be designed. These nanoscale innovations enhance drug delivery systems, enabling targeted therapies with reduced side effects and improved efficacy. In diagnostics, nanotechnology allows for the development of highly sensitive biosensors, capable of detecting biomolecules at very low concentrations, thereby facilitating early disease detection. Furthermore, the integration of nanomaterials in tissue engineering has led to the creation of biocompatible scaffolds that support cell growth and tissue regeneration. Despite these advancements, challenges such as toxicity, scalability, and regulatory approval remain. Continued interdisciplinary research is essential to fully exploit the potential of nanotechnology in biotechnology, offering new avenues for personalized medicine and sustainable bioprocesses.

Analysis of Pulmonary Surfactant Protein C during Aging

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Aging is characterized by a decline in the activity of various biological mechanisms and an increased susceptibility to diseases1. Alveolar Epithelial Cells Type II (AECII) are responsible for the biosynthesis and secretion of Pulmonary Surfactant (PS), as well as in the regenerative capacity of alveolar epithelium. PS is a dynamic system that reduces surface tension within the alveoli. It is composed of phospholipids and hydrophobic proteins (SP-B, SP-C) that play a role in the surfactant's biophysical activity, along with hydrophilic proteins (SP-A, SP-D) associated with the lungs' innate immunity. Pulmonary surfactant

proteins SP-B and SP-C are stored in Multilamellar Bodies (MLB) within AECII.2 Their suppression is implicated in the disruption of pulmonary homeostasis3–5, a characteristic often linked to aging in the literature but supported by limited evidence. Therefore, it is crucial to study the changes in surfactant during aging to understand how these changes might contribute to aberrant changes in the alveolar epithelium. Methods. Immunodetection assays and relative gene expression analysis were performed to determine the expression status of SP-C in whole lungs and AECII from mice with accelerated aging (Zmpste24-/-) and natural aging (wild type, C57: 18-28 months). Ultrastructural analysis using Transmission Electron Microscopy (TEM) was conducted to identify changes in MLB. In organoid models, immunostaining assays for SPC and cell senescence markers were performed. Results. SPC expression was increased in AECII and lungs in both natural and accelerated aging models. Moreover, cells in culture from both models demonstrated the ability to proliferate and regenerate. Contrary to some studies, natural aging is associated with an increase in SP-C expression. The isolated cells retained proliferative capacity, suggesting that in diseases where SP-C activity is reported to be altered with aging, other initiating factors may be required. The knockout model also appears to successfully mimic what occurs with this system during natural aging. The presence and number of MLBs suggest the maintenance of surfactant activity; however, the presence of AM may indicate a more active role of the surfactant surveillance system. Conclusions. In summary, our study highlights an increase in SP-C expression during both natural and accelerated aging in the lung, contrary to previous findings. This suggests ongoing cellular regeneration despite aging. 1. López-Otín, C., Blasco, M. A., Partridge, L., Serrano, M. & Kroemer, G. The hallmarks of aging. Cell 153, 1194–1217 (2013). 2. Mason, R. J. & Dobbs, L. G. Alveolar Epithelium and Pulmonary Surfactant. in Murray and Nadel's Textbook of Respiratory Medicine134-149.e5 (Elsevier, 2016). doi:10.1016/B978-1-4557-3383-5.00008-7. 3. Nureki, S.-I. et al. Expression of mutant Sftpc in murine alveolar epithelia drives spontaneous lung fibrosis. J Clin Invest 128, 4008–4024 (2018). 4. Foster, C. D., Zhang, P. X., Gonzales, L. W. & Guttentag, S. H. In vitro surfactant protein B deficiency inhibits lamellar body formation. Am J Respir Cell Mol Biol 29, 259–266 (2003). 5. Ruwisch, J. et al. Air Space Distension Precedes Spontaneous Fibrotic Remodeling and Impaired Cholesterol Metabolism in the Absence of Surfactant Protein C. Am J Respir Cell Mol Biol 62, 466–478 (2020).

Enhancing Critical Thinking in Undergraduate Biology Students: The Effect of Gradual Active Learning Strategies

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Evidence has consistently shown that active learning (AL) is a more effective instruction method for developing higher cognitive skills than traditional lecturing across science, technology, engineering, and mathematics (STEM) disciplines. Despite this evidence, traditional lecturing remains in many universities worldwide. One way to overcome this problem is to offer more practical training for STEM faculty on implementing AL in their classrooms. At the Facultad de Ciencias (UNAM), we developed an AL training program for faculty members at the Facultad de Ciencias-UNAM in which we guided lecturers in planning and conducting evidence-based AL activities to assess critical thinking in undergraduate biology students. Here we report on the preliminary challenges, obstacles, limitations, and strategies faculty experience during their participation in the program.

Fibrillarin: Evolution, function and its implications in cancer

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Fibrillarin is a well-characterized protein primarily known for its role as a rRNA 2'-O-methyltransferase. In the well-described canonical complex, it interacts with NOP58, NOP56, p15.5, and guide RNAs (such

as U3, U6, U13, etc.) to form a ribonucleoprotein complex that methylates preribosomal RNA. However, fibrillarin also plays several other crucial roles, including epigenetic functions, where it methylates the H2A histone of ribosomal promoters, and transcriptional regulation at various stages, involving interactions with transcriptional mediators of RNA pol II promoters. Additionally, it is implicated in responses to viral and bacterial infections, where variations in fibrillarin levels and their mutations significantly influence viral progression and bacterial response. Furthermore, fibrillarin has a role in selective RNA processing, which is dependent on specific types of nuclear lipids, in particular phosphatidylinositol 4,5-bisphosphate. These diverse functions make fibrillarin an interesting subject of study and together with well-defined expression patterns of each of its domains, make it a valuable model for studying nuclear compartments, such as Cajal bodies and nucleoli due to its liquid-liquid phase separation properties. Fibrillarin is often considered an essential protein across a wide range of organisms, from archaea to humans. In this study, we present the dynamics and ultra-resolution structures of fibrillarin as well as selected mutants that affect the cell nucleus. Moreover, we show the evolution of both human fibrillarin genes and their syntenic-associated genes and explore how their expression differs across various types of cancer.

Mechanisms of neuronal senescence

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Cellular senescence is a phenotype characterized by a secretory phenotype that modifies the surrounding tissue. In aging, senescent cells are not efficiently cleared by the immune system leading to their accumulation in tissues, including the brain. Persistent senescent cells contribute to aging and age-related diseases, as they promote local inflammation and induce paracrine senescence. Therefore, it is fundamental to understand the molecular mechanisms of cellular senescence establishment and maintenance. On the other hand, the efficiency of autophagy decreases during aging in several species, yet the cause of autophagy dysfunction is still poorly understood. Neurons are particularly dependent on autophagy because they contribute to processes such as axon growth, synaptic formation and plasticity. We discovered that age-associated autophagy dysfunction contributes to neuronal senescence. Autophagic failure seems to be a consequence of exacerbated nuclear-cytoplasmic transport. Exportin-1/CRM1 regulates nucleus-cytoplasmic traffic of hundreds of proteins, including TFEB, the principal transcription factor involved in autophagy and lysosomal biogenesis genes expression. We found enhanced Exportin-1/CRM1 activity in old mice brains, concomitant with an increase of cytoplasmic TFEB in senescent neurons having autophagic flux impairment. By a pharmacological inhibition of Exportin-1/CRM1 activity in an in vitro model of neuronal senescence, TFEB was retained in the nucleus, autophagy function was restored and neural senescence was prevented. Currently, we are targeting Exportin-1/CRM1 to reduce its activity in middle-aged mice to evaluate if preventing autophagic failure prevents neuronal senescence in vivo. This work is funded by UNAM-PAPIIT IN205324, CONAHCyT CBF2023-2024-1669 and Pew Innovation Fund 2022.

Association between *Helicobacter pylori* infection and diabetes type 2 in a murine model

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Diabetes mellitus type 2 (DM2) is a multifactorial metabolic disease with a 13% worldwide prevalence, while in Mexico is around 10.3%. Otherwise, the *Helicobacter pylori* (Hp) infection affects more than half of the world's population, being even higher in our country (>70%). The prevalence of both diseases displays a great impact on public health, and many epidemiological studies are being carried out in several countries. To understand the development of these illnesses several animal models are employed. The gerbils have been infected with Hp and mice have developed DM2. However, there is no

animal model that encompasses both DM2 with Hp infection, so far. For this reason, the aim here was the generation of an in vivo model to elucidate their relationship, and the molecular mechanisms involved between both pathologies. Mice C57/BL6 strain were fed with a high glucose diet (HGD) and treated with streptozotocin to damage the pancreatic b-cell, in addition to being infected with Hp PMSS1 strain. After three months of treatment, the zoometric results showed that diabetic, infected/diabetic and infected animals presented an increase in body weight, waist circumference, Lee index and adipose epididymis, compared to the control group (fed with a normal diet). In addition, the histological analysis of the gastric tissue of these mice showed an inflammatory process. Moreover, the biochemical analyses of infected groups, showed an increase in the levels of glucose, triglycerides and total cholesterol. A reduction of insulin in the islets of Langerhans and a decrease of the GLUT2 receptor in the pancreatic acini were also observed, both in infected and diabetic mice. Derived from these findings, we can conclude that we generated a murine model that combines both pathologies (bacterial infection and DM2). In these animals, significant morphological changes, alterations in glucose and lipid metabolism, and pancreatic functions were observed, suggesting that Hp infection could be a risk factor for DM2 development. Acknowledgments We acknowledge support from CINVESTAV in the development of this research work.

Molecular regulation of mitochondrial homeostasis and its contribution to aging associated diseases

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Mitochondria are highly dynamic and undergo constant turnover through two opposing processes: mitophagy, which selectively removes superfluous and damaged mitochondria by the autolysosomal pathway, and mitochondrial biogenesis, which generates fresh, functional ones. We have previously characterized that FUNDC1functions as a mitophagy receptor to mediate hypoxia-induced mitophagy. Interestingly, we found that PGC-1α and NRF1, master regulators of mitochondrial biogenesis and metabolic adaptation, also transcriptionally up-regulate Fundc1 in response to cold stress in brown fat tissue (BAT). Specific knockout of Fundc1 in BAT results in reduced mitochondrial turnover and accumulation of functionally compromised mitochondria, leading to impaired adaptive thermogenesis. Recently, we showed that inducible loss of endothelial Fundc1 in postnatal mice was sufficient to cause spontaneously Pulmonary Arterial Hypertension (PAH), currently non-curable disease. Fundc1 deficiency in endothelial cells, but not in smooth muscle cells, leads to metabolic reprogramming, pseudohypoxia and senescence. We further characterize the molecular pathways driving pulmonary arterial remodeling and suggest new strategies to ameliorate PAH. Collectively, our results demonstrate that FUNDC1 dictates mitochondrial quantity, quality and turnover and contributes to adaptive thermogenesis and aging related diseases.

Bro1 proteins govern multivesicular body fate switch to regulate exosome secretion and cancer progression

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Exosomes play pleiotropic tumor-promoting functions and are secreted by fusion of multivesicular bodies (MVBs) with the plasma membrane. However, MVBs are also directed to lysosomes for degradation and the mechanism controlling different fates of MVBs remains elusive. Here, we show that the pro-tumor protein WDR4 enhances exosome secretion through degrading the ESCRT-associated Bro1-family protein PTPN23. Mechanistically, PTPN23 and its paralog ALIX compete for binding to syntenin, thereby directing MVBs towards degradation and secretion, respectively. ALIX, but not PTPN23, recruits actin-capping proteins CAPZA1/CAPZB to prevent branched F-actin accumulation around MVBs, thus enabling MVBs trafficking to cell periphery for secretion. Functionally, WDR4/ALIX-dependent exosomes preferentially load a set of pro-tumor proteins to potentiate metastasis and

immune evasion. Our study highlights a previously unprecedented coupling between biogenesis mechanism and fate decision of MVBs and its importance in exosome cargo landscape, which have potential broad impacts on boosting exosome production and devising cancer therapeutic strategy.

Linking Nuclear Envelope Dysfunction to Cellular Senescence

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The nuclear lamina is a meshwork of type V intermediate filament proteins consisting primarily of A and B type lamins. In addition to providing mechanical strength to the nucleus, the nuclear lamina is associated with several cellular processes such as gene expression, DNA repair, cell cycle progression, and chromatin organization. Mutations in nuclear lamin A are casual for the Hutchinson-Gilford progeria syndrome (HGPS), a rare disease that displays several features associated with aging including muscle weakness, atherosclerosis, low-frequency conductive, hearing loss, elevated blood pressure, reduced vascular compliance, and adventitial thickening. Using human HGPS skin fibroblasts and two mouse models, Lmna-/- and Lmnadel9 that respectively represent dystrophic and progeric laminopathies, we report that a common explanation for these lamin-associated dysfunctions appears to be the over-accumulation of the Sun1 protein. We determined that farnesylation of progerin, the lamin A mutant protein that causes HGPS, enhances its interaction with SUN1, forms aggregates with SUN1 in the endoplasmic reticulum (ER) cisternae during mitosis, and leads to the accumulation of SUN1, thereby disturbing the nuclear envelope and ER structure. We also created a mouse model in which progerin can be inducibly overexpressed and discovered that muscle-specific overexpression of progerin was sufficient to induce muscular dystrophy and alter whole-body energy expenditure, leading to premature death. An examination at the molecular level revealed that progerin recruits Sln and Calnexin to the nuclear periphery. Furthermore, progerin-expressing myoblasts presented enhanced store-operated Ca2+ entry, as well as increased co-localization of STIM1 and ORAI1. These findings suggest that progerin dysregulates calcium homeostasis through an interaction with a subset of ER-associated proteins, resulting in thermogenic and metabolic abnormalities.

Na+/K+-ATPase: a unique multifunctional protein, serving as a sodium pump, an adhesion molecule, and a signaling receptor, crucial in controlling cell adhesion

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The Na+/K+-ATPase is the ion transporter of the plasma membrane responsible for generating the characteristic Na+/K+ gradient of animal cells that cardiotonic steroids inhibit. Blanco and collaborators' contributions are essential for understanding the biological roles of the Na+/K+-ATPase α4 subunit, including motility, that specifically expresses spermatozoa. Besides its crucial role as an ion transporter, the Na+/K+-ATPase is also an adhesion molecule in astrocytes and epithelia. In the last tissue, the adhesion conferred by its β-subunit is necessary for the polarized expression of the enzyme in the lateral membrane; recent studies by Shoshani and collaborators unravel the structural details of the Na+/K+-ATPase β-subunit that confers cell adhesion in astrocytes and epithelial cells and, therefore, the polarized expression of the enzyme in the lateral membrane of epithelial cells, necessary for the vectorial transport of nutrients and waste substances through epithelia. Work by Barbosa and collaborators also shows how the chemically modified cardiotonic steroid 21-benzylidene digoxin (21-BD) regulates the expression of claudins, the transmembrane proteins of the tight junctions of epithelial cells. In our laboratory, we study how the Na+/K+-ATPase is also a receptor that signals the presence of environmental cardiotonic steroids, principally the prototypical compound ouabain, to control migration, differentiation, adhesion, and death in epithelial cells. All this research shows that the

Na+/K+-ATPase is a multifunctional enzyme whose biological roles include ion transport, adhesion, and cell motility.

SIAH2 is potentially related to HIF-α increase in human and rat experimental renal cell carcinoma cell lines

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Even though renal cell carcinoma (RCC) is the main kidney cancer worldwide, molecular mechanisms involved remain largely unexplored. More than 70% of the clear cell RCC (ccRCC) cases, the most common subtype, exhibit an inactivation of VHL gene, which codes for a ubiquitin ligase which marks the alpha subunit of hypoxia-inducible factor (HIF-α) for proteasomal degradation. Although this seems to explain the widely reported increased HIF-α levels in ccRCC, it is not rare to find this alteration even in the presence of an apparently functional pVHL. The seven in absentia homolog (SIAH) is a family of ubiquitin ligases that have been proposed for explaining the HIF-α accumulation in different cell types without alterations in pVHL since one of their substrates, the prolyl hydroxylases (PHD), promote HIF-α degradation. SIAH1 and SIAH2 role has been described in various types of cancer, however, their behavior in RCC is yet unknown. The HIF-1α, HIF-2α, SIAH1 and SIAH2 protein levels, as well as the pVHL status were analyzed by western blot in two non-cancerous rat (NRK-52E) and human (HK-2) kidney cell lines as references, a rat chemically induced RCC cell line (RC5E) and two human RCC cell lines with different VHL genotypes: ACHN (VHL+/+) and 786-O (VHL-/-). High levels of HIF-α were observed in 786-O and RC5E, although the presence of pVHL was confirmed in the latter, as well as in ACHN where no changes in HIF-α were detected. Regarding SIAH1 levels, no significant differences were identified, while high levels of SIAH2 were found in RC5E and 786-O, but not in ACHN. Our results reinforce the idea that SIAH2 may be involved in the pVHL-independent HIF-α regulation in certain RCC cases, by participating in its stabilization as observed in other types of cancer; however, further experimental evidence is required. Supported by: CONACYT-F003-284155, UNAM-DGAPA-PAPIIT IN-228716 and FQ-PAIP 5000-9109.

Viridicatin isolated from the antarctic fungus *Penicillium* sp. shows neuroprotective effect on HMC-3 microglia cells and prevents HO-1 traslocation to the nucleus under H2O2 oxidative stress

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Neurodegenerative diseases have increased due to the systematic aging of the population. Microglia plays a critical role in the central nervous system's (CNS) homeostasis. Aging significantly affects the optimal functioning of microglia due to sustained oxidative stress, which activates the Nrf2/HO-1 antioxidant pathway. After its synthesis, HO-1 protein is normally delivered and anchored to the membrane of the smooth endoplasmic reticulum (SER) to exert its antioxidant activity. However, under extreme stress conditions, its nuclear translocation has been observed, which is associated with the development of neurodegenerative diseases. The search for new drugs capable of protecting these CNS resident cells is vital for the prevention of aging-related cognitive impairment. Antarctic fungi represent a novel source of molecules with biotechnological potential for the treatment of priority diseases. The Antarctic fungus *Penicillium* sp. was cultured in 80 flasks with YM medium for 28 days, for subsequent extraction with EtOAc. The total organic extract was fractionated by affinity chromatography with increasing polarity mobile phase. After purification, compounds isolated structures were analyzed by crystallographic analysis (XRD), NMR (600 MHz), and EI-ESI-HRMS. Three molecules were obtained as crystalline solids: two benzodiazepines Cyclopenin and Cyclopenol, and the quinoline Viridicatin. The cell protective effects of isolated compounds were assassed in HMC-3 microglia cells, pre-incubated with the molecules, and then treated with H2O2 (300 μM) for 24 hours with Sytox in INCUCYTE. In

addition, immunofluorescence microscopy was performed in confocal microscopy to observe and quantify Nrf2, HO-1, and NOQ1. Viridicatin showed a protective role against damage caused by oxidative stress induced by H2O2, with an IC50 of 50 µM and almost 100% protection at a concentration of 100 µM. Furthermore, it was observed that this molecule prevents nuclear translocation of HO-1 compared to the control (microglia treated with H2O2). Therefore, Viridicatin has potential as a future neuroprotective drug.

Towards a systems-level understanding of cellular aging in yeast

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A major challenge in aging research is to describe the way in which different genetic factors are interconnected to one another and the environment. We will present results of functional genomics screens using large collections of budding yeast single- and double-gene knockouts coupled to automated chronological lifespan profiling. Our genomewide assays and meta-analyses expose a consistent set of conserved genetic drivers of aging and longevity in this simple unicellular model. Genetic screens of longevity by metformin coupled to genetic epistasis analyses reveal novel players orchestrating postmitotic cellular survival, which includes chromatin-remodeling, retrotransposon expression, and non-coding RNAs. I will present evidence that lifespan extension by metformin and Set3-mediated histone deacetylation involves a complex cellular crosstalk involving mitochondrial function, Ty retrotransposition, and tRNA pool balance. In addition, Swr1C histone-exchange impairment also extends lifespan by altered tRNA pool balance, with impacts on cellular proteostasis. The chronological lifespan paradigm in budding yeast provides an unprecedented systems-level perspective on the genetic players involved in promoting longevity and their regulatory cross-talks in aging cells.

Three-dimensional analysis of interchromatin granules in the reptile *Sceloporous torquatus*

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The cell nucleus contains several ribonucleoprotein particles involved in RNA metabolism. Among them are the perichromatin fibres and granules and interchromatin granules. Interchromatin granule clusters (IGCs) and perichromatin fibres are known also as speckled pattern when analyzed by immunofluorescence microscopy using antibodies against pre-mRNA processing factors. In mammals, interchromatin granules are constituted by about 15 nm in diameter granules associated to fibers. To see whether granules and fibres are spatially associated in reptiles, we prepared samples from liver of the lizard *Sceloporous torquatus* to transmission electron microscopy. Thin sections were treated with standard technique and for EDTA regressive staining for ribonucleoproteins. Images were taken using a goniometer coupled to the microscope and displayed as stereopairs. Results show that interchromatin granules are connected to surrounding fibers within the IGCs (supported by PAPIIT-DGAPA-UNAM IN223223).

The parabasal filaments of *Trichomonas vaginalis*: a new filament and observations using ultra-high-resolution Scanning Electron Microscopy

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Trichomonas vaginalis is the etiologic agent of trichomoniasis, the most common nonviral sexually transmitted infection worldwide, with an estimated 260 million new cases annually. T. vaginalis contains

a complex and elaborate cytoskeleton constituting the mastigont system, which is mainly formed by several proteinaceous structures associated with basal bodies, the pelta-axostylar complex made of microtubules, and striated filaments named the costa and the parabasal filaments (PBs). Although the structural organization of trichomonad cytoskeletons has been analyzed using several techniques, observation using a new generation of scanning electron microscopes with a resolution of below 1 nm has allowed more detailed visualization of the three-dimensional organization of the mastigont system. In this study, we have investigated the cytoskeleton of T. vaginalis using a diverse range of scanning probe microscopy techniques, which were complemented by electron tomography and Fast-Fourier methods. This multi-modal approach has allowed us to characterize an unknown parabasal filament and reveal the ultrastructure of other striated fibers that have not been published before. Here, we show the differences in origin, striation pattern, size, localization, and additional details of the PBs, thus improving the knowledge of the cell biology of this parasite.

Evaluation of fibrillar components of extracellular matrix in the vaginal wall in women with pelvic organ prolapse

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Pelvic organ prolapse (POP) is a pathology characterized by the descent of one or more vaginal compartments. The vagina plays an important role in balancing and supporting the pelvic organs and viscera in women. The fibrillar organization of the extracellular matrix (ECM) provides structural integrity to tissues. Then changes in their ECM components, can lead to the genesis or progression of POP. Then, our aim is to understand how the organization of fibrillar components of ECM can provide a basis for understanding POP. A prospective study was carried out in a consecutive sampling of 30 women with genital prolapse and 30 women from the control group (without prolapse) assisted at the Women's Hospital at State University of Campinas (UNICAMP). This study was approved by the Research Ethics Committee (under the protocol 59334021.0.0000.5404). During surgery, vaginal wall biopsies were taken to carry out morphological analyzes using light microscopy for Masson's Trichrome, resorcin fuchsin and Eosin and Hematoxylin staining for second harmonic generation microscopy, and Transmission Electron Microscopy. Our morphological analyzes show that the vaginal wall mucosa of women with POP, when compared to the control group, presents loose connective tissue with spaced and thinner collagen fibers and with probably an increase in ground substance in the lamina propria. Second harmonic generation microscopy shows thinner bundles of collagen fibers in the case group. In the control group, the elastic fibers are long and thin, while in the case group, they become apparently longer and thicker. Our ultrastructural analyses show small clumps containing elastin and microfibrils interspersed between collagen fibrils in the control group and in the case group, large arrays of microfibril bundles were observed with amorphous clumps of elastin. Therefore, our preliminary morphological results indicate that POP leads to a loss of homeostasis of ECM components in the vaginal wall.

Fructan dietary fibers and immunological effects

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In recent years, it has become increasingly evident that many food components play a crucial role in influencing health, either by affecting gut microbiota or directly interacting with the cells of the gastrointestinal immune barrier. The gut, which regulates a significant portion of the body's immune responses, is home to approximately 80% of the human immune system's cells. These immune cells work in a highly coordinated manner, equipped with specialized receptors that allow them to distinguish between beneficial and harmful entities. This precision is essential, as the gut must tolerate over 100

trillion commensal bacteria that support metabolism and immunity, producing vital compounds that the human body cannot generate on its own. At the same time, the gut immune system must eliminate pathogens and toxins hidden among these beneficial bacteria. Advances in recent years have shed light on how specific food components can bolster gut immunity. By leveraging cutting-edge technologies and improved analytical methods to characterize dietary fibers, we are now better equipped to identify immune-regulating carbohydrates. Our research has led to several groundbreaking discoveries with significant practical applications. We have found that not all carbohydrates exert the same effects across different age groups or health conditions, with efficacy being heavily dependent on carbohydrate chemistry. For instance, we demonstrated that the molecular length of certain inulins plays a critical role in preventing type 1 diabetes in animal models. More recently, in a groundbreaking collaboration with the National Autonomous University of Mexico (UNAM), we discovered that specific inulins derived from Agave possess remarkable immunomodulatory properties. Together with our colleagues at UNAM, we were able to pinpoint binding sites on specific pattern recognition receptors, marking a significant step forward in our understanding of how dietary fibers can influence immunity. In human studies conducted at our own institute, we have shown that carefully selected carbohydrates can support immune function, even delaying age-related immune decline through targeted interventions. Our findings highlight the importance of food component chemistry in regulating immune responses and underscore the need for tailored approaches to maximize the health benefits for specific populations.

NOX-mediated Downregulation of VEGF function by Omega-3-fatty acid in aortic smooth muscle cell cultures treated with High Glucose and Palmitic acid

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Background and aims: High glucose (HG) and palmitic acid (PA) are believed to be responsible for diabetes- and obesity-related risk of cardiovascular complications. Vascular endothelial growth factor (VEGF) is vital for formation, growth and function of vasculature. We examined the effect of Omega-3-fatty acid (OFA), on mitogenic function of VEGF in rat aortic smooth muscles (VSMC) cells treated HG and PA. Methods: Primary cultures of VSMC were set up by enzymic digestion of the medial layer and maintained in DMEM F-12 medium. Confluent cell cultures were treated with PA (500 µM) and /or OFA (100 µM) with / without HG (25 mM). Mitogenic function of VEGF (50ng/ml) was measured by BrdU incorporation and NADPH oxidase (NOX) activity was assayed by chemiluminescence method. VEGF receptor (VEGFR) expression was assessed by Western blotting. Results: VEGF-induced BrdU incorporation was increased ($p < 0.01$) by HG which was further enhanced by PA. Both HG and PA significantly increased the NOX enzymatic activity and VEGFR levels. Diphenyliodonium (DPI), a NOX inhibitor, attenuated PA- and HG-mediated increase in VEGF-mediated BrdU incorporation. OFA (100 µM) markedly reduced ($p < 0.01$) the incremental effect of PA and/or HG on NOX activity, VEGFR expression and VEGF mitogenic function. Conclusion: These results suggest that NOX-mediated increase in VEGF mitogenic activity by PA and HG might have a role in pathogenesis of diabetes and obesity, and OFA is a potential therapeutic agent.

Annexin A1 Deficiency Exacerbates Liver Pathology in a Type 1 Diabetes Mouse Model: Insights into Hepatocyte Protection Mechanisms

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Diabetes mellitus (DM) is a global public health issue causing systemic dysregulations, including severe liver complications. Type 2 diabetes (DM2) patients show elevated annexin A1 (AnxA1) levels, and in murine DM2 models, AnxA1 mitigates insulin resistance effects like hepatosteatosis. However, its role in DM1 is underexplored. This study investigates AnxA1's role in hepatocyte biology in a streptozotocin (STZ)-induced DM mouse model. Male C57BL/6 mice (AnxA1+/+ and AnxA1-/-) were divided into control (CTR) and DM groups. DM was induced via STZ injection (65 mg/kg for 5 days). After 12 weeks, livers were collected for analysis. Both DM AnxA1+/+ and AnxA1-/- mice showed weight loss and increased blood glucose, reflecting typical diabetic metabolic disruptions. Morphological evaluations revealed normal hepatocytes in AnxA1+/+ CTR mice, while 70% of AnxA1-/- CTR mice showed cytoplasmic vacuolation. In DM groups, 50% of AnxA1+/+ mice had vacuolated, damaged hepatocytes, and this increased to 75% in AnxA1-/- DM mice, highlighting AnxA1's protective role in diabetic livers. Glycogen levels in hepatocytes were lower in DM mice, more so in AnxA1-/- DM mice. Collagen deposition in centrilobular veins and portal triads was higher in AnxA1-/- DM mice, indicating worsened fibrosis without AnxA1. Both DM AnxA1+/+ and AnxA1-/- livers showed reduced fibroblast growth factor 2 (FGF2) and vascular endothelial growth factor A (VEGF-A) levels, suggesting impaired regeneration. Inflammation varied between genotypes: AnxA1+/+ DM mice had higher IL-10 and TNF-α, while AnxA1-/- DM mice showed increased monocyte chemoattractant protein-1 (MCP-1). Oxidative stress markers indicated increased reactive oxygen species (ROS) in AnxA1-/- DM hepatocytes, along with differing trends in nitric oxide (NO), superoxide dismutase (SOD), and catalase (CAT) activities between groups. This suggests AnxA1 is crucial in liver protection under diabetic conditions. Keywords: type 1 diabetes; hepatocyte; oxidative stress; cytokines; angiogenic factors. Funding: CAPES (code 001) and FAPESP (2023/16282-7)

Morphological aspects and immunolocalization of hormone receptors in the uterine cervix of primiparous and multiparous senescent mice during pregnancy and postpartum

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During a woman's reproductive life, various factors, such as age, pregnancy, and childbirth, contribute to changes in reproductive organs. The uterine cervix is crucial for supporting pelvic organs, with hormones like estrogen, progesterone, and relaxin influencing cervical tissue remodeling. Loss of hormonal homeostasis may compromise the integrity of the cervix, potentially causing pelvic floor dysfunction (PFD). This study aims to evaluate the immunolocalization of hormone receptors in the uterine cervix of primiparous and multiparous senescent mice during pregnancy and postpartum. Histological evaluation using Hematoxylin-Eosin (HE) staining, morphometric analysis, and localization of estrogen, progesterone, and relaxin receptors were performed on cervix samples. The study was approved by the Ethics Committee for Animal Use CEUA-IB/Unicamp No. 5542-1/2020, with tissue collection at day zero (D0) and day 18 of pregnancy (D18), as well as 3 (3dpp) and 21 days postpartum (21dpp). HE-stained sections revealed that multiparous senescent mice exhibited increased epithelium height and stromal disorganization, indicating delayed remodeling compared to primiparous mice. The cervical lumen area and lamina propria thickness were altered in multiparous mice, especially at 21dpp, indicating altered recovery to the pre-pregnancy state. Immunohistochemical analysis revealed distinct hormone receptor expression patterns in multiparous senescent mice compared to primiparous. Estrogen and progesterone receptors were more prominently expressed in the epithelial layer at D18 in multiparous mice, while relaxin receptors showed increased localization in the stroma during postpartum recovery. These findings suggest that multiparity significantly alters hormone receptor expression in the uterine cervix, which may contribute to delaying its return to the pre-pregnancy state, highlighting the role of hormone receptor regulation in cervical homeostasis and its impact on PFD.

Analysis of oxidative stress in MCF-7 cells treated with L-arginine

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Cancer is one of the leading causes of mortality across the world and is caused due to faulty cellular processes, which result in uncontrolled cell growth and therefore affect normal cell function. Therapeutic approaches against cancer have been the subject of several studies, because in many cases the forms of treatment are effective, but the comorbidities are aggravating for the prognosis and survival, including breast cancer. The use of L-arginine (L-ARG) in dietary supplementation for cancer patients has shown positive results due to its relevance in different body systems, but its use may have direct effects on tumor cells. Objective: Investigate the effects of treatment with L-arginine in the tumor lineage MCF-7 and expression of enzymes related to oxidative stress. The tumor cell line used was MCF-7. Methodology: The treatment consisted of exposure to concentrations of 800/1600/3200 µg/mL of L-ARG for 48 hours. The values of advanced oxidation protein products (AOPP) lineage MCF -7 were established by spectrophotometry in a microplate reader. The AlamarBlue assay was used to measure proliferation and assess cell viability. Evaluation of catalase, NRF2 and NOX2 expression was performed by Western blot and ImageJ software was used to measure the amount of each protein. Data were analyzed using the GraphPad Prism 8.0.2 software and using the one-way ANOVA test. Results: Changes in MCF-7 cells were reduction in cell viability when treated with L-arginine at doses of 1600ug/mL and 3200 µg/mL, increase in advanced protein oxidation products. There was also a reduction in the expression of catalase and NRF2 and an increase in the expression of NOX2. L-ARG as a complementary therapy to conventional treatments can prove to be effective, since changes in the concentration of proteins related to the formation of reactive oxygen species and in the antioxidant system (Catalase) that configure a holistic vision, favorable to the promotion of research clinic.

A tale of two amebas

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Next year will be 100 years of the pioneering observation of a French scientist, Dr. Emile Brumpt, that almost 70 years later led to a groundbreaking accomplishment in the field of amoebiasis research. In 1925, Dr. Brumpt suggested that there were two species of amebas, one capable of causing invasive disease and one that never causes disease, which he called E. dispar. His hypothesis was dismissed by other scientists, until biochemical, immunological and genetic data accumulated in the 1970's and, in 1993, a formal redescription of E. histolytica was published, distinguishing it from E. dispar. I will present a brief account of the events that led to the recognition of the two amoebic species as separate entities, and I will also describe the work that our laboratory has done over the years - some of it unpublished - to characterize the two species.

Understanding TGF-β Actions in Cancer

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Loss of the antiproliferative response is a hallmark in human cancers. Tumor cells have developed a number of strategies to escape from antigrowth control. One major mechanism to resist the cytostatic effect of antigrowth factor such as TGF-□ is through inactivating mutations/deletions in the TGF-□ signaling pathway, which frequently occur in gastrointestinal and pancreatic cancer. For example, tumor suppressor Smad4/DPC4, the central transducer of TGF-□ signaling, is deleted in more than half of pancreatic cancer patients. However, deletion or mutations in the Smad4 gene are rare in other types of cancers. We have studied how the tumor suppressor function is regulated in normal and cancer cells. We found that TGF-□ signaling is fined by multiple oncoproteins. For instance, hyperactivation of many

oncoproteins can cause TGF-β resistance. Our novel studies gain conceptual insights into the oncoprotein-tumor suppressor interplay in tumorigenesis and provide guidance to logical therapeutic designs in cancer prevention, diagnostics and treatment.

Immune intestinal rescuing effects of two Agave fructans against the Giardia virulence factor arginine deiminase

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INTRODUCTION. *Giardia lamblia* is the causative agent of human giardiasis and represents a crucial cellular reference for studying the infection process caused by protozoans. The parasite's ability to impact the host's immune system to sustain the infection is essential to its pathogenicity, being arginine deiminase (GlADI), one of its virulence factors that participates in the crosstalk during host-parasite interaction. It is not entirely understood how natural products such as dietary fibers regulate key pathophysiological processes related to parasitosis and inflammation. Dietary fibers such as graminan-type fructans (GTFs) extracted from agave plants are immunomodulators that can attenuate proinflammatory responses by inhibiting the activation of Toll-like receptors 2 and 4 (TLR2 and TLR4) and protect the intestinal epithelial barrier. OBJECTIVE. To investigate the effects of GlADI on intestinal immune response and the potential protective role of two GTFs with different chain lengths. METHODS. Caco-2, goblet, HEK and dendritic cells were incubated in presence of recombinant GlADI, or pre-incubated with GTFs followed by the addition of GlADI and 24 more hours of incubation. In Caco-2 cells, transepithelial resistance (TEER), expression of tight junctions (TJ), and interleukin-8 (IL-8) production were determined. In goblet cells the expression of mucus- and endoplasmic reticulum stress-related genes, and IL-8 production were measured. The activation or inhibition of TLR 2 and TLR 4 were quantified in HEK cells. The cytokine release by dendritic cells was determined. RESULTS. GlADI neither changed TEER nor TJ distribution, however it significantly decreased IL-8 production in Caco-2 cells, this effect was prevented by pre-incubating cells with GTFs. GlADI increased the expression of MUC2 and XBP1 genes in Goblet cells, whereas GTFs rescued them from this adverse effect. In dendritic cells, GlADI increased the production of the proinflammatory cytokine IL-1β, which was counteracted by GTF II. Activation of TLR2 and TLR4 was significantly increased by GlADI, and pre-incubation with GTF I inhibited TLR2 activation, whereas pre-incubation with both GTFs inhibited the activation of TLR4 in a dose-dependent manner. DISCUSSION AND CONCLUSIONS. GlADI strongly influences the intestinal immune response towards a tolerogenic response decreasing IL-8 production, while GTFs rescue enterocytes of such an infective parasite strategy. Until the best of our knowledge, this is the first time GlADI is proposed as responsible for this effect, since it previously was attributed to the degradation of IL-8 by Giardia proteases.

Pathogenic *E. coli*- A master cell biologist

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Pathogenic *E. coli* causes worldwide diarrhea and significant moridity and mortality. As part of its pathogenesis, it establishes close contact with host epithelial cells. While adhering to the host cell, it has a type III secretion system that injects greater than 20 bacterial molecules ("effectors") into host cells that have many cell biology effects. It rearranges cytosekeletal actin to build a pedestal upon which it resides, it dampens the immune response, it affects mitochondria and cell death, and overtakes several cell biology pathways to exploit host cells. A general discussion of how this pathogen rewires the cell

biology of its host cell will be given. These effectors are excellent ways to learn more about cell biology in a rather non-conventional way- through infection.

Differentiation into osteoblasts of Wharton's Gelatin-derived Mesenchymal cells on gelatin-hyaluronic acid scaffolds loaded with microspheres containing BMP-2 and VEGF

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Scaffolds made of natural materials such as gelatin (Ge) and hyaluronic acid (HA) were used for the treatment of bone defects. Chitosan was also used for the fabrication of microspheres and the delivery of growth factors due to its mucoadhesive properties. Furthermore, the growth factors BMP-2 and VEGF encapsulated and included in Ge-HA scaffolds can be gradually released and induce cell differentiation and proliferation.

XAANTAL1 and 2 in flowering transition and meristems maintenance

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MADS-box transcriptional factors are relevant in proliferation/differentiation decisions among eukaryotes. XAANTAL1 or AGAMOUS-like 12 (AGL12) and XAANTAL2 (AGL14) are involved in root development and flowering transition. In the root, xal1mutant alleles show a short-root phenotype with a smaller meristem, and one-third of the mutants showed abnormal root apical meristem organization. In agreement with these phenotypes, XAL1 positively regulates some cell-cycle components. Also, in the xal1-2 root, cells start elongating before WT cells stop growing before reaching the differentiation zone. In the shoot apical meristem, XAL1 positively regulates FD, a critical transcription factor that can bind to FT or TFL1 cofactors. In the presence of the first one, flowering transition takes place in response to long-day photoperiod, but in the presence of TFL1, plants remained longer in the vegetative phase. Interestingly, TFL1-FD is necessary for the identity of the inflorescence meristem, while AP1 gives identity to the flower meristem. These proteins repress each other expression. We have found that XAL2 regulates TFL1, and constitutive overexpression of XAL2 alters the flower meristem, acquiring vegetative and inflorescence traits due to up-regulation of TFL1 in this meristem. Moreover, XAL2 overexpression up-regulates AP1, generating early flowering plants instead of late flowering. XAL2 mediates ageing signals and response to phytohormones such as gibberellins, whereas XAL1 is regulated by light and warm temperatures. This way, XAL1 and XAL2 help fine-tune flowering transition to different external and internal signals. This research has been possible with financial support from the UNAM (latest project PAPIIT, DGAPA IN203223) and CONAHCyT.

Ultrastructural Stereopairs analysis of the archaea *Haloferax volcanii*

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Ultrastructure of archaea cells is scarce. To better understand the internal structure of *H. volcanii*, we performed standard transmission electron microscopy of cells growing in log pase. We fixed cells with glutaraldehyde and paraformaldehyde; grids were contrasted with uranyl acetate and lead citrate. We then made images by using the goniometer coupled to the electron microscope, by tilting the sample at 15 degrees. Stereopairs were also presented after using the program. Results indicate a widht external layer of nm and a heterogeneous cytoplasm including electron opaque and also translucid areas. All

these área are connected. Future studies will include a detailed analysis of several of these cytoplasmic areas.

Rules of engagement for condensins and cohesins guide mitotic chromosome formation

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During mitosis, interphase chromatin is rapidly converted into rod-shaped mitotic chromosomes. Using Hi-C, imaging, proteomics and polymer modeling, we determine how the activity and interplay between loop-extruding SMC motors accomplishes this dramatic transition. Our work reveals rules of engagement for SMC complexes that are critical for allowing cells to refold interphase chromatin into mitotic chromosomes. We find that condensin disassembles interphase chromatin loop organization by evicting or displacing extrusive cohesin. In contrast, condensin bypasses cohesive cohesins, thereby maintaining sister chromatid cohesion while separating the sisters. Studies of mitotic chromosomes formed by cohesin, condensin II and condensin I alone or in combination allow us to develop new models of mitotic chromosome conformation. In these models, loops are consecutive and not overlapping, implying that condensins do not freely pass one another but stall upon encountering each other. The dynamics of Hi-C interactions and chromosome morphology reveal that during prophase loops are extruded in vivo at ~1-3 kb/sec by condensins as they form a disordered discontinuous helical scaffold within individual chromatids.

A Novel Chimeric Transcriptional Modulator Maintains Mitochondrial DNA Integrity in the Yeast *Saccharomyces cerevisiae*

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In *Saccharomyces cerevisiae*, the transcriptional repressor Nrg1 (Negative Regulator of Glucose-repressed genes) and the b-Zip transcription factor Rtg3 (ReTroGrade regulation) mediate glucose repression and signaling from the mitochondria to the nucleus, respectively. We will present a novel function of these proteins, wherein alanine promotes the formation of a chimeric Nrg1-Rtg3 regulator. The neo-functionalization of Nrg1 enables it to interact with Rtg3, leading to significant biological implications. Both the nrg1Δ and rtg3Δ single mutant strains exhibited an inability to utilize ethanol as a carbon source and displayed a petite phenotype on glucose media, indicative of mitochondrial dysfunction. Notably, neither wild-type gene was able to complement the petite phenotype in these mutants, suggesting irreversible damage to mitochondrial DNA. Direct measurements of mitochondrial DNA gene complements confirmed this irreversible damage in both mutant strains, underscoring the essential role of the chimeric Nrg1/Rtg3 regulator in maintaining mitochondrial DNA integrity. The findings suggest that the interaction between Nrg1 and Rtg3 is critical for cellular responses to metabolic changes, particularly under conditions where alanine is present. This study was funded by the Dirección General de Asuntos del Personal Académico, UNAM (DGAPA-PAPIIT, GRANTS: IN202521 and IN207424),Consejo Nacional de Humanidades Ciencias y Tecnologías (CONAHCyT, grant:101729)

Ultrastructure of the nucleolus of *Taxodium mucronatum*

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The eukaryotic cell nucleus has several membrane-less bodies with different functions. The nucleolus is the most prominent and dynamic nuclear body where the transcription and maturation of preribosomal RNAs occurs. Also the nucleolus is related with other cellular functions. Although it is composed of three different ultrastructural elements, there is evidence that differences are present between animals and

angiosperm plants, such as the presence of heterogeneous fibrillar centers. Therefore, it is essential to understand how these characteristics differ among species. Here we focused on the gymnosperm *Taxodium mucronatum*, well known as Ahuehuete, the Mexican National tree which has an enormous value to national history. There is no data about the nucleolar organization of this modern gymnosperm. We prepared samples of leaf meristems for transmission electron microscopy and used cytochemical techniques including EDTA regressive contrast and AgNOR staining for nucleolar organizers. Our results showed in most cases, a central, spheroid and compact nuclear body displaying fibrillar and granular elements, as described in other eukaryotic species. This body is also discontinuous and positive to EDTA regressive procedure and also to silver staining. Furthermore, we also observed a smaller body associated. We concluded that *T. mucronatum* presents a compact, fibrogranular nucleolus and Cajal bodies associated with it. This nucleolus is a tripartite structure since it shows fibrillar centers, dense fibrillar and granular components. It remains to be studied the process of nucleologenesis in *T. mucronatum*.

New applications in cellular immunology: Gut and respiratory microbiota and role of probiotics on human immunology

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New applications in cellular immunology: Gut and respiratory microbiota and role of probiotics on human immunology. Pedro Gutiérrez Castrellón MD; PhD. International Scientific Council for Probiotics. Mexico City. Mexico There are approximately 100 trillion symbiotic microorganisms in the human body, which are ten times larger than human cells and mainly located in the gastrointestinal tract, known as the gut microbiota. (1) They play an important role in maintaining human health, such as promoting mucosal development, modulate immunity and increase resistance to intestinal infections, and maintaining intestinal barrier function. (2-4) Additionally, beyond local effects, the gut microbiota also has systemic impacts outside the gastrointestinal tract, producing short-chain fatty acids (SCFAs) and other metabolites that play significant anti-inflammatory roles. (5-7) Recent studies have highlighted the gut microbiota's impact on distant organs like brain, lungs, and liver, leading to concepts such as the gut-brain, gut-lung, and gut-liver axis. (8-10) Probiotics are live microorganisms, which when administered in proper amounts, confer a health beneft on the host. (11) Among different probiotics, Lactobacillus and Bifdobacterium have been studied enormously and shown to confer significant immune health benefits to multiple hosts with distinct diseases. (12-14) From in vitro and in vivo to clinical studies, utilized probiotics have demonstrated the different effects on activation mechanisms of the immune system, ranging from eliciting B cells, T cells, and DC cells activation to mucosal and system immune response regulation. (15) In order to function properly in the host, probiotics should first survive and be recognized by individual host systems. For this, probiotics should become recognized in the gut lumen through various pathways. At first, probiotics are engulfed by either macrophages (Mfs) or dendritic cells (DCs) present immediately below M cells. Then, probiotics or their components directly or by assisting DC cells are recognized through interaction with intestinal epithelial cells. This is followed by triggering the secretion of an array of cytokines that further impact the immune functions of DCs, T cells, and B cells in the gut-associated lymphoid tissue (GALT). (16,17) In addition, activation of the innate immune response has been another confirmed probiotic impact on immune compartments. Since probiotic contains a conserved microbial-associated molecular pattern (MAMP) such as peptidoglycan (PGN) and cell wall polysaccharides (CPs), they seem to modulate and activate innate immune response through different pattern recognition receptors (PRR). One of the well-studied PRRs is toll-like receptors (TLRs), performing a critical role in linking innate and adaptive immune system arms. Upon interaction of probiotics and their components with different TLRs, many multiples signaling pathways are conveyed to cells by transmembrane (TM) protein and induce immune responses in receptor-expressing cells, leading to transcriptional pathways regulation, such as MAPK and NF-κB, against pathogenic and non-pathogenic diseases. (18,19) In 2014 we conducted a randomized controlled trial (RCT) to evaluate

whether daily administration of *Limosilactobacillus reuteri* (*L. reuteri*) DSM 17938 reduces the frequency and duration of gastrointestinal (GIs) and respiratory infections (RTIs) and other health outcomes in day school children in Mexico. Healthy children (born at term, aged 6–36 months) attending day care centers were enrolled in this randomized, double-blind, placebo-controlled trial. They received *L. reuteri* DSM 17938 (dose 108 colony-forming unit; n = 168) or identical placebo (n = 168) by mouth, daily for 3 months, after which they were followed-up after a further 3 months without supplementation. The number of days with GIs per child was reduced by *L. reuteri* supplementation from 0.96 (0.2) to 0.32 (0.1), p = .03 during the intervention period (0–12 weeks) and from 1.1 (0.1) to 0.5 (0.2), p = .01 during follow-up (12–24 weeks). Significant reductions in the number of episodes and the duration of GIs by *L. reuteri* supplementation were also observed during the intervention and follow-up periods. The number of days with RTI per child was reduced by *L. reuteri* supplementation from 4.6 (1.8) to 1.5 (0.6), p = .01 during the intervention period and from 4.4 (1.1) to 2.1 (0.8), P = .01 during follow-up. Significant reductions in episodes and duration of RTI by *L. reuteri* supplementation were also observed in both periods. (20) In 2021 we conducted a second RCT aimed to evaluate the safety and efficacy of *Limosilactobacillus reuteri* ATCC PTA 5289 and DSM 17938 to reduce the duration and severity of ARI symptoms. This randomized controlled trial included children aged from 6 months to 5 years, with pharyngitis or tonsillitis, who were randomised to receive a probiotic product containing *L. reuteri* ATCC PTA 5289 and *L. reuteri* DSM 17938 or placebo, as drops, ingested orally for 10 days as adjuvants to the use of non-steroidal anti-inflammatory drugs. The main outcomes were the duration and severity of ARI symptoms. The secondary outcomes were changes in salivary immunoglobulin A and inflammatory biomarkers. There was no fever on day 2 and subsequent days in the *L. reuteri* group (37.3 ±0.5 °C vs 38.6±0.3 °C, P<0.05). Beginning on day 3, the severity of sore throat (5±0.9 vs 8±1.2, P<0.05) was lower in the *L. reuteri* group. Significant differences in the days with runny nose, nasal congestion, days of non-programmed visits to the medical office or emergency department, levels in tumoral necrosis factor-alpha (TNF-alpha) and related costs of treatment were observed in the *L. reuteri* group. The frequency of adverse events was similar between the groups. (21) In 2023 we conducted a single-center, quadruple-blinded, randomized trial in adult symptomatic Coronavirus Disease 2019 (Covid19) outpatients. Subjects were allocated 1:1 to probiotic formula (strains *Lactiplantibacillus plantarum* KABP022, KABP023, and KAPB033, plus strain *Pediococcus acidilactici* KABP021, totaling 2 × 109 colony-forming units (CFU)) or placebo, for 30 days. Co-primary endpoints included: i) proportion of patients in complete symptomatic and viral remission; ii) proportion progressing to moderate or severe disease with hospitalization, or death; and iii) days on Intensive Care Unit (ICU). Three hundred subjects were randomized (median age 37.0 years [range 18 to 60], 161 [53.7%] women, 126 [42.0%] having known metabolic risk factors), and 293 completed the study (97.7%). Complete remission was achieved by 78 of 147 (53.1%) in probiotic group compared to 41 of 146 (28.1%) in placebo (RR: 1.89 [95 CI 1.40-2.55]; P < .001), significant after multiplicity correction. No hospitalizations or deaths occurred during the study, precluding the assessment of remaining co-primary outcomes. Probiotic supplementation was well-tolerated and reduced nasopharyngeal viral load, lung infiltrates and duration of both digestive and non-digestive symptoms, compared to placebo. No significant compositional changes were detected in fecal microbiota between probiotic and placebo, but probiotic supplementation significantly increased specific IgM and IgG against Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV2) compared to placebo. It is thus hypothesized this probiotic primarily acts by interacting with the host's immune system rather than changing colonic microbiota composition. (22) References 1. Alvarez J., Fernandez R.J., Guarner F., et al. Gut microbes and health. Gastroenterol. Hepatol. 2021;44(7):519–535 2. Groves H.T., Higham S.L., Moffatt M.F., et al. Respiratory viral infection alters the gut microbiota by inducing inappetence. mBio. 2020;11(1) 3. Sekirov I., Russell S.L., Antunes L.C., et al. Gut microbiota in health and disease. Physiol. Rev. 2010;90(3):859–904 4. Hagan T., Cortese M., Rouphael N., et al. Antibiotics-driven gut microbiome perturbation alters immunity to vaccines in humans. Cell. 2019;178(6):1313–1328 5. Avery E.G., Bartolomaeus H., Maifeld A., et al. The gut microbiome in hypertension: recent advances and future perspectives. Circ. Res. 2021;128(7):934–950 6. Campbell C., Mckenney P.T.,

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Mutational analysis of genes related to the antibiotic resistance in different Helicobacter pylori strains

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Helicobacter pylori is a Gram-negative bacterium with a 50% worldwide prevalence. This bacterium can reach the stomach, colonize the gastric mucosa and epithelium, and cause gastric diseases such as chronic gastritis, peptic ulcer, and gastric cancer. However, it has recently been linked to extragastric diseases. In 2018, the WHO considered *H. pylori* as a pathogen of global relevance due to its high prevalence and increase in the rate of antimicrobial resistance (AMR). Eventually, the AMR could intensify the H. pylori virulence and enhance the incidence of conditions related to this infection. This

work aimed to induce resistance in different strains of *H. pylori* against traditional antibiotics and analyze the presence of genetic mutations related to AMR. *H. pylori* strains 51932 and 43504 were treated with 3 antibiotics: clarithromycin (CLR), amoxicillin (AMX), and levofloxacin (LVX) at different sub-minimum inhibitory concentrations (MIC). The MICs were evaluated by the agar dilution method, and the obtained values were 15-62, 31-62, and 15-62 ng/mL for CLR, AMX, and LVX, respectively. AMR induction was produced by treating the two bacterial strains with increasing concentrations of the antibiotics for 3 cycles. However, both strains did not increase their MICs to be considered resistant to these antibiotics. The AMR-related genes 23s, pbp1a, gyrA, and gyrB were then identified. By PCR assays and Sanger sequencing, it was determined that only the gyrB gene presented mutations. Furthermore, bacterial treatment with AMX and LVX induced the conversion of its common bacillary morphology to a coccoid form, which is another known mechanism of antimicrobial resistance. The findings of this work indicated that the interaction of *H. pylori* with antibiotics induces mutations in genes related to AMR in a short period, adding the change to coccoid morphology, which provides a mechanism to evade the damage caused by these drugs. It remains to be determined whether these strains present differences in their virulence in various human tissues during persistent infections. Acknowledgements. I would like to express my gratitude to Cinvestav for all the support provided during the master's degree and the preparation of this work.

Mitochondrial Calcium Homeostasis and Fusion Dynamics

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Calcium oscillations control mitochondrial motility along the microtubules and in turn, support on-demand distribution of mitochondria. Calcium-dependent regulation of mitochondrial movements is mediated by the Miro-Milton complex that links mitochondria to kinesin and dynein motors. Both mitochondrial motility and fusion-fission dynamics seem to be sensitive to a Ca(2+)-dependent switch by this complex. Calcium also affects mitochondrial fusion-fission by several mechanisms. A fraction of calcium during calcium oscillations is accumulated by the mitochondria impacting the calcium regulation of both cytoplasmic and intramitochondrial targets. In this talk, some novel mechanisms of the interaction of calcium with the components of the mitochondrial dynamics will be described.

Analysis of TFIID Transcriptional Complex Components and Their Contribution to Heat Shock Response in Breast Cancer Cells

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Breast cancer is the leading cause of death from malignant tumors in women worldwide. An altered transcriptional program characterizes cancer cells. In eukaryotes, protein-coding genes are transcribed by RNA polymerase II and several general transcription factors, including TFIID. TFIID is a multisubunit complex involved in promoter recognition and the transcriptional preinitiation complex assembly. In humans, this transcriptional factor comprises TBP, and thirteen TBP-associated factors called TAFs. Recent studies have shown the existence of alternative TFIID complexes active in human embryonic cells, lymphocytes, and male germ cells. Some subunits of TFIID are not observed in these non-canonical complexes, or their isoforms replace these. Differences in TFIID subunit expression levels have been observed in breast cancer cells, suggesting the existence of non-canonical TFIID complexes in this disease as well as the cytoplasmic overexpression of the major subunit of TFIID (TAF1) under heat shock conditions, suggesting a role for the complex in stress response conditions. Nevertheless, the role of this transcription factor has not been determined in breast cancer cells, nor the implications it could

have in the heat shock stress response. Therefore, in this work, we will determine whether TAF1 plays a role in breast cancer cells' heat shock stress response.

Marchantia polymorpha GOLDEN2-LIKE transcriptional factor is a key protein for chloroplast differentiation and plant vegetative development

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Plastids support plant growth and development by providing carbon sources and essential compounds. The GOLDEN2-LIKE (GLK) transcription factors promote chloroplast biogenesis and the expression of photosynthesis-related nuclear genes in response to chloroplast status and environmental cues; acting as central regulatory nodes involved in both development and environmental responses. In this study, we examined the function of the single GLK representative of *Marchantia polymorpha* (MpGLK). Decreasing MpGLK levels impairs chloroplast differentiation and alters the expression of photosynthesis-associated nuclear genes. Conversely, overexpressing MpGLK results in ectopic chloroplast biogenesis. These findings demonstrate that MpGLK is a bona fide GLK protein, despite containing an additional domain in its structure, compared to Angiosperms. Furthermore, we show that altering MpGLK levels leads to diverse defects in Marchantia polymorpha development, particularly in dorsal structures such as air pores and gemma cups. Transcriptomic profiling and chromatin immunoprecipitation sequencing demonstrate that MpGLK is a direct activator of the MpMAX2 gene and thereby regulates gemma cup development through the KARRIKIN-INSENSITIVE2 receptor-mediated pathway. We propose that MpGLK functions as a master regulator, potentially coupling chloroplast development with vegetative reproduction.

Biomaterials for Tissue Engineering

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Biomaterials play a pivotal role in tissue engineering as scaffolds to provide structural support and influencing cell behaviour. Biomaterials, depending on the type (metals, polymers, ceramics, or composites) offer different strength, durability, flexibility and biodegradability. Composites combine the benefits of different materials, enhancing mechanical properties and biodegradability. In cardiac tissue engineering, biomaterials must replicate the mechanical and biochemical properties of native cardiac tissue. Hydrogels, decellularised matrices, and synthetic polymers support cardiomyocyte growth and function. Recent innovations include conductive biomaterials that enhance electrical signal propagation, crucial for synchronized heart contractions. Additionally, 3D bioprinting techniques enable precise fabrication of cardiac tissue constructs, offering promising avenues for creating functional heart tissue. Bone tissue engineering focuses on repairing and regenerating bone defects using biomaterials for bone engineering to foster osteogenesis. Bioceramics and self-assembly hydrogels support bone cell attachment and growth. Composite materials combining bioceramics with polymers enhance mechanical properties and biodegradability. Innovations in scaffold design, such as porous structures and bioactive coatings, further improve the integration and functionality of engineered bone tissue. The interdisciplinary nature of tissue engineering, integrating principles from biology, materials science, and engineering is underpinned by the advances in biomaterials, driving progress in cardiac and bone tissue engineering, offering hope for effective treatments and improved patient outcomes. Ongoing research and collaboration are essential to overcome current challenges and translate these innovations into clinical applications.

PNC, nucleoli, and anti-cancer therapeutics

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Cancer metastasis remains a leading cause of cancer mortality due to the lack of specific inhibitors against this complex process. To identify compounds selectively targeting the metastatic state, we used the perinuclear compartment (PNC), a complex nuclear body at the periphery of nucleoli that associated with metastatic behaviors of cancer cells, as a phenotypic marker for the development of selective anti-metastatic drugs. A high-content screen of over 140,000 structurally diverse compounds searching for those removed PNCs without cytotoxic properties yielded several candidates. Metarrestin, resulting from optimization of a screening hit, disassembles PNCs in multiple cancer cell lines, inhibits invasion in vitro, blocks metastatic development in several mouse models of human cancer, and extends survival of mice in a metastatic pancreatic cancer xenograft model with no organ toxicity or discernable adverse effects. Metarrestin disrupts the nucleolar structure and inhibits RNA polymerase (Pol) I transcription, in part by interacting with the translation elongation factor eEF1A2. Altogether, metarrestin represents an alternative therapeutic approach for the treatment of metastatic cancer.

Leeuwenhoek and the microscopy tradition in Mexico: novel approaches and discoveries in the history of microscopy in the Americas for innovation of cell biology education

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The key distinction between scientific education and technical training is the cultivation of critical thinking through an understanding of the genesis of scientific theories, the logic of experimental design, and the significance of scientific discovery within the cultural context. It is imperative that cell biology education adopt a comprehensive approach to scientific and cultural encyclopedism, particularly in light of the pivotal role played by the cell theory, which, alongside evolutionary theory, constitutes the most fundamental and axial corollary of past and current biology. It is proposed that cell biology education and outreach can benefit from the incorporation of novel historical narratives, which may be described as 'scientific heritage perspectives'. For life scientists to gain an understanding of the cultural and epistemological value of cell theory and discovery, it is necessary for them to engage with such narratives. The latter is based on the open science international agenda and posits that the history of science should not be constrained to the Eurocentric view of science. Instead, local narratives of discovery and circulation of ideas can be embraced as heritage in the Global South and put forward to inspire scientific vocations, critical thinking and novel trends in cell biology. These can be researched based on observable evidence. Considering this we propose that the rediscovery of an original Leeuwenhoek book at the National Library of Mexico can enhance cell biology education by establishing a robust narrative of microscopic tradition in Mexico, which can be contextualized with reference to contemporary Mexican advances in cell biology, such as the discovery of a nucleolus in Archaea. This provides our community with a meaningful and profound context of transcendence of cell biology in our countries.

Automated, correlative lamella production with Arctis

Matt Joens, Thermo Fisher Scientific, USA.

From recent advances in automated lamella production to correlative imaging techniques to machine learning-powered analysis, cryo electron tomography (cryoET) has evolved into a robust technology that is enabling researchers to probe the mysteries within cells like never before. Here we will explore the latest innovations in automated, high-quality lamella production using a fully automated plasma FIB with integrated fluorescence targeting. Come see how the Arctis is redefining what is possible with a lamella production tool.

Cellular machinery revealed by cryo electron tomography and cryo spin mill tomography

Matt Joens, Thermo Fisher Scientific, USA.

Conventional electron microscopy has long been the gold standard for resolving the nanoscale structures between organelles, however, the use of chemical fixatives and stains often begs the question of what is real or artifactual. Cryo preservation circumvents these issues, but sample preparation, imaging, and analysis have historically been cumbersome and time consuming - until now. Cryo-plasma FIBs have redefined what is possible by combining volume imaging techniques like cryo spin mill tomography with gentle and targeted sample preparation abilities to enable cryo-electron tomography at the TEM level. From probing the spatial relationship of cells and organelles at the tissue level to visualizing tethering proteins and organelle contact points, the next generation of tools combined with machine learning analysis is opening new doors for what is possible with cellular and structural biology.

Electron Microscopy and the Frontiers of Plant Research

Matt Joens, Thermo Fisher Scientific, USA.

Electron microscopy has revolutionized plant research due to its ability to cover large fields of view and high depth of field at resolutions often far greater than what is achievable with light microscopy. It has been used in almost all major areas of the plant sciences, from agricultural development to the characterization of pathogens. Over the years, these techniques have evolved; from determining high-resolution structures that reveal the molecular organization of macromolecules, to volume electron microscopy techniques that unlock fine details of ultrastructural organization in cells, tissues, and even whole organisms. Here we explore the latest innovations in volume imaging for plant biology, from recent advances in plasma milling and spin mill tomography to machine learning-powered analysis.

Beyond the classical pathway: Understanding unconventional protein secretion in plant cells

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For over 50 years, the conventional protein secretion (CPS) pathway has been the model for understanding how soluble secretory and membrane proteins reach their destinations. CPS involves proteins with a signal peptide (SP) recognized by the signal recognition particle (SRP) during translation. SRP directs the nascent protein-ribosome-mRNA complex to the endoplasmic reticulum (ER) lumen, where the SP is cleaved, and translation continues. Proteins then exit the ER in vesicles, travel through the Golgi apparatus, and reach the plasma membrane. Secretory proteins are released into the extracellular space, while integral membrane proteins remain in the plasma membrane. The identification of numerous SP-lacking secretory proteins suggests the occurrence of alternative pathways, termed unconventional protein secretion (UPS). Some UPS have been described, while others remain unclear. These pathways bypass at least one of the CPS elements, such as the ER and/or Golgi, to reach the extracellular space. Although more than 50% of the plant secretome comprises SP-lacking proteins, UPS research has mainly focused on yeast and animal cells. In studying self-incompatibility in Nicotiana alata, a type h thioredoxin (Trxh) –NaTrxh– was identified as crucial in self-pollen rejection. Trxh was originally categorized as a cytosolic group; however, NaTrxh is secreted into the extracellular space of the stylar transmitting tissue in N. alata. NaTrxh belongs to Trxh subgroup 2, featuring N- and C-terminal extensions. Its N-terminal extension includes two motifs: Na and Nb. The Nb motif, despite its hydrophilic profile and atypical position –unlike typical SPs– functions as an SP. Cellular evidence from NaTrxh-GFP and Nb-GFP fusion proteins shows that NaTrxh secretion occurs via CPS. However, the Nb motif reveals an SRP-independent and post-translational, rather than co-translational, ER translocation. This suggests a “semi-CPS” pathway, combining CPS and UPS elements, where NaTrxh might serve as a model for exploring this novel secretory pathway in plants. Analysis the Nb motif has also revealed that variations might lead to different trafficking roles among Trxh, providing further insight into the complexity and specialization of plant thioredoxins. Grant: PAPIIT IN207823.

Chromatin-associated lncRNA-splicing factor condensates regulate hypoxia responsive RNA processing of genes pre-positioned near nuclear speckles

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Hypoxia-induced differential mRNA processing contributes to tumor progression and metastasis. Presently, little is known about how hypoxia-responsive alternative splicing (AS) is achieved, and whether higher order genome and nuclear domain organization dictate these processes. We observe that hypoxia-responsive genes pre-position at nuclear speckle (NS) neighborhood as “hubs”. MALAT1 long noncoding RNA regulates hypoxia responsive AS of genes located at NS proximity. Hypoxia-induced MALAT1 spatially organizes splicing factor SRSF1 in the NS neighborhood and specifies the binding of SRSF1 with its substrate RNAs. MALAT1 by nucleating SRSF1 condensates near NS-associated genes, achieves the critical local concentration, required for efficient interaction between SRSF1 and elongating RNA polymerase II. Our studies reveal that during hypoxia, MALAT1 regulates spatially organized AS, by establishing the threshold concentration of SRSF1 at NS proximity, which is critical for its interactions with RNA polymerase II, splicing partners and substrate pre-mRNAs.

Triggering nuclear body formation with extracellular signals

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Macrophages are key phagocytic and signaling cells in the innate immune system, contributing to the first line of defense against pathogens. Macrophages differentiate into distinct active states upon treatment with different stimuli. I will present data about nuclear bodies known as paraspeckles (PSs) that frequently form during pro-inflammatory macrophage differentiation driven by bacterial lipopolysaccharide, but not during anti-inflammatory (“M2”) differentiation driven by interleukin-4. PS RNA-binding proteins and the scaffold lncRNA Neat1_2 colocalize in differentiated macrophages, and chemical inhibition of distinct kinases strongly reduces PS formation during inflammatory macrophage differentiation, suggesting a role for PS protein phosphorylation during differentiation.

Studying dynamic cell interactions with Celldetective

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A current bioimaging challenge in immunology and immunotherapy research is the analysis of multimodal and multidimensional data recording of dynamic interactions between diverse cell populations. We developed Celldetective, a software that integrates AI-based cell segmentation and tracking algorithms as well as automated signal analysis into a user-friendly graphical interface. It offers complete interactive visualization, annotation, and training capabilities. We present original experimental data of spreading immune effector cells on activating surfaces imaged by interferential microscopy. Harnessing Celldetective, we extract a detailed chronological description of immune cell interactions and decisions within large populations. We also showcase antibody-dependent cell cytotoxicity events using multimodal fluorescence microscopy, with the aim of characterizing new potential anticancerous agents.

Precise Control of Microtubule Structure and Intracellular Trafficking in Living Cells and Behaving Animals

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Microtubules play a crucial role in regulating various cellular activities, including intracellular transport, mitosis, and cell migration. Dysfunctions in microtubules are associated with diseases such as neurodegenerative disorders, cancer, and aging. The diversity of microtubule subtypes, defined by

tubulin isotypes and post-translational modifications (PTMs), adds complexity to their cellular functions. Furthermore, microtubules form structures such as primary cilia, centrosomes, and mitotic spindles to fulfill specific functions. A major challenge in the field is understanding how these subtypes regulate cellular activities in a spatiotemporal manner, a task complicated by technical limitations in precisely manipulating them. Our team has developed tools to control tubulin PTMs and microtubule disassembly with precision (Hong et al., Nat. Commun., 2018; Liu et al., EMBO J., 2022) and to perturb intracellular trafficking along specific subtypes in a spatiotemporal manner (Chen et al., Advanced Science, Accepted). These methods enable rapid and reversible manipulation of microtubule properties and functions, providing new insights into their underlying mechanisms. Our research has uncovered how microtubules and intracellular trafficking influence cellular architectures and activities. At the organismal level, we have demonstrated that controlling microtubule disassembly and synaptic vesicle transportation can reversibly affect locomotion in C. elegans and Drosophila. Furthermore, we have shown potential therapeutic applications, such as specifically inhibiting ACE2-mediated viral entry without disrupting other endocytic pathways. These innovative tools open new avenues for understanding how different microtubule subtypes and intracellular trafficking regulate cellular architecture and function across various physiological and pathological conditions.

Roles of Cardiolipin Conversion in Mitochondrial Dynamics and Quality Control

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Mitochondria are the powerhouse of eukaryotic cells, responding and adapting to various stressors and metabolic demands through their dynamic nature. Mitochondrial dynamics rely on the coordinated regulation of proteins and phospholipids to control the processed of fission, fusion and transport. Cardiolipin, a unique phospholipid enriched in the inner mitochondrial membrane, promotes inner membrane fusion but facilitates outer membrane fission when externalized upon mitochondrial stress. Interestingly, cardiolipin can be hydrolyzed by phospholipase D 6 (PLD6) into phosphatidic acid (PA), promoting mitochondrial fusion; however, the exact mechanism by which cardiolipin conversion drives mitochondrial fusion remain unclear. In this work, we discover nucleotide diphosphate kinase NME3 as a key player in PLD6-mediated mitochondrial fusion. NME3 directly binds to PA via its N-terminal amphipathic helix and is enriched at the contact sites between closely positioned mitochondria in a PLD6-dependent manner. The ability of NME3 to bind PA and form hexamers enables its mitochondrial tethering activity. Notably, nutrient starvation enhances NME3 enrichment at mitochondrial contact sites, and its ability to promote fusion is critical for mitochondrial fusion and cell survival under starvation. Our findings reveal a mechanism by which cardiolipin conversion selectively drives mitochondrial fusion and contributes to mitochondrial quality control.

Cryo-EM and Cryo-ET in visualizing host interaction and genome ejection mechanism of mycobateriophage

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Recent reports have highlighted the use of engineered siphophages to treat antibiotic-resistant mycobacterial infections, revitalizing interest in phage therapy. However, the current phage engineering approach relies on random mutations, primarily due to the lack of comprehensive structural information of siphoviridae, which feature a long non-contractile tail. Herein, we elucidate the cryo-EM structure of the siphophage which consists of 1105 proteins at resolutions ranging from 2.18 to 4.0 Å. Our atomic model reveals that the assembly of this 400 nm-long, pin-shaped phage only requires thirteen symmetric protein building blocks conjoined by various symmetry-mismatched interfaces. While receptor-binding proteins fully cover the phage surface for host recognition, our cryoET image shows two type of phage-host orientation, indicating the flexible tail tube for increased infectivity. The structure also reveals a

unique way of the tape measurement protein (TMP) for DNA gating mechanism while the phage tail tube and neck structures remain unchanged before and after DNA ejection. The DNA passthrough channel is sealed mostly by trimeric TMPs, preventing DNA release. The host interaction opens the baseplate hub and triggers TMP release, leading to the unsealing of the dodecameric portal ring for DNA ejection. In conclusion, our atomic structure of siphophage and high-resolution images of phage-host interaction not only provide insights into how the siphophage infects the host but also provide a structural basis for future mechanistic studies and phage engineering.

A plant stress protein: structural flexibility and cellular organization under water deficit

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A plant stress protein: structural flexibility and cellular organization underwater deficit Coral Martínez-Martínez1, Teresa Nava-Ramírez1, Marisa S. Otegui2 and Alejandra A. Covarrubias1* 1Plant Molecular Biology Department, Instituto de Biotecnología, Universidad Nacional Autónoma de México; 2Department of Botany, University of Wisconsin-Madison. During development, orthodox seeds undergo a unique event characterized by substantial water loss, eventually reaching a desiccated state. Despite this extreme dehydration, the process is essential for seed germination. At the onset of dehydration, in the late stages of embryogenesis, these seeds accumulate a group of proteins known as Late Embryogenesis Abundant (LEA) proteins. These proteins also accumulate in response to water deficit in both vegetative and reproductive tissues of plants. Due to their amino acid composition and physicochemical properties, LEA proteins are classified as Intrinsically Disordered Proteins (IDPs). LEA proteins, like other IDPs, display remarkably structural flexibility, allowing them to interact with a wide variety of molecular components in their environment. This structural adaptability facilitates dynamic interactions with multiple molecular partners, influencing both the nature and organization of their intra- and inter-molecular associations. In this study, we focused on the AtLEA4-5 protein, a member of the LEA4 protein family from Arabidopsis thaliana. LEA4 proteins are found throughout the plant kingdom, including angiosperms, gymnosperms, and bryophytes. These proteins share a conserved N-terminal region with a high propensity to form alpha-helical structures, while their C-terminal region is more variable and disordered. AtLEA4-5 is highly abundant in dry seeds and is also present in vegetative tissues subjected to dehydration. Arabidopsis mutants lacking members of these family show reduced tolerance to water deficit conditions. To investigate the function of this protein in planta, we used AtLEA4-5-GFP fusion to study its tissue and intracellular localization in seeds, during early developmental stages after germination, and in response to water limitation. Our results indicate that the AtLEA4-5 protein is present in all tissues of imbibed seeds and this localization persists during the first few days after germination under non-stressful conditions. In seedlings exposed to water deficit, the protein localizes to tissues more affected by the stress. At subcellular level, AtLEA4-5 is found in both the nucleus and the cytosol. Notably, in imbibed seeds, AtLEA4-5 not only abundant but also forms large aggregates that behave as biomolecular condensates. Similar condensate formation was observed in vegetative tissues under water deficit. Furthermore, in the cytosol, the protein exists in at least two distinct forms: as condensates and in a more dynamic, non-aggregated state. The relationship between the cellular organization of this protein and its functional role will be discussed.

Cysteine protease CP A4: Study of its participation as autophagin in *Trichomonas vaginalis* autophagy

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In our working group we studied the protozoan parasite *Trichomonas vaginalis* (*T. vaginalis*), a causal agent of trichomoniasis, one of the four most prevalent sexually transmitted infections worldwide. In

women with trichomoniasis, T. vaginalis is exposed to different glucose concentrations (0.3 -36.65 mM), one of the main carbon and energy source. Under metabolic stress by glucose restriction, *T. vaginalis* activates several adaptive mechanisms, such as metabolic reprogramming, improved antioxidant capacity, and autophagy, for cellular homeostasis that promotes cell survival. In T. vaginalis two autophagy markers, TvAtg8a and TvAtg8b have been identified. For the formation of autophagosomes, processing of TvAtg8a or TvAtg8b should be necessary and must be carried out by a cysteine protease ATG4-like, belonging to the C54 family or autophagin. In *T. vaginalis* five genomic sequences coding for proteases of the C54 family are found in the genome database. At the transcript level under glucose restriction (GR) conditions a higher expression of CP A1 followed by CP A4 has been reported, while at the protein level proteomes only showed expression of CP A4 under normal iron conditions. Thus, our goal in this work was study one of the five trichomonad autophagin, CP A4. Our approach was to express and purify recombinant CP A4 to generate polyclonal antibodies and used in WB and IFI assays to determine its amount and localization in trichomonads, as well its *in vitro* proteolytic activity, respectively. We also checked its expression by qRT-PCR under different glucose conditions. Our results showed that cp a4 gene has higher expression in GR than under HG conditions. At the protein level, no significant differences were observed under different glucose conditions. However, at the localization level, under GR conditions more protein was observed in the Golgi apparatus and cytoplasm than under HG conditions, whereas under HG conditions more vesicles were observed than under GR conditions that corresponded to autophagic vesicles, hydrogenosomes, as well as in the Golgi complex and endoplasmic reticulum. These data suggest that CP A4 also participates in the first step of Atg8 processing during *T. vaginalis* autophagy.

Role of Gp32 protein in the induction of H66 prophage to the lytic cycle

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Pseudomonas aeruginosa is a gram-negative bacterium and the leading cause of morbidity and mortality in patients with cystic fibrosis, whose abnormal airway epithelium allows long-term colonization of the lungs. The genomic plasticity observed in this, and other species can be attributed to horizontal gene transfer events, including those mediated by bacteriophages. As a temperate phage H66 can undergo two alternative developmental pathways: the lytic or lysogenic cycle. The lysogenic state is reversible and therefore the lytic pathway can resume upon activation of the host SOS response, a phenomenon termed prophage induction. In the genome of phage H66, a Hollowayvirus isolated from a clinical strain of *P. aeruginosa*, homologous elements of the bacterial SOS response have been identified and characterized including a homolog of the LexA repressor (Gp14) and LexA recognition sites (SOS boxes) overlapping phage gene promoters. These boxes seem to regulate the expression of the ORF32 and ORF70 genes, whose function is unknown. In this regard, it has been demonstrated the change in the expression of ORFs 32 and 70 of H66 in response to damage by MMC, resembling the expression pattern of bacterial SOS genes. Additionally, Gp32 is located adjacent and opposite the lysogeny repressor gene (gp33). For all the above, the present work aims to experimentally analyze the function of the Gp32 protein in the induction of the lysis cycle of the prophage H66. To determine the function of Gp32, molecular cloning of ORF32 into an inducible expression vector was performed. Furthermore, we intend to evaluate the MMC-dependent induction capacity by generating a mutant phage lacking the Gp32 gene using homologous recombination and Cas13a-based counterselection. The above strategies aim to gain insights into the role of Gp32 on the induction of H66 virion formation coupled to SOS response.

Mitochondrial Dysfunction at the Crossroad of Aging and Lung Fibrosis

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Idiopathic Pulmonary Fibrosis (IPF) is a lethal chronic age-related lung disease characterized by progressive scarring of the lung. Age-related perturbations are increasingly found in epithelial cells and fibroblasts from IPF lungs and are believed to play a critical role in the predisposition to lung injury, disrepair, and fibrosis. Our studies show that mitochondrial dysfunction and metabolic distress potentiate senescence, aberrant lung repair, and susceptibility to lung fibrosis. Elucidation of the mechanisms that regulate the mitochondrial and metabolic adaptations to aging, injury and repair might help to define potential therapeutic approaches that target aging processes and might be beneficial for halting the progression of the fibrotic disease.

The Role of SBiP1 in Protein Synthesis and Nitrogen Metabolism in *Symbiodinium microadriaticum*

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Coral reef ecosystems depend heavily on the mutualistic relationship between cnidarians and photosynthetic algae from the Symbiodiniaceae family. *Symbiodinium microadriaticum* contributes up to 95% of the host's energy requirements through photosynthesis, in exchange for shelter and inorganic nutrients. However, the molecular pathways involved in how S. microadriaticum responds to environmental cues like light and nutrient availability are still not fully understood, particularly in relation to protein synthesis and nitrogen metabolism. In my research, a yeast two-hybrid screen revealed eight potential ligands interacting with the light-regulated chaperone protein SBiP1 (from the HSP70 family) in *S. microadriaticum*. SBiP1 phosphorylation is influenced by light, as well as by exposure to inhibitors such as cycloheximide (which blocks mRNA translation) and glufosinate-ammonium (which inhibits nitrogen metabolism). These findings suggest that SBiP1 may have a dual role in light responsiveness and nitrogen metabolism. Several of SBiP1's identified ligands are directly involved in protein synthesis, including HSP70 (a chaperone), MAP2 (an aminopeptidase), and EFL1-α (which facilitates tRNA transfer to ribosomes). Another ligand, POX18, a peroxisomal multifunctional enzyme involved in lipid biosynthesis, shows differential expression depending on nitrogen levels, further linking SBiP1 to metabolic regulation. In this lecture, I will present data on SBiP1 gene expression under conditions of nitrogen stress, exploring how SBiP1's interactions suggest its involvement in nascent protein synthesis, highlighting the essential role of nitrogen in this process. Keywords: coral symbiosis, nitrogen metabolism, gene expression, chaperone proteins

Unlocking Symbiosis: How SnRK1 Regulates *P. vulgaris* Partnerships

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Sucrose nonfermenting 1 (SNF1)-related protein kinase (SnRK1), along with its homologues AMP-activated protein kinase (AMPK) in animals and SNF1 kinase in yeast, are conserved serine/threonine protein kinases that act as cellular energy sensors, promoting catabolism and inhibiting anabolic processes. SnRK1 is known to regulate both enzymatic activities and the expression of numerous genes, particularly in response to abiotic stress. However, its role in legume symbiosis is less understood. In this study, we overexpressed SnRK1 in the hairy root system of *Phaseolus vulgaris* and observed a significant increase in primary and lateral root growth and density, which also enhanced the aerial growth of the composite plants. Upon inoculation with nitrogen-fixing *Rhizobium tropici*, these plants exhibited a six-fold increase in nodule numbers, leading to higher nitrogen fixation without any anatomical differences in the nodules compared to controls. In contrast, SnRK1-overexpressing plants showed impaired arbuscule development during mycorrhization. RNA-Seq analysis of roots from both symbiotic conditions revealed distinct transcriptional responses: 35% of genes were upregulated and 6% downregulated in rhizobial symbiosis, while 23.5% were upregulated and 30% downregulated under mycorrhization. These findings highlight a crucial role for SnRK1 in the symbiotic associations of P. *vulgaris*.

Stress-Induced Biomolecular Condensates in the *C. elegans* Germline

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In certain conditions, diffused RNAs and proteins can form liquid droplets, like oil in water, to perform specific functions. This phenomenon is transient and is known as liquid-liquid phase separation (LLPS). Membrane-less organelles, or biomolecular condensates, such as RNA granules, are formed through LLPS. We are using the adult hermaphrodite *C. elegans* gonad as a model to study LLPS due to several advantages, including its transparency and the presence of germ cells that share cytoplasm, forming a syncytium. This allows for in vivo study of this phenomenon. The *C. elegans* gonad contains various types of RNA granules. Under control conditions, the germ cells in their cytoplasm exhibit germ granules, also known as P granules, which are important for establishing the germline, sorting mRNAs and processing piRNAs. Like cells in other organisms, germ cells also contain processing bodies, which are important for mRNA turnover and for temporarily storing mRNA. Under stress conditions, we and other groups have observed at least two different classes of condensates known as stress granules, which protect germ cells from harmful conditions. One type of stress granules is mainly located in the distal core of the gonad while the other kind is found in the most proximal part of the gonad where oocytes are forming. Germ granules have been studied with more detail in the nematode in contrast processing bodies and stress granules formation, function and composition are not well understood yet. We are studying how the formation of stress granules is regulated in the *C. elegans* gonad. We have observed that the RNA-binding proteins TIAR-1 and GLA-3 (homologs of TIA1/TIAR and TTP in mammals, respectively) are required for stress granule formation in the *C. elegans* gonad. We are currently studying how aging and exposure to certain drugs affect the formation of stress granules. We would like to thank Dr. Beatriz Aguilar Maldonado and M. En C. Laura Silvia Salinas Velázquez for her technical support in the realization of our projects. Our research is sponsored by PAPIIT-UNAM (IN199324) and the Cell Physiology Institute from UNAM.

Enteroaggregative *E. coli* Pic is a key factor for interacting with intestinal goblet cells

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A hallmark of enteroaggregative *Escherichia coli* (EAEC) infection is the formation of an intestinal biofilm, which comprises a mucus layer with immersed bacteria. Pic is an autotransporter secreted by EAEC, and other E. coli pathotypes, and has been involved in two apparently contradictory phenotypes, as a mucus secretagogue and as a mucinase. During my talk, I will show our investigation to understand this Pic dual activity, mucus secretagogue capability and mucinolytic activity, in human goblet cells that secrete MUC2 and MUC5AC. Pic induced mucus hypersecretion directly in the goblet cells, without other intestinal cell types involved. At the same time, Pic exhibited strong proteolytic activity on the secreted mucins. These activities were independent since a mutation in the serine protease motif (PicS258I) abolished mucin degradation while maintaining the mucus secretagogue activity intact. Furthermore, deoxycholic acid (DCA)-induced mucins were proteolytically degraded when goblet cells were co-incubated with DCA/Pic, while co-incubation with DCA/PicS258I induced a synergistic effect on mucus hypersecretion. Pic was more efficient degrading MUC5AC than MUC2, but no degradation was detected with Pic inactivated at the active site by mutation or pharmacological inhibition. Remarkably, Pic cleaved MUC2 and MUC5AC in the C-terminal domain, leaving N-terminal subproducts, impacting the feature of gel-forming mucins and allowing mucus layer penetration by EAEC. Astonishingly, Pic stimulated rapid mucin secretion in goblet-like cells by activating the intracellular calcium pathway resulting from the PLC signal transduction pathway, leading to the production of DAG and releasing IP3, a second messenger of calcium signaling. Therefore, the dual activity of Pic, as a mucus secretagogue and a mucinase, is relevant in the context of carbon source generation and mucus layer penetration, allowing EAEC to live within the layer of mucus but also access epithelial cells.

Identification of Integrins associated with metastasis in breast cancer cell extracellular vesicles by flow cytometry

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Extracellular vesicles (EV) are lipid bilayer-delimited micro and nano-sized particles that cannot replicate and are released by all cell types, with a significant role in cell-to-cell communication. There has been a growing interest in exosomes because these nano EVs have been proposed as tools with diagnostic and prognostic value in several pathologies. EV molecular cargo includes proteins, lipids, and nucleic acids. These biomolecules can be transferred horizontally between cells and contribute to microenvironment modulation. Molecular characterization of EVs can reflect cellular status associated with different pathological conditions and can also provide insights about potential effects on target cells. The role of EV in breast cancer (BCa) has been studied and is now considered a key factor in tumor progression and metastasis. Several molecules transferred by EV have been associated with this process. Such is the case of integrins, membrane receptors involved in cellular adhesion, extracellular cell matrix interactions and tumor-promoting signaling pathways. Integrin EV's are implicated in metastasis and organotropism. However, an extensive and individualized identification of integrins in BCa-derived EVs has not yet been established. Nano flow cytometry has recently been developed and emerges as a methodology that can contribute to provide a detailed phenotypic and functional characterization of exosomes and other EVs. The aim of this work was to identify the selective profile of integrins in EVs from BCa cells and to identify novel candidates as biomarkers for BCa progression. EVs were obtained from BCa spheroids, stained with several antibodies directed to integrins and analyzed by nano flow cytometry in a Cytoflex LX cytometer (Beckman Coulter). Preliminary results show a differential profile of integrin and tetraspanins in EV from different BCa cell lines, which correlated with a reported aggressive phenotype and metastatic potential. In this context, EVs from MDA-MB-231 cells exhibited an increased content of CD63 and β1 integrin than EVs from MCF7 cells. Furthermore, when comparing EVs from MDA-MB-231, MCF7, and MCF10A cells, the latter showed no detectable levels of β1 integrin, suggesting that β1 integrin may play a critical role in cancer. Additionally, differential expression of α3 integrin is observed among EVs from MDA-MB-231, MCF and MCF10A cells which has been associated with invasiveness and metastasis. These preliminary findings emphasize the potential of selective integrin profiles from EVs as biomarkers for BCa progression, highlighting their relevance for the design of new potential therapeutic tools for clinical intervention in cancer patients.

A Therapeutic Chagas Vaccine: more than a decade of a multidisciplinary effort

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Chagas disease, caused by the protozoan parasite *Trypanosoma cruzi*, is one of the most important neglected parasitic diseases. It has an annual incidence of 30,000 new cases and around 12,000 deaths, and it mainly affects marginated regions in Latin America. Due to the low efficacy of the available drug-based therapy, therapeutic vaccines based on recombinant antigens have been proposed as a novel complementary treatment for controlling *T. cruzi* infection. For more than a decade, a multidisciplinary effort by a consortium of public and private institutions had been working to develop a therapeutic vaccine based on recombinant antigens. In this talk, I will show the advances of this multidisciplinary effort in developing the technology to produce recombinant antigens, test them in animal models, and make it possible for one antigen to be produced at GMP conditions by a Mexican biotechnology company ready for clinical studies.

Tissular Biology and Tissue Engineering

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Tissue biology focuses on the study of tissues in living organisms, examining and analyzing their structure, function, and repair, covering everything from the tissue level to organs and systems. The analysis of tissues performed in tissue biology aids in understanding biological processes at these levels, providing the in-situ context where functions occur, which in turn offers a comprehensive view of the interactions established with components located in a tissue. These interrelations revealed by tissue biology allow us to understand how tissues and organs respond to stimuli, injuries, and diseases. Therefore, it is vital to know and understand the structural foundations of tissue and organ function.

In this sense, tissue biology, also known as histology, shows its relevance for the development of tissue engineering by demonstrating the arrangement and distribution of the extracellular matrix, innervation, and vascularization that exist among cells. Histology not only details the histological structure of various tissues but also deciphers the functioning of heterogeneous associations of different tissues and the high level of organization they achieve in whole animals in situ. Tissue engineering, on the other hand, combines principles from tissue biology, medicine, materials science, and engineering to develop biological substitutes that restore, maintain, or enhance the function of damaged tissues or organs. Tissue engineering is based on three essential components: cells, scaffolds or biomaterials, and growth factors. Tissue engineering has been a substantial tool in regenerative medicine, enabling the development of various organs such as artificial skin, cartilage, and even complete organs. Due to the relevance of tissue engineering in regenerative medicine, it is a priority to deepen the study of the histological structure of tissues in organisms, as this knowledge is fundamental for the design and manufacturing of scaffolds. In other words, the more complete and in-depth the knowledge of tissues, the closer we can get to imitating the native architecture of tissues. For example, if we consider the type of mechanical support provided by connective tissue, the porosity that facilitates the diffusion of nutrients, oxygen, and waste products that vascularization provides, as well as the tensegrity between various components, scaffold designs will increasingly resemble the structure found in an organism's tissues. If these qualities are met in scaffolds, emulating the conditions of a tissue, they promote the necessary tissue microenvironment for cell proliferation, migration, and differentiation. In conclusion, tissue biology is relevant because it emphasizes histological structure, providing a model to design scaffolds that efficiently support cell growth in a way that guides tissue elements to form functional tissues and facilitate their integration with surrounding tissues in the body. The imitation of three-dimensional structure, mechanical properties, and the ability to interact with biochemical signals are key factors for the success of scaffolds in tissue engineering.

SYCP3 yields accumulations in primary spermatocytes during the first spermatogenic wave in murine models (*Rattus norvegicus* and *Mus musculus*)

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Meiosis is a specialized kind of cell division in which reproductive cells rise to egg cells or sperm cells that are responsible for sexual reproduction. A key structure in this process is the Synaptonemal Complex (SC). The SC is a scaffolding structure that interacts with various proteins in a specific way to achieve its main function, this is, to keep homologous chromosomes attached during homologous recombination. This means, this union promotes the exchange of genetic material between homologous chromosomes known as crossing over. Despite this is the main role of SC, this structure is also involved in the correct disjunction of chromosomes during the first meiotic division. These functions of SC make it a conserved structure in evolution with a canonical structure that has several homolog proteins among

vertebrates, invertebrates, plants and yeast. This canonical structure consists of three main components; first, the lateral element (LE) which is formed by CS Protein 2 (SYCP2) and the SC Protein 3 (SYCP3). Second component is the Central Region which is formed by the space between the LE of each chromosome, this space is transversely crossed by structures called transverse filaments formed by SC Protein 1 (SYCP1). And the third component is the Central Element which is another filament structure composed by Central Element Proteins 1, 2 and 3 (SYCE1, SYCE2 and SYCE3) and the TEX12. The SC assemblage takes place during leptotene of meiotic prophase I, when SYCP2 and SYCP3 join to chromosome axes independently to start the recombination process, however, the assembly completes in pachytene stage which is also the stage when recombination takes places, this means that the assembly of the synaptonemal complex is crucial for meiosis and for sexual reproduction and therefore, SYCP3, as a major component of SC is also very important in this processes. Different studies have been carried out to understand the mechanisms of action and function of these proteins, which still set certain questions. To study SYCP3 during meiotic prophase I, we analyzed a prepubertal murine model. We observed performing primary mouse spermatocytes surface spreads, that SYCP3 forms accumulations at all stages of meiotic prophase I that show different morphologies at each stage of meiotic prophase I. We also found a high percentage of SYCP3 accumulations in primary mouse spermatocytes, that is higher in spermatocytes of 14-day mice, when zygotene stage spermatocytes are more abundant than any other stage spermatocyte. Interestingly, this accumulations don´t affect meiotic progress In the rat model, both spermatocytes in zygotene and pachytene exhibit the highest percentage of presence of SYCP3 accumulations. Western-blot and real-time PCR were performed for a better understanding of this protein. We found a peak amount of SYCP3 protein in spermatocytes of 27 days, this is, when late pachytene and diplotene of first spermatogenic wave are more abundant, but this specific age also includes a great abundance of zygotene spermatocytes form the second spermatogenic round. We also found a peak of sycp3 in spermatocytes of 20 days, despite, nor the peak of protein nor the peak of the gene were statistically significant, it clearly shows a particular behavior of the protein during the first spermatogenic wave. Also, an *in-silico* proposal of the structure of Rat SYCP3 was created using predictive bioinformatic tools. Interestingly, we found several similarities between our prediction of Rat SYCP3 and human SYCP3 whose structure has already been crystalized. In addition, ELISA was performed to measure the concentrations of hormones related to the spermatogenic process (FSH, LH and testosterone). These assays were significant only in the 14 days aged mouse, where testosterone has a significant decrement. Our results indicate that SYCP3 accumulations are a particular feature of murine prepubertal organisms. However, more studies should be performed to understand the reason. The contribution of our work in the behavior of murine SYCP3, relates it with the known structure of human SYCP3 and relates its auto-assembly intrinsic capacity with the ability of forming accumulations. Also we found that these accumulations doesn't seem to be a consequence of protein or gene expression itself and none of the sexual hormones seems to have a relation with SYCP3 accumulations neither. Remarkably, we found that, in murine models, accumulations of this protein have different ratio and a specifical structure depending on the analyzed stage of meiotic prophase I.

Fibroaging a dismissed player regulated by extracellular matrix

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Aging is a multifactorial biological process leading to a progressive decline of physiological functions. The process of aging includes numerous changes in the cells and the interactions between cell-cell and cell-microenvironment remaining a critical risk factor for the development of chronic degenerative diseases. Systemic inflammation, known as inflammaging, increases with aging contributing to age-related morbidities. Just as well, persistent and uncontrolled activation of fibrotic pathways, with excessive accumulation of extracellular matrix (ECM) and organ dysfunction is markedly more frequent in the elderly. In this context, we proposed the concept of “Fibroaging”, that is, the propensity to develop tissue fibrosis associated with aging, and propose that ECM is a key player underlying this process.

During aging, molecules of the ECM become damaged through many modifications including glycation, crosslinking, and accumulation, leading to matrix stiffness which intensifies ageing-associated alterations. We suggest that ECM accumulation and stiffening, often viewed as a consequence of tissue fibrosis, play a critical role in the initiation and progression of fibrogenesis by promoting mechano-activation of pro-fibrotic signaling pathways. Thus, stiffer ECM activates several fibrotic amplification feedback loops including among others, the release of growth factors, primarily TGF-β, and inducing the nuclear translocation of YAP and TAZ which target profibrotic gene expression. The cells not only respond to the mechanical stress by themselves with changes in proteostasis, mitochondrial function, metabolism, and nuclear architecture among others but also generate multiple signals to communicate with neighbor cells activating fibrotic positive feedback loops and amplifying the fibrotic response.

Characterization of the effect of white, red and blue LED light in the growth, photosynthesis and phycocyanin content in *Arthrospira maxima*

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Phycocyanin is a primary photosynthetic pigment found in *Arthrospira maxima*. Its water solubility makes it valuable in various industries, including food, pharmaceutical, cosmetic, and medical. Consequently, the production of phycocyanin-rich *Arthrospira maxima* biomass has become a significant biotechnological challenge. This study investigated the impact of white, red, and blue LED lights on the growth, photosynthesis, and phycocyanin production of *Arthrospira maxima*. The chromatic induction protocol consisted of two phases: 1) Cells were cultivated in 12 experimental units for 7 days at 35°C in Zarrouk medium with a white light (400-700nm) phototonic density of 100 ± 5 µmol/m2.s; 2) the culture was maintained throughout 7 days with the same experimental condition, exposing 3 experimental units to different wavelengths. White light (400-700nm), red (600-800), blue (450-550) and red+blue. Growth was determined by dry weight, phycocyanin content by UV-VIS spectrophotometry, and photosynthesis by chlorophyl a fluorescence emission. Our results show that maximum growth was achieved in white light and red+blue combination, decreasing 20% in blue and 45% in red light compared to the control. Blue light exposure induced a significant increase of 30% of phycocyanin content per dry biomass in comparison to the control. Photosynthetic activity increased in all treatments, being the highest in blue light, which was 135% superior to white light. These results indicate that *Arthrospira maxima* adjusts its cellular processes and light harvesting structures (phycobilisomes and antennae complexes) as a function of the wavelength it grows under, therefore, the chromatic induction in two phases biotechnological protocol is useful for biomass obtention with higher phycocyanin content.

Blood-brain barrier-on-a-chip: A novel tool to study neuroinflammation

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Neuroinflammation (NI) plays a role in the pathogenesis of various neuropathologies and addictions. Acute inflammation aims to restore homeostasis, but when it becomes chronic, it may sustain or exacerbate tissue damage. Currently, the development of molecules with anti-inflammatory activity has grown. These require evaluation *in vivo* experimental models, which, although they allow assessments within a physiological context, present various limitations. To address these limitations, platforms known as “organ-on-a-chip” have been developed, based on the combination of cell cultures with physiologically relevant stimuli. Given the fundamental role of the blood-brain barrier (BBB) in modulating and limiting the exchange of molecules, cells, and exogenous agents, such as drugs, between peripheral circulation and the central nervous system, we have designed a blood-brain barrier-on-a-chip (BBBCh) to study new anti-inflammatory molecules. In this BBBCh, murine endothelial cells,

astrocytes, pericytes, and microglia were co-cultured. Subsequently, the functionality, phenotype expression, cytokine secretion, and de novo production of basal membrane proteins of the BBB were evaluated. It was demonstrated that the BBBCh limits the diffusion of 10, and 40 kDa dextran/fluorescein and promotes the expression of ZO-1, Claudin-5, and nidogen. An LPS stimulus was applied, followed by treatment with dexamethasone (Dex). It was observed that LPS increases the permeability of the BBBCh, which is restored with Dex treatment. In both cases, the concentration of various cytokines was modified, mainly TNFα and IL-6. Additionally, the expression of Claudin-5 increased after Dex treatment. These results show the capability of this BBBCh to analyze molecules that modulate inflammation and modify the functionality and phenotype expression of the BBB. Furthermore, they pave the way for using this system to discover new experimental molecules with similar effects.

Cross-Neutralizing Anti-Dengue 2 IgG Antibodies from Patients and BALB/c Mice against Chikungunya Virus

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Dengue (DENV) and Chikungunya (CHIKV) viruses can be transmitted simultaneously by Aedes mosquitoes, and there may be co-infections in humans. However, how the adaptive immune response is modified in the host has yet to be known entirely. In this study, we analyzed the crossreactivity and neutralizing activity of anti-Dengue 2 IgG antibodies against CHIKV in sera of patients from the Mexican Institute of Social Security in Veracruz, Mexico, collected in 2013 and 2015 and using IgG antibodies from 6–8-week-old male BALB/c mice inoculated with 30 ng of total viral protein of purified DENV and then with CHIKV. The neutralizing activity of 2013 anti-DENV IgG antibodies from both primary and secondary infection was higher than in sera from patients with positive anti-CHIKV IgG antibodies. The anti-DENV2 IgG neutralizing antibody titers of sera from 2015 patients positive for both anti-DENV IgG and anti-CHIKV IgG antibodies (7.88 Log2) were significantly higher than those of sera from patients positive for anti-CHIKV IgG antibodies but negative for anti-DENV IgG antibodies (1.78 Log2; $p < 0.01$). Mice that were first inoculated with DENV2 and then with CHIKV showed a significant increase in anti-DENV2 IgG antibodies (19.67 ± 10.25 AU) compared to the mice of the control group (2.549 ± 1.35 AU, $p < 0.0001$). The evaluation of anti-DENV2 IgG antibody neutralizing capacity showed that IgG antibodies of the group of mice that were first inoculated with DENV2 and then with CHIKV showed a higher DENV2 neutralization than the IgG antibodies of the group of mice that were only inoculated with DENV2 up to a dilution of 1:320 (23.5% vs. 2.5%, $p < 0.0001$). In serum samples from 2013 patients, we found that anti-DENV IgG antibodies crossreacted with CHIKV and had neutralizing capacity, even though there were no reported cases of Chikungunya in Mexico. Mice that were only inoculated with DENV2, which presented a stronger IgG cross-reactive and neutralizing capacity with CHIKV.

Some aspects of T cells mechanobiology

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The responses of immune cells are mediated largely through direct cell-cell contacts, which allow cells to exchange biochemical signals and also exert and sense mechanical force. The contribution of mechanical forces to molecular and cellular function has fascinated biologists and physicists alike for more than a decade, and in recent years it has become evident that mechanical forces are fundamental to immune cell function. We will present some experimental approaches and results which all together try to dissect the mechanobiology of T lymphocytes during the early stages of their recognition of a partner cell or substrate. Such biophysical methodologies may be applicable to other immune cells, or other fields of cell biology.

Maternal mRNA localization in embryos

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The localization of maternal mRNA is a hallmark in oocyte maturation and early embryogenesis. In C. elegans, a subset of maternal mRNAs is compartmentalized into germ granules, membraneless organelles enriched in RNA and proteins. This process is mediated by the intrinsically disordered binding protein, MEG-3, which plays a key role in stabilizing germ granules and driving mRNA localization. Using a combination of genetics, microscopy, and biochemistry, we are investigating how intrinsically disorder regions bind to RNA and drive biological phenotypes.

Three-dimensional reconstruction and cytochemical analysis of the nucleolus in the *Ustilago maydis* fungus

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The nucleus of eukaryotic cells contains dynamic, membraneless subcompartments within the nucleoplasm, where the nucleolus is the largest intranuclear body. The ultrastructural organization of the nucleolus in mammals and plants is characterized by three morphological elements: dense fibrillar component, fibrillar centers, and granular component. These compartments, primarily composed of ribonucleoproteins and nucleic acids, play critical roles in pre-ribosomal RNA synthesis, processing, and ribosome assembly. Although the nucleolus in the ascomycete *Saccharomyces cerevisiae* is recognized as a bipartite compartment, nucleolar properties in basidiomycete fungi remain largely unexplored. Here we show the nucleolar architecture in yeast-like cells of the basidiomycete *Ustilago maydis*, known for causing the corn smut, or “Huitlacoche,” in maize. Our findings reveal a prominent, peripheral intranuclear nucleolus with a spheroid shape and fibro-granular elements. Cytochemical analysis indicates nucleolar structure is composed of ribonucleoproteins and positive for specific argyrophilic nucleolar proteins, while it is partially surrounded by DNA. Furthermore, FIB-SEM and electron tomography analyses allowed us to identify a subnucleolar body at the periphery of the nucleolus. Notably, treatment with low doses of actinomycin D resulted in nucleolar segregation. This work improves our knowledge to understand the nucleolar and nuclear structure in basidiomycete fungi.

Identification of peroxisomal proteins (Peroxin 4 and ACSL4) in *Entamoeba histolytica*

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The *Entamoeba* genus includes a group of unicellular, anaerobic parasitic organisms capable of infecting vertebrates such as human, non-human primates, reptiles and fish. Members of this genus seem to lack peroxisomes, which are single-membrane organelles involved in several metabolic processes, including the degradation of hydrogen peroxide and lipid metabolism. In this work, we identified in Entamoeba histolytica, the etiological agent of amoebiasis, two peroxisome-specific enzymes, Peroxin 4 (PEX4) -

involved in the biogenesis of the organelle- and acyl-CoA synthetase long chain family member 4 (ACSL4) -which participates in fatty acid metabolism within peroxisomes. Bioinformatics analysis revealed that E. histolytica PEX4 possess a ubiquitin domain and the conserved cysteine residue reported in all peroxines, while ACSL4 has a transmembrane region and an AMP-binding site. Using polyclonal antibodies against *Giardia lamblia* PEX4 and ACSL4, we identified PEX4 and ACSL4 in the cytoplasm of *E. histolytica* trophozoites by confocal microscopy. Western blot experiments confirmed the presence of both proteins in E. histolytica protein extracts. Finally, by transmission electron microscopy of 3-3'-diaminobenzidine (DAB)-stained *E. histolytica* trophozoites, we identified a dark-brown insoluble precipitate within cytoplasmic vesicles, suggesting the presence of peroxisomal enzymatic proteins, as these enzymes are known to react with DAB and generate insoluble precipitates. Our results suggest that E. histolytica may possess simplified peroxisomal machinery for the biogenesis and metabolism of this organelle.

Evaluation of Oxidative Stress and Therapeutic Resistance in MCF-7 cells Treated with L-arginine and Doxorubicin

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ROS are elevated in cancer cells and their effects on cell growth are concentration dependent. Therapeutic approaches against cancer have been the subject of several studies, because in many cases the forms of treatment are effective, but the comorbidities are aggravating for the prognosis and survival, including breast cancer. Doxorubicin (DOX) has been used to treat a broad spectrum of cancers for many years for be a highly potent compound, being one of the most extensive chemotherapy drugs used. The use of L-arginine (L-ARG) in dietary supplementation for cancer patients has shown positive results due to its relevance in different body systems, but its use may have direct effects on tumor cells. Objective: To investigate the effects of combined treatment with L-arginine and doxorubicin in the tumor lineage MCF-7 and expression of enzymes related to oxidative stress and therapeutic resistance. Methodology: The treatment consisted of exposure to concentrations 800ug/ml L-arg + 1uM Dox, 1600ug/ml L-arg + 2uM DOX e 3200ug/ml L-arg + 4uM Dox. The AlamarBlue assay was used to measure proliferation and assess cell viability. Evaluation of catalase, NOX2 and NRF2 expression was performed by Western blot and ImageJ software was used to measure the amount of each protein. Data were analyzed using the GraphPad Prism
8.0.2 software and using the one-way ANOVA test. Results: In our results, cell viability was reduced and oxidative stress increased with the treatment in combination of L-arginine and Doxorubicin dose dependently. Furthermore, the treatment reduced CAT, increased NOX2 and reduced NFR2 at all concentrations. In conclusion, the combined treatment of L-arginine with DOX may be an indication to overcome therapeutic resistance and be possible therapy in the treatment of breast cancer.

Identification of peroxisomal proteins (Peroxin 4 and ACSL4) in *Entamoeba histolytica*

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The *Entamoeba* genus includes a group of unicellular, anaerobic parasitic organisms capable of infecting vertebrates such as human, non-human primates, reptiles and fish. Members of this genus seem to lack peroxisomes, which are single-membrane organelles involved in several metabolic processes, including the degradation of hydrogen peroxide and lipid metabolism. In this work, we identified in *Entamoeba histolytica*, the etiological agent of amoebiasis, two peroxisome-specific

enzymes, Peroxin 4 (PEX4) -involved in the biogenesis of the organelle- and acyl-CoA synthetase long chain family member 4 (ACSL4) -which participates in fatty acid metabolism within peroxisomes. Bioinformatics analysis revealed that *E. histolytica* PEX4 possess a ubiquitin domain and the conserved cysteine residue reported in all peroxines, while ACSL4 has a transmembrane region and an AMP-binding site. Using polyclonal antibodies against *Giardia lamblia* PEX4 and ACSL4, we identified PEX4 and ACSL4 in the cytoplasm of E. histolytica trophozoites by confocal microscopy. Western blot experiments confirmed the presence of both proteins in *E. histolytica* protein extracts. Finally, by transmission electron microscopy of 3-3'-diaminobenzidine (DAB)-stained *E. histolytica* trophozoites, we identified a dark-brown insoluble precipitate within cytoplasmic vesicles, suggesting the presence of peroxisomal enzymatic proteins. These enzymes are known to react with DAB and generate insoluble precipitates. Our results suggest that E. histolytica may possess simplified peroxisomal machinery for the biogenesis and metabolism of this organelle.

Evidence that the EPO receptor is necessary for the cellular protective activity of EPOrh in Neuro-2a cells exposed to damage by oxygen and glucose deprivation

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In studies of ischemic damage, it has been shown that the administration of recombinant human erythropoietin (rhEPO) has neuroprotective effects (Sun et al., 2023). However, the dose necessary to induce neuroprotection is associated with unwanted side effects and its neuroprotective activity is related to the expression of its canonical receptor (EPOR) (Sanchez et al., 2009; Ogunshola et al., 2009). In addition, it has been reported that rhEPOR acts through a heteroreceptor formed by at least one EPOR subunit and the common beta receptor (CD131) (Brines et al., 2004).Currently, there is only one study without damage induction in which an increase in cell viability and proliferation was reported, related only to the protein expression of the CD131 receptor when Neuro-2a cells were incubated with 5 ng/mL rhEPO for 24 h (Ding et al., 2015). In this sense, the objective of the present study was to evaluate the effect on cell viability of exposure for different times to damage by oxygen deprivation, glucose and reoxygenation (OGD/R), and the subsequent administration of different doses of rhEPO during re-oxygenation, as well as to evaluate the changes in EPOR gene expression. Neuro-2a cells were exposed to 4, 6 or 9 h of OGD and 12 h of reoxygenation, respectively; While to determine the decrease in cell viability loss upon administration of rhEPO, cells were treated with different concentrations (80, 60, 40, 20, and 10 U/mL) of rhEPO during the 12 h of reoxygenation after 4 or 6 h of OGD. Cell viability was assessed by MTT assay and relative gene expression analysis was performed by real-time PCR using the 2-ΔΔCt method. When evaluating the response of Neuro-2a cells to OGD/R, a lower percentage of viability was observed which was dependent on the exposure time, compared to the Ctrl group ($p<0.05$). Furthermore, unlike the report by Ding et al, in our work we did not observe a significant increase in cell viability in any of the experimental or pharmacological study groups evaluated, damage times and doses of rhEPO administered compared to the control groups. Regarding changes in EPOR gene expression, mRNA expression was not observed in any group evaluated at 6h OGD and 12h of reoxygenation, administering 80 U/mL of rhEPO), with respect to the reference gene. In addition, this is the first report of EPOR gene expression in this cell line, and there are currently no studies supporting that rhEPO administration can mediate signaling only through the CD131 receptor. However, further investigations with longer erythropoietin treatments at different time points post-damage and assessment of EPOR variant expression are needed to understand the effects of rhEPO in this cell line.

Analysis of virulence factors in extracellular vesicles secreted by *Naegleria fowleri*

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Naegleria fowleri is the etiologic agent of Primary Amoebic Meningoencephalitis, an acute and fulminant infection of rapid progression that affects the Central Nervous System, mainly in children and young adults with a history of having performed aquatic activities in natural or artificial freshwater bodies. This disease has a mortality rate greater than 95%. One of the main problems is its symptomatic similarity with other meningitis caused by viruses or bacteria, which makes it difficult to make a rapid and timely diagnosis that prevents the progression of this infection. Therefore, it is necessary to know the antigenic determinants as well as the pathogenicity mechanisms of this amoeba in order to implement strategies that allow better therapeutic targets and antiamoebic diagnoses. Therefore, the aim of this work was to analyze some virulence factors as part of the extracellular vesicle (EVs) cargo secreted by *N. fowleri*. The secretion as well as the identification of EVs cargo was performed by immunocytochemistry, SDS-PAGE, Western blot and RT-PCR. Our results showed that *N. fowleri* secretes a wide variety of vesicle sizes ranging from 0.2 to more than 2 micrometers. In addition, these EVs were recognized by anti-N. fowleri, anti-Naegleropore B, anti-19 kDa polypeptide band, anti-membrane protein Mp2CL5, anti-protease cathepsin B and anti-actin antibodies. Specifically in relation to small vesicles, our purified exosomes were recognized by CD63 and Hsp70 markers, along with the previously mentioned proteins. RT-PCR analysis was made through the isolation of EVs from *N. fowleri* trophozoite culture by concentration, filtration and ultracentrifugation. Interestingly, we obtained PCR products for Nfa1, NPB, Mp2CL5, and CatB genes as part of exosomes cargo. This suggests that the molecules identified in this work could play an important role in communication as well as in infectious processes caused by this amoeba.

Introduction

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Cell Aging is a complex biological process characterized by a progressive decline of cellular functions, leading to an increase in chronic age-related diseases and ultimate death. Several hallmarks of aging have been described as influenced by environmental and genetic factors resulting in changes in metabolism, mitochondrial function, and ER stress, leading the cell into cellular senescence. Understanding the mechanisms underlying these changes will promote healthy aging and will contribute to mitigating the consequences of age-related diseases.

Lung regeneration via 3D cultures of progenitor cells

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Lung regeneration is a fundamental challenge that has begun to be undertaken thanks to the discovery of the stem cells involved. It has been proposed that the interaction of type 2 epithelial cells and fibroblasts is responsible for recovering the alveolar epithelium involved in the pathophysiology of different lung diseases. During regeneration, particularly in aged tissues, progenitor cells experience alterations in their microenvironment that affect their functionality. In this sense, the notion has been consolidated that these cells as facultative progenitors require a supportive microenvironment or "niche" to maintain their function. In the case of the lung, the cells that have been described are fibroblasts, although it is not exclusively these cells that make up the niche. Even when epithelial cells have a primary role, studies have been insufficient because in conventional culture these cells rapidly lose their phenotype. However, the culture system known as 'organoids' offers a novel alternative. Alveolar organoids are formed from the small number of progenitor cells that proliferate with the support of mesenchymal cells and matrigel, these cells subsequently differentiate and assemble in three-dimensional structures. When cells are cultured separately, these structures are not formed, demonstrating that physical and/or paracrine interaction is necessary. Therefore, understanding the mechanisms by which these cells lose their functionality in specific contexts is of paramount importance. This knowledge could elucidate how alterations in the microenvironment contribute to

impaired alveolar regeneration and stem cell exhaustion, ultimately guiding the development of specific therapeutic strategies.

Long distance transport of RNA and proteins in plants

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Long distance communication between tissues and organs is essential for plants to maintain homeostasis, regulate its development and to respond to changes in environmental conditions. There are many examples of this, such as flowering, systemic silencing, responses to pathogens and more. It is also evident that the vasculature, both phloem and xylem, is essential for the transport of the potential systemic signals. Thus, the vascular tissue harbors signals involved in long range signaling, such as phytohormones, proteins and small RNAs although many of these have yet to be described. We have previously described the long-distance transport of mRNAs through the phloem and found that some may have a role in developmental regulation. For instance, we have shown that one of these, encoding a Translationally Controlled Tumor Protein (TCTP), promotes proliferation as well as whole plant regeneration. This will be described in more detail in this presentation. On the other hand, we have found that certain viral genomic components are transported systemically in the absence of proteins required for long distance movement, supporting the notion that plants may harbor a machinery involved in the delivery of RNAs to distant tissues. Finally, some applications of this knowledge will be presented as well.

The testis specific Na,K-ATPase; a "pump" specifically dedicated to sperm function

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The Na,K-ATPase (NKA) transporters comprise a group of plasma membrane isozymes that use the energy from the hydrolysis of ATP to exchange cytoplasmic Na+ for extracellular K+. Na,K-ATPase isozyme diversity results from association of different molecular forms, or isoforms of the catalytic α and glycosylated β subunits that constitute the transporter. Among these isoforms, Na,K-ATPase α4 (NKAα4) has the most restricted pattern of expression, and is specifically found in male germ cells of the testis. NKAα4 is expressed after cell meiosis and is particularly abundant in spermatozoa. Within sperm, NKAα4 is mainly localized to the midpiece of the sperm flagellum. After heterologous expression in insect cells, we have shown that NKAα4 exhibits biochemical characteristics that are unique, including apparent affinities for the transported ions and a sensitivity to the NKA inhibitor ouabain, which are different from those of the other NKA isoforms. Selective dose dependent inhibition of NKAα4 with ouabain showed that this isoform is essential for sperm motility through the control of membrane potential, [Na2]i, [Ca2+]i and [H+]i in the cells. Importantly, transgenic mice in which NKAα4 expression was deleted or was over-expressed showed sperm functional phenotypes. Thus, knockout male mice lacking NKAα4 were completely sterile and spermatozoa from these mice lost most of their motility and were unable to fertilize eggs and in vitro. In contrast, NKAα4 over-expression in mice, had sperm with increased sperm motility. Overall, these results demonstrate the importance of NKAα4 in sperm function and its requirement for male fertility, a function which cannot be compensated by the somatic Na,K-ATPase α1 (NKAα1) isoform also expressed in sperm. Future experiments are now focused on using NKAα4 as a marker for male fertility and as a target for male contraception. [Supported by NIH grant HD080423].

***Trichomonas vaginalis* aspartic proteinase (Tv-AP): an analysis of the protein expression, localization and enzymatic activity in different trichomonad isolates and its regulation by iron**

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Trichomoniasis is the most common non-viral sexually transmitted infection in the world, which is caused by the protozoan parasite *Trichomonas vaginalis*. *T. vaginalis* has various virulence factors to colonize tissues, such as adhesins, phospholipases, porins, proteases, lipoglycan, exosomes, and others. An aspartic protease (Tv-AP) positively regulated by glucose was described by our group. Tv-AP degrades human hemoglobin, is in vitro secreted and localized in multiple subcellular sites: cytoplasm, lysosomes, Golgi apparatus, endoplasmic reticulum, vacuoles, and nucleus. Our goals were to determine whether there are differences in the protein expression, localization and proteolytic activity of Tv-AP among distinct *T. vaginalis* isolates and to analyze the effect of iron on the Tv-AP expression and localization in one of the Tv isolates. Thus, we performed western blot, indirect immunofluorescence assays, and AP enzymatic kinetics determined by spectrofluorimetry. In a selected trichomonad isolate, we also evaluated the iron effect in the Tv-AP protein expression and localization in parasites grown under different iron conditions by western blot and indirect immunofluorescence assays. Our results showed that Tv-AP is differentially expressed in all *T. vaginalis* isolates analyzed, localized in multiple subcellular sites (cytoplasm, vacuoles, ER, Golgi apparatus and nucleus), and was enzymatically active at pH 4.5. Additionally, Tv-AP was positively regulated by iron, a greater amount of protein was observed in parasites grown under high iron than under iron restriction conditions. These results show that each *T. vaginalis* isolate has a characteristic behavior regarding to Tv-AP, which could be related to the parasite virulence. Moreover, Tv-AP is positively regulated by iron. However, the mechanism of regulation by iron is still unknown. Thus, future work will be directed to address these questions.

Autophagy Activation in a Mouse Model of Hypersensitivity Pneumonitis induced by *Saccharopolyspora rectivirgula* exposure

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Autophagy Activation in a Mouse Model of Hypersensitivity Pneumonitis induced by Saccharopolyspora rectivirgula exposure Andrea Montserrat Sánchez Barajas, Miguel Gaxiola, Annie Pardo Cemo, Moises Selman Lama, Sandra Cabrera Benítez. Laboratorio de Biopatología Pulmonar Ciencias-INER, Facultad de Ciencias, UNAM, Circuito interior S/N,Ciudad Universitaria,Coyoacán, 04510 CDMX E-mail: scb@ciencias.unam.mx Tel. 56224800 #84076 Hypersensitivity pneumonitis (HP) is a complex interstitial lung disease (ILD) caused by exposure to an inhaled antigen or mixture of antigens in susceptible individuals that develop an exaggerated immune response. In order to reduce the multivariable challenge in human disease, such as genetics, environment, age, gender and lifestyle, that impact autophagy flux, we develop a HP mouse model with the aim to evaluate autophagy regulation in lung, analyzing established periods of time and one antigen. Autophagy is a degradation pathway characterized by the formation of a double-membrane vesicle that fuses with lysosomes for the cargo degradation and its role in HP has not been studied yet. C57BL6 mice were intranasally instilled with 50 µg of Saccharopolyspora rectivirgula (SR) in combination with 5µg of LPS three times a week for three and twelve weeks and mice were euthanized three days after the last exposure. Mouse lungs were stained with hematoxylin-eosin and Masson's trichrome stain, and histological changes, autophagic flux and cytokine profile were analyzed. The lung of SR+LPS-exposed mice exhibited cellular infiltrates in the pulmonary parenchyma, mainly lymphocytes and macrophages, as well as thickening of the alveolar epithelium and collagen deposition. In the LBA we observed a significant increase in the cytokines CXCL1, CCL1, TNF-α, CCL25 compared to non exposed controls. We evaluated changes in the autophagic flux using GFP-LC3 transgenic mice, where we observed an increase in the autophagosomes formation in mice exposed to SR+LPS compared to the controls. By immunofluorescence we detected colocalization of LC3B with ATG4B and p62 in macrophages, as well as bronchial and alveolar

epithelium. In addition, by immunoblot, we observed an increase of LC3B, p62, ATG4B and ATG5 protein levels in SR+LPS mouse lungs compared with control tissues, confirming the findings obtained by immunohistochemistry, and suggesting autophagy is being activated in lung after SR and LPS exposure. With the aim of evaluating the role of autophagy impairment in the disease, we developed the HP model in autophagy-deficient mice (atg4b-/-), these mice demonstrated less inflammation i n lung parenchyma than WT mice, and a significant decrease in CXCL1, CCL1, CCl25 compared to WT mice in the BAL, showing that autophagy could have a detrimental role in the pathogenesis of the disease. This abstract is funded by: DGAPA UNAM PAPIIT IN224323

Glycine effects on orphan receptors and APP expression in Wistar rat neonatal glial cells culture

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Glycine functions as a neurotransmitter in the central nervous system and plays various roles in peripheral and nervous tissues, including antioxidant, anti-inflammatory, cryoprotective, and immunomodulatory effects. Alzheimer's disease (AD) is a progressive brain disorder primarily associated with memory decline and cognitive degeneration, impacting the ability to perform simple tasks. AD affects cognitive functions such as behavior, personality, and memory. Studies have linked several receptors to the pathology of AD, including glutamate and GABA receptors. Additionally, the nerve growth factor receptor-interacting protein and orphan receptors like GPR3, GPR6, and GPR12 have been shown to increase amyloid precursor protein (APP) expression and the formation of amyloid plaques in the brain, a hallmark of Alzheimer's pathology. Objective: to analyze the effect of glycine on the expression of APP and orphan receptors GPR3, GPR6, and GPR12 in neonatal glial cell cultures. Methods: neonatal glial cells were cultured in 6-well plates (4 x 10^5 cells per well) using DMEM/F12 medium. Astrocytes were stimulated with glycine (20 mM, 40mM and 60mM) for two different time points: 1 hour and 24 hours. Gene expression levels of APP and the orphan receptors were measured using RT-qPCR. Results: our results showed that glycine increased the expression of the orphan receptors GPR3, GPR6, and GPR12, while it decreased APP expression. Conclusion: this study suggests that glycine enhances the expression of orphan receptors GPR3, GPR6, and GPR12, while downregulating APP in neonatal glial cells. These findings imply that glycine could play a modulatory role in pathways associated with amyloid plaque formation in Alzheimer's disease.

Cellular immunomodulation exerted by Agave fructans and probiotic exopolysaccharides through the NF-kB/AP1 pathway

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Eukaryotic cells establish communication with other cells through molecules like cytokines. A vital signaling pathway involved in such cellular communication is that mediated by Toll-like receptors (TLRs). A variety of carbohydrates of natural origin can exert immunomodulatory effects on cells. These effects can help develop new strategies for diseases of inflammatory origin. Mexican agaves are enriched in non-digestible carbohydrates (NDCs) known as fructans, which have demonstrated anti-inflammatory properties. Moreover, probiotic bacteria produce another kind of NDCs, exopolysaccharides (EPS), which are secreted to the milieu, showing immunomodulatory effects, too. Each probiotic strain produces specific EPS depending on the carbon source that is accessible during its growth. The present study explores the immunomodulatory effects of agave fructans, probiotic strains, and EPS through the NF-kB/AP1 pathway. This pathway is a central axis in both innate and adaptive immune responses. Experiments were conducted using human TLR4 and TLR2 reporter HEK293 Cells (NF-κB) expressing TLR2 and TLR4. These cells allowed the quantitative evaluation of the activation and inhibition of the NF-kB/AP1 pathway. The THP1-Blue NF-κB Cells human monocyte cell line, which expresses all TLRs, was used to provide a comprehensive view of the immune modulation mediated by these receptors. The

fructans used in the study were obtained from three different agave species. Regarding probiotics, two strains were used: one isolated from pig feces and *Lactobacillus reuteri*. These strains were selected for their ability to produce EPS with potential immunomodulatory properties. Our results indicate that agave fructans, probiotics, and EPS exert different immunomodulatory effects. These effects suggest that regulation of the NF-kB/AP1 pathway by these biomolecules may lead to more specific regulation of inflammation. Notably, a potent inhibitory effect on the NF-kB/AP1 pathway was detected by the supernatant of the *L. reuteri* cultures on TLR4 and THP-1 when agave fructans were used as a carbon source. This suggests that the combination of probiotics and fructans enhances the anti-inflammatory effects. These results have the potential for therapeutic strategies, especially in managing chronic inflammatory and autoimmune diseases. Our findings open new perspectives for developing therapeutic approaches based on natural products to improve immune system regulation and human health.

Antibody neutralizing capacity of anti-Chikungunya IgG against Dengue Virus type 2 from patients and BALB/c mice

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Arboviruses is a group of viruses transmitted by arthropod vectors like sand flies, ticks, or mosquitoes. The latter, species such as Aedes albopictus and Aedes aegypti can transmit Dengue (DENV) and Chikungunya (CHIKV) viruses. Some factors such as climate change, international travels and the occupation of natural spaces, have led to an increase in the number of cases, making this virus infections a global health problem. DENV and CHIKV have in common the geographical regions in which they are present, so this virus can be transmitted simultaneously by Aedes mosquitoes, and there may be co-infections in humans in which the most severe clinical forms of fevers caused by these two viruses are manifested. It is essential to understand the alterations in the mechanisms involved in the scenario of a co-infection with these two viruses. In this sense, one of the most important aspects is the immune response, in particular, how the adaptive immune response is modified in the host has yet to be known entirely. Therefore, in this study, we analyzed the crossreactivity and neutralizing activity of anti-Chikungunya IgG antibodies against DENV in sera of patients from the Mexican Institute of Social Security in Veracruz, Mexico, collected in 2013 and 2015 and using anti-Chikungunya IgG antibodies of BALB/c against CHIKV. Mice were inoculated with CHIKV, and then with DENV had IgG antibodies with more significant anti-CHIKV IgG antibody neutralizing activity. However, the inoculation only with CHIKV resulted in better neutralization of DENV2. In sera obtained from patients in 2013, significant cross-reactivity and low anti-CHIKV IgG antibody neutralizing activity were observed. These results suggest that CHIKV stimulates DENV2-induced memory responses and vice versa. Furthermore, cross-reactivity between the two viruses generated neutralizing antibodies, but exchanging CHIKV for DENV2 generated a better anti-CHIKV neutralizing response.

Microscopic evidence of contamination in hepatocytes of Anolis porcatus (SQUAMATA: POLYCHROTIDAE)

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The use of chemical compounds to improve production and to enrich the soil in cultivated areas has harmful consequences for the fauna associated with these areas, and reptiles are particularly vulnerable. Among the indicators of contamination in organisms exposed to these degraded habitats is

the pigmentation of liver cells and tissues, which act as a defense against harmful compounds. The objectives of this work were to demonstrate the presence of pigments in hepatocytes of *Anolis porcatus* collected in anthropized areas. The work was carried out in a cultivated area of the province of Havana with apparent levels of contamination due to the use of fertilizers. The specimens were treated ethically following the established protocols, they were anesthetized and dissected, and liver fragments were fixed in 4% paraformaldehyde and subjected to the classic paraffin embedding technique. The sections were stained with hematoxylin and eosin and Masson's trichrome. Other portions of this tissue were fixed in 2.5% glutaraldehyde and processed for transmission electron microscopy (TEM). Energy Dispersive Spectroscopy (EDS) was also performed to identify possible contaminants. The results showed the presence of numerous pigmented bodies in the liver, melanomacrophage centers (MCCs) derived from Kupffer cells, whose presence and abundance are analyzed in this work, as well as evidence of potassium, iron, zinc, cadmium, titanium and other elements into the hepatocytes.

Scanning electron microscopy of the undulating membrane in spermatozoa of amphibians from Cuba

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Amphibians are a special class of vertebrates that have demonstrated a variety of evolutionary tendencies in their reproductive biology. Besides, the sperm cell morphology provides important insights into fertilization mechanisms. Cuba has some 68 recognized species of amphibians, with a high percentage of endemism of 95.3%. Only the order Anura is represented in the Cuban archipelago. The genus *Eleutherodactylus* stands out for its diversity. Meanwhile, the archipelago is home to the greatest wealth of *Peltophryne* species. The ultrastructure of anuran spermatozoa has been associated with phylogenetic relationships and with fertilization environment. Also, it has been postulated that a complex spermatozoon displays a distinctive condition in their axial and juxtaxonemal fibers.

Male specimens, three for each species from *Eleutherodactylus*, and *Peltophryne*, also *Osteopilus septentrionalis* were collected during the breeding seasons of 2024. Sperm suspensions were prepared by crushing a testis or a piece of testis in a small quantity of Ringer rana. They were spread over cover slips and 4% paraformaldehyde was added for fixation. Spermatozoa were coated with gold and observed with a scanning electron microscope (JEOL 2100).

Results shown sperm cell from these Cuban amphibians characterized by the presence of both accesory fibers an undulating membrane was also related to internal fertilization or viscous fertilzation background as observed in anurans. The findings are discussed in this work by analyzing the fertilization strategies of these amphibians.

Parthenogenetic eggs of Bombyx mori L. (LEPIDOPTERA, BOMBYCIDAE)

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Bombyx mori L., the silkworm, is a holometabolous insect of great importance in the silk industry and presents a complete metamorphosis from egg to adult stage. Its life cycle presents four defined stages: egg, larva, pupa or chrysalis and moth. Most animals reproduce sexually and, in 1% of the species on the planet, eggs can develop without fertilization through parthenogenesis. In B. mori, this was first observed in the 18th century and its artificial induction in 1847 from females kept under sun exposure. The silkworm also uses itself as a bioreactor to produce recombinant proteins; several vectors have been developed and it has been shown that they influence the production of mammalian proteins: interferon, human albumin, mouse antibodies and human collagen. The insertion of transgenes is the critical step in the production of transgenic silkworms. These procedures could be simplified using parthenoclones.

The advantage of using parthenogenetic strains is that after successful integration of the transgene into the silkworm genome, a single female initiates a clonal lineage with a genome completely identical to that of the mother, which can be easily maintained as pure female populations without sexual reproduction. Subsequent generations have the same expression as the inserted gene. Therefore, the objective is to obtain eggs from parthenogenetic females for the subsequent production of proteins used in medicine. In the experiments, eggs from three pure bivoltine strains were activated by acid treatment: J7, J3 and C6. The acid treatment was carried out in the CIPB laboratory under controlled conditions. This work shows optical and scanning electron micrographs of parthenogenetic eggs obtained in Cuba and their color changes that indicate the fertility of the embryo.

Correlation between *Helicobacter pylori* Infection with Metabolic Syndrome in a population of Mexico City

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The *Helicobacter pylori* infection presents a high prevalence in Mexico (>70% in adults) and is associated with various metabolic diseases, such as the Metabolic Syndrome (MS). The relationship between infection an MS has been detected in Eastern countries, but in Mexico there are no similar studies, even when the MS prevalence in the Mexican population is worrying (54%). Here, the aim was to establish the association between these two diseases in a vulnerable population of Mexico City. An epidemiological cross-sectional study was conducted, sampling 515 individuals during two health events carried out at UACM, which allowed us to obtain their medical history and anthropometric and biochemical parameters. Based on the results obtained which were scrutinized with robust and complex statistical analyses, we determined that the majority of individuals were young women (64.7%) and a high percentage of subjects were overweight or obese (53% men vs 56% women). The MS prevalence was 30.3%, the older adults being the most affected. Although gender did not predispose individuals to develop MS, overweight, obesity, dyslipidemia, hyperglycemia, and high HbA1c levels contributed to the manifestation of this condition. Moreover, some individuals with MS, tested positive for H. pylori (29.4% vs 7.6% with and without MS, respectively). In conclusion and thanks to the statistical analyses performed, our findings demonstrated that H. pylori infection, age, and lack of physical activity are risk factors for the MS development in the examined population. We would like to thank the Cinvestav for their invaluable support and contribution to this research.

Nucleolar compartments visualized by atomic force microscopy

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The nucleolus is a plurifunctional intranuclear organelle where synthesis and processing of pre-rRNA as well as ribosome assembly take place. It is composed by three different ultrastructural elements known as fibrillar centers, dense fibrillar and granular components. In addition, the nucleolus contains more than 700 proteins, pre-rRNA, rRNA, rDNA and several UsnoRNAs. To characterize nucleolar components a nanoscale, we use animal and plant cells processed as for transmission electron microscopy. Semithin sections were scanned with an atomic force microscope operating in contact mode. Results indicate that dense fibrillar and granular components display different nanometric surface profiles. We propose that nucleolar profiles may be used to analyzed nucleolar structure under different conditions.

New insights on cell-cell adhesion mediated by AMOG/b2 subunit of Na,K-ATPase

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AMOG/β2, the β2 isoform of Na+,K+-ATPase, has long been recognized as an important adhesion molecule on glia, facilitating interactions between astrocytes and neurons in the central nervous system (CNS). Despite this, the neuronal receptor that interacts with β2/AMOG remains unidentified. In this talk,

I will discuss the potential of β2/AMOG to form homophilic trans-dimers, drawing comparisons with the β1-subunit, a known homophilic adhesion molecule. Using computational modeling and in vivo assays, I will highlight key differences in the stability and behavior of β1 and β2 trans-dimers, particularly the distinct role of N-glycosylation in mediating these interactions. Our findings open new avenues for understanding β2/AMOG's role in neuron-glia communication, synapse formation, and CNS pathologies like glioblastoma. Ultimately, our work will shed light on the elusive binding partner of β2/AMOG and its broader implications for CNS adhesion dynamics.

The Role of the Flagellar Adhesion Zone in Trypanosoma cruzi Infectivity: New Insights from TcFLA-1BP and TcGP72 Knockouts

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Chagas disease, caused by the protozoan parasite *Trypanosoma cruzi* (Tc), is a major public health problem. The parasite exhibits a complex life cycle with different morphological stages. Tc cell organization is supported by the cytoskeleton, where the flagellar adhesion zone (FAZ) plays a central role in the adhesion of the flagellum to the cell body, which is essential for parasite motility and host cell invasion. Despite the structural characterization of the FAZ in T. cruzi, the protein composition and dynamics of this structure during the biological cycle remain poorly understood. In this study, we characterized the role of the FAZ during *T. cruzi* infection in mammalian host cells using knockout parasites for these proteins (TcFLA-1BP-/- and TcGP72-/-) generated by the CRISPR/Cas9 editing system. We observed that the absence of TcFLA-1BP led to heterogeneity in flagellar adhesion, with parasites presenting attached, partially detached, or completely detached flagella. On the other hand, TcGP72-/- parasites exhibited a more homogeneous phenotype, with the flagellum consistently detached. Functional analyses revealed that TcFLA-1BP knockout parasites had a lower infection capacity and longer generation and egress time, while TcGP72-/- showed a similar infection pattern to the control. Immunofluorescence (IF) analysis showed alterations in the distribution of the FAZ in knockout parasites, with intense labeling near the flagellar pocket in TcGP72-/-. However, TcFLA-1BP-/- showed labeling along the FAZ in the cell body even with the detached flagellum. Additionally, ultrastructural analysis revealed alterations in the organization of the kinetoplast and nucleus in knockout parasites in the trypomastigote forms. Our results demonstrate that TcFLA-1BP and TcGP72 are essential proteins for maintaining the integrity of the FAZ and play a crucial role in the infectivity and morphology of *T. cruzi*.

An up-close look at the larval development of red harvester ants

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Reproductive division of labor is a defining characteristic of eusocial animals such as ants, bees and wasps and is characterized by colonies consisting of non-reproductive workers and reproductive queens, which are distinguished from one another by a variety of morphological and molecular features. Caste differentiation during development is influenced by various molecular mechanisms, often in response to environmental and social cues, ultimately affecting the development of the individual within the colony. In some species, larval size and early developmental signals are crucial in channeling gene expression toward specific castes. In many species the precise moment of caste differentiation is unknown. An understanding of how larval morphology changes during development will help to focus efforts to determine how and when this differentiation occurs and will open the way for more detailed molecular and cellular studies on caste differentiation and determination. The red harvester ant, *Pogonomyrmex barbatus*, is widely distributed across northern Mexico and the southwestern United States. The mechanisms underlying caste differentiation in this species remain poorly understood, including the number of larval instars and the developmental cues that could influence the final

development of each individual. In this study, we characterized the total number of larval instars present in workers of *Pogonomyrmex barbatus* ants, identifying a total of 4 instars based on measurements of head capsule width from multiple individuals collected across 3 different laboratory colonies. Mean head capsule width was 253.7(±3.1SE)µm, 312.6(±5.0SE)µm, 401.6(±3.5SE) µm and 501.7(±1.9SE)µm for instars 1, 2, 3 and 4, respectively. The length of individuals increases linearly, although there is a large overlap in length between instars. We also conducted a morphological description of each instar using scanning electron microscopy (SEM) to highlight the morphological differences that distinguish one instar from another. With this information, our study contributes to the understanding of larval development and sets the basis for future descriptions of larvae of reproductive individuals that will be crucial for our studies on the molecular and epigenetic basis of caste differentiation in this ant species. This work, then, has the potential to contribute to a broader understanding of phenotypic plasticity and the development of specialized reproductive castes within eusocial species.

Role of Motors in Genome Folding

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Condensation of hundreds of mega-base-pair-long human chromosomes in a small nuclear volume is a spectacular biological phenomenon. This process is driven by the formation of chromosome loops. The ATP consuming motor, condensin, interacts with chromatin segments to actively extrude loops. I will present an analytically solvable model for loop extrusion (LE) [1], which was motivated by real-time imaging experiments. The theory suggests that condensin must undergo a large conformational change, induced by ATP binding, bringing distant parts of the motor to proximity. Simulations using a simple model confirm that the motor transitions between an open and a closed state in order to extrude loops by a scrunching mechanism, similar to that proposed in DNA bubble formation during bacterial transcription. Changes in the orientation of the motor domains are transmitted over ~50 nm, connecting the motor head and the hinge, thus providing an allosteric basis for LE. Extension of the theory to multiple motors [2], which was used to obtain mitotic structures will be presented.

References:

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Gastrointestinal Melioidosis and the role of the T6SS in *Burkholderia pseudomallei* pathogenesis

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Burkholderia pseudomallei (Bpm) has a complex lifestyle that culminates in cell-to-cell fusion and intracellular dissemination, regardless of the route of infection. Using a gastrointestinal melioidosis model of infection and a dual RNA-seq analysis, we identified BicA as a regulator of type 3 (T3SS) and type 6 secretion systems (T6SS) and a factor upregulated in a T6SS mutant. To fully dissect the role of BicA and T6Ss during systemic infection, we have used two macrophage cell lines paired with a pulmonary in vivo challenge murine model. Thusly, we investigated how the BicA and T6SS influences cell death, apoptosis, and inflammation within the macrophage and pulmonary models of infection. We found that ΔbicA has a distinct intracellular replication defect in both immortalized and primary macrophages, which is linked with the lack of cell-to-cell dissemination as well as a defect in T3SS expression. The in vitro phenotype translated in vivo as ΔbicA was attenuated in a pulmonary model of infection, demonstrating a distinct macrophage activation profile and a lack of pathological features present in the wild type. Further, the T6SS is responsible for exacerbating apoptotic cell death during infection in both macrophages and the lungs of infected mice. Overall, these results highlight the role of BicA and T6SS in regulating intracellular virulence, significantly impacting on host response and Bpm pathogenesis.

Mitochondrial dysfunction in ovary of offspring of mice with polycystic ovary syndrome

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Mitochondrial dysfunction is related to various diseases, such as polycystic ovary syndrome (PCOS), characterized by hyperandrogenism and ovulatory dysfunction. Women with PCOS as well as animal models present mitochondrial alterations in dynamics, biogenesis, and respiratory capacity. Since mitochondria are maternally inherited, the aim was to evaluate reproductive and morphological mitochondrial changes in the ovaries of a mouse model of PCOS and the offspring. The PCOS model was created using 25-day-old female Balb/c mice treated with dehydroepiandrosterone for 20 days. The mice were mated with healthy males. Mice with PCOS (M-PCOS) and adult offspring (O-PCOS) were employed for the study. The estrous cycle was evaluated for two weeks. During this time, M-PCOS group remained in metaestrous and O-PCOS in the diestrus stage, while the control groups presented a normal estrous cycle. Besides, ovaries were obtained and processed for optical and transmission electron microscopy. Morphological and ultrastructural analysis of the M-PCOS group showed similar characteristics to the control group. However, O-PCOS showed a decrease in primordial and primary follicles, but a greater number of secondary and antral follicles, along with a higher number of atretic follicles. Additionally, no healthy oocytes were observed. Ultrastructural characteristics of granulosa cells (GC) and oocytes in the offspring of O-PCOS group included endoplasmic reticulum and mitochondrial swelling, in addition to loss of mitochondrial cristae. Moreover, immunolocalization of CLPP, an indicator of the mitochondrial unfolded protein response, was positive in oocytes and GC of O-PCOS. In conclusion, maternal PCOS is associated with mitochondrial dysfunction in GC and oocytes and loss of estrous cycle in the offspring.

Morphological and metabolic characteristics in the pancreas and liver of male offspring of a polycystic ovary syndrome mouse model

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Hyperandrogenism is one of the main features in women with polycystic ovary syndrome (PCOS), with a negative impact throughout a woman's life, including pregnancy. In prenatal models of maternal hyperandrogenism, changes in the weight and size of the pancreas have been reported, as well as dyslipidemia. However, the effects of hyperandrogenism on the metabolism of male offspring are not fully understood. Therefore, this work aims to evaluate pancreas morphology, liver ultrastructure, hepatic lipid, and gluconeogenic enzymes expression in the offspring of a postnatal mouse model of PCOS. In this study we administered dehydroepiandrosterone (DHEA) to Balb/c mice for 20 consecutive days, subsequently, and the mice mated with control males. One-month-old and adult offspring of the group treated with DHEA (O-DHEA) presented insulin resistance and increased hepatic lipid accumulation. Furthermore, the O-DHEA group showed higher expression of fatty acid synthase and gluconeogenic enzymes, pyruvate carboxylase and phosphoenolpyruvate carboxykinase. In addition, endoplasmic reticulum and mitochondrial swelling occurred in hepatocytes. Morphological analysis of the pancreas showed an increase in islet area in the O-DHEA group. Immunofluorescence showed loss of the pancreatic architecture with a higher percentage of cells positive for insulin and glucagon. These results suggest a role of maternal PCOS in the offspring pancreatic function that trigger insulin resistance, impairs hepatic metabolism and induces mitochondrial and endoplasmic reticulum stress.

Cryo-EM in Enzymology and Conformational Dynamics

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In addition to structures of large protein complexes, cryo-EM is also breaking new grounds in the fields of enzymology and protein dynamics, particularly in the identification of intermediates of enzymatic catalysis (1). Recently Nature published a News Feature with a heading “Welcome to the age of protein cinematography” (2). This lecture will illustrate these applications of cryo-EM with two stories: First is the use of cryo-EM to dissect intermediate structures of temperature-dependent conformational changes of Ketol-acid reductoisomerase (KARI) from the thermophilic archaeon Sulfolobus solfataricus (Sso) (3). Secondly, we used cryo-EM to show that African swine fever virus type 2 topoisomerase (AsfvTop2) pre-exists in six conformers that mimic the reaction intermediates with substrates (4). References: 1. “Enzymology and Dynamics by Cryo-EM”. Ming-Daw Tsai, Wen-Jin Wu, and Meng-Chiao Ho. Annu. Rev. Biophys. 51,19–38 (2022). 2. “Welcome to the age of protein cinematography” Nature 627, 480 (2024) 3. “Temperature-resolved Cryo-EM Uncovers Structural Bases of Temperature-Dependent Enzyme Functions.” Chen CY, Chang YC, Lin BL, Huang CH, Tsai MD. J. Am. Chem. Soc. 141, 19983-19987 (2019). doi: 10.1021/jacs.9b10687. 4. “A unified view on enzyme catalysis by cryo-EM study of a DNA topoisomerase”. Chiung-Wen Mary Chang, Shun-Chang Wang, Chun-Hsiung Wang, Allan H. Pang, Cheng-Han Yang, Yao-Kai Chang, Wen-Jin Wu, Ming-Daw Tsai. Communications Chemistry (2024) 7:45.

A genetic platform for functional profiling and visualization of the sphingolipid metabolic network

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Sphingolipids (SPLs) govern diverse cellular processes, and their dysregulation underlies numerous human diseases. Despite extensive biochemical characterizations, understanding the orchestration of the SPL metabolic network within living organisms remains challenging. To systemically investigate SPL metabolism, we established a versatile genetic platform that included CRISPR-engineered reporters of 52 SPL regulators, recapitulating endogenous gene activity and protein distribution. The platform also allows conditional protein degradation for functional assays. Additionally, we developed a biosensor for visualizing sphingomyelin/ceramide phosphoethanolamine (SM/CerPE) dynamics in vivo. Using this platform, we revealed the spatiotemporal heterogeneity of the SPL metabolic network in the brain, even within the same branch of the SPL regulatory pathways. For example, we identified distinct cellular requirements for SMases, particularly the microcephaly-associated CG6962/SMPD4. Furthermore, neurons and glia degrade SM/CerPE at specific organelles with coordinated lipoprotein-mediated intercellular transfer. This platform enables comprehensive investigation of SPL metabolism in vivo, offering new avenues for deciphering its pathophysiological mechanisms.

Aging related epigenetic derepression of LINE-1 retrotransposon in Idiopathic Pulmonary Fibrosis

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Idiopathic Pulmonary Fibrosis (IPF) is a chronic, progressive, irreversible and usually lethal interstitial lung disease, of unknown etiology and very limited therapeutic options. The main risk factor and driving force of this disease is aging. Most of the hallmarks of accelerated aging have been identified in IPF lungs, including cellular senescence and aging-related epigenetic reprogramming involving altered methylation patterns and histone modifications. Transposable elements are nucleic acid sequences that can move from one place to another in the genome, for example, Long Interspersed Element-1 (LINE-1) is the only active autonomous retrotransposon (constitutes 17% of the human genome) and its activity can cause mutagenesis, DNA damage and genomic instability. During aging, the epigenetic mechanisms that keep retrotransposons silenced become less efficient which may suggest a previously unreported LINE-1 activation in IPF. The aim of this study was to study the epigenetic modifications involved in the derepression of LINE-1 retrotransposon in IPF. Methods: LINE-1 ORF1 protein was examined by immunohistochemistry on human IPF lung tissue sections. Relative LINE-1 expression was assessed by

RT-qPCR in IPF lung fibroblasts. Methylation analysis was carried out via MS-PCR using bisulfite-converted DNA from IPF and control lung fibroblasts. Results: We observed LINE-1 mainly in the epithelium of human IPF lung tissue with a lesser presence in fibroblast foci, while absent in controls. LINE-1 expression was increased in IPF lung fibroblasts compared to controls. MS-PCR revealed LINE-1 to be unmethylated in IPF. Conclusion: An aging-related hypomethylation allows for the derepression of LINE-1 in IPF.

Neuronal differentiation of pluripotent stem cells in monolayer and organoids

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Mammalian pluripotent stem cells can be induced to differentiate to several neuronal subtypes present in the central nervous system. We have used normal and transgenic embryonic stem cells to produce spinal motor and dopamine neurons that express appropriate trajectories of neural induction, as well as markers of maturity. Initial protocols used terminal differentiation on monolayer to produce dopamine neurons that, after grafting, can improve the behavioral and biochemical alterations caused by lesioning the endogenous dopaminergic neurons. By reprogramming fibroblasts of Parkinson disease Mexican patients, we have generated induced pluripotent stem cells and confirmed that dopamine neurons can be obtained from these cells. Using midbrain organoids, we have started to study dopamine neuron function with cells differentiated from normal and patient´s pluripotent cells. Also, we have produced organoids of neural origin containing spinal motor neurons that were placed together with muscle cells aggregates to form assembloids, to study axonal regeneration.

Neocartilage by tissue engineering. Potential applications for auricular reconstruction

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Tissue engineering has emerged as a promising strategy for the regeneration and repair of auricular cartilage, as it is an avascular tissue with limited regenerative capacity. In cases of congenital deformities such as microtia or traumatic injuries, conventional auricular reconstruction faces significant challenges. Tissue engineering aimed at forming elastic neocartilage combines the use of autologous cells, biomimetic scaffolds, and biochemical signals, offering a viable alternative for clinical applications. This approach is based on a deep understanding of cellular biology, focusing on how cells and growth factors interact to form functional tissue. Neocartilage engineering combines advances in cellular biology and biomimetic materials, offering an innovative platform for auricular reconstruction. Although challenges such as vascularization and integration with surrounding tissues persist, recent advances suggest that this technology holds great clinical potential for the auricular reconstruction of both acquired and congenital auricular defects. One of the key aspects for the formation of neocartilage is the selection of the source cells. Although autologous auricular chondrocytes are specialized, they present limitations in terms of proliferation. Patients with unilateral microtia are a viable option, while in cases of anotia, cell availability remains a significant limitation. For this reason, mesenchymal stem cells (MSCs) have gained prominence due to their ability to differentiate into chondrocytes under appropriate stimuli. MSCs are easily obtained from sources such as bone marrow or adipose tissue, and with proper signaling through growth factors, they can generate cartilage that mimics native auricular tissue. The cellular microenvironment is essential for inducing chondrogenic differentiation and extracellular matrix (ECM) synthesis. Growth factors such as TGF-β and BMP-2 are fundamental in guiding MSCs toward a chondrogenic lineage. Additionally, creating a low-oxygen environment, like the native cartilage setting, and applying mechanical stimuli promote the production of type II collagen, proteoglycans, and elastin—key components for cartilage structure and functionality.

Characterization of transmigration and effect of NK cell geometry

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Characterization of transmigration and effect of NK cell geometry NK (natural killer) cells are a type of lymphocyte belonging to the innate immune system, whose main function is to identify and destroy virus-infected cells and tumor cells. NK cells play a crucial role in immune surveillance against cancer and viral infections. The study of NK cells is complicated by the fact that murine models do not always faithfully replicate the behavior of human NK cells, in vivo monitoring and in two-dimensional cell culture it is difficult to replicate physiological environments. The development of experimental platforms, such as microfluidic devices, that simulate cellular interactions in a controlled environment is essential to study NK cell biology under conditions as close as possible to physiological ones. To understand the activation and transmigration of these cells, two microfluidic devices were designed in this study. In the first, fibronectin microstamping was performed to fix and organize MCF7 cells, with the aim of altering their morphology and clustering. This approach allowed studying the interaction between NK-92 cells and MCF7 cells, measuring the activation of the former, through the intracellular concentration of calcium. To verify the microstamping patterns with fibronectin, tests were performed using GFP protein (Green Fluorescent Protein), which made it possible to determine the optimal parameters of pressure and stamping time. Once these values were established, fibronectin patterns were created in the microfluidic device to adhere MCF7 cells in a controlled manner. In the second device, an environment was designed to assess NK cell migration. This device includes a vertical barrier of collagen membrane and endothelial cells that simulates a capillary wall. Additionally, a recirculation system was integrated to replicate a physiological environment closer to that faced by NK cells in the human body. Migration was assessed by measuring both the speed and distance traveled by NK-92 cells in the presence of MCF7 cells. Preliminary results from microstamping with GFP protein allowed optimization of pressure and stamping time values to obtain clear and reproducible fibronectin patterns on the microfluidic device. By using these patterns to adhere MCF7 cells, it was observed that modifications in the morphology and clustering of these cells influence the intracellular concentration of calcium in NK-92 cells. As for the device designed to study migration, it was observed that NK-92 cells were able to migrate across the endothelial barrier, simulating their behavior in a realistic vascular environment. The integration of the recirculation system allowed for a more mimetic environment that contributed to the accurate assessment of NK cell migration. By assessing how cancer cell morphology and clustering affects NK cell migration and activation, this research may influence the development of new targeted immunotherapies, in the development of microfluidic devices to assess the effectiveness of different drugs.

Phase separation in the control of cilia formation

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The primary cilium serves a crucial function in detecting and transmitting extracellular signals within cells. Initiation of ciliogenesis hinges on the recruitment of Tau-tubulin kinase 2 (TTBK2) via CEP164 at centriole distal appendages (DAs). However, the precise mechanism of the CEP164/TTBK2 interaction at DAs during ciliogenesis remains unclear. In this study, we unveil that CEP164 features a long intrinsically disordered region (IDR) and forms dynamic condensates with TTBK2 through liquid-liquid phase separation (LLPS). Additionally, our investigation demonstrates that CEP164 undergoes LLPS with TTBK2 through multivalent electrostatic interactions. This interaction is a pivotal determinant for the recruitment of TTBK2 to DAs, thereby kickstarting the process of cilia formation. Therefore, our findings provide valuable insights into the molecular regulation of CEP164/TTBK2 interaction at DAs and highlight the pivotal role of LLPS in facilitating cilia formation.

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Poster no. 41(abstract below, pag. 95)

***Actinobacillus seminis* testosterone receptor identification**

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Poster no. 42 (abstract below, pages 95-96)

NaTrxh extensions: key mediators of cellular localization and specificity towards S-RNase in *Nicotiana alata*

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Poster no. 43 (abstract below, pag. 96)

The parabasal filaments of *Trichomonas vaginalis*: a new filament and observations using ultra-high resolution Scanning Electron Microscopy

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Poster no. 44 (abstract below, pages 98-97)

Analysis of DAPK1 Methylation in Apoptosis Resistance in Idiopathic Pulmonary Fibrosis derived Fibroblasts

Mariana Río de la Loza 1 , David A. Lizcano 1 , Remedios Ramirez 1 , Uriel Camacho-Silverio 1 , Tania Valdivia-Herrera 1 , Fernanda Toscano-Marquez 2 , Marco Espina-Ordoñez 2 , Moises Selman 2 , Annie Pardo 1 , Yair Romero 1.
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Actinobacillus seminis testosterone receptor identification

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Actinobacillus seminis is a commensal opportunistic pathogen of rams and other ruminants that causes epididymitis in males and abortions in female ovines. This microorganism ascends from prepuce to epididymis and testis when males attain sexual maturity. Low fertility, infertility, and abortions are the main damage of the diseases this microorganism causes. A. seminis pathogenicity mechanisms are unknown, and virulence factors are scarcely known. However, it is known that when this microorganism is grown with testosterone, its growth duplicates; it expresses putative adhesins, biofilm production increases, and in preformed biofilm, dispersion is induced. The present work describes the purification and identification of putative testosterone protein receptors. Protein receptors were isolated from total cell extracts of *A. seminis* grown overnight in the presence of 5 ng/ml testosterone. Samples were added to 24 well plates in which testosterone was previously coupled. Proteins adhered to testosterone were released with SDS 1%. A cytoplasmic protein was purified by passing samples through Sepharose-CLB6 column coupled with triazine orange dye and eluted with KCl 150 mM, Na2HPO4 0.2 M, NaCl 1 M, TRIS 20 mM buffer. Purified proteins were analyzed by spectrometer masses assay, and peptides presented identity with OmpF porin and LsrB (autoinducer-2 receptor) protein. By molecular docking in silico assay, it was determined that testosterone-OmpF and -LsrB present a 59% and 50% interaction, respectively. Those proteins are scarcely expressed or not in the absence of hormones, and their expression is inhibited in the presence of 2(5H)-furanone, a quorum-sensing inhibitor that can bind to different two-component systems participating in quorum-sensing signaling. The presence of furanone also dimidiases the growth, adhesins, and protease expression to levels of cultures without testosterone. Identifying A. seminis host molecule receptors could pave the knowledge in microbial pathogenesis.

NaTrxh extensions: key mediators of cellular localization and specificity towards S-RNase in Nicotiana alata

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Self-incompatibility (SI) is a genetic mechanism in flowering plants that prevents inbreeding by distinguishing between self- (incompatible) and non-self-pollen (compatible). SI is controlled by the multiallelic S locus, which encodes S determinants in the pistil and pollen. In *Nicotiana alata*, the female S determinant, S-RNase, a pistil-specific ribonuclease, enters the pollen tube (PT) and degrades RNA in incompatible PTs, inhibiting them. Other genes, such as NaTrxh (*N. alata* thioredoxin type h), are also crucial for self-pollen rejection. NaTrxh localizes to the stylar extracellular matrix, interacts with S-RNase and specifically reduces one of its four disulfide bonds. This reduction significantly increases S-RNase ribonuclease activity, which is essential for self-pollen rejection. NaTrxh has N- and C-terminal extensions with distinctive roles. The N-terminal extension (27 residues) contains two motifs –Na and Nb–. Both are required for the NaTrxh-S-RNase interaction allowing S-RNase reduction, while the Nb motif (A17-P27) also acts as a secretion signal. The C-extension (E136-Q152) is important in stabilizing the interaction between both proteins but has no effect on the reduction of S-RNase. Regarding its extracellular localization, the Nb motif exhibits features distinct from typical signal peptides, suggesting that NaTrxh secretion is SRP-independent. Nb directed secretion involves a post-translational incorporation to the ER, indicating that it acts as an ER transit peptide rather than a typical signal peptide. This hypothesis is reinforced by analysis of similar Trxs h (subgroup 2), which suggests the possibility that variations within Nb sequence might influence cellular localization, such as mitochondrial targeting. To understand how the N- and C-terminal extensions participate in the NaTrxh specificity to S-RNase and

its correct orientation for specifically reduce the single disulfide bond target, we are working on achieving the structure of the NaTrx-S-RNase complex. CONAHCYT postgraduate fellowship; PAPIIT IN207823.

The parabasal filaments of Trichomonas vaginalis: a new filament and observations using ultra-high-resolution Scanning Electron Microscopy

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Trichomonas vaginalis is the etiologic agent of trichomoniasis, the most common nonviral sexually transmitted infection worldwide, with an estimated 260 million new cases annually. T. vaginalis contains a complex and elaborate cytoskeleton constituting the mastigont system, which is mainly formed by several proteinaceous structures associated with basal bodies, the pelta-axostylar complex made of microtubules, and striated filaments named the costa and the parabasal filaments (PBs). Although the structural organization of trichomonad cytoskeletons has been analyzed using several techniques, observation using a new generation of scanning electron microscopes with a resolution of below 1 nm has allowed more detailed visualization of the three-dimensional organization of the mastigont system. In this study, we have investigated the cytoskeleton of *T. vaginalis* using a diverse range of scanning probe microscopy techniques, which were complemented by electron tomography and Fast-Fourier methods. This multi-modal approach has allowed us to characterize an unknown parabasal filament and reveal the ultrastructure of other striated fibers that have not been published before. Here, we show the differences in origin, striation pattern, size, localization, and additional details of the PBs, thus improving the knowledge of the cell biology of this parasite. The parabasal filaments of *Trichomonas vaginalis*: a new filament and observations using ultra-high-resolution Scanning Electron Microscopy.

Analysis of DAPK1 Methylation in Apoptosis Resistance in Idiopathic Pulmonary Fibrosis derived Fibroblasts

Mariana Río de la Loza1, David A. Lizcano1, Remedios Ramirez1, Uriel Camacho-Silverio1, Tania Valdivia-Herrera1, Fernanda Toscano-Marquez2, Marco Espina-Ordoñez2, Moises Selman2, Annie Pardo1, Yair Romero1.
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Introduction: Idiopathic pulmonary fibrosis (IPF) is a progressive, irreversible, and fatal disease characterized by the aberrant activation of epithelial cells. This activation promotes the formation of fibroblast/myofibroblast foci that uncontrollably synthesize extracellular matrix components, reducing elasticity, altering lung architecture, and ultimately impairing gas exchange. It is known that IPF fibroblasts are resistant to apoptosis, with molecular mechanisms of evasion or resistance that have not yet been fully elucidated. This resistance leads to their accumulation and persistent activation, turning regeneration into progressive fibrosis. Specifically, these changes may result from epigenetic alterations acquired with aging, preventing proper tissue regeneration. Therefore, it is of great interest to investigate what prevents these cells from entering the apoptotic stage. The aim of this project is to analyze the relationship between the expression and methylation of genes that promote programmed cell death (apoptosis), specifically the "DAPK1" gene, in fibroblasts from patients with IPF.
Methods: For this analysis, primary cell cultures derived from patients with IPF and controls were used, following bioethical protocols. DNA and RNA were extracted and purified using the TRIzol method. The obtained DNA was subjected to RT-qPCR with specific primers, and the amplified regions underwent bisulfite treatment, revealing methylation with MS-PCR.
Results: The fibrotic cell lines showed significant differences in DAPK1 expression compared to controls. Additionally, methylation supported the expression results, as higher methylation was observed in IPF.

Similarly, applying the genetic or pharmacological inhibitor of DAPK1 in control cells inhibited cellular apoptosis.
Conclusions: The presence of DAPK1 gene methylation is a strong inhibitor of apoptosis in fibrotic cells, promoting disease progression.

The locus of enterocyte effacement of enteropathogenic E. coli: a unique toolbox for virulence gene regulation

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Enteropathogenic *Escherichia coli* (EPEC) and enterohemorrhagic E. coli (EHEC) are major causes of infectious diarrhea in humans, characterized by a distinct histopathology known as the attaching and effacing (A/E) lesion. EPEC primarily causes severe and persistent diarrhea in children under two years old, especially in developing countries, while EHEC can lead to bloody diarrhea and hemolytic uremic syndrome, which can be fatal. The mouse pathogen *Citrobacter rodentium*, which shares with EPEC and EHEC the genes responsible for generating the A/E lesion, has facilitated studies of the mechanisms underlying infections caused by A/E pathogens. The A/E lesion is characterized by the localized elimination of the enterocyte microvilli and the formation of actin rich, pedestal-like structures beneath the adherent bacteria. The ability to form A/E lesions is conferred by proteins encoded on a pathogenicity island known as the LEE ("Locus of Enterocyte Effacement"). The expression of LEE genes is regulated by a complex network involving specific regulators exclusive to A/E pathogens, encoded within the LEE. These include Ler and GrlA, which act as positive regulators, and GrlR, a negative regulator. Additionally, global regulators such as H-NS and IHF modulate LEE expression in response to environmental and physiological cues encountered inside and outside the host. Ler, encoded by the first gene of the LEE1 operon, serves as the master regulator of LEE genes and thus the virulence of A/E pathogens. Ler is not a typical activator, as it acts as a derepressor by destabilizing nucleorepressor complexes formed by H-NS on LEE-controlled genes. The LEE also contains the grlRA operon encoding two additional regulatory proteins. Under inducing conditions, GrlA upregulates Ler expression, which in turn enhances grlRA operon expression, creating a positive feedforward loop that amplifies LEE gene expression. In contrast, depending on the growth conditions, GrlR can repress LEE gene expression by interacting with the DNA-binding domain of GrlA. Recent evidence indicates that the regulatory mechanisms of the LEE, mediated by GrlA and GrlR, are more intricate than previously understood. Here, we discuss new findings that propose promising directions for future research.