VIII Congreso de Especies Reactivas del Oxígeno en Biología y Medicina

Proceedings of the VIII Congress of Reactive Oxygen Species in Biology and Medicine

IX Workshop on Comparative Aspects of Oxidative Stress in Biological Systems

ORGANIZING COMMITTEE

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Sept 26-29, 2023, Tlaxcala, Tlaxcala. México

Welcome

Dear participants of the Congress on Reactive Oxygen Species in Biology and Medicine. It is a pleasure to welcome you to this important event that brings together experts and scholars in the field.

The significance of this congress lies in its crucial role for the scientific community, as reactive oxygen species have become a multidisciplinary field in constant evolution.

With great pleasure, we have observed how our field has experienced significant growth, becoming a manifestation of progress in this domain and a forum for sharing ideas, collaborating, and enriching our study perspectives.

Over the next few days, you will have the opportunity to hear from internationally renowned experts, present your latest research, and establish valuable connections with colleagues. We hope this congress will be an enriching experience for all of you and that your contributions will further advance science.

Your contributions and participation are essential to the success of this event.

We wish you all a productive and enriching congress!

Welcome to the Congress on Reactive Oxygen Species in Biology and Medicine!

Sincerely,

Organizing Committee

Acknowledgments

Dear representatives of the institutions and sponsors, esteemed researchers, postgraduate and undergraduate students,

It is an honor and a privilege to address all of you now to convey our most sincere gratitude for your support and dedication to the realization of the VIII Congress on Reactive Oxygen Species in Biology and Medicine. This event has not only been possible due to the support of the institutions and sponsors. Still, it has also been greatly enriched by the active participation and enthusiasm of all attendees, from esteemed researchers to postgraduate and undergraduate students.

We wish to take a moment to highlight and extend our gratitude to the following institutions and sponsors for their contribution:

***Sociedad Mexicana de Bioquímica**: We deeply value and appreciate your expertise, involvement, and dedication to promoting biochemistry in Mexico. Your support has been instrumental and added significant value to our congress.*

***Consejo Nacional de Humanidades, Ciencias y Tecnologías (CONAHCYT):** Your generosity and financial support have been crucial for this congress's materialization and promotion of scientific research in our country.*

***Society for Free Radical Biology and Medicine (SFRBM):** We thank you for your consistent support and commitment to advancing research in this field.*

***Universidad Autónoma Metropolitana -Iztapalapa (UAM-Iztapalapa):** Your support has elevated the quality and prestige of our event.*

***Universidad Autónoma de Tlaxcala (UATx):** Our most profound gratitude for providing the space and resources essential for organizing this event. Your collaboration and willingness have been invaluable.*

Centro INAH Tlaxcala and Museo Regional de Tlaxcala: *Thank you for enriching our experience with cultural and artistic aspects that have added a unique value to our congress.*
Centro Tlaxcala de Biología de la Conducta. *Universidad Autónoma de Tlaxcala (UATx): We appreciate your participation in our congress.*
La colmena. Centro de tecnologías creativas Grace Quintanilla (UATx): *Your contribution has significantly enhanced our experience.*
Sponsors: Cipquim, BiAssys, Medix, LABEQUIM, Uniparts, MASTER LEAV, Crissof. *Your generosity and ongoing support have made this congress a reality.*

Beyond institutional collaboration and financial backing, we express our profound gratitude to all attendees, who, as researchers, postgraduate, and undergraduate students, have enriched this congress with their knowledge, enthusiasm, and passion for scientific research. Your presentations, discussions, and active participation are essential in cultivating a collaborative and enriching learning atmosphere.

This congress represents a venue for knowledge exchange and a testament to the significance of the scientific community and collaboration across generations. Your contributions have enabled the realization of an event that we are confident will leave a lasting impact on the advancement of research in Reactive Oxygen Species in Biology and Medicine.

We anticipate your involvement in future events and look forward to continuing our collaboration to advance scientific knowledge.

With gratitude and appreciation,

Congress Organizing Committee

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VIII Congreso de Especies Reactivas del Oxígeno en Biología y Medicina

Program

Sept 26-29, 2023, Tlaxcala, Tlaxcala. México

PROGRAM

Tuesday, September 26th, 2023	
13:00-17:00	**REGISTRATION –** Lobby del Auditorio anexo de rectoría "Luis Carbajal Espino"
16:30-17:00	**OPENING CEREMONY** Rector UATx Doctor Serafín Ortiz Ortiz, Secretaria Académica UATx Dra. Margarita Martínez Gómez, and Comitte organizing.
17:00-18:30	**CULTURAL CONFERENCE** Cultural Conference and Guided Tour in: *El Conjunto Conventual Franciscano de Nuestra Señora de la Asunción de Tlaxcala* (UNESCO World Heritage Site of Humanity). **Lic. José Vicente de la Rosa Herrera,** *Director del Centro INAH Tlaxcala, y* **Diego Martín Medrano**, *Director del Museo Regional de Tlaxcala. Investigadores del Instituto Nacional de Antropología e Historia (INAH).*
18:30-20:00	**WELCOME COCKTAIL** **Claustro Alto del Museo Regional de Tlaxcala**

Wednesday, September 27th, 2023	
7:30-9:00	**IX WORKSHOP ON COMPARATIVE ASPECTS OF OXIDATIVE STRESS IN BIOLOGICAL SYSTEMS** *Los microarreglos en el estudio del estado redox* **Instructora QFB Alejandra Idan Valencia Cruz** *Unidad de Microarreglos, Instituto Nacional de Medicina Genómica (INMEGEN), México.*

9:00-10:00	**BREAKFAST**
	ORAL PRESENTATIONS **Students selected to compete for the award for the best oral presentation.**
10:00-10:15	*NADPH oxidases and dynamin are required for epiboly in zebrafish* **Arlen Ramírez Corona** *Universidad Nacional Autónoma de México (UNAM), México.*
10:15-10:30	*Differential effect of exposure to phthalates in skeletal muscle cells from terrestrial and marine mammals* **Elizabeth Brassea Pérez** *Centro de Investigaciones Biológicas Del Noroeste S.C. (CIBNOR), México.*

10:30-10:45	*He is a ten, but…: prooxidants enhanced attractiveness in mealworm beetles at the cost of diminished antioxidants in ejaculate* **María del Sagrario Cordero Molina** *Universidad Nacional Autónoma de México (UNAM), México.*
10:45-11:00	*Impact of oxidative stress on the prostate transcriptome* **Kevin Samael Olascoaga Del Ángel** *Universidad Autónoma Metropolitana, Unidad Iztapalapa (UAM-I), México.*
11:00-12:00	**KEYNOTE LECTURE 1** *Thiol Redox Codes of Cellular Signaling* **Dr. Francisco R. M. Laurindo** *Heart Institute (INCOR), University of São Paulo,* *São Paulo, Brasil.* ***President of the Society for Redox Biology And Medicine (SfRBM).***
12:00-12:30	**COFFEE BREAK**
	SYMPOSIUM 1 **Diseases associated with oxidative stress**

12:30-13:00	*Oxidative stress and redox regulation, early stellar in chronic liver diseases* **Dr. Jaime Arellanes Robledo** *Laboratorio Enfermedades Hepáticas Crónicas, Instituto Nacional de Medicina Genómica (INMEGEN), México.*
13:00-13:30	*Copper redox chemistry in neurodegenerative diseases* **Dra. Liliana Quintanar Vera** *Departamento de Química, Centro de Investigación y de Estudios Avanzados (CINVESTAV), México.*
13:30-14:00	*Role of oxidative stress from exposure to xenobiotics in the development of nephropathies* **Dr. Oliver Christophe Barbier** *Laboratorio de Toxicología Renal, Centro de Investigación y de Estudios Avanzados (CINVESTAV), México.* **Remote conference via ZOOM**

14:00-16:00	**LUNCH**
16:00-17:00	**KEYNOTE LECTURE 2** *Redox signaling in breast cancer: from basic science to drug discovery* **Dra. Carola Neumann** *University of Pittsburgh, Pittsburgh, PA. EUA.* ***Vice-president of the Society For Redox Biology And Medicine (SfRBM).***
17:00-18:00	**WOMEN IN SCIENCE ROUND TABLE** **Conversatorio:** *Ciencia en Femenino y el poder de las mentorías* Moderadora: Dra. Norma Edith López Díaz-Guerrero *Universidad Autónoma Metropolitana, Unidad Iztapalapa (UAM-I), México.* **Dra. Margarita Martínez Gómez** *Investigadora del Instituto de Investigaciones Biomedicas de la UNAM.* *Secretaria Académica, Universidad Autónoma de Tlaxcala (UATx), México.* **Dra. Arely Anaya Hernández**

	Centro de Investigación en Genética y Ambiente, Universidad Autónoma de Tlaxcala, (UATx), México. **Dra. Verónica Reyes Meza** *Centro Tlaxcala de Biología de la Conducta. Universidad Autónoma de Tlaxcala, (UATx), México.*
18:00-20:00	**POSTERS**

Thursday, September 28th, 2023	
7:30-9:00	**IX WORKSHOP ON COMPARATIVE ASPECTS OF OXIDATIVE STRESS IN BIOLOGICAL SYSTEMS** *Medición de copias mitocondriales y longitud telomérica* **Instructor Dr. Mauricio Guillén** *Laboratorio de Comportamiento Animal, Instituto de Ecología, Universidad Nacional Autónoma de México (UNAM), México.*
9:00-10:00	**BREAKFAST**

	ORAL PRESENTATIONS **Students selected to compete for the award for the best oral presentation.**
10:00-10:15	*Quantification of plasma polysulfides in mother-neonate pairs with and without preeclampsia diagnosis* **Ricardo Antonio Rojas Pérez** *Universidad Nacional Autónoma de México (UNAM), México.*
10:15-10:30	*Effects of particulate matter PM2.5 on the antioxidant status in A459 lung carcinoma epithelial cells: glutathione S-transferase and NAD(P)H: Quinone Oxidoreductase* **Jesús Valencia Cervantes** *Instituto Nacional de Enfermedades Respiratorias Ismael Cosio Villegas (INER), México.*

10:30-10:45	*Role of reactive oxygen species in breast and ovarian cancer* **Fabiola Lilí Sarmiento Salinas** *Centro de Investigación Biomédica de Oriente (CIBIOR), México.*
10:45-11:00	*Antioxidant activity in Senna mutiglandulosa seedlings exposed to Li, Ag, and Cu: anin vitro study* **Dolores Itzel López Nicolás** *Universidad Nacional Autónoma de México (UNAM), México.*
11:00-12:00	**KEYNOTE LECTURE 3** *Antioxidant capacity of phytochemicals from Mexican Semidesert plants and their biological activities* **Dr. Raúl Rodríguez Herrera.** *Facultad de Ciencias Químicas, Universidad Autónoma de Coahuila (UadeC), Coahuila, México.*
12:00-12:30	**COFFEE BREAK**
	SYMPOSIUM 2 **Effect of oxidative stress on fungi and plants**
12:30-13:00	*The role of the respirasome in the control of mitochondrial production of ROS in Ustilago maydis* **Dr. Óscar Flores Herrera** *Facultad de Medicina, Universidad Nacional Autónoma de México (UNAM), México.*

13:00-13:30	*Caenorhabditis elegans* a model to study antioxidant activity of native tomatoes **Dr. Darío Gómez Linton** *Departamento Ciencias de la Salud, Universidad Autónoma Metropolitana, Unidad Iztapalapa (UAM-I), México.*
13:30-14:00	*The dynamics of ROS during polarized growth and its role as a signal molecule* **Dr. Luis Cárdenas Torres** *Departamento Biología Molecular de Plantas, Instituto de Biotecnología (IBT), UNAM, México.*
14:00-16:00	**LUNCH**

16:00-17:00	**KEYNOTE LECTURE 4** *Design of multifunctional antioxidants with potential application in Parkinson's and Alzheimer's diseases* **Dra. Annia Galano Jiménez** *Depto. Química, Universidad Autónoma Metroplitana, Unidad Iztapalapa (UAM-I), México.*
17:00-18:00	**Business session**
18:00-20:00	**POSTERS**

Friday, September 29th, 2023	
	IX WORKSHOP ON COMPARATIVE ASPECTS OF OXIDATIVE STRESS IN BIOLOGICAL SYSTEMS **TALLER TÉCNICAS Y EQUIPOS** **Impartidas por tres Patrocinadores**
7:30-8:00	*Aplicaciones del digital droplet PCR* **CRISSOF DE MÉXICO**
8:00-8:30	*Dale un glow-up a tus ensayos con PROMEGA Glomax Systems: ROS (Reactive Oxygen Species)* **UNIPARTS**
8:30-9:00	*La fuerza de la importación para la consolidación de nuestros científicos* **CIPQUIM**
9:00-10:00	**BREAKFAST**
	SYMPOSIUM 3 **Oxidative stress generating toxicants**
10:00-10:30	*Cadmium-mediated oxidative effect of smoking on musculoskeletal diseases* **Dra. Karina Martínez Flores** *Laboratorio de Líquido Sinovial, Instituto Nacional de Rehabilitación (INR), México.*
10:30-11:00	*Ozone environmental pollution and neurodegenerative disease* **Dra. Selva Lucía Rivas Arancibia**

	Laboratorio estrés oxidativo y plasticidad cerebral, Facultad de Medicina, Universidad Nacional Autónoma de México (UNAM), México.
11:00-11:30	*Antioxidant effect of low-intensity exercise in a rat model of osteosarcopenic obesity* **Dr. Armando Luna López** *Departamento de Biología del Envejecimiento, Instituto Nacional de Geriatría (INGER), México.*
11:30-12:30	**KEYNOTE LECTURE 5** *The oxidative cost of reproduction as a mechanism implicated in evolutionary theoriesof aging: is there strong evidence?* **Dr. Carlos Alonso-Álvarez** *Museo Nacional de Ciencias Naturales de Madrid (MNCN), Consejo Superior de Investigaciones Científicas (CSIC), España.*
12:30-13:00	**COFFEE BREAK**
	SYMPOSIUM 4 **Oxidative stress in Ecology and Evolution**
13:00-13:30	*Mitochondrial coupling efficiency and ROS production: key candidate mechanisms underlying variation in life-history trajectories?* **Dr. Antoine Stier** *Institut Pluridisciplinaire Hubert Curien - CNRSUniversity of Strasbourg, Francia.* **Remote conference via ZOOM**
13:30-14:00	*Oxidative stress in insects in times of global change* **Dr. Daniel González Tokman** *Instituto de Ecología A.C., Xalapa, México.*
14:00-14:30	*Oxidative stress indicators of green turtles (Chelonia mydas) are biomarkers of habitat conditions in the Mexican Caribbean* **Dra. Vanessa Labrada Martagón** *Facultad de Ciencias, Universidad Autónoma de San Luis Potosí (UASLP), SLP, México.*
14:30-16:00	**LUNCH**
16:00-17:00	**KEYNOTE LECTURE 6**

	The role of necroptosis and oxidative stress in aging and age-related diseases **Dra. Deepa Sathyaseelan** *University of Oklahoma Health Sciences Center, Oklahoma City, Oklahoma, EUA.* **Remote conference via ZOOM**
17:00-17:30	**CLOSING CEREMONY** Rector UATx Doctor Serafín Ortiz Ortiz, Secretaria Académica UATx Dra. Margarita Martínez Gómez, and Comitte organizing.
20:00	**DINNER DANCE PARTY, AND MEXICAN SHOW** **Instituto Tlaxcalteca de Cultura**

Oral and Poster presentations

ORAL PRESENTATIONS BY STUDENTS

Wednesday, September 27, 2023

Free Radicals in Cell Biology (FRCB)			
Number	**Authors**	**Institution**	**Title**
FRCB01O	Arlen Ramírez-Corona, Francisco Javier Méndez- Cruz, Denhí Schnabel- Peraza, Hilda Lomeli- Buyoli1, Enrique Salas-Vidal.	Department of Development Genetics and Molecular Physiology. Universidad Nacional Autónoma de México.	NADPH oxidases and dynamin are required for epiboly in zebrafish.
FRCB03O	Elizabeth Brassea-Pérez, Vanessa Labrada-Martagón,José Pablo Vázquez- Medina, Ramón Gaxiola- Robles, Claudia Hernández-Camacho, Tania Zenteno- Savín.	Centro de Investigaciones Biológicas Del Noroeste S.C.	Differential effect of exposure to phthalates inskeletal muscle cells fromterrestrial and marine mammals.
Free Radicals in Aging and Age-Related Diseases (AARD)			
Number	**Authors**	**Institution**	**Title**
AARD04O	Kevin Samael Olascoaga Del Angel, Jorge I. Castañeda-Sánchez, Mina Königsberg, Humberto Gutiérrez, Norma Edith López-Diazguerrero.	Universidad Autónoma Metropolitana. Unidad Iztapalapa.	Impact of oxidative stress onthe prostate transcriptome.

Free Radicals in Exercise and Physical Performance (EPHP)			
Number	**Authors**	**Institution**	**Title**
EPHP01O	Sagrario Cordero-Molina, Jorge Contreras-Garduño.	Escuela Nacional de Estudios Superiores. Universidad Nacional Autónoma de México	He is a ten, but…: prooxidants enhanced attractiveness in mealwormbeetles at the cost of diminished antioxidants in ejaculate.

Thursday, September 28, 2023

Free Radicals in Medicine (FMED)			
Number	**Authors**	**Institution**	**Title**
FMED13O	Ricardo Antonio Rojas Pérez, Alberto Martín Guzmán Grenfell,Silvia Fuentes García, Alondra Cruz Vázquez, Yessica Dorin Torres Ramos.	Facultad de Estudios Superiores Cuautitlán. Universidad Nacional Autónoma de México	Quantification of plasma polysulfides in mother-neonate pairs with and without preeclampsia diagnosis.
Free Radicals in Cancer (FRCN)			
Number	**Authors**	**Institution**	**Title**

FRCN04O	Jesús Valencia Cervantes, Yazmín Debray García, María de los Angeles Andrade Oliva, Ana Larissa Barbosa Sánchez, Alejandra Loaeza Román, Octavio Gamaliel Aztatzi Aguilar, Margarita Isabel Palacios Areola, Martha Patricia Sierra Vargas.	Instituto Nacional de Enfermedades Respiratorias Ismael Cosio Villegas.	Effects of particulate matter $PM_{2.5}$ on the antioxidant status in A459 lung carcinoma epithelial cells: glutathione S-transferase and NAD(P)H: quinone oxidoreductase.
FRCN05O	Fabiola Lili Sarmiento Salinas,Paola Maycotte.	Centro de Investigación Biomédica de Oriente.	Role of reactive oxygen species in breast and ovariancancer.
Free Radicals in Plants (FRPS)			
Number	**Authors**	**Institution**	**Title**
FRPS01O	Dolores I. López-Nicolás, Fernando Rivera-Cabrera, Juan Orozco-Villafuerte, Leticia Buendía-González.	Universidad Autónoma delEstado de México.	Antioxidant activity in *Senna mutiglandulosa* seedlings exposed to Li, Ag, and Cu: an *in vitro* study.

POSTERS SESSION

Wednesday, September 27, 2023

Emergent Topics, Methods, and Models for the Study of ROS (EMTM)			
Number	**Authors**	**Institution**	**Title**
EMTM01C	Lesly Adamari Cueto Covarrubias, Mónica Andrea Valdez Solana, María del Carmen Reza Vargas, Jorge Armando Meza Velázquez, Miguel Aguilera Ortiz, Erick Sierra Campos.	Facultad de Ciencias Químicas, Universidad Juárez del Estado de Durango.	Susceptibility of *M. oleifera* seed oil to oxidative stress.

EMTM02C	Paola Lima-Hernández, Fabiola Lilí Sarmiento-Salinas, Paola Maycotte.	Facultad de Medicina, Benemérita Universidad Autónoma de Puebla.	Reactive oxygen species (ROS) regulate macrophage migration inhibitory factor (MIF) secretion in breast cancers.
EMTM03C	Berenice González-Magallanes, Fátima Sofía Magaña-Guerrero, Víctor Manuel Bautista-de Lucio y Jimmy Giovanni Hernández-Gómez, Ángel Gustavo Salas-Lais, Humberto Hernández-Sánchez.	Unidad de Investigación del Instituto de Oftalmología.	New double labeled technique with probe for detection of intracellular reactive oxygen species (ROS) and reactive nitrogen species (RNS) in activated J774A.1 macrophages.
EMTM04C	Berenice González-Magallanes, Claudia Cruz-Rodríguez, Fátima Sofía Magaña-Guerrero, Víctor Manuel Bautista-de Lucio, Jimmy Giovanni Hernández-Gómez, Ángel Gustavo Salas-Lais, Humberto Hernández-Sánchez.	Unidad de Investigación del Instituto de Oftalmología.	Determination of intracellular reactive oxygen (ROS) and nitrogen (ERN) species production in activated J774A.1 macrophages using flow cytometry.

EMTM05C	Amparo Celene Razo Estrada, Iván Aranda González, Rocío GuzmánIbarra, José Antonio Velázquez-Domínguez.	Escuela Nacional de Ciencias Biológicas. Instituto Politécnico Nacional.	Oxidative stress induced in tadpoles of *Lithobates catesbeianus* by diatomaceous earth.
EMTM06C	María José Martín Martínez, Lorena Alvarez Contreras, Beatriz Liliana España Sanchez, Noé Arjona.	Centro de Investigación y Desarrollo Tecnológico en Electroquímica S. C	Hydrogen peroxide detection on interface-engineered atomically dispersed Co material / N-doped carbon composites.

EMTM07C	Segoviano-León Juan Paulino, Valdez-Morales Maribel, Perea-Domínguez Xiomara Patricia	Universidad Autónoma de Occidente, Instituto Politécnico Nacional, Centro Interdisciplinario de Investigación para el Desarrollo Integral Regional Unidad Sinaloa.	Myoglobin protection ratio of Lippia alba extracts against reactive oxygen species.

Free Radicals in Medicine (FMED)

Number	Authors	Institution	Title
FMED01C	Erick Sierra Campos, Mónica Andrea Valdez Solana, Jorge Armando Meza Velázquez, Miguel Aguilera Ortiz.	Facultad de Ciencias Químicas. Universidad Juárez del Estado de Durango.	In silico assays suggest that morintides from *Moringa oleifera* digested with trypsin produce peptides with antioxidant, anti-inflammatory and anti-cancer properties.
FMED02C	Wylly Ramsés García-Niño, Francisco Correa, Adrián Patricio Castañeda-Gómez, Edson Mejía-Díaz, Aldo José-Rodríguez, Angélica Ruíz-Ramírez.	Department of Cardiovascular Biomedicine. Instituto Nacional de Cardiología "Ignacio Chávez".	L-theanine: a cardioprotective agent against ischemia/reperfusion-induced mitochondrial damage.
FMED03C	Luis Alberto Ayala-Ruiz, Oliver Rafid Magaña-Rodríguez, Patricia Ríos-Chávez.	Facultad de Biología. Universidad Michoacana de San Nicolas de Hidalgo.	Effect of major terpenes of *Callistemon citrinus* on fat deposition and IL-6, TNF-α, adiponectin and, leptin levels in the liver of rats fed with high-fat diet.
FMED04C	Juan Carlos Avilés García, Silvia Fuentes-García, Alondra Cruz Vázquez, María Fernanda Aguilar Dueñas, Ricardo Antonio Rojas Pérez,Yessica Dorin Torres-Ramos.	Escuela Nacional de CienciasBiológicas. Instituto Politécnico Nacional	Evaluation of oxidizing-reducing system alterations inerythrocyte cytosol of neonates born to women withpreeclampsia.

FMED05C	Luis Gerardo Ortega-Pérez,Daniel Godínez-Hernández,Patricia Ríos-Chávez.	Instituto de Investigaciones Químico Biológicas. Universidad Michoacana de San Nicolas de Hidalgo.	Anti-obesogenic effect of leafphytosomes of *Callistemon citrinus* on anthropometric and biochemical parameters in wistar rat.
FMED06C	Jessica Ortega Pérez, Patricia Ríos-Chávez.	Instituto de Investigaciones Químico Biológicas. Universidad Michoacana de SanNicolas de Hidalgo.	Antioxidant effect of the mainterpenes of *Callistemon citrinus* on the liver of rats fedwith a hypercaloric diet.
FMED07C	S. Bautista-Pérez, G. Chávez-Chavira, A. Ortiz-Plata, R. Santana-Martínez, C. Silva-Islas, D. Barrera-Oviedo, P.D. Maldonado.	Department of Pharmacology, Faculty of Medicine. Universidad Nacional Autónoma de México.	S-allylcysteine treatment decrease the alterations and the increase in RIP3 levels induced by ischemia/reperfusion.
FMED08C	Jorge Armando Rojas Rivera,José Luis Saucedo Hernández, Silvia Fuentes-García, Brayan Uriel Salazar González, Yessica Dorin Torres-Ramos.	Escuela Nacional de Ciencias Biológicas. Instituto Politécnico Nacional.	Analysis of oxidative stress markers in colostrum of women with obesity followingpregnancy.
FMED09C	Iván Ignacio-Mejía, Itzel Jatziri Contreras-García, Julieta Griselda Mendoza-Torreblanca, Mercedes Edna García-Cruz, Antonio Romo- Mancillas, Saúl Gómez- Manzo, Cindy Bandala, MaríaElena Sánchez-Mendoza, Luz Adriana Pichardo-Macías, Saray Quintero Fabián, Noemí Cárdenas-Rodríguez.	Laboratorio de Neurociencias, Instituto Nacional de Pediatría. Secretaría de Salud.	Evaluation of the antioxidant activity of levetiracetam in a temporal lobe epilepsy model.

FMED10C	Salvador Pérez-Mora, María del Consuelo Gómez-García,David Guillermo Pérez- Ishiwara.	Escuela Nacional de Medicina y Homeopatía. Instituto Politécnico Nacional	Challenging baldness: hair growth by BFBP through activation of NRF2 and inhibition of lipoperoxidation and apoptosis.
FMED11C	Astrid Mayleth Rivera Antonio, Yazmín K. Márquez-Flores, Alan Hipólito Juárez Solano, Mónica Adriana Torres-Ramos, Itzia Irene Padilla Martínez, Martha Cecilia Rosales Hernández.	Escuela Superior de Medicina. Instituto Politécnico Nacional.	Evaluation of the effect of hydroxycinnamic acids 2c and2f in a model of ulcerative colitis.
FMED12C	Adriana Alarcón Aguilar, Armando Luna-López, Raúl Librado-Osorio, Luis AlbertoErosa-Haro, Mina Konigsberg.	Universidad Autónoma Metropolitana. Unidad Iztapalapa.	Respuesta hormetica antioxidante inducida con metotrexato.
FMED14C	Brayan Uriel Salazar González, José Luis SaucedoHernández, Silvia Fuentes García, María Fernanda Aguilar Dueñas, Jorge Armando Rojas Rivera, Yessica Dorin Torres Ramos.	Facultad de Estudios Superiores Cuautitlán. Universidad Nacional Autónoma de México.	Evaluation of antioxidant capacity in breast milk from women with obesity.
FMED15C	Elian Yuritzi Alegría Herrera,Laura Josefina Pérez Flores,Maribel Lucila Herrera Ruiz, Adriana Alarcón Aguilar, Mayra Alejandra Santillán Urquiza, Jesús Enrique Jiménez Ferrer.	Universidad Autónoma Metropolitana. Unidad Iztapalapa.	Evaluation of antioxidant and anti-inflammatory properties of *Ocimum selloi* (lamiaceae)in mice TPA model.

FMED16C	Alizon Sujey Morales Guzmán, Adriana Alarcón Aguilar, Armando Luna López, Daniel Santana Vargas, Max Julio SchmulsonWasserman.	Universidad Autónoma Metropolitana. Unidad Iztapalapa.	Correlation of oxidative stress(OE) with interleukins in irritable bowel syndrome (IBS).
FMED17C	Lorena De La Cruz Mendoza,Marlen Valdés-Fuentes, ErikaRodríguez-Martínez, Eduardo Hernández-Orozco, Alfredo Miranda-Martínez, Selva Rivas-Arancibia.	Facultad de Medicina. Universidad Nacional Autónoma de México.	Effect of chronic exposure to low doses of O_3 on NFκB activation in the jejunum and hippocampus.
FMED18C	Yessica Dorin Torres-Ramos,Alondra Cruz Vázquez, Juan Carlos Avilés García, María Fernanda Aguilar Dueñas, Ricardo Antonio Rojas Pérez.	Instituto Nacional de Perinatologia Isidro Espinosa de los Reyes.	Evaluation of ischemia-modified albumin and its relationship with free fatty acids in neonates born from women with preeclampsia.
FMED19C	David Calderón Guzmán, Norma Osnaya Brizuela, Maribel Ortiz Herrera, Armando Valenzuela Peraza,Francisca Trujillo Jimenez, Gerardo Barragan Mejia.	Instituto Nacional dePediatría.	5-HIAA and ATPase increaseduring N-Acetylcysteine and Cisplatin administration and induce neuroprotection in brain.
FMED20C	Ana M. Nava-Cabrera, MiguelVázquez-Moreno, Daniela Orozco-Colín, Araceli Pérez- Bautista, Brenda Valdez- Feregrino, Margarita Díaz- Flores, Miguel Cruz-López.	Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social.	Total antioxidant capacity modifies the correlation between body mass index and HDL cholesterol in Mexican children.
FMED21C	Clara Ortega-Camarillo, Guadalupe Diaz-Rosas, Mayra Cruz-Hernández, Agustin Pedraza-Galeana, Adolfo López-Torres, Alejandra Contreras-Ramos.	Unidad de Investigación Médica en Bioquímica. Instituto Mexicano del Seguro Social.	Effect of borax on mouse cardiac hypertrophy induced during pregnancy.

FMED22C	Laura Denise López Barrera,Rafael Alejandro Rodríguez Salazar, Miriam Guadalupe López Luna, Roberto Diaz Torres, Patricia Ramírez Noguera.	Facultad de Estudios Superiores Cuautitlán. Universidad Nacional Autónoma de México.	Characterization of antioxidant nanostructured systems and their cytotoxic effect in primary culture of ratchondrocytes.
FMED23C	Armando Ordaz Hernández, Mayra Herrera Martínez, Leidy Diana López Ignacio, Juan Manuel Loeza Corte, Emmanuel Correa Solis, Carolina Calderón Chiu, LibniIsrael Ramírez Acevedo.	Instituto de Farmacobiología. Universidad de la Cañada.	Antioxidant properties of Stevia lucida in murine macrophages J774A.1 induced with LPS.
FMED24C	Eduardo Madrigal-Santillán, Jacqueline Portillo-Reyes, José Antonio MoralesGonzález, Eduardo Madrigal-Bujaidar, Ángel Morales-González, Manuel SánchezGutiérrez, Jeannett Alejandra Izquierdo-Vega.	Escuela Superior de Medicina. Instituto Politécnico Nacional.	Antigenotoxic capacity of chayote juice (*Sechium edule*)against oxidative damage produced by benzopyrene.
FMED25C	Cristina Salinas-Nolasco, Elizabeth Pérez-Hernández, Nury Pérez-Hernández, Aracely Evangelina Chávez-Piña.	Escuela Nacional de Medicina y Homeopatía, Instituto Politécnico Nacional.	Alpha-linolenic fatty acid (ALA) protects against indomethacin-induced gastricdamage in murine model.
FMED26C	Sánchez Aguilar Demetrio Guadalupe, García Benavides Leonel, CastañedaArellano Rolando, Méndez Del Villar Miriam, Trujillo Rangel Walter Ángel.	Centro Universitario de Tonalá, Departamento de Ciencias Biomédicas.	Effect of L-Arginine in oxidative stress index and oxidative damage to DNA on Hind-Limb ischemia-reperfusion model.
FMED27C	Trujillo Rangel Walter Ángel, Leonel García Valdés, Castañeda Arellano Rolando,Méndez Del Villar Miriam, Demetrio Sánchez Aguilar, García Benavides Leonel.	Centro Universitario de Tonalá, Departamento de Ciencias Biomédicas.	Therapeutic targets for regulating oxidative damage induced by ischemia-reperfusion injury: a study from a pharmacological perspective

FMED28C	Jessica Baldriche-Acosta, Marisela Uribe-Ramírez, Juana Narváez-Morales, Olivier Christophe Barbier, Octavio Gamaliel Aztatzi-Aguilar.	Centro de Investigación y de Estudios Avanzados del Instituto Politécnico Nacional.	Oxidative stress in kidney damage from acute inhalationexposure to endotoxin in a ratmodel.
FMED29C	Ángeles Suárez Santos, Nadia Lizeth Soto Gómez, María Cristina Ortíz León, Minerva Hernández Lozano, Clara Luz Sampieri, Pedro Zapater Hernández, Eduardo Rivadeneyra Domínguez, Oscar Rosales Sánchez, Emmanuel Severino Quintana, Rebeca García Román.	Instituto de Salud Pública. Universidad Veracruzana.	Liver damage in obesity due to frequent consumption of acetaminophen (generator of oxidative stress).
FMED30C	Julia Yazmín Morales Domínguez, Eduardo Rivadeneyra Domínguez, Omar Elind Arroyo Helguera, Rebeca García Román.	Instituto de Salud Pública. Universidad Veracruzana.	Glutathione level at the stageof steatohepatitis in patients with metabolic fatty liver disease.
FMED31C	José Antonio Guevara-García.	Facultad de Ciencias Básicas, Ingeniería y Tecnología. UniversidadAutónoma de Tlaxcala.	Vanadium compounds: potential medicine applications as a source of ROS.
FMED32C	Tonantzi Guadalupe Osorio Pérez, Eliut Pérez Sánchez, Vanessa Guadalupe Nolasco Garduño, Leticia Nicolás Toledo.	Universidad Autónoma de Tlaxcala.	Influence of chronic stress onoxidative stress in epididymalspermatozoa of young rats.

Free Radicals in Cell Signaling (FRCS)			
Number	**Authors**	**Institution**	**Title**
FRCS01C	Manuel Martínez Ramírez, Yessica Zamudio Cuevas, Karina Martínez Flores, Javier Fernández Torres, María Fernanda Pérez Ruíz, Ambar López Macay.	Universidad Autónoma Metropolitana. Unidad Xochimilco.	Study of the role of the urate transporter ABCG2 in the regulation of the immune response in knockout models of human colon and kidney cells.
FRCS02C	Yazmín Contreras-Bravo, Alejandro De las Peñas Nava, Guadalupe Gutiérrez-Escobedo.	Instituto Potosino de Investigación Científica y Tecnológ ica A.C.	Msn2 and msn4 regulation during oxidative stress response
FRCS03C	Jorge Damián Ramírez Robles, Francisco Torres Quiroz.	Instituto de Fisiología Celular. Universidad Nacional Autónoma de México.	Redox regulation of DNA repair by peroxiredoxin Tsa1 and cysteine oxidation in *Saccharomyces cerevisiae.*
FRCS04C	Lizbeth Espinosa-García, Isela Álvarez-González, JoséDavid García-García, Felipe de Jesús Carrillo-Romo, Antonieta García-Murillo, Eduardo Madrigal Bujaidar.	Escuela Nacional de Ciencias Biológicas. Instituto Politécnico Nacional.	Oxidative stress induced by gadolinium-based nanoparticles in cultured human lymphocytes.

Thursday, September 28, 2023

Free Radicals in Medicine (FMED)			
Number	**Authors**	**Institution**	**Title**
FMED33C	Reyna Hernández Calderón, Jaime Morales Romero, Pamela Romanque Ulloa, Julio Isael Pérez Carréon, Jonathan García Román, Rebeca García Román.	Instituto de Salud Pública. Universidad Veracruzana.	Expression of redox genes involved in the lipid storage mechanism in fatty liver disease associated with metabolic dysfunction

FMED34C	José Abraham Marín Carmona, María del Socorro Fernández, Elda María del Rocío Coutió Rodríguez, Ana María Aguirre Martínez, Silvia Ivonne Mora Herrera3, Clara Inés Espitia Pinzón, Jonathan García Román, Rebeca García Román.	Instituto de Salud Pública. Universidad Veracruzana.	Glutathione peroxidase-1 mRNA levels in patients with comorbidity: fatty liver disease associated with metabolic dysfunction and type 2 diabetes mellitus.
FMED35C	Eliut Pérez Sánchez, Jorge Rodríguez Antolín, Estela Cuevas Romero, Leticia Nicolás Toledo.	Universidad autónoma de Tlaxcala.	Chronic unpredictable mild stress causes oxidative stress in kidneys in adults rats.
FMED36C	Leticia Nicolás Toledo, Bibiana Montoya, Eliut Pérez Sánchez, Jorge Rodríguez Antolín.	Universidad autónoma de Tlaxcala.	Adult rats exposed to a high-sugar diet early in life have permanent histomorphological changes and oxidative stress in the testis.
FMED37C	Sarmiento-Ortega Victor Enrique, Moroni-González Diana, Diaz Alfonso, Treviño Samuel.	Faculty of Chemical Sciences. Meritorious Autonomous University of Puebla.	Adipose tissue as a target organof reactive oxygen species by cadmium exposure in Wistar rat.
FMED38C	Aguilar-Gamas C.F, Gómez-Crisostomo, N.P, Lopez-Diazguerrero N.E, Martinez-Abundis E.	Universidad Juarez Autónoma de Tabasco, Universidad Autónoma Metropolitana-Iztapalapa	Evaluation of the electron transport chain activity and production of oxidative stress in the brains of rats with obesity Induced by a high-fat and high-sugar diet

Free Radicals in Cell Biology (FRCB)

Number	Authors	Institution	Title
FRCB02C	Ana Gabriela Santana Chávez, Teresa Montiel, Lourdes Massieu.	Instituto de Fisiología Celular. Universidad Nacional Autónoma de México.	Recurrent moderate hypoglycemia generates changes in glutamate transport and cysteine uptake for glutathione synthesis.

FRCB04C	María Dolores Caballero Sánchez, Pedro Antonio Hernández Cruz, Berenice Fernández Rojas, Itandehuí Belem Gallegos Velasco.	School of Medicine and Surgery. Universidad Autónoma Benito Juárez de Oaxaca.	The role of O-GlcNAcylation in the development of oxidative stress in MCF-7 cells.
FRCB05C	Melani León Martínez, Rubio Ruíz María Esther, Vicente Castrejón Téllez.	Instituto Nacional de Cardiología "Ignacio Chávez".	Effect of cyanidin on oxidative stress and mitochondrial biogenesis in an aortic endothelial cell line (PAE) during hypoxia-reoxygenation.
FRCB06C	Mariana García, Elías Nieto-Zaragoza, Francisco Torres-Quiroz.	Instituto de Fisiología Celular. Universidad Nacional Autónoma de México.	Effect of potential hydrogen on hydrogen sulfide production in *Saccharomyces cerevisiae*.
FRCB07C	Camila Roa Gutiérrez, Jorge Damián Ramírez Robles, Francisco Torres Quiroz.	Instituto de Fisiología Celular. Universidad Nacional Autónoma de México.	Characterization of Sod1 in a non RSS accumulative *S. cerevisiae* strain.
FRCB08C	Marijose López Miranda, Elías Nieto Zaragoza, Francisco Torres Quiroz.	Instituto de Investigaciones Biomedicas. Universidad Nacional Autónoma de México	Visualization of yeast cystathionine beta synthase is dependent on its native 3´ UTR.
FRCB09C	Nancy Sánchez Fuentes, Carolina López Rubalcav, Francisca Pérez Severiano.	Departamento de Farmacobiología, Cinvestav-Sede Sur.	Oxidative damage in adult mice caused by chronic exposure to lead since pregnancy and its association with anxiety-like anddepression-like behaviors.
FRCB010C	Jesus Javier Delgado Reyna, María de los AngelesAndrade Oliva, Octavio Gamaliel Aztatzi Aguilar, Elvira Gómez Guerrero, Yazmín Debray García.	Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas.	Effect of vitamin E acetate exposure on the antioxidant response and tight junction integrity of a549 cells in co-culture with Thp1.

FRCB011C	Yessica Caballero-Vázquez, Ahiezer Rodriguez-Tobón, Fausto R. Méndez-de la Cruz, Edith Arenas-Rios.	Lab. Morfofisiología y Bioquímica del Espermatozoide UAM-I. Lab. de Biología y Ecología de Mamíferos UAM-I. Laboratorio de Herpetología UNAM.	Participation of antioxidant enzymes in the protection of the testis and epididymis against temperature changes in the lizard *Holbrookia propinqua*

Free Radicals in Cancer (FRCN)

Number	Authors	Institution	Title
FRCN01C	Gareth Omar Rostro-Alonso, Erick Fernando Aguilar-Llanos, Edgar Yebrán Villegas-Vázquez, Laura Itzel Quintas-Granados, Daniela Silva-Adaya, Gabriela Figueroa-González, Octavio Daniel Reyes-Hernández.	Facultad de Estudios Superiores Zaragoza. Universidad Nacional Autónoma de México.	I3C modulates tumor capacities in an *in vitro* model of cervical cancer: possible role of AhR in the induction of cell arrest and autophagy.
FRCN02C	Montes-Alvarado José, Domínguez-Avilés Irma, Milflores-Flores Lorena, Maycotte-González Paola.	Centro de Investigación Biomédica de Oriente. Instituto Mexicano del Seguro Social.	Antioxidant effect of isothiocyanates from brassicas vegetables on benzo(a)pyrene-induced reactive oxygen species production in a cell transformation model.
FRCN03C	Karen Andrea Larrauri Rodríguez, Lourdes Millán Pérez Peña, Paola Maycotte.	Centro de Investigación Biomédica de Oriente. Instituto Mexicano del Seguro Social.	Analysis of oxidation and ERK activation markers in databases of patient samples and cervical cancer cell lines.
FRCN06C	Aldair Soria Contreras, Diana Guadalupe Meza Hernández, Citlali Ekaterina Rodríguez Pérez, Mónica Adriana Torres Ramos.	Instituto Nacional de Neurología y Neurocirugía Manuel Velasco Suárez.	Hormetic response to oleic acid on human glioma cells in a medium enriched with reactive oxygen species.

Free Radicals in Neurodegeneration (FRND)			
Number	**Authors**	**Institution**	**Title**
FRND01C	Mayra Lizeth López Cervantes, Andrés Quintanar Stephano, Jorge Larriva Sahd, Raúl Aguilar Roblero, Gema Martínez Cabrera, Olivia Vázquez Martínez, Mauricio Díaz Muñoz.	Institute of Neurobiology. Universidad Nacional Autónoma de México.	Evaluation of mitochondrial structure and function in a model of cerebellar vacuolar degeneration in rats with portocaval anastomosis.
FRND02C	Julián de Jesús Lira-Rotstein, Roberto Santín-Márquez, Adriana Alarcón Aguilar, Raúl Librado Osorio3; Oscar Rosas Carrasco, Mina Konigsberg, Armando Luna López.	Universidad Autónoma Metropolitana. Unidad Iztapalapa.	Evaluation of the neuroinflammatory state and oxidative stress in older adult women with obesity.
FRND03C	Claudia Dorado-Martínez, Enrique Montiel-Flores, José Luis Ordoñez-Librado, Ana Luisa Gutiérrez-Valdez, Jesús Espinosa Villanueva, Ana Karen Sánchez-Medina, Denisse Cortés-Velázquez, Maria Rosa Avila-Costa.	Facultad de Estudios Superiores Iztacala. Universidad Nacional Autónoma de México.	Vanadium pentoxide (V2O5) inhalation as an induced model of Alzheimer's disease.

Free Radicals in Aging and Age-Related Diseases (AARD)			
Number	**Authors**	**Institution**	**Title**
AARD01C	Verónica Salas-Venegas, Roberto Santín-Márquez, Pamela Flores-Torres, Marisol De la Vega-Tinajar, Ángeles Fortis-Barrera, Arturo Belmont, Raúl Librado-Osorio, Norma E. López-Díazguerrero, Armando Luna-López, Mina Königsberg.	Universidad Autónoma Metropolitana. Unidad Iztapalapa.	Effects of senolytic and senomorphic treatments on systemic redox state in an obesity model of middle-aged female rats.
AARD02C	Osiris Germán Idelfonso-García, Brisa Rodope Alarcón-Sánchez, Verónica Rocío Vásquez-Garzón, Saúl Villa-Treviño, Pablo Murie, Héctor Serrano, JulioIsael Pérez-Carreón, Jaime Arellanes-Robledo.	Instituto Nacional de Medicina Genómica.	Nucleoredoxin activity is altered by oxidative stress generated bychronic alcohol consumption andis associated with increased cellular senescence in the liver of aged mice.
AARD03C	Michel López Teros, Karla Estephania Ávila, Raúl Librado-Osorio, Adriana Alarcón Aguilar, Armando Luna López, Mina Königsberg.	Universidad Autónoma Metropolitana. Unidad Iztapalapa.	Differences in redox state in primary rat cerebral cortex astrocytes induced to cellular senescence or gliosis with palmitate.
AARD05C	Gavia-García, David Hernández-Álvarez, Taide Laurita Arista-Ugalde,Itzen Aguiñiga-Sánchez, Edelmiro Santiago-Osorio, Víctor Manuel Mendoza- Núñez, Juana Rosado- Pérez.	Universidad Nacional Autónoma de México.	The consumption of *Sechium edule* (chayote) prevents telomere attrition in older adults with metabolic syndrome.

AARD06C	Haisha Cortés Carrasco, Samuel Treviño Mora.	Facultad de Ciencias Químicas, Benemérita Universidad Autónoma de Puebla.	Effect of chronic administration of resveratrol on the redox balance of the prefrontal cortex of the male wistar rat during aging.
AARD07C	David Hernández Álvarez, Nayeli Anaí Vaquero Barbosa, Juana Rosado Pérez, Graciela Gavia García, Taide Laurita Arista Ugalde, Itzen Aguiñiga Sánchez, Edelmiro Santiago Osorio, Víctor Manuel Mendoza Núñez.	Universidad Nacional Autónoma de México.	Tai chi training vs strength training on markers of oxidative stress and inflammation.
AARD08C	Esmeralda Lira Romero, Itzel Guadalupe Rebollo Ruiz, Misato González Kawahara, Ramírez Sánchez, Francisco Javier Estrada Mena.	Universidad Panamericana. School ofMedicine.	Preventive effects of (-)-epicatechin in a model of retinaldegeneration induced by $NaIO_3$.

Free Radicals in Microorganisms (FRMS)			
Number	**Authors**	**Institution**	**Title**
FRMS01C	Ma. Guadalupe Gutiérrez Escobedo, Ana López Marmolejo, Irene Castaño Navarro, Alejandro De Las Peñas Nava.	Instituto Potosino de Investigación Científica y Tecnológica A.C.	Glutathione and thioredoxin pathways in *Candida glabrata.*
FRMS02C	Antonia Herrera-Ortiz, Shou-Jiang Gao.	Keck School of Medicine, University of Southern California.	Nitric oxide mediates Kaposi's sarcoma-associated herpesvirus lytic replication.
FRMS03C	Leslie Azucena Sámano Rodríguez, Santa Garcia Cisneros, Maria Olamendi Portugal, Miguel Angel Sánchez Alemán, Antonia Herrera Ortiz.	Centro de Investigaciones sobre Enfermedades Infecciosas. Instituto Nacional de Salud Pública.	Nitric oxide and SARS-CoV-2, evidence of high serum nitrite concentrations in hospitalized patients with COVID-19.

FRMS04C	James González, Miguel Rosas-Paz, Alberto Zamora-Bello, Diana Villarreal-Huerta, Nayeli Torres-Ramírez, Lucero Romero-Aguilar, Claudia Segal-Kischinevzky.	Facultad de Ciencias. Universidad Nacional Autónoma de México.	characterization of the antarctic yeast *Rhodotorula mucilaginosa*: lipid droplets, expression profiles, and fatty acids synthesis.

Free Radicals in Plants (FRPS)

Number	Authors	Institution	Title
FRPS02C	Jesús David García Ortiz, Sendar Daniel Nery Flores, Juan Alberto Ascacio Valdés, Raúl Rodríguez Herrera.	Facultad de Ciencias Químicas. Universidad Autónoma de Coahuila.	Antioxidant effect of pigments obtained from red corns.
FRPS03C	Carlos Neftali Cano-Gonzalez, Lluvia de Abril Alexandra Soriano-Melgar, Juan Carlos Contreras-Esquivel, Raul Rodríguez-Herrera, Rocio Yaneli Aguirre-Loredo.	Facultad de Ciencias Químicas. Universidad Autónoma de Coahuila.	Microwave-assisted extraction of phenolic compounds with potential antioxidant activity from grape residues.
FRPS04C	Janeth Karina Jacuinde-Guzmán, Héctor Bernardo Escalona-Buendía, Fernando Rivera-Cabrera, Denise Raddatz-Mota, Lluvia de Abril Alexandra Soriano-Melgar.	Universidad Autónoma Metropolitana. Unidad Iztapalapa.	Effect of calcium nanoparticles on antioxidants compounds, enzymes, and capacity of minimally processed watermelon (*Citrullus lanatus*).
FRPS05C	Gina Carolina Bandera Rojas, María Guadalupe Neira Velázquez, Gustavo Soria Argüello, Raúl Rodríguez-Herrera, Lluvia de Abril Alexandra Soriano-Melgar.	Facultad de Ciencias Químicas. Universidad Autónoma de Coahuila.	Plasma activated water as a postharvest treatment for gerbera (*Gerbera jamesonii* l.)

FRPS06C	Sonia Estela Mar-Ramírez, Lluvia de Abril Alexandra Soriano-Melgar, Carlos Neftali Cano Gonzalez, Raúl Rodríguez Herrera.	Facultad de Ciencias Químicas. Universidad Autónoma de Coahuila.	Oligosaccharins and polyethylene glycol on chlorophylls, phenolic compounds, and antioxidant capacity of candelilla (*Euphorbia antisyphilitica* zucc.) *In vitro*.
FRPS07C	Eduardo Ruíz Hernández, Alma Socorro Sobrino-Figueroa, José Roberto Villagómez Ibarra.	Universidad Autónoma del Estado de Hidalgo. Universidad Autónoma Metropolitana Iztapalapa.	Effect of Sodium Dodecyl Sulphate on the production of Thiobarbituric Acid Reactive Substances and photosynthetic pigments in the alga *Cladophora sp* and the macrophyte *Egeria densa*.

Abstracts

FRCB01O

NADPH oxidases and dynamin are required for epiboly in zebrafish

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Reactive oxygen species (ROS) are known to be involved in regulating several cellular functions, such as proliferation, cell death, migration, and adhesion among others, all essential to orchestrate early developmental morphogenesis. Recently we reported that reactive oxygen species generated by NADPH oxidases (Nox) are required for cell motility and the progression of epiboly, the first morphogenetic movement carried out by the zebrafish embryo. Epiboly consists of the thinning and spreading of the blastoderm over the yolk cell during gastrulation.

We have reported that the pharmacological downregulation of Nox activity by treating embryos with VAS2870 delays migration of blastoderm cells and produces lethality before 24 hrs of development. We also found that endocytosis and intracellular trafficking of E-cadherin are processes regulated by Nox produced ROS.

All these effects observed after Nox inhibition are rescued after treatment with H_2O_2. We have also found that inhibition of Dynamin 2 activity and, as a result, inhibition of endocytosis fully rescues E-cadherin localization, epiboly progression and survival.

Additionally, we have found as well that inhibition of proteasomal protein degradation, a process downstream of the endocytic flow, partially rescues the epiboly delay elicited by Nox pharmacological inhibition. Overall, our results suggest that H_2O_2 produced by Nox participates in regulating E-cadherin trafficking, which is necessary for epiboly progression. These results provide new information towards elucidating the pathways through which ROS participate in regulating normal early development. This work was supported by UNAM-PAPIIT IN212820 and IN227223. A.R.C. acknowledges fellowship 720706 and PhD scholarship from CONAHCyT.

FRCB03O

Differential effect of exposure to phthalates in skeletal muscle cells from terrestrial and marine mammals

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The plasticizer bis (2-ethylhexyl) phthalate (DEHP) dysregulates the balance between reactive oxygen species production and antioxidant defenses. Despite the information available on DEHP's hazardous effects in model species, the cellular response to DEHP in organisms naturally adapted to tolerate oxidative stress remains unexplored. Marine mammals are naturally adapted to cope with oxidative stress derived from diving-induced ischemia/reperfusion. The objective of this study was to compare changes in oxidative stress indicators induced by DEHP in human (*Homo* sapiens) and California sea lion (*Zalophus californianus*) skeletal muscle cells. Abdominal muscle samples were collected from healthy women undergoing planned cesarean surgery at full term after obtaining informed consent. Sea lion muscle samples were obtained from recently deceased pups at Los Islotes rookery, Gulf of California. Skeletal muscle cells were isolated and cultured under standard conditions. Cells were divided into two groups. One group was exposed to 1 mM DEHP for 13 days (n = 25; treatment), and the other served as a control (n = 25; no DEHP). Superoxide radical ($O_2^{\bullet-}$) production, oxidative damage, antioxidant enzyme activities and gene expression were measured using spectrophotometric methods and RT-qPCR. DEHP exposure increased $O_2^{\bullet-}$ production and superoxide dismutase (*SOD*) activity in both species. While the activity of glutathione S-transferase (*GST*) and protein carbonyls (*PC*) levels significantly increased in human cells, these indicators showed a non-significant elevation in sea lion cells. In contrast, Glutathione peroxidase (*GPx*) and catalase (*CAT*) activities increased significantly in sea lion but not in human cultures exposed to DEHP. Glutathione reductase (*GR*) did not show significant changes in neither species. Genes involved in redox metabolism showed differential expression between culture conditions and species. DEHP exposure led to widespread modifications in gene expression patterns, with 27 and 18 differentially expressed genes in humans and sea lion cells, respectively. In human cells, DEHP increased microsomal *GST1* and *GST* (κ, μ, θ, ω, and z), while suppressing 8-oxoguanine DNA glycosylase (*OGG1*), *CAT*, *GR*, and nuclear factor erythroid 2-related factor 2 (*Nrf2*) expression, suggesting increased oxidative stress and phase two detoxification processes. In contrast, DEHP increased *OGG1*, *Nrf2*, *GPx2* and *SOD3* expression, suggesting that DEHP activates antioxidant defenses in sea lion cells, potentially contributing to maintain redox homeostasis and avoid oxidative damage. Keywords: Antioxidant enzymes, emerging pollutants, reactive oxygen species.

AARD04O

Impact of Oxidative Stress on the Prostate Transcriptome

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This study focused on elucidating the transcriptomic changes occurring in human prostate epithelial cells when exposed to hydrogen peroxide, a Reactive Oxygen Species (ROS), leading to cellular senescence. ROS are extremely reactive molecules that can interact with cellular components and disrupt cellular homeostasis, ultimately resulting in cellular senescence. The accumulation of senescent cells in tissues and organs is closely associated with chronic-degenerative diseases. To induce senescence, human prostate epithelial cell cultures were established and subjected to hydrogen peroxide exposure. After 9 days, total RNA was extracted, and the Affymetrix Clariom™ D Assay platform was employed to assess gene expression and measure alternative splicing events. Furthermore, gene co-expression networks and functional genomics techniques were utilized to characterize the genes impacted by hydrogen peroxide exposure. The findings revealed the identification of 2,493 genes with differential expression. Co-expression networks highlighted variations in gene interactions across different age groups. Moreover, some of these genes exhibited notable correlations with age, indicating their involvement in aging-related physiological changes. Furthermore, 1,889 significant alternative splicing events were detected, and functional enrichment analysis unveiled that the differentially expressed genes are associated with biological processes related to oxidative stress and aging. In summary, this study demonstrates that hydrogen peroxide-induced oxidative stress can result in substantial alterations in gene expression and gene interactions within human prostate cells, and these changes are closely linked to the aging process. The discovery of alternative splicing events and the enrichment of biological processes associated with oxidative stress offer valuable insights into the potential contributions of these factors to age-related changes in prostate tissue. These findings hold significant implications for comprehending prostate diseases related to aging and present new opportunities for exploring therapeutic targets in this context.

Acknowledgements This work was supported by Consejo Nacional de Ciencia y Tecnología (CONACYT) grant FORDE-CYT-PRONACES/263957/2020. Olascoaga-Del Angel KS is a CONACyT scholarship holder.

EPHP01O

He is a ten, but…: prooxidants enhanced attractiveness in mealworm beetles at the cost of diminished antioxidants in ejaculate

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Variations in extrinsic environmental factors, such as food or parasite load, can affect the homeostatic state of organisms, putting their survival at risk. On some occasions, when the damage caused by the environment exceeds a certain level, organisms invest their resources to maximize their present reproduction rather than future reproduction. This phenomenon is known as terminal investment (TI). One of the environmental factors to which many organisms in nature can be exposed is xenobiotic contaminants, which can cause oxidative stress and jeopardize survival. However, oxidative stress has not been described as a trigger for TI. In this study, we investigated whether the commonly used prooxidant herbicide, paraquat (PQ) can induce TI in the beetle *Tenebrio molitor*. In this species it has been found that males that are about to die produce more sexual pheromones and are more preferred by females than healthy males. We applied a PQ in a dose gradient and evaluated whether, at any of these doses, males were more preferred by females and whether females laid more eggs. Subsequently, we investigated whether oxidative stress and massive investment in attractiveness affect male fertility. To do this, we measured the levels of oxidants and antioxidants in the ejaculate of males that heavily invest in being chosen for copulation compared to males that first recover from oxidative stress before copulating. Our results showed that TI due to oxidative stress is dose-dependent on the prooxidants received by males, although it did not affect the number of eggs. We also found that the ejaculate of males engaging in TI has less peroxidase enzyme, while males capable of recovering before mating provide females with ejaculate containing higher antioxidant protection. These findings highlight the influence of oxidative stress on the reproductive strategies of males facing mortality, as well as potential reproductive costs incurred by females when choose males engaged in terminal investment of their pheromones.

FMED13O

Quantification of Plasma Polysulfides in Mother-Neonate Pairs with and without Preeclampsia Diagnosis

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Preeclampsia (PE) is characterized by increased oxidative stress levels. Polysulfides, a novel group of biomolecules, have recently emerged as potent reductive agents. It is hypothesized that under conditions of oxidative stress, the synthesis of polysulfides is stimulated from various proteins and non-protein biomolecules, serving as a compensatory protective response. This study aimed to quantify plasma polysulfides, total antioxidant capacity, Malondialdehyde (MDA) as a marker of lipid damage, and protein carbonylation as a marker of protein damage in 88 mother-neonate pairs, including 44 cases with a PE diagnosis and 44 cases without PE diagnosis. Statistical analysis using the ANOVA test and Bonferroni's multiple comparisons post-hoc test revealed no significant difference in plasma polysulfide production among the four study groups. However, a statistically significant increase in total antioxidant capacity was observed in both mother-neonate pairs with and without PE diagnosis ($p=0.0409/p=0.0069$, respectively). While no significant difference in MDA levels was found between the control group of mothers and the group of mothers diagnosed with PE, a significant difference was observed between the control group of neonates and the group of neonates born to mothers diagnosed with PE ($p=0.0005$). Protein carbonylation exhibited a statistically significant increase in both mother-neonate pairs with and without PE diagnosis ($p=0.0113/p=0.0001$, respectively). The results demonstrate evident oxidative damage to lipids and proteins in mother-neonate pairs affected by PE, along with activation of the antioxidant capacity. However, the contribution of polysulfides as reducing agents appears to be insignificant, potentially due to the moderate intensity of the oxidative damage observed. It is plausible that the existing antioxidant capacity is sufficient to counteract the oxidative damage, while polysulfides may be produced under more severe oxidative conditions.

Keywords: Preeclampsia, Oxidative Stress, Polysulfides

FRCN04O

Effects of particulate matter $PM_{2.5}$ on the antioxidant status in A459 lung carcinoma epithelial cells: glutathione S-transferase and NAD(P)H: Quinone Oxidoreductase

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Pollution is the largest environmental cause of diseases and premature deaths worldwide, with an estimated 9 million deaths in 2019. The evidence suggests that particulate matter (PM) pollution, especially $PM_{2.5}$ is a main risk for lung cancer; it is also associated with the increased incidence of other diseases such as cardiovascular diseases or asthma. Lung cancer is the leading cause of cancer deaths (2,206,771 new cases and 1,796,144 deaths), representing a public health problem. Therefore, it is necessary to identify the mechanisms that support the association between PM and lung cancer. This study aimed to analyze the protein expression and enzymatic activity of glutathione S-transferase (GST) and NAD(P)H: Quinone Oxidoreductase (NQO1) in A549 lung carcinoma epithelial cells after the treatment with $PM_{2.5}$ obtained from three regions of Mexico City, from November 2021 to April 2022. The cells were exposed for 24 h to 10 µg/ml of $PM_{2.5}$ with simulated epithelial lung fluid formulation with DMEM. The cell viability was evaluated by MTT assay and protein expression by Western Blot for GST isoforms M1, T1, P1, and NQO1. The enzymatic activity of glutathione S-transferases was measured through 1-Chloro-2,4-dinitrobenzene as a substrate and the enzymatic activity of NQO1, through the reduction of menadione. The results indicated that 10 µg of $PM_{2.5}$ does not compromise cell viability ($p>0.05$). However, the protein expression of GST isoforms M1, T1, P1, and NQO1 decreased modestly with the $PM_{2.5}$ exposure of the different regions. Likewise, the enzymatic activity of GST and NQO1 decreased after treatment. Our results propose that $PM_{2.5}$ reduces the antioxidant capacity of lung cancer cells, which could allow disease progression (PROYECT NUMBER: CF-MG-20191024195736984-840342).

FRCN05O

Role of reactive oxygen species in breast and ovarian cancer

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Breast and ovarian cancer are among the leading causes of death by neoplasia in women worldwide. Breast cancer is classified by immunohistochemistry into three subtypes: the hormone receptor positive (RE+) subtype, which responds to hormonal therapy; the epidermal growth receptor type 2 subtype positive (HER2 +), which is treated with the monoclonal antibody trastuzumab and triple negative breast cancer (NT), which lacks biomarkers for diagnosis and targeted therapy. On the other hand, ovarian cancer is a histologically, clinically, and molecularly heterogeneous disease, for which there are few detection methods and few treatment options, with high-grade subtypes having the worst prognosis.There is extensive information about alterations present in cancer cells, as an example, an increase in reactive oxygen species (ROS), which has been associated with DNA damage, genomic instability, the activation of different signaling pathways related to survival and cell proliferation and increase in the secretion of proinflammatory cytokines, promoting the origin and progression of cancer. In the present work, ROS levels and potential sources were determined in different breast cancer cell lines, which allowed us to establish a correlation between basal levels of ROS and the breast cancer subtype, with NT showing the highest levels of ROS, compared to the ER+ subtype and the non-tumorigenic control. In ovarian cancer cell lines, we demonstrated that lines corresponding to the high-grade serous subtype had basal ROS levels similar to those of breast cancer NT lines. Breast and ovarian cell lines were shown to be sensitive to treatment with the general antioxidant N-acetylcysteine, but some were also found not to respond equally to the mitochondrial antioxidant MitoTempo. On the other hand, a correlation was established between the elevated levels of ROS and the increase in the secretion of different proinflammatory cytokines, since the NT lines and the serous-type ovarian lines showed high rates of MIF, IL-6 and IL- 8. Importantly, the secretion of some of these cytokines was reduced by providing antioxidant treatment. Therefore, it is proposed that ROS could have high potential as biomarkers of malignancy and as a target of therapy in NT cancer and epithelial ovarian cancer, which currently have the worst prognoses and have limited options for targeted therapies.

Key words: ROS, proinflammatory interleukins, antioxidants, MIF, NAC, MitoTEMPO.

FRPS01O

Antioxidant activity in *Senna mutiglandulosa* seedlings exposed to Li, Ag, and Cu: an *in vitro* study

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The increase in technological development and industrial growth generates a lot of solid waste, including electronic waste. The latter, at the end of their useful lives, are deposited in landfills (87%) and open-air dumps (13%), and their poor management and regulation represent a serious problem of pollution that is increasing; only in 2019 was the generation of 45 million tons worldwide estimated. In addition, these e-wastes release high concentrations of metals, which are incorporated into the environment in manufacturing processes, during their use, and at the end of their useful lives. Once the metals are inserted, they persist indefinitely, as they are not biodegradable and bioaccumulate, generating major disturbances in ecosystems. Among the methods to remediate contaminated zones, we find phytoremediation, is a technique based on the use of plants that can tolerate, bioaccumulate, stabilize, or degrade pollutants. Therefore, the aim of this study was to evaluate the potential of *Senna multiglandulosa* to tolerate and accumulate Li, Ag, and Cu. *S. multiglandulosa* seeds were inoculated in culture tubes containing MS medium and different concentrations of Li, Ag, and Cu (0, 0.01, 0.1, and 1 mM) for 50 d. The seedling showed a high degree of tolerance to treatments with metals, with germination rates of 95-100%, a high survival rate (>82%), and no effect on growth. While a pattern of accumulation was observed for Ag>Cu>Li (379.53, 276.1, and 12.69 mg/kg biomass, respectively), having a higher concentration in the root in all treatments generated a translocation factor (FT)<1, suggesting that *S. multiglandulosa* is a phytostabilizing species; however, it shows a bioaccumulation factor (BF)>1, so it can be considered a hyperaccumulation species. Regarding the physiological responses, the highest content of phenols was determined in the root compared to the shoot and the control. On the other hand, the highest content of flavonoids was observed in the shoot of the seedling exposed to the metals. Regardless of the concentrations and type of metal, the content increased at least four times compared to the control treatment. These results demonstrate that *S. multiglandulosa* seedlings are tolerant to Li, Ag, and Cu with a phytostability strategy in the process of metal extraction and that the content of phenols, flavonoids, and antioxidant activity is associated with the responses to counter oxidative stress resulting from the extraction of metals.

EMTM01C

Susceptibility of M. oleifera seed oil to oxidative stress

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Lipids are a concentrated source of energy, fat-soluble vitamins, essential fatty acids, flavor carriers, and many bioactive compounds with an important role in maintaining the body's physiological functions. *Moringa oleifera* is widely cultivated in Mexico with a seed that contains oil from 35 to 40%. *M. oleifera* oil (MOO) has a light yellow color with a slightly nutty flavor and the fatty acid composition suggests that MOO is well suited for edible and non-edible applications.

Extraction by pressing at high temperature is an efficient method for obtaining MOO that avoids the use of solvents. However, high temperature can affect both the physicochemical and antioxidant properties of the product and affect its quality. In addition, it is known that “trans fatty acids” and conjugated linoleic acid can be altered by the thermal process. Therefore, the acidity index, peroxide, iodine, and saponification were evaluated as physicochemical tests, and the DPPH, ABTS, FRAP, and total phenol content assays to determine the presence of biomolecules with antioxidant capacity. Finally, the susceptibility of MOO to tert-butyl hydroperoxide (tBuOOH, 0.42 mM) as an initiator of oxidative stress was evaluated. The results were 0.71 mg KOH/g for acid number and 1.74 meq O_2/kg for peroxide, which suggests that the oil presents a higher percentage of fatty acids. On the other hand, flavonoids and alkaloids present an antioxidant activity of 17.30% and the phenol content was 8.58 mg trolox equivalents. In adittion, the results of MOO susceptibility to tBuOOH were determined by the change in absorbance at 220 nm after 30 min, MOO neutralized 25% of tBuOOH and this result was similar to the effect of trolox (30 µM) with 18% neutralization.

Finally, dry extraction at high temperatures maintains low levels of oxidation and acidity while also preserving the antioxidant qualities of the oil's phenols and tocopherols. MOO is exceptionally resistant to auto-oxidation, it can be utilized as an antioxidant to stabilize commercial edible oils over time.

Key words: *Moringa oleifera* oil, oxidative stress, and oxidative stability.

EMTM02C

Reactive oxygen species (ROS) regulate macrophage migration inhibitory factor (MIF) secretion in breast cancer

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Introduction: Macrophage migration inhibitory factor (MIF) is a cytokine that plays a significant role in cancer progression and immune response regulation. It has been associated with promoting tumor growth, angiogenesis, and metastasis in various types of cancer, including breast cancer. Reactive oxygen species (ROS), which are generated as byproducts of cellular metabolism, can influence MIF expression and secretion. High levels of ROS have been related to MIF production in cancer cells. **Methodology:** Four breast cancer cell lines were used (Estrogen receptor positive MCF-7 cell line and triple negative MDA-MD-231, BT-549 and MDA-MB-468 breast cancer cells), which were treated with n-acetylcysteine (NAC) at concentrations of 1, 3.5, 7 and 15 µM. Viability was estimated using cristal violet staining and its espectrophotometrical quantification. To quantify MIF, we used the enzyme-linked immunosorbent assay (ELISA) MIF human protocol. For protein quantification, the Bradford protocol was performed. **Discussion:** NAC treatment at concentrations of 3.5 and 7 micromolar in breast cancer cells led to a significant decrease in MIF secretion, probably by reducing ROS levels and restoring cellular redox balance. NAC treatment also decreased cell proliferation, probably related to the reduction in MIF secretion, suggesting a potential role for MIF in the promotion of cancer-related processes. **Conclusion:** Antioxidant treatment modulated proliferation and MIF secretion from breast cancer cell lines, potentially impacting tumor growth. By reducing ROS-induced MIF secretion, antioxidants may help disrupt the pro-tumorigenic effects of MIF in breast cancer.

EMTM03C

New double labeled technique with probe for detection of intracellular reactive oxygen species (ROS) and reactive nitrogen species (RNS) in activated J774A.1 macrophages

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Introduction. The detection of reactive species *in vitro* (cell lines) is mainly based on using colorimetric chemical reactions which aren't specific for the detection of intracellular reactive species content. The use of probe label is a technique with greater sensitivity and specificity activity compared with chemical reactions. In this work was innovating in the development of a new double labeling technique with probes for the detection of two types of reactive species: reactive oxygen (ROS) and nitrogen (RNS) species in macrophages activated trough of a challenge between enterohemorrhagic *E. coli* and different stimuli from *E. coli* Nissle 1917 probiotic and subsequently evaluation through flow cytometry. **Methodology.** The macrophages were stimulated with EHEC for 6 h of incubation at 37 °C/5% CO_2. Then every assay was stimulated with *E. coli* Nissle 1917 immunobiotic (IB: viable probiotic), paraprobiotic (PP: inactivated probiotic) and postbiotic (PB: cell free supernatant probiotic) for 24 h under the same conditions. The labeling procedure was started 60 min before the end of the incubation time for each assay. The CellROX® Deep Red (5 µM) probe was added, and the tubes were incubated for 30 min at 37 °C in 5% CO_2 in the dark. After that, the DAF-FM DA probe (5 µM) was added to the tubes, and they were incubated for another 30 min. A separated tube of macrophages with the same assay conditions were labeled with 4 µL of conjugated-APC anti-mouse F4/80 antibody incubating this for 30 min in darkness. The analysis was performed on a BD FACSLyric™ Flow Cytometry System and the data analyzed in GraphPad Software. Results. The results show that there is a decrease in the RNS production when macrophages once stimulated with the EHEC strain, were stimulated with each of the probiotic strain EcN stimuli, presenting decreasing percentages of 29.36% for EHEC-IB, 27.71% for EHEC-PP and 31.93% for EHEC-PB, taking as reference macrophages stimulated only with the EHEC strain. In the case of the ROS detection only EHEC-PB presented a decrease of 73.42% meanwhile a ROS increased was presented in EHEC-IB and EHEC-PP. In general, most of the assays showed a higher production of ROS compared to RNS. There was a significative difference between EHEC and post treatment with all EcN stimuli for ROS and for RNS there's no significative difference. **Conclusion.** The immunoregulatory capacity of probiotic stimuli was demonstrated in macrophages previously activated with an EHEC strain. In most of the experiments, the amount of ROS was higher than that of RNS, indicating the presence of different activation mechanisms and a certain selectivity.

EMTM04C

Determination of intracellular reactive oxygen (ROS) and nitrogen (ERN) species production in activated J774A.1 macrophages using flow cytometry

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Introduction. *Bifidobacterium* genre has shown ability for immune system regulation through anti-inflammatory effects which are helpful in the treatment of infection or inflammatory diseases. Between immune cells involved there are macrophages, which once are activated they performed the Reactive Oxygen Species (ROS) and Reactive Nitrogen Species (RNS) production which is a process involved in the pathogen defense tactic and the induction and outlining of the immune response. **Methodology.** The macrophages had the first activation with EHEC for 6 h of incubation at 37 °C/5% CO_2. Then the second stimuli with *Bifidobacterium animalis* subsp. *lactis* HN019 immunobiotic (IB: viable probiotic), paraprobiotic (PP: inactivated probiotic) and postbiotic (PB: cell free supernatant probiotic) was contacted for 24 h under the same conditions. The labeling procedure was started 60 min before the end of the incubation time for each assay. The CellROX® Deep Red (5 µM) probe was added, and the tubes were incubated for 30 min at 37 °C in 5% CO_2 in the dark. After that, the DAF-FM DA probe (5 µM) was added to the tubes, and they were incubated for another 30 min. A separated tube of macrophages with the same assay conditions were labeled with 4 µL of conjugated-APC anti-mouse F4/80 antibody incubating this for 30 min in darkness. The analysis was performed on a BD FACSLyric™ Flow Cytometry System and the data analyzed in GraphPad Software. Results. The results show that there is a decrease in the ROS production when macrophages previously stimulated with the EHEC strain, were stimulated with each of the probiotic strain HN019 stimuli, presenting decreasing percentages of 30.58% for EHEC-IB, 28.58% for EHEC-PP and 11.53% for EHEC-PB, taking as reference macrophages stimulated only with the EHEC strain. In the case of the RNS detection only EHEC-PP presented a decrease of 24.95% meanwhile a RNS increased was presented in EHEC-IB and EHEC-PB. For ROS there was a significative difference between macrophages stimulated with EHEC and post treatment with IB and PP HN019 stimuli, meanwhile for RNS there's no significative difference. **Conclusion.** The activation of the macrophages measured through the generation of RNS and ROS depends on probiotic stimuli. It was detected an immunoregulatory activity in the probiotic stimuli through ROS and RNS detection with probe label.

EMTM05C

Oxidative stress induced in tadpoles of Lithobates catesbeianus by diatomaceous earth

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Sustainable agriculture requires new control alternatives that support and eliminate the use of agrochemicals (persistent and with environmental impact). Currently, integrated pest management promises a sustainable change where pesticides of natural origin with low toxicity such as diatomaceous earth (TD) are applied. The TD is of sedimentary origin (fossil remains of unicellular algae, composed of silicates), each silicate (Si) has a complex tetrahedral ion as its main compound; which consists of the union of a Si ion with four oxygen atoms. They have unique surface acidity and ion exchange properties. Si has been shown to protect against aluminum toxicity (Al) in various plants and fish. Bullfrog (*Lithobates catesbeianus*) tadpoles were used to assess whether TD generates oxidative stress, since during their aquatic life cycle they are very sensitive to physicochemical changes in water. If the optimal water conditions are adverse, its life cycle is prolonged longer, otherwise it stops. The objective of this work was to test whether TD generates oxidative stress in *L. catesbeianus* during 96 h of exposure. A TD sedimentation test was performed at concentrations (100, 1000, and 10,000 ppm). Oxidative stress was determined by spectrophotometry (lipoperoxidation (LPPx), superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) activity). In addition, acetylcholinesterase (AChE) activity was evaluated. The data suggest that during 96 h of exposure to TD in *L. catesbeianus*, the 10,000 ppm concentration remains in suspension. While the LPPx reports damage from 24 h to 96 h with a significant difference compared to the control. For its part, the activity of the SOD reports a gradual increase in damage from 6 h and had an increase of almost three times at 96 h; Regarding the activity of the CAT, this increased gradually until 96 h; Finally, the activity of the GPx showed a gradual increase in its activity from 24h to 96h. Finally, the evaluation of neurotoxicity did not show significant differences with respect to the control.TD has been considered an effective agent to control various pests, whose mechanism of action is mainly mechanical, damaging the anatomical structure of the organisms to which it is directed, tadpoles are organisms sensitive to any xenobiotic found in the water, under Under Sotkes' law conditions no significant changes were observed in sedimentation of TD concentrations. The TD caused oxidative stress at a concentration of 10,000 ppm from 6 h, gradually up to 96 h of the test.

EMTM06C

Hydrogen peroxide detection on interface-engineered atomically dispersed Co material / N-doped carbon composites

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Atomically dispersed materials (ADMs) present low coordination numbers, relatively high surface area, and high metal availability, making them highly active materials for electrochemical reactions. In this work, we obtained Co ADMs using the zeolitic imidazolate framework (ZIF-67), analyzing the calcination temperature (500-700 °C). Additionally, we performed the interface engineering by combining the ADM with a mixture of multi-walled carbon nanotubes and graphene containing N heteroatoms (G-CNT). The ZIF-67, a promising material for peroxide detection, was synthesized using a hydrothermal method and then calcined at different temperatures (500, 600, and 700 °C) in a nitrogen atmosphere. The electrocatalytic activity of the materials was assessed in a 0.1 M phosphate buffer (pH 7.4). The results indicated that the best detection was achieved with the material calcined at 700 °C, presenting a low detection limit of 2.88 ppm (S/N=3), a broad linear range between 20 and 100 ppm, and a limit of quantification of 9.61 ppm obtained). The effect of the calcination temperature on the detection limits of Co ADMs was studied by Differential Pulse Voltammetry (DPV). The DPV voltammogram presented two well-defined peaks, one related to a chemisorptive process and the other to a Faradic process. The first was related to the chemical adsorption of H_2O_2, and the second to the further reduction of OH- species. The materials presented shifts in the potential for the adsorption process, which together with chemical calculations, was attributed to changes in the electronic structure of Co ADM. These findings validate the results obtained through Cyclic Voltammetry (CV) and agree with the calculated absorption energy values. The herein synthesized material also displayed excellent reproducibility, while the good detection limit was attributed to the electronic enhancement from the interface engineering, and the better cobalt dispersion over the carbonaceous material.

EMTM07C

Myoglobin protection ratio of *Lippia alba* extracts against reactive oxygen species

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There are multiple research lines associated with reactive oxygen species (ROS), one of them is the search for antioxidants of natural origin, either in foods or plants that, through their regular consumption, help to reduce oxidative stress. In this context, *Lippia alba* is a plant that has been widely studied in South America, becoming recognized for presenting multiple therapeutic properties and an important antioxidant potential. Although the plant has not been widely studied in Mexico, inhabitants of some regions have used it for centuries to treat multiple diseases; however, the way it is used and its effects are not consistent with what has been reported in South America. There are several *In vitro* and *in vivo* methods that allow the evaluation of the antioxidant capacity of a sample, each with its respective advantages and disadvantages. The myoglobin protection ratio method is a spectrophotometric technique proposed by Terashima et al. (2012), in which physiological conditions of PH and temperature are simulated and the myoglobin protein (naturally responsible for transporting oxygen to muscle tissue) is exposed to the action of different ROS, allowing the evaluation of multiple samples and a closer approximation to their possible mechanisms before *in vivo* methods. Under this method, different concentrations of methanolic extracts from the stem, flower and sheet of the plant were evaluated, as well as an aqueous infusion obtained by the method reported by the inhabitants of the region against hypochlorite ion, peroxyl and hydroxyl radicals. The flower get an IC50 value significantly lower than the other samples, nevertheless infusion present great values, with not significantly differences with sheet. This study suggests that the antioxidant capacity of *Lippia alba* infusion could be significantly affected according to the abundance of flowers in it.

FMED01C

In silico assays suggest that morintides from *Moringa oleifera* digested with trypsin produce peptides with antioxidant, anti-inflammatory and anti-cancer properties

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The growing need to combat redox imbalance in chronic diseases has prompted researchers to concentrate their efforts on molecules with antioxidant activity. Bioactive peptides are gaining popularity as natural compounds with health-promoting effects on a variety of physiological systems. They are protein fragments containing 2-20 amino acids that can be released through a variety of methods including gastrointestinal digestion, food processing, and microbial fermentation. Recent investigations on the impact of bioactive peptides in the cellular environment show that these molecules might exert their action by modulating particular molecular pathways (Tonolo F., et al., 2023). Accurate prediction of ACPs is of great importance for the exploration of their mechanism of action and for the development of therapeutic ACPs. As the experimental identification and development of novel ACPs is a very time-consuming and labor-intensive process, therefore bioinformatics tools are needed for effective analysis of the available big data on existing peptides so as to allow the identification of novel ACPs while also shedding light about their mechanism of action. Shruthi G. Kini et al., (2017) isolated and characterized of two novel 8C-hevein-like peptides, designated morintides (mO1 and mO2), from the drumstick tree *Moringa oleifera*. Proteomic analysis revealed that morintides comprise 44 amino acid residues and are rich in cysteine, glycine and hydrophilic amino acid residues such as asparagine and glutamine. To find out if mO1 and mO2 have potential therapeutic and biotechnological effects, we carried out a bioinformatics study to perform a simulated digestion with trypsin using PeptideCutter of Expasy and later analyze the medicinal properties of the resulting peptides using multiple web servers. Six peptides of multiple sizes were obtained, of which the peptide GGGGAGGAGGGAGGGSP presented the most interesting characteristics because it has antioxidant (score of 0.9993), anti-angiogenic (score of 0.55) and anti-cancer (score of 0.57) properties with the use of AnOxPP and CSM-peptides servers, respectively. In addition, the anticancer activity was reaffirmed with the servers ACPred (score of 0.756) and AntiCP (score of 0.65) and through AcPEP predicts that the peptide has a high therapeutic activity against breast cancer of 84.953 µM, while for the treatment of cervical, colon, skin and prostate cancer, the concentration is in the range of 240.07 to 450.02 µM are required. Keywords: Biopeptides, bioinformatics tools, morintides, Moringa oleifera.

FMED02C

L-theanine: a cardioprotective agent against ischemia/reperfusion-induced mitochondrial damage

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Ischemia and reperfusion (I/R) injury to the myocardium is caused by the exacerbated generation of reactive oxygen species that promote loss of mitochondrial function, making cardiomyocytes more susceptible to the deleterious effects of I/R. In this sense, L-theanine (TEA), a non-protein amino acid isolated from *Camellia sinensis* leaves, has antioxidant and anti-inflammatory properties that could prevent mitochondrial damage generated during I/R. Therefore, we explored the effect of TEA treatment on mitochondrial function, the ability to buffer calcium overload and preserve membrane potential in mitochondria isolated from rat hearts undergoing I/R. Four groups of rats were used: control (CT) and I/R received intragastric distilled water for 10 days, while TEA and TEA+I/R were treated with 250 mg/Kg of TEA. On day 11, hearts were removed and mounted in a Langendorff perfusion system. CT and TEA hearts were perfused for 90 min, while I/R and I/R+TEA hearts underwent global ischemia for 30 min, followed by 60 min of reperfusion. The hemodynamic parameters and the size of the infarct in the hearts were measured. Subsequently, cardiac mitochondria were isolated and their function was assessed by measuring the rate of oxygen consumption, calcium retention capacity, and inner mitochondrial membrane potential (ΔΨm). I/R hearts had a significant reduction in cardiac function and increased infarct size compared with CT, TEA, and TEA+I/R hearts. In addition, mitochondrial dysfunction was evidenced in I/R hearts due to the significant reduction in State 3 respiration, poor respiratory control, reduced phosphorylation efficiency, alterations in ΔΨm, and inability to buffer calcium release; such changes were not observed in CT and TEA hearts. Remarkably, mitochondrial function was significantly improved in the TEA+I/R group compared to the I/R group ($p<0.05$). Taken together, our findings demonstrate that TEA protects hearts against I/R-induced mitochondrial damage.

FMED03C

Effect of major terpenes of *Callistemon citrinus* on fat deposition and IL-6, TNF-α, adiponectin and, leptin levels in the liver of rats fed with high-fat diet

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Intake of high fat and sugar diets produces an excess of adipose tissue, which is associate with obesity, insulin resistance, cardiovascular diseases an oxidative stress. The increase in saturated fat activates various signaling pathways that promote an increase in reactive oxygen species and inflammation in different cells, affecting cell function. Terpenes are a natural product found in plants these compounds have many biological activities. *Callistemon citrinus* is belonging to the Myrtaceae family, which has a high number of these compounds. In the present study, the effects of the major terpenes of *Callistemon citrinus* (1-8-cineol, limonene, and α-terpineol) on fat deposition and interleukin (IL-6), tumor necrosis factor alpha (TNF-α), leptin and adiponectine were determined in rats fed with a high-fat diet model. 36 male albino Wistar rats were randomly divided into six groups (n=6). Group I was the control; group II was a high-fat diet (63% of the Purina® Rodent Chow food, 41.66% of INCA® vegetable fat, 41.66% of lard, and 16.66% of sucrose). Group III, IV, V, and VI were administered orally with terpenes at different concentrations (1-8-cineol at 0.88 mg/kg, limonene at 0.43 mg/kg, α-terpineol at 0.32 mg/kg, and terpenic mixture of those), and the high-fat diet daily for 15 weeks. The results showed increased of fat deposition and high levels of pro-inflammatory cytokines: TNF-α, Il-6, and, leptin was found in rats liver of the high-fat diet group compared to the control group. Conversely, the levels of the pro-inflammatory cytokines and, fat deposition in the groups with 1,8-cineole, α-terpineol, and limonene plus the high-fat diet were decreased. Low adiponectin level was found in the high-fat diet group. Inconclusion, major terpenes of *Callistemon citrinus* reduced the pro-inflammatory cytokines and fat deposition, indicating its potential anti-inflammmatory and anti-obesogenic properties.

Keywords: Natural compounds, Inflammation, Cytokines.

FMED04C

Evaluation of Oxidizing-Reducing System Alterations in Erythrocyte Cytosol of Neonates Born to Women with Preeclampsia

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Oxidation-reduction (redox) reactions play a crucial role in fundamental redox regulation processes known as "redox signaling" and "redox control." However, an imbalance in this redox balance towards oxidation, commonly referred to as "oxidative stress," can have detrimental effects. Oxidative stress occurs when there is an imbalance between pro-oxidant secretion and antioxidant capacity and is a significant factor in the pathophysiology of various pregnancy complications. Preeclampsia (PE), characterized by arterial hypertension after 20 weeks of gestation, is a severe pregnancy disorder that falls under hypertensive disorders and affects approximately 15% of pregnancies worldwide. The main aim of this study was to evaluate alterations in the cytosol of erythrocytes obtained from umbilical cord arterial blood by assessing antioxidant enzymatic activity and markers of oxidative damage. A case-control study design was implemented, consisting of neonates born to healthy women (n=30) and neonates born to women with preeclampsia (n=30). The activity of antioxidant systems was measured using Caiman kits, while the concentration of lipid and protein oxidation markers was quantified through spectrophotometry. The results revealed a significant increase in antioxidant activity in cases of Superoxide Dismutase (SOD, p=0.0001), Glutathione peroxidase (GPx, p=0.0001), and Glutathione reductase (GR, p=0.0001). However, there was no significant change in Catalase (CAT, p=0.22). Comparing neonates born to women with PE with the control group, there was a significant increase in oxidation markers: Malondialdehyde (MDA), protein carbonylation, and Lipohydroperoxides (LPH) (p=0.0087, p=0.0365, p=0.0222, respectively). Based on the results, it can be inferred that the concentrations of H_2O_2 present in the cytosol of these neonates are sufficient for GPx to effectively reduce it to water, making the involvement of CAT enzymatic activity unnecessary. The increase in antioxidant enzyme activity observed suggests an effort to counteract oxidative damage in these neonates. Therefore, it is essential to consider the implementation of antioxidant therapy to restore the imbalance in oxidation-reduction in future projects. Keywords: Oxidative Stress, Antioxidant Enzymes, Preeclampsia, Neonates, Erythrocytes.

FMED05C

Anti-obesogenic effect of leaf phytosomes of *Callistemon citrinus* on anthropometric and biochemical parameters in Wistar rat

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Phytosomes are standardized plant extracts or water-soluble phytoconstituents with phospholipids to produce lipid-compatible vesicular structures. *Callistemon citrinus* leaf extract has demonstrated its potential antioxidant effect and as a possible anti-obesogenic agent in *in vivo* studies. The biological activity of *C. citrinus* has been attributed mainly to phenolic compounds and terpenoids previously described. In the present work, the anti-obesogenic effect of *Callistemon citrinus* leaf phytosomes on anthropometric and biochemical parameters in Wistar rats was evaluated. Forty-eight two months old male Wistar rats were randomly divided into 8 treatments of 6 rats. Only the groups were fed with a high-fat diet (HFD): group 1 (HFD control), 3 (HFD plus anti-obesogenic drug), 6 (HFD plus 50 mg/kg phytosomes), 7 (HFD plus 100 mg phytosomes) and 8 (HFD plus phytosomes 200 mg/kg). Group 3 was administered Orlistat (5 mg/kg of body weight/orally daily), in the case of group 6, 7 and 8 the phytosomes loaded with the ethanolic extract of the *Callistemon citrinus* leaf (50, 100 and 200 mg/kg body weight/orally daily, respectively). Group 2 was given balanced pellet (normal control), in addition, group 4 was given pellet plus vehicle (oral liposome daily) and group 5 was given pellet *plus Callistemon citrinus* leaf extract (200 mg/kg body weight/orally daily). At 15 weeks of the experimental procedure, the morphometric parameters were measured, and the blood was taken to obtain the biochemical parameters. The results showed that the rats treated with the phytosomes (50, 100 and 200 mg/kg) of the *Callistemon citrinus* leaf had significantly lower body weight, morphometric parameters and biochemical parameters than the group fed a high-fat diet ($p>0.05$). This study suggests that *C. citrinus* leaf phytosomes could be used as treatment or prevention of obesity, due to the antioxidant effect on biochemical indicators and anti-obesogenic effect on anthropometric parameters during this condition.

Keywords: Ethanolic extract, Pharmacological vehicle, Antioxidant.

FMED06C

Antioxidant effect of the main terpenes of *Callistemon citrinus* on the liver of rats fed with a hypercaloric diet

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Free radicals (RL) are unstable molecules associated with cell damage. These LR are generated through endogenous and exogenous factors, including a hypercaloric diet. As a consequence of a high concentration of RL, it leads to an imbalance of the normal redox state, causing oxidative stress and with it chain reactions in multiple biomolecules. However, the body has a protection system made up of an enzymatic and a non-enzymatic system. Superoxide dismutase, catalase, glutathione peroxidase, glutathione-s-transferase, paraoxonase, and quinone reductase are antioxidant and detoxifying enzymes capable of stabilizing and protecting living organisms against cell damage.

In the present work, groups were developed which was the antioxidant effect of the main Callistemon citrinus terpenes on the liver of male Wistar rats fed with a hypercaloric diet, for which 6 were divided with an n=6; Control, high-fat diet, limonene, α-terpineol, 1,8-cineole and the mixture of all 3. The experimental treatments were administered orally daily at different concentrations for 15 weeks. Finally, the enzymatic activity will be extended in all the treatments, achieving that the groups treated with limonene, 1,8-cineole, α-terpineol and the mixture increased the enzymatic activity in the different treatments with respect to the control group. Last but not least, we can conclude that the terpenes in C. citrinus favor antioxidant activity against oxidative damage caused by RL induced by a hypercaloric diet.

Keywords: Free radicals, oxidative stress, hypercaloric diet, enzymatic activity, terpenes.

FMED07C

S-allylcysteine treatment decrease the alterations and the increase in RIP3 levels induced by ischemia/reperfusion

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Stroke is a leading cause of death and disability worldwide. Stroke is caused by the obstruction of a blood vessel, leading to death of cells by different mechanisms, such as the necroptosis. Morphologically, necroptosis is like necrosis; however, it is not an accidental event, and requires the activation of a signaling pathway. Phosphorylation of RIP3 kinase is required for activation of necroptosis as well as the formation of pores around the cell that allow the uncontrolled passage of cell content, increasing inflammation and tissue damage. In this context, necroptosis is a pathway that represent a therapeutic target. S-allylcysteine (SAC) is a molecule that has shown a protective effect on the alterations induced by ischemia/reperfusion (I/R), effect mainly associated with its antioxidant properties; however, the mechanisms involved in this protection are not completely known, so in this work the effect of SAC on necroptosis activation was explored using a model of middle cerebral artery occlusion. Male Wistar rats were used and divided into 3 groups: control (subjected to the surgical procedure without occlusion of the artery), and the IR and IR+SAC groups, both groups were subjected to 1 h of ischemia and 48 h of reperfusion. IR+SAC group received 3 doses of 100 mg/kg SAC; the first, onset reperfusion and two more every 24 h; the animals were sacrificed 48 h after the start of reperfusion.
In this work, the protective effect of SAC was corroborated, since the treatment reduced motor deficit, morphological damage, and the number of cells in neurodegeneration induced by IR. In addition, SAC treatment decreased the increase in RIP3, 8-hydroxy-2'-deoxyguanosine and p65 levels induced by IR in the cortex and striatum. Nitric oxide synthase 1 (NOS1) levels did not change after IR in the cortex; however, the percentage of positive cells to NOS1 was higher in the striatum, and this increase was decreased by SAC treatment.The protective effect of SAC in the middle cerebral artery occlusion could be related with the reduction of the necroptosis, oxidative stress and the proinflammatory pathway of NFkB in striatum and cortex. The mechanism by which SAC decreases necroptosis could be through the indirect reduction of oxidant stress and inflammation.
Necroptosis, antioxidant, stroke.

FMED08C

Analysis of Oxidative Stress Markers in Colostrum of Women with Obesity Following Pregnancy

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Oxidative stress, characterized by an imbalance between reactive oxygen species (ROS) and the antioxidant system, can lead to damage to various biomolecules, including lipids and proteins. Obesity, an inflammatory condition associated with increased ROS production, can have implications during and after pregnancy, potentially affecting breast milk composition. This study aimed to investigate markers of oxidative stress in the colostrum of women with obesity compared to women with normal weight. To assess the oxidative stress status, markers such as lipohydroperoxides (LHP), malondialdehyde (MDA), protein carbonylation (PC), dityrosines (DT), and total antioxidant capacity (CUPRAC) were measured in the colostrum of 21 obese women and 17 women with normal weight as the control group. The results revealed a significant increase in markers of oxidative damage to lipids in the colostrum of women with obesity compared to the normal weight group, specifically for LHP ($p=0.0067$) and MDA ($p=0.0201$). Additionally, markers of protein damage, PC and DT, were significantly higher in the obese group compared to the normal weight group ($p=0.0133$, $p=0.0006$, respectively). Furthermore, the obesity group exhibited a decreased antioxidant activity, as quantified by the CUPRAC method, in comparison to the control group ($p=0.0011$). Based on these findings, it can be inferred that obesity during pregnancy contributes to the modification of colostrum biomolecules, resulting in an elevation of oxidative damage markers in lipids and proteins, accompanied by a decrease in protein antioxidant systems. However, it should be noted that breast milk is a dynamic biological fluid, and it is possible that non-protein antioxidant mechanisms are implemented to counteract oxidative damage during the colostrum stage. Considering the vital importance of colostrum as the first source of nutrition for newborns, further research is warranted to elucidate these non-protein antioxidant mechanisms in breast milk.

Keywords: Obesity, Oxidative Stress, Colostrum, Lipid Damage, Protein Damage, Antioxidant Capacity.

FMED09C

Evaluation of the Antioxidant Activity of Levetiracetam in a Temporal Lobe Epilepsy Model

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Temporal lobe epilepsy (TLE) accounts for approximately 60% of all focal epilepsies. TLE is a neurological disorder in which oxidative stress (OS) has been shown to be involved in epileptogenesis. Recently, it has become clear that seizure control with anticonvulsant drugs (ASD) to treat epilepsy is insufficient, as comorbidities do not tend to diminish, on the contrary they often progress. Therefore, research into undescribed properties of known ASD that can be used to benefit patients has been encouraged. The literature has shown that some ASD have neuroprotective properties. Levetiracetam (LEV) is a second-generation and first-line ASD for the treatment of TLE; its efficacy, tolerability, and pharmacokinetic profile have been favorable in the treatment of this type of epilepsy, and in some studies, it has been found to possess antioxidant properties. Since the antioxidant effects of LEV have not been demonstrated in the chronic phase of epilepsy, the objective of this study was to evaluate, for the first time, the effects of LEV on the oxidant-antioxidant status in the hippocampus of rats with chronic phase TLE, by quantifying the antioxidant activity of four antioxidant enzymes and the level of three markers of oxidative stress in the hippocampus of an animal model of chronic phase TLE. LEV administration in rats with TLE significantly increased superoxide dismutase (SOD) activity, increased catalase (CAT) activity, but did not change glutathione peroxidase (GPx) activity, and significantly decreased glutathione reductase (GR) activity in comparison with epileptic rats. LEV administration in rats with TLE significantly reduced hydrogen peroxide (H_2O_2) levels but did not change lipoperoxidation and carbonylated protein levels in comparison with epileptic rats. LEV showed significant antioxidant effects in relation to restoring the redox balance in the hippocampus of rats with TLE. However, further studies in experimental models and clinical trials are needed to demonstrate the antioxidant capacity of LEV to explain the biochemical and molecular mechanisms underlying the direct and indirect antioxidant effects of this drug. And in the future to use the antioxidant properties of LEV to improve the treatment of patients not only with ELT, but also with other types of epilepsy.

FMED10C

Challenging baldness: hair growth by BFBP through activation of NRF2 and inhibition of lipoperoxidation and apoptosis

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Introduction. One of the main factors contributing to hair loss is a redox imbalance, where the activity of the endogenous antioxidant system is reduced, while lipoperoxidation and apoptosis inhibit hair growth. Therefore, the objective of this study was to investigate the potential effects of a nanostructured formulation derived from the bioactive fraction of *Bacopa procumbens* (BFNB) to promote hair growth in a murine model, through activation of the antioxidant system via NRF2 and inhibition of lipoperoxidation and apoptosis.

Methodology. C57BL/6 mice were depilated on their back and head, and then topically applied with BFNB for 30 days. The study encompassed a comprehensive characterization of the follicular phases and a histomorphological analysis to evaluate the effects of topical application of BFNB. The study was reinforced by molecular evaluation of the activation of the endogenous antioxidant system, lipoperoxidation, and apoptosis. **Results.** After 15 days of BFNB application to the back and head of mice, remarkable hair growth was observed, along with increased follicle count, hair length, diameter, and thickening of the epidermis and hypodermis, compared to mice treated with vehicle or minoxidil. At the molecular level, BFNB stimulates the protein expression of PI3K and AKT. AKT, in turn, promotes the activation of NRF2, leading to a significant decrease in the major products generated by lipoperoxidation, such as 4-hydroxynonenal and malondialdehyde. Furthermore, there was an inhibition of the apoptosis process, as evidenced by a decrease in the protein expression of Caspase 3-pS11, Caspase 9-pS10, and P53. Conversely, the survival biomarker BCL2 increased.

Conclusion. Our results suggest that BFNB could represent an innovative therapy to promote hair growth by activating the endogenous antioxidant system mediated by NRF2, resulting in a reduction in lipoperoxidation and apoptosis.

FMED11C

Evaluation of the effect of hydroxycinnamic acids 2c and 2f in a model of ulcerative colitis

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Background: Ulcerative colitis (UC) is an inflammatory bowel disease (IBD) that causes long-lasting inflammation and ulcers in the digestive tract, due to excessive production of proinflammatory interleukins and reactive oxygen species such as hypochlorous acid (HOCl) that inactivates protease inhibitors and activates collagenolytic metalloproteinase, favoring protein-mediated degradation of the interstitial matrix of mucosa and epithelial cells. The current study is aimed to evaluate the protective effects of hydroxycinnamic acids on UC induced by 2,4,6-trinitrobenzenesulfonic acid (TNBS) in rats. **Methods:** Thirty-six adult Wistar rats were divided into six groups (n=6) and UC was induced in four groups using TNBS solution (50 mg/kg) in 50% of ethanol. They received different daily doses of hydroxycinnamic acids (**2c** and **2f**) with 50 mg/kg/ day. On the 15th day, the colon tissues were removed and examined regarding the macroscopic and histopathology lesions. Also, Disease Activity Index (DAI) and Myeloperoxidase (MPO), malondialdehyde (MDA), reduced glutathione (GSH) and superoxide anion (O_2^-) activities were measured in the colon homogenate. **Results:** Rectal administration of TNBS caused the induction of colon ulcers with a significant increase in MPO and MDA, and a decrease in GSH. The administration of compounds **2c** and **2f** significantly decreased the presence of ulcers, inhibited MPO in peroxidation activity and in immunofluorescence, increased GSH and decreased MDA.
Conclusion: Compounds **2c** and **2f** showed MPO inhibitory and antioxidant activity in a model of ulcerative colitis, similar to the reference compound (5-ASA).

FMED12C

Methotrexate induced antioxidant hormetic response

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Neurodegenerative diseases have increased worldwide in recent years. Its relationship with oxidative stress has motivated research to find therapies and drugs capable of suppressing oxidative damage and, therefore, delaying its progression of these diseases. Glutathione (GSH) is the most important cellular antioxidant in living beings and is responsible for regulating the cellular redox state. However, GSH cannot be administered by any route of administration, so molecules that increase its concentration have been recently studied. The main molecules used to increase GSH levels are those that activate the Nrf2-ARE signaling pathway; since Nrf2 regulates the main genes involved in the synthesis and recycling of GSH. Astrocytes are the main cells in the nervous system that participate in the antioxidant response, this response is performed by providing GSH and other substrates so that neurons have an efficient antioxidant response. Methotrexate (MTX) is a drug that is mainly used as an anti-inflammatory agent at low concentrations, however at high concentrations it is used as an anti-neoplastic agent, which makes MTX an excellent hormetic agent. With what was previously described, the objective of this work was to use MTX as an inducer of the antioxidant hermetic response. Therefore, in this study we used different concentrations of MTX at different exposure times to induce a hormetic antioxidant response in primary rat astrocytes. Our results showed that 20 nM MTX preconditioning for 12 h increased the GSH/GSSG ratio and protected cell viability against lethal MTX and H_2O_2 treatments. The antioxidant hormetic mechanism was mediated by an increase in the activation of the Nrf2 pathway. This mechanism was verified when we inhibited the activation of Nrf2 by administering Brusatol.
The results obtained are a first step in the search for alternative treatments to reduce or delay the oxidant effect in neurodegenerative diseases, where MTX could be an excellent option, since it has been shown that in addition to having this interesting antioxidant response, it is also an excellent anti-inflammatory drug.

FMED14C

Evaluation of antioxidant capacity in breast milk from women with obesity

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Obesity, a metabolic disorder, is increasingly prevalent among pregnant women. Apart from its inflammatory nature, obesity is closely associated with oxidative stress caused by an elevation in reactive oxygen species, which can have implications for processes such as breastfeeding. Considering that colostrum in breast milk contains lipids, proteins, and immunological factors that offer protection to the newborn, assessing the antioxidant capacity of colostrum in women with obesity becomes crucial. Thus, this study aimed to evaluate the protein and non-protein antioxidant activity, as well as the activity of the enzymes glutathione peroxidase and glutathione reductase, in the colostrum of 22 women with obesity and 17 women with normal weight serving as a control group. The data were subjected to statistical análisis using Student's t-test. The results indicated no significant statistical difference in non-protein antioxidant activity between the control and obese groups. However, a significant difference ($p<0.01$) was observed in protein antioxidant activity between the two groups. Furthermore, both glutathione peroxidase enzyme activity ($p<0.0001$) and glutathione reductase enzyme activity ($p<0.05$) demonstrated a statistically significant difference between the control and obese groups. These findings suggest that the antioxidant capacity of colostrum may be altered in the presence of conditions such as obesity during pregnancy. This alteration may arise frpm the increased production of reactive oxygen species in the placenta due to heightened mitochondrial activity as the maternal body mass index increases. Consequently, the health of both the mother and the newborn can be compromised.

Keywords: Antioxidant Capacity, Obesity, Breast Milk.

FMED15C

Evaluation of antioxidant and anti-inflammatory properties of *Ocimum selloi* (Lamiaceae) in mice TPA model

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Species of the genus *Ocimum* have an extensive ethnomedical and culinary use. They contain secondary metabolites useful in the treatment of chronic degenerative diseases[1]. Previous studies have shown that ethyl acetate extract of *O. selloi,* regulates the damage responses generated by chronic arterial hypertension, which was generated in a mice model by the administration during 12 weeks of angiotensin II[2].
In the present study, we have assessed the ability of ethyl acetate extract of *O. selloi* (OsEA) and fractions; OsF3, OsF7, OsF10, OsF13, OsF17 and OsF18, to inhibit the nitric oxide (ON) promotion by TPA (12-O-tetradecanoylphorbol-13-acetate) in the mouse auricular pavilion. Method: 10 µl of the TPA (2.5 µg/kg) solution was applied to the left auricular pavilion. 10 min later the treatments were placed. After 4 hours, the animals were euthanized due to overexposure to anesthetic and central sections of 6 mm in diameter were taken from the auricle, the differential of weight was a parameter of inflammation. Results: Topical administration of indomethacin (1mg/ear) inhibited 45% ear edema promotion by TPA. For the experimental groups administrated by OsF7 and OsF17 (1mg/ear) inhibited edema 53% and 74% respectively. In addition, TNFα and IL10 was measured by enzyme-linked immunosorbent assay (ELISA): OsEA (508.2 pg TNFα/mg prot), OsF7 (678.6 pg TNFα/mg prot) and OsF17 (422.8 pg TNFα/mg prot) effectively blocked TPA-induced TNFα production (1172.3 pg TNFα/mg prot). Besides, OsEA (411.3 pg IL10/mg prot) and OsF3 (423 pg IL10/mg prot) increases the concentration of IL10 compared to TPA (327 pg IL10/mg prot). Nitric oxide was measured by Griess reagent: OsF3 (14 µM NO_{2^-}), OsF7 (8.7 µM NO_{2^-}), OsF10 (9 µM NO_{2^-}), OsF13 (10.8 µM NO_{2^-}) inhibited TPA-induced NO production (18.4 µM NO_{2^-}). Conclusion: OsEA and fractions showed not only antioxidant, but also anti-inflammatory effects. The modulating activity of OsEA in the TPA-induced oxidative stress, could be associated with compounds of the polyphenol type, such as, rosmarinic acid, caffeic acid and oleanolic acid. Besides, in a thin layer chromatography of OsF13 and OsF17, we found flavonoids and derivates of caffeic acid.

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FMED16C

Correlation of oxidative stress (OE) with interleukins in Irritable Bowel Syndrome (IBS)

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Introduction: IBS is a multifactorial disorder that includes immune activation/low-grade inflammation. (Bashashati et al. NGM 2014) Also, we have previously shown greater oxidative stress (OS) in IBS vs. controls given by higher levels of malondialdehyde (MDA) and lower antioxidant systems: reduced glutathione (GSH), and GSH/GSSG ratio. (Morales-Guzmán et al. Panamerican Congress of Gastroenterology 2021) Further, OS has been related to inflammation (Nathan et al. Cell 2010), however, this has not been evaluated in IBS. **Aims:** To determine the molecular processes related to the activation of the immune system, OS, and low-grade inflammation to try to clarify the pathophysiological mechanisms of IBS. **Methods:** We included 30 patients with IBS-Rome III from the Gastroenterology outpatient clinic of the General Hospital of Mexico and 30 IBS-negative controls. Those with organic diseases (diabetes, autoimmune, IBD, celiac, recent infections) were excluded. Previously, the venous-blood level of MDA was determined by the Draper and Hadley method, and GSH, GHSSG, by the commercial quantification kit Sigma-Aldrich. IL-10, IL-4, IL-6, and TNF-α were determined by a milliplex immunoassay [Cat. HCYTOMAG-60K-06]. The expression of the transcription factor nuclear factor kappa B (NF-kB) and nuclear factor 2 derived from erythroid 2 (Nrf2) was evaluated by means of western blot. Statistical analysis was conducted by the Mann-Whitney U test and Tukey's HSD when appropriate. A $p \leq 0.05$ was considered significant. **Results:** The IBS were classified in IBS with Constipation (IBS-C), Diarrhea (IBS-D), Mixed (IBS-M), and Unclassified (IBS-U). In IBS vs. controls, there were lower levels of IL-10: $p<0.001$ and IL-4: $p<0.01$; and higher IL-6: $p<0.001$ and TNF-α: $p<0.001$. According to the IBS subtypes, there were higher levels of TNF-α in IBS-D vs IBS-C: $p=0.05$. OS did not correlate with cytokines in IBS or controls; however, within IBS-D there was a high correlation in GSH and IL-4: $r=0.99$ ($p=0.04$); while in IBS-C, MDA correlated with IL-4: $r= 0.77$ ($p=0.03$) and TNF-α with IL-10: $r= 0.74$ ($p<0.001$). There were no correlations in IBS-U. Finally, a higher expression of the NF-kB was observed in IBS compared to controls: $p=0.00$; and a lower expression of the Nrf2 in IBS vs. controls: $p=0.01$. **Conclusion:** Our data confirm that with IBS there is an imbalance in the physiological homeostasis of the cellular redox state and the response to stress that can generate inflammation and immune activation. On the other hand, the differences in the relationship between OS and IL according to the IBS subtypes suggest different pathophysiological mechanisms in the IBS subgroups.

FMED17C

Effect of Chronic Exposure to Low Doses of O_3 on NFκB activation in the Jejunum and Hippocampus

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Environmental pollution by ozone produces an increase in the formation of reactive oxygen species (ROS) in the organism and a deficit in antioxidant defenses, which causes a state of oxidative stress, which is present in the maintenance and evolution of chronic degenerative diseases. ROS are known to participate in the cellular and molecular mechanisms that lead to loss of intestinal permeability. Lately, there is evidence that relates alterations in both the microbiota and intestinal permeability with neurodegenerative diseases. In these degenerative processes, both the chronic state of oxidative stress and the loss of regulation of the inflammatory response form a vicious circle that maintains and increases the degenerative process over time. On the other hand, the nuclear factor that enhances the kappa light chains of activated B cells (NF-kB) is a regulator of the immune response, since when translocated to the nucleus it regulates the synthesis of cytokines. The objective of this work was to study the effect of repeated exposure to ozone on the NFkB factor and its translocation to the nucleus, both in the jejunum and in the hippocampus of rats. For this purpose, 24 male Wistar rats were used, with water and food ad-libitum, which were divided into 6 groups that received one of the following treatments: Group 1, Control; Groups 2, 3, 4, 5 and 6 exposed to ozone for 7, 15, 30, 60, and 90 days respectively (the ozone dose was 0.25 ppm for 4 h daily). Once the treatment was finished, the jejunum and hippocampus were processed, with the Western-Blot technique and immunohistochemistry according to NOM062-ZOO-1999 adjusted to the international guidelines of ethics and animal management. The data obtained were analyzed and the Kruskal Wallis and Mann-Whitney U test were used. The results show that both NFkB and its translocation to the nucleus show a significant increase in the jejunum at 7 and 30 days, in addition to a significant decrease at 90 days of exposure ($p < 0.05$). The hippocampus shows a significant increase at 7 and 30 days ($p < 0.05$). These results show that repeated exposure to low doses of ozone causes an increase in NFkB as well as its translocation to the nucleus in both jejunum and hippocampus. This implies an increase in the synthesis of inflammatory response cytokines, which generates the loss of regulation of the inflammatory response that occurs in chronic degenerative processes. This progressive degeneration process may be similar to what occurs in Parkinson's disease.
Keywords: Ozone, oxidative stress and degenerative disease.

Acknowledgment: Project funded by the Dirección General de Asuntos del Personal Académico-UNAM (PAPIIT IN221521) a S.R-A

FMED18C

Evaluation of Ischemia-Modified Albumin and its Relationship with Free Fatty Acids in Neonates Born from Women with Preeclampsia

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Preeclampsia (PE) is a multifactorial disease characterized by placental ischemia, resulting in generalized vascular endothelium dysfunction and increased oxidative damage. This damage may be associated with the chemical modification of various molecules, including ischemia-modified albumin (IMA). However, recent studies have suggested that IMA might be generated because of elevated free fatty acids (FFAs) rather than direct oxidative stress. In line with this, our study aimed to evaluate albumin modified by ischemia and its relationship with free fatty acids in neonates born to women with preeclampsia, comparing them with neonates born to women without PE (control group). We included 30 neonates born to women without a diagnosis of PE (control group) and 30 neonates born to women with a diagnosis of PE. Ischemia-modified albumin (IMA), free fatty acids (FFAs), as well as biomarkers of lipid damage including conjugated dienes (CD), malondialdehyde (MDA), and lipohydroperoxides (LHP), were quantified in plasma. Additionally, biomarkers of oxidative damage to proteins, such as reduction of nitroblue tetrazolium (NBT), protein carbonylation (PC), and dityrosines (DT), were assessed. Our findings revealed a significant increase in IMA ($p=0.0001$) and FFAs ($p=0.0023$) in neonates born to women with PE compared to the control group. Furthermore, the problem group exhibited increased concentrations of CD (41%), LHP (38%), and MDA (57%), along with similar patterns in biomarkers of oxidative damage to proteins. These observations indicate evident oxidative damage to lipids and proteins in neonates due to increased systemic oxidative stress associated with PE. The elevated levels of IMA support the presence of placental ischemia resulting from inadequate trophoblast invasion. Hence, IMA can be proposed as a marker of placental ischemia-induced damage in neonates born to women with PE. However, the close correlation between IMA and FFAs suggests that the conversion of albumin to IMA is likely due to FFA binding rather than increased oxidative stress. These findings provide insight into new mechanisms for preventing the development of fetal pathologies.

Keywords: Ischemia-Modified Albumin, Preeclampsia, Free Fatty Acids, oxidative stress, lipoperoxidation

FMED19C

5-HIAA and ATPase increase during N- Acetylcysteine and Cisplatin administration and induce neuroprotection in brain

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Neurotoxicity is a major obstacle to the effectiveness of cisplatin (CDDP) in cancer chemotherapy. In this process, oxidative stress and inflammation are considered to be the major mechanisms involved. The aim was to study the influence of protein quantity on some antioxidant biochemical parameters in brain and lung of 50-day old rats that were treated with cisplatin and N-Acetylcysteine (NAC). Methods. Twenty-four young male Wistar rats weighing 200g each were used. The animals were randomly distributed in 4 groups. Each group was fed with a protein diet at 7% for 15 days. Thereafter, the groups were given either a single dose of cisplatin® 5mg/kg b.w. i.p. or Acetylcysteine® 5mg/kg b.w. i.p. as follows: group 1 (control), NaCl 0.9% vehicle; group 2, Cisplatin; group 3, NAC and group 4, NAC + Cisplatin. The animals were sacrificed at the end of treatment, and their blood were collected and used to measure blood triglycerides and glucose levels. The brain and lung were used to assay peroxidation of lipids (TBARS), glutathione (GSH), serotonin metabolite (5-HIAA), catalase and the activity of Ca^{+2}, Mg^{+2} ATPase, using previously validated methods. Results. TBARS, H_2O_2 and GSH decreased significantly in cortex and cerebellum/medulla oblongata regions of groups treated with CDDP and NAC. Total ATPase showed a significant increase in the lung and cerebellum/medulla oblongata of the experimental animal groups while 5-HIAA depicted the same tendency in cortex of the same group of animals. Conclusion. 5-HIAA and ATPase increase during NAC and Cisplatin administration resulted in brain protection. This effect could even be more powerful when membrane fluidity is increased. Thus, probed efficacy of combined NAC and CDDP drug therapy and appears to be a promising strategy for future chemotherapy in malnourished patients.

FMED20C

Total antioxidant capacity modifies the correlation between body mass index and HDL cholesterol in Mexican children

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Introduction. Oxidative stress occurs during obesity and is associated with dysregulation of cellular metabolism and the early development of insulin resistance, type 2 diabetes, and cardiovascular disease. The total antioxidant capacity (TAC), which is associated with healthy lifestyles, could reduce the risk of metabolic complications related to oxidative stress. **Objective.** To assess whether TAC modifies the association between obesity and cardiovascular risk factors in Mexican children. **Methods.** 218 children with normal weight (NW) and 232 with obesity (OB) where included in the study. To determine cardiovascular risk factors, anthropometric and blood pressure measurements were taken. Serum glucose concentration (GLUC), total cholesterol (TC) and its high (HDL-C) and low (LDL-C) density fractions, and triglycerides (TG) were also measured. The percentage of TAC was measured in serum using 2,2-diphenyl-1-picrylhydrazyl (DPPH). **Results.** OB was negatively associated with the serum concentration of HDL-C ($p<0.05$) and positively associated with the increase in systolic and diastolic blood pressure, the serum concentration of GLUC, TC, LDL-C and TG, and the percentage of TAC ($p\leq0.05$). The serum concentration of HDL-C was negatively correlated with the percentage of TAC (Rho= -0.202, p= 0.004). To assess whether TAC modified the association between obesity and cardiovascular risk factors, the correlation between body mass index (BMI) and cardiovascular risk factors was analyzed in the low, medium, and high TAC groups (according to tertiles of TAC). The BMI only was significantly correlated with the decrease in HDL-C, specifically in the groups of low (PN: Rho= -0.218, p= 0.040; OB; Rho= -0.357, p= 0.006) and medium (PN: Rho= -0.314, p= 0.014) TAC. The correlation between BMI and HDL-C was not significant in the high TAC groups ($p> 0.05$). **Conclusion.** Our results suggest that TAC modifies the association between BMI and HDL-C in Mexican children. It is important to continue analyzing the relationship between TAC and lifestyles in order to support possible strategies for the prevention of metabolic complications related to OB.

FMED21C

Effect Of Borax on Mouse Cardiac Hypertrophy Induced During Pregnancy

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Fetal and postnatal hypertrophy develops in response to such different exposures or illnesses the mother suffers during gestation as anti-infectious and physical agents, obesity, hypertension, diabetes, and even advanced maternal age. This gives rise to high comorbidities in the newborn; therefore, looking for alternatives that contribute to cardiac homeostasis is quite necessary to inhibit the overgrowth of myocytes. Boron-derivative compounds could play a key role in exerting a repairing effect on chronic cardiac damage induced during gestation.

The cardioprotective effect of Boron was tested in the treatment of pregnant mice previously subjected to isoproterenol (fetal hypertrophy model) on the fifth-day post-coitus. Prior to the sacrifice of the pups of mice, an electrocardiography (ECG) was done.

Morphological and histological changes of the heart were assessed in newborn pups. As a damage marker, the concentration of p38 MAPK was evaluated by Western Blot, and the levels of malondialdehyde (MDA) as well as glutathione (GSH) and glutathione peroxidase (GPx) were tested by spectrometry. mRNA expression for early response genes (c-jun, c-fos y c-myc), late response (GATA-4, Mef2c, NFAT), and heart damage (ANP and BNP) was measured by qPCR real-time.

Pregnant females treated with isoproterenol were treated with 100 mg/kg sodium tetraborate during the fetal stage, an improvement in contractility was detected in the pups with an actual reduction in myocardial fibrosis and oxidative stress, but cardiac mass increased. In addition, the expression of c-jun, c-myc, GATA-4, MEF2c, and ANP mRNA declined in comparison with CTR. However, the hypertrophic damage mechanism was sustained by c-fos, NFAT, and BNP expressions.

This result suggests that sodium tetraborate mitigates hypertrophy induced during pregnancy, thereby improving contractibility, reducing oxidative stress, and stimulating cell proliferation. Therefore, sodium tetraborate could be an excellent prophylactic treatment administered by delivery oral route during pregnancy when there is a risk of developing fetal left ventricular hypertrophy.

Key words: Boron, hypertrophy cardiac, oxidative stress

FMED22C

Characterization of antioxidant nanostructured systems and their cytotoxic effect in primary culture of rat chondrocytes

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Objetives:
Prepare and Physicochemically Characterize Glutathione-Loaded Nanoparticles and Liposomes
To evaluate the cytotoxic effect of exposure to different concentrations of liposomes and nanoparticles in primary culture of rat chondrocytes.
Methods:
Nanoparticles were prepared by the ionic Gelacio method, while the liposomes were prepared by the Bangham method and subsequently sonicated to reduce the particle size. Characterization (size and Zeta potential) was carried out in the Malvern zeta sizer equipment, while the encapsulation percentage of GSH was developed indirectly by the DTNB method. To assess the biological effects, a primary culture of rat cartilage was performed. The cytotoxic effect will be prolonged by measuring cell viability using the Resazurin and crystal violet techniques. A one-way ANOVA was performed with a ($p<0.05$), and Fisher's multiple means analysis.
Results and discussion:
Regarding the characterization, nanoparticles had an average size of 272.9 nm, while the liposomes show a size greater than 493.4 nm, the percentage of encapsulationin the two systems obtained above 99% and in terms of the Z potential the NP had 23 Vm and the liposomes -8 Vm. These physicochemical characteristics suggest that nanostructural systems could uptake by cell for endocytosis and, due to the Z potential, be stable systems. In the resazurin reduction assay, no significant differences were observed in the metabolic activity of the cells in the concentrations used ($p<0.05$) and in the viability evaluation crystal violet does not show a reduction in cell density when compared with with the positive control, so it is suggested that the systems have low toxicity.
Conclusions:
Results of the physicochemical characterization suggest that these systems are stable in solution due to the Z potency obtained and also due to their size they could uptaken by the cell by endocytosis None of the concentrations used in the two different systems showed to have a cytotoxic effect in primary cultures of rat chondrocytes.

FMED23C

Antioxidant properties of *Stevia lucida* in murine macrophages J774A.1 induced with LPS

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Introduction. The medicinal plants *Parkinsonia praecox* and *Stevia lucida* have been used in tradicional medicine to treat inflammation, nevertheless, the presence of compounds with antioxidant properties is unknown. Thus, the present study focused on evaluating the effect of methanolic extract from this plant on the generation of reactive oxygen species (ROS) in a cell model of murine macrophage.

Methods. The bark of *P. praecox* and the aereal parts of *S. lucida* were pretreated with several unit operations and the solids obtained were mixed with methanol to recover the plant extracts (PpBM and SlAM, respectively) by ultrasound-assisted extraction. Then, the cytotoxicity of the extracts was assessed againts murine macrophages J774A.1 using the MTT colorimetric assay. Additionally, the effect of plant extracts on the ROS generation were measured directly in murine macrophages stimulated with LPS, using 2', 7'-dichlorodihydrofluorescein diacetate (H2DCFDA) assay.

Results *Parkinsonia praecox* and *S. lucida* extracts exhibited low cytotoxicity against murine macrophages (<20%) at concentrations of 31 µg/mL and 15 µg/mL, respectively. Under these conditions, the PpBM reduced the ROS production by 82% in murine macrophages stimulated with LPS, while that SlAM showed the highest antioxidant activity with a decrease of ROS levels by 95%.

Conclusions. This is the first report about the antioxidant activity of *P. praecox* and *S. lucida* in macrophages. Finally, the macrophage-based model provided direct and reliable information on the antioxidant potential of extracts, since cells resemble physiological conditions. Further studies are required to isolate the responsable compounds and use them to pharmacological applications *in vivo* models.

Keywords: *Parkinsonia praecox* and *Stevia lucida* extracts, antioxidant potential, macrophages

FMED24C

Antigenotoxic capacity of chayote juice (*Sechium edule*) against oxidative damage produced by benzopyrene

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Traditional medicine continues to be used to treat and/or prevent the appearance and progression of chronic diseases. Specifically, chayote (*Sechium edule*) has shown diuretic, anti-inflammatory, hypotensive, antiulcer, antihyperlipidemic, antimicrobial, and antioxidant activity. Beneficial effects related to the presence of its bioactive compounds (flavonoids, triterpenes and cucurbitacins).

The aim of this study was to determine the anticlastogenic potential of two types of fresh chayote juice [filtered (ChJ-f) and unfiltered (ChJ-uf)] against oxidative damage induced by benzo(a)pyrene [B(a)P] using the micronucleus (MN) assay. The juices (ChJ-f and ChJ-uf) were freely consumed by the animals for 2 weeks. We include a negative control (water), a positive batch [B(a)P, dose 250 mg/kg/week], and two combined lots [B(a)P plus ChJ-f or ChJ-uf]. Blood smears were taken at different times (0/day 1, 48h/day 3, 96h/day 5, 144h/day 7, 192h/day 9, 240h/day 11, 288h/day 13). The slides were stained and observed microscopically to quantify the number of micronucleated normochromic erythrocytes (MNE). The results indicated: a) B(a)P increased the frequency of MNE with the time of administration, b) None of the chayote juices (ChJ-f and ChJ-uf) induced micronuclei; on the contrary, both juices were genoprotective. However, the highest protection was with ChJ-uf at the end of the experiment (80%).

These results suggest that avoiding filtration favors a greater number of bioactive compounds; allowing the combination of its antioxidant compounds to reduce the damage induced by B(a)P. Therefore, consumption of chayote (especially fresh and unfiltered) can be considered a a chemopreventive process, and its mechanism of action is probably related to its antioxidant capacity.

Keywords: *Sechium edule*, micronucleus assay, antioxidant capacity

FMED25C

Alpha-linolenic fatty acid (ALA) protects against indomethacin-induced gastric damage in murine model

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Non-steroidal anti-inflammatory drugs (NSAIDs) are a group of drugs widely used worldwide for their anti-inflammatory and analgesic properties; however, their prolonged use causes gastrointestinal damage for people who consume them. Therefore, natural alternatives are sought to reduce gastric damage caused by NSAIDs, such as omega-3 polyunsaturated fatty acids (ω-3 PUFAs), which induce anti-inflammatory and antioxidant effect. Among the ω-PUFAs, docosahexaenoic acid (DHA) has recently demonstrated gastroprotective effect; however, the effect of α−linolenic acid (ALA), which is mainly found in seeds such as chia, has not been studied so far. The objective of this work was to evaluate the gastroprotective effect of ALA in a murine model of gastric damage induced by indomethacin in female Wistar rats. ALA was administered orally acutely (300 mg/kg, p.o.) or for 10 days (20 mg/kg, p.o.), two hours later indomethacin (INDO) was administered orally to induce acute gastric damage (30 m/kg, p.o.). Three hours after INDO administration, the rats were euthanized, and the gastric lesions were evaluated to obtain the total area of damage. Gastric tissue was collected to quantify levels of leukotriene B4 (LTB4) by enzyme-linked immunosorbent assay, and reduced glutathione (GSH), myeloperoxidase (MPO), malondialdehyde (MDA) and oxide nitric (NO) by colorimetric assay. ALA exhibits gastroprotective effect both macroscopically and microscopically due to a single oral administration and 10 days oral administration against acute gastric damage caused by indomethacin. LTB4, MPO, MDA and NO levels decrease significantly with ALA treatment, while GSH levels increase with both treatments. ALA showed gastroprotective effect against indomethacin. Our results suggest that ALA induces gastroprotective action through the antioxidant pathway activation.

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Keywords: Alpha-linolenic fatty acid, gastric injury, antioxidant.

FMED26C

Effect of L-Arginine in oxidative stress index and oxidative damage to DNA on Hind-Limb ischemia-reperfusion model

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Ischemia-reperfusion (I-R) is a phenomenon that is defined as tissue damage induced by consecutive interruptions of blood flow (ischemia) that is so aggravated by the restoration of blood flow (reperfusion). I-R is linked to multiple pathologies related to morbidity and mortality. According to earlier studies, the I-R phenomenon is strongly linked to oxidative stress modification.

Oxidative stress is characterized as an imbalance between oxidant species such as reactive oxygen species (ROS) or reactive nitrogen species and antioxidant species such as nitric oxide. A high level of oxidizing species generates a negative impact on biological systems, which is presented as damage to lipids, proteins, and DNA. There are several biomarkers for measuring oxidative stress like 8-hydroxy-2-deoxyguanosine (8OHdG), a useful molecule as a marker of oxidative damage in genetic material, and peroxides, which are the oxidation products of several molecules. For the analysis we selected four different groups: I) Control/sham without I-R, II) I-R, III) I-R + 100mg L-arginine, and IV) I-R + 200mg L-arginine. All procedures were carried out under anesthesia with Zelasol 5mg/kg weight. Femoral occlusion was carried out by ligation for six hours. Once the ischemia time elapsed, reperfusion was carried out in periods of seven and fourteen days, where groups III and IV received treatment with L-arginine. We measured DNA damage using an 8OHdG assay. Also, we measured the oxidative stress index through the total oxidant status and total antioxidant status kits. We saw a significant difference between treated groups and non-treated groups with L-arginine ($P<0.05$). The subjects treated with 200mg of L-arginine showed a lower oxidative stress index (5.5 ± 0.71) than group I (8.83 ± 0.30), group II (11.13 ± 0.50) and group III (8.11 ± 0.51). For its part, DNA damage measured by 8OHdG showed similar results where group IV had a lower presence of 8OHdG (134.33 ± 2.61) than group I (146.88 ± 8.27), group II (185.78 ± 10.18) and group III (140.81 ± 6.94). These results offer us an encouraging outlook on the possibilities of reducing damage from oxidative stress using L-arginine.

Keywords: L-arginine, ischemia-reperfusion, oxidative stress index.

FMED27C

Therapeutic Targets for Regulating Oxidative Damage Induced by Ischemia-Reperfusion Injury: A Study from a Pharmacological Perspective

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Ischemia-reperfusion (I-R) injury is damage caused by restoring blood flow into ischemic tissues or organs. This complex and characteristic lesion accelerates cell death induced by signaling pathways such as apoptosis, necrosis, and even ferroptosis. In addition to the direct association between I-R and the release of reactive oxygen species and reactive nitrogen species, it is involved in developing mitochondrial oxidative damage. Thus, its mechanism plays a critical role via reactive species scavenging, calcium overload modulation, electron transport chain blocking, mitochondrial permeability transition pore activation, or noncoding RNA transcription. Other receptors and molecules reduce tissue and organ damage caused by this pathology and other related diseases. These molecular targets have been gradually discovered and have essential roles in I-R resolution. Therefore, the current study is aimed at highlighting the importance of these discoveries. In this work, we inquire about the oxidative damage receptors that are relevant to reducing the damage induced by oxidative stress associated with I-R. Several complications in surgical techniques and pathology interventions do not mitigate the damage caused by I-R. Nevertheless, these therapies developed using alternative targets could work as coadjutants in tissue transplants or I-R-related pathologies.

Therapeutic Targets in Oxidative Damage. A detailed review of the therapeutic targets that can be a receptor for some antioxidant drugs should be performed. This allows redirecting research to the establishment of therapeutic alternatives that can have more interesting effects. Calcium overload, which triggers the formation of reactive species in the mitochondria, and MPTP opening, which is involved in the release of Ca^{2+} from the mitochondrial matrix to the cytosol, are relevant. Regulating the ferroptosis process, one of the key pathways in nonapoptotic death that is strictly correlated with cellular antioxidant capacity could be an interesting alternative for different pathologies involving I-R. Undoubtedly, the modulation of ROS/RNS in the cytosol, which can prevent all types of cell damage, is a strategy that remains undiscovered due to a large number of possibilities. Notably, future research must focus on the different types of tissues, variations in I-R injury, and modulating strategies.

Keywords: oxidative stress, ischemia-reperfusion, oxidative stress index.

FMED28C

Oxidative Stress in kidney damage from acute inhalation exposure to endotoxin in a rat model

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Oxidative stress (OxS) is a known mechanism involved in the toxicity resulting from exposure to particulate matter (PM), including endotoxin as one of its components. However, limited evidence exists regarding the impact of inhalation exposure to endotoxin on nephrotoxicity and the involvement of OxS. In this study, we investigated the effects of acute inhalation exposure to endotoxin on kidney damage and its relationship with OxS in a rat model. Adult male Sprague-Dawley rats (n=6) were exposed to endotoxin (lipopolysaccharide; *Escherichia coli* 055:B5) using a nebulized inhalation exposure system (in-Expose, SCIREQ) for four days, with a duration of 7.5 minutes per day. The rats were divided into four groups: a control group exposed to sterile water and three exposure groups exposed to different atmospheric endotoxin concentrations of 2 EU/mg $PM_{2.5}$ (low), 300 EU/mg $PM_{2.5}$ (high), and a mixed group exposed to alternating concentrations above the threshold. At the end of the exposure, urine samples were collected from the rats using metabolic cages to assess water consumption, urinary flow, and glomerular filtration rate (GFR). Myeloperoxidase (MPO) and methylglyoxal (MGO) were measured in serum, urine, lung homogenates, and renal cortex samples, both biomarkers related to OxS, as well as inflammation and metabolism, respectively. Non-parametric statistics were employed for data analysis, and significant differences were observed between the exposed groups and the control group for both biomarkers evaluated. Serum MPO activity and MPO levels in renal cortex homogenates were significantly higher in the mixed group compared to the control group. While pulmonary MPO levels did not show significant changes, an increase in MPO activity was observed in the renal cortex of the high-dose exposure group, accompanied by elevated urinary excretion in both the high-dose and mixed groups. Serum MGO levels increased in the high-dose exposure group, but decreased in the lung and renal cortex of the low-dose and high-dose groups, respectively. However, urinary MGO levels were elevated in all three exposure groups compared to the sterile water control. These findings suggest the involvement of OxS in kidney damage resulting from inhalation exposure to endotoxin. Additionally, all exposed groups exhibited hyperfiltration, as evidenced by increased GFR. Keywords: endotoxin, kidney, oxidative stress.

FMED29C

Liver damage in obesity due to frequent consumption of acetaminophen (generator of oxidative stress)

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Acetaminophen (APAP) is one of the drugs frequently prescribed in Mexico and the whole world, due to its use to treat most of the general ailments associated with common diseases. When APAP is metabolized, it produces a toxic reagent called N-acetyl-p-benzoquinone imine (NAPQI), which under normal conditions binds with glutathione (GSH) and is easily neutralized and excreted via the kidneys by the body. However, people with obesity present a decrease in GSH levels, which can lead to oxidative stress and consequently hepatotoxicity. Furthermore, the only treatment for acute hepatotoxicity is the antioxidant N-acetyl cysteine (NAC), which is indicated for those patients at risk of liver injury. NAC acts by detoxifying NAPQI, restores GSH reserves and can also be directly conjugated with NAPQI serving as a GSH substitute. **Aim**: To evaluate whether obesity is associated with the causality of liver injury in patients with chronic consumption of acetaminophen at therapeutic doses. **Methods:** The association between obesity and the causality of liver damage was analyzed in patients from Veracruz with chronic consumption of acetaminophen at therapeutic doses. The study was a cross-sectional prospective cohort and was carried out in patients from the ISSSTE Xalapa, Ver., clinic-hospital, who presented obesity and chronic consumption of APAP at therapeutic doses. The causality of drug-induced hepatotoxicity was determined by applying the RUCAM questionnaire (*Roussel Uclaf Chance Assessment Method*), which is a globally established questionnaire in cases of suspected drug-induced liver injury. The association between obesity and chronic APAP consumption was determined through a multiple logistic regression statistical analysis. **Results and conclusions:** a prevalence of suspected liver injury was obtained in people with obesity and chronic consumption of APAP of 40.62% compared to the prevalence of liver injury in normal-weight people of only 13.63%. Multiple logistic regression analysis, with which body mass index (BMI) was compared between the groups with and without liver injury; obtained an odds ratio (OR) value of 4.333 (1.061-17.7) and *p= 0.023*, which show a significant association between BMI (≥ 30 kg/2) and liver injury, that is, the higher the BMI, the greater the probability of liver injury caused by consumption of APAP at therapeutic doses. The use of acetaminophen at therapeutic doses (< 4 g/d) in people with obesity, must be carefully regulated and monitored to avoid generating long-term liver damage.

FMED30C

Glutathione Level at the Stage of Steatohepatitis in Patients with Metabolic Fatty Liver Disease

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Metabolic Dysfunction Associated Liver Disease (MAFLD), formerly known as non-alcoholic fatty liver disease; is defined by an excessive accumulation of fat (steatosis) in the liver that can progress to steatohepatitis (NASH) and hepatocellular carcinoma. It has been shown that the population at risk of developing MAFLD is overweight, obesity, type 2 diabetes mellitus, and patients with dyslipidemia. In recent years it has been discovered that oxidative stress (OS) plays a very important role in the origin and evolution of MAFLD. Even, it has been shown that obese patients have low reserves of the antioxidant glutathione (GSH). However, to date the redox mechanisms that lead to the development of this disease are unknown. **Aim:** quantify the glutathione level in patients with NASH and compare with healthy patients without MAFLD. **Methods:** Patients diagnosed with and without MAFLD were included from the family medicine units of ISSSTE-Xalapa, Veracruz. The probable state of NASH was analyzed by the following methods: standardized panel of Hypertension, Alanine aminotransferase (ALT) and insulin resistance (HAIR), the triglyceride and glucose index (TyG Index), the aspartate ratio aminotransferase / alanine aminotransferase (AST/ALT) > 2, and the hypoalbuminemia and thrombocytopenia. In patients with a high probability of suffering from steatohepatitis, glutathione levels were analyzed using the ELLMAN technique. **Results:** Of the 5 criteria used for the diagnosis of NASH, in 4 of them more than 50% of the population presented NASH; however, only those patients with a positive result in each panel performed were considered as NASH criteria. No statistically significant difference was found between the mean GSH concentrations between the control group and the group with possible NASH, but it was observed that the mean GSH concentration value in the group with probable NASH was significantly lower with compared to the control group. **Discussion:** No statistical significance in GSH level could be due to the low number of samples used, therefore, if a larger population sample were used, it is very possible to reach said significance, since a clear pattern of decreased GSH was observed in the group with probable NASH. **Conclusion:** The low level of GSH in the group with probable NASH shows that the antioxidant defense system is altered and that the cellular response capacity to this environment is not taking place normally.

FMED31C

Vanadium compounds: potential medicine applications as a source of ROS

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Vanadium is an ultra-trace element present in higher plants, animals, algae, and bacteria, mainly since it constitutes a well-known phosphate analogue. Hence, its study offers possibilities to design promising vanadium-containing metallo-drugs. Doing so, I begin, in the year 1996 [1], a large journey in the research of vanadium compounds with biological activity. In first place, interested in the antidiabetic properties [2, 3], we design a new vanadium antidiabetic pro-drug which acts inhibiting the negative regulator of insulin signaling PTP1B [4]. Then, we turn our faces to see vanadium compounds are considered a representative of a new class of non-platinum metal anticancer drugs do to its activity over cell signaling pathways on cancer cells and of the underlying mechanisms [5]; therefore, we proposed ALA-peptide-VO(O2)2·H2O complexes as prodrugs with enhanced uptake to the hPepT1 channel, reducing the pharmacologically effective dose and reducing side effects of traditional cisplatin [6]. In 2019, responding to the call of the international scientific community to confront the COVID-19 pandemic, we joined forces to develop an alternative treatment based on the similarities the SARS-CoV-2 main protease (Mpro) with the PTP1B protein family [7], fighting against the virus but also with the hidden reluctance of the big pharmaceutical companies to incorporate vanadium in their formulations [8]. Already before the appearance of the virus, we had realized that the signal transduction pathway of PTP´s have another form of activity control inside the cell which is generated by reactive oxygen species. ROS are known to be involved in metabolic cascades that lead to cellular apoptosis but could also influence insulin-like activities [9]. Very at the beginning, the source of vanadium's strong biological activity lies in its affinity for incorporating oxygen atoms and presence of the oxo group induces the electron donating process over complexes that in first instance does not present it. We theoretical demonstrate show that the vanadyl ion promotes the production of OH· radicals, while vanadium IV and V complexes produce reactive oxygen species (OH· and O2−·) by Fenton and Haber-Weiss mechanism [10]. Now the scientific community knows that biological benefits vanadium exerts is likely the underlying cause for some of the beneficial effects reported in cancerous, diabetic, neurodegenerative conditions and other diseased tissues impacted by LPO processes [11]. We are moving towards the knowledge of the molecular bases of the interaction of vanadium with the primordial structures of life such as DNA [12]. But along the way we have learned a bit about the reactivity of vanadium and how we can use this tool, as a ROS gun machine, to control it and direct it towards biological molecular targets that are of interest in medicine.

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FMED32C

Influence of chronic stress on oxidative stress in epididymal spermatozoa of young rats

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In adult rats, chronic stress has been reported to affect spermatogenesis, with the loss of germ cells and the initio of an apoptotic process. We have found that chronic stress increases aldosterone levels in young adult rats. Leydig cells have aldosterone receptors, and this hormone is a regulator of fluid and electrolyte balance in the epididymis. In addition, the proliferation and maturation of the Leydig cells are inhibited by an increase in aldosterone. The present study aimed to evaluate the effects of elevated aldosterone levels induced by chronic stress on oxidative stress and sperm quality. Eplerenone, a selective aldosterone blocker, was used. Two experiments were designed. The first consisted of control and chronic unpredictable mild stress (CUMS). The second consisted of control + vehicle (C + veh), CUMS + eplerenone (CUMS + EP, 100 mg/ kg/día of eplerenone diluted in 1 mL of tap water). On postnatal day 51, eplerenone was administered orally through a gastric tube two hours before the start of the stress. The stress stimuli consisted of 5 different stimuli: reduction of space in a clean cage for 5 hours, restriction of movement for 3 hours, forced swimming in cold water for 10 minutes, forced swimming in warm water for 10 minutes, and reduction of space in a dirty cage for 5 hours. The order of the stressor stimuli was different during each week, excluding the weekend. In the first experiment, sperm motility, viability, concentration, and morphology were lower the JE4 group than in the control group. In the JE4 group, SOD activity was lower than in the control group. At the same time, the CAS3 activity was higher compared to the control group. In the second experiment, sperm viability and sperm concentration were lower in the JE4 + EP group than in the control +Veh group. In the JE4 + EP group, TBARS, SOD, CAT, and CAS3, were higher than in the control group + Veh group. In conclusion, aldosterone is increased by chronic stress-induced oxidative stress however, the eplerenone potentiates oxidative stress, sperm cell death, and thus reduced sperm quality.

FRCS01C

Study of the role of the urate transporter ABCG2 in the regulation of the immune response in knockout models of human colon and kidney cells

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Introduction.- Gout is a multifactorial metabolic disease, characterized by intra articular pain attacks due to inflammatory activation induced by monosodium urate (MSU) crystals. These crystals accumulate due to the patient's frequent hyperuricemia. ABCG2 is a membrane transporter protein that clears uric acid mainly in the intestine and kidney. *ABCG2* gene has been related to hyperuricemia and gout due to polymorphism studies in different populations, however its role in the development and progression of the disease is not clear, a recent study where *ABCG2* gene was silenced in THP1 cells stimulated with MSU, it suggested a possible role of *ABCG2* in the regulation of inflammation in gout. **Objective**.- To study the role of *ABCG2* as a possible regulator of inflammation induced by the presence of MSU crystals and soluble uric acid (sUA) crystals in two *in vitro* models of gout. **Methodology**.- Until now cultures of the human colon HCT15 and the kidney cell line HEK293T was been grown in specific media until the day of the tests with or without activation with MSU for 48 hours. Genome editing of cultures of these same cells was performed using the CRISPR CAS9 recombinant protein to silence the *ABCG2* gene with Edit R- human *ABCG2* crRNA and track RNA. The identification of transfected cells with non-functional *ABCG2* will be through growth in culture plates to evaluate the gene and protein expression of *ABCG2*, later sequencing of the target area will be carried out. Activation assays with MSU of the cells silenced for 48 h will be carried out, later the expression of inflammation and *ABCG2*, *IL-1*beta, *IL-6*, *S-100*, *p53* and *TL3* genes will be analyzed by immunofluorescence, westen blot and real-time PCR. **Preliminar results**.- Until now, transfected cells and not transfected cells have been stimulated with MSU and SUA, we observed that vesicles with internal crystals are formed in the colon HCT15 cells after 24 hours of exposure with MSU. *ABCG2 ,S-100* and *p53* gene expression was measured without finding differences in expression at 24 and 48 hours by immunofluorescence. Transfected HCT15 cells had higher expression of phosphorylated p53 than non-transfected cells without stimulation. **Conclusions**.- Human colon and kidney non transfected cells respond to the presence of MSU by forming vesicles with MSU crystals without finding differences in the expression of *ABCG2* at the exposure times used up to now. The transfected HCT15 cells maintain their normal growth with active p53 expression.

FRCS02C

Msn2 and Msn4 regulation during oxidative stress response

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Candida glabrata is an opportunistic fungal pathogen, it is the second specie of its gender that causes invasive candidiasis. During the infection process, *C. glabrata* is internalized by host phagocytic cells, which induce the production of reactive oxygen species (ROS). However, *C. glabrata* survives these conditions because it has an efficient oxidative stress response (OSR) that consists of the induction of genes that encode antioxidant proteins. Msn2 and Msn4 are transcriptional factors that control the expression of several genes under different stress conditions. Under oxidative stress, Msn2 and Msn4 partially induce *CTA1* that codes for the main antioxidant enzyme of *C. glabrata*, although Msn2 and Msn4 are two transcription factors that respond to different types of stress. Their mechanisms of action and their regulation are not known in *C. glabrata*. The aim of this work is to elucidate the signaling pathways that regulate the activity of Msn2 and Msn4 in response to oxidative stress.

FRCS03C

Redox regulation of DNA repair by peroxiredoxin Tsa1 and cysteine oxidation in *Saccharomyces cerevisiae*

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DNA repair is a group of highly conserved mechanisms required to respond to DNA damage and maintain DNA integrity. Recent studies in mammals have noted the regulatory effect of cysteine oxidation of Rad51 and peroxiredoxins on DNA repair by homologous recombination. The yeast *Saccharomyces cerevisiae* shares the same mechanisms of DNA repair as in mammals; nevertheless, there is a lack of information about cysteine oxidation in yeast, despite the fact that many of these cysteines are conserved between human and yeast. Besides, there is a peroxiredoxin in *S. cerevisiae* named Tsa1 that helps to maintain DNA integrity, but until now this molecular mechanism has not been elucidated. Our main objective in this project is to demonstrate that Tsa1 affects cysteine oxidation to promote efficient DNA repair, just as peroxiredoxins do in humans. We performed growth curves and drop assays to evaluate a strain of *Δtsa1* in DNA damage provoked by MMS and single double strand break by endonuclease HO. And the cysteine oxidations, S-sulfenylation and S-persulfidation, were detected in gel by direct reaction with dimedone and dimedone switch, respectively. We found that *Δtsa1* behaves differently from the wild type when its genome is cut by endonuclease HO, that its susceptibility to MMS increases, and that TSA1 influences total cysteine oxidation. These findings confirm that peroxiredoxin Tsa1 influences mechanisms of DNA repair such as homologous recombination and may be linked to cysteine oxidation.

This project was financed by UNAM-DGAPA-PAPIIT through the research programs IN208922 and by the Consejo Nacional de Humanidades, Ciencias y Tecnologías through the Ciencia de Fronteras 2023 program CF-2023-I884.

FRCS04C

Oxidative stress induced by gadolinium-based nanoparticles in cultured human lymphocytes

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Gadolidium is an earth rare metal used as contrast in magnetic resonance studies. However, it has been demonstrated toxicity and health damage by the agent, therefore, alternatives to use nanoparticles (NPs) of Gd2O3 and its derivatives have been initiated. In these efforts, preclinical assays to examine the NPs safe use are mandatory. In the present report we synthetized aerogel Gd2O3NPs, as well as those doped with europium, and functionalized with tenoyl-trifluoroacetone in order to increase the fluorescence power. The three NPs were examined to determine their lipid and protein oxidative capacity in the lymphocytes of three male donors. Moreover, we also examined their genotoxic and cytotoxic capacity in the same cells.

The NPs were determined as macro and microporous structures, with a variable size (100-140 nm), with a fluorescent emission spectra between 257-350 nm. The experimental groups for the biological assays included a control group administered saline solution, a positive control group treated with mitomycin C (0.01 µg/ml) and three groups of NPs (1, 10, and 100 µg/ml). For the oxidation assays, we first separate the lymphocytes, which were cultured for 24 h to follow the method of Levine et al., (1990) and of Shalini et al (2017). For the geno/cytotoxic studies, we applied the cytokinesis block micronucleus assay in human lymphocytes. Each parameter was evaluated per concentration/per donor, and statistically examined with an ANOVA followed by the Tukey tests. Our results in protein oxidation showed a significant increase of carbonyl groups mainly in cells treated with Gd2O3NPs, while the malondialdehyde level were highly increased in the doped NPs, as well as in the doped and functionalized NPs. The increase was more than 100% with respect to the control level. These data suggest that oxidized molecules could interact with other molecules to produce undesired toxicity. This effect was confirmed with our geno/cytotoxic studies where we found an increase of nuclear bridges and nucleoplasmic buds, as well as of necrotic cells. Our results, therefore, showed a significant increase of lipid and protein free radicals that were expressed in DNA and cytotoxicity effects.

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Shalini, D., Senthilkumar, S., Rajaguru, P. (2017). Toxicol Mech Methods

Key words: Gadolinium, nanoparticles, lymphocytes, oxidation, geno/cytotoxicity

FMED33C

Expression of redox genes involved in the lipid storage mechanism in fatty liver disease associated with metabolic dysfunction

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Metabolically Dysfunction-Associated Fatty Liver Disease (MAFLD) is defined by an excessive accumulation of fat in the liver (5-10% of the organ's weight). Its spectrum can range from steatosis to metabolic steatohepatitis (MeSH) with risk of progression to fibrosis, cirrhosis and hepatocellular carcinoma. At the national level, cirrhosis, along with other chronic liver diseases, ranks fourth and third in deaths, respectively. It is believed that 70% of cryptogenic cirrhosis cases are caused by MAFLD, for which it is important to know and understand the molecular mechanisms by which fat accumulates in the liver giving rise to MAFLD. A general hypothesis for the etiology of MAFLD is the “two-hit hypothesis”, where insulin resistance and oxidative stress play the main roles in causing this disease. These mechanisms are related to the deregulation of lipid homeostasis and oxidative stress causing a lack of control in the metabolism of S-Adenosyl-L-Methionine (SAM) where the methionine-adenosyl-transferase 1a and 2a (Mat1a, Mat2a), the phosphatidylethanolamine-N-methyltransferase (Pemt), Glycine-N-methyl-transferase (Gnmt), S-adenosyl-homocysteine-hydrolase (Sahh), betaine-homocysteine methyltransferase (Bhmt) and many other gene methyl transferases participate. The objective of this work was to determine the expression level of the Mat1a, Mat2a, Pemt, Gnmt, Sahh and Bhmt genes involved in the mechanism of fat storage in MAFLD in liver tissue biopsies. In situ RT-PCR assays were performed to evaluate the transcription of the genes: Mat1a, Mat2a, Pemt, Gnmt, Sahh and Bhmt; in samples from patients with MAFLD. Results: 3 samples with an anatomo-histopathological diagnosis of MAFLD were analyzed. The Pemt gene presented the highest expression with a staining intensity of moderate to intense; the gene that encodes for the Sahh enzyme presented strong expression, with moderate to intense staining, while the Mat2a gene was widely expressed, showing weak to intense signal intensity, while the Mat1a gene exhibited low expression with a weak to moderate signal, finally the Gnmt gene showed the lowest expression of the genes studied, with a weak staining intensity.

FMED34C

Glutathione peroxidase-1 mRNA levels in patients with comorbidity: fatty liver disease associated with metabolic dysfunction and type 2 diabetes mellitus

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Fatty liver disease associated with metabolic dysfunction (MAFLD) is characterized by a spectrum that includes simple hepatic steatosis, steatohepatitis, fibrosis, cirrhosis, and even hepatocarcinoma. Its main risk factor is metabolic syndrome, which involves obesity, insulin resistance, dyslipidemia and arterial hypertension. This disease is closely related to type 2 diabetes mellitus (DM2), since it induces insulin resistance and other alterations. Insulin resistance and other factors produce perturbations in the synthesis and oxidation of fatty acids, inducing their accumulation in the liver. The mentioned process increases the formation of reactive oxygen species (ROS), producing high levels of oxidative stress and thus increasing lipid peroxidation. The produced oxidative stress is counteracted by an antioxidant defense system, which has enzymes and other factors that are responsible for detoxifying ROS from the cell to prevent oxidative damage. These enzymes include glutathione peroxidase, which is responsible for reducing hydrogen peroxide to water, if the synthesis of this enzyme is altered, accumulation of peroxides occurs, which tend to interact with free fatty acids and Mitochondrial DNA, increasing oxidative damage in the cell. In this work we propose to determine the levels of the mRNA of glutathione peroxidase-1 in patients with the following co-morbidity: MAFLD and DM2, and to compare them with those who only present MAFLD, as well as to relate these levels with certain sociodemographic, anthropometric and biochemical variables. A total of 28 subjects were analyzed, which were divided into 3 groups: the MAFLD and DM2 group, the MAFLD-only group, and the reference control group (healthy). At the end of this work, it was found that none of the sociodemographic variables yielded significant differences. On the other hand, an increase in the levels of TG and cholesterol was found in both groups MAFLD and DM2, and only MAFLD, also ALT and GGT were elevated. The levels of glutathione peroxidase-1 mRNA expression were slightly higher in the MAFLD-only group, compared to the MAFLD and DM2, however, these differences were not statistically significant.

FMED35C

Chronic unpredictable mild stress causes oxidative stress in kidneys in adults rats

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Stress is a response needed to survive. Animals can either adapt to the stressor and restore homeostasis. Or they can continue to experience stress and develop health problems. The unpredictable chronic mild stress (CUMS) protocol increases insulin and serum corticosterone and decreases serum leptin. It causes late effects such as elevated cholesterol, triglycerides, and glucose intolerance. Early and late effects of CUMS on the oxidative stress kidney have not been studied. Therefore, this work aims to evaluate the early and late effects of CUMS on the kidney of adult rats. The experimental design consisted of three groups of 81-day-old male rats: control group (n= 7-8), adult male rats subjected to CUMS during the last two weeks of the experiment (AE2, n=7-8), and adult male rats subjected to CUMS until the last four weeks of the experiment (AE4, n= 7-8). The stress stimuli consisted of 5 different stimuli: reduction of space in a clean cage for 5 hours, restriction of movement for 3 hours, forced swimming in cold water for 10 minutes, forced swimming in warm water for 10 minutes, and reduction of space in a dirty cage for 5 hours. The order of the stressor stimuli was different during each week. Serum and urinary creatinine concentrations were assessed at study termination. The right kidney was used to identify tubulointerstitial fibrosis (Masson's trichrome) and tubular damage around the corpuscles (Schiff's periodic acid staining, tubules showing one or more of the following characteristics were counted: wrinkled and thickened basement membrane, shrunken and simplified epithelium). Kidney triglycerides content, thiobarbituric acid reactive substances (TBARS), superoxide dismutase activity (SOD), catalase activity (CAT), ferric reducing power assay (FRAP), and glutathione (GSHt) were determined in the left kidney. In the AE2 group, the percentage of tubulointerstitial fibrosis tubular damage and the concentration of triacylglycerides in the kidney was increased. An increase in the percentage of TBARS and a decrease in the percentage of FRAP were observed. In the late form, all variables were like the control group. In conclusion, CUMS causes early changes in the kidney of adult rats, which subsequently adapt and recover homeostasis without affecting renal function. These findings are important because if a precipitating factor is introduced at any point in time, the adaptation may be impaired, and kidney damage may continue to progress at a late stage.

FMED36C

Adult rats exposed to a high-sugar diet early in life have permanent histomorphological changes and oxidative stress in the testis

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During childhood and adolescence, excessive food consumption stimulates the expansion of adipose tissue. This promotes obesity in humans, rats, and mice. Obesity in children and adolescents is a predictor of the presence of obesity in adulthood. A high-sugar diet has been identified as a potential factor in the increase in the prevalence of obesity, type 2 diabetes, and metabolic syndrome in children and adolescents. These conditions are associated with poor sperm quality. We hypothesized that early life exposure to a high-sugar diet (HSD) causes permanent damage to testicular histomorphology and oxidative stress, which reduces sperm quality in adulthood. Wistar rats (aged 21 days) were divided into four groups (n = 6-8). In the first experiment, the rats received tap water (control) and a 30% sucrose diet for one month (S30). In the second experiment, the control and 30% sucrose diets were fed for two months (2S30). In the third experiment, the control and 30% sucrose diets were fed for two months, followed by replacement with tap water for two months (IS30). The animals were then euthanized. The testes were collected, and the epididymal spermatozoa were excised. Testicular samples were used to describe morphology using H&E staining and oxidative stress. Epididymal spermatozoa were used to assess sperm quality. Our results show that sperm quality was impaired by consuming HSD and could not be restored by dietary intervention. Overall the HSD caused permanent changes in seminiferous tubule cross-sectional area, oxidative stress, and sperm quality. Therefore, a high-sugar diet in early life causes permanent damage to sperm quality in adulthood.

FMED37C

Adipose tissue as a target organ of reactive oxygen species by cadmium exposure in Wistar rat

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Cadmium exposure is involved in the development of obesity and diabetes in Wistar rats mainly through oxidative stress. However, there are no studies of its effect on adipose tissue. Adipose tissue plays an essential role in energy homeostasis and metabolism in mammals, and its dysfunction is key to the development of metabolic disorders. The objective of this work was to evaluate the effect of cadmium exposure on redox metabolism in the adipose tissue of Wistar rats. In this study, we exposed Wistar rats to two concentrations of Cadmium in drinking water for 2 and 5 months. After this time, the visceral adipose tissue was extracted to evaluate the accumulation of Cadmium by ICP-MS, pro-oxidant molecules (ROS, MDA, 4HDA, N02), and antioxidants (CAT, SOD, GSH) by spectrophotometry, the expression of NRF2 and NF-κB was evaluated by Western Blot and by Finally, we evaluated the morphology of the tissue by histology. The results show that cadmium exposure increases ROS, NF-κB and Cadmium concentration in adipose tissue in a time and dose-dependent manner, coupled with decreased antioxidant activity, as well as NRF2, finally, the tissue hypertrophies after the second month of exposure, but it suffers hyperplasia over time, this could indicate loss of function. These results indicate that adipose tissue could be a target tissue for cell damage generated by exposure to cadmium, in the development of obesity and diabetes.

FMED38C

Evaluation of the Electron Transport Chain Activity and Production of Oxidative Stress in the Brains of Rats with Obesity Induced by a High-Fat and High-Sugar Diet

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Obesity is a public health issue resulting from sedentary lifestyles and excessive consumption of sugars and fats, leading to hyperglycemia, hypertension, and dyslipidemia, promoting an environment of oxidative stress with mitochondrial dysfunction in the central nervous system. For this study, recently weaned male Wistar rats were fed a high-sucrose and high-fat diet for 12 months. Body weight, blood pressure, and glucose tolerance curve were determined. Subsequently, the abdominal fat percentage was determined; the brain was dissected, and mitochondria from the hippocampus (HP) and cerebral cortex (CC) were extracted. The activity of complexes I-IV of the mitochondrial respiratory chain (MRC) in HP and CC was measured by spectrophotometry. To determine oxidative stress, carbonylated proteins in mitochondria and homogenate of HP and CC were measured, and lipid peroxidation using the TBARS test. It was shown that after consuming high-calorie diets, the animals developed obesity, low glucose tolerance, and hypertension. In the evaluation of the MRC complexes from the HP, there was a higher activity of complex I in both diets and complex II only increased in the sucrose group.

In contrast, in CC, sucrose consumption increased the activity of complex I and decreased it in complex III. Finally, oxidative stress indicators showed an increase in carbonylated proteins and peroxidized lipids in the mitochondria of HP and CC in both diets, while in the total homogenate, these markers only increased with the consumption of the high-fat diet. In conclusion, the experimental diets induced changes in MRC activity associated with oxidative stress.

FRCB02C

Recurrent moderate hypoglycemia generates changes in glutamate transport and cysteine uptake for glutathione synthesis

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Moderate hypoglycemia (MH), where blood glucose levels drop between 40-60 mg/dl, is a frequent complication in diabetic patients, who can suffer from up to two hypoglycemic events per week. Recurrent moderate hypoglycemia (RMH) has been associated with increased oxidative stress, lipid peroxidation and a decrease in glutathione (GSH) levels, one of the most important antioxidants in the central nervous system. These changes are involved in the exacerbation of brain damage induced by severe hypoglycemia, whenever it is preceded by RMH. The synthesis of GSH is limited by the transport and availability of cysteine, which can be transported to the brain in the form of cystine through the cystine-glutamate exchange system (Xc^- transporter) in astrocytes, microglia, and cells from the blood-brain barrier. Since the Xc^- transporter releases glutamate to the extracellular space, its activity is closely linked to the astrocytic glutamate transporter GLT-1, main mediator of the extracellular glutamate reuptake in cortex and hippocampus preventing glutamate accumulation and excitotoxicity. Deficient cysteine transport has been associated with a decrease in GSH and neuronal death, and it has been shown that increasing the levels of this amino acid exerts a protective effect on neurons against oxidative stress. Thus, investigating whether RMH can generate changes in the abundance of cysteine transporters is the aim of the present work. Three-month-old male Wistar rats were injected with insulin (6.5 IU) for 1, 3, and 7 consecutive days, and blood glucose level was monitored from the tail vein, every 30 min for 2.5 to 3 h. Animals were sacrificed after 3 h or were recovered by food ingestion and sacrificed 24 h later. Parietal cortex and hippocampus were dissected for immunoblot analysis of the protein levels of xCT functional subunit Xc^- and GLT-1. Also, the abundance of two transcription factors that can mediate the expression of cysteine and glutamate transporters, and are related with the antioxidant response, Nrf2, and NF*k*B p65, were determined.The data show that RMH generates a decrease in GLT-1 levels in the parietal cortex and the hippocampus after 1 and 3 events, which might contribute to the accumulation of extracellular glutamate. However, after 7 RMH events, GLT-1 increases, an effect possibly mediated by NFkB p65, which also increases. GLT-1 and NFkB p65 levels return to basal levels at 24 h when the animals are recovered and glucose is restored. On the other hand, after 7 MH events, the levels of xCT moderately increased in the hippocampus possibly to compensate the decrease in GSH, an effect that was lost 24 h after recovery. In the cortex, the increase in xCT is more delayed as it was observed at 24 h when glycemia is restored. Taken together, our data show that RMH modifies the abundance of glutamate and cystine transporters (xCT) both in the cortex and in the hippocampus, possibly contributing to changes in the availability of cysteine and GSH.
Keywords: hypoglycemia, cysteine and transporter.
Acknowledgements: This study was supported by CONAHCYT A1-S-17357 grant to LM and CONAHCYT 793650 scholarship to AGS.

FRCB04C

The role of O-GlcNAcylation in the development of oxidative stress in MCF-7 cells

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O-N-acetyl-β-D-glucosamine binding, better known as O-GlcNAc, consists of the addition of O-GlcNAc to serine (Ser) and threonine (Thr) residues, is a nutrient-dependent post-translational modification by the hexosamine biosynthetic pathway (HBP). The activity of O-GlcNAcylated proteins has been attributed to processes such as cellular pathway regulation, signal transduction, mitochondrial bioenergetics and cell cycle. Several experimental data have associated O-GlcNAc as part of cancer progression following nutrient availability and demand, exerting modifications in cellular processes or protein-protein activity, such as energy deregulation and thus reactive oxygen species (ROS) production. Some studies have shown that O-GlcNAc regulation influences ROS homeostasis. That said, the present work shows some results on O-GlcNAc expression in an in vitro culture with MCF-7 cells by immunocytochemistry, flow cytometry and cell viability assays. The results have shown O-GlcNAcylation at concentrations of 5mM Glucosamine.

Keywords: O-GlcNAcylation; Oxidative stress; Glucosamine.

FRCB05C

Effect of Cyanidin on oxidative stress and mitochondrial biogenesis in an aortic endothelial cell line (PAE) during Hypoxia-reoxygenation

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Diets rich in fruits and vegetables have beneficial effects on human health they are made up different phytonutrients such as anthocyanins whose beneficial properties include being antioxidants, anti-inflammatory, preventing mutations, interfering with cancer development, and regulating cellular enzymatic functions1. Inflammation is one of the main elements in the development of pathologies such as hypertension, atherosclerosis, endothelial dysfunction, myocardial infarction, and heart failure. Anthocyanins have been shown to have cardioprotective effects2. Part of these mechanisms are also related to cell function and cell organelles such as mitochondria, it is there where most of the cell´s free radicals are generated, in such a way that any alteration in their function can generate stress. oxidative and therefore cell malfunction. The effect of anthocyanins in protecting against damage during ischemia-reperfusion is very important at the mitochondrial level since processes such as cell death are regulated in this organelle3. Anthocyanins can intervene as substrates in complex I of the respiratory chain mitochondrial4. They also participate as uncoupling agents of oxidative phosphorylation and cytosolic cytochrome c5 reduction. In this work, we study the effect of cyanidin on oxidative stress and mitochondrial biogenesis during the hypoxia-reoxygenation process. For this purpose, the expression of antioxidant proteins such as catalase, SOD-2, UCP-2, and GPX-4 was determined by western blot, as well as the proteins related to mitochondrial biogenesis PPARγ, NRF-2, PGC1α, and TFAM. Free radical levels were also determined by flow cytometry using the CellRox and MitoSox fluorescent indicators and, finally, mitochondrial stability was studied by analyzing the transmembrane potential and the expression of respiratory chain proteins. The results show an increase in the expression of mitochondrial antioxidant enzymes at different concentrations of cyanidin, a decrease in the levels of free radicals detected by flow cytometry was also observed, indicating a regulation in mitochondrial activity, finally, stability was observed in the mitochondria because it detected an increase in fluorescence in the presence of MitoTracker and the presence of cyanidin, an indicator of the generation of transmembrane potential after hypoxia/reoxygenation, there is also an increase in the expression of proteins related to mitochondrial biogenesis PPARγ, NRF-2, PGC1α, and TFAM. This result demonstrates the important protection in processes such as hypoxia-reoxygenation, exerted by natural products such as cyanidin, on cellular activity related to mitochondrial stability, both in the reduction of free radical levels and in the increase in expression of free radicals which may be related to the increase in the number of mitochondria due to the increase in the expression of proteins related to mitochondrial biogenesis.

Keywords. Mitochondria, free radicals, cyanidin

FRCB06C

Effect of potential hydrogen on hydrogen sulfide production in *Saccharomyces cerevisiae*

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The vacuole has a pH value of around 5, which is possible due to the vacuolar ATPase (V-ATPase), whose function is to generate electrochemical and proton gradients across the membranes of the vacuolar system. The *VMA1* gene codes for the A subunit of the V-ATPase which contains a cysteine residue (C261) that is related to the catalytic activity of the vacuolar ATPase. It was shown that *VMA1* presents a genetic interaction with *CYS4*, which codes for cystathionine β-synthase. A link between the vacuole and cysteine degradation to release H_2S was identified. Deletion of each of the subunits that make up the V1 subcomplex of V-ATPase, resulted in low H_2S production from cysteine. However, the mechanism by which V-ATPase regulates this is not yet known; recently obtained results seem to indicate that one of these factors is pH. Since the 1990s, it has been determined that deleting any of the genes that code for V-ATPase (*VMA* genes) results in the strain's inability to grow at a pH greater than 7.5.

The results obtained in this work could indicate that the inability of the Δ*vma1* strain to grow in YPD pH 7.5 is due to the fact that the strain does not have the capacity to acidify the medium because the vacuole is not active. On the other hand, it was determined that adding glutathione (GSH) and cysteine (cys) to the medium helps the strain recover the growth phenotype, this indicates that the *CYS4* gene positively regulates, in some way, the *VMA1* gene. We speculat that GSH and cys modify the V-ATPase.

This project was financed by UNAM-DGAPA-PAPIIT through the research programs IN208922 and by the Consejo Nacional de Humanidades, Ciencias y Tecnologías through the Ciencia de Fronteras 2023 program CF-2023-I884.

FRCB07C

Characterization of Sod1 in a non RSS accumulative *S. cerevisiae* Strain

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Cells are constantly exposed to the production and accumulation of reactive oxygen species (ROS). Having to deal with this oxidative stress, the organism has mechanisms that counteract the instability of these molecules by converting them into a more stable form, which is therefore more secure for the cell.

One of these structures is the cytosolic protein superoxide dismutase 1 (Sod1), its main activity is to reduce the superoxide to hydrogen peroxide. Besides, it was recently shown that it has the capability to reduce sulfur compounds.

Most of the experiments done to figure out how Sod1 works are done with a yeast strain that accumulates reactive sulfur species (RSS). Thus, we wanted to determine the Sod1 phenotype in a genetic background that does not retain RSS. For this, we evaluated the behavior of *Δ*sod1 under different stress conditions as well as measured hydrogen sulfide production under these conditions.

This project was financed by UNAM-DGAPA-PAPIIT through the research programs IN208922 and by the Consejo Nacional de Humanidades, Ciencias y Tecnologías through the Ciencia de Fronteras 2023 program CF-2023-I884.

FRCB08C

Visualization of yeast cystathionine beta synthase is dependent on its native 3´ UTR

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Introduction: In this project, emphasis is placed on the Scarless tagging method, which consists of tagging genes by inserting a fluorescent tag at the C-terminal or N-terminal end of a gene of interest without disrupting the endogenous 5' UTR and 3' UTR regulatory sequences and without leaving a "scar" sequence that could affect transcription or translation of the initial gene.
CBS is one of the enzymes of the transsulfuration pathway that produces hydrogen sulfide (H_2S), which is an excellent reductant that readily binds to sulfenic acids (RSOH) and disulfides (RSSR). On these thiol groups on cysteine residues, thanks to the H2S produced, a post-translational S-persulfuration modification is generated. This modification is important because it protects proteins from ROS by preventing their irreversible oxidation. **Methodology:** Two rounds of PCR amplification are used to flank the selection marker URA3 with the tagging protein GFP. The construction is then transformed into bacteria and yeast before the marker is cut. When the selection marker is removed with the appropriate restriction enzymes, the two separate pieces merge by recombination and come together as a full-length fluorescent protein that sticks directly to the gene of interest. **Results:** The use of a newly constructed scarless plasmid on the CBS protein (CBS scarless GFP), has shown a significant difference in the fluorescence signal when compared to a canonical construction of the same protein (CBS GFP) which could perhaps be linked to protein abundance. In addition, differences in localization were also found between the traditional genomic construct and the scarless construct.
Conclusion: It can be expected that genomic integration by the scarless method will be a much more effective and faster alternative for tagging and tracking genes of interest in *Saccharomyces cerevisiae* and in the case of CBS, this technique can be applied to further study the oligomeric regulation and its involvement in enzyme regulation and ROS response.
This project was financed by UNAM.DGAPA-PAPIIT through the research program IN208922 and by the Consejo Nacional de Humanidades, Ciencias y Tecnologías through the Ciencia de Fronteras 2023 program CF-2023-I884.

FRCB09C

Oxidative damage in adult mice caused by chronic exposure to lead since pregnancy and its association with anxiety-like and depression-like behaviors

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Lead is a heavy and toxic metal abundant in the environment; it can hurt several organs, mainly the brain. Some works have shown that lead produces oxidative stress. For example, workers exposed occupationally to lead in a steel factory exhibited high levels of peroxidized lipids in their serum. Also, it decreased the enzyme activity of SOD (superoxide dismutase) compared to the control group (office workers). Similarly, in mice exposed to lead acetate (50 mg/Kg) for 28 days, it was observed a decrease in enzyme activity of SOD, in the whole brain. On the other hand, male adolescent rats exposed to lead acetate (0.2%) in drinking water for 28 weeks exhibited anxiety-like behaviors, while male Wistar rats exposed to lead acetate (500 ppm) in drinking water for five weeks exhibited depression-like behaviors. Because there are reports that women during pregnancy can be exposed to lead, the present study analyzes the effect of lead exposure from pregnancy to adulthood on oxidative damage in the brain and its impact on anxiety- and depression-like behavior. Also, no studies have analyzed if there are sex differences in the effect to lead exposure from pregnancy to adulthood.

In the present study, we analyzed if chronic exposure to lead, from the prenatal stage to adulthood, induces anxiety-like and depression-like behaviors in adult male and female mice. We also examined the effects of lead exposure in oxidative stress in brain areas associated with behavior regulation. We used C57BL/6 mice divided into two groups, one was administered with lead acetate (250 ppm) in the drinking water, and the second was administered only with purified water. We performed the behavioral tests when the mice reached adulthood at 75 postnatal days. We used light-dark and defensive burying behavioral tests to assess anxiety-like behavior. At the same time, the depression-like behavior was evaluated with the tail suspension and the forced swimming tests. After the behavioral tests, mice were euthanized by decapitation, and the following brain areas were extracted: prefrontal cortex, hippocampus, amygdala, and brain stem. In these brain areas were measured reactive oxygen species (ROS).

We found that mice exposed to lead exhibited depression-like behavior but did not exhibit anxiety-like behavior. Also, the mice exposed to lead exhibited high levels of reactive oxygen species in the cortex and hippocampus.

FRCB010C

Effect of vitamin E acetate exposure on the antioxidant response and tight junction integrity of A549 cells in co-culture with THP1

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In September 2019, the U.S. Centers for Disease Control and Prevention warned of a significant increase in e-cigarette or vaping-associated lung injury (EVALI) cases, an acute respiratory failure syndrome characterized by monocytic and neutrophilic alveolar inflammation. The frequency and use of electronic cigarettes have increased among adolescents and young adults in recent years. Vitamin E acetate (VEA) is used as a thickening product in the manufacture of vape liquids containing tetrahydrocannabinol (THC) and some containing nicotine. So far, it is the main substance associated with EVALI and has been found in the bronchioalveolar lavage of patients using electronic cigarettes. Therefore, it is important to evaluate the alterations that may exist in tight junction proteins in the coculture of monocytic cells (THP-1) and human basal alveolar epithelial cells (A549) in the presence of VEA; the latter being responsible for the diffusion of substances through the lung alveoli. A549 cells co-cultured with THP-1 were exposed to different concentrations of VEA for 24 hours and then, their viability was measured by the 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) method, an IC_{50} was determined and the concentration that did not affect viability by 20% or more was selected. The co-culture was exposed to 0.002% VEA for 24 hours, and protein was extracted and quantified by the Lowry method. Changes in the expression of tight junction proteins Claudin 1, 2, 4, and Ocludin were determined by the Western Blot method. Finally, Malonaldehyde (MDA) levels and enzyme activity were measured by Myeloperoxidase (MPO), Gamma Glutamyl Transferase (GGT) and Glutathione S-transferase (GST) assays in culture medium supernatant. The results showed that the explosion to VEA induced an increase in Ocludin and a decrease in Claudin 1, with no significant changes in Claudin 2 or 4. In the enzyme activity assays, a statistically significant increase in GST and MPO was observed, and a decrease in GGT. There was no change in MDA. The data indicate that VEA can be metabolized by GST, generates oxidative stress, and induces the antioxidant activity of MPO, but does not generate lipid damage. Oxidative stress could be involved in modulating the expression of tight junction proteins. (Proyect: FOP16-2021-01 with number 000000000320841.

FRCB011C

Participation of antioxidant enzymes in the protection of the testis and epididymis against temperature changes in the lizard *Holbrookia propinqua*

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The effect of heat stress on the testis, epididymis, and spermatozoa shows as loss of testicular mass, increased cell apoptosis (Lue et al., 1999), decreased sperm concentration, viability, and motility. (Banks et al., 2005; Zhu et al., 2004), which turn in reduction of spermatozoa capacity to fertilize (Yaeram et al., 2006). The cellular response to stress will depend on responsiveness, DNA repair, recombination, and cell cycle regulation (Rockett et al., 2001). The rise in temperature at the gonadal level has negative implications on the secretion of testosterone (T) (Hansen, 2009) which, in turn, has been reported to interfere with the antioxidant capacity of the cell (Izquierdo et al., 2020), generating oxidative stress. Therefore, the cell contains a complex antioxidant defense network that is based on SOD, CAT and GPX enzymes that are responsible for avoiding the harmful effects of ROS (Ortega et al., 2003). *Holbrookia propinqua* is a lizard morphologically adapted to live in sand dunes with warm temperature substrates. Its thermal preferences define it as a thermophilic lizard, considering that occurs in temperatures range between 31°C - 43.1°C (Cooper, 2000). Due to its affinity for temperatures >40°C, it is an ideal model for exploring the mechanisms that maintain the processes of spermatogenesis, epididymal maturation, and sperm fertile potential at high temperatures. This work aims to determine the specific activity of SOD, CAT and GPX in testes and epididymis of *H. propinqua*. For this study, 25 adult individuals were collected in the Playa Escondida town, Tamaulipas. They were transported to the UAMI laboratory, somatic data were taken. Five specimens were sacrificed upon arrival at the laboratory whereas 4 more groups were formed by placing the organisms at different temperatures (28, 32, 37 and 41°C) inside a Hova Bator incubator. After 7 days of acclimation, the organisms were sacrificed by decapitation to extract the organs. For the determination of SOD, xanthine oxidase (XOD) was used. The main function of SOD is the dismutation of O_2- to a more stable ROS, H_2O_2. The activity of GPX is based on the reduction of H_2O_2 and its transformation into water, and for CAT a reaction system containing $KMnO_4$ was carried out. It was found that, in the SOD activity, there are significant differences between the treatments only in the tail of the epididymis, thus demonstrating that this area is where there is greater activity of this enzyme and consequently a greater amount of O_2- generation. Regarding GPX and CAT, no significant differences were found between treatments, indicative that the activity of the different enzymes is participating correctly for the inhibition of H_2O_2 in this specimen, even under warmer temperature conditions.

FRCN01C

I3C modulates tumor capacities in an *in vitro* model of cervical cancer: possible role of AhR in the induction of cell arrest and autophagy

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Cervical cancer (CC) is associated with infection by the human papillomavirus (HPV), from which the E6 and E7 oncoproteins are expressed. E6 participates in the degradation of p53, promoting the evasion of death by apoptosis; E7 induces the release of the E2F oncoprotein, which exacerbates sustained cell proliferation of host cells. Together, these oncoproteins favor two of the most representative tumor capacities. An alternative to control the tumor phenotype promoted by these oncoproteins is through the receptor for aryl hydrocarbons (AhR), which is a ligand-dependent transcriptional factor, which has been shown in *in vitro* and *in vivo* models to be capable of regulating the protein levels of p53 and the E7 oncoprotein. This regulation of the protein levels of p53 and E7 results in the control of tumor phenotypes, in accordance with previous functional studies that have evaluated the potential capacity of the AhR to modulate the levels of sustained cell proliferation, as well as the levels of apoptosis. However, it has not been shown that AhR activation is capable, in the same study model, of controlling the levels of proliferation and non-necrotic cell death (apoptosis and autophagy). In the present work, we evaluated whether the activation of the AhR can lead to the inhibition of cell proliferation and the promotion of non-necrotic death, particularly apoptosis or autophagy, which are two types of ordered death that do not generate secondary effects. HeLa cell cultures were used to determine the effect of the natural AhR ligand indole-3- carbinol (I3C) on cell viability. At the same time, the changes in the time in which the different stages of the cell cycle were developed, as well as the potential capacity of I3C for the induction of apoptosis, autophagy and necrosis were determined. So far, a decrease in cell number has been observed with treatment with 150 uM I3C. Subsequently, we evaluated whether this decrease was due to alterations in the cell cycle (cell arrest) or to the induction of cell death (apoptosis, autophagy or necrosis). We observed that there was a slight arrest in the cell cycle, however, the most notable changes by the treatment with I3C 150 uM were observed in the induction of autophagy, the above due to the increase in p62 according to the WB. Apoptosis - evaluated through flow cytometry, immunofluorescence and the detection of molecular markers such as Caspase 3 - did not show substantial changes compared to the control groups; necrosis was also not increased in the groups treated with the AhR ligands. According to the results obtained so far, it can be concluded that the AhR is a protein that can become a potential therapeutic target, since it has been shown to participate in more than one tumor phenotype, which can be activated through exposure with natural ligands, without the risk of developing side effects.

FRCN02C

Antioxidant effect of isothiocyanates from *Brassicas* vegetables on benzo(a)pyrene-induced reactive oxygen species production in a cell transformation model

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Epidemiologic studies have revealed an inverse correlation between dietary intake of cruciferous vegetables and the risk of breast cancer. Crucifers, such as broccoli and cauliflower, contain bioactive compounds that are believed to have protective effects against cell transformation and the development of cancer. These compounds, such as glucosinolates and their derivates, including isothiocyanates (ITC), have been shown to have antioxidant and detoxifying properties that can counteract the negative effects of reactive oxygen species (ROS) generated during cell transformation. In particular, the exposure of MCF-10 A cells (a non-tumorigenic mammary cell line) to benzo(a)pyrene, a common environmental carcinogen, can induce the formation of ROS and increase oxidative stress. However, studies have shown that the compounds present in cruciferous vegetables can modulate ROS levels and reduce harmful effects. It has been observed that sulforaphane, an isothiocyanate present in cruciferous plants, can increase the activity of antioxidant enzymes, such as superoxide dismutase, which reduces the accumulation of ROS and protects cells from oxidative stress. This study we found that different concentrations of *Brassicas* ectracts modulated the production of ROS and cellurar proliferation in cell exposed to benzo(a)pyrene. This suggests that the ITC present in *Brassicas* could have a protective effect against carcinogens by preventing ROS generation and by modulating celular proliferation. These findings highlight the potential of ITC as agents that can help counteract the harmful effects of carcinogens on cells. In summary, the compounds present in cruciferous extracts regulated the production of ROS during cell transformation mediated by the carcinogen benzo(a)pyrene in MCF-10 A cells, exerting antioxidant and detoxifying effects that could help to protect against the development of cancer. However, it is important to emphasize that research in this field is still ongoing and more studies are needed to fully understand the specific mechanisms and effects of these interactions.

FRCN03C

Analysis of oxidation and ERK activation markers in databases of patient samples and cervical cancer cell lines

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Among the types of cancer (Ca) affecting women, cervical cancer (CC) is a public health problem with high incidence and mortality in the world. It is currently classified into two main subtypes according to their histological type: squamous cell carcinoma (SCC), which is the most prevalent, and adenocarcinoma (AC), which has the worst prognosis; but there are also other less frequent cancers such as adenosquamous (AdSq) and neuroendocrine. There are several risk factors associated with the development of this pathology, being the Human Papilloma Virus (HPV) the main one, whose infection in cervical cells results in inflammation and the subsequent generation of reactive oxygen species (ROS). ROS have been related to malignant transformation in several types of cancer such as breast cancer (BrCa), gastric cancer and lung cancer. At basal levels, ROS can act as second messengers in signaling pathways, and an increase in their concentration has been linked to the overactivation of signaling pathways such as ERK. The ERK pathway is a signaling cascade involved in cell proliferation and differentiation processes that has been found to be altered in several types of Ca, favoring the malignant transformation of cells. Therefore, different studies have suggested the supplementation of antioxidants or inhibitors of the ERK pathway as a targeted therapy. In this project we analyzed the basal levels of oxidation and ERK activation in patient databases and in CC cell lines with different histological type, HPV genotype and integrated viral genome copy number. The results showed that, contrary to expectations, there appears to be no relationship between ROS levels and ERK activation in *in vitro* models of CC, however, we found a relationship between high levels of ROS with HPV genotype and integrated viral genome copy number. Interestingly, differences in ERK activation were observed between the histological types of CC: SCC and AC, while ROS levels between these histological types do not differ and were only related to HPV genotype or viral copy number. Furthermore, antioxidant treatment on HaCaT, SiHa, CaSki and HeLa cell lines decreased proliferation and survival, suggesting an important role for ROS in the proliferation and survival of these cell lines.

FRCN06C

Hormetic response to oleic acid on human glioma cells in a medium enriched with reactive oxygen species

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Introduction.

Oleic acid (OA) is a monounsaturated omega-9 fatty acid in plant and animal sources. Evidence shows that OA can reduce the risk of cancer, such as breast, colorectal, and prostate cancer. It is known that an increase in reactive oxygen species (ROS) has a pivot effect in cancer cells that produce nanomolar concentrations in the tumor environment, and it can induce apoptosis. On the other hand, ROS also acts as a second messenger in biological processes.

Objective

To evaluate the effect of oleic acid on different human glioma cell lines in an environment enriched with reactive oxygen species

Methods

Human glioma cell lines LN18 and MO59K were stimulated with different concentrations of H_2O_2 [10,12.5,25,50,100,200] µM for 30 min, 2 h, and 24 h followed by OA treatments at different concentrations [10, 25, 100, 200] µM. Cell viability was evaluated with the MTT assay. For each trial, the statistical tests were performed.

Results and discussion

It was observed that the OA at 24 h had a hormetic effect in a medium with an increase in ROS induced by the presence of H_2O_2. The damage produced by the stimulus of H_2O_2 [50µM] for 30 min was rescued by concentrations of 50, 100, and 200 µM of OA; instead, the high concentrations of OA that increased the cytotoxicity induced by H_2O_2 in 2 and 24h, synergistically. The OA showed no effect on the cell lines incubated for 24 h.

FRND01C

Evaluation of mitochondrial structure and function in a model of cerebellar vacuolar degeneration in rats with portocaval anastomosis

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Portosystemic Shunt (PCS) consists of the partial or complete diversion of portal blood to systemic circulation without passing through the liver. PCS model is widely used to study the effects of liver dysfunction (Papamichail et al., 2018). In addition, this model is associated with Hepatic Encephalopathy (HE), affecting the quality of life and daily function activities in patients with chronic liver disease (Ochoa-Sanchez & Rose, 2018). The underlying mechanism responsible for HE is not fully characterized. In studies carried out in our laboratory after 13 weeks of treatment, a vacuolar/spongiform degeneration was evident only in the cerebellum cortex. Physiopathology was characterized by cellular, metabolic, and behavioral damage, as well as edema formation followed by a neuroinflammatory process. Therefore we suggest hyperammonemia caused by PCS promotes cerebellar damage (López-Cervantes et al., 2021). In this context, the aim of this project was to characterize mitochondrial dysfunctions and the prooxidant state in the cerebellum cortex. *In vivo*, using mitochondrial and cytosolic fluorescence dyes. *In vitro*, using classical biochemical methods, ultrastructural analysis, and molecular biology techniques. We found there is a differential response of mitochondrial and cytosolic reactive oxygen species (ROS) between each of the cellular layers of the cerebellum cortex as well as ultrastructural abnormalities, resulting in a change of their shape in mitochondrial networks (in Purkinje neurons). Also, an increase in the number of lysosomes Additionally, ROS increased lipid oxidation, however, carbonylated protein levels decreased in PCS rats. In conclusion, there are mitochondrial dysfunctions and high levels of ROS in the cerebellar cortex with PCS. Thereby, hyperammonemia may contribute to the vulnerability of the cerebellum to PCS-induced ammonia damage oxidative stress (Kosenko et al., 2017). Our current results suggest differential management of ROS in the cerebellar cortex in the same way in protein degradation associated with morphological changes in mitochondrial networks of PCS rats and antioxidant defense.

Keywords: Portosystemic Shunt, Cerebellum, Reactive Oxygen Species, Mitochondrial dysfunctions, Neuroinflammation.

FRND02C

Evaluation of the neuroinflammatory state and oxidative stress in older adult women with obesity

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Obesity has been related to different pathologies such as diabetes, sarcopenia, and osteoporosis among the most important. Obesity is characterized by presenting systemic, chronic, and low-grade inflammation, which has been related to increasing the decline of obesity-associated diseases. During normal conditions, the brain is protected by the blood-brain barrier, which is responsible for regulating the passage of a large number of toxic substances and molecules, which could damage the nervous tissue. However, it has been reported that during obesity, some inflammatory molecules are capable of crossing the blood-brain barrier and causing neuroinflammation, mainly by promoting the expression of Reactive Oxygen Species (ROS), as occurs with the pro-inflammatory cytokine TNF-α. The ROS-generated low-grade chronic inflammation is a risk factor for cognitive damage and neurodegeneration, as observed in different pathologies such as Alzheimer's and Parkinson's. One of the groups most likely to be affected by obesity may be adult women over 60 years of age, since after menopause, when hormone levels such as estradiol decrease, women tend to increase body fat, which leads to an increase in the inflammatory state and oxidative stress. That is why the aim of this work is to evaluate the inflammatory state, oxidative stress, and neuroinflammation in elderly women with obesity and relate these factors to their cognitive state. Our results showed that obese women have a higher systemic pro-inflammatory profile compared to non-obese older adult women. In addition, it was observed that the group of obese women present a more oxidized redox state than the non-obese and a higher degree of oxidized proteins. This suggests that obesity in older women is a risk factor in the development of neuroinflammation and cognitive damage during aging.

This Project is financed by FORDECYT-PRONACES/263957/2020 and Ayudas a la investigación Ignacio H de Larramendi 2022. Fundación MAPFRE.

FRND03C

Vanadium pentoxide (V2O5) inhalation as an induced model of Alzheimer's disease

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Alzheimer's disease (AD) is the most common neurodegenerative pathology worldwide, it has been reported that approximately 15 million people suffer from this disease, the incidence annually increases 0.5% in 65 year old people and 8% in 85 year olds; although it was described more than a 100 years ago and there is a lot of research being done about this pathology, it has been difficult to find an animal model that replicates all the characteristics of the neurodegenerative process of Alzheimer's. Previous experiments in our laboratory have shown that chronic exposure to vanadium pentoxide in rats causes morphological and behavioral changes similar to those seen in Alzheimer's disease. To this end 40 male Wistar rats were randomly divided into two control and two experimental groups (n = 10) with an initial weight of 180-200 gr. The experimental groups were exposed to V_2O_5 0.02M for 1 h, 3 times a week, for 6 months, after 6-month exposure one experimental group was leaved in a 6-month recovery phase. To measure behavioral changes, the four groups were trained in the T-maze test that assesses spatial behavior and an open field test for 10 mins. All groups were evaluated once a month for 6 or twelve months. To measure histological alterations, after 6 or 12 months of inhalation, frontal and entorrinal cortex, CA1, subiculum and amygdala, underwent Congo red or argentic Bielschovsky impregnation and were analyzed. Memory results in the T-maze show memory impairment since the group had been exposed for three months to V_2O_5. Chronic inhalation of V2O5 is a model of induced AD that causes alterations in spatial memory and motor behavior, accumulation of βA in the vascular endothelium and pyramidal neurons of the frontal and entorhinal cortex and accumulation of intraneuronal NFTs in pyramidal neurons of CA1 and subiculum and loss of dendritic spines, which allows us to propose a model that relates clinical manifestations with histological changes. Being an induced model represents sporadic AD which, although it represents more than 95% of cases, there are few models for it. This model represents neurodegenerative changes specific to AD in which we observe production and accumulation of amyloid and plaques first in certain structures, production, and accumulation of NFTs in other structures, and damage to certain types of memory. There is a decrease in the number of neurons in the cortex and CA1, these alterations are not reversible after six months, indicating that once the neurodegenerative process is established and homeostasis is broken, the damage multiplies. Our model is compatible with Braak stage IV of Alzheimer's disease, which represents a moment where it is feasible to propose therapies that have a positive impact on stopping neuronal damage.

Alzheimer´s Disease, Vanadium Pentoxide, NFTs
PAPIIT-DGAPA IN216821

AARD01C

Effects of Senolytic and Senomorphic Treatments on Systemic Redox State in an Obesity Model of Middle-Aged Female Rats

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Overweight and obesity are a growing problem worldwide and are associated with various comorbidities, with a higher prevalence among women. In recent years, adipose tissue, particularly in the visceral compartment, has been recognized not only as a simple energy storage tissue but also as an active endocrine organ that releases a variety of biologically active molecules known as cytokines. Due to the complex interplay of these cytokines, obesity is characterized by chronic low-grade inflammation and persistently elevated oxidative stress. Cellular senescence is a response to increased oxidative stress levels, and emerging evidence suggests that different cell types are highly susceptible to senescence with both aging and obesity. Senescence is associated with adipocyte hypertrophy (inappropriate expansion), insulin resistance, dyslipidemia, and various diseases. Significant efforts have been made to identify approaches to eliminate senescent cells, including the use of "senolytic and senomorphic" compounds. The most well-established senolytic treatment to date is the combination of dasatinib and the antioxidant quercetin, which reduces cellular senescence and improves chronic disorders in experimental animal models. Sulforaphane, derived from edible cruciferous vegetables, is one of the most potent inducers of phase II enzymes and acts as a strong activator of the Nrf2-Keap1 signaling pathway. This activation enables Nrf2 to evade Keap1-dependent degradation, leading to the stabilization and nuclear accumulation of Nrf2. The aim of our study was to determine how the administration of dasatinib+quercetin and sulforaphane treatments could affect the systemic redox state in an obesity model using middle-aged female Wistar rats. Female Wistar rats were fed a hypercaloric diet (HD) from 21 days after birth until they were euthanized at 14 months of age. SFN (0.5 mg/kg) was administered subcutaneously, five days a week for two months, while D+Q (5 mg/kg and 50 mg/kg, respectively) were administered via nasogastric tube once a month for two months. At 14 months of age, the rats were euthanized, and blood samples were collected to separate the serum and plasma. In the serum samples, we evaluated the levels of oxidized glutathione and reduced glutathione (GSH/GSSG) and observed changes in the induction of antioxidant response and the redox state (GSH/GSSG) levels.

We thank Dr. Guerrero-Aguilera from UAM-I for animal supply. This work was supported by CONACyT grant FORDECYT-PRONACES/263957/2020T. Salas-Venegas, Santín-Márquez &Flores-Torres are CONACyT scholarship holders.

AARD02C

Nucleoredoxin activity is altered by oxidative stress generated by chronic alcohol consumption and is associated with increased cellular senescence in the liver of aged mice

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Background and Aims: Aging, a natural process associated with abnormal oxidative stress production, involves several cellular and molecular changes that ultimately compromise the well-functioning of organs and systems in multicellular complex organisms. Aging also declines the liver metabolic capability and as a consequence, the efficiency to eliminate toxic agents is significantly decreased. These alterations are closely related with an increased production of reactive oxygen species (ROS) which is exacerbated by the chronic alcohol consumption; however, the underlying mechanisms had not been clarified. Nucleoredoxin (NXN) is both an oxidoreductase that targets ROS and a redox-sensitive enzyme that regulates key cellular processes trough redox protein-protein interactions. Here, we aimed to determine the involvement of NXN in alcoholic liver disease (ALD) during aging in the mouse liver. **Methods:** ALD was recapitulated in 7-week-old (young) C57BL/6J female mice, 12- month-old (adult) and 18-month-old (aged) by the chronic ethanol consumption (20%) in 20% of sucrose for eight weeks and a single dose of lipopolysaccharide (1 mg/kg). We evaluated histological and cellular alterations, cellular senescence markers, NXN protein level and NXN-dependent protein-protein interaction status, as well as, the gene expression profile changes. **Results:** During aging, the ALD model increases the levels of cellular senescence markers, such as senescence-associated beta-galactosidase and H2A.X levels. Interestingly, the level of oxidized proteins was significantly increased, and NXN protein level decreases; as well as, the ratio of NXN/FLII, NXN/MYD88 and NXN/PFK1 interaction complexes were significantly disrupted by ALD during aging. A RNA-seq analysis revealed that the ALD model modified the expression of genes related to cell cycle arrest, oxidative stress regulation, induction of cellular senescence and lipid metabolism in aged mice. **Conclusions:** Our results show that the ALD model increases oxidative stress during aging. Of note, the level of NXN redox-sensitive enzyme was decreased in the liver of aged mice subjected to ALD model, but more interestingly, the interaction ratio of NXN- regulated proteins such as FLII was importantly modified by the ALD model. Thus, this evidence strongly suggests that ROS produced by the ALD oxidize proteins and sensitize the liver cells and as a result, the NXN-dependent regulation is altered, a phenomenon that might accelerate the establishment of cellular senescence in the liver of aged mice.

AARD03C

Differences in redox state in primary rat cerebral cortex astrocytes induced to cellular senescence or gliosis with palmitate

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Since obesity is a global health problem, in this study we used palmitate to simulate this pathological environment in a cellular context to study the effects of obesity on neuroinflammation. Astrocytes are the most abundant cells of the Central Nervous System (CNS) and show an adaptive plasticity that defines the functional maintenance of said system. When a stressful stimulus happens in the CNS, astrocytes can enter in a state of cellular senescence (SC) or be activated to gliosis. Both phenotypes play an important role in the processes of neuroinflammation and neurodegenerative diseases associated with aging. Therefore, in this study we compared the redox state of this phenotypes to help to understand their differential participation in neuroinflammation caused by obesity.
To achieve this, primary cultures of neonatal Wistar rat astrocytes (3-5 days) were performed. Cells were exposed to a Palmitate-Albumin conjugate (1:6) at different concentrations: 200μM for senescence induction, and 40μM for gliosis, during 24 h. SC and gliosis markers were evaluated, as well as viability, proliferation, and SA-β-Gal. Finally, the redox state of both inductions was determined by evaluating the GSH/GSSG ratio by HPLC. Using the Oily Red technique, we confirmed that palmitate (PA) was introduced into the cells. Significant differences were found in the treatment cells proliferation with respect to the control, both in senescent and gliosis. The SA-β-Gal and safranin assay showed that the treatment with PA increased the number of senescent cells, but not the control or gliotic cells. The senescence state and gliosis were confirmed using immunofluorescence tests with markers of senescence (γH2AX, Lamin B1, p16 and B-gal) and gliosis (C3, S100A and GFAP). Finally, the redox state of both was evaluated and differences were found between both phenotypes.
We thanks to Dr. Ángeles Guerrero-Aguilera. This Project is financed by - FORDECYT-PRONACES/263957/2020. LTM and AGKE are CONAHCyT scholarship holders.

AARD05C

The consumption of *Sechium edule* (chayote) prevents telomere attrition in older adults with metabolic syndrome

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Metabolic síndrome (MetS) is characterized by a combination of metabolic abnormalities and is related to the accumulation of oxidized products and deficiency of antioxidant mechanisms. One of the most accepted theories of aging is associated with shortening of telomere length (TL) during each cell duplication. Leukocyte telomere length (LTL) is considered a biomarker of cellular aging that also has a close relationship with MetS. Also, is well established that the increase in the number of MetS components is associated with a shortening of the LTL. In this context, it is necessary to find therapeutic strategies that counteract OxS, improve antioxidant protection mechanisms, and avoid or prevent the shortening of LTL and MetS related complications. In this regard, *Sechium edule* (chayote) is an edible fruit of the *Cucurbitaceae* family, to which various beneficial properties for human health have been attributed. However, it is still unknown what role it may play in TL dynamics and if it could counteract telomere attrition, and what biochemical, OxS or inflammation markers might be involved. Hence, the objective of this study is to determine the effect of *Sechium edule* consumption on OxS, inflammatory markers, and its association LTL attrition in older adults with MetS. **Objective:** To determine the effect of the consumption of *Sechium edule* (1.5 g/day) for six months on oxidative stress (OxS) and inflammation markers and its association with telomere length (TL) in older adults with MetS. **Methods:** The study was conducted in a sample of 48 older adults: placebo (EP) and experimental (EG) groups. Lipoperoxides, protein carbonylation, 8-OHdG, total oxidant status (TOS), SOD, GPx, CAT, total antioxidant status (TAS), inflammatory cytokines (IL6, IL10, TNF-α), and TL were measured before and six months post-treatment. **Results:** We found a significant decrease in the levels of lipoperoxides, protein carbonylation, 8-OHdG, TOS in the EG in comparison PG. Likewise, a significante increase of TAS, IL-6, and IL-10 levels was found at six months post-treatment in EG in comparison with PG. TL showed a statistically significant decrease in PG compared to post-treatment EG. **Conclusions:** Our findigns showed that the supplementation of *Sechium edule* has antioxidant, and anti-inflammatory effects, and diminushion of shortening of telomeric DNA in older adults with MetS. This would shows that the intervention with *Sechium edule* has a posible geroprotective effect by preventing telomeres from shortening as usually happens in these patients. Therefore, suggesting a protection of telomeric DNA and genomic DNA.

Key words: *Sechium edule*; metabolic syndrome; telomeric length.

AARD06C

Effect of chronic administration of resveratrol on the redox balance of the prefrontal cortex of the male Wistar rat during aging

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Aging is a dynamic process characterized by a progressive deterioration of physiological functions and is one of the most important risk factors for the appearance of neurodegenerative diseases. Cognitive functions decline with age due to the structural and functional changes generated in aging brain. In this sense, oxidative stress is one of the most relevant factors involved on the appearance of aging, based on the damage generated by reactive oxygen species (ROS) and nitrogen species (RNS) in biomolecules, compromising the redox state in tissues. The prefrontal cortex (PFC) is the most developed area of the human brain, responsible for higher cognitive functions. Stress generated in aging leads to changes in the function and morphology of the PFC, compromising behavior and memory. Various strategies have emerged to reduce the negative physiological impact of stress derived from aging, such as the use of antioxidant molecules, and research suggests that chronic administration of molecules, such as resveratrol, maintains the redox balance of the PFC. The objective of this work was to evaluate the effect of resveratrol on the redox balance during aging of the PFC in male Wistar rats. 135 male Wistar rats were randomly divided in 3 groups: control, vehicle (7.5% ethanol), and resveratrol (7.5% ethanol + 10 mg/kg/d of resveratrol). The administration was evaluated every 2 months, for 18 months. Biochemical and enzymatic tests were performed at CPF for the evaluation of markers of oxidative damage (•NO, MDA, 4-HNE), antioxidant function (SOD, CAT, GPx) and glutathione system (GT, GSH, GSSG, GST, GR). Results showed that aging increases •NO levels and generates a maximum accumulation of MDA and 4-HNE at 10 months in control group. Likewise, it decreases the activity levels of SOD, CAT and GPx. Total and reduced glutathione (GSH) levels fluctuated throughout the intervention period, maintaining a prooxidant redox index. Resveratrol promoted the decrease of •NO levels and the maintenance of MDA and 4-HNE levels with a similar pattern to the control group. The activity levels of SOD and GPx increased towards the end of the treatment, as well as the levels of total glutathione and GSH, observing an antioxidant redox index towards 16 months of treatment. In conclusion, in the PFC, the aging process registered modifications and adaptations of the redox system to prevent neurodegeneration. The chronic administration of resveratrol at a dose of 10mg/kg prevented the overproduction of •NO and lipid peroxidation in the PFC, improving the activity of the detoxifying enzymes SOD, CAT and GPx and preserving the glutathione system with a greater impact in the stage of late old age.

AARD07C

Tai Chi Training vs. Strength Training on Markers of Oxidative Stress and Inflammation

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Aging is associated with the development of chronic-degenerative diseases (cardiovascular, neurodegenerative, type 2 diabetes mellitus (T2DM), and cancer), inflammation, oxidative stress (OxS), and a negative impact on muscle tissue. Loss of skeletal muscle mass has been associated with sedentary lifestyles, inflammation, and OxS. The latter is associated with damage to various biomolecules such as lipids, proteins, and DNA; For its part, inflammation favors the loss of strength, muscle mass, and functionality. Clinical studies; have shown that physical training exerts beneficial effects on antioxidant and anti-inflammatory status. Tai Chi (TC) is a type of physical exercise of moderate intensity and its practice in recent years has been increasing among the elderly; since it has a positive impact on the functioning of the cognitive, cardiovascular, respiratory, and neuromuscular systems. Another type of moderate-intensity exercise is strength training (ST), which optimizes cardiorespiratory and muscular function and increases muscle mass and strength. Objective: To evaluate the effect of moderate intensity (Tai Chi) versus resistance exercise on markers of oxidative stress and inflammation and their relationship with muscle mass in older adults. Methods: Plasma TOS determination was performed using the commercial kit (Rel Assay Diagnostics, Gaziantep, TR). Total plasma antioxidant status (TAS) levels were quantified using 2,20-azino-bis (3 ethylbenzthiazoline-6-sulfonic acid) (ABTS) (Randox Laboratories Ltd., Antrim, UK). Measurement of 8-OHdG was carried out with an ELISA kit (Wuhan Fine Biotech Co., Ltd., Wuhan, Hubei, CHN) according to the manufacturer's instructions. Carbonylated proteins were evaluated using 2,4-dinitrophenylhydrazine (DNPH). The levels of interleukin-6 (IL-6), interleukin-10 (IL-10), interleukin-8 (IL-8), and tumor necrosis factor-alpha (TNF-α) were determined in serum samples by the cytokine bead array (CBA) Human assay; using the human inflammatory cytokine kit (BD Biosciences, San Jose, CA, USA). The body composition was determined by evaluating the total fat content: fat mass (FM) and skeletal muscle mass index (SMMI), using a bioelectrical impedance analysis (BIA), using four-pole mono-frequency equipment (50 kHz, Quantum X, RJL System). **Results:** The results showed a decrease in the amount of 8-OHdG in the serum of TC practitioners from 26.97 ± 3.53* (ng/mL) at baseline to 19.76 ± 3.89 (ng/mL). mL) after six months of training. People who did ST had an increase in the number of carbonylated proteins from 35.64 ± 13.96 to 47.68 ± 16.85* (nmol/mg); Regarding the TOS of the same group, an increase from 5.28 ± 2.55 to 11.42 ± 6.17* and a reduction from 1.05 ± 0.206 to 0.86 ± 0.124* was also observed for the same group of strength training for TAS. Inflammation markers showed an increase in IL-8 in people who practiced TC after six months of training from 16.93 ± 5.05 to 29.90 ± 14.23*. ANOVA of repeated measures test, significance level 95%,* Statistical significance based on their respective group basal ($p < 0.05$). **Keywords:** Exercise, Oxidative Stress and Inflammation.

AARD08C

Preventive Effects of (-)-Epicatechin in a model of retinal degeneration induced by $NaIO_3$

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Age-Related Macular Degeneration (AMD) is the leading cause of irreversible blindness in adults, and its incidence has been increasing. One of the hypotheses regarding the pathophysiology of AMD is the presence of an imbalance of Reactive Oxygen Species (ROS) in the Retinal Pigment Epithelium (RPE) and photoreceptors, which leads to loss of central vision in advanced stages. In this study, we aimed to evaluated the potential of the flavanol (-)-Epicatechin (Epi) to reduce the detrimental effects of ROS on mitochondrial biogenesis, as well as on the structure and function of the RPE and photoreceptors, using a mouse model of retinal degeneration induced by sodium iodate ($NaIO_3$). Female C57BL/6 mice were randomly assigned to one of three treatment groups: 1) $NaIO_3$ group, where a single intraperitoneal dose of $NaIO_3$ at 35 mg/kg was administered on day 15 of treatment. 2) Epi/$NaIO_3$ group, receiving a daily oral dose of 1mg/kg of Epic from day zero to day 20 of treatment, plus 35mg/kg of $NaIO_3$ intraperitoneally on day 15. 3) Control group, receiving a daily administration of 100 µl of water for 20 days. After treatment period, the mice were euthanized by cervical dislocation and the eyes were collected for further analysis. Phalloidin and hematoxylin-eosin staining were performed to compare the epithelial structure and conduct a nuclei count between groups. Additionally, the expression of proteins related to mitochondrial biogenesis and oxidative stress was quantified, and a TUNEL analysis was conducted. Our findings demonstrated that treatment with (-) Epi can reduces structural damage and cell death, suggesting a potential early prophylactic benefit of dietary supplementation with (-)-Epi in mice. These results may have significant implications for AMD patients. keywords: Oxidative stress, Age-Related Macular Degeneration, Flavonoids.

FRMS01C

Glutathione and thioredoxin pathways in *Candida glabrata*

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Redox homeostasis systems regulate cellular biological responses. These systems are present in all living organisms and are divided into two pathways: the glutathione pathway (GSH/Glr), which includes the antioxidant molecule glutathione (GSH) and the enzyme glutathione reductase (Glr); and the thioredoxin pathway (Trx/Trr), which includes the thioredoxin (Trx) and thioredoxin reductase (Trr) enzymes. Both pathways proceed through sequential oxidation-reduction reactions once they react with their target. As a result of the importance of maintaining redox balance within the cell, the glutathione and thioredoxin pathways function as background systems for each other. In the pathogen yeast *Candida glabrata*, the Trx/Trr system has two cytosolic thioredoxin reductases (Trr1 and Trr2) and a cytosolic (Trx2), and mitochondrial (Trx3) thioredoxins; while the GSH/Glr1 system has GSH and glutathione reductase (Glr1) located in cytosol and mitochondria. To elucidate the contribution of these systems to the different responses that *C. glabrata* activates during interaction with its host, we have generated different mutants in the components of the GSH/Glr and Trx/Trr pathways. We found that *GSH1*, which encodes for the γ-glutamyl-cysteine synthetase, is an essential gene. A null mutation in *GSH2* (*gsh2*Δ), which encodes for glutathione synthetase shows a phenotype of slow growth, sensitivity to metals, oxidative and heat stress. The single mutants *trr1*Δ and *trx3*Δ show growth and response to oxidative or heat stress like the wild type strain, while the mutants *trr2*Δ and *trx2*Δ show a slight decrease in growth and are highly sensitive to oxidative stress, and only *trx2*Δ is sensitive to heat stress. Interestingly, Trr1, Trr2, Trx2 and Trx3 are required for survival in the presence of human polymorphonuclear cells. The double mutant *trr1*Δ *trr2*Δ is sensitive to heat stress. We constructed double mutants affecting both the GSH and Trx systems and were able to only get *gsh2*Δ *trr1*Δ, *gsh2*Δ *trr2*Δ, and *gsh2*Δ *trx3*Δ. These double mutants show slow growth and cannot use non-fermentable carbon sources (glycerol or ethanol) indicating dysfunction in the mitochondria. Null mutations in the mitochondrial Glr1 and Grx2 (glutaredoxin 2) have an increased resistance to H_2O_2 stress, which indicates a deregulation of the H_2O_2 signaling circuitry. Interestingly, we have observed that mitochondrial Prx1 (peroxiredoxin), which has a key function in signaling the oxidative state towards the cytosol, interacts with Glr1, Grx2, and Trx3. This indicates that there is an overlap between the GSH/Glr1 and Trx/Trr systems in mitochondria maintaining the redox balance to withstand different growth and stress conditions.

FRMS02C

Nitric oxide mediates Kaposi's sarcoma-associated herpesvirus lytic replication

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Introduction: Kaposi's sarcoma-associated herpesvirus (KSHV) is an oncogenic virus associated with several human malignancies, including Kaposi's sarcoma (KS), a vascular cancer commonly seen in AIDS patients, and a rare type of lymphoma named primary effusion lymphoma. The KSHV life cycle consists of two phases, one latent and one lytic, this late has a critical role in KS progression by disseminating the virus and producing viral or inducing cellular proinflammatory and proangiogenic factors. Numerous viral and cellular factors have been shown to regulate KSHV lytic replication, including reactive oxygen species. In this study, we investigated the role of nitric oxide (NO) in lytic viral replication. **Methods:** Two different cell culture systems, TREx BCBL1-Rta cells and iSLK- RGB-Bac16 cells, were used to examine lytic replication of KSHV. Nitric oxide generation during reactivation was measured by Griess reaction and by fluorescence microscopy using DAF-2DA (4,5-diaminofluorescein diacetate solution). Both cell types were treated with N(ω)-nitro-L-arginine methyl ester (L-NAME), a competitive inhibitor of nitric oxide synthase (NOS), or with a NO donor, S-Nitroso-N- acetylpenicillamine. Viral replication was analyzed by measuring levels of transcription and protein production using RT-PCR and Western Blotting, respectively. Virion production was measured by titration in MM cells and the results were confirmed by flow cytometry analysis in iSLK-RGB-Bac16 cells. **Results:** It was found that after lytic reactivation, both TREx BCBL1-Rta and iSLKRGB- Bac16 cells increased endogenous NO and L-NAME treatment reduced it. Interestingly, chemical inhibition of NO generating with L-NAME blocked KSHV lytic replication as observed by reduced levels of viral lytic protein and transcripts and production of infectious virions. In contrast, exogenous addition of a NO donor (SNAP) was sufficient to trigger lytic replication of KSHV. **In conclusion**, these results demonstrated that NO has a crucial role in KSHV lytic replication and that NO inhibition could be a new strategy to control KSHV-induced malignancies. However, the mechanism of NO induction during KSHV lytic replication as well as the mechanism of NO regulation of KSHV lytic replication remain unclear.

FRMS03C

Nitric oxide and SARS-CoV-2, evidence of high serum nitrite concentrations in hospitalized patients with COVID-19

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INTRODUCTION: Nitric Oxide (NO) is a free radical with multiple functions, acting as an antimicrobial and anti-inflammatory agent in adequate concentrations. Its role in defense against respiratory viruses, including SARS-CoV and more recently as a possible treatment against SARS-CoV-2 has been documented. However, there is no evidence on the generation of NO in patients with CoVID-19, so the objective of this study was to determine the plasma concentration of nitrites (main metabolite of NO) as an indirect measure of NO generation, in hospitalized patients with COVID-19. **METHODS:** A cross-sectional study was conducted from August 2021 to January 2022 at the Cuernavaca General Hospital including 144 patients with COVID-19 and 381 healthcare workers without COVID-19 (control group). The concentration of NO in previously deproteinized serum was quantified by measuring nitrites using the Griess reaction. Statistical analysis was performed for comparison. **Results:** Among the control group (uninfected people) an average serum nitrite of 13 µM (±11.3) was obtained, considering this value as a reference value for the population without COVID-19. In contrast, hospitalized patients with COVID-19 had an average of 22.6 µM nitrites (±19.1). This means that the patients presented 1.7 times more nitrites than the uninfected people, this difference being statistically significant ($p<0.0$). However, when analyzed by sex and age among the patients, no differences were found, suggesting that NO generation does not dependent from sex and age. Patients with comorbidities such as diabetes and arterial hypertension had slightly higher NO concentrations, although the association was not significant. **Conclusion:** These findings suggest that SARS-CoV-2 infection induces NO generation and increases serum nitrite concentration. It remains to be elucidated whether these NO-induced physiological changes and signaling pathways are involved in defense against the virus or contribute to the pathogenesis of CoVID-19.

FRMS04C

Characterization of the Antarctic Yeast *Rhodotorula mucilaginosa*: Lipid Droplets, Expression Profiles, and Fatty Acids Synthesis

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Rhodotorula mucilaginosa M94C9 is an oleaginous yeast isolated from Antarctic soil and considered polyextremotolerant that grows under cold, osmolar, salinity, and oligotrophic conditions. This yeast can accumulate large amounts of lipids in response to nutritional stress factors such as nitrogen limitation in the presence of glucose as a carbon source. Under these conditions, oleaginous yeasts can lead carbon flux to triacylglyceride biosynthesis through some enzymes such as acetyl-CoA carboxylase (Acc1) and fatty acid synthase 1 and 2 (Fas1 and Fas2) and diacylglycerol acyltransferase (Dga1). However, studies of lipid biosynthesis in *R. mucilaginosa* at the molecular level are scarce because most of the genes involved in lipogenesis have yet to be completely identified in this genome. Here we characterized the effect of stress by nitrogen limitation and analyzed the expression profile of genes involved in synthesizing neutral lipids. For this, growth, biomass, and lipid droplet (LDs) dynamics were first evaluated under different conditions of nitrogen availability. Likewise, the fatty acid profile was determined under nitrogen stress conditions, showing an accumulation of oleic acid. Growth and biomass production depend on nitrogen availability, and the most significant accumulation of LDs occurs under nitrogen starvation. In addition, an increase in the size of the LDs was found during the stationary phase in any medium, being more evident under nitrogen starvation. Subsequently, the ORFs of the RmACC1, RmFAS1, RmFAS2, and RmDGA1 genes were identified by in silico analysis, and their expression was measured by qPCR. During the exponential phase, RmACC1, RmFAS1, RmFAS2, and RmDGA1 are downregulated, while in the stationary phase, they are upregulated. The expression of these genes during the stationary phase allows the redirection of carbon metabolism toward the production of neutral lipids. This study includes the synthesis of neutral lipids in *R. mucilaginosa*, which could be helpful for the oleochemical industry.

This project is financed by Dirección General de Asuntos del Personal Académico (DGAPA-UNAM), PAPIIT IA208820 and IA204923.

FRPS02C

Antioxidant effect of pigments obtained from red corns

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Currently, several pigments used in food production have shown to have proinflammatory properties by promoting oxidative stress through the generation of reactive oxygen species (ROS)(Kwon et al., 2022); therefore, several studies have sought to obtain different pigments with anti-inflammatory and antioxidant properties that can be used as pigments. The objective of this work was to determine the phytochemical content and antioxidant potential of pigments from three different red colored corn (RA= high pigmentation, RM= medium pigmentation and RB= low pigmentation) obtained by hybrid microwave-ultrasound technique(Garcia-Ortiz et al., 2022). For which the hydrolyzable phenol content was quantified by the folin-ciocalteu technique, the condensed tannin content by HCl-butanol and the antioxidant potential by ABTS, FRAP and DPPH assays. The results indicated a higher content of hydrolyzable phenols and condensed tannins in the pigment obtained by RA with values of 0.33 mg GA Eq/g sample and 1.49 mg Cat Eq/g sample, as well as better results in the antioxidant potential assays with values of 15. 09, 13.01, and 13.17 mmol TE/100 g dry sample, respectively, showing values of 70.45% (DPPH), 100.8% (FRAP), and 104.75% (ABTS) higher than those of RB and 17.70% (DPPH), 11.52% (FRAP), and 14.41% (ABTS) higher than those of RM. This shows that red corn pigments with high phytochemical content are a viable option to produce pigments with better antioxidant properties.

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FRPS03C

Microwave-assisted extraction of phenolic compounds with potential antioxidant activity from grape residues

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ABSTRACT

Utilization of waste to produce by-products with added value is an alternative to reduce pollution [1]. Agro-industrial residues such as grapes present different compounds of interest to the food industry, highlighting phenolic compounds. Due to their chemical structure, these phenolic compounds present bioactivities and physicochemical properties with potential applications. Microwave-assisted extraction is an alternative method for obtaining active compounds, preserving their composition and bioactivity [2,3]. The aim of this work was to obtain phenolic compounds from grape residues assisted by microwaves to take advantage of their ability free radical scavenging activity. The raw material was dried and ground to a particle size <355 µm to extract phenolic compounds. Extraction factors studied were time (150 and 300 s) and power (400 and 800 W). The most efficient extraction conditions were 800 W-150 s and 400 W-300 s, with similar responses in the extracted compounds and bioactivity. Results obtained were: total phenol content 58.24 ± 5.96 and 57.86 ± 5.61 mg gallic acid equivalent (GAE)/g of dry sample, the content of hydroxycinnamic and hydroxybenzoic acids was 47.4 and 45.9 mg GAE/g of dry sample and the free radical scavenging activity by DPPH (2,2-diphenyl-1-picrylhydrazyl) was 85.9 and 84.0 %, respectively. In conclusion, microwave-assisted extraction promotes a high recovery of phenolic compounds in short periods, mainly hydroxycinnamic and hydroxybenzoic acids, with a high free radical scavenging activity.

Keywords: Phenolic compounds; Hydroxycinnamic; Hydroxybenzoic Microwave, Free radicals.

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FRPS04C

Effect of calcium nanoparticles on antioxidants compounds, enzymes, and capacity of minimally processed watermelon (*Citrullus lanatus*)

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Today´s lifestyle in big cities demands ready-to-eat products that also maintain nutritional quality, which is why minimally processed products (MPP) are marketed. These products are prepared through a series of steps such as washing, peeling, scraping, and packaging. However, the processing induces stress due to cutting, reducing its shelf-life. There are different postharvest technologies to extend the useful life of MPP which combine the application of natural additives and refrigeration. In recent decades, the use of nanoparticles (NP) has become a very promising technology in several fields, including MPP postharvest. On the other hand, watermelon is a very attractive fruit due to its refreshing flavor and texture, it is composed of a large amount of nutrients such as vitamins (A, B6, and C), lycopene, and other antioxidants. Regarding, the use of nanoparticles and their effect on nutritional quality and the response to postharvest stress, the information is still very scarce and even more so concerning MPP. The aim was to evaluate the effect of calcium oxide (CaO) and calcium hydroxide (CaOH) NP at 200 ppm on the phenolics compounds, antioxidant enzymes, and total antioxidant capacity of MPP watermelon fruits, using as controls treatment without NP and another with 1% $CaCl_2$. The results obtained showed a higher activity of catalase and peroxidases in the fruits treated with CaO-NP and CaOH-NP during the first 4 days of storage. As well as a higher antioxidant capacity in watermelon fruits treated with Ca-NP compared to controls. The content of total phenolic compounds was affected in the first 4 days by the action of cutting but recovered values similar to the initial day after day 8 of storage, and was maintained for the storage period in all treatments. Finally, the antioxidant capacity and the content of total phenolic compounds are higher in fruits treated with CaOH-NP at the end of storage compared to the other treatments. Therefore, it is concluded that CaOH-NP treatment can be an alternative treatment for preserving antioxidant compounds in minimally processed watermelon fruit for up to 12 days after cutting.

FRPS05C

Plasma Activated Water as a Postharvest Treatment for Gerbera (*Gerbera jamesonii* L.)

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The ornamental and cut flower trade have an impact on the world economy, being an increasingly profitable business in the agricultural sector. Among the main cut flowers are gerberas, which are appreciated for their variety of colors. However, the main problem is their short shelf life (3-5 days), which is why genetic improvement and treatments have been required to extend their shelf life. Among the emerging technologies, in recent years the use of atmospheric plasma and vacuum plasma has been highlighted for various agricultural and industrial applications, from materials to food; being reported as a successful treatment for conservation of different foods of vegetable origin. The application of atmospheric plasma in water modifies its chemical properties, generating new chemical groups based on reactive oxygen species (ROS) and reactive nitrogen species (RNS). Equilibrium ROS are necessary for the cell because of their role as messenger molecules in regulating a wide variety of cellular physiological processes. A low or absent antioxidant mechanism can cause an imbalance in ROS triggering irreversible damage to the cell. In this study, plasma-activated water (PAW) at 50, 60, 70, and 80 volts at 5, 15, and 30 min of activation was used as a vase solution to improve the shelf life of a cultivar of *Gerbera jamesonii* cv H. Bolus variety 'Amlet' where the response of chlorophylls a, b and total, anthocyanins, phenols, and antioxidant capacity was evaluated. Results indicate a longer shelf life of gerberas up to day 21 using PAW treated at 60 volts and 5 min. As a biochemical response for this treatment, chlorophylls a, b, and total chlorophylls were maintained concerning day zero flowers, while for other treatments with PAW, chlorophyll b increased. Anthocyanins decreased in response to the antioxidant effect against ROS and for PAW treated at 70 and 80 volts they increased significantly. Phenolic compounds and total antioxidant capacity evaluated in the pedicel suffered the greatest decrease when directly exposed to PAW, while their activity increased in the ligule. Therefore, it is concluded that treatment with PAW, mainly at 60 volts and 5 min, is an alternative treatment for postharvest preservation of gerbera flowers for up to 21 days.

FRPS06C

Oligosaccharins and Polyethylene Glycol on Chlorophylls, Phenolic Compounds, and Antioxidant Capacity of Candelilla (*Euphorbia antisyphilitica* Zucc.) *in vitro*

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Candelilla plants (*Euphorbia antisyphilitica* Zucc.) are of great ecological and economic importance, mainly for the state of Coahuila de Zaragoza, where most of the plant material of this species is concentrated. Natural wax is obtained from it, which is used in a large number of industries. The overexploitation of its wild populations has generated a great ecological impact, reducing it considerably in the last years, together with its low germination percentage and its slow development. Oligosaccharins (OGS) are plant elicitors, which generate an increase in growth and induce oxidative stress. Polyethylene glycol (PEG) generates a water deficit, which causes an increase in the antioxidant response and induces physiological changes related to an increase in biomass. Therefore, the objective was to evaluate the effect of OGS, PEG, and their combination in candelilla plants under *in vitro* conditions. For this purpose, the following treatments were applied in Murashige-Skoog (MS) culture medium: 1) control (MS medium), 2) 10 mg L^{-1} of OGS (derived from pectin, Pectimorf®), 3) 2 % (w/v) of PEG 6000, and 4) 10 mg L^{-1} of OGS + 2 % (w/v) of PEG 6000. The content of chlorophylls (a, b, and total), carotenes, phenolic compounds, and antioxidant capacity were evaluated. Results showed that candelilla seedlings developed with OGS + PEG treatment had an increase in chlorophylls a, b, and total chlorophylls, and phenolic compounds. Candelilla seedlings developed with the 2 % PEG treatment showed an increase in carotene content and antioxidant capacity. Therefore, it is concluded that the treatments with PEG and the combination (OGS + PEG) showed a greater induction to oxidative stress, increasing the content of the main photosynthetic pigments, antioxidant compounds, and total antioxidant capacity, which could modify the development of candelilla plants under *in vitro* conditions.

Keywords: *Euphorbia antisyphilitica* Zucc, candelilla, oligosaccharins, polyethylene glycol, *in vitro*, water deficit.

FRPS07C

Effect of Sodium Dodecyl Sulphate on the production of Thiobarbituric Acid Reactive Substances and photosynthetic pigments in the alga *Cladophora sp* and the macrophyte *Egeria densa*

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Surfactants represent a pollutant that is discharged in large volumes in different aquatic bodies, with inhibitory effects on the growth, reproduction, and physiological functions of organisms by altering membrane permeability, enzymatic activity, and tissue structure. Even though several toxicological studies with surfactants have been carried out, the evaluation of oxidative damage in plants and algae, as well as the role of photosynthetic pigments as antioxidants are still limited. Therefore, in order to determine the effects of Sodium Dodecyl Sulphate (SDS) on the production of Thiobarbituric Acid Reactive Substances (TBARS) and photosynthetic pigments, bioassays were performed in two aquatic species: *Egeria densa* and *Cladophora sp*. The study was based on the guidelines 221 and 238 of the Organization for Economic Cooperation and Development (OECD), where at least six concentrations of SDS were used (0, 0.1, 1, 5, 10, 50, 100 mg/l) and three replicates per repetition in a completely randomized design. The bioassays were carried out in reconstituted water with a hardness of 160 mg of CaCO3, pH between 7.6 and 8.2, temperature of 25 ± 1 °C, illumination of 4000 lx and a cycle of 16:8h of light and darkness. At the end of the exposure period, growth inhibition and the content of chlorophyll A, chlorophyll B, carotenes and TBARS were evaluated in both species. The results revealed a decrease in the growth of both organisms as the concentration of SDS increased. *Cladophora sp* showed a significant decrease in pigment production when it was exposed to a concentration of 100 mg/l, while TBARS production increased significantly from a concentration ≥ 5 mg/l. On the other hand, *E. densa*, chlorophylls A and B, and carotenes showed a similar behavior to hormesis, with an increase of 0.5 mg/l and a significant decrease at concentrations ≤ 50 mg/l. Regarding the TBARS production, the macrophyte showed a significant increase in concentrations higher than 0.5 mg/l. Both species exhibited an imbalance in the antioxidant and oxidative processes in their plant cells, reflected in the increase in TBARS. However, the plant proved to be more tolerant to the contaminant than the algae. Also, exposure to SDS caused physiological changes in both species, with a decrease in growth and an increase in TBARS production. This study highlights the report of oxidative damage in a filamentous alga exposed to surfactants.

Keynote Lectures

KEYNOTE LECTURE 1

Dr. Francisco R. M. Laurindo

Associate Professor and Director, Vascular Biology Laboratory
University of São Paulo School of Medicine, São Paulo, Brazil
President of Society for Redox Biology and Medicine

Dr. Laurindo is Associate Professor at the University of São Paulo School of Medicine and Head of the Vascular Biology Laboratory. He has had a long-standing interest to investigate redox signaling in vascular cells and tissues in physiology and disease, with particular emphasis on mechanisms of oxidant generation. More recently, his group has focused on the thiol redox effects of endoplasmic reticulum chaperones from the protein disulfide isomerase family. He has authored or co-authored > 180 publications and supervise among 50 PhD students and post-doctoral fellows. He serves at the Editorial Board of FRBM, Clinical Science and Circulation Research and is the current President of the Society of Redox Biology and Medicine. He has served at the Board of Directors of the Brazilian Academy of Sciences from 2016 to 2022 and as an Advisory Board member for Fapesp Research Agency since 2008.

"Thiol Redox Codes of Cellular Signaling"

Dr. Francisco R. M. Laurindo

Abstract

Many signaling proteins are regulated via cysteine thiols (RSH) and their several oxoforms (RSSR'; RSOH; RSO2H…etc). Thiol code integration is provided by so-called "professional" thiol redox proteins. E.g., abundant peroxiredoxins react fast with peroxides (107 to 108 M-1.s-1) and qualify as redox sensors and disulfide relay-proteins. We have focused on the family of Protein Disulfide Isomerases (PDIs), composed by >21 proteins, which are dithiol oxidoreductase chaperones from thioredoxin superfamily. As redox protein folding catalysts from the endoplasmic reticulum (ER), their roles in ER-related proteostasis are well-studied. PDIA1, the family prototype, exerts thiol oxidation, reduction and isomerization, plus chaperone effects. PDIs are not poised for mass-effect redox sensor roles such as peroxiredoxins, due to kinetic constraints (rate constant of reaction with peroxide of 17 M-1.s-1). Meanwhile, substantial evidence indicates that PDIA1 regulates thiol-disulfide switches in specific cell locations at the cell surface and cytoplasmic structures, with implications in vascular (patho)physiology. Subcellular PDIA1 translocation routes remain unclear and seem Golgi-independent. Our work has shown that PDIA1 is required for agonist-triggered superoxide production via Nox NADPH oxidase activation and vascular smooth muscle cell (VSMC) migration. PDIA1 is also involved in VSMC phenotype regulation, with PDI overexpression in VSMC and in transgenic mouse model causing upregulation of differentiation markers and Nox4 NADPH oxidase. In parallel, we showed that PDIA1 is involved in vascular remodeling and required for fine-tuning cytoskeletal adjustments in directional VSMC migration and adhesion force distribution, minimizing the noise of polarized cytoskeletal organization in response to a variety of mechanostimuli. These data allow the proposal of a redox/biomechanical paradigm for vascular

remodeling. Such effects associate with redox modulation of integrins and RhoGTPases; physical association between PDIA1 and the RhoGTPase regulator RhoGDIalpha was reported by us. Moreover, the PDI family genes display a remarkable microsyntenic arrangement with RhoGDI genes, conserved through >800 million-years of evolution. PDIA1 is implicated in diseases. In human plasma, lower PDIA1 levels reveal proteomic signatures involved in immune-inflammatory and thrombogenic phenotypic endothelial markers. We show that PDIA1 is a novel protective factor against aortic dissection. In turn, cell-surface PDIA1 is required for enhanced platelet adhesion to hyperglycemic endothelial cells. Thus, PDIA1 and its interactors establish a unique redox-sensitive regulatory signaling code. The “all-in-one” organization of its peculiar redox/chaperone properties provide PDIs with precision, versatility and focused effects in redox signaling, making them promising therapeutic targets.

KEYNOTE LECTURE 2

Dra. Carola Neumann

University of Pittsburgh, Pittsburgh, PA. EUA.

Vicepresident of the Society for Redox Biology And Medicine (SfRBM).

Dr. Neumann's research focuses on the role oxidative stress, the imbalance between free radicals and antioxidants in the body, plays in breast cancer. Currently, her laboratory is focused on moving a new drug for triple negative breast into pre-clinical trials. Triple negative breast cancer is an extremely aggressive form of the disease, and for the cancer cell to stay alive, it needs to continuously repair its DNA. Dr. Neumann's lab has identified this DNA repair dependency as an opportunity for targeted treatment. With the drug developed and the drug target established, researchers now have a therapy that only effects cancer cells.

In addition, Dr. Neumann's laboratory investigates the role oxidative stress plays on the tumor microenvironment on cancer-associated fibroblasts. The majority of a breast tumor is made up of fibroblasts, but they can be difficult to target. Researchers in Dr. Neumann's laboratory are exploring what turns a normal mammary fibroblast cancerous, and what role oxidative stress plays in that process.

"Redox signaling in breast cancer: from basic science to drug discovery"

Dra. Carola Neumann

Lisa Hong, Dennis C. Braden, Yaoning Zhao, John J. Skoko, David Gaboriau, Myriam Attar, Fei Chang, Steven R. Woodcock, Crystall Uvalle, Allison Casey, Katherine Wood, Candice E. Paulsen, Hongqiang Ma, Yang Liu, Hanzhi Wu, Cristina M. Furdui, Yefim Manevich, Ciaran G. Morrison, Erika T. Brown, Daniel Normolle, Maria Spies, Michael Ashley Spies, Kate Carroll, Sonia R. Salvatore, Alparslan Asan, Trey Harkness, Adeola Fagunloye, Mortezaali Razzaghi, Adam Straub, Francisco Schopfer, Bruce A. Freeman, and Carola A Neumann

Abstract

RAD51 is a critical recombinase that functions in concert with auxiliary mediator proteins to direct the homologous recombination (HR) DNA repair pathway. We show that Cys319 RAD51 possesses nucleophilic characteristics and is important for irradiation-induced RAD51 foci formation and resistance to inhibitors of poly (ADP-ribose) polymerase (PARP). We have previously identified that cysteine (Cys) oxidation of proteins can be important for activity and modulated via binding to peroxiredoxin 1 (PRDX1). PRDX1 reduces peroxides and coordinates the signaling actions of protein binding partners. Loss of PRDX1 inhibits irradiation-induced RAD51 foci formation and represses HR DNA repair. PRDX1-deficient human breast cancer cells and mouse embryonic fibroblasts display disrupted RAD51 foci formation and decreased HR, resulting in increased DNA damage and sensitization of cells to irradiation. Following irradiation cells deficient in PRDX1 had increased

incorporation of the sulfenylation probe DAz-2 in RAD51 Cys319, a functionally-significant, thiol that PRDX1 is critical for maintaining in a reduced state. Molecular dynamics (MD) simulations of dT-DNA bound to a non-oxidized RAD51 protein showed tight binding throughout the simulation, while dT-DNA dissociated from an oxidized Cys319 RAD51 filament. These novel data establish RAD51 Cys319 as a functionally-significant site for the redox regulation of HR and cellular responses to IR. Based on these findings we have initiated a library screen to target RAD51 recognized as a sought-after drug target in cancer therapy. Screening a library of 55 compounds, we identified the nitroalkene [(E) 8-nitro-nonadec-7-enoic acid (CP-8)] and Michael Acceptor, to inhibit RAD51 function. CP-8 kills cancer cells, synergizes with three different inhibitors of the poly ADP-ribose polymerase (PARP) and □-IR. In vivo, CP-8 showed promising anticancer activities alone and in combination with the PARP inhibitor talazoparib in an HR-proficient cancer mouse model. As preliminary preclinical toxicology analysis also suggests CP-8 as safe, our data endorse CP-8 as a novel anticancer molecule for treating cancers sensitive to homologous recombination-mediated DNA repair inhibitors.

KEYNOTE LECTURE 3

Dr. Raúl Rodríguez Herrera

Facultad de Ciencias Químicas, Universidad Autónoma de Coahuila (UadeC),Coahuila, México.

Dr. Rodríguez Herrera is a professor at the Universidad Autónoma de Coahuila. He received a degree in Agricultural Engineering and a Master's in Plant Breeding from UAAAN, a Ph.D. in Plant Breeding from Texas A&M-USA, and a postdoctoral degree in Molecular Genetics from Texas A&M-USDA. He was involved in establishing the Master's programs in Chemical Sciences and Food Science and Technology and the Ph.D. programs in Biotechnology and Food Science and Technology at UAdeC. He collaborated on over 25 patents and mentored over 250 undergraduate, Master's, and doctoral students. He has published over 345 articles in indexed journals, 61 outreach articles, 120 book chapters, and seven books. He has received awards such as Level 3 Researcher from SNI, Best Research Award from the PCCMCA Meeting in San José, Costa Rica, 1988 and San Salvador, El Salvador, 1990. National Award in Food Science and Technology 2003, National AgroBIO Award 2005, State Award for Best Completed Project COECYT 2005, Miguel Ramos Arizpe and Mariano Narváez Medals-UAdeC, and UAdeC Researcher of the Year (Chemistry-Biology) 2021 and Host of Honour, Kannur University India. He has reviewed over 25 scientific journals in JCR and institutions such as: Southern Illinois University-USA, Texas A&M University-CONACYT, UC-Mexus-CONACyT, Ministry

of Science, Technology, and Productive Innovation-Argentina, Universidad Técnica Estatal de Quevedo-Ecuador, Universities of Cauca and Valle in Colombia, Pondicherry and Kerala Universities-India, INRA-France, CONACyT, SAGARPA, CONAFOR, Universidad Autónoma de Aguascalientes, CIQA, and UAAAN. He has also evaluated awards such as: "National Award in Sciences 2019", "Potosino Award in Science, Technology and Innovation 2019 and 2020", and "Eustaquio Buelna Award-Sinaloa 2019".

"Antioxidant capacity of phytochemicals from Mexican Semidesert plants and their biological activities"

Dr. Raúl Rodríguez Herrera

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Abstract

Plants from the semi-desert have been adapted to survive long periods of drought, high temperatures during the day and low temperatures at night, and high osmotic pressures. Due to these conditions, these plants have developed a very efficient metabolism, which allows them to defend themselves against pests and diseases, withstand high temperatures, and combat reactive oxygen species (ROS), which include oxygen ions, free radicals, and peroxides, which can be organic or inorganic. The flora in the semi-desert is very different in morphology and in its applications. There are plants that are used mainly for their leaves, such as creosote bush (Larrea

tridentata), and tarbush (Fourencia cernua), their flowers, for example palms of different species of the Yucca genus, and others for their fruits, for example cacti species such as nopal [Opuntia ficus-indica (L.) Mill.], and Xoconostle (Cylindropuntia imbricata) all these plants have antioxidants, among which polyphenols, ascorbic acid and pigments stand out. Polyphenols are compounds of plant origin that are synthesized during growth, such is the case of carotenoids, tocopherols, flavonoids, tannins, sterols, etc., these chemical compounds protect plants against UV light stress, infection, injury, attack of pests, they also act as a photochemical. The insoluble phenols are in the cell walls because they provide mechanical resistance, protecting the integrity of the tissues, while the soluble polyphenols are in the vacuoles of the cells. Among the many positive effects that polyphenols have on health, antioxidant activity, reduction of hypertension, atherosclerosis, thrombosis, and inflammatory processes in relation to cardiovascular diseases, anticancer, antidiabetic, antiaging, antibacterial and antiviral stand out. This conference will provide information on obtaining polyphenols from various semi-desert plants (creosote bush, tarbush, xoconoxtle, palm, etc.), their antioxidant activity and their applications as anticancer, anti-inflammatory and antimicrobial agents.

KEYNOTE LECTURE 4

Dra. Annia Galano Jiménez.

Depto. Química, Universidad Autónoma Metroplitana, Unidad Iztapalapa, México

Dr. Galano is currently a Full-Time Tenured Professor C at Universidad Autónoma Metropolitana, Iztapalapa. Bachelor's Degree: Universidad de la Habana 1990, Ph.D.: Universidad de la Habana 2000. SNI level 3, since 2014. Indexed publications: over 200. Citations to publications: over 10,000. H-Index: 58. Highly Cited Researcher 2021 and 2022, according to Clarivate. She served on the Evaluating Commission of SNI, Area 2, in 2014, 2015, 2016, 2020, and 2021 and was the commission's president in 2021. She also joined the Reviewing Commission SNI, Area 2, in 2017. Reviewer for more than 50 indexed journals, including JACS and Angewandte Chemie. She is a member of the editorial boards of Computational and Theoretical Chemistry, Theoretical Chemistry Accounts, and Melatonin Research. She has been a member of the editorial boards of the Journal of Pineal Research and the Journal of Mexican Chemical Society. Elected member of the WATOC Board since 2017. (WATOC = World Association of Theoretical and Computational Chemists) and Academic Secretary of the Sociedad Química de México. Theses supervised: 10 Ph. Ds, four master's, and four bachelor's degrees.

"Design of multifunctional antioxidants with potential application in Parkinson's and Alzheimer's diseases"

Dra. Annia Galano Jiménez

Abstract

The CADMA-Chem computational protocol is introduced, designed for the search of multifunctional antioxidants with potential application as neuroprotectors. To illustrate the details of this protocol, the melatonin derivative dM38 is used. Currently, CADMA-Chem is the only computational protocol that considers chemical processes in addition to receptor-ligand affinities. It simultaneously analyzes physicochemical properties related to the behavior of oral drugs, toxicity, manufacturability, versatile antioxidant capacity (clearance of free radicals, redox metal chelation, repair of damaged biological targets), and polygenic neuroprotection. It is hoped that it will help speed up the discovery of drugs capable of preventing, or slowing the progression of, multifactorial diseases and reduce research costs.

KEYNOTE LECTURE 5

Dr. Carlos Alonso-Álvarez

Museo Nacional de Ciencias Naturales de Madrid (MNCN), Consejo Superior de Investigaciones Científicas (CSIC), España.

He conducted his doctoral thesis on the eco-physiology of the yellow-legged gull, focusing then on blood biochemistry and steroid hormones to address issues related to adaptation to hunger, reproductive behavior, and, ultimately, the application of this knowledge to wildlife conservation. After presenting this thesis, he carried out postdoctoral stays at the Estación Biología de Doñana (Seville, Spain) and the Université Pierre et Marie Curie in Paris (France). In the latter, he undertook a project examining the role of oxidative stress in the evolutionary trade-offs of life history, particularly the one established between the investment of resources in reproduction or the maintenance of the organism (longevity), using cage birds as a model. Another significant line of research is focused on studying the most important pigments in vertebrates, melanins, and carotenoids, and their relationship with the antioxidant machinery and the production of colored traits involved in sexual selection. The aim is to explain how this machinery can make the trait a reliable signal of the individual's quality, promoting its evolution.

"The oxidative cost of reproduction as a mechanism implicated in evolutionary theories of aging: Is there solid evidence?"

Dr. Carlos Alonso-Álvarez

Abstract

Nearly seventy years ago, it was suggested that free radicals can cause damage that accumulates over a lifetime, leading to the overall loss of function we know as aging (free radical theory). Although widely criticized, this theory remains a touchstone in gerontological research. On the other hand, evolutionary biology has sought to explain aging from a natural selection standpoint. According to this perspective, the selection of traits that favor homeostasis weakens over time because the likelihood of reaching old age decreases by sheer chance due to accidents or predation. In this context, genes with harmful mutations only expressed in old age are not purged from the population, accumulating in the gene pool and promoting aging (mutation accumulation theory). It has also been proposed that genes favoring early reproduction might promote aging-related diseases (antagonistic pleiotropy theory). From a phenotypic viewpoint, investing physiological resources in early reproduction would detract from the maintenance of the organism, reducing longevity. Investing in somatic maintenance at an advanced age would be irrelevant if there is no possibility of reproduction: the soma becomes disposable (disposable soma theory). At least the last two theories are based on the trade-off between investing resources in reproduction versus longevity. Such a trade-off was traditionally interpreted in terms of scarce energy resources. However, 20 years ago, some experimental results suggested that reproductive investment might involve antioxidant resources or directly imply an oxidative cost. If we accept

the free radical theory, such a cost would promote aging. Since the hypothesis of the oxidative cost of reproduction was formulated, many studies have attempted to test it, obtaining inconsistent results. Different explanations have been argued for this lack of consistency. In this talk, I will review the state of the matter, addressing the ideas that try to understand why this oxidative cost remains elusive and what consequences of employing evolutionary logic can be drawn for the general understanding of the causes of aging.

KEYNOTE LECTURE 6

Dra. Deepa Sathyaseelan

University of Oklahoma Health Sciences Center, Oklahoma City, Oklahoma, EUA.

Dr. Deepa Sathyaseelan holds a Ph.D. in Biochemistry from the University of kerala, India, and has pursued postdoctoral research at prestigious institutions such as the University of Cambridge, Kobe Pharmaceutical University in Japan, and the University of Texas Health Sciences Center. Dr. Sathyaseelan currently serves as an Assistant Professor at the University of Oklahoma Helath Sciences center, contributing significantly to the understanding of aging-related processes and their impact on health. Her research is supported by grants form the NIH, and she is an active member of various scientific organizations. Dr. Sathyaseelans´s work has been published extensively in reputable journals, and she continues to make notable contyributions to the field of geroscience, with a particular focus on necroptosis, chronic inflammation, and their implications in aging and age-associated diseases.

"The Role of Necroptosis and Oxidative Stress in Aging and Age-associated diseases"

Deepa Sathyaseelan, Ph.D.

University of Oklahoma Health Sciences Center, OK, USA.

Abstract

Age-associated increase in low-level chronic inflammation, termed sterile inflammation or 'inflammaging' is an important risk factor for most age-related diseases (e.g. cancer, type 2 diabetes, cardiovascular diseases, cancer, neurodegenerative diseases etc.). Although the exact cause of inflammaging is not known, age-associated accumulation of damage associated molecular patterns (DAMPs) is reported to be a strong inducer of inflammation. Necroptosis is a non-apoptotic form of programmed cell death pathway that plays a major role in inflammation through generation of DAMPs. We demonstrated for the first time that necroptosis increases with age, and dietary restriction reduces age-associated increase in necroptosis and inflammation[1]. We also found that necroptosis and inflammation are increased in a mouse model of accelerated aging and increased oxidative stress: mice deficient in Cu/Zn-superoxide dismutase (*Sod1*$^{-/-}$ mice) that exhibit increased inflammation as indicated by high levels of circulating pro-inflammatory cytokines. Inhibition of necroptosis using necrostatin-1s (pharmacological inhibitor of necroptosis pathway) reduced necroptosis and transcript levels of pro-inflammatory cytokines and M1 macrophage/microglia markers in the tissues of *Sod1*$^{-/-}$ mice as well as old wild type mice[2,3,4]. Similar results were obtained when necroptosis was blocked genetically in old wild type mice. Blocking necroptosis improved chronic liver disease conditions associated with aging: non-alcoholic steatohepatitis and hepatocellular carcinoma, which are driven

primarily by inflammation[4,5]. We also identified a novel crosstalk between necroptosis and cellular senescence, a key pathway involved in inflammaging. Thus, our study identifies necroptosis as a major contributor to age-associated inflammation and diseases.

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Symposia
and
Round Table

SYMPOSIUM 1

"Diseases associated with oxidative stress"

Dr. Jaime Arellanes Robledo

Laboratorio Enfermedades Hepáticas Crónicas, Instituto Nacional de Medicina Genómica (INMEGEN), México.

Dr. Arellanes obtained the degree of Chemical Biologist Parasitologist from the Faculty of Chemical Biological Sciences of the Universidad Autónoma de Guerrero (UAGro) in 1997. Between 2002 and 2009, he pursued his master's and doctoral studies in Cellular Biology at the Centro de Investigación y de Estudios Avanzados of the Instituto Politécnico Nacional under the guidance of the Emeritus Researcher Dr. Saúl Villa Treviño. He conducted studies on the chemoprevention of experimental hepatocarcinogenesis through transcriptomic analyses carried out during a doctoral stay at the Genopole-Institut National des Sciences Appliquées (INSA) in Toulouse, France. He is a member of the National System of Researchers Level II. Since 2015, Dr. Arellanes has led the research line "Redox regulation in chronic liver diseases and cellular senescence" and serves at the Instituto Nacional de Medicina Genómica (INMEGEN) as an associated researcher for INMEGEN-CONACYT in the "Researchers for Mexico" program.

“Oxidative stress and redox regulation, early players in chronic liver diseases”

Dr. Jaime Arellanes Robledo

Laboratorio de Enfermedades Hepáticas Crónicas. Instituto Nacional de Medicina Genómica. Periférico Sur No. 4809, Col. Arenal Tepepan, Delegación Tlalpan, Ciudad de México. C.P. 14610. Teléfono: +52 (55) 5350-1900, ext. 1218.

Abstract

Chronic liver diseases progress through functional and structural alterations of cells, which manifest as well-characterized and chronologically identified pathological processes such as steatosis, fibrosis, steatohepatitis and cirrhosis, that may eventually lead to liver cancer. These alterations are mainly promoted in a microenvironment of exacerbated oxidative stress induced by different etiological agents including chronic alcohol consumption, among others. Since alcohol is mainly metabolized by the liver, this organ is the most exposed to the adverse effects of this hepatotoxic. Alcohol metabolism is a free radical generating process par excellence, which reacts with macromolecules, compromises cellular functioning, causes tissue injury and alters the liver redox balance, the latter, interferes with redox-sensitive molecular interactions modifying the activity of signaling pathways downstream. However, little is known about how the alteration of redox-sensitive molecular interactions induced by early oxidative stress contributes to the establishment of chronic liver diseases. In this talk, we will discuss the impact of oxidative stress on these interactions during the early progression of chronic liver diseases.

Dra. Liliana Quintanar Vera

Departamento de Química, Centro de Investigación y de Estudios Avanzados(CINVESTAV), México.

Dr. Quintanar Vera completed her undergraduate studies in chemistry at the Faculty of Chemistry of UNAM (1993-1998), earning honors and the Gabino Barreda medal. Since 2005, she has been a researcher in the Department of Chemistry at the Centro de Investigación y de Estudios Avanzados (Cinvestav) of the IPN, where she leads a research group focused on the spectroscopic study of metal-protein interactions that are relevant in the development of neurodegenerative diseases (Alzheimer's, Parkinson's, and prion diseases) and degenerative diseases (diabetes and cataracts). Her work has been published in the most distinguished journals in the field of inorganic chemistry, and she has been invited to give lectures at the most important international conferences in bio-inorganic chemistry. She is a member of the Editorial Advisory Board of the Journal of Biological Inorganic Chemistry (JBIC) and ChemBioChem and the editor of Avance y Perspectiva, the science outreach journal of Cinvestav. She is a National System of Researchers (level II) member. She has been involved in organizing several conferences and workshops, both national and international, promoting academic exchange between Mexico, the United States, and Latin America.

"Copper redox chemistry in neurodegenerative diseases"

Dra. Liliana Quintanar Vera

Department of Chemistry, and Center for Research in Aging, Cinvestav (Center for Research and Advanced Studies), Mexico City, México lilianaq@cinvestav.mx

Abstract

Copper is essential for life as cofactor of a wide range of metalloproteins, yet it has also been implicated in several degenerative diseases associated to the deposition of protein aggregates. Examples include Alzheimer´s, Parkinson´s, prion diseases, diabetes type 2, and cataracts, where the implicated proteins are the b-amyloid peptide (Ab), a-synuclein, prion protein (PrP), islet amyloid polypeptide (IAPP) or amylin peptide, and b/g-crystallin proteins, respectively. Our research group studies the interaction of copper ions with some of these proteins, using a wide range of spectroscopic tools to understand their coordination chemistry, and the impact of the metal ion in protein folding, stability and aggregation properties. In some of these copper-protein interactions, it becomes evident the role of the copper redox chemistry in protein oxidation and aggregation processes.

In this presentation, the Cu binding properties of two proteins at the synapse, PrP and Ab, and their competition for the metal ion will be discussed.[1] This competition gains relevance as Cu-PrP interactions play a key role in neuroprotective mechanisms at the synapse that are likely affected in Alzheimer´s disease.[2] On the second part of this presentation, the mechanisms of copper-induced aggregation of human lens b/g-crystallins, associated to cataract disease, will be briefly discussed.[3]

Copper ions activate different site-specific mechanisms, including: protein unfolding, metal bridging, disulfide bridges, and copper redox processes that lead to formation of free radical species. The role of Cys residues in this interesting redox chemistry will be discussed.[4] Overall, understanding how copper ions impact protein stability and aggregation provides further insights into the bioinorganic chemistry of copper in these degenerative diseases.

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Dr. Oliver Christophe Barbier

Laboratorio de Toxicología Renal, Centro de Investigación y de Estudios Avanzados(CINVESTAV), México.

Dr. Oliver Christophe Barbier is a member of the SNI level 2 and a member of the Sociedad Mexicana de Toxicología (MX), among others. He studied at the Universidad de Niza (France), obtaining his master's and doctoral degrees in

Sciences with honors and special commendations from the jury (2004). He undertook a postdoctoral stay at the Department of Physiology of Cinvestav (2005-2006). He has also been a visiting professor in the Renal Division of Brigham and Women's Hospital and the School of Medicine at Harvard University (2016-2017). In recent years, Dr. Barbier and his group have focused on the early diagnosis of kidney disease and its association with environmental risk factors. He acts as the leader and co-leader of research projects funded by CONACyT (Basic Science, Pronaii), the IPN, the Secretaria de Ciencia Tecnología e Innovación de la CDMX, the European Union, and the Fundación Gonzalo Rio Arronte. In terms of his scientific, academic, and educational production, he has published five book chapters and 48 articles in high-impact international journals, cited 2,850 times; he is the thesis advisor for doctoral (9), master's (19), and undergraduate (9) programs.

"Role of oxidative stress from exposure to xenobiotics in the development of nephropathies"

Dr. Oliver Christophe Barbier

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Abstract

The role of oxidative stress and environmental pollutants in the development of kidney diseases is an area of growing interest in medical research. Oxidative stress

is an imbalance between the production of reactive oxygen species and the antioxidant capacity of the organism, which can damage many organs, including kidney cells, by triggering various adaptive/repair responses or cell damage, including inflammatory processes. Environmental pollutants, such as metals, some minerals, industrial chemicals, and particulate matter in the air, can enter into contact with the renal system through ingestion, inhalation, or dermal absorption, contributing to oxidative stress generation and renal dysfunction.

For example, chronic inflammation induced by oxidative stress and environmental pollutants can lead to endothelial dysfunction in renal blood vessels, disrupting blood flow and regulating blood pressure. This phenomenon can aggravate hypertension, a key risk factor in kidney diseases. In addition, damage at the microvascular level can affect glomerular filtration and tubular function, compromising renal health.

During the conference, the topics of the role of oxidative stress generation by exposure to environmental pollutants, alterations of their regulatory mechanisms (mitochondrial damage, generation of reactive oxygen species, antioxidant system, expression of miRNAs), and their modes of action in the development of acute and chronic nephropathies were be addressed.

SYMPOSIUM 2

"Effect of oxidative stress on fungi and plants"

Dr. Óscar Flores Herrera

Facultad de Medicina, Universidad Nacional Autónoma de México (UNAM), México.

Dr. Flores Herrera is a biologist from the Facultad de Ciencias of UNAM, with a master's and doctoral degree in Basic Biomedical Research from UNAM. He is a Full Professor A and head of laboratory 36/4 in the Department of Biochemistry, Facultad de Medicina, UNAM. His research focus is on mitochondrial bioenergetics. He described the transport of calcium and the mechanisms regulating the mitochondrial synthesis of progesterone in the human placenta. He is currently studying the respirasome and the dimer of the F1FO-ATP synthase (V2) from the basidiomycete Ustilago maydis, a fungus gastronomically known as huitlacoche, as well as the ascomycetes Yarrowia lipolytica, Saccharomyces cerevisiae, and Debaryomyces hansenii, and the heart, liver, and kidney of the Wistar rat. He collaborates with Dr. Michael Feldbrügge's group from Heinrich-Heine University, Düsseldorf, Germany, in constructing mutants in U. maydis to study the role of the F1FO-ATP synthase dimers in this basidiomycete's metabolism; and with Dr. James A. Letts' group from the University of California, Davis, USA, with whom he is defining the three-

dimensional structure of the respirasome and the dimer of complex V of U. maydis (MEXUS - U. California Program). In 2017, he was awarded the "Dr. José Laguna" medal by the Bioenergetics and Biomembranes Branch of the Sociedad Mexicana de Bioquímica. His work has been published in 46 international articles, with just over 500 citations. He is SNI-II, and as an undergraduate, master's, and doctoral tutor, he playfully "torments" his cherished students.

"The role of the respirasome in the control of mitochondrial production of ROS in Ustilago maydis"

Oscar Flores-Herrera

Oscar Flores-Herrera, Jaime A. de Lira-Sánchez, Mercedes Esparza-Perusquía, Giovanni García-Cruz, S. Paola López-Cervantes, Federico Martínez, Juan P. Pardo. Departamento de Bioquímica, Facultad de Medicina, Universidad Nacional Autónoma de México.*Corresponding author. oflores@bq.unam.mx*

Abstract

The F1FO-ATP synthase uses the energy stored in the electrochemical proton gradient to synthesize ATP. This complex is found in the inner mitochondrial membrane as a monomer and dimer. The dimer is essential for cristae folding and the monomer-monomer interface is constituted by subunits a, i/j, e, g, and k. Deletion of the ATP20 gene, encoding the g subunit, in Ustilago maydis, a strict respiratory organism, did not affect cell growth or glucose consumption, but biomass production

was lower in the mutant strain (g□ strain). Ultrastructure observations showed that mitochondrial size and cristae shape were similar in wild-type and g□ strains.

Although the mitochondrial membrane potential in both strains had a similar magnitude, the ATP synthesis was 20% lower in the g□ strain. The mutant strain expressed the alternative oxidase in the early stages of growth (exponential phase), as a response to increasing mitochondrial ROS production. Several enzymes have been proposed as a hot point for ROS production, particularly the complexes of the electron transport chain. Recently, stable associations between respiratory complexes have been described, suggesting a role in the control of ROS production. The respirasome is constituted by complexes I, III2, and IV, and we have determined that it has a higher NADH:DBQ oxidoreductase activity coupled to a lower ROS production than the free complex I. To verify the role of respiratory complexes in ROS production, the respirasome, and free-complex I were isolated and analyzed. The isolated respirasome or free-complex I were incubated in the presence of seven of the most toxicologically relevant heavy metals to generate oxidative stress. We showed a deactivation of the respirasomal-complex I by heavy metals without increasing ROS production. In sharp contrast, the free-complex I was more resistant to heavy metals but was 30 times more ROS-producing. These results underly the preventive role of the respirasome in mitochondrial electron leak and ROS production and recall its disassembled in some pathologies which involve mitochondrial damage and oxidative stress.

Acknowledgment: *This work was supported by Dirección General de Asuntos del Personal Académico (DGAPA, IN206320 and IN201923) from Universidad Nacional Autónoma de México (UNAM), Consejo Nacional de Ciencia y Tecnología (CONACyT, 87160), and UC MEXUS-CONACYT collaborative grant (CN-20-327).*

De Lira-Sánchez and García-Cruz are Ph.D. students from Programa de Doctorado en Ciencias Biomédicas, Universidad Nacional Autónoma de México (UNAM) and received fellowship (666472, DL-S; 1103534, G-C) from CONACyT. López-Cervantes is a Ph.D. student in the Programa de Doctorado en Biología Experimental from Universidad Autónoma Metropolitana-Iztapalapa and supported by CONACyT through a doctoral scholarship (868620).

Dr. Darío Rafael Gómez Linton

Universidad Autónoma Metropolitana, Iztapalapa, CDMX México.

Dr. Gómez-Linton, a Ph.D. in Biotechnology, completed his professional education at the Universidad Autónoma Metropolitana, Iztapalapa campus, in the field of bioactive compounds from plants and their use in aging studies, particularly using the C. elegans model. He has published and presented several works on these topics at both national and international academic venues. Currently, he is an associate professor at UAM-I, where he collaborates with the Physiology, Biochemistry, and Molecular Biology of Plants group.

"Caenorhabditis elegans: A model for studying the antioxidant activity of native tomatoes"

Darío R. Gómez-Linton; Laura J. Pérez-Flores

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Abstract

The nematode Caenorhabditis elegans is one of the most widely used animal models in biological research. Its applications include the study of genetics, cellular and molecular biology, reproduction, neurology, toxicology, pharmacology, host-pathogen interactions, oxidative stress, degenerative diseases, and aging. Some of their advantages are: fully sequenced genome, with nearly 80% of human genes having homologs in the worm; evolutionarily-conserved signaling pathways; ability to generate mutant individuals; small size; rapid propagation; cost-effective maintenance; easy handling; and a variety of standardized methodologies.

Tomato fruits are highly relevant sources of carotenoids and vitamin E. Both groups of compounds are known for their antioxidant properties. While commercial varieties of tomatoes are well characterized, studies highlighting relevant features of native genotypes such as color, flavor or nutritional and functional properties are highly appreciated. These genotypes are significantly relevant in Mexico, since it is a domestication site for this species.

To evaluate the antioxidant potential of the lipophilic fraction of tomato fruits, optimized organic extracts were obtained and tested using C. elegans. The extracts increased the nematodes' resistance to stress induced by H_2O_2, thermal shock, and

UV radiation. The involvement of the insulin signaling pathway and the stress response transcription factors DAF-16, SKN-1, and HSF-1 were studied using mutant strains. It was found that, while carotenoids and vitamin E can neutralize and scavenge free radicals in vitro, part of their in vivo activity is mediated by stress response transcription factors since the protective effect was lost in some mutant individuals. These results demonstrate the ability of native tomato fruit extracts to enhance the antioxidant defenses of nematodes, increasing their lifespan under conditions of exogenous stress. Achiote seeds, which like tomatoes are rich in carotenoids and vitamin E, have also been studied using a similar approach to the one described, obtaining results in line to those mentioned.

In addition to the mentioned assays, the range of possibilities to test oxidative stress using nematodes includes testing with antioxidant enzymes mutants, fluorescent mutants (GFP or YFP), autofluorescence (lipofuscin) measurements, the use of other stress-inducing agents such as juglone or paraquat, mitochondrial chain mutants, GSH/GSSG ratio, as well as the measurement of lipoperoxidation, protein oxidation, and DNA oxidation products, making the nematode a highly versatile model to study oxidative stress.

Dr. Luis Cárdenas Torres

Biología Molecular de Plantas, IBT, UNAM, México

Dr. Luis Cárdenas Torres is a biotechnology researcher, holding a bachelor's degree from the University of Veracruz and a master's and doctorate from the Institute of Biotechnology at UNAM. He is a Principal Investigator (level B) at the Department of Molecular Plant Biology at the same institute, where he leads a globally recognized research group. His research focuses on the molecular mechanisms of plant-bacteria interaction and polar growth, utilizing novel molecular probes to study signaling in plant cells. Dr. Cárdenas has received prestigious accolades, including the Weizman Award in Natural Sciences in 1998 and UNAM's Alfonso Caso Medal in the same year. He was also honored as a Young Brilliant Scientist by the American Society of Microbiology and received a scholarship for a research stay at the University of Pennsylvania. He has authored over 45 scientific articles and has participated in international conferences such as the Gordon Research Conference and Cold Spring Harbor symposia. Additionally, he has organized scientific conferences and is a member of the National System of Researchers at level II. His research is centered on the symbiotic association between soil microorganisms and leguminous plants, particularly in the biological nitrogen fixation process, a key aspect of plant biology. In 2019/2020, he also embarked on a sabbatical to edit NADPH oxidases in the model moss Physcomitrella patens using Crispr/Cas techniques, aiming to comprehend the oxidative response to pathogens in basal plants. His work has significantly impacted Plant Biology and has been acknowledged by the American Society of Plant Biologists (ASPB).

"The dynamics of ros during polarized growth and its role as a signal molecule"

Dr. Luis Cárdenas Torres

Diego Flores-Bautista [1], Hen-Ming Wu [2], Alice Cheung[2], Janet Palacios-Martínez[1], Ana I. Chávez-Martínez [1], Samantha Ryken[3], Magdalena Bezanilla[3] and Luis Cardenas[1].

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[2]Department of Biochemistry and Molecular Biology, University of Massachusetts, Amherst.

[3]Department of Biological Sciences, Dartmouth College. New Hampshire, USA.

Abstract

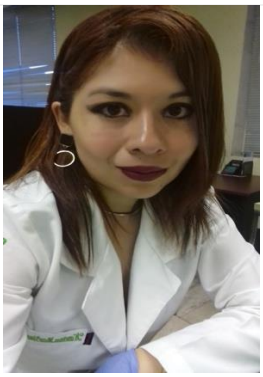
The apical growth of root hairs and caulonema moss cells involves the regulation of the ions flow, calcium homeostasis, exocytosis, and cytoskeleton. Reactive Oxygen Species (ROS) also play an important role in the growth of the root and the root hairs. The production of the ROS occurs as a result of aerobic metabolism during respiration and photosynthesis, and its distribution is higher in organelles such as mitochondria, chloroplasts, and peroxisomes. However, ROS can be produced by the enzymatic activity of NADPH oxidase (RBOHs in plants for respiratory burst oxidase homologue), these enzymes transfer electrons from NADPH to an acceptor, the oxygen, to form the superoxide radical from which other ROS originate as H_2O_2. In this work we measured the dynamics of H_2O_2 levels in living plant roots and root hairs from Arabidopsis expressing a ROS sensor "Hyper 1". This sensor can be studied by a ratiometric analysis and thus renders quantitative data. This approach allows the measurements of ROS intracellular ROS levels in living plant cells under the microscope. Furthermore, we also generated moss transgenic lines which also

express an improved version of the sensor “Hyper 3” which present a superior dynamic range and sensitivity. Since moss experiment a polar growth in caulonema cells, the dynamic and role of ROS can be studied and correlated with intracellular calcium changes during polar growth and responses to external pathogenic elicitors. Our results depict an apical distribution of H_2O_2 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 growth rate.

This work was funded by DGAPA IN-210321 and CV200519 to LC, Conahcyt 253247 and 319643. Conahcyt support for postdoctoral position to JPM and ACM.

SYMPOSIUM 3

"Oxidative stress generating toxicants"



Dra. Karina Martínez Flores

Laboratorio de Líquido Sinovial, INR, México

Doctorate in Experimental Biology from the Universidad Autónoma Metropolitana, she is currently a researcher at the Instituto Nacional de Rehabilitación and a member of the Sistema Nacional de Investigadores Level I. She has over 30 publications in indexed journals and an H-index of 14 with more than 600 citations. Additionally, she has received external funding from CONACyT. For over six years, she has led the research line related to cadmium toxicity from smoking in developing osteoarthritis and other musculoskeletal diseases. Her in vitro work was honored as the best biomedical research work of 2019 by the Universidad Autónoma de Baja California.

"Cadmium-mediated oxidative effect of smoking on musculoskeletal diseases"

Karina Martínez Flores[1]

Karina Martínez Flores[1], Yessica Zamudio Cuevas[1], Octavio Gamaliel Aztatzi Aguilar[2], Gabriela Angélica Martínez Nava[3], Ambar López-Macay[1], Luz María Del Razo Jiménez[4], Javier Fernández Torres[1]

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Abstract

Introduction. Smoking has been implicated in the development of osteoporosis and rheumatoid arthritis, but its role in the etiology of osteoarthritis (OA) is understudied and controversial. It has been reported that exposure to tobacco smoke promotes oxidative stress (OS) as part of its damage mechanism and that this may be caused by some of its compounds. In vitro studies from our research group have shown that cadmium and tobacco smoke extract affect chondrocyte viability by generating OS. Objective. To evaluate whether smoking increases cartilage damage by OS generation. Materials and Methods. Peripheral blood (PB) and synovial fluid (SF) samples from patients with OA were analyzed. The samples were stratified according to smoking habit, Kellgren-Lawrence score, pain, and cotinine

concentrations in PB. Malondialdehyde (MDA), methylglyoxal (MGO), advanced protein oxidation products (APOPs), and myeloperoxidase (MPO) were assessed; the activity of antioxidant enzymes such as gamma-glutamyl transferase (GGT), glutathione-s-transferase (GST) and catalase (CAT), as well as the activity of arginase, which favors the destruction of cartilage, were determined. Results. When stratified by age, for individuals <60 years, the levels of MDA and APOPs and the activity of MPO and GST were higher, as well as antioxidant system activity in the smoking group (OA-S). A greater degree of pain in the OA-S group increased the concentrations of APOPs and arginase activity ($p<0.01$ and $p<0.05$, respectively). Arginase activity increased significantly with a higher degree of pain ($p<0.01$). Conclusion. Active smoking may be an important risk factor for the development of OA by inducing systemic OS in young adults, in addition to reducing antioxidant enzymes in older adults and increasing the degree of pain and cartilage loss, probably due to the presence of cadmium in tobacco.

Dra. Selva Lucía Rivas Arancibia

Laboratorio estrés oxidativo y plasticidad cerebral, Facultad de Medicina, Universidad Nacional Autónoma de México (UNAM), México.

Dr. Rivas Arancibia holds a Medical Surgeon degree and a Doctorate in Biomedical Sciences from the Universidad Nacional Autónoma de México. She is currently a Full Professor C in the Department of Physiology, PRIDE D. She is a Sistema Nacional de Investigadores member. She leads the Oxidative Stress and Brain

Plasticity laboratory at the Facultad de Medicina of UNAM. Her research is focused on understanding how repeated exposure to low ozone doses causes an imbalance in the body's redox state, leading to a chronic oxidative stress state. This results in the loss of inflammatory response regulation, causing degenerative diseases. From the findings of these studies, she has supervised over 20 postgraduate theses, participated in more than 200 conferences, and published over 80 articles in international journals. Currently, her work has received over 2250 citations.

"Ozone environmental pollution and neurodegenerative disease"

Dr. Selva Rivas Arancibia

Department of Physiology, Facultad de Medicina. Universidad Nacional Autónoma de México. CDMX. Mexico

Abstract

Environmental pollution by ozone represents a very complex health issue to address both for our country and the world, as repeated inhalation of low doses of ozone causes the body's antioxidant systems to lose their ability to maintain redox balance. This leads to a chronic state of oxidative stress, which is implicated in chronic-degenerative diseases. Added to this, there are other significant factors that contribute to enhancing these deleterious effects, such as the global temperature rise caused by climate change, which contributes to increased air pollution by ozone. On the other hand, the aging of the population is also critical in the development and progression of neurodegenerative diseases. Moreover, the increase in reactive oxygen species leads to the oxidation of biomolecules, changes in cell signaling, causing alterations in the regulation of metabolic pathways, cell survival, and the immune response, among others. In our laboratory, we use a murine model to study

the effect of repeated exposure to low doses of ozone. The results obtained indicated: severe decrease in brain repair response (neurogenesis), increase in neuronal death, ATP deficiency, reduction in the activity of superoxide dismutase 1 and 2, alterations in the transcription factor NFkB, and an increase in Th1 and Th17 responses and a decrease in the Th2 response. Besides, there is evidence of an altered response from T cells and regulatory T cells. In conclusion, repeated exposure to ozone in healthy animals produces a progressive neurodegeneration process, characterized by neural death and loss of brain repair mechanisms. During this process, the chronic state of oxidative stress and the loss of inflammatory response regulation are critical in the maintenance and progression of the neurodegenerative process, similar to what can occur in Parkinson's and Alzheimer's diseases. *Funded by DGAPA PAPIIT- IN221521 to S.R-A UNAM.*

Dr. Armando Luna López

Departamento de Biología del Envejecimiento, Instituto Nacional de Geriatría(INGER), México.

Researcher in Medical Sciences "E." National Institute of Geriatrics. Distinction in the National System of Researchers Level II. Bachelor's, Master's, and Doctorate in Experimental Biology from the Universidad Autónoma Metropolitana. Research Lines: Molecular and cellular mechanisms of aging, Oxidative stress, and aging, Molecular mechanisms of osteosarcopenic obesity in aging, exercise as a therapy to delay the effects of aging, hormesis. Participation in the publication of 42 indexed

articles. Direction of undergraduate and postgraduate theses. Research stay at the Medical College of Georgia. Acquisition of various research funds.

"Antioxidant effect of low-intensity exercise in a rat model of osteosarcopenic obesity"

Armando Luna López

Beatriz Mena Montes[2], Julián de Jesús Lira Rotstein[2], Raúl Alejandro Librado Osorio[2], Gibrán Pedraza Vázquez[1], Adriana Alarcón Aguilar[1]. Mina Konigsberg Fainstein[1]. Armando Luna López[2].

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Abstract

Aging is a natural and irreversible process that is characterized by the progressive decline of an individual's physiological, biochemical, and structural functions, which makes the organisms more susceptible to acquiring chronic degenerative diseases related to aging, such as diabetes, obesity, osteoporosis, neurodegenerative diseases, and sarcopenia. This decline has been linked to the accumulation of oxidative damage throughout life. Physical exercise and nutritional management, mainly protein and/or amino acid supplementation, are currently the most widely

used interventions for the treatment and prevention of sarcopenia and other age-related disease. In particular physical activity, specifically, exercise, can modulate ROS production and clearance. The impact of exercise on ROS varies from harmful to beneficial and depends on the type of exercise performed. Long-term exercise regulates signaling pathways that enhance antioxidant defense systems and control ROS production. In this study, we developed a model of low-intensity exercise for 20 months to counteract oxidative damage and increment antioxidant response. Oxidative damage and cellular redox state were evaluated, as well as the expression of the main antioxidant enzymes and the regulatory mechanisms of miRNAs. The results showed a decrease in oxidized proteins and a better redox state (GSH/GSSG). Antioxidant enzyme´s expression was higher in exercised rats and miRNA´s regulatory participation was confirmed. Moreover, exercised rats showed increased muscle functionality and survival. In conclusion, we can suggest that performing a low-intensity physical activity might help improve functionality in the elderly since it represents a low-risk and easy-to-perform activity.

This work was partially supported by the CONAHCYT grants: ALL FOSSIS-262302 and MKF FOSSIS-272256, Ciencia de Frontera 2019 (263957), as well as the National Institute of Geriatrics SIRES-DI-PI-021/2015.

SYMPOSIUM 4

"Oxidative stress in Ecology and Evolution"

Dr. Antoine Stier

Institut Pluridisciplinaire Hubert Curien - CNRS University of Strasbourg, Francia.

My research is at the crossroad between physiology, ecology and gerontology. I am broadly interested in understanding the impact of environmental conditions on physiological mechanisms contributing to the ageing process, and how such processes shape organism's phenotype from the conception to the death. My research focus on the contribution of mitochondrial function, oxidative stress, glucocorticoid hormones and telomere dynamics as proximate mechanisms shaping avian health, ageing and life histories. I am a CNRS researcher at the IPHC in Strasbourg (France) and an Adjunct Professor of Physiological Ecology at the University of Turku (Finland).

"Mitochondrial coupling efficiency and ROS production: key candidate mechanisms underlying variation in life-history trajectories?"

Dr. Antoine Stier

Abstract

Understanding why variations in animal life histories exist, have evolved and what are the underlying physiological mechanisms remains a key challenge for biologists. Mitochondria are the powerhouse of eukaryotic cells, providing ~90% of the cellular energy as ATP, but they also produce reactive oxygen species (ROS) that can damage biomolecules and hasten the ageing process. A mechanistic trade-off exists between the efficiency at which mitochondria convert nutrients to ATP and the amount of ROS being produced. Individuals with consistently higher mitochondrial efficiency would therefore be predicted to have a fast pace of life characterized by high reproduction rate, fast growth, early maturity and short lifespan. Yet, empirical tests of this appealing hypothesis remain scarce at best.

I will present data from captive and wild bird species showing the existence of a within-individual and within-family consistency in mitochondrial traits. In captive zebra finches, mitochondrial efficiency was experimentally decreased using a chemical treatment (2,4-dinitrophenol) over the lifespan of individuals. I will present the impact of such treatment on ROS production and life-history traits including lifespan, and the limitations of such an experimental approach.

Dr. Daniel González Tokman

Instituto de Ecología A.C. Xalapa, México

Daniel González Tokman is a biologist with a doctorate in Biological Sciences from the UNAM. Since 2014, he has been a researcher in the Ecoethology Network of the Institute of Ecology (INECOL) in Xalapa, Veracruz. He has undertaken research stays in Mexico, Brazil, Switzerland, Canada, and Australia and is an associate editor of the journal Behaviour. He has over 50 publications on ecology, evolution, toxicology, and insect physiology.

"Oxidative Stress in Insects in Times of Global Change"

Dr. Daniel González Tokman

Abstract

Currently, exposure to high temperatures and other stressors of natural and anthropogenic origin combine and threaten biodiversity. Insects are susceptible to global change due to their ectothermic nature, and combining heat with other stressors can have synergistic effects. In this talk, I will present the risk factors for oxidative stress in insects, current knowledge of antioxidant response mechanisms, and how antioxidants and other mechanisms help prevent oxidative damage. I will

present case studies on insects of economic importance and discuss the significant questions that remain to be resolved to understand better the role of oxidative stress in the evolution of insects in times of global change.

Dra. Vanessa Labrada Martagón

Facultad de Ciencias, Universidad Autónoma de San Luis Potosí (UASLP), SLP,México.

Full-time Research Professor at the Faculty of Sciences of the Universidad Autónoma de San Luis Potosí. Head of the "Ecología de la Salud" Laboratory at the same institution, and holds a Ph.D. in the Use, Management, and Preservation of Natural Resources from the Centro de Investigaciones Biológicas del Noroeste (CIBNOR). Research focuses on the Health Ecology of wildlife, primarily marine megafauna, with over ten years of experience in ecological studies on sea turtles. She has published 26 scientific articles, five outreach articles, 1 Biostatistics book, and three book chapters. She has delivered over 100 talks, including academic seminars, outreach sessions, and presentations at national and international conferences. She has been a member or chair of over 30 thesis committees for undergraduate or postgraduate research projects. She is currently a Level II member of the Sistema Nacional de Investigadores a professor with a PRODEP profile. She is on the editorial committee of the scientific journal "Frontiers in Physiology," Aquatic

Physiology section. Member of the Ibero-American Network RIESCOS: Network for the Evaluation of the Effects of Emerging Contaminants on Human Health.

"Oxidative stress indicators of Green turtles (Chelonia mydas) are biomarkers of habitat conditions in the Mexican Caribbean"

Vanessa Labrada-Martagón[1], Nadia Luisa Islas Madrid[2], Bárbara Cúmez Caté[2], Fernando Alberto Muñoz Tenería[3] Montserrat Solé[4], Tania Zenteno-Savín[2]*

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Abstract

In Mexican Caribbean region, located at the Mesoamerican Reef System, are found the most important foraging and nesting habitats for green sea turtles (Chelonia mydas) characterized for hectares of seagrasses bed. During the last 25 years, urban development and touristic activities have been increasingly stablished in the Quintana Roo state. Additionally, this region has been ecologically afflicted in the last decade by pelagic Sargassum blooms. Information about the biochemical responses of green turtles from the Caribbean is nil, impeding the use of the oxidative stress biomarkers for the evaluation of the health state of this population.

The goal of this study was to quantify the biochemical oxidative stress indicators in intracellular material of red blood cells of green turtles (C. mydas) inhabiting the coast of Mexican Caribbean and characterize the variations in relation to biotic (sex, size, age class, fibropapilloma) and abiotic (years, areas and sampling duration) factors. Free-living green turtles (n=195) were captured in four foraging bays during 2015-2018. Activities of GPx ($r=-0.61$, $p=0.03$) and SOD ($r=0.54$, $p=0.04$) were correlated with handling time taken for bleeding in Akumal and Xcalak, respectively.

Activity of SOD was correlated with body size of the individuals ($r=0.21$, $p=0.04$), thus, green turtle adults captured in the northern region (Punta Arenas) presented the highest activity of this enzyme ($p=0.02$). Levels of carbonyl proteins presented a decrescent pattern from north (Punta Arenas) to south (Xcalak). Annual variability in oxidative stress responses of green turtles from Akumal and Punta Herrero was found; in the former sites, individuals captured during 2015 were characterized for presenting highest activities of GST, GPx, and highest levels of TBARS. A multivariate analysis discriminated the biochemical responses of green turtles captured in Akumal bay during 2015 (Wilks' Lambda=0.06, $p<0.001$), where their production rate of $O2^{\bullet-}$, activities of SOD, GPx, GST, and the levels of TBARS were highest than 2016 and 2018. Biochemical responses of green turtles captured during 2015 in Akumal and Punta Herrero bays coincided with one of the first peaks reported of massive atypical influx of pelagic sargassum, which in turn decreased in 2016 and 2017. Results of this study corroborates the utility of the oxidative stress indicators as biomarkers of environmental conditions in this sentinel's species.

WOMEN IN SCIENCE ROUND TABLE

"Science in Feminine and the Power of Mentorship"

Moderator: Dr. Norma Edith López Díaz-Guerrero, UAM-I

Dr. Margarita Martínez Gómez

Dr. Margarita Martínez Gómez is a senior researcher at the Instituto de Investigaciones Biomédicas of UNAM and assigned to the Unidad Foránea located at the Universidad Autónoma de Tlaxcala (UATx), where she currently holds the position of Academic Secretary. She obtained her Bachelor's degree in Biology from Universidad Veracruzana, a Master's degree in Reproductive Biology from UATx, and a PhD in Physiological Sciences from Universidad Nacional Autónoma de México. She had research stays in Canada, the USA, and Hungary during her training. She has been a Researcher of the Sistema Nacional de Investigadores with Level III since 2011. She served as the president of the Academia de Investigación en Biología de la Reproducción A.C.. As of September 2023, she is the Sociedad Mexicana de Ciencias Fisiológicas AC president. She is also a member of the Seminario Universitario Interdisciplinario de Envejecimiento y Vejez of UNAM. Her primary research focus is on female reproductive physiology. She has supervised dozens of student theses and collaborates with USA, Spain, and France researchers. She founded the Unidad de Atención Integral a la Mujer, a program dedicated to the comprehensive health of elderly women in Tlaxcala and another promoting scientific vocations among young girls. She initiated and now coordinates the activities of the Estación Científica la Malinche UATx-UNAM, which advocates for the conservation of this mountain's natural resources and their connection to human health. She is the founder of the postgraduate program in Biological Sciences

at UATx (programs within the Sistema Nacional de Posgrados of CONACyT). She has received several awards: El Faro de la Sabiduría and the Reconocimiento Paulina Maraver by UATx; the "Patria" Award and "Xochitecatcihuatl" Award from the Government of the State of Tlaxcala; the "Sor Juana Inés de la Cruz" acknowledgment by UNAM; and the research group award "Por Amor al Planeta" granted by Volkswagen de México.

Dr. Arely Anaya Hernández

Dr. Arely Anaya Hernández holds a degree in Pharmaceutical Biology Chemistry from the Benemérita Universidad Autónoma de Puebla, a Master's in Biological Sciences from Universidad Autónoma de Tlaxcala, and a Ph.D. in Neuroethology from Universidad Veracruzana. She has conducted research at the Instituto de Neurociencias de Alicante and Universidad Miguel Hernández de Elche in Spain. Dra. Anaya completed a postdoctoral stay at the Centro de Investigación en Genética y Ambiente of Universidad Autónoma de Tlaxcala, where she is currently a full-time academic. She has taught courses and supervised theses at both undergraduate and postgraduate levels. Her current research line is in Molecular Toxicology, where she evaluates the effects of environmental pollutants on biota, including humans, focusing on exposure and effect biomarkers. Among her accolades, she received the Tlaxcala State Youth Award in 2015 for Academic Merit, was selected by the British Council to be part of the first generation of the "Mentoras en la Ciencia" program for 2021-2022, and was chosen by the Global Young Academy for the first Scientific Leadership Program of Latin America and the Caribbean 2022-2023. She is a Sistema Nacional de Investigadores of CONACyT Level I member. Additionally, she has been an invited evaluator for national and

international research projects, national conference papers, and international scientific journals.

Dr. Verónica Reyes Meza

Dr. Verónica Reyes Meza is a psychologist with a Master's in Neuropsychological Diagnosis and Rehabilitation. She holds a doctorate in Neuroethology and a postdoctorate in Biological Sciences. Dra. Anaya is a member of SNI, Level 1. She has had research stays at the University of California, Instituto Nacional de Astrofísica Óptica y Electrónica, Université Sorbonne Paris Nord, and Università degli Studi della Tuscia. In 2006, she won the Ibero-American contest Leamos la Ciencia para Todos. She is currently a full-time professor in the postgraduate program in Biological Sciences at the Centro Tlaxcala de Biología de la Conducta, UATx.

VIII Congreso de Especies Reactivas del Oxígeno en Biología y Medicina

VIII Congress of Reactive Oxygen Species in Biology and Medicine

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