The 17 th International Conference of
the Society of Neuroscientists of Africa

Proceedings

BRAIN and ENVIRONMENT:
"The Challenge of the Future"

April 17th - 20th, 2025 | Marrakesh, Morocco

Welcome message by the president of SONA2025

As the Chair of the Organizing Committee and President of the 17th Society of Neuroscientists of Africa (SONA) Conference in 2025, I extend a warm invitation to all neuroscientists from Africa and around the world to join us for the Joint SONA – MAN Conference 2025. This exciting event will take place at the Conference Center of Cadi Ayyad University in Marrakech from April 17th to 20th, 2025, under the theme "BRAIN and ENVIRONMENT: The Challenge of the Future." This international conference will be the third one to be hosted in Morocco, after the SONA 1995 in Marrakesh and the SONA 2013 in Rabat. It could also be the last SONA Conference organized before the 2027 IBRO World Congress, which will be held in Africa.

Morocco has consistently garnered the attention and support of the International Brain Research Organization (IBRO) and its distinguished representatives. Over the years, we have had the honor of hosting eminent figures such as David Ottoson from Sweden, Pierre Magistretti from Switzerland, Carlos Belmonté from Spain, Marina Bentivoglio from Italy, and many others. Their presence has enriched our scientific discourse and strengthened our ties with the global neuroscience community.

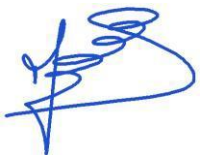
This Conference aims to be a crossroads for exchanging scientific information, ideas, and expertise between African neuroscientists and those from worldwide. The program will particularly highlight significant research, recently conducted, covering various fields, from fundamental to clinical neurosciences. It will offer, through plenary lectures, symposia, oral, and poster presentations, an opportunity for discussion and exchange of information between clinical and neuroscientists on the issues and challenges of the future in understanding the mysteries of the brain. In the margins of this conference, several pre- and post-conference satellite activities, including schools and workshops, will be held.

Attending SONA 2025 is also an opportunity to explore the beauty and magic of the imperial city of Marrakech and southern Morocco, as well as to experience its gastronomic, cultural, and touristic diversities through a social program proposed by our tourism operator to participants.

We sincerely thank you for joining us and being a part of this exciting international neuroscience event in Africa, held in Marrakesh, "the Red City", Morocco.

The combination of the participants' involvement and the total devotion of the organizing and scientific committees was the key to the success of our SONA2025.

Warm regards,



Professor Mohamed BENNIS
President of the Moroccan Association of Neurosciences
President of the SONA
President of the SONA Conference 2025
Chair of the Organizing Committee

Preface to the SONA2025 Conference Proceedings

We are genuinely enthusiastic to introduce the compilation of papers from the 17th International Conference of the Society of Neuroscientists of Africa (SONA2025). This significant event unfolded at the lively Cadi Ayyad Conferences Center in Marrakesh, Morocco, between the 17th and 20th of April, 2025. Anchored by the thought-provoking theme, "Brain and Environment: The Challenge of the Future," this key assembly tackled the pressing global issue of brain-related illnesses. These conditions stand as the primary driver of disability worldwide, even overshadowing the impact of cancer, heart disease, and infectious ailments. Through vibrant, interdisciplinary conversations and a rich exchange of knowledge, SONA2025 acted as a pivotal springboard. It spurred the pinpointing of core origins, forged essential connections for the sharing of technology, and ignited crucial collaborations. These cooperative endeavors aimed at crafting effective strategies for treatment, preventative measures, and interventions, touching upon vital areas like research, policy-making, education, and advocacy.

Our sincere gratitude extends to the esteemed individuals who participated from across Africa, Europe, and the Americas. Their varied presentations and insightful dialogues significantly enriched the fabric of the conference. Their contributions shed light on the complex relationship between the human brain and its surroundings across a diverse range of subjects. A particular highlight of SONA2025 was the honored presence and invaluable contributions of our distinguished invited speakers:

- Prof. Wail Benjelloun, Emeritus Professor at the University of Mohamed V of Rabat, Morocco.
- Prof. Rosa Cossard, Director at INMED and Director of Development of the Hippocampal Cognitive MAPS Lab at Aix-Marseille University, France, and also a Member of the French Academy of Sciences.
- Prof. Khalid El Allali, Head of the Comparative Anatomy Unit at the School of Veterinary Medicine, Hassan II Institute of Agronomy and Veterinary Medicine, Rabat, Morocco.
- Prof. Tracy Bale, The Anschutz Foundation Endowed Chair in Women's Integrated Mental & Physical Health Research at the Ludeman Centre; Director of Inter-Generational Stress and Health; and Director of the Department of Psychiatry Sex Differences Research at the University of Colorado.
- Prof. Toshihide Yamashita, Distinguished Professor and Chairman of the Department of Molecular Neuroscience and the Department of Neuro-Medical Science, Graduate School of Medicine, Osaka University, Osaka, Japan.
- Prof. Juan Lerma Gomez, Vice President of the European Brain Council EMBO and member of the Instituto de Neurociencias CSIC-UMH San Juan de Alicante, Spain.
- Prof. Elizabeth Ngo Bum, Dean of the Faculty of Sciences at the University of Maroua; Head of the Laboratory of Medicinal Plants and Galenic Formulation at the University of Ngaoundéré; and a Member of the Cameroon Academy of Science.
- Prof. William Wisden, PhD FMedSci, from the Department of Life Sciences & UK Dementia Research Institute, Imperial College London, UK.

Within these pages lies a collection of abstracts representing the 19 key themes explored during the SONA2025 conference. We once again express our profound appreciation to all attendees for their active involvement and contributions to the conference program. Finally, we would like to extend our warmest thanks to everyone who contributed with their unstinting dedication and considerable efforts, which were essential in making this important event a resounding success.

Sponsors

On behalf of the organizing committee of the SONA 2025 Conference and the attendees, we extend our deepest gratitude for the invaluable support of our sponsors. Your generous moral and financial support played a crucial role in the success of the conference. Thank you for being such a valuable partner.

Local Organizing Committee

Name	*Institution*
Mohamed BENNIS (Chair)	SONA+AMN
Saadia Ba-M'HAMED	FSSM, Marrakech
Loubna BOUKHZAR	FSSM, Marrakech
Fatima-Zohra LAMGHARI	FSSM, Marrakech
Abderrazzak GHANIMA	FST, Marrakech
Najib KISSANI	FMPM, Marrakech
Zakaria OUHAZ	ISPITS, Marrakech
Noureddine KAIKAI	ENS, Marrakech
Nezha BOUHADDOU	Fac. Sci., Rabat
Hicham FARSI	IAV Hassan II, Rabat
Yassine AIT-BALI	ENS, Rabat

Scientific Committee

Name	*Country*	*Name*	*Country*
Nouria LAKHDAR-GHAZAL (Chair)	Morocco	Athanase MILOGO	Burkina Faso
Mohamed BENNIS (Loc Chair)	Morocco	Amira ZAKY	Egypt
Lihle QULU	South Africa	Mahmoud MANIA	United Kingdom
James OLOPADE	Nigeria	Salimata DIANG SAGNA	Senegal
Fatiha CHIGR	Morocco	Willias MASOCHA	Zimbabwe/ Kuwait
Amadi O IHUNWO	South Africa	Ines ELBINI DHOUIB	Tunisia
Gwladys Temkou NGOUPAYE	Cameroon	Wael MOHAMMED	Malysia
Nilesh Bhalalbhai PATEEL	Kenya	Robert BINEY	Ghana
Lumbuka KAUNDA	Zambia	Rajesh KALARIA	United Kingdom
Samson SAHILE	Ethiopia	Désiré Tschala KATUMBAY	USA
Zeinab KONE	Mali		

Overview
of the SONA 2025 main activities

Covered topics

- Neurogenetic, Neurodevelopment & Stem Cells
- Neuroanatomy, Structural & Functional connectomics
- Sensory & Motor Systems
- Emotion, Cognition & Behaviour
- Neuroendocrine Systems, Biological Rhythms & Sleep
- Neuroimmunology, Neuroinflammation & Neuroinfection
- Neurochemistry
- Neuropharmacology & Ethnopharmacology
- Neuroethology
- Nutrition, Metabolic disorder & Brain disease
- Stress & Mental Health
- Neurodegeneration, Neuroplasticity & Repair
- Pollutants, Neurotoxicity & Brain Disorders
- Drug Addiction & Drug of Abuse
- Clinical Neuroscience
- Computational & Theoretical Neurosciences
- Robotic, Artificial Intelligence & Neurosciences
- Neuroethics
- History, Education & Teaching

Plenary Lectures

ISN-Keynote

Rosa Cossart

Director of INMED
Director of Development of Hippocampal Cognitive MAPS Lab, Aix-Marseille University, France
Mmember of the Academy of Sciences, France

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Khalid EL Allali

Head of Comparative Anatomy Unit School of Veterinary Medicine Hassan II Institute of Agronomy and Veterinary Medicine, Rabat, Morocco

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Tracy Bale

The Anschutz Foundation Endowed Chair in Women's Integrated Mental & Physical Health Research at the Ludeman Centre; Director, inter Generational Stress and Health; Director of Department of Psychiatry Sex Differences Research University of Colorado,

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IBRO-Sponsored

Toshihide Yamashita

Distinguished Professor and Chairman Department of MolecularNeuroscience Department of Neuro-Medical Science. Graduate School of Medicine, Osaka. University Osaka, Japan

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EMBO Keynote

Juan Lerma Gomez,

Vice President of European Brain Council
EMBO member
Instituto de Neurociencias CSIC-UMH San Juan de Alicante, Spain

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IBRO-ISN Sponsored

Elisabeth Ngo Bum

Dean of Faculty of Sciences, University of Maroua. Head of laboratory of medicinal plant and Galenic formulation, University of Ngaoundéré, Member of the Cameroon Academy of Science.

eli_bum@yahoo.fr

IBRO-Sponsored

William Wisden

PhD FMedSci
Department of Life Sciences & UK Dementia Research Institute
Imperial College London UK

w.wisden@imperial.ac.uk

Special lecture

Wail A. Benjelloun

wbenjelloun@gmail.com
Emeritus Professor
Mohammed V University
Rabat, Morocco

Tribute in memory of James Kimani

James Kimani Lecture

Turning toxins into treatment: The therapeutic potential of animal venoms in neurodegeneration

Ines ELBINI
Institut Pasteur de Tunis, Tunisia

Special Lecture

Are there really non-psychedelic psychedelics? A comparison of lisuride and LSD

Scott Thompson, PhD
Center for Novel Therapeutics
University of Colorado School of Medicine
Anschutz Medical Campus, USA

Speaking Science or Letting Science Speak

28 Symposia
3 to 5 talks/Symposium

9 Oral Presentations sessions

3 Poster sessions

The SONA 2025 Events Calendar

Thursday, April 17, 2025

08:00 | Whole Day Registration

09:00 – 11:00 | Symposia: Session 1

- **SP1 (Room1)**: "Brain Circuits for Motor and Social Behaviors"

Organizer: ***Abdeljabbar El Manira*** *(Sweden)*

- **SP2 (Room2)**: "Serotonergic Modulation and Neurochemical Pathways in Neurological Disorders"

Organizers : ***Philippe De Deurweardere*** *(France)* ***& Victor O. Olotu*** *(Nigeria)*

11:00 – 11:15 Coffee Break

11:15 – 11:45 | Opening Ceremony

- **Prof. Belaid Bougadir,** *President of the Cadi Ayyad University*
- **Prof. El Hassan El Mouden,** *Dean of the Faculty of Sciences Semlalia*
- **Mohamed Bennis,** *President of SONA2025 and President of the organizing committee*
- **Nouria Lakhdar-Ghazal,** *President of the Scientific Committee*
- **Sadiq Yousuf,** *General Secretary of the SONA*
- **Hon. Mai Mala Buni,** *Executive Governor of Yobe State, Nigeria*

11:45 – 12:45 | Opening Lecture:

"**How GABAergic neurons shape cortical circuit assembly in health and disease**"

Speaker: ***Rosa Cossart*** *(France)*

sponsored keynote

12:45 – 14:30 | Welcome Reception

14:30 – 15:15 | Plenary Lecture 1:

"**Stress across generations: Mechanisms for altering brain development**"

Speaker: ***Tracy Bale*** *(USA)*

sponsored keynote

15:30 – 17:30 | Symposia: Session 2

- **SP3 (Room1)**: "Emerging Effectors in Neurodegeneration: From Molecular to Therapeutic Targets"

*Organizers: **Inès El Bini Dhouib & Rym Benkhalifa** (Tunisia)*

- **SP4 (Room2)**: "Molecular Targets of Addictive Drugs and Associated Behaviors"

*Organizer: **Mohammed Kabbaj** (USA)*

- **SP5 (Room3)**: "Young Women Investigators in Africa"

*Organizer: **Nouria Lakhdar-Ghazal** (Morocco)*

- **SP6 (Room4)**: "The Science of Burnout: From Brain Mechanisms to Holistic Management Approaches"

*Organizer: **Daniel Gams Massi** (Cameroon)*

17:30 – 18:00 Coffee Break

18:00 – 19:30 | Oral Presentations: Sessions 1&2

- **Room 1/ Session 1.** Topic: "Clinical Neurosciences and Epidemiology of Neurological and Psychiatric Disorders"
  Chair of the session: **Fatima Zahra Lamghari-Moubarrad**, *Cadi Ayyad University, Marrakech, Morocco*

Time Slot	Speaker	Title of the Oral Communication
18:00 -18:10	Igor Branchi (Italy)	Plasticity as a clinical marker: implications for resilience and vulnerability to psychopathology
18:10 -18:20	Irina Shoshina (Russia)	Ocular microtremor in assessing the dynamics of the condition in schizophrenia
18:20 -18:30	Sonia Bansal (Usa)	Clinical, perceptual, and neural features associated with hallucinations in clinical and non-clinical voice hearers
18:30 -18:40	Chaimae Bouab (Morocco)	Relationship between thyroid dysfunction in a Moroccan population and behavioral status
18:40-19:00	**General Discussion**	

- **Room 2 / Session 2.** Topic: "Ethics, Sleep, and Neuroscientific Insights from Animal and Computational Models"
  Chair of the session: **Rachida Roky**, *Hassan II University, Casablanca, Morocco*

Time Slot	Speaker	Title of the Oral Communication
18:00-18:10	Tom Buller (Usa)	Brain and environment: can neuroethics meet the challenge of the future?
18:10-18:20	Victoria Williams (South Africa)	Artificial intelligence: 'Exploring empathy in language models'

18:20-18:30	Rachida Roky (Morocco)	Sleep, Chronotype, and Stress Among Students: A Sex-Based Analysis
18:30-18:40	Hicham Farsi (Morocco)	Circadian Regulation of Rumination in Desert Black Goats (*Capra hircus*): Evidence for an Endogenous Rhythm
18:40-18:50	Mohammed El Mehdi M'hani (Morocco)	Entrainment of the master circadian clock by the timing of food distribution in goats (*Capra hircus*)
18:50-19:00	**General Discussion**	

Room 3 /ALBA Network-IBRO Workshop: 18:00-19:30

"Equity in publishing: strategies for inclusive publishing and knowledge sharing".

Organizer: ***Francesca Cirulli*** *(Italy)*

19:45 – 21:30 | IBRO Connect: Networking Event

Friday, April 18, 2025

09:00 – 11:00 | Symposia: Session 3

- **SP7 (Room1)**: "An update on the pathophysiology of the non-motor symptoms in Parkinson's disease"
  Organizer: ***Abdelhamid Benazzouz (France)***
- **SP8 (Room 2)**: "Molecular Mechanisms of Heavy Metal Toxicity in Neurodegenerative Diseases: Translational Medicine and the Role of Natural Antidotes".
  Organizer: ***Chinna Orish*** *(Nigeria)*
- **SP9 (Room3)**: "Early life stress, Sex differences, and Maternal influences: Insights into Brain and Behavioral Disorders"
  Organizer: ***Fatiha Chigr*** (Morocco)
- **SP10 (Room4)**: "The neurobiology of animals' adaptations to harsh African biotopes"
  Organizer: ***Khalid El Allali*** (Morocco)

11:00 – 11:15 Coffee Break

11:15 – 12:00 | Plenary Lecture 2:

"Neuro-adaptation to the desert biotope: An overview on the recent data of camel and goat"

Speaker: ***Khalid El Allali*** *(Morocco)*

12:00 – 12:30 | James Kimani Lecture Session

Lecture giving by**: *Inès El Bini-Dhouib*** *(Tunisia)*

12:30 – 14:00 | Lunch and Posters Session 1

14:00 – 14:45 | Plenary Lecture 3:

"Impact of the traditional medicine in Cameroon"

*Speaker: **Elisabeth Ngo Bum** (Cameroon)*

sponsored keynote

15:00 – 17:00 | Symposia: Session 4

- **SP11(Room1)**: "Natural Products Research, Characterization and Therapeutic Role".

*Organizer: **Martha Davilla-Garcia (USA)***

- **SP12 (Room2)**: "Brain Pathologies - Neuron and Glia Diversity in Regeneration".

*Organizers: **Frank Kirchhoff** (Germany) **& Luciana Politti Cartarozzi** (Brasil)*

- **SP13 (Room3)**: "Emerging regenerative medicine for CNS repair".

*Organizers: **Fatiha Nothias & Afsaneh Gaillard** (France)*

- **SP14 (Room4)**: "The signaling system of the neuropeptide relaxin 3/rxfp3 in cognitive and pain processes".

*Organizers: **Monica Navarro & Olucha Bordonau** (Spain).*

17:30 – 18:00 Coffee Break

17:15 – 19:15 | Oral Presentations: Sessions 3, 4, 5

- **Room 1 / Session 3.** Topic: "Neuroprotection and Neurobiological Disruption in Neurodegenerative Diseases"

Chair of the session: **Samir Ahboucha**, *FPK, USMS, Khouribga, Morocco*

Time Slot	Speaker	Title of the Oral Communication
17:15 -17:25	Adejoke Elizabeth Memudu (Nigeria)	Protective Effects of Lutein-20 Against Cadmium-Induced Oxidative Stress and NeuroinflammationinthePrefrontalCortexandHippocampusofAdultMale Wistar Rats
17:25 -17:35	El Hafedh El Mouhab (Mauritania)	Parkinson's Disease in Mauritania: Epidemiological Profile and contribution to the genetic analysis (the G2019S mutation).

17:35 -17:45	Hiba Midouane (Morocco)	In Silico ModelingofLRRK2G2019SProtein-ProteinInteractionsinParkinson'sdisease: A MachineLearningApproach
17:45 -17:55	Kaoutar Aalilouch (Morocco)	Neonatal microglia enhance aged opc differentiation via autophagy, and metformin restores differentiation capacity in aged opcs: insights from a mouse co-culture model
17:55 -18:05	Oussama Duieb (Morocco)	Systematic review of the effect of environmental enrichment on brain-derived neurotrophicfactor(BDNF)activityformitigatingbehavioralmanifestationsin an autism spectrum disorder (ASD) model.
18:05 -18:15	Raphael Takyi (France)	Longitudinal brain aging after stroke: a marker for neurodegeneration and its relevance for motor outcome
18:15 -18:25	Rebecca Casterton (Uk)	Investigating neuronal cell cycle re-entry in C9ORF72 frontotemporal dementia
18:25 -19:35	Samir Ahboucha (Morocco)	Neurosteroids and Brain Injuries: Are they protective or deleterious?
19:35	**GeneralDiscussion**	

- **Room 2 / Session 4.** Topic: "Neuropharmacology and Traditional Ethnobotanical Agents in Modulating Cognitive and Behavioral Disorders.

Chair of the session: **Nikolaos Pitsikas,** *University of Thessaly, Larissa, Greece*

Time Slot	Speaker	Title of the Oral Communication
17:15 -17:25	Nicolas Guyon (France)	Manipulating endogenous nicotinic receptors and associated behaviors with photoactivatable agonists
17:25 -17:35	Nikolaos Pitsikas (Greece)	The nitric oxide synthase inhibitor 7-nitroindazole counteracts social withdrawal and cognition deficits induced by the blockade of the NMDA receptor in the rat.
17:35 -17:45	Hanane El Fatimi (Morocco)	A Novel Benzodiazepine Derivative: Exploring Anxiolytic-like effect and potential side effect.
17:45 -17:55	Ifeoma Felicia Chukwuma (Nigeria)	Bioassay-guided Identification of Potential Alzheimer's Disease Therapeutic Agents from Kaempferol-Enriched Fraction of *Aframomum melegueta* Seeds using in Vitro and Chemoinformatics Approaches.
17:55-18:05	Isaac Blessed Mensah (Ghana)	Evaluating the Potential of Stigmasterol as a Scaffold for Novel Antidepressants
18:05 -18:15	Samuel Onasanwo (Nigeria)	Impact of Ocimum gratissimum-supplemented diet on scopolamine-induced Alzheimer's disease-like memory impairment in Swiss mice.
18:15 -18:25	Soumia Ed-Day (Morocco)	Neuroprotective effects of Schinus terebinthifolius Raddi on streptozotocin-induced Alzheimer's disease-like symptoms in rats.
18:25 -19:35	Stève Brunel Ngoufack Kenfack (Cameroon)	Preventive effect of Graptophyllum grandulosum on Conditioned place preference-induced nicotine addiction on pubescent adolescent rats
19:35	**GeneralDiscussion**	

- **Room 3 / Session 5.** Topic: "Neurobiological and Psychosocial Impacts of Stress, Trauma, and Therapeutic Interventions"

Chair of the session: **Hassan Ainani**, *Mohamed VI University Benguerir*

Time Slot	Speaker	Title of the Oral Communication
17:15 -17:25	Matthias Ertl (Switzerland)	Vestibular perceptual training enhances vestibular perception, posture, and gait in older adults
17:25 -17:35	Hassan Ainani (Morocco)	Potential role of Piezo1 in the mechanical responses of neurons and glial cells in dorsal root ganglia
17:35 -17:45	Hidaayah Jimoh-Abdulghaffaar (Nigeria)	Aspirin as a modifier of epigenetic responses: DNA methylation changes in a social instability stress model of depression in female Wistarrats
17:45 -17:55	Yassine Bentefour (Morocco)	Effects of early-life stress on reproductive behavior in female mice
18:05 -18:15	Hamza Tahiri (Morocco)	Evaluation of the anxiolytic potential of passionflower, valerian, and lemon balm on physiological parameters and neurobehavioral effects in mice subjected to stress
18:15	**General Discussion**	

19:15 | AMN General Assembly

Saturday, April 19, 2025

09:00 – 11:00 | Symposia: Session 5

- **SP15 (Room1)**: "Neuro-immune dysfunction and mental health outcomes: advances in immune-psychiatry in Africa".
  *Organizer: **Arish Mudra Rakshasa-Loots** (UK)*

- **SP16 (Room2)**: "Neuroinfections".
  *Organizers: **Francesca Cirulli & Rachael Dangarembizi** (Italy)*

- **SP17 (Room3)**: The Pathophysiology & challenges in therapy of neurodegenerative disease.
  *Organizer: **Abdelhamid Benazzouz** (France)*

09h00 – 11h00 | Oral Presentations: Session 6

- **Room 4, Session 6** / Topic: "Pollutants, Neurotoxicity, and Brain Disorders".

  Chair of the session: **Said Galai**, *National Neurological Institute, Tunis, Tunisia*

Time Slot	Speaker	Title of the Oral Communication
9:00 -9:10	Emeka Chika Igwe (Nigeria)	High-fat diet and Bisphenol A interactions sustained the physiological state of microglia and elicitedtheexpressionofalpha-synucleinandparvalbuminproteinsinthecerebellum of male Wistar rats
9:10 -9:20	Asmaa Lafram (Morocco)	Effects of exposure to micro/nanoplastics of polystyrene on neuronal oxidative stress, neuroinflammation, and anxiety-like behavior in mice.
9:20 -9:30	Nour-Eddine Kaikai (Morocco)	Maternal Exposure to Metam Sodium-Based Pesticides Induces Long-Term Neurodevelopmental Disorders Linked to Oxidative Stress and Neuroinflammation
9:30 -9:40	Steeve Thany (France)	Understanding the neurotoxic impact of pesticides on mammalian brain nicotinic acetylcholine receptors
9:40 -9:50	Said Galai (Tunisia)	Implementation of new protocols for Glyphosate and its byproduct detection. Application for demonstration of neurotoxicity by in vitro N2a cell culture
9:50 -10:00	Meriem Laaroussi (Morocco)	Multigenerational Reproductive and Neuroendocrine Effects of Chronic Mercury Chloride Exposure in Female Mice
10:10 -10:20	Paul ADELEKE (Nigeria)	Roles of oxidative stress and pro-inflammatory cytokines in copper sulfate-induced depression-like disorders and abnormal neuronal morphology in mice
10:20 -10:30	Hafsa MALQUI (Morocco)	Effect of Aging and mercury chloride intoxication interaction in mice
10:30 -10:40	Assmaa TALI (Morocco)	Lambda-cyhalothrin sex-dependently alters behavioural abilities in Swiss mice
10:40	**GeneralDiscussion**	

11:00 – 11:15 Coffee Break

11:15 – 12:00 | Plenary Lecture 4:

"Kainate Receptors: A Double-Edged Sword in Brain Health and Disease" Speaker:
Juan Lerma Gomez *(Spain)*
EMBO *IBRO-sponsored keynote*

12:00 – 12:30 | ISN-Lunch Sponsored Talk:

12:30 – 14:00 | Lunch and Posters Session 2

14:00 – 14:45 | Plenary Lecture 5:
"Repulsive guidance molecule regulates glial and immune function under neurological diseases"
Speaker: ***Toshihide Yamashita*** *(Japan)*
IBRO *sponsoredkeynote*

15:00 – 17:00 | Symposia: Session 6

- **SP18 (Room 1)**: "The rising Star symposium: Neuroimmunology Schools in Africa Alumni Research and Progress".
  IBRO *- **ARC** - Organizers: **Willias Masocha** (Kuwait) **& Roberto Furlan** (Italy)*
- **SP19 (Room 2)**: "Neurological disorders caused by Mediterranean pollutants".
  (PsyCoMed) *Organizer: **Marc Landry** (France)*
- **SP20 (Room 3)**: "Environmental toxins and brain alterations: from mild cognitive effects to severe consequences on neuronal cell death".
  *Organizer: **Samir Ahboucha** (Morocco)*
- **SP21 (Room 4)**: "Artisanal mining and its impact on the brain in the eastern region of Cameroon"
  *Organizers: **Ayissi Rigobert Espoir & Eric Bila Lamu** (Cameroon)*

17:00 – 17:15 Coffee Break

17:15 – 19:00 | Networking Events and Oral Presentations (Sessions 7 & 8)

- **Rooms 1 & 2: World Women in Neuroscience Mentoring Circle (WWN)**
  Title: **"*The Art of Building a Career in Neuroscience*"**
  *Organizer: **Martha Dávila-García** (USA)*

- **Room 3 / Session 7** / Topicof: "Neurobiological mechanisms of behavior, emotion, and addiction across diverse models"

Chair of the session: **Abderrazzak Ghanima**, *Cadi Ayyad University, Marrakech Morocco*

Time Slot	Speaker	Title of the Oral Communication
17:15 -17:25	Mohamed Aly Zahran (Spain)	Peptidergic systems involved in aggressive processes after alcohol intoxication
17:35 -17:45	Cathy Perrina Ranaivomanana (Morocco)	The Role of Nicotinic Receptors in Nicotine-Induced Drug-Seeking Behavior in Drosophila melanogaster
17:45 -17:55	Ilias Chaibi (Morocco)	Topiramate inhibits aggressive behavior through targeting morphological and functionnal alterations of the anterior cingulate cortex in socially isolated mice
17:55 -18:05	Mónica NAVARRO SÁNCHEZ (Spain)	Neuropeptidergic Modulation and Neural Pathways in Contextual Fear Conditioning and Extinction: Insights from the Retrosplenial Cortex and Nucleus Incertus
18:15 -18:25	Prune Mazer (Portugal)	Predictive processing and autistic traits: Neurophysiological evidence across sensory modalities and task demands
18:25 – 19:00	**General Discussion**	

- **Room 4 / Session 8** / Topic: "Neurodevelopmental, Neuroinflammatory, and Neurodegenerative Mechanisms in Disease Models and Behavioral Outcomes."

Chair of the session: **Silvestre Sampino**, *Institute of Genetics and Animal Biotechnology, Poland*

Time Slot	Speaker	Title of the Oral Communication
17:15 -17:25	Hiroaki Taniguchi (Morocco)	Dysfunction of the Nfe2l1 Gene Disrupts Parkinson's Disease-Related Gene Expression Under Ubiquitin Stress and Impairs Neuronal Differentiation in P19 Cells
17:25 -17:35	Silvestre Sampino (Poland)	Prenatal determinants of neurodevelopmental programming in the BTBR mouse model of autism
17:35 -17:45	Binu THARAKAN (USA)	NLRP3 Inflammasome Signaling Promotes Blood-Brain Barrier Dysfunctions

17:45 -17:55	Nahla Ouard (Morocco)	Different cortical and subcortical astroglial responsiveness in rats with acute liver failure
17:55 – 18:15	**GeneralDiscussion**	

20h 00 GALA DINNER

Sunday, April 20, 2025

09:00 – 11:00 | Symposia: Session 7

- **SP 22 (Room1):** "The Brain, Environment and Nutraceuticals; Combating Neurodegeneration through Neuroprotection"

Organizer: ***Abel N. Agbon (Nigeria)***

- **SP 23 (Room2):** "Epidemiology, Physiopathological and Therapeutic Aspects of Multiple Sclerosis: An Emerging Disease in Africa"

Organizers: ***Fatiha Chigr & Samir Ahboucha*** (Morocco)

- **SP 24 (Room3):** "Management of Neurodegenerative Disorders by Potential Bioactive Compounds"

Organizer: ***Olfa Masmoudi & Taoufik Ghrairi (Tunisia)***

- **SP 25 (Room4):** ISN sponsored UM5 School Alumni symposium

Organizer: ***Nouria Lakhdar-Ghazal*** (Morocco)

11:00 – 11:15 | Coffee Break

11:15 – 12:00 | Plenary Lecture 6:

"How do we sleep?"

Speaker: ***William Wisden*** *(UK)*

sponsored keynote

12:00 –12h30 *Special* Lecture

"Are there really non-psychedelic psychedelics? A comparison of lisuride and LSD"

Speaker: ***Scott Thompson*** *(USA)*

11:00 – 11:15 | Lunch + Posters session 3

14:00 – 14:45 | *Plenary lecture 7*

"African Neuroscience and Human Development Strategies"

*Speaker****: Wail Benjelloun*** *(Morocco) Special Keynote*

15:00 – 17:00 | Symposia*: session 8 & Oral presentations: Session 9*

- **SP 26** (Room 1): "Recent Advances At The Interface Of Neuroscience And AI (NeuroAI)"

Organizer: ***Sricanth Ramaswamy*** *(UK)* ***& Jie Mei*** *(Austria)*

- **SP 27** (Room 2): "Advancing Neuroscience Education in Africa through Capacity Building"

Organizers: ***Sharon Juliano*** (USA)

- **SP 28** (Room 3): "The Brain Research International Data Governance & Exchange (BRIDGE): African perspectives"

Organizer: ***Amadi Ihunwo*** (South Africa)

- **Room 4 / *Session 9*** / Topic: "Neurobiological Impacts of Environmental, Genetic, and Nutritional Factors on Brain Structure and Function."

Chair of the session: **Toluwalope Ajonijebu**, *Northwest University South Africa*

Time Slot	Speaker	Title of the Oral Communication
15:00- 15:10	Elmehdi Hamouda (China)	The impact of Preterm Birth on Gyri and Sulci' Structural and Functional Connectivity
15:10 -15:20	Hanane Iben Daoudi (Morocco)	Dissociating sensory-discriminative and affective-emotional pain processing in the mediodorsal thalamus
15:20 -15:30	Ihsane Ait Mansour (Morocco)	Preventive effects of environmental enrichment on behavioral impairment and morphological alterations of the midcingulate cortex in aggressive socially isolated mice
15:30 -15:40	Olanrewaju Fatola (Nigeria)	Neuroanatomical adaptations in the ocular and visual systems of African tree squirrels, crucial for their arboreal lifestyle.
15:40- 15:50	Oumaima Moutayb (Morocco)	Morphological alterations of the pyramidal cells in the anterior cingulate cortex in Monosodium Iodoacetate-Induced Osteoarthritis in Mice
15:50 -16:00	Chiara Musillo (Italy)	Protective effects of prenatal Omega-3 supplementation on brain and behavioral changes in mouse offspring exposed to a maternal high-fat diet
16:00- 16:10	Edoardo Pisa (Italy)	Brain-specific and time-dependent KCNQ1 knockout leads to cognitive and metabolic alterations in mice
16:10 -16:20	Mohamed Amine Zkim (France)	Role of the glucose transporter GLUT3 in neuronal energy metabolism and somatosensory cortex cognitive functions
16:20 -16:30	Toluwalope Ajonijebu (South Africa)	Increased activation of glucagon-like peptide-1 signaling mediates maternal high-fat induced oxidative…
16:30 -16:40	Fatima Ezzahra KACIMI (Morocco)	Impact of Vitamin A Modulation on Biochemical and Behavioral Abnormalities in a Valproic Acid-Induced Autism Model in Wistar Rat Offspring
16:40 – 16:50	**General Discussion**	

17:15 _ | Closing Ceremony

17:45 | SONA General Assembly

Major Sections of the Proceedings

Section 1:

Section 2:

Section 3:

Section 4:

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The SONA 2025 Conference

Lectures

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Lectures

Opening Lecture

How GABAergic neurons shape cortical circuit assembly in health and disease

Rosa Cossart

Institute of Mediterranean Neurobiology (INMED), Development of Hippocampal Cognitive MAPS Lab, Aix-Marseille University, France

The early postnatal period is marked by structured patterns of spontaneous neural activity that are essential for cortical circuit assembly. GABAergic neurons play a central role in organizing these developmental transitions, first by contributing excitatory drive and later by sculpting inhibitory networks. In this talk, I will present recent findings using longitudinal calcium imaging to track the maturation of individual neurons in vivo. We reveal a sharp and conserved transition during the second postnatal week characterized by the emergence of somatic inhibition, sparsification of network activity, and the formation of highly connected GABAergic hub cells. I will also explore how these processes may be altered in neurodevelopmental disorders, focusing on evidence from a mouse model of autism. These findings suggest that GABAergic neurons serve as key integrators between environmental inputs and internal circuit refinement.

Lectures

Lecture 1

Stress across generations: Mechanisms for altering brain development

Tracy L. Bale

Department of Psychiatry Sex Differences Research
University of Colorado

Talk abstract

Parental lifetime experiences to perturbations such as stress, infection, malnutrition, and advanced age are linked with an increased risk for offspring disease, including a strong association with neuropsychiatric disorders. In somatic cells involved in germ cell maturation, lasting changes to cellular function and energy metabolism are found following cumulative stress or trauma. We have focused on identifying the causal biological mechanisms whereby information in the environment can be transmitted to germ cells and ultimately alter the course of embryo development, affecting the brain health of the next generation. In our preclinical animal studies, we examine causal roles for somatic-to-germline transmission of stress signals capable of altering the rate of fetal neurodevelopment via specific types of secreted nanoparticles, or extracellular vesicles (EVs). In the somatic cells along the male reproductive tract, we previously identified broad histone and transcriptomic alterations in mouse epididymal epithelial cells (EECs) in vivo and in vitro. Further, these cells produced significant changes in the secreted EV proteome cargo and when incubated with sperm, these EVs increase sperm motility via targeted enhancement of mitochondrial respiration. Following fertilization, these sperm produce embryos with faster rates of development, including for the brain. As adults, these mice have a reduced stress reactivity, altering their ability to homeostatically respond to a changing environment. Translationally, we have found similar effects of chronic perceived stress changes in human sperm motility and EV cargo, suggesting that stress has a significant effect on reproductive processes to alter the embryonic developmental trajectory. Together, these studies support a causal importance of EV sperm interactions in the intergenerational delivery of environmental signals critical to brain development.

Lectures

Lecture 2

Neuroadaptation to the desert biotope: An overview on the recent data of camel and goat

Khalid El Allali

Hassan II Agronomy and Veterinary Institute, Comparative Anatomy Unit (URAC CNRST 49) and Medecine and Surgical Unit of domestic animals, Rabat, Morocco.

Talk abstract

Circadian rhythms in Mammals are driven by a central circadian clock located in the suprachiasmatic nucleus of the hypothalamus. It is well documented that the molecular and physiological mechanisms of the circadian clock are synchronised by the Light Dark cycle but the role of other environmental cues such as changes of the ambient temperature (Ta) as a synchronizer of circadian rhythms remains very poorly studied. In this lecture, we will show how the circadian timing system can be modulated by ambient temperature in two diurnal desert mammals, the camel and the goat. Notably, the melatonin rhythm, a well-studied output of the circadian system, has been observed to be significantly synchronized by ambient temperature under specific conditions. Furthermore, we will present data demonstrating that these two desert species exhibit a particular sensitivity to Ta and dehydration under desert conditions and a specific thermoregulatory state of heterothermy when subjected to dehydration and heat stress. We have also characterized the circadian rhythm of sleep-wake and the activity and rumination in these species. The data show that camels are the least prolific sleepers, with a total sleep duration of only 1 hour and 42 minutes per night. In addition, dehydration in these desert animals induces a shift in the circadian rhythms of activity and sleep. Finally, subjecting dehydrated animals to heat stress has been observed to result in an adaptation characterized by an increase in daytime sleep and a shift towards nocturnal behavior.

Key words: desert, camel, goat, circadian-clock, zeitgeber, melatonin, activity, sleep, body temperature,

Lectures

Lecture 4

Impact of the traditional medicine in Cameroon

Elisabeth Ngo Bum[1,4],
Fleur Clarisse Moto Okomolo[2], Gwladys Ngoupaye Ntemkou[3], Jacqueline Stéphanie Njapdounke Kameni[4], Antoine Kandeda Kavaye[5]

1. Faculty of Science, University of Maroua, Cameroon
2. Higher Training College, University of Yaounde I, Cameroon 3Faculty of Science, University of Dschang, Cameroon
4. Faculty of Science, University of Ngaoundéré, Cameroon
5. Faculty of Science, University of Yaounde I, Cameroon

Talk abstract

Traditional medicine has been used for many centuries by communities as they are derived from longstanding practice and are natural. Its practices include the use of plants, animals, fungi, or other components of nature (rocks, minerals….). Many plant-based traditional medicines showed promising potential in efficacy due to their bioactive compounds. Traditional medicine occupies a central place in the health system in Cameroon, where it is widely practiced, particularly in rural areas. Its impact is multiple and manifests itself in several areas such as culture, the treatment of common diseases and its complementarity with conventional medicine. In addition, the landscape of the use of plant-based traditional medicine has been improved during and after the COVID-19 pandemic, giving more credit to the use of natural products. Despite these advantages, traditional medicine has limitations such as the lack of regulation and formal training of practitioners to guarantee the safety and effectiveness of the care provided. This presentation will show different studies on natural products issued from plant-based traditional medicine that have shown pharmacological potency.

Keywords: plant-based traditional medicine, natural product, bioactive compounds, Cameroon

Lectures

Lecture 5

Kainate Receptors: A Double-Edged Sword in Brain Health and Disease

Juan Lerma

Instituto de Neurociencias, CSIC-UMH.
San Juan de Alicante, 03550 Spain

Talk abstract

Kainate receptors (KARs) are essential components of the glutamatergic synapse, playing pivotal roles in both pre- and postsynaptic signaling. Proteins interacting with KAR subunits have been identified, and functional studies have demonstrated a dual signaling system in which KARs can signal through ion flux or by activating G-proteins. While they are crucial for normal brain function, including synaptic plasticity and network excitability, dysregulation of KARs has now been implicated in various neurological and psychiatric disorders. In this presentation, I will explore the multifaceted roles of KARs in health and disease. I will discuss recent findings demonstrating the involvement of KARs in organizing synaptic connections and regulating synaptic plasticity in the cerebellum, independent of their traditional ionotropic and metabotropic function. Furthermore, I will delve into the growing body of evidence linking genetic variations in KAR subunits to disorders such as autism, anxiety, and Down syndrome. These genetic alterations often lead to subtle changes in KAR expression levels, rather than affecting their biophysical properties. These subtle changes can have profound consequences for synaptic function, leading to impaired neurotransmission, unbalanced circuit operations, and cognitive deficits. Indeed, a single gene variation in the glutamatergic system results in behavioural symptomatology that may account for the behavioural abnormalities observed in DS, autism, and schizophrenia. By understanding the intricate mechanisms underlying KAR function and dysfunction, we are gaining valuable insights into the pathophysiology of neurological and psychiatric disorders.

Lectures

Lecture 5

Repulsive guidance molecule regulates glial and immune function under neurological diseases

Toshihide Yamashita

Department of Molecular Neuroscience, Graduate School of Medicine, Osaka University Graduate School of Frontier Biosciences, Osaka University

Laboratory of Molecular Neuroscience, WPI Immunology Frontier Research Center, Osaka University

Talk abstract

Repulsive guidance molecule-a (RGMa), which is a glycosylphosphatidylinositol-linked glycoprotein, is expressed in glial cells and immune cells. RGMa was previously recognized as the protein that regulates axon growth negatively in the adult central nervous system (CNS). Enhanced recovery of skilled forelimb movement, as well as neural rewiring, was observed after spinal cord injury (SCI) in adult macaque monkeys following anti-RGMa antibody treatment. Based on the findings by the preclinical studies, the international clinical trials of humanized anti-RGMa monoclonal antibody (Unasnemab) for SCI is currently ongoing.

Furthermore, RGMa was shown to be involved in immune regulation. RGMa expressed in dendritic cells promotes activation of T cells, leading to deterioration of autoimmune encephalomyelitis. Further, under the condition of neuromyelitis optica (NMO), anti-RGMa antibody treatment significantly suppressed neutrophil infiltration, and decreased the expression of neutrophil chemoattractants. The multiple modes of action of anti-RGMa antibody may explain the potent effects on the neurodegenerative and neuroimmune diseases, as well as the CNS injuries. The clinical trial of Unasnemab for HTLV-1-associated myelopathy is also ongoing.

We recently reported that RGMa regulates blood-brain barrier integrity and cell survival in the CNS. Intravenous, administration of anti-RGMa antibodies reduced the loss of tyrosine hydroxylase (TH)-positive neurons and accumulation of Iba1-positive microglia/macrophages in the substantia nigra (SN) in a mouse model of Parkinson's disease (PD). Selective expression of RGMa in TH-positive neurons in the SN induced neuronal loss/degeneration and inflammation, resulting in a progressive movement disorder. Increased RGMa expression upregulated pro-inflammatory cytokine expression in microglia. Our observations suggest that the upregulation of RGMa is associated with the PD pathology; furthermore, inhibitory RGMa antibodies are a potential therapeutic option.

Lectures

Lecture 6

How do we sleep?

William Wisden

FMedSci FRS, Dept Life Sciences & Interim Director, UK Dementia Research Institute
Imperial College London, SW7 2AZ, UK

Talk abstract

There is no consensus on if sleep is for the brain, body or both. But the difference in how we feel following disrupted sleep or having a good night of continuous sleep is striking. Understanding how and why we sleep will likely give insights into many aspects of health. In this talk I will outline our recent work on how the prefrontal cortex can signal to the hypothalamus to regulate sleep preparatory behaviours and sleep itself, and how other brain regions, including the ventral tegmental area, respond to psychosocial stress to induce beneficial sleep. I will also outline our work on examining the function of the glymphatic system, and whether clearance of molecules from the brain is enhanced during sleep or wakefulness.

Lectures

Lecture 7
African Neuroscience and Human Development Strategies

Wail Benjelloun
Mohammed V University, Rabat
wbenjelloun@gmail.com

Talk abstract

More than 3.4 billion persons around the world suffered from a neurological condition in 2021, placing these disabilities as the leading cause of ill health globally. 80% of those affected were in low and middle-income countries, with Africa disproportionately affected. Diseases such as epilepsy are two to three times more prevalent in Sub-Saharan Africa than in Europe, and around 50% of people who go to the emergency room in Africa have some sort of neurological complication. This differential susceptibility may be related to the stigma frequently associated with mental disorders which prevents timely treatment, and the prevalence of diseases having neurological consequences such as malaria and HIV/AIDS, in addition to unsanitary living conditions and insufficient health systems. The cost of these neurological diseases, both socially and economically, constitutes a heavy burden for the continent and leads to higher health expenditures and to decreased productivity of large sectors of the population.

The net consequence is a compromised human development. The approach to the treatment of these neurological disorders thus necessitates a multi-faceted strategy, in which the key elements involve improved education and research and better health services, combined with social support, public awareness, social activism and high ethical standards that protect citizens and ensure equitable access to treatment.

The IBRO – UNESCO commitment to African neuroscience in 1988, and IBRO's continued support since then, have contributed effectively, in coordination with SONA and the national associations, to laying the groundwork for significant advances in neuroscience education and research. Initiatives such as the IBRO high-level schools have trained a cadre of qualified specialists. Alongside, and certainly as important, has developed a greater government interest and more support for hospitals, and neuroscience education and research. These developments have been accompanied in parallel by African human development initiatives by universities and social action groups throughout the continent, to increase the awareness of the general public as well that of national decision makers. These human development initiatives include, but are not limited to, the public dissemination of information, destigmatization, neuroscience diplomacy, neuroscience economy, and equitable access to the benefits of neuroscience research. The work accomplished and the results presented here point to an expanding role for these initiatives in future.

Lectures

James Kimani Lecture

Turning toxins into treatment: The therapeutic potential of animal venoms in neurodegeneration

Ines ELBINI

Institut Pasteur de Tunis, Tunisia

Talk abstract

Background and Objectives: Neurodegenerative diseases pose significant health and societal challenges due to their progressive nature and the lack of curative treatments. Recent studies have highlighted animal venom as a unique source of bioactive compounds that can specifically target membrane receptors, such as ion channels and integrins, which play critical roles in cellular signaling and neuroprotection. This lecture aims to explore the therapeutic potential of purified biomolecules from animal venoms, focusing on their high affinity and specificity for neuronal receptors and their capacity to modulate neuroinflammatory pathways, protect neurons, and influence disease progression. Methods: Purified biomolecules derived from animal venoms were studied for their interactions with key molecular targets, including voltage- gated potassium channels and integrins. Experimental evidence was gathered from in vitro and in vivo models of Parkinson's disease to evaluate their effects on neural communication, oxidative stress, and neuroinflammation. Results: Venom-derived biomolecules demonstrated high specificity and affinity for ion channels and integrins, effectively modulating neuroinflammatory pathways and providing neuroprotection. In experimental models of Parkinson's disease, these compounds were found to stabilize neural communication, reduce oxidative stress, and inhibit neuroinflammation, offering promising evidence for their therapeutic potential. Discussion: The findings underscore the translational potential of venom- derived biomolecules in neurodegenerative disease treatment. These natural compounds leverage precise mechanisms of action, such as targeting voltage-gated potassium channels and regulating immune responses through integrins, to address critical aspects of disease pathology. Despite their promise, challenges remain in advancing these compounds from experimental models to clinical applications, including issues of safety, delivery, and scalability. Embracing venom-based therapeutics could open new avenues for innovative treatments, utilizing nature's molecular toolkit to meet the pressing needs of patients with neurodegenerative diseases.

Keywords: Neurodegenerative diseases, animal venom, bioactive compounds, Parkinson's disease.

Lectures

Special Lecture

Are there really non-psychedelic psychedelics? A comparison of lisuride and LSD

Scott Thompson

University of Colorado, School of Medicine, USA
scott.m.thompson@cuanschutz.edu

Talk abstract

Psychedelics provide rapid and persistent symptom improvements for many neuropsychiatric disorders, including depression. However, their implementation as a widespread clinical intervention may be hindered by the characteristic alterations in perception and consciousness they produce (i.e. the hallucinations). Psychedelics are potent agonists at many serotonin receptors (5HTRs). Specifically, 5HT2ARs mediate psychedelic-induced perceptual alterations and may also be essential to the therapeutic response. To harness the clinical benefits of psychedelics while lowering economic barriers, biased 5HT2AR agonists that do not elicit hallucinations are currently being commercially developed. The absence of a head twitch response (HTR) is a widely used preclinical assessment of non-psychedelic compounds. However, it is unknown whether the absence of a HTR is sufficient to identify truly non-hallucinogenic compounds. Here we report results comparing the effects of lysergic acid diethylamide (LSD) and lisuride, a partial 5HT2AR agonist that does not cause HTRs, in a variety of assays in mice. Lisuride (0.5mg/kg), but not LSD (0.1mg/kg), produced a profound impairment of motor behavior in the open field and on the rotarod. Lisuride and LSD both impaired performance in an ethologically relevant cognitive task, the puzzle box. Both motor and cognitive effects of lisuride persisted when 5HT2ARs were pharmacologically blocked with MDL100,907 (0.5mg/kg). Lisuride and LSD produced unique alterations in electroencephalograms recorded over the prefrontal cortex. Combined, these results suggest that although lisuride may not be acting as a traditional psychedelic, it does impact both motor and cognitive function. Further, robust preclinical phenotypic characterization beyond the HTR is important for identifying non-hallucinogenic compounds to move forward for clinical development.

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The SONA 2025 Conference

Symposia

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Symposia

Symposium 1

"Brain Circuits for Motor and Social Behaviors"

Organizer: **Abdeljabbar El Manira**

Karolinska Institute, Stockholm, Sweden
email: abdel.elmanira@ki.se

Abstract:

The integration of motor and social behaviors is vital for survival, enabling organisms to dynamically interact with their environment and social groups. Both behaviors depend on intricate neural circuits that seamlessly coordinate sensory, motor, and cognitive processes. This symposium will bring together leading experts to offer cutting-edge insights into how brain circuits drive motor and social behaviors, uncovering their underlying mechanisms and revealing the interconnectedness of these processes. This symposium will provide a comprehensive view of the neural circuits that drive motor and social behaviors, with each speaker contributing distinct yet complementary perspectives. It will highlight the interconnectedness of motor and social circuits, exploring how they influence each other and adapt to various contexts. By bringing together outstanding researchers in key areas of contemporary neuroscience, this symposium will address current challenges and define future directions for understanding how neural circuits orchestrate complex behaviors, with potential clinical implications.

Speakers

Number	Speaker	e-mail	Title of the communication
SP1_1	Silvia Arber	silvia.arber@unibas.ch	Generating movements with brainstem circuits
SP1_2	Rui Costa	ruimcosta@gmail.com	Executing, reinforcing and refining actions
SP1_3	Nirao Shah	nirao@stanford.edu	Neural circuit architecture for a social behavior
SP1_4	Ole Kiehn	ole.kiehn@sund.ku.dk	Motor Circuits Prioritizing Safety-Seeking

Symposia

SP1_1

Title

Generating movements with brainstem circuits

Authors

Silvia Arber, Antonio Falasconi, Harsh kanodia, Irene Pallucchi, Laura VrijBloed and Haohao Wu, Biozentrum University of Basel, Switzerland

Abstract

Movement is the behavioral output of the nervous system. This talk will focus on recent work elucidating the organization and function of neuronal circuits central to the regulation of distinct forms of body movements, with a focus on skilled forelimb movements. It will show that dedicated circuit modules in different regions of the brainstem and their interactions within the motor system play key roles in the generation of diverse movements encompassing the overall behavior. Keywords: Motor behavior, brainstem, neuronal circuits.

SP1_2

Title

Executing, reinforcing and refining actions

Author

Rui Costa Allen, Institute for Brain Science, Seattle, USA

Abstract

The ability of animals to build individual repertoires based on the consequences of their actions is fascinating, and essential for survival. Understanding this process requires mechanistic insight into how self-paced actions are initiated, how they can be selected/initiated again, and how feedback can refine their execution and organization. We use behavioral, genetic, electrophysiological, and optical approaches to gain this mechanistic insight. The combination of these approaches allowed us to uncover that dopaminergic neurons are transiently active before self-paced movement initiation. This activity is not action-specific and modulates both the probability of initiation and the vigor of future movements, but does not affect ongoing movement. Dopamine is supposed to have opposite effects on downstream striatal direct and indirect pathways. Contrary to what is classically postulated, we found that both striatal direct and indirect pathways are active during movement initiation. The activity in both pathways is action-specific and has complementary but different roles in movement, which are enabled by specific basal ganglia output circuits. Input from cortex seems to be critical to organize striatal activity, and cortico-striatal plasticity is necessary to select, reinforce and refine the specific neural and behavioral patterns that lead to desirable outcomes. These data invite new models on the mechanisms underlying self-paced movement initiation, and motor dysfunction in Parkinson's disease. They also suggest that cortico-basal ganglia circuits play a generic role in learning to reinforce and refine task-relevant neural activity and behavioral patterns.

SP1_3

Title

Neural circuit architecture for a social behavior

Author

Nirao Shah, Department of Psychiatry and Behavioral Sciences and Department of Neurobiology, Stanford University, USA.

Abstract

Mating behavior is fundamental to survival and propagation of a species. Despite its centrality to reproductive success, how the mammalian brain generates this primal behavior has been elusive. We have employed recent advances in deep sequencing and brain mapping in mice to define a neural circuit that transforms sensory information into motor components of mating. This neural pathway spans multiple synapses, and it encodes the key elements of male sexual behavior: it is developmentally wired, it recognizes potential mates, it is male-specific, and it governs mating displays, libido, and the hedonic aspects of this behavior. I will describe these and other unpublished findings from my group.

SP1_4

Title

Motor Circuits Prioritizing Safety-Seeking

Author

Ole Kiehn Department of Neuroscience, Faculty of Health and Medical Sciences, University of Copenhagen, Denmark

Abstract

Animals continuously adapt their behavior to balance survival and fulfilling essential needs. This balancing act involves prioritization of safety over the pursuit of other needs and involves ambulatory movement towards desired objects, amid a constant conflict with need for safety. Initiating ambulatory movement is crucial not only for approaching targets but also for shifting priorities to ensure safety when necessary. The specific deep brain circuits that regulate safety-seeking behaviors in conjunction with motor circuits are not well understood. In this talk I will focus on work that has identified neuronal hypothalamic circuits that bridge with brainstem circuits and eventually spinal executive motor circuits to implement safety seeking. The lecture will report the finding of a distinct glutamatergic neuron population in the lateral hypothalamus (LHA) that targets locomotor initiating circuits in the pedunculopontine nucleus. This LHA-PPN orchestrates context-dependent locomotion and governs the prioritization of safety over essential needs. This circuit may be trigger both intrinsically and by external cues. Our study reveals a crucial link between neuronal circuits and adaptive actions. The findings

demonstrate how brain circuits orchestrate context-dependent locomotion, bypassing cortical or basal ganglia inputs to the brainstem.

MNS-sponsored Symposium 2

"Serotonergic Modulation and Neurochemical Pathways: Implications for Antidepressant Efficacy, Epilepsy, and Neurotoxicity"

Organizer: **De Deurwaerdere** & **Bamidele Victor Owoyele**

University of Bordeaux ; CNRS UMR 5287 ;
2 rue du Docteur Hoffmann Martinot, 33076
Bordeaux Cedex; France
email: pdeurwaer@gmail.com; owoyele@unilorin.edu.ng

Abstract:

This symposium will showcase research from young researchers and leading experts exploring the role of serotonergic receptors in various neurological and psychiatric conditions, including depression, epilepsy, and the impact of neurotoxins on brain function.

1. Soukaina Es-Safi (University of Bordeaux) will discuss the rapid antidepressant efficacy of 5-HT4 receptors, focusing on the neuro-plasticity mechanisms that could accelerate the onset of antidepressant action. Her work, conducted as part of her PhD, emphasizes novel therapeutic targets for rapid-acting antidepressant action.
2. Sabrine Ben Slimen (University of Sfax) will present findings on how pesticides, individually or in combination, affect brain monoamine function in rats. Her research addresses critical concerns about environmental toxins and their impact on neuropsychiatric health, using rodent models to explore the underlying neurochemical disruptions.
3. Patrick Oluwole Abolarin: (University of Ilorin/Chrisland University) will present his findings on how tannic acid administration influenced the brain serotonin availability and depressive like behaviour in rats. His work, which was conducted as part of his recently defended Ph.D. thesis identify a pivotal role for interventions using tannic acid in glyphosate-based pesticide induced neurotoxicity and spread awareness on the toxicity pesticides.
4. Professor Philippe De Deurwaerdaere (University of Bordeaux) will lead a discussion on the neurochemical responses of brain-wide neurotransmitter systems to psychedelic drugs and the role of 5-HT2A receptor antagonism. His extensive work in neurotransmitter analysis bridges basic and clinical research, offering insight into potential psychiatric applications of psychedelics.
5. Professor Giuseppe Di Giovanni (University of Magna Graecia) will present his research on the modulation of GABAA receptor tonic inhibition and GAT1 function by 5-HT2A receptors, with a focus on absence epilepsy. His groundbreaking studies propose new therapeutic targets for epilepsy and related neuropsychiatric comorbidities.

This symposium promises to deepen our understanding of serotonergic modulation across different neurological disorders and offer novel perspectives on therapeutic interventions.

Symposia

Speakers

Number	Speaker	e-mail	Title of the communication
SP2_1	Soukaina Es-Safi	soukaina.es-safi@u-bordeaux.fr	5-HT4 receptors of the medial Prefrontal Cortex impair the induction of a long-term potentiation within the Dentate Gyrus
SP2_2	Sabrine Ben Slimen	sabrinebenslimene1@gmail.com	Effect of pesticides alone or combined on monoamines and behavior in rats
SP2_3	Patrick Oluwole Abolarin	wolexpatrick@gmail.com	Reversibility of Glyphosate-induced serotonin depletion and depressive-like behaviour in mice: Tannic acid intervention study
SP2_4	Philippe De Deurwaerdere	pdeurwaer@gmail.com	Brain wide neurochemical analysis of neurotransmitters in response to psychedelic drugs and 5-HT2A receptor antagonist

Symposia

SP2_1

Title

5-HT4 receptors of the medial Prefrontal Cortex impair the induction of a long-term potentiation within the Dentate Gyrus

Authors

Soukaina Es-Safi; Guillaume LUCAS (University of Bordeaux)

Abstract

In order to determine to what extent, the rapid antidepressant (AD)-like effects related to (i) increased synaptic plasticity or (ii) the administration of 5-HT4 agonists share common mechanisms, we aimed to investigate the role of 5-HT4 receptors (5-HT4-R) in the modulation of synaptic plasticity within the dentate gyrus (DG). The first step was to dissociate the global influence of 5-HT4 agonists from their direct effects on DG neurons. Indeed, these compounds are known to increase central 5-HT activity through long-loop feedback originating within the medial prefrontal cortex (mPFC). To this end, we used small hairpin RNAs (shRNA) expressed by lentiviruses (LV) to inhibit the expression of 5-HT4 receptors specifically in the mPFC. Then, we assessed the effect of the 5-HT4-R selective agonist prucalopride on the long-term potentiation (LTP) induced within the DG of anesthetized rats by high-frequency stimulation (HFS) of its main afference, the perforant pathway. In animals non injected with LV, an acute treatment with prucalopride (5 mg/kg, i.p.) reduced the success rate of LTP compared to the vehicle (field potentials: 54 vs 82%, population spike: 55 vs 100%), without any significant effect on LTP amplitude. The continuous treatment for 3 days with prucalopride (5 mg/kg, s.c.) further reduced the success rate of LTP to 27% for the field potentials, without changing its amplitude. For spike population, the success rate of LTP was the same as in acute conditions, but with a significant decrease of its amplitude. In LV-treated rats, acute prucalopride induced a successful LTP in 91% and 86 % of the cases for field potentials and spike population, respectively (no change in LTP amplitudes). Experiments addressing the effects of a 3-day treatment in LV-injected animals are currently underway. Together, our results support an important role for 5-HT4-R in the modulation of hippocampal synaptic plasticity. It appears that 5-HT4-R located in the mPFC hinder the induction of LTP, which is restored to normal levels after their knockdown.

Symposia

SP2_

Title

Effect of pesticides alone or combined on monoamines and behavior in rats

Authors

Sabrine Ben Slimen, Philippe De Deurwaerdere (University of Bordeaux),
HAMADI Fetoui (University of Sfax)

Abstract

Synthetic pyrethroids are widely used as plant protection agents in agriculture. However, several epidemiological studies have suggested that these chemicals are considered potential contributors to neurodegenerative diseases. Given the critical role of the brain's monoaminergic systems in regulating brain functions, pyrethroids may impact neurochemistry. Our research aimed to investigate the effects of the exposure to 2 types of pyrethroids and their combination on the serotonergic and dopaminergic systems and the related cognitive and behavioral outcomes in Wistar rats, with a particular emphasis on gender-specific impacts. Rats were divided into four groups: control, permethrin group (34 mg/kg), deltamethrin group (1.35 mg/kg), and a mixture group receiving both pyrethroids during the lactation period (PND7 to PND21). Behavioral assessments were conducted at PND150, and monoamine levels in various brain regions were quantified using HPLC-ECD. Our findings revealed that early exposure to synthetic pyrethroids leads to alterations in brain monoaminergic systems, which were associated with the disruption of cognitive behavior in a sex-dependent manner in the rats.

KEYWORDS: Lactation period, permethrin, deltamethrin, mixture, monoamines, behavior, HPLC-ECD

SP2_

Title

Reversibility of Glyphosate-induced serotonin depletion and depressive-like behaviour in mice: Tannic acid intervention study

Authors

Patrick Oluwole Abolarin, Bamidele Victor Owoyele, Department of Physiology, Neuroscience and Pain Laboratory, College of Health Sciences, University of Ilorin, Ilorin, Kwara state, Nigeria

Abstract

Glyphosate (Gly) is a widely used herbicide whose exposure has been linked to serotonin depletion, depressive-like behaviour, and associated memory decline in mice, raising concerns about human and animal health risks. The underlying mechanisms of Gly action involve oxidative stress and neuroinflammation. Despite the identified deleterious effects of Gly, research on effective interventions against Gly-induced neurotoxicity is limited. Tannic acid (TA), a potent polyphenolic antioxidant and anti-inflammatory agent, may therefore offer a protective intervention by reversing glyphosate-induced neurotoxicity. This study explored the neuroprotective effect of TA in counteracting Gly-induced depressive-like behaviour and memory decline in mice. Male Swiss mice were randomly divided into six groups (n=8): control (distilled water 0.2 ml/kg), Gly (Gly 500 mg/kg), Pre-TA + Gly (TA 50 mg/kg pre-treatment, afterwards Gly-administered), TA + Gly (TA 50 mg/kg and Gly co-administered), Pre-AA + Gly (ascorbic acid (AA) 100 mg/kg pre-treatment, afterwards Gly-administered), and AA + Gly (AA 100 mg/kg and Gly co-administered). Six weeks post-treatments, forced swim, tail suspension, sucrose spray, and open field tests were performed for depressive-like behaviours, while Y-maze and Barnes maze tests were used to evaluate memory function. Animals were subsequently euthanised for brain biochemical and histological evaluations. Results revealed that TA treatment prevented the manifestation of depressive-like behaviour and memory decline as evidenced through behavioural tests. Molecular analysis revealed decreased neuroinflammation (TNF-α, IL-1β, and IL-6) and increased serotonin concentration and neurotrophic factor concentration, BDNF, in the prefrontal cortex and the hippocampus following TA treatment. Immunohistochemistry analysis revealed astrogliosis in the prefrontal cortex and the hippocampus following Gly exposure, suggesting enhanced neuroinflammation, while TA caused a reversal effect. Our findings demonstrate that TA can effectively mitigate glyphosate-induced neurotoxicity, providing preclinical evidence for potential therapeutic interventions. These results have significant implications for understanding the mechanisms underlying glyphosate-induced neurotoxicity and developing strategies to protect human health.

Keywords: glyphosate, serotonin, depressive-like behaviour, Tannic acid, neurotoxicity.

SP2_4

Title

Brain wide neurochemical analysis of neurotransmitters in response to psychedelic drugs and 5-HT2A receptor antagonist

Authors

Philippe De Deurwaerdere, Jasmine Jade Butler, Margherita Virgili
(university of Bordeaux)

Abstract

The serotonergic receptor subtype 2A (5-HT2AR) is an intriguing pharmacological target because it binds classical psychedelics, which are 5-HT2AR agonists, as well as antipsychotic drugs that antagonize 5-HT2AR function. The widespread influence of these compounds on the activity of neurotransmitter systems across the brain remains unknown. In mice, we studied the effect of non-selective 5-HT2AR agonist TCB-2 (0.3, 3, and 10 mg/kg) alone or in combination with 5-HT2AR antagonist MDL100,907 (0.2 mg/kg) with TCB-2 (3 mg/kg) on the tissue level of neurotransmitters [GABA, glutamate, noradrenaline (NA), dopamine (DA), serotonin (5-HT) and their metabolites] 1-h after agonist administration. Post-mortem, tissue content was measured by HPLC in 28 brain regions belonging to various neurobiological networks. Ten minutes before the sacrifice, we placed the animals on a textured floor to promote forced investigatory behaviour after having measured the head twitches from 20 to 50 minutes to determine the efficacy of the treatments. Quantitatively, TCB-2 dose-dependently decreased 5-HT turnover (usually an increase in 5-HT) in all brain regions. It reduced the ratio 3-methoxytyramine/DA in the striatum, and enhanced markers of the DA system in a few cortices (cingulate, somatosensorial) and NA in the cingulate cortex and the ventral hippocampus. Despite the ability of MDL100,907 to prevent TCB-2-induced head twitches in these animals, the decrease in 5-HT turnover induced by TCB-2 was generally insensitive to MDL100,907. However, MDL100,907 blocked TCB-2-induced 5-HT increase in the cingulate and auditory cortices and the ventral hippocampus. It also blocked some DA and NA effects, notably in the anterior cingulate cortex. TCB-2 alone or combined with MDL-100,907 did not modify amino acid tissue contents. Qualitatively, an impressive number of organized correlations assessed by Pearson's correlations was found for all neurotransmitters between brain regions in vehicle-treated animals. TCB-2 dramatically decreased the correlative links. MDL100,907 also reduced the correlative links. The disruptive effect of TCB-2 was partially counteracted by MDL100907 for 5-HT, glutamate, and GABA. Irrespective of its questionable action beyond 5-HT2AR, the data indicate that TCB-2 dramatically alters the activity of 5-HT neurons in the brain and disrupts the correlative links between brain regions for all neurotransmitters.

Symposia

Symposium 3

" Emerging effectors in neurodegeneration: from preclinical to clinical models"

Organizer: **Ines Elbini** & **Rym Benkhalifa**

Institut Pasteur de Tunis, Tunisia
email: ines.bini@pasteur.tn & rymbkh@gmail.com

Abstract:

This symposium explores the impact of emerging effectors such as ion channels, receptors, and cytokines on neurodegeneration within preclinical and clinical models. A particular emphasis will be placed on the underlying pathophysiological mechanisms driving these processes. The symposium will be chaired by a panel of scientists from the Institut Pasteur of Tunis (IPT), Tunisia, and UM6P, Morocco. Dr. Rym Benkhalifa will elucidate the role of Kv3.1 potassium channels in specific pathophysiological conditions and recount the process of investigating this channel within the context of her research team's work. Dr. Meriam Belghith will tackle the practical challenges in understanding the molecular mechanisms of neuroinflammation, a critical aspect of neurodegeneration. Miss Nour-elhouda Neili will present her research on the role of ion channels in Parkinson's disease using induced pluripotent stem cell (iPSC)-derived dopaminergic neurons as a model system. Her work aims to elucidate how dysregulation of these channels contributes to neuronal dysfunction, providing insights into potential therapeutic interventions. Finally, Dr. Mohamed Taha Moutaoufik from UM6P will explore systems biology approaches to understand mitochondrial functions and their implications in neurodegenerative diseases, with a particular focus on Parkinson's disease. Youssef Anouar "will describe the discovery of a new enzyme belonging to the selenoprotein family which plays an essential role in the protection of dopaminergic neurons and how his work led to the development of a new therapeutic candidate for this disease.
Together, these presentations will offer a comprehensive overview of emerging effectors that could serve as potential targets for future therapeutic strategies, utilizing either natural or chemical molecules. This symposium aims to provide fresh insights into neurodegenerative disease management by highlighting innovative approaches to counteract the pathophysiological mechanisms at play.

Speakers

Number	Speaker	e-mail	Title of the communication
SP3_1	Rym Benkhalifa	rymbkh@gmail.com	When the secondary role plays its full part: the story of the Kv3.1 channel in neuropathologies.
SP3_2	Meriam Belghith	belghith.meriam@gmail.com	Adaptive immune cells in the cerebrospinal fluid of neuroinflammatory disorders: a comparative study between neuro-Behçet and Multiple Sclerosis
SP3_3	Nour-elhouda Neili	neilinourelhouda1@gmail.com	Targeting Ion Channels in Parkinson's Disease: Insights from iPSC-Derived Dopaminergic Neurons
SP3_4	Mohamed Moutaoufik	mohamed.moutaoufik@um6p.ma	Decoding Mitochondrial Interactomes: Insights into Neurodegenerative Disease Mechanisms
SP3_5	Youssef Anouar	Youssef.Anouar@univ-rouen.fr	Role of a selenoprotein in the pathophysiology of PD: therapeutic application after intranasal administration

SP3_1

Title

When the secondary role plays its full part: the story of the Kv3.1 channel in neuropathologies

Authors

Rym Benkhalifa, Amani Cheikh, Sonia Maatoug and Hager Tabka

Lab. Biomolecules, venom and theranostic applications, Pasteur Institute of Tunis, University Tunis ElManar, Tunisia

Abstract

Introduction and objectives: Kv3.1 channels are of critical importance to the functioning of the nervous system. These channels, which are abundantly expressed in fast-firing neurons, as inhibitory GABAergic interneurons and cerebellar neurons, are essential for the rapid repolarization of action potentials. Neurological disorders are marked by neuronal loss leading to cognitive and motor dysfunctions. Recent research highlights the involvement of oligodendrocytes in the pathogenesis of these disorders due to their rising susceptibility to damage. This vulnerability might be exacerbated by the dysregulation of voltage-gated potassium channels like Kv3.1, impacting cell proliferation, migration, and axon myelination. Our research has focused on examining the various pathophysiological implications of this channel, including K+ hemostasis, neurodegeneration in Alzheimer's disease cell models, and its relationship with serotonergic mechanisms. We finally used an active scorpion venom fraction to identify a molecule that modulates Kv3.1 currents (Cheikh et al., 2013). Results: Our research findings suggest that the lipid environment affects Kv3.1b channel expression and/or functionality and that subsequent rupture of K? homeostasis is associated with oligodendrocytes and microglial cell damage. Actually, a positive correlation seems to exist between Kv3.1b or the intracellular K? concentration, loss of transmembrane mitochondrial potential, and increased plasma membrane permeability in these cells (Bezine et al., 2018) We have also confirmed that Kv3.1b channel is significantly expressed in 1C11 cell line, as an in vitro model for serotonergic release, and that fluoxetine affects Kv3.1b expression but increases cell proliferation. Finally, we highlighted its role during cell differentiation (Tabka etal., 2020). Moreover, we used a combined optimization through advanced biochemical purification and patch-clamp screening steps to characterize, for the first time, the peptide in Aah venom active on Kv3.1 channels (Maatoug et al., 2021). Discussion: Kv3.1's frequently secondary or indirect contribution to pathophysiological mechanisms does not in any way diminish its crucial role. Targeting Kv3.1 inhibition selectively could yield treatments for diseases involving altered neuronal excitability, such as epilepsy, ataxia, or auditory processing disorders. While these findings provide promising perspectives, advances in pharmacology and personalized medicine will likely to make Kv3.1 modulation a viable therapeutic strategy in the near future.

Symposia

SP3_2

Title

Adaptive immune cells in the cerebrospinal fluid of neuroinflammatory disorders: The control of cytokine production

Authors

Meriam Belghith[1], Rafika Ben Laamari[1], Olfa Maghrebi[3], Zakaria Saied[2], Samia Ben Sassi[2], and Mohamed Ridha Barbouche[4].

1-Laboratory of Transmission, Control and Immunobiology of Infections. (LTCII), Institut Pasteur de Tunis, Tunisia. 2-Mongi Ben Hamida National Institute of Neurology, Tunis, Tunisia. 3-University of Florence, Firenze, Italy. 4-Department of Microbiology, Immunology and Infectious Diseases, Manama, Bahrain.

Abstract

Background and objectives: Multiple Sclerosis (MS) is a demyelinating and neurodegenerative disease of the central nervous system (CNS). Neuroinflammation induced by genetic variations in CNS cells or by peripheral immune cells plays a crucial role in the process of neurodegeneration. This inflammation involves a different panel of cytokines and cellular players of innate and adaptive immunity. In order to study these players in the CNS of MS patients, we compared them with patients suffering from a chronic relapsing multisystem inflammatory disease which can affect the CNS, the neurological manifestation of Behçet disease (NBD). Methods: This study included blood and cerebrospinal fluid (CSF) samples from 35 MS patients and 25 NBD patients from Institute of Neurology of Tunis. We measured and compared cytokine signatures related to Th1, Th2, Th17, Th9, Th22, T regulatory and inflammatory response. A broad panel of selected genes was compared between MS, NBD, and non-inflammatory neurological disorders. T populations were studied by qRT-PCR, ELISA and multiplexes beads. To reach this aim, bivariate and multivariate analysis were applied. Results: In initial CSF samples, ROR-γt, IL-17A and IFN-γ were significantly elevated in patients compared to controls. This group displayed activation of the Th1/Th2 and Th17 axes in the CSF. The Principal Analysis Component (PCA) highlighted distinct profiles betweenNBD, MS, and controls. Parameters related to cellular activation and inflammatory cytokines within the CSF clearly differentiate between the two inflammatory diseases and the controls. Moreover, foxp3 in the blood along with IL-4, IL-10, and IL-17 expressions were the parameters that are the main contributor to the segregation between MS and NBD clustering. We proceeded to ROC analysis in order to identify the most distinctive parameters between both disorders. The latter analysis suggested that IL-17, CD73 in the blood as well as IL-1γ and IL-10 in the CSF were the most discriminating parameters between MS and NBD. Discussion: The study of inflammatory response in the CNS of MS patients showed increased expression of IL-1β. Moreover, a comprehensive analysis of multiple cellular markers by combined multi-dimensional analysis suggests distinct mechanisms governing the pathophysiology of these two neuro-inflammatory disorders.

Symposia

SP3_3

Title

Targeting Ion Channels in Parkinson's Disease: Insights from iPSC-Derived Dopaminergic Neurons

Authors

Nour-elhouda Neili[1], Razan Sheta[2,3], Abid Oueslati[2,3], Ines ELBini[1]

1 Laboratory of Biomolecules, Venoms and Theranostic Applications (LR20IPT01), Pasteur Institute of Tunis, University of Tunis, El Manar, Tunis, Tunisia. 2 CHU de Québec Research Center, Axe Neurosciences, Quebec City, Canada 3 Department of Molecular Medicine, Faculty of Medicine, Université Laval, Quebec City, Canada.

Abstract

Parkinson's Disease (PD) is a widespread neurodegenerative disorder affecting over 10 million individuals worldwide, leading to significant disability. Current treatments alleviate symptoms but do not address disease progression or target underlying mechanisms like protein misfolding. Indeed, a hallmark of PD is the accumulation of α-synuclein (α-syn) aggregates and Lewy bodies, which contribute to neuronal dysfunction and degeneration, highlighting α-syn as a key therapeutic target. Building on evidence suggesting the toxic nature of extracellular α-syn aggregates binding to cell surface receptors, this study explores the role of ion channels, specifically the potassium channel Kv1.3, in the formation of pathological α-syn aggregates. Kv1.3 is upregulated in experimental PD models and post-mortem PD brain tissue, making it a promising therapeutic target. We investigated the impact of selective Kv1.3 blockers, the chemical compound PAP-1, on α-syn aggregation. Utilizing a novel PD cell model with light-induced α-syn aggregation, we demonstrated, via confocal microscopy, that these compounds significantly reduced α-syn aggregates in both neuron-like N2A cells and induced pluripotent stem cell (iPSC)-derived dopaminergic neurons (iDA). Although additional studies are needed to confirm these results, our findings highlight Kv1.3 blockade as a potential strategy for targeting α-syn aggregation in PD. This approach may pave the way for novel therapies that address the underlying pathology of PD and improve patient outcomes. Keywords: Parkinson's Disease, α-synuclein, protein aggregation, ion channels, iPSC-Derived Dopaminergic Neurons.

SP3_4

Title

Mitochondrial Interactomes and Neurodegenerative Disease Mechanisms

Authors

Mohamed Taha Moutaoufik,

Institute of Biological Sciences (ISSB), Faculty of Medical Sciences (FMS), Mohammed VI Polytechnic University (UM6P), Ben Guerir, Morocco

Abstract

Mitochondria are essential organelles involved in various cellular processes, and their dysfunction is linked to numerous human diseases, including neurodegenerative disorders. Advancing our understanding of these conditions necessitates a comprehensive mapping of protein-protein interaction networks that encompass both mitochondrial and non-mitochondrial proteins. However, these networks remain only partially characterized. In this presentation, I will discuss our research on the mitochondrial interactome, emphasizing its dynamic reorganization during neuronal differentiation. Our studies have revealed significant rewiring of mitochondrial protein interactions, shedding light on the mechanisms that govern mitochondrial functions in neuronal development and the maintenance of cellular health. Additionally, I will present our findings on the role of the CHCHD2 gene in Parkinson's disease. Our research has demonstrated that CHCHD2 mutant mice exhibit mitochondrial protein accumulation and impaired energy metabolism, providing valuable insights into the molecular mechanisms of Parkinson's disease.

SP3_05

Title

Role of a selenoprotein in the pathophysiology of PD: therapeutic application after intranasal administration

Authors

Youssef Anouar, Univ. Rouen Normandy, INSERM, Nordic, U1239, F-76000 Rouen France

Abstract

Oxidative stress is central to the pathogenesis of different diseases affecting the central nervous system, but therapeutic strategies targeting this pathological process have been difficult to design. We have previously demonstrated that selenoprotein T (SELENOT), a new thioredoxin-like protein of the ER, is essential for embryonic development and dopaminergic neuron survival and function. SELENOT exerts its neuroprotective activity by reducing oxidative stress and improving tyrosine hydroxylase activity. In an animal model of PD, targeted SELENOT gene disruption in the brain provoked rapid and severe parkinsonian-like motor deficits. Based on these findings, we designed a 10-amino acid peptide named PSELT as a potential mimic of SELENOT catalytic site to test its activity in PD animal models. PSELT proved to be efficient in protecting dopaminergic neurons in vitro and in vivo and could improve motor skills in animal models of PD. Transcriptomics studies revealed that PSELT acts through an original epigenetic regulatory mechanism. These results uncover the role of SELENOT as a neuroprotective enzyme and indicate that PSELT is a new therapeutic candidate for Parkinson's disease and other neurodegenerative disorders associated with oxidative stress.

Symposia

Symposium 4

"Molecular targets of addictive drugs and associated neuropsychiatric disorders"

Organizer: **Mohamed Kabbaj**
1115 W Call Street-Tallahassee 32312 USA
email: mkabbaj@fsu.edu

Abstract:

For most abused drugs, there are still no effective treatments. In this symposium, the presenters will describe some of the molecular mechanisms behind novel targeted therapies for substance abuse as well as associated neuropsychiatric disorders in humans and animal models. Specifically, Dr. Nicoletti will start the session by discussing the role of metabotropic glutamate receptors in Alcohol Use Disorders (AUD) and psychostimulants addiction. Dr. Maccari will then discuss how prenatal stress affects alcohol consumption and sleep in male and female rats and discuss the role of the glutamate receptor mGlu1 in mediating some of the effects of stress and alcohol. Afterward, Dr. Naasila will present his recent findings comparing the effects of psychedelics like psilocybin, LSD, and ketamine in treating Alcohol Use Disorders (AUD) and addiction-related behavior. Dr. Kabbaj will then present a study exploring some of the mechanisms behind ketamine's abuse potential with a focus on the role of medium spiny neurons (MSNs) in the nucleus accumbens. Finally, Dr Matrisciano will show some recent human studies where they used ATAC-seq to examine epigenetic changes in the prefrontal cortex of individuals with AUD. His work points to a link between neuroinflammation and epigenetic regulation in alcohol addiction. Overall, the 5 presenters will shed some light on the complex interplay between stress, sex differences, neurotransmitter systems, and genetic/epigenetic factors in the context of alcohol and substance use disorders.

Speakers

Number	Speaker	e-mail	Title of the communication
SP4_1	Jun-Xu Li	junxuli@buffalo.edu	TAAR1 agonists for opioid use disorder (OUD): a new approach to mitigate OUD while sparing opioid analgesia
SP4_2	Stefania Maccari	stefania.maccari@univ-lille.fr	Prenatal stress effects on alcohol drinking: role of oxytocin and metabotropic receptors
SP4_3	Mickael Naassila	mickael.naassila@u-picardie.fr	Psychedelics as treatment of alcohol use disorders: Insights from preclinical models and comparison of the psychedelics psilocybin, LSD and Ketamine.
SP4_4	Mohamed Kabbaj	mkabbaj@fsu.edu	Role of medium spiny neurons in ketamine reinstatement

Symposia

SP4_1

Title

TAAR1 agonists for opioid use disorder (OUD): a new approach to mitigate OUD while sparing opioid analgesia

Authors

Jun-Xu Li, Jianfeng Liu, Ruyan Wu, Robert Seaman Jr. Yanan Zhang, Jun-Xu Li Department of Pharmacology and Toxicology, University at Buffalo, Buffalo, NY, USA 14203

Abstract

Opioid use disorder (OUD) affects millions globally. In the United States, opioid abuse has reached epidemic level with over 100,000 Americans died from opioid overdose in 2023 alone. Currently available treatments of OUD are only partially effective, and new and effective approaches for treating OUD are in dire need. Trace amine-associated receptor 1 (TAAR1) is an emerging G-protein coupled receptor that can directly modulate the central dopaminergic activity, suggesting that pharmacological agonists of TAAR1 may be able to modulate opioid-related effects. In this study, we examined the effects of a TAAR1 agonist RO5263397 on abuse-related effects of opioids. RO5263397 attenuated the expression of morphine-induced behavioral sensitization in wildtype but not TAAR1 receptor knockout mice. RO5263397 shifted the dose-effect curve of morphine self-administration downward and reduced the breakpoint in a progressive ratio schedule of reinforcement but did not affect food self-administration in rats. RO5263397 decreased the cue- and drug-induced reinstatement of morphine-seeking behavior in rats. However, RO5263397 did not affect the analgesic effects of morphine in an acute nociception model in mice and a chronic pain model in rats. These results indicated that TAAR1 agonists selectively attenuated the reinforcing, but not antinociceptive effects of morphine, suggesting that selective TAAR1 agonists might be useful to combat opioid addiction, while sparing their analgesic effects.

SP4_2

Title

Prenatal stress effects on alcohol drinking: role of oxytocin and metabotropic receptors

Authors

Stefania Maccari, University of Lille, France

Abstract

The animal model of maternal stress (PRS) induces chronic stress and sleep abnormalities that are associated with an increased vulnerability to psychostimulants and natural rewards. We have previously demonstrated that PRS increases the preference for ethanol in heavy-drinking females following severe stress. Conversely, in PRS males, activation of the stress axis is reduced in response to alcohol consumption. We investigated the link between early-life stress and alcohol consumption/sensitivity in both sexes, as well as the strategies implemented in the sleep-wake cycle by exposing PRS rats to chronic intermittent alcohol consumption (20%) in a two-bottle choice paradigm during either adolescence or adulthood. Our findings reveal the following: 1) Adolescent PRS rats consume more alcohol than non-stressed controls, with a more pronounced effect observed in PRS females. 2) Adult females exhibit higher alcohol consumption than males. 3) Analysis of sleep architecture reveals increased REM sleep and sleep fragmentation in both male and female PRS animals compared to controls. 4) After two months of intermittent alcohol intake, PRS rats consume less alcohol than control rats that have been drinking alcohol. 5) Additionally, PRS groups experience a reduction in REM sleep compared to controls. 6) In adult PRS rats that consume water, we observed an increase in glutamate mGlu 1 receptor expression in the striatum compared to controls. This increase is more pronounced in male PRS rats than in PRS females following alcohol intake. In conclusion, our results support the evidence of an interplay between glutamate and early-life stress in alcohol dependence, which is sex-dependent.

Symposia

SP4_3

Title

Psychedelics as treatment of alcohol use disorders: insights from preclinical models and comparison of the different psychedelics psilocybin, LSD and ketamine

Authors

Mickael, Naassila, Université de Picardie Jules Verne

Abstract

The use of psychedelics to treat alcohol use disorders (AUDs) is very promising, but the mechanisms of action remain poorly understood. We combined behavioral, pharmacological and gene expression analyses to decipher the mechanisms of action of psilocybin, LSD and ketamine in different animal models of AUDs. Male Long Evans rats underwent chronic operant ethanol self-administration before testing the effect of intraperitoneal psilocybin or LSD or different enantiomers of ketamine. Psilocybin or LSD were also directly injected within the nucleus accumbens core or other brain region. Transcripts from the dopaminergic or neuroplasticity pathways were quantified in the nucleus accumbens and prefrontal cortex. Psilocybin or LSD significantly reduced (50%) ethanol self-administration when injected 4 hours before the session either intraperitoneally (1mg/kg) or directly within the left nucleus accumbens (0.15microg) but not the right nucleus accumbens or the left ventral tegmental area. The effect of intraperitoneal injection of psilocybin was prevented by intra left nucleus accumbens injection of 0.3microg of the 5-HT2AR antagonist ketanserin. In rats that self-administered ethanol but not in those self-administering saccharin, dopamine D2 receptor mRNAs were increased in both the nucleus accumbens and the prefrontal cortex by psilocybin, while D1R mRNA was increased only in the prefrontal cortex. Regarding ketamine enantiomers, we found specific effects depending on sex and depending on the enantiomers. As in humans, psilocybin reduced ethanol self-administration in rats through the 5-HT2AR within the left nucleus accumbens possibly through increased D2R expression. Our results open unexpected perspectives regarding the hemispheric lateralization of psychedelic effects.

SP4_4

Title

Role of medium spiny neurons in ketamine reinstatement

Authors

Mohamed Kabbaj, Florida State University College of Medicine

Abstract

With the growing number of clinical studies showing promising effects of repeated, low-dose ketamine treatment across various psychopathologies -including depression and drug addiction- it is critical to determine the potential addictive properties and their associated mechanisms in both sexes. Accordingly, the present work examined the abuse potential of repeated low to moderate doses of ketamine in male and female rats, and molecular profiles associated with the development of behavioral sensitization in D1 and D2 dopamine receptor-expressing medium spiny neurons (MSNs) of the nucleus accumbens (NAc). Here, following bilateral intra-NAc infusions of a Cre-inducible ribosomal tagged virus, locomotor activity was measured in adult Drd1a-iCre and Drd2-iCre transgenic male and female rats in either diestrus or proestrus following repeated administration of ketamine (0, 10, or 20 mg/kg, i.p.) to evaluate the development of locomotor sensitization. To examine enduring context- and cell-type-specific transcriptomic changes associated with sensitization to ketamine in NAc MSNs, tissue was collected four days after the final dose of ketamine in a drug-free state and RNAseq was performed on polyribosome-bound mRNA. Under this treatment protocol, female' but not male' rats developed sensitization to the locomotor-activating effects of ketamine at both doses examined. Further, proestrus female rats developed sensitization more rapidly to 10 mg/kg ketamine than those in diestrus. Accordingly, a greater effect of treatment was observed in the NAc of proestrus versus diestrus females at this dose, characterized by a greater number of differentially expressed genes primarily in D1 MSNs which were broadly related to synaptic plasticity, neurotransmission, DNA transcription, and signal transduction. Robust enrichment and clear separation of D1 versus D2 MSN transcriptomes support the cell-type specificity of the sensitization-associated changes observed. Taken together, these findings are consistent with earlier reports of sex-dependent sensitivity to behavioralsensitization to intermittent low-dose ketamine and provide novel evidence of associated cell-type-specific and estrous-cycle-dependent molecular profiles in female rats. Additional studies are warranted to further delineate the functional contributions of NAc D1 MSN populations to the abuse liability of low-dose ketamine in both sexes.

Symposia

SP4_5

Title

ATAC-seq analysis unveils epitranscriptomic alterations in the prefrontal cortex of alcohol use disorder and control subjects: potential link to neuroinflammation

Authors

Francisco Matrisciano, University of Illinois Chicago, Chicago, IL 60612, USA

Abstract

AUD is a disorder characterized by numerous factors, including inflammation in the brain, potentially associated with epigenetic mechanisms. However, the overall role of the epigenome AUD is underexplored. Assay for Transposase-Accessible Chromatin followed by high-throughput sequencing (ATAC-seq) unveils changes in open chromatin regions mediated by posttranslational modifications to histones and DNA. We analyzed ATAC-seq data obtained from the prefrontal cortex (PFC) of AUD subjects and identified open chromatin domains associated with genes of potential interest in inflammatory processes. Post-mortem PFC (BA 10) from AUD subjects (n=30) and controls (n=30) were obtained from the University of South Wales Tissue Center. Nuclei were isolated and tagmented using Illumina's Tn5 Transposase. Paired-end sequencing (150 nucleotides) of the libraries generated over 6.3 billion reads. Bioinformatics analysis was performed and identified numerous open and closed chromatin peaks. The largest changes were located more distal to transcription start sites. We validated significant peaks using chromatin immunoprecipitation (ChIP) assays with H3K27ac and H3K4me1 antibodies using primers specific to the peak regions. Open chromatin ATAC-seq peaks associated with enhancer regions were identified and validated in the PFC of AUD subjects. Changes in H3K27ac and H3K4me1 marks were validated and confirmed the ATAC-seq findings. Significant increases were identified by H3K27ac and H3K4me1 precipitation in AUD samples for LHFPL6 (Lipoma HMGIC Fusion Partner, Tetraspan Subfamily Member 6), ADAMTSL1 (a disintegrin and metalloproteinase with thrombospondin motif), and OS9 (OsteosarcomaAmplified 9, Endoplasmic Reticulum Lectin). Our data and network analysis showed that LHFPL6, ADAMTSL1 and OS9 interact with several key molecular factors involved in inflammatory processes, including TP53, a tumor suppressor protein, EGR1, a nuclear protein and transcriptional regulator, or NF-kB, MEF2, and PPAR involved in inflammation and cell signaling in human PFC.

Symposia

IBRO-sponsored
Symposium 5
Young Women Investigator in Africa

Organizer: **Nouria Lakhdar-Ghazal (Morocco)**

African Center for Advanced Training in Neuroscience,
Mohammed V University, Rabat, Morocco
email: **nlakhdarghazal@gmail.com**

Abstract:

This symposium will be funded by IBRO and is being organized to bring together 5 young female neuroscientists from different parts of Africa who are at the start of their career, at the end of their thesis or post-doctorate to present their research work, which is being developed in their laboratory in Africa. This symposium encourages research carried out by women in a context where scientific research is dominated by men. The symposium will include a speaker representing North Africa (Morocco), a speaker representing South Africa (RSA), a speaker representing East Africa (Kenya), a speaker representing West Africa (Nigeria) and finally a speaker representing Central Africa (Cameroon). The symposium is proposed and will be coordinated by Professor Nouria Lakhdar-GHazal (Chair of the African Centre for Advanced Training in Neuroscience).

Speakers

Number	Speaker	e-mail	Title of the communication
SP5_1	Siham Boumhaoued	siham_boumhaouad @um5.ac.ma	Melatonin modulates the diurnal variations in both cholinergic and dopaminergic release in the mouse striatum
SP5_2	Amalia Awala	awlama001 @myuct.ac.za	Silent sentinels: microglial (in)action during cryptococcal infection
SP5_3	Nelly Murugi Nyaga	Nellyflashier @gmail.com	Translational Research: SMART Guidelines for Psychiatric Diseases
SP5_4	Kom Diebo Bibiane Tatiana	tatianakom717 @gmail.com	Antidepressant-like effects of the aqueous lyophilisate of Momordica foetida (Cucurbitaceae) in rats
SP5_5	Adedunsola Obasa	Dunsolaobasa @gmail.com	Neuropathological Impacts of Pre-Injury Stress in Ferrets (Mustela putorius furo) with Traumatic Brain Injury (TBI).

Symposia

SP5_1

Title

Melatonin modulates the diurnal variations in both cholinergic and dopaminergic release in the mouse striatum.

Authors

Siham Boumhaouad[12], Anika Frank[13], Nezha Bouhaddou[2], Jihane Balla[2], Khalid Taghzouti[2], David Sulzer[1], Eugene V. Mosharov[1]

[1] Departments of Psychiatry and Neurology, Division of Molecular Therapeutics, New York State Psychiatric Institute, Columbia University Medical Center, New York, NY, USA. [2] Physiology and Physiopathology Team, Genomics of Human Pathologies Research Center, Faculty of Sciences, Mohammed V University in Rabat, Rabat, Morocco. [3] Department of Neurology, Faculty of Medicine and University Hospital Carl Gustav Carus, Technische Universität Dresden, Dresden, Germany

Abstract

Background: Melatonin, an indolamine, is derived from the precursor tryptophan through a series of enzymatic steps. In a commonly used strain of C57BL/6J mice, the genes for these enzymes are truncated, resulting in a lack of circadian melatonin rhythmicity. By introducing these alleles, a C57BL/6J congenic line known as CBA/CaJ was created, with the ability to synthesize melatonin. It has been shown that extracellular striatal dopamine levels fluctuate during the day, as a result of changes in the cholinergic neurons' circadian rhythm activity, which itself would be controlled by melatonin's rhythmicity. As diurnal changes in DA have been shown to alter synaptic connectivity and animal behaviour, understanding this modulation has consequences for diagnostics and treatment of neurodegenerative diseases such as Parkinson's disease and psychiatric disorders. Methods: In this study, we examined the impact of melatonin on evoked DA release using fast-scan cyclic voltammetry in acute striatal slices from CBA/CaJ and C57BL/6J mice. Slices were prepared at two distinct points during the light/dark cycle, corresponding to the lowest and highest MLT levels. Results: Our findings reveal that during the dark cycle, when MLT peaks, DA release decreases in CBA mice and not in C57BL6 mice. Furthermore, when physiological concentrations of exogenous MLT were applied to the slices, it inhibited DA release mainly in CBA/CaJ mice. In contrast, C57BL/6J mice did not display a substantial response, likely due to their reduced sensitivity of receptors to melatonin. Conclusion: Our results confirm that melatonin receptors? activation plays a vital role in modulating striatal DA release and establishing the dose- and time-dependent kinetics of release inhibition and recovery. Additionally, we showed that this regulation of DA rhythmicity by melatonin is mediated through the activity of cholinergic interneurons in the striatum.

Symposia

SP5_2

Title

Silent sentinels: microglial (in) action during cryptococcal infection.

Authors

Awala AN[1234], Higgitt ER[234], de Lange A[1234], Kauchali M[234], Pato Y[234], Raimondo JV[12], Dangarembizi R[234]

[1] Division of Cell Biology, Department of Human Biology, Faculty of Health Sciences, University of Cape Town, South Africa. [2] Neuroscience Institute, Faculty of Health Sciences, University of Cape Town, Groote Schuur Hospital, South Africa. [3] Division of Physiological Sciences, Department of Human Biology, Faculty of Health Sciences, University of Cape Town, South Africa. [4] CMM AFRICA Medical Mycology Research Unit, Institute of Infectious Disease and Molecular Medicine, Faculty of Health Sciences, University of Cape Town, South Africa

Abstract

Background: Cryptococcal meningitis (CM) is a fatal fungal infection of the brain and the leading cause of HIV-related mortality in the world. Brain injury in CM is thought to be of an inflammatory origin; however, substantial gaps exist in the neuropathogenesis of the disease. Particularly unknown is how microglia, the resident immune cells of the brain, respond to brain invasion by Cryptococcus neoformans, the causative fungus for CM. We therefore employed rodent and human brain-based experimental models to characterize the interaction between microglia and C. neoformans in vitro. Methods: We stimulated cultured rodent and human organotypic brain slices (OBSs) with a GFP/mCherry expressing reporter H99 strain of C. neoformans and characterized fungal-cell interactions using cell-type-specific immunofluorescent markers for microglia (IBA1). Inflammatory activation of microglial cells within C. neoformans-treated OBS was measured by dual immunofluorescence staining with the inflammatory transcription factor nuclear factor for interleukin 6 (NF-IL6), a robust marker for tracking inflammation. We also performed single-nuclei RNA-sequencing to determine inflammatory responses at the transcriptomic level. C. neoformans-treated slices were compared to untreated slices and slices treated with a known immunogen, lipopolysaccharide (LPS). To investigate potential neuroimmune modulation, we co-stimulated C. neoformans-treated slices with LPS. Inflammation was further confirmed by measuring proinflammatory cytokine release using a multiplex assay. Results: C. neoformans was internalized by microglia but infected microglia were paradoxically inactive. There was minimal NF-IL6 activation and proinflammatory cytokine release in C. neoformans-treated slices and controls as was observed in LPS-treated slices. The slices co-stimulated with LPS and C. neoformans showed a significant reduction in NF-IL6 and cytokines suggesting that C. neoformans may possess immunomodulatory properties. Conclusions We conclude that C. neoformans engages in a unique immunomodulatory interaction with microglial cells, being internalized however failing to induce a robust proinflammatory response.

Symposia

SP5_3

Title

Translational Research: SMART Guidelines for Psychiatric Diseases

Authors

Nelly Nyaga, Steven Wanyee. IntelliSOFT Consulting Limited, Nairobi, Kenya

Abstract

An important element in enhancing global healthcare delivery is the digitalization of healthcare systems. This has led to improved access, enhanced quality of care, greater accountability, and reduced costs. This transition presents an opportunity for healthcare systems worldwide to digitize existing paper-based processes and leverage the additional benefits offered by digital tools. The benefits include the integration of clinical decisions in line with evidence-based treatment guidelines and the standardization of approaches in calculating health indicators for more accurate public health reporting and monitoring. However, integrating clinical guidelines derived from research into digital healthcare systems remains unsatisfactory as the integration is often marred by errors and inconsistencies that compromise the quality of care. This absence of standardized documentation for translating research-based guidance into digital systems frequently results in subjective interpretations by the implementers and the software vendors, leading to erroneous representations of clinical content. To address this challenge, our implementation research utilizes the WHO SMART (Standards-based, Machine-readable, Adaptive, Requirements-based, and Testable) guideline framework to develop a digital adaptation kit designed for managing depressive and psychotic disorders. This digital adaptation kit will serve as a bridge to translate clinical guidelines into precise technical specifications for software development to be used to manage psychiatric disorders. Guided by global normative clinical guidelines for psychiatric disorders, this digital adaptation kit will include standards-based clinical workflows, data dictionaries, and algorithms for clinical decision-making and treatment of these disorders. This approach has been applied in the development of the WHO Digital Adaptation Kit for Family Planning and Antenatal Care whichare now global references for the development of digital tools to support these program areas. Our aim is to ensure that digital health tools for psychiatric conditions strictly adhere to clinical guidelines, leading, ultimately, to improved patient outcomes.

SP5_4

Title

Antidepressant-like effects of the aqueous lyophilisate of Momordica foetida (Cucurbitaceae) in rats

Authors

Bibiane Tatiana Diebo Kom1, Gwladys Temkou Ngoupaye2, Elisabeth Ngo Bum1

1Department of Biological Sciences, Faculty of Science, University of Maroua, Maroua, Cameroon; 2Department of Animal Biology, Animal Physiology and Phytopharmacology Research Unit, University of Dschang, Dschang, Cameroon.

Abstract

M. foetida (Cucurbitaceae) is a perennial climbing herb, known in traditional medicine for the treatment of certain diseases, such as malaria, headaches, skin-related problems and many others. The objective of this work is to evaluate the antidepressant effect of the aqueous lyophilisate of the mixture of leaves and stems of M. foetida, as well as the signaling pathway by which this effect is mediated. The antidepressant effect of the aqueous lyophilisate of M. foetida at different doses (25 mg/kg, 50 mg/kg, and 75 mg/kg) was evaluated in Wistar rats of both sexes submitted to chronic restriction for 14 days using the forced swimming test, open field test and sucrose preference test. One hour after the last behavioural test, animals were sacrificed and the hippocampi were collected for biochemical assessment of oxidative parameters such as malondialdehyde (MDA), reduced Glutathione (GSH), Catalase activity, superoxide dismutase (SOD) and nitric oxide (NO) as well as monoamines levels such as serotonin, noradrenaline and dopamine. The aqueous lyophilisate of M. foetida significantly decreased the immobility time and significantly increased sucrose consumption ($P<0.001$) and no alteration of locomotor activity. The aqueous lyophilisate of M. foetida significantly increased the concentrations of GSH, SOD, as well as catalase activity, while reducing the concentrations of MDA and NO at all doses ($P<0.001$). M. foetida at the doses 25 mg/kg and 50 mg/kg significantly increased the concentration of serotonin and dopamine, and the dose 75 mg/kg significantly increased the concentration of noradrenaline ($p<0.001$). These results suggest the antidepressant-like effects of M. foetida through the modulation of the oxidative stress and monoamine pathways.

Keywords: Depression; M. foetida; Monoamines; Chronic restriction; Oxidative stress.

Symposia

SP5_5

Title

Neuropathological Impacts of Pre-Injury Stress in Ferrets (Mustela putorius furo) with Traumatic Brain Injury (TBI)

Authors

Adedunsola Obasa, Susan C. Schwerin; Michael Ray; Mitali Chatterjee; Sharon L. Juliano (Uniformed Services University of the Health Sciences, USA); James O. Olopade (University of Ibadan, Nigeria)

Abstract

Background & Objectives: Traumatic Brain Injury (TBI) leads to significant disability and death, and is linked to neurodegenerative diseases and sleep disorders. Sleep activates the glymphatic system, which removes brain metabolites via aquaporin-4 (AQP4) channels. Chronic stress disrupts this process, leading to sleep disorders and exacerbating stress, which negatively impacts health. Despite the intrinsic relationship between stress and TBI, few studies have explored the influence of pre-existing stress on TBI outcomes. This study investigated the impacts and temporal dynamics of pre-injury stress exposure in ferrets with TBI, focusing on activity/sleep patterns, components of the glymphatic system and various neurological markers. Methods: Adult male ferrets were divided into three cohorts (C1, C2, C3) and assigned to Control, Injury (IN), or Injury+Stress (I+S) groups. Each cohort underwent different injury and stress protocols. Activity/sleep patterns were monitored. Brain tissue was analysed using immunofluorescent markers for astrocytes, AQP4 and microglia. Results and Discussion: At 1MPI, CON exhibited distinct clusters of sustained high-activity with 50% being significantly active for over 50 minutes. Clustering was greatly reduced and more random in IN and I+S. At 6MPI, I+S had more high activity clusters than IN but e less sustained and more random than CON. Changes in activity patterns inferred sleep disturbances, with reduced activity indicating potential fatigue and reduced functionality. Elevated astrocytic and AQP4 immunoreactivity persisted in IN and I+S up to 6MPI. An important feature of TBI is reactive astrogliosis and an upregulation of AQP4 expression, impacting neuroinflammation, sleep regulation, post-traumatic oedema and secondary injury progression. IN exhibited microglial activation at 1MPI, transitioning to amoeboid morphology by 6MPI. I+S showed reduced microglial immunoreactivity compared to IN. Chronic microglial activation contributes to secondary injury and chronic neurodegenerative pathology after TBI. Stress seemed to reduce IBA1 immunoreactivity, with previous studies showing that chronic stress elevates microglia in stress-sensitive brain regions and shifts them to an active state. In conclusion, pre-existing stress can influence TBI outcomes over time, affecting activity/sleep patterns, astrocytic reactivity and microglial response. Paradoxically, pre-injury stress showed some neuroprotective effects in TBI, highlighting the complex relationship between stress and brain injury outcomes.

Keywords- TBI, Ferrets, Stress.

Symposia

Symposium 6

The Science of Burnout: From Brain Mechanisms to Holistic Management Approaches.

Organizer: **Daniel Gams Massi**

Douala General Hospital,
University of Douala, Douala, Cameroon
email: **danny.gamsmassi@gmail.com**

Abstract:

Burnout is a syndrome resulting from chronic workplace stress not being successfully managed. Three dimensions characterize it: feelings of energy depletion or exhaustion; increased mental distance from one's job, or feelings of negativism or cynicism related to one's job; and reduced professional efficacy1. The burden of burnout increased during the COVID-19 pandemic2. Despite the importance of the data in the literature, understanding the pathophysiological, clinical, and therapeutic characteristics of burnout remains a challenge for many scientists.

In this symposium, we intend to provide some relevant elements to appreciate the extent of this problem in Africa. To do this, we will address the fundamental mechanisms underlying the consequences of burnout on the brain. Then, we will present the physical expression of these alterations related to burnout and the diagnostic strategies. We will also discuss pharmacological treatments and the contributions of alternative medicine to the management of burnout. It will be a 1.5-hour symposium; with four speakers from different backgrounds with a session chair, (a co-chair will be optional).
References: 1WHO keyfacts and details; 2Alanazy ARM, Alruwaili A. Healthcare (Basel). 2023

Speakers

Number	Speaker	e-mail	Title of the communication
SP6_1	Oritoke M. Okeowo	omokeowo@futa.edu.ng	Basic Neurological Mechanisms underlying Burnout
SP6_2	Francky Teddy Endomba	franckyteddyea@gmail.com	Burden and clinical manifestations of burnout : a focus on Africa.
SP6_3	Daniel Gams Massi	danny.gamsmassi@gmail.com	The burden and clinical manifestations of burnout in clinical settings.
SP6_4	Gwladys Temkou Ngoupaye	gtngoupaye@gmail.com	Burn out and resilience: The two faces of a coin.

Symposia

SP6_1

Title

Basic Neurobiological Mechanisms Underlying Burnout

Author

Oritoke M. Okeowo 1Department of Physiology, School of Basic Medical Sciences, Federal University of Technology, Akure, Ondo State, Nigeria 2Laboratory for Experimental and Translational Neurobiology, University of Medical Sciences, Ondo, Ondo State, Nigeria

Abstract

Burnout is a psychological condition characterized by emotional exhaustion, depersonalization, and diminished personal accomplishment, recognized as a growing public health challenge in high-demand environments. Emerging evidence underscores the role of neurobiological mechanisms in the development and progression of burnout, particularly emphasizing the hypothalamic-pituitary-adrenal (HPA) axis, neuroinflammation, and neurotransmitter dysregulation. Prolonged activation of the HPA axis due to chronic stress leads to excessive cortisol release, resulting in structural and functional impairments in brain regions such as the hippocampus, amygdala, and prefrontal cortex. These changes disrupt critical processes, including emotion regulation, memory consolidation, and decision-making. Simultaneously, chronic stress triggers neuroinflammation, characterized by the activation of microglia and the release of pro-inflammatory cytokines such as IL-1α and TNF-α. This inflammatory response exacerbates neural dysfunction and contributes to the cognitive and emotional symptoms observed in burnout. Alterations in neurotransmitter systems, including serotonin, dopamine, and norepinephrine, further illuminate the link between chronic stress and burnout. Dysregulation of these systems is associated with decreased motivation, anhedonia, and cognitive impairments. Neuroimaging studies corroborate these findings, revealing structural atrophy in the hippocampus, reduced connectivity in the prefrontal cortex, and heightened amygdala activity in individuals experiencing burnout. Understanding the neurobiological mechanisms underlying burnout offers critical insights into its pathophysiology and lays the groundwork for developing biomarkers and targeted interventions. This perspective also bridges the gap between neuroscience and mental health, emphasizing the importance of addressing burnout to improve individual well-being and organizational productivity.

Symposia

SP6_2

Title

Burden and clinical manifestations of burnout: a focus on Africa

Authors

Francky Teddy ENDOMBA (MD, MSc) Dijon Bourgogne University Hospital and University of Burgundy, Dijon, France, Daniel Gams Massi (MD) Douala General Hospital and University of Douala, Douala, Cameroon, Oritoke M. OKEOWO (PhD) Federal University of Technology, Akure, Nigeria

Abstract

Included in the 11th revision of the International Classification of Diseases (ICD-11) as an occupational phenomenon, burnout (syndrome) has been widely studied worldwide, especially in Western settings. In this presentation, we aim to highlight the epidemiological burden of this phenomenon in Africa and provide a global view of its manifestations. To explore the epidemiology of burnout in Africa, we conducted a narrative review of articles published in PubMed/MEDLINE using a search strategy designed by combining terms related to African regions/countries and burnout (using the title filter). In just under 200 papers retrieved using our strategy, the majority of articles concerned southern Africa, followed by eastern and western Africa. Almost 90% of the published papers focused on health care workers and health care students. The prevalence of significantly high levels of burnout among health care workers varies widely between studies, generally ranging from 20% to 80%. Factors associated with burnout include low social support, workload as reflected in the number of hours worked per week, interpersonal and professional conflicts, poor relationships with supervisors, work environment, number of years in the job, night shifts, alcohol consumption, low physical activity, and mental/physical health status. The COVID-19 pandemic also influenced the burden of burnout, particularly among doctors and nurses, with higher frequencies during and after the pandemic compared with the pre-pandemic period. For burnout in populations other than health care providers/students, data were available for teachers, university students in academic fields other than health care, police officers, bankers, and drivers/conductors. In terms of burnout manifestations, most studies published in African settings have used the clinical dimensions of the Maslach Burnout Inventory. These dimensions, which are close to those in the ICD-11, include emotional exhaustion, depersonalisation (replaced by cynicism in the revised version) and low personal accomplishment. Burnout can have a negative impact on work, but also on health and quality of life. In conclusion, although burnout is not a disease in the international nomenclature, it is a highly prevalent phenomenon in Africa. There seems to be a need to optimise research and management of this occupational phenomenon in the African context.

Symposia

SP6_3

Title

The burden and clinical manifestations of burnout in clinical settings

Authors

Daniel Gams Massi, University of Douala, Douala General Hospital

Abstract

Burnout is a state of fatigue or frustration related to a commitment to a cause, a lifestyle or a relationship that has not brought the expected gratification. Burnout has been frequently studied in 3 specific situations: in professionals, in parents and in caregivers. It is a condition of recent interest, but is especially common in situations of chronic stress. Burnout syndrome is based on 3 fundamental dimensions: emotional exhaustion, dehumanization of the helping relationship and decreased personal accomplishment. Physical manifestations can be at the forefront. These include recurrent headaches; abdominal pain, nausea, bloating, diarrhea, various food intolerances; itching, irritation, burning sensations, tingling; psychogenic frequent urination, psychogenic dysuria, painful periods; back or joint pain; palpitations, sweating, hot flashes, tremors, cold sensations. Scales are used to assess professional burnout (Maslach Burn-out inventory, Shirom-Melamed Burn-out Measure, Oldenburg Burn-out Inventory, Job Burn-out Inventory), parental burn-out (Parental burn-out inventory, Parental Burn-out Assessment), and caregiver burnout (Zarit Burden Interview, Maslach Burn-out Inventory Informal Caregiver). While many studies have been conducted in developed countries, the clinical characteristics of burnout in the African population are still poorly described by studies. In this presentation, we will develop the different clinical aspects of burnout and its assessment in clinical practice in an African context.

Symposia

SP6_4

Title

Burnout and resilience: The two faces of a coin

Authors

GWLadys T. Ngoupaye; Department of Animal Biology, University of Dschang

Abstract

Prolonged exposure to work-related stress leads to Burnout. Burnout is a work-related stress syndrome characterized by fatigue and withdrawal. This syndrome tends to decrease feelings of worthiness and successful performance, which leads to behaviors of withdrawing from work, such as absenteeism and running away from work. Three main dimensions of burnout include emotional exhaustion, depersonalization, and reduced personal accomplishment. Regardless of sex, burnout is observed in both men and women. Resilience which is the capacity to recover quickly from difficulties, has been reported by studies to be a key component of well-being under prolonged adversity. Although burnout and resilience seem to look as two faces of a coin, should this be actually the case regardless of the magnitude of the burnout and predisposing factors to that? In this presentation, we will discuss how developing resilience could meet physiological disturbances induced by the leading factors of burnout syndrome and highlight some limitations.

Symposia

ALBA Network-IBRO

ALBA Network-IBRO workshop: Equity in publishing: strategies for inclusive publishing and knowledge sharing.

Organizer: **Francesca Cirulli (Italy)** & John Foxe (USA)

Centre for Behavioural Sciences and Mental Health, Instituto Superior di Sanita, Italy
Department of Neuroscience, University of Rochester, New York, USA
email: **francesca.cirulli@iss.it; cogneurolabrochester@gmail.com**

Abstract:

This session, organised by the ALBA Network with the support of IBRO, aims to address the significant challenges faced by neuroscience researchers from low-to-middle-income countries (LMICs) in accessing equitable opportunities in publishing.
Targeted at local researchers from all career levels, the workshop will examine the systemic barriers within the global publishing industry that limit diverse representation and perspectives in scientific publications. It will explore how some publishing practices perpetuate harmful stereotypes, and exclude diverse voices.
Participants will engage in discussions led by experienced publishing professionals, on exploring the challenges that they face when publishing, bridging the publishing gap, and the advantages of publishing in society journals. The workshop aspires to empower attendees to foster a more equitable publishing ecosystem that amplifies underrepresented perspectives and knowledge from the Global South. This event is organised with the support of the International Brain Research Organization (IBRO). The ALBA Network is a division of the Federation of European Neuroscience Societies (FENS).

Speakers:

- **Francesca Cirulli**, Centre for Behavioural Sciences and Mental Health, Instituto Superior di Sanita, Italy - ALBA Network Chair / Editor in Chief of Neuroscience. francesca.cirulli@iss.it
- **Rachael Dangarembizi**, Neuroscience Institute, University of Cape Town, South Africa – ALBA Board of Directors / Senior Editor of Neuroscience. rachael.dangarembizi@uct.ac.za
- **John Foxe**, Department of Neuroscience, University of Rochester, New York, USA - Editor in Chief of the European Journal of Neuroscience (EJN). cogneurolabrochester@gmail.com
- **Ying Shing Chan**, The University of Hong Kong Li Ka Shing Faculty of Medicine, Hong Kong, Hong Kong SAR China - Founding Editor-in-Chief for IBRO Neuroscience Reports / Associate Editor for European Journal of Neuroscience (EJN). yschan@hku.hk

Programme:

10 min: Opening remarks and introduction (R. Dangarembizi, F. Cirulli)
15 min: **Benefits of publishing in Society Journals** (F. Cirulli, J. Foxe)
10 min: **The publishing gap** (R. Dangarembizi)
20 min: Open discussion with the audience – **Challenges in publishing for researchers from LMIC** (R. Dangarembizi)
25 min: Open discussion with the audience – **Strategies for inclusive publishing and opportunities for researchers from LMIC** (Y.S. Chan, F. Cirulli, J. Foxe)
10 min: Conclusions and identified solutions (Y.S. Chan, F. Cirulli, R. Dangarembizi, J. Foxe)

Symposia

Symposium 7

"An update on the pathophysiology of the non-motor symptoms in Parkinson's disease"

Organizer: **Abdelhamid Benazzouz**

Neurodegenerative diseases institute,
Bordeaux University, Bordeaux, France
email: abdelhamid.benazzouz@u-bordeaux.fr

Abstract:

Parkinson's disease (PD) is a neurological disorder well known for its disabling motor symptoms and also for the wide range of non-motor symptoms, including anxiety, depression, apathy, sleep disorder, pain and others. These non-motor symptoms, which contribute to the deterioration of patients' quality of life, are less well treated than motor symptoms, due to a poor understanding of their pathophysiology.
In this symposium, we will present the results of recent works to clarify the pathophysiological mechanisms of these disabling non-motor symptoms. Houyam Tibar will present a clinical overview of the non-motor symptoms with a focus on the most frequent in moroccan parkinsonian patients. Then, Karim Fifel will present new experimental data on the role of motivational deficits in sleep/wake disorders in PD. This presentation will be followed by three talks focusing on the control of nociceptive abnormalities by descending pathways from the brain to the dorsal horn of the spinal cord (DHSC) in the context of PD. Pascal Fossat and Alexia Duveau will present new data highlighting the key r ole of the serotonergic and the dopaminergic (respectively) control of pain and spinal cord integration in the 6-OHDA mouse model of PD. Finally, Abdelhamid Benazzouz will highlight an important mechanism of interplay between the subthalamic nucleus and DHSC in controlling nociceptive circuits in the context of PD.
Overall, our symposium will highlight the important pathophysiological mechanisms of the non-motor symptoms related to PD, which may contribute to the development of new therapeutical approaches.

Speakers

Number	Speaker	e-mail	Title of the communication
SP7_1	Houyam Tibar	tibarhouyam@gmail.com	An overview on the non-motor symptoms of Parkinson's disease: Moroccan experience.
SP7_2	Karim Fifel	karim.fifel@um6p.ma	The role of Motivational deficits in sleep/wake disorders in PD
SP7_3	Pascal Fossat	pascal.fossat@u-bordeaux.fr	Brainstem serotonin amplifies nociceptive transmission in a mouse model of Parkinson's disease
SP7_4	Alexia Duveau	alexia.duveau@u-bordeaux.fr	The A11 hypothalamic dopaminergic nucleus and the control of nociception abnormalities in a mouse

Symposia

SP7_1

Title

An overview on the non-motor symptoms of Parkinson's disease: Moroccan experience

Authors

Houyam tibar, wafa regragui, hopital des spécialités, ibn sina university hospital, medical school of rabat, rabat, morocco

Abstract

The non-motor symptoms (NMS) of Parkinson's disease significantly impact patients' quality of life and often precede the classical motor manifestations. Despite their prevalence, these symptoms remain underrecognized and undertreated. This presentation will focus on the clinical spectrum of NMS in Moroccan Parkinsonian patients, highlighting their frequency, impact, and management challenges in our specific context. Based on our clinical experience and research data from Moroccan centers, we will present the most frequent NMS encountered in our population, including mood disorders, sleep disturbances, autonomic dysfunction, and pain. Special attention will be given to cultural and socioeconomic factors that may influence the presentation and recognition. We will also discuss the screening tools used in our practice and share our approach to early identification and management of NMS in resource-limited settings. This overview aims to contribute to the better understanding of NMS in different populations and emphasize the importance of their systematic assessment in routine clinical practice.

SP7_2

Title

The role of motivational deficits in sleep/wake disorders in PD r

Authors

Karim Fifel, Masashi Yanagisawa, Tom Deboe

Abstract

Sleep/wake alterations are predominant in neurological and neuropsychiatric disorders involving dopamine dysfunction. Unfortunately, specific, mechanisms-based therapies for these debilitating sleep problems are currently lacking. The pathophysiological mechanisms of sleep/wake alterations within a hypodopaminergic MitoPark mouse model of Parkinson’s disease (PD) are investigated. MitoPark mice replicate most PD-related sleep alterations, including sleep fragmentation, hypersomnia, and daytime sleepiness. Surprisingly, these alterations are not accounted for by a dysfunction in the circadian or homeostatic regulatory processes of sleep, nor by acute masking effects of light or darkness. Rather, the sleep phenotype is linked with the impairment of instrumental arousal and sleep modulation by behavioral valence. These alterations correlate with changes in high-theta (8-11.5 Hz) electroencephalogram power density during motivationally-charged wakefulness. These results demonstrate that sleep/wake alterations induced by dopamine dysfunction are mediated by impaired modulation of sleep by motivational valence and provide translational insights into sleep problems associated with disorders linked to dopamine dysfunction.

SP7_3

Title

Brainstem serotonin amplifies nociceptive transmission in a mouse model of Parkinson's disease.

Authors

Pascal Fossat[1-2], Zoé Grivet[12], Franck Aby[1-2], Aude Verboven[12], Rabia Bouali-Benazzouz[1-2], Benjamin Sueur[1-2], François Maingret[1-2], Frédéric Naudet[1-2], Thibault Dhellemmes[1-2], Philippe De Deurwaerdere[3-4], Abdelhamid Benazzouz[1-2]#, Pascal Fossat[1-2]#*

[1] Université de Bordeaux, Institut des Maladies Neurodégénératives, UMR 5293, F-33000 Bordeaux, France. [2] CNRS, Institut des Maladies Neurodégénératives, UMR 5293, F-33000 Bordeaux, France. [3] Université de Bordeaux, Institut des Neurosciences Cognitives et Intégratives d'Aquitaine, UMR 5287, F-33000 Bordeaux, France. [4] CNRS, Institut des Neurosciences Cognitives et Intégratives d'Aquitaine, UMR 5287, F-33000 Bordeaux, France

Abstract

Parkinson's disease arises from the degeneration of dopaminergic neurons in the substantia nigra pars compacta, leading to motor symptoms such as akinesia, rigidity and tremor at rest. The non-motor component of Parkinson's disease includes increased neuropathic pain, the prevalence of which is 4 to 5 times higher than the general rate. By studying a mouse model of Parkinson's disease induced by 6-hydroxydopamine, we assessed the impact of dopamine depletion on pain modulation. Mice exhibited mechanical hypersensitivity associated with hyperexcitability of neurons in the dorsal horn of the spinal cord (DHSC). Serotonin (5-HT) levels increased in the spinal cord, correlating with reduced tyrosine hydroxylase (TH) immunoreactivity in the nucleus raphe magnus (NRM) and increased excitability of 5-HT neurons. Selective optogenetic inhibition of 5-HT neurons attenuated mechanical hypersensitivity and reduced DHSC hyperexcitability. In addition, blockade of 5-HT2A and 5-HT3 receptors reduced mechanical hypersensitivity. These results reveal, for the first time, that PD-like dopamine depletion triggers spinal-mediated mechanical hypersensitivity, associated with serotonergic hyperactivity in the NRM, opening up new therapeutic avenues for Parkinson's disease-associated pain targeting the serotonergic systems.

Symposia

SP7_3

Title

The A11 hypothalamic dopaminergic nucleus and the control of nociception abnormalities in a mouse

Authors

Alexia Duveau, A. Duveau (université de bordeaux, CNRS), R. bouali-Benazzouz (université de bordeaux, Cnrs), J. Viellard (université de bordeaux, cnrs), F. Naudet (université de bordeaux, cnrs), J. Bonneau (université de bordeaux, cnrs), H. Jadot (université de bordeaux, cnrs), P. Fossat (université de bordeaux, cnrs), A. Benazzouz (université de bordeaux, cnrs)

Abstract

Parkinson's disease (PD) is a neurodegenerative disease characterized by the manifestation of motor and non-motor symptoms. Among the latter, pain is one of the most frequent symptoms, affecting around 80% of patients. Nowadays, their treatment is still inefficient due to the lack of knowledge of the underlying mechanisms. However, we know that pain can be modulated by the monoaminergic descending pathway at the level of the spinal cord. Thus, the aim of the present study is to unravel the involvement of the hypothalamic A11 nucleus, one of the only sources of dopamine in the dorsal horn of the spinal cord (DHSC), in the control of the nociceptive impairments in 6-OHDA mouse model of PD. After characterizing the anatomical, behavioral and electrophysiological disruption in this model, we used optogenetic approaches to selectively modulate the A11 dopaminergic neurons projecting in the lumbar DHSC. We first demonstrated that the activation of A11 dopaminergic descending pathway is able to improve mechanical allodynia in 6-OHDA mice without affecting motor symptoms. Furthermore, we also show that this activation may have an effect on nociceptive integration in the DHSC. Those new findings, may lead to find new therapeutics target in order to efficiently treat pain as a non-motor symptom of PD.

Symposia

Symposium 8

Molecular Mechanisms of Heavy Metal Toxicity in Neurodegenerative Diseases: Translational Medicine and the Role of Natural Antidotes

Organizer: **Chinna Orish**

Department of Anatomy.University of PortHarcourt.Rivers State
email: chinna.orish@uniport.edu.ng

Abstract:

Neurodegenerative diseases, marked by progressive neuronal degeneration, represent an escalating global public health crisis. Emerging evidence strongly links exposure to heavy metals like lead, mercury, cadmium, and aluminum with the onset and progression of these disorders. This symposium will examine the molecular mechanisms by which heavy metals induce neurotoxicity, emphasizing their role in the pathogenesis of neurodegenerative diseases. Heavy metal toxicity initiates a cascade of detrimental molecular events, including oxidative stress, chronic inflammation, mitochondrial dysfunction, and disrupted metal homeostasis. These processes contribute to misfolded protein accumulation, synaptic dysfunction, and neuronal cell death hallmarks of conditions such as Alzheimer's, Parkinson's, and ALS. A thorough understanding of these pathways is critical for the development of targeted therapeutic interventions. Translational medicine is essential in transforming laboratory research into clinical applications, offering new hope for those affected by neurodegenerative diseases. Advances in understanding heavy metal-induced neurotoxicity have led to innovative diagnostic tools, biomarkers, and therapeutic strategies.

Approaches like nanotechnology, gene therapy, and pharmacological interventions show promise in counteracting the neurotoxic effects of heavy metals, potentially slowing or halting disease progression ting agents exhibit neuroprotective properties by scavenging free radicals, modulating inflammation, and enhancing metal excretion. These natural compounds provide valuable insights for developing safe, adjunctive therapies. This presentation will synthesize the current understanding of the molecular mechanisms underlying heavy metal toxicity in neurodegenerative diseases. It will also highlight the translational potential of recent scientific advancements and the role of natural antidotes in creating effective treatment strategies. By elucidating these complex interactions, this symposium aims to contribute to the development of targeted interventions that could significantly alleviate the global burden of neurodegenerative diseases.

Cancelled

Symposia

Symposium 9

Early life stress,Sex differences, and Maternal influences: Insights into Brain and Behavioral Disorders

Organizer: **Fatiha Chigr**

Biological Engineering Laboratory,
Faculty of Sciences and Techniques,
Sultan Moulay Sliman, University, Beni Mellal, Morocco
email: **F.CHIGR@usms.ma**

Abstract:

Early life stress (ELS), which includes prenatal and postnatal stress, is a significant factor profoundly affect brain development, leading to long-lasting alterations in neurobiological functions and behavior. During critical developmental periods, adverse experiences are associated with an increased risk of neuropsychiatric and mental disorders, including anxiety, depression, long-term cognitive deficits, and metabolic dysregulation.

This symposium will explore the impact of ELS, with a focus on the underlying neurobiological mechanisms, it will also provide an overview of our current understanding of the neurodevelopmental, molecular, and environmental factors that mediate the effects of perinatal insults on neurobiological and behavioral outcomes, ranging from metabolic to behavioral disorders.

The invited speakers are leading experts in developmental programming. They will address various aspects of the maternal environment, including nutrition, hormonal imbalances, psychological and social stress, and a range of lifelong disorders in offspring, such as obesity, anxiety, depression, memory deficits, emotion-related behaviors, and sociability. They will Additionally, we will highlight recently identified mechanisms and explore potential intervention strategies.

These emerging concepts offer new perspectives on managing obesity, metabolic, and behavioral disorders, as observed in human life. This symposium is noteworthy for its interdisciplinary focus and potential for real-world applications, and it is likely to attract a wide audience of neurobiologists.

Speakers

Number	Speaker	e-mail	Title of the communication
SP9_1	Sebastien BOURET	Sebastien.bouret@inserm.fr	Sex specific differences in developmental programming of metabolism
SP9_2	Oumaima ESSAIDI	essaidioumaima1996@gmail.com	Sex-specific long-term effects of prenatal stress in miceÂ offspring.
SP9_3	Liana FATTORE	liana.fattore@in.cnr.it	Sex-dependent differences in the effect of pre-weaning social isolation and enrichment
SP9_4	Stefania MACCARI	stefania.maccari@univ-lille1.fr	The intergenerational inheritance of early life stress is transmitted by maternal care via oxytocin.

Symposia

SP9_1

Title

Sex specific differences in developmental programming of metabolism

Authors

Sebastien Bouret, Inserm UMR-S1172, Lille Neuroendocrinology lab, Lille, France

Abstract

The early life environment, particularly during fetal development and the neonatal period, plays a crucial role in shaping an individual's susceptibility to metabolic diseases later in life, including obesity. Interestingly, the incidence of metabolic disorders differs between males and females, raising the need to investigate how early life experiences contribute to these sex-specific differences. There is general recognition that the developing brain is more susceptible to environmental insults than the adult brain. In particular, there is growing appreciation that the developmental programming of hypothalamic neuroendocrine systems by the perinatal environment represents a possible cause for metabolic diseases. This presentation will summarize the major stages of hypothalamic development and provide an overview of evidence concerning the sex-specific action of perinatal hormones, neurohormones (such as oxytocin) and maternal nutrition in programming the development and organization of hypothalamic circuits involved in feeding and energy balance regulation. It will also discuss possible mechanisms responsible for mediating these sexually dimorphic effects on hypothalamic development and metabolic programming. Further investigation on how early life environmental changes differently impact long-term health in males and females will be crucial for developing sex-specific strategies for metabolic risk monitoring and control, particularly during critical periods of life.

Symposia

SP9_2

Title

Sex-specific long-term effects of prenatal stress in mice offspring

Authors

Oumaima Essaidi[1], Meriem Laaroussi[1], Laila Berroug[2], Hammou Anarghou[1,3], Fatiha Chigr[1].

1. Biological Engineering Laboratory, Faculty of Science and Technology, Sultan Moulay Slimane University, Beni mellal, Morocco 2. Department of Cell Biology and Anatomy, New York Medical College, Valhalla, New York, USA 3. High Institute of Nursing Professions and Health Techniques Dakhla Annex, Dakhla, Morocco

Abstract

Prenatal stress (PS), in both humans and animals, presents a potential risk threatening the mother and her fetus throughout gestation. PS is always associated with physiological changes that alter embryonic development and predispose the individual to lifelong health problems, including susceptibility to mental illness, in a sex-specific manner. This study investigated the long-term effects of prenatal restraint stress on memory and depression-like behaviors. Pregnant mice were subjected to restraint stress from embryonic day 7.5 (E7.5) until delivery for three hours daily, and their offspring were evaluated for discrimination memory and depression-like behaviors. Stressed females exhibited impaired discrimination memory, an effect that persisted into adulthood. Both male and female mice displayed increased depression-like behaviors during adolescence and adulthood. At the adult stage, hippocampal analysis revealed elevated levels of pro-inflammatory cytokines and reduced brain-derived neurotrophic factor (BDNF) expression in stressed animals. These findings suggest that PS induces neuroinflammatory and neuroplasticity alterations, contributing to long-term, sex-specific behavioral and molecular outcomes.

Symposia

SP9_3

Title

Sex-dependent differences in the effect of pre-weaning social isolation and enrichment

Authors

Liana Fattore, Bratzu J, Pisanu A, Porcu P, Fumagalli F, Romualdi P, Trezza V, Ciccocioppo R, Sanna F, Fattore L

Abstract

Early social stress (ESI) interferes with neurodevelopmental processes and can lead to long-lasting emotional, cognitive, and hormonal alterations in adulthood. Communal nesting (CN) is a form of alloparenting in which 2 or more lactating female conspecifics rear their offspring within a common nest while sharing caregiving behavior from birth to weaning, and provides a socially stimulating environment, which was shown to affect social and anxiety-like behaviors. This study investigated whether pre-weaning ESI affects reward-related processing for natural rewards, compulsive tendencies, sensorimotor gating, and basal stress levels in adolescence and adulthood, and whether CN could reverse the impact of ESI on behavior and corticosterone plasma levels. Both male and female rats were used to detect potential sex-dependent differences. In a food self-administration paradigm, both adolescent and adult rats reared in CN conditions showed a significantly slower acquisition and lower active responding than standard housed (SH) animals, while ESI led to a steeper curve in adulthood. During the maintenance period of the training, CN led to a general decrease in active responding in adolescence, while ESI increased responding in adolescent and adult females, an effect that was reversed by CN in adult females. Under a progressive ratio protocol, non-stressed CN animals showed lower breakpoints than SH animals, and ESI increased the breakpoint in all groups, although to a greater extent in females than in males. Notably, ESI-induced effect was reverted by CN. The Marble Burying test revealed an obsessive-compulsive trait in ESI adolescent males, but also this effect was fully prevented by the CN condition. The Prepulse Inhibition (PPI) test showed lowered the PPI in ESI adolescent animals, an effect that was long-lasting in males and reverted by CN. Finally, female ratsshowed higher plasma corticosterone levels, independently from housing and stress conditions. Altogether, our findings indicate that social isolation and communal nesting have long-lasting effects on sensorimotor gating, reward-seeking, and compulsive-like behaviors, with males and females showing different vulnerabilities in these domains.

SP9_4

Title

The intergenerational inheritance of early life stress is transmitted by maternal care via oxytocin.

Authors

Stefania Maccari, A GAETANO, S Morley-Fletcher, R Benlakehal, H Bouwalerh, F Nicoletti;

1-UGSF, UMR 8576, CNRS, Univ. Lille, FR; 2-Dept. Pharmacology, Sapienza University of Rome, IT; 3-IRRCS Neuromed, IT; 4-Dept. Science and Medical - Surgical Biotechnology, University Sapienza of Rome, IT; *LIA-CNRS, PSND, France-Italy

Abstract

Perinatal stress (PRS) in rats induces enduring alterations that can be predicted by reduced maternal behavior due to gestational stress. This study examined the intergenerational effects of PRS by mating first-generation (F1) PRS-exposed female rats with naïve males and evaluating the phenotypes of both F1 and second-generation (F2) offspring. PRS was associated with diminished maternal behavior in F1 mothers and F0 grandmothers. Both F1 and F2 offspring exhibited consistent changes in behaviors, linked to neurobiological alterations affecting stress responses across generations. Notably, F2 offspring were not directly exposed to restraint stress during gestation, suggesting an indirect transmission of PRS effects via maternal care. Given the established role of maternal care in epigenetic transmission, we investigated epigenetic modifications influenced by maternal behavior. Differential gene expression patterns were identified across F1 and F2 generations, implicating key pathways related to glutamatergic synaptic transmission and the regulation of stress systems. These findings highlight the interplay between maternal care and epigenetic mechanisms in shaping the offspring's hypothalamic-pituitary-adrenal (HPA) axis function. Postpartum treatments with carbetocin (an oxytocin analog) or the probiotic Lactobacillus reuteri, known to enhance oxytocin system activity and consequently maternal care, successfully reversed PRS-induced long-term effects in F1 and F2 offspring. Theseinterventions restored normal maternal care and mitigated neurobehavioral and epigenetic alterations. The results underscore the critical role of maternal care in mediating the intergenerational transmission of PRS effects and the plasticity of these outcomes through targeted interventions. Moreover, the findings emphasize the therapeutic potential of modulating the oxytocinergic system to counteract the adverse consequences of PRS, offering promising avenues for addressing stress-related neuropsychiatric disorders across generations.

Symposia

IBRO-sponsored

Symposium 10

The neurobiology and neurochemistry of animals' adaptations to harsh African biotopes

Organizer : **Khalid EL ALLALI**

Institut Agronomique et Vétérinaire Hassan II
RABAT-INSTITUTS 10101 Rabat, Morocco
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Abstract:

Organisms are subject to the effects of changes in their surrounding environment. The majority of vertebrates are equipped with the capacity to anticipate circadian and seasonal changes, which enables them to adapt their biological functions involving time-keeping mechanisms. In mammals, circadian rhythms are driven by a central clock located in the suprachiasmatic nucleus, which modulates the peripheral oscillators, including those in muscle and liver tissue. Melatonin is the primary output of the central clock, serving to synchronize the various peripheral functions.

Melatonin is also understood to convey photoperiodic data to the hypothalamus, thereby enabling seasonal regulation of physiological processes, including reproduction. The primary environmental cue influencing circadian and seasonal rhythms in mammals is photoperiod. However, in African ecosystems, other environmental cues, including ambient temperature, social interactions, and predation, appear to be sufficiently potent to drive the rhythms of native animals.

The symposium will concentrate on the neurochemistry and neurobiological adaptations of native species inhabiting African biotopes. Organizing this symposium aims to convene a select group of experts in the field to engage in a collaborative exchange of ideas, insights, and findings with the present scientists at the conference. The symposium will address aspects concerning the peculiarities of structural, behavioral, and physiological mechanisms in the circadian and seasonal rhythms that have evolved in species living under specific African ecological niches, including those found in deserts and savannas.

Symposia

Speakers

Number	Speaker	e-mail	Title of the communication
SP10_1	Nouria Lakhdar-Ghazal	nlakhdarghazal@gmail.com	Neurochemical Mechanisms of the Jerboa (Jaculus orientalis) to its harsh biotope: focus on the suprachiasmatic nucleus
SP10_2	Nigel Bennett	ncbennett@zoology.up.ac.za	Thermoregulation Meets Chronobiology: How Temperature, Not Light, Governs Circadian Rhythms in Subterranean Mole-Rats
SP10_3	Jérémy TERRIEN	jeremy.terrien@mnhn.fr	Life under the tropics : sex-specific and photoperiod-dependent regulation of metabolism in the grey mouse lemur (*Microcebus murinus*)
SP10_4	Younes BENIAICH	y.beniaich@iav.ac.ma	Effects of the Onset of Rumination on Sleep/Wake Architecture in Camel and Sheep Newborns

Symposia

SP10_1

Title

The suprachiasmatic nucleus- driven Locomotor Activity Rhythm in the male jerboa (Jaculus orientalis) is synchronized by melatonin and depressed by testosterone

Authors

Nouria Lakhdar-Ghazal

Africa Center for Advanced Training in Neuroscience, Mohammed V University, Rabat, Morocco

Abstract

The jerboa (Jaculus orientalis) is a wild species of rodent living in high continental shelves of the Middle Atlas Mountains in Morocco. In this seasonal breeder, short autumnal photoperiod is correlated with high pineal and plasma melatonin, which induces low testosterone concentrations. In these conditions, the suprachiasmatic nucleus metabolic activity is high. We thus investigated whether melatonin and testosterone would act as non-photic cue on the SCN-driven general locomotor activity recorded by the Circadian Activity Measuring System (CAMS). Fifty sexually active (SA) male jerboas caught in the field in early spring were maintained in animal facilities for two weeks, then divided in 3 groups: SA controls, gonadectomised, and gonadectomised with testosterone implant. Locomotor activity was recorded in constant Long Photoperiod for two weeks, then in darkness (DD) for 3 months. All parameters of the rhythms were analysed with Clock Lab software. Actograms show that locomotor activity is perfectly synchronized to 24h by light/dark cycle in all animals. Under constant darkness, the rhythm was fundamentally circadian with an endogenous period around 23.9 h. Two phenotypes were obtained in the dark. Most animals expressed phase advance in their activity, while a few animals expressed phase delay. The period of the rhythm did not change in the 3 groups; however, the duration of night activity and the global locomotor activity were lower in gonadectomised jerboas. Under darkness, melatonin injected 4 hours before subjective light-on synchronises the locomotor activity rhythm in control animals, expressing phase delay in their activity. Testosterone implants restored this night activity to control levels. Gonadectomised jerboas showed a high onset error when compared to controls and the testosterone group. These results showed that in the SA Jerboa, locomotor activity rhythm is circadian and synchronized to 24h by light/dark cycle. In the Jerboa, the activity rhythm in under the modulatory effect of melatonin and testosterone. Testosterone depressed the quantities of locomotor activity by probably impacting SCN activities. Testosterone didn't however, synchronize the activity of the rhythm in constant darkness.

Key words: Circadian rhythm, locomotor activity, light/dark cycle, darkness, melatonin, testosterone

SP10_2

Title

Thermoregulation Meets Chronobiology: How Temperature, Not Light, Governs Circadian Rhythms in Subterranean Mole-Rats

Authors

Nigel C. Bennett; Kerryn L. Grenfell; Barry van Jaarsveld; Daniel W. Hart. Mammal Research Institute, Department of Zoology & Entomology, University of Pretoria, Pretoria, South Africa

Abstract

Traditionally, circadian rhythms in mammals have been thought to be primarily entrained by light, but recent research highlights the increasingly recognized role of ambient temperature (Ta) as a powerful environmental cue. In our study, we investigated Ta as a key zeitgeber for locomotor activity (LA) in several species of subterranean African mole-rats, which live in environments with minimal light exposure. By subjecting these species to varying Ta cycles and light conditions, we found that LA was predominantly synchronized with the Ta cycle, demonstrating strong thermal entrainment even in the absence of light. When both Ta and light cues were presented, Ta consistently emerged as the primary entrainer, overriding light cues. To explore the evolutionary drivers of this pattern, we investigated the role of thermoregulatory traits, such as heat dissipation abilities, in shaping temperature entrainment of circadian rhythms. By artificially altering the thermoregulatory capacity of mole-rats through modifications to insulation, we observed a reduction in the strength of Ta as a zeitgeber. This further supports the hypothesis that a species; sensitivity to temperature fluctuations influences its circadian regulation. Our findings challenge the long-standing dominance of light as the primary circadian cue in mammals and emphasize the critical role of temperature. This research advances our understanding of the intricate link between chronobiology and thermoregulation, suggesting that temperature may play a more significant role in mammalian circadian rhythms than previously acknowledged.

Symposia

SP10_3

Title

Life under the tropics: sex-specific and photoperiod-dependent regulation of metabolism in the grey mouse lemur (*Microcebus murinus*)

Authors

Jérémy TERRIEN

Abstract

Symposia

SP10_4

Title

Effects of the Onset of Rumination on Sleep/Wake Architecture in Camel and Sheep Newborns

Authors

BENAICH Younes1, FARSI Hicham1, PIRO Mohammed1, ACHAABAN Mohamed Rachid1, PEVET Paul2, CHALLET Etienne2, SATTE Amal3, and EL ALLALI Khalid1. 1 Hassan II Agronomy and Veterinary Medicine Institute, Morocco. 2 Institute of Cellular and Integrative Neurosciences, CNRS and University of Strasbourg, France. 3 Department of Neurophysiology, Military Hospital Mohammed V, Morocco

Abstract

Wake/sleep architecture changes throughout the lifespan of many species, due to several factors, including ontogeny, anatomical and physiological function development. Ruminants possess an essential component of digestive function, rumination, which is a distinctive trait exclusive to these species. Rumination manifests when the forestomach has reached its full developmental maturity. However, it remains unclear how the onset of this function affects sleep during the transition from a monogastric physiological state (newborn-ruminant) to a polygastric one (young-ruminant having acquired the rumination function). The objective of the present study was to assess the rumination onset impact on vigilance states in two ruminant species. Neonates of dromedary camel (n=3) and sheep (n=3) were subjected to two non-invasive sessions of polysomnographic (PSG) and behavioral recording. The initial session was conducted immediately following the animals' birth and spanned 72 consecutive hours (3 days and 3 nights). The subsequent session was initiated after acquiring the rumination function (1-1.5 months postnatal) and entailed a similar 72-hour continuous recording period. Immediately after birth, sheep and camel newborns showed polyphasic sleep, divided between nocturnal and diurnal periods. The animals spent 48.4% and 45.6% of the 24-hour in sleep respectively, of which 10.2% and 12.1% in drowsiness, 27.1% and 25.1% in NREM-sleep, and 11% and 8.4% in REM-sleep. Several weeks later, the onset ofrumination was associated with changes in distribution of vigilance states over a 24-hour period, with rumination accounting for 21.0% and 12.8% of total time in sheep and camel neonates respectively. Rumination onset was accompanied by a reduction in the drowsiness percentage to 4.8% and 6.0%, in the NREM-sleep percentage to 19.4% and 15.8%, and in the REM-sleep percentage to 5.7% and 3.8%, respectively. No effect was observed on wakefulness. The results of the present study demonstrate that rumination had a comparable impact on the sleep architecture of both species. This was reflected in a reduction in the proportion of time spent in the different sleep states and, in some cases, in the synchronization of rumination and SWS. Our study suggests that this physiological process in ruminant newborns may interfere or compete with sleep stages without affecting wakefulness.

Symposia

Symposium 11

Natural Products Research, Characterization and Therapeutic Role

Organizer: **Martha Dávila-García**

Howard University College of Medicine, Washington DC USA
email: **mdavila-garcia@howard.edu**

Abstract:

Natural products serve an indisputably important role in the development of therapeutics for treating a variety of neurological and mental conditions. Many of the compounds used today as neurobiologically active, were discovered in nature or modified from natural compounds, others have been synthesized only after careful study of the biological molecules involved in the various diseases and disorders that affect the brain. Humans have used a variety of these natural compounds for centuries, the preparation methods and dispensation of the compounds have been known to communities, but have not received adequate recognition because of a lack of careful and systematic documentation and scientific evidence of their efficacy. We are presenting 5 scientific research studies to demonstrate the characterization and therapeutic uses of some natural compounds.

1. Dr. Okeowo, "Methyl jasmonate characterization in stress-related disorders".
2. Dr. Biney, "Semi-synthetic derivatives of plant diterpene xylopic acid as scaffolds for novel antidepressants".
3. Dr. Da Silva, "Ethnopharmacology of neuroprotective compounds in the state of Bahia, Brazil".
4. Dr. Adebowale, "Exploring derivatives of nicotine as novel targets to inhibit PLK-1-PBD to modulate brain tumors".
5. Dr. Da¡vila-Garcia, "Cannabis and its derivatives; A historical persective of a therapeutic treasure chest".

Speakers

Number	Speaker	e-mail	Title of the communication
SP11_1	Martha Dávila-García	mdavila-garcia@howard.edu	Natural products research, characterization and therapeutic role
SP11_2	Robert P. Biney	robert.biney@ucc.edu.gh	Derivatives of plant diterpene xylopic acid as scaffolds for novel antidepressants
SP11_3	Oritoke M. Okeowo	theoritoke@gmail.com	Methyl Jasmonate Characterization in Stress-Related Disorders
SP11_4	Victor D. A. Da Silva	vdsilva@ufba.br	Ethnopharmacology of neuroprotective compounds in the state of Bahia, Brazil
SP11_5	Adebowale	deboogunjirin@yahoo.co	Exploring Derivatives of Nicotine as Novel Targets

	Ogunjirin	m	to Inhibit PLK-1-PBD to Modulate Brain Tumors

SP11_1

Title

"Natural products research, characterization and therapeutic role"

Authors

Martha I Davila-Garcia, Howard University College of Medicine, Washington, DC USA; Oritoke Okeowo, Federal University of Technology, Akure, Nigeria; **Robert P. Biney**, School of Pharmacy and Pharmaceutical Sciences, University of Cape Coast, Cape Coast, Ghana**; Victor D. A. Da Silva**, Federal University of Bahia, Salvadore, Brasil; **Adebowale Ogunjirin**, Gallaudet University, Washington, DC USA

Abstract

Natural products serve an indisputably important role in the development of therapeutics for treating a variety of neurological and mental conditions. Many of the compounds used today as neurobiologically active, were discovered in nature and used as extracted complexes, modified from natural compounds, or used as lead compounds to produce synthetic medicines. Others have been synthesized only after careful study of the biological molecules involved in the various diseases and disorders that affect the brain. Humans have used a variety of these natural compounds for centuries, the preparation methods and dispensation of these compounds have been known to communities, but have not received adequate recognition because of a lack of careful and systematic documentation and scientific evidence of their efficacy. We are presenting 5 scientific research studies to demonstrate the characterization and therapeutic uses of some such natural compounds. Dr. Davila-Garcia will present "Epibatidine and its derivatives; A historical perspective of a therapeutic treasure chest"; Dr. OrKeowo will talk about the "Methyl jasmonate characterization in stress-related disorders"; Dr. Biney will talk about his research with "Semi-synthetic derivatives of plant diterpene xylopic acid as scaffolds for novel antidepressants"; and Dr. Da Silva will present his recent research on the "Ethnopharmacology of neuroprotective compounds in the state of Bahia, Brazil"; and Dr.Adebowale will discuss "Exploring derivatives of nicotine as novel targets to inhibit PLK-1-PBD to modulate brain tumors".

Symposia

SP11_2

Title

Derivatives of plant diterpene xylopic acid as scaffolds for novel antidepressants & quot

Authors

Robert Peter Biney, Department of Pharmacotherapeutics and Pharmacy Practice School of Pharmacy and Pharmaceutical Sciences, University of Cape Coastt

Abstract

Diterpenes are isoprene molecules with a characteristic four isoprene units that occur in nature and exert several biological activities such as anti-inflammatory action, antimicrobial and antispasmodic activities. The naturing occurring plant diterpene, xylopic acid, is a small molecule with significant potential in drug discovery pipelines for neurological disorders. In this talk we will review semi-sythetic derivatives of xylopic acid and characterise the neuropharmacological and pharmacokinetic properties of four of its derivatives that highlight them as potential scaffolds for novel antidepressants. Their structure activity relationships are discussed and a case is made for increased awareness and research on similar plant diterpenes and their derivatives as scaffolds for future CNS drug discovery platforms.

Symposia

SP11_3

Title

Methyl Jasmonate Characterization in Stress-Related Disorders

Author

Oritoke M. Okeowo Department of Physiology, School of Basic Medical Sciences, Federal University of Technology, Akure, Ondo State, Nigeria 2Laboratory for Experimental and Translational Neurobiology, University of Medical Sciences, Ondo, Ondo State, Nigeria

Abstract

Methyl jasmonate (MJ), a plant-derived cyclopentanone compound, has shown promise as a therapeutic agent in managing stress-related neurological disorders. Research has demonstrated MJ's ability to enhance stress resilience, which is evident in preclinical models such as the forced swim endurance test and anoxic tolerance test. MJ administration significantly delayed the onset of immobility, reduced immobility duration, increased active swimming time, and prolonged the latency to exhaustion, highlighting its adaptogenic properties and potential to modulate energy during stress. MJ's neuroprotective effects extend to its role in modulating neurotransmitter systems and reducing oxidative stress, key factors in the pathophysiology of many neuropsychiatric conditions. Studies have revealed that MJ may exert antidepressant and antipsychotic effects through its impact on stress-related biochemical pathways. By mitigating oxidative damage and influencing neurochemical balance, MJ emerges as a promising candidate for the development of novel therapeutics aimed at treating complex mental health conditions associated with chronic stress. Future research directions include detailed exploration of MJ's molecular mechanisms, optimization of its pharmacokinetic profile for enhanced stability and bioavailability, and rigorous clinical trials to evaluate its safety and efficacy in humans. Additionally, investigations into MJ's synergistic effects with existing therapies could provide insights into its potential for integrated treatment strategies. The findings underscore MJ's therapeutic potential, offering a foundation for its application in managing stress-related disorders and advancing our understanding of natural product-based interventions in neuropsychopharmacology.

Symposia

SP11_4

Title

Ethnopharmacology of neuroprotective compounds in the state of Bahia, Brazil

Authors

Victor D. A. DA Silva1, Florisvaldo Ramos1, Flávia Santos Sanches, Rafael Short Ferreira, Aimeé P. Alves dos Santos, Gloriene C. de Jesus, Suzana Braga, Deise S. Vilas Boas, Clarissa Schitine, Paulo Ribeiro. Affiliantion: Federal University of Bahia, Brazil.

Abstract

Medicinal plants have long been used for stroke therapy in Brazilian folk medicine. Stroke is a leading cause of death and disability worldwide. A major factor in brain damage following ischemia is excitotoxicity caused by elevated levels of the neurotransmitter glutamate. The aim of the present study was to protect neuroprotective compounds derived from medicinal plants used for stroke in black communities in the State of Bahia, Brazil. We interviewed twelve communities that make frequent use of medicinal plants. We prepared extracts from parts of the most commonly used plant, the Amburana cearensis, and tested its neuroprotective action in Pc12 cells. Our results demonstrated that the most bioactive extract, a dichloromethane extract (EDAC) from A. cearensis seeds, is rich in coumarin by 1H and 13C-NMR. Furthermore, we showed that EDAC and isolated coumarin protect PC12 cells against glutamate excess and oxygen-glucose deprivation and prevent the production of ROS in PC12 cells. This study provides evidence that A. cearensis is a medicinal plant commonly used for stroke in Bahia-Brazil, and coumarin from EDAC is cytoprotective against ischemia and glutamate excitotoxicity.

Symposia

SP11_5

Title

Exploring Derivatives of Nicotine as Novel Targets to Inhibit PLK-1-PBD to Modulate Brain Tumors

Authors

Adebowale Ogunjirin School of Science, Technology, Accessibility, Mathematics, & Public Health Hall Memorial Building N447 Gallaudet University, NE Washington

Abstract

Polo-like kinase 1 (PLK1) is a key serine/threonine kinase involved in cell cycle regulation and is frequently overexpressed in aggressive tumors, including brain cancers. Targeting the Polo-box domain (PBD) of PLK1 presents a promising therapeutic strategy by selectively disrupting its scaffolding function, potentially reducing off-target effects. This study investigates the potential of nicotine derivatives as novel inhibitors of PLK1-PBD, aiming to modulate tumor growth through targeted interference with mitotic progression. Using fluorescence polarization binding assays, nicotine analogs was screened for their affinity toward PLK1-PBD, with thymoquinone as a reference inhibitor. Our accelerated screening identified key candidates exhibiting significant binding affinity, with subsequent competition assays revealing IC50 values indicative of promising inhibitory activity. Future studies will involve computational docking and structure-activity relationship (SAR) analysis to further characterize ligand-protein interactions and refine these inhibitors for enhanced potency and selectivity. These findings lay the groundwork for developing nicotine-based small-molecule inhibitors as potential therapeutic agents against brain tumors, warranting further preclinical evaluation.

Symposia

NWG-sponsored
Symposium 12
Brain Pathologies - Neuron and Glia Diversity in Regeneration

Organizer: **Frank Kirchhoff** & **Luciana Politti Cartarozzi**

Centre for Behavioural Sciences and Mental Health, Instituto Superior di Sanita, Italy
Department of Neuroscience, University of Rochester, New York, USA
email: **frank.kirchhoff@uks.eu; lpcarta@unicamp.br**

Abstract:

On behalf of the German Neuroscience Society (NWG), I am proposing a symposium for SONA 2025 titled "Brain Pathologies - Neuron and Glia Diversity in Regeneration." This symposium will bring together a diverse group of scientists from four continents, each contributing unique insights into brain pathologies and regeneration. Aligned with the conference theme "Brain Health and Environment: The Challenge of the Future," we will explore how neuron-glia interactions and brain organoids can inform future therapies for neurological diseases. The symposium will be co-chaired by myself, Prof. Frank Kirchhoff (University of Saarland, Germany), and Dr. Luciana Politti Cartarozzi (University of Campinas, Brazil), who will also present her research on "Exploring glial and immune mechanisms in spinal motoneuron regeneration." Our speaker lineup reflects a strong commitment to diversity: Dr. Mubeen Goolam (University of Cape Town, South Africa) will present "Developing African origin brain organoids as a genetically diverse preclinical model of neurological disease," focusing on stem cell models for neurological conditions like epilepsy. Dr. Priscilla Kolibea Mante (Kwame Nkrumah University of Science and Technology, Ghana) will discuss "Harnessing Phytochemicals for Neuroprotection," highlighting plant-based compounds for treating epilepsy, anxiety, depression, and pain.
Ms. Zainab Faik (University of Zurich, Switzerland), originally from Morocco, will present "Fueling the White Matter: Distinct Metabolism in Oligodendrocytes, Astrocytes, and Axons," using advanced imaging techniques to explore brain energy metabolism. Dr. Xianshu Bai (University of Saarland, Germany) will conclude with "The role of neuron-oligodendrocyte communications in the developing mouse brain and during remyelination processes."
This symposium showcases cutting-edge research while emphasizing global representation in neuroscience. By including speakers from Africa, Europe, Asia, and South America, we aim to foster international collaboration and highlight diverse perspectives on brain health challenges. Our multidisciplinary approachâ€”spanning organoid models, neuroimmunology, phyto-chemistry, and glial biology will engage a broad audience and contribute to advancing neuroscience research in Africa.

Symposia

Speakers

Number	Speaker	e-mail	Title of the communication
SP12_1	Priscilla Kolibea Mante	pkmante.pharm @knust.edu.gh	Harnessing Phytochemicals for Neuroprotection.
SP12_2	Mubeen Goolam	mubeen.goolam @uct.ac.za	Developing African origin brain organoids as a genetically diverse preclinical model of neurological disease.
SP12_3	Luciana Politti Cartarozzi	Lpcarta @unicamp.br	Exploring glial and immune mechanisms in spinal motoneuron regeneration: insights from the ventral root crush model
SP12_4	Zainab Faik	zainab.faik @pharma.uzh.ch	Fueling the White Matter: Distinct Metabolism in Oligodendrocytes, Astrocytes, and Axons
SP12_5	Xianshu Bai	xianshu.bai@uks.eu	Plasticity of oligodendrocyte lineage cells during development and acute brain injury

Symposia

SP12_1

Title

Harnessing Phytochemicals for Neuroprotection

Authors

Priscilla Kolibea Mante, Department of Pharmacology, Kwame Nkrumah University of Science and Technology, Kumasi, Ghana

Abstract

Neurodegenerative disorders such as Alzheimer's, Parkinson's, and stroke remain major challenges due to the limited regenerative capacity of the central nervous system (CNS). These diseases are driven by oxidative stress, neuroinflammation, and impaired glial support, leading to progressive neuronal loss. Research from our lab investigates the neuroprotective effects of phytochemicals isolated from indigenous Ghanaian plants, such as Cryptolepis sanguinolenta and Pseudospondias microcarpa. The objective is to elucidate how these compounds modulate neuroinflammation, oxidative stress, and neurotrophic mechanisms to support neuronal regeneration. Methods: Phytochemicals were isolated and characterized using chromatographic methods. Their antioxidant and anti-inflammatory properties were evaluated using in vitro assays, including ROS scavenging and cytokine inhibition tests. In vivo models of neurodegeneration and chronic stress were employed to assess behavioral, biochemical, and molecular outcomes. Zebrafish models were used to examine anxiolytic and anti-stress effects. Techniques such as immunohistochemistry and enzyme-linked immunosorbent assays (ELISA) were employed to analyze glial activation and neurotrophic factor expression. Key molecules like brain-derived neurotrophic factor (BDNF) and glial cell-derived neurotrophic factor (GDNF) were measured to validate neuroregenerative effects. NMR-based metabolomics were used to measure drug action. Results: Cryptolepine and flavonoid-rich extracts demonstrated significant antioxidative and anti-inflammatory effects, including downregulation of TNF-? and IL-6. Behavioral studies showed that Pseudospondias microcarpa extracts reversed stress-induced deficits and modulated GABAergic and serotonergic pathways, promoting anxiolytic effects. Enhanced glial activation created a favorable microenvironment for neuronal survival, while upregulated BDNF and GDNF expression facilitated synaptic repair and neurogenesis. These compounds improved the neuronal microenvironment, promoting axonal regeneration and reducing neuronal death. Discussion: Phytochemicals offer a multifaceted approach to neuroprotection by addressing oxidative stress, inflammation, and neurotrophic deficits. Their role in modulating glial and neuronal pathways underscores their potential as cost-effective, sustainable therapies for neurodegenerative diseases. These findings highlight the promise of integrating phytochemicals into therapeutic strategies, particularly in resource-constrained settings.

Keywords: Phytochemicals, Neuroprotection, Neuronal Regeneration, Oxidative Stress

Symposia

SP12_2

Title

Developing African origin brain organoids as a genetically diverse preclinical model of neurological disease

Authors

Mubeen Goolam Room 6.02.3, Level 6, Anatomy Building

Abstract

In recent years there has been a rapid development of stem cell-based models of neurological development. These model systems, collectively known as brain organoids, have generated much interest for their ability to overcome the limitations of other models and their potential to be the definitive system within which to study neural development and disorder. Brain organoids are generated as 3D in vitro aggregates of human neurological tissue derived from human pluripotent stem cells that can model the human brain microenvironment. Critically, cerebral organoids can be derived from induced pluripotent stem cells (iPSCs). allowing for the possibility of generating patient specific brain organoids and represents a powerful approach to study the genetic, proteomic, and structural changes caused by disease pathogenesis with little to no little to no ethical drawbacks. In the context of critical brain health challenges, particularly those faced on the African continent, brain organoids present as a uniquely accessible and physiologically relevant model system within with we can develop crucial insights into the disease pathogenesis and potential treatment options for brain diseases. Despite their immense potential, cerebral organoids, have yet to be developed in any African university or institution and, critically, never from iPSCs of African origin. Our research focuses on generating African origin iPSCs and using them to develop novel models of neurological development. We make use of various signalling molecules and ligands and investigate how they drive anterior differentiation in iPSCs. We show that Wnt signalling has a posteriorising effect on stem cells and directs differentiation towards the mesoderm while N2/B27 can mitigate these effects and drives anterior differentiation. Critically we find that BMP4 signalling as a pulse can drive neural differentiation, while signalling centresresulted in better recapitulation of aspects of anterior-posterior axis formation. With the immense genetic diversity within African populations and the important role this can play in both disease progression and treatment there is significant scope to develop African specific brain organoids to investigate brain disease and development. Our findings are the first steps in generating diverse brain organoids that can be used as a preclinical model of neurodevelopmental diseases.

Symposia

SP12_3

Title

Exploring glial and immune mechanisms in spinal motoneuron regeneration: insights from the ventral root crush model

Authors

Luciana Cartarozzi, University of Campinas, SP/Brazil.

Abstract

In the context of motor system repair, the ventral root crush (VRC) model has emerged as a valuable experimental tool that provides a controlled and reproducible platform to selectively study the degenerative and regenerative dynamics of motoneurons, and their interactions with glial cells. Herein, the time course of motoneuron degeneration and microglial and astroglial responses after VRC will be demonstrated. Furthermore, the influence of immune molecules, such as MHC-I, TLR2, and TLR4, in the context of motoneuron degeneration and glial responses will be presented. In addition, the VRC model will be explored as a preclinical platform for testing neuroprotective and pro-regenerative therapies, with results from stem cell-based interventions and pharmacological modulation. By elucidating neuron-glia interactions and therapeutic strategies, the VRC model bridges basic research and clinical applications, allowing insights into the mechanisms underlying neuronal and glial diversity in regeneration.

Symposia

SP12_4

Title

Fueling the White Matter: Distinct Metabolism in Oligodendrocytes, Astrocytes, and Axons

Authors

Zainab Faik[1], konstantin satushev[1], jeanne droux[1], Jan Dernic[1], Luca Ravotto[1], Bruno Weber1,2; Aiman S. Saab[1,2]. 1- University of Zurich, Institute of Pharmacology and Toxicology, Zürich, Switzerland 2- Neuroscience Center Zurich, University and ETH Zurich, Zurich, Switzerlandand

Abstract

Axonal integrity is crucial for brain function. However, the mechanisms by which oligodendrocytes and astrocytes support axonal health in white matter, especially their metabolic interactions and energy sources, remain poorly understood. In this study, we used two-photon imaging of the optic nerve alongside compound action potential (CAP) recordings to monitor both cellular ATP homeostasis and axonal spiking activity. We developed novel adeno-associated virus (AAV) delivery strategies to express the FRET-based ATP sensor Ateam1.03 specifically in optic nerve oligodendrocytes or astrocytes. Our findings revealed distinct metabolic capacities in glial cells and axons. Both oligodendrocytes and astrocytes were able to maintain some ATP levels in the absence of glucose, while axonal conduction ceased entirely. Oligodendrocytes were more efficient in switching to lipid metabolism during aglycemia compared to astrocytes, which required more time to initiate this pathway. We also identified differences in the ability of axons and glial cells to metabolize other substrates for ATP production. Our findings suggest that both oligodendrocytes and astrocytes can metabolize fatty acids or other substrates during hypoglycemia, potentially allowing the precious glucose to be delivered to axons to support critical antioxidant functions. Such glial metabolic flexibility, and possibly their glucose-sparing capacity, could be impaired in white matter diseases or in inflammatory conditions, which requires further research.

Symposia

SP12_5

Title

Plasticity of oligodendrocyte lineage cells during development and acute brain injury

Authors

Xianshu Bai Molecular Physiology, CIPMM, Geb. 48, Saarland niversity, 66421 Homburg, Germany.

Abstract

OPCs and oligodendrocytes (OLs) exhibit heterogeneity in their location, function, and origin. However, whether their responses to acute brain injuries are similarly heterogeneous remains unclear. Recently, we demonstrated that a subset of OPCs temporarily downregulate the expression of the Olig2 transcription factor, previously thought to be ubiquitously expressed in OPCs. Unlike Olig2-expressing OPCs, these Olig2-negative OPCs exhibit limited proliferation and simpler morphologies. Interestingly, one week after injury, these cells begin to re-express Olig2, indicating a high degree of OPC plasticity in response to injury. Moreover, through the use of multiple transgenic mouse models and in vivo two-photon laser scanning microscopy (2P-LSM), we revealed that a subset of mature OLs in the mouse cortex undergoes plastic changes and gives rise to astrocytes following acute injury. Notably, we identified a transitional cell stage termed AO cells (cells exhibiting both astrocytic and oligodendroglial properties). These AO cells arise from oligodendrocytes and have the capacity to differentiate into astrocytes. Our findings suggest a significant plasticity within the oligodendrocyte lineage and offer potential therapeutic avenues for acute brain injuries by targeting these cells.

Symposia

Symposium 13

Emerging regenerative medicine for CNS repair

Organizer: **Fatiha Nothias** & Afsaneh Gaillard

CNRS UMR8246/Inserm U1130, Sorbonne Université, Paris
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Abstract:

In humans, traumatic, neurovascular, and neurodegenerative diseases have significant social consequences and can lead to life-long disability. This is due to the limited ability of the adult CNS for self-repair after injury, also linked to the complexity of these progressive diseases. This symposium will focus on recent progresses in CNS repair, addressing challenges such as limited axonal regeneration, poor revascularization, scarring, demyelination, and chronic inflammation. Despite the lack of available effective treatments, research is making progress in areas like immune modulation, axon regeneration, remyelination, and cell-based therapies. Physical activity-based therapies and neuromodulation, that reached the clinical practice, have also shown promise in re-establishing neuronal networks, further advancing neuro-restoration efforts.

This symposium will bring together leading experts to provides a comprehensive overview of the current challenges and advancements in CNS repair. As the field progresses, it will also emphasize on the combination of approaches with cutting-edge technologies such as bioengineering, stem cell therapies and neuromodulation that offer great potential for CNS repair solutions in the near future: Loubinoux, will describe the effect of "Regenerative implants in Stroke and TBI"; A. Gaillard, will share her recent data on combined strategies to treat TBI in animal models; "Stem cells and biomaterial-based regenerative therapeutic strategies for the treatment of cortical injury"; I. Vivodtzev, will talk about Combinatorial therapies to improve respiratory recovery after cervical SCI: from bedside to pre-clinical studies and back to human translation; the title will be "Neurostimulation and respiratory recovery after cervical spinal cord injury"; J. Martin, will describe the advantage of "Combining Biomaterial Scaffold and Neuromodulation Strategy to Promote Tissue Repair and Corticospinal Connectivity after Spinal Cord Injury".

Finally, Neuromodulation has emerged as a promising therapeutic approach for managing symptoms of Parkinson's disease; thus A. Benazzouz will give us "An update on the pathophysiology and treatment of Parkinson's disease".

Symposia

Speakers

Number	Speaker	e-mail	Title of the communication
SP13_1	Abdelhamid Benazzouz	abdelhamid.benazzouz @u-bordeaux.fr	Neuromodulation and an update on the pathophysiology and treatment of Parkinson's disease
SP13_2	Isabelle Loubinoux	isabelle.loubinoux @inserm.fr	Regenerative implants in Stroke and TBI
SP13_3	Afsaneh Gaillard	afsaneh.gaillard @univ-poitiers.fr	Combination of biomaterial and stem cell-based strategies to improve cortical repair
SP13_4	Isabelle Vivodtzev	isabelle.vivodtzev @sorbonne-universite.fr	Neurostimulation and respiratory recovery after cervical spinal cord injury
SP13_5	John Jack H Martin	Jmartin @med.cuny.edu	Combined Biomaterial Scaffold and Neuromodulation Strategy to Promote Tissue Repair and Corticospinal Connectivity after Cervical Spinal Cord Injury.

Symposia

SP13_1

Title

Neuromodulation and an update on the pathophysiology and treatment of Parkinson's disease

Authors

Abdelhamid Benazzouz, Keri-Ann Charles*, Elba Molpeceres Sierra*, Rabia Bouali-Benazzouz*, Houyam Tibar, Khalid Oudaha, Frédéric Naudet, Alexia Duveau, Pascal Fossat and Abdelhamid Benazzouz (* Equal contribution)

Abstract

Parkinson's disease (PD) is a neurological disease characterized by the manifestation of motor and non-motor symptoms. Over several decades, a considerable amount of research in animal models, particularly rodents and non-human primates, and in patients has contributed to our understanding of the pathophysiology of the motor symptoms of PD and the development of new therapeutic approaches. Numerous studies have demonstrated the key role of the subthalamic nucleus (STN) in the manifestation of these symptoms, and their alleviation by high frequency electrical stimulation of this nucleus. Recently we also discovered that the STN is also involved in the pathophysiology of the non-motor symptoms of PD, including pain which is affecting around 85% of patients and contributing to a deterioration in their quality of life. Using in vivo extracellular electrophysiology, we have shown that STN neurons are able to detect nociceptive stimuli, encode their intensity and generate windup-like plasticity. However, these phenomena are impaired in dopamine-depleted animals. Indeed, the intensity response is altered in both STN and wide dynamic range (WDR) neurons of dorsal horn of the spinal cord (DHSC). Moreover, neuromodulation of STN by high frequency electrical stimulation, named also deep brain stimulation, in dopamine-depleted animals showed an improvement in mechanical allodynia and thermal hyperalgesia compared to sham animals. This effect is mediated by descending brainstem projections leading to normalization of nociceptive integration in DHSC neurons. Our study highlights the centrality of the STN in nociceptive circuits, its interaction with the DHSC and its key involvement in pain sensation in Parkinson's disease. Furthermore, our results provide for the first-time evidence that subthalamic DBS produces analgesia by normalizing the responses of spinal WDR neuronsvia descending brainstem pathways.

Symposia

SP13_2

Title

Regenerative implants in Stroke and TBI

Authors

Isabelle Loubinoux, ToNIC, Toulouse, France **; Clauzel Julien, Colitti Nina, Combeau Maylis, Labriji Wafae, Robert Lorenne, Brilhault Adrien, Cirillo Carla, Desmoulin Franck**. LabHPEC, Université de Toulouse, ENVT, Toulouse, France : Raymond-Letron Isabelle.

Abstract

Background: The limited capacity of brain tissue to regenerate after acute injury, hampered by cell death, edema and inflammation, has led to an interest in promising and innovative approaches such as implantable regenerative scaffolds designed to improve brain plasticity. Either guiding or non-guiding, degradable or non-degradable, fabricated by molding or 3D printing, these scaffolds can be tailored to match the intricate architecture of the brain. Methods: we performed in vivo biocompatibility assessments after a brain lesion on distinct biomaterials, a non-degradable one, silicone (PDMS) and bioeliminable or bioresorbable materials: Poly (ethylene glycol) diacrylate (PEGDA), Polycaprolactone (PCL) and a PEGDA mixed with gelatin methacrylate (PEGDA-GelMA). These implants were inserted in a brain lesion, one week after the insult. Results: We made twice the proof of concept the guiding PDMS implants seeded with neural cells improved grip force in rats (1,2). They improved formation of neotissue within the lesion and neovascularization (3,4). Security of this therapeutic strategy was proven: no tumor, no fibrosis, no inflammation, and a reduced glial reaction was observed. With 3D printing, a scaffold with a complex shape was printed with patterns, spatial resolution and porosity adapted to cerebral cortex reconstruction (5). In vivo evaluations were complemented by behavioral monitoring, affirming the safety of these degradable materials. High-resolution T2 MRI imaging effectively captured scaffold structures and demonstrated their non-invasive utility in monitoring degradability. ASL MRI imaging quantified cerebral blood flow and was positively and significantly correlated with lectin immunofluorescent labeling. It may be used to non-invasively monitor progressiverevascularization of implants. PEGDA produced an intense foreign-body response, PCL provoked a controlled inflammatory reaction and facilitated cell migration into the scaffold, although it induced a fibrotic response. Conversely, the PEGDA-GelMA composite emerged as a promising candidate for intracerebral implantation. Conclusion: Behavior, MRI monitoring and histology allowed a thorough following of biomaterial biocompatibility. The collective findings position PDMS and PEGDA-GelMA as convincing biomaterial options as a basis for treating severe brain lesions. 1. Vaysse. Biomaterials 2015; doi: 10.1016/j.biomaterials.2015.04.019. 2. Davoust, StemCellRes&Ther 2017. DOI 10.1186/s13287-017-0702-3. 3. Le Friec, NeuralPlasticity 2017. DOI 10.1155/2017/2545736 4. Accardo; BrainResearchBulletin. 2019. DOI 10.1016/j.brainresbull.2019.07.020 5. Clauzel, RegenerativeTherapy 2024 doi: 10.1016/j.reth.2024.10.004

Symposia

SP13_3

Title

Combination of biomaterial and stem cell-based strategies to improve cortical repair

Authors

Afsaneh Gaillard, Annousha Devi Govindan, Oriane Rabesandratana, Anaïs Lainé, Antoine Retho, Marie-Laure Bonnet, Sébastien Brot, Afsaneh Gaillard, Laboratory of Experimental and Clinical Neurosciences INSERM1084, FRANCE

Abstract

Traumatic brain injury (TBI) is among the leading cause of death and disability with limited treatment options available. Given the limited capacity of the adult brain for self-repair, cell transplantation is a potential strategy to repair the degenerated brain pathways following TBI. One clinically relevant cortical tissue in the context of cortical lesion is the motor cortex, however, there is no reliable protocol to generate cortical neurons of motor identity. Recently, we have generated a 3D motor cortical neurospheres (mCO) from human induced pluripotent stem cells (hiPSC) using a well-established protocol in our laboratory. These mCO grafted into injured motor cortex of adult Rag2??? mice, develop axonal projections to appropriate cortical and subcortical host targets. However, a major limiting factor after transplantation is cell death of grafted neurons. To enhance graft survival and functional recovery post TBI, mCO were embedded with hyaluronic acid (HA)-based hydrogel at day 18 of differentiation in vitro. In the present study, we investigated the impact of HA-hydrogel on cell survival and maturation of hiPSC derived motor cortical neurons in vitro and in vivo after transplantation. We first analyzed in vitro the cellular composition of generated cortical neurons in combination with or without HA-Hydrogel at day 46. In vitro, we found that the HA-hydrogel romotes the survival and the maturation cortical neurons derived hiPSC. Next, we grafted mCO treated +/- HA-Hydrogel into injured motor cortex of adult Rag2??? mice. Interestingly, we found that 2 months after transplantation, the combination of HA-Hydrogel with mCO improves dramaticallythe connectivity of the grafted neurons in term of density and the extent of repair. These results reported a beneficial effect of HA-hydrogel on hiPSC-derived mCO in vitro and in vivo after transplantation. Further studies are underway to assess long-term connectivity and functionality of the grafted neurons.

SP13_4

Title

Neurostimulation and respiratory recovery after cervical spinal cord injury

Authors

Isabelle Vivodtzev, Thibaut Coustillet, Wei Chen, Sylvia Soares & Fatiha Nothias. Affiliation: Dev2A, Thibaut Coustillet, Wei Chen, Sylvia Soares & Fatiha Nothias. Affiliation: Dev2A, CNRS UMR8263, Inserm U1345, Institute of Biology Paris Seine, IBPS, Sorbonne Université, Paris, France

Abstract

A majority of spinal cord injury (SCI) happen at a cervical level, resulting in a marked respiratory dysfunction. When injury is high (4th cervical or above), disruption of the spinal cord integrity not only paralyzes the limb but it alters phrenic nerves which innervate the diaphragm, the main inspiratory muscle. Survivors can be rendered ventilator dependent, a sequela that dramatically compromises quality-of-life and increases mortality rate. Today, mechanical ventilation (MV) is the only treatment available for these patients to ensure breathing. Alternative solutions such as diaphragm pacing or nerve transfer approaches are strongly limited by MV-induced atrophy of the respiratory muscles over time. Only one patient out of ten has sufficient phrenic conduction and diaphragmatic function to benefit from it. In our lab, one current project is to develop and optimize neuromodulation techniques to improve respiratory recovery after cervical SCI. Based on clinical observation, we have developed a sensori-motor stimulation allowing to indirectly preserve diaphragm activity and restore ventilatory capacity in a mouse model of tetraplegia1. This approach aims at reactivating the lost connections with a breathing-synchronized neuromuscular electrical stimulation algorithm for intercostal and abdominal muscles (rSynES), built on mathematical modeling of ventilatory flow, adjusted to mirror real ventilation patterns2. This symposium will present current approaches for treating respiratory impact of cervical SCI and how this innovative concept could be promising for future treatment of respiratory deficits in patients with tetraplegia and respiratory deficiency. In particular, we will discuss how it could promote respiratory recovery when combined to other neuromodulation approaches such as repetitive trans-spinal magnetic stimulation, known to increase the corticospinal network3, ortissue engineering, known to improve spinal tissue restoration and neuro-inflammation4. The ultimate goal is to treat respiratory deficits in patients with SCI to deliver patients from whole life respirator dependency. 1. Bajjig A. et al. Biology 2022, 11, 558. doi.org/10.3390/biology11040558 2. Coustillet T. *et al*. European Respiratory Journal, Abstarct of the ERS conference, (2021) 3. Michel-Flutot P. et al., Respiratory Physiology & Neurobiology 292 (2021) 103704 4. Chedly J. *et al.* Biomaterials 138, 91-107 (2017).

Symposia

SP13_5

Title

Combined Biomaterial Scaffold and Neuromodulation Strategy to Promote Tissue Repair and Corticospinal Connectivity after Cervical Spinal Cord Injury

Authors

John Martin Williams[1], Ahmanna C.[2], Kante K.[2], Soares S.[2], Sharif H.[1-3], Zareen N.[1], Nothias F.[2]. 1-City University of New York School of Medicine; 2-Sorbonne Université Sciences; 3-Merz Therapeutics

Abstract

Spinal cord injury (SCI) damages tissue at the trauma site, producing chronic motor, sensory, autonomic impairments. The damage triggers neurodegeneration and inflammation and produces vascular damage that leads to further impairments. Axonal injury interrupts motor and sensory signaling and disrupts constitutive activity-dependent processes, resulting in changes in spinal neuron excitability, sprouting of spared axons, and trans-neuronal degeneration. Although numerous strategies have been developed to improve recovery after injury, there remains a tremendous gap in functional restoration after cervical SCI. I will present novel approaches for treating the injury site and the loss of neural connections after SCI. Injury site repair is targeted using a uniquely engineered biomaterial Scaffold-Chitosan Fragmented Physical Hydrogel Suspension (CFPHS) that is injected directly into the site of trauma. Our studies show this stimulates local tissue remodeling, including lesion cavity and glial scar abatement and axon outgrowth (1,2). Promoting functional neural connections after SCI is targeted with electrical neuromodulation to stimulate the motor cortex, the origin of the corticospinal tract, and the cervical spinal cord. Motor cortex stimulation, using the theta burst protocol, activates the corticospinal system and promotes corticospinal tract axon outgrowth and long-term potentiation (LTP) (3,4). Spinal cord stimulation, using targeted direct current stimulation, enhances the actions of motor cortex stimulation. I will first discuss the separate actions of the biomaterial scaffold on cervical SCI repair and neuromodulation, on promoting CST axon outgrowth and producing a highly persistent form of muscle response LTP. Then I will present recent findings when the biomaterial and neuromodulation are combined (2). This combined approach offers key benefits for translating a novel SCI therapy to improve motor recovery. 1. J. Chedly et al. Biomaterials 138, 91-107 (2017). 2. P. Williams et al. Exp Neurol 382, 114965 (2024). 3. N. Zareen et al. Proc Natl Acad Sci U S A 121, e2408508121 (2024). 4. A. Amer et al. Proc Natl Acad Sci U S A 118, DOI: 10.1073/pnas.2113192118 (2021).

Symposia

Symposium 14

The signaling system of the neuropeptide relaxin 3/rxfp3 in cognitive and pain processes

Organizer: **Monica Navarro & Olucha Bordonau (Spain)**

Universitat Jaume I, Av Vicent Sos Baynat sn, Castellón, Spain
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Abstract:

The modulation of forebrain activity allows to an adaptation of the ongoing behavior to several environmental conditions. The nucleus incertus in the pontine tegmentum displays a widespread system of ascending projections to forebrain areas involved in emotional and cognitive processing.

These areas include the cingulate cortex, the hippocampus, the medial septum and the supramammillary nucleus. As the nucleus incertus contains a high concentration of the corticotropin releasing factor-receptor 1, this nucleus could play a role of an extended modulation of the forebrain function under a stress condition. Cells in the nucleus incertus produce and delivers through axon terminals the neuropeptide relaxin3 which is the ligand of the RXFPÂ·G-protein coupled receptor.

Interference of this signaling system has an impact on the hippocampal theta rhythm associated with learning processes, disruption of anxiety, social behavior and food and alcohol intake. In this symposium, F. Olucha-Bordonau will give a general overview of the system and its evolutive meaning. M. Zahran will provide data on the effect of the alcohol withdrawal on the aggressive behavior related to relaxin3 expression in forebrain areas and the relationship to c-fos expression in these areas. M. Navarro-Sanchez will provide new data on relaxin3 activation in the retrosplenial cortex and its impact on contextual fear conditioning acquisition, retrieval and extinction. Finally, S. Scoffier will analyze the effect of interfering relaxin3/rxfp3 transmission in the anterior cingulate cortex and in the amygdala on mechanical and thermic pain. The aim of this symposium is to provide data about the feasibility of the relaxin3/RXFP3 as a potential target to treat cognitive and emotional disfunctions.

Speakers

Number	Speaker	e-mail	Title of the communication
SP14_1	Francisco Olucha Bordonau	Folucha @uji.es	The evolutive meaning of the relaxin3-rxfp3 system and new ways on cognition, emotion and metabolic processing
SP14_2	Mohamed Zahran	Zahran @uji.es	Peptidergic systems involved in aggressive processes after alcohol intoxication
SP14_3	Solene Escoffier	solene.escoffier @u-bordeaux.fr	Neuroanatomical basis for the anti-nociceptive role of the relaxin-3/RXFP3 peptidergic system
SP14_4	Monica Navarro-Sánchez	Monavarr @uji.es	Neuropeptidergic Modulation and Neural Pathways in Contextual Fear Conditioning and Extinction: Insights from the Retrosplenial Cortex and Nucleus Incertus

Symposia

SP14_1

Title

The evolutive meaning of the relaxin3-rxfp3 system and new ways on cognition, emotion and metabolic processing.

Authors

Olucha-Bordonau F E[1,2]; Navarro-Sánchez M[1,2]; Gil-Miravet I[1,2]; Zahran MAE[1,2]; Hidalgo-Cortes J[1,2],; Mañas Ojeda A[1,2]; Castillo-Gómez E.[1,2] 1-Department of Medicine, School of Medical Sciences, Universitat Jaume I, 12071 Castelló de la Plana, Spain. 2- Spanish National Network for Research in Mental Health (CIBERSAM), Instituto de Salud Carlos III, Madrid, Spain

Abstract

Relaxin3 was the latest neuropeptide of the relaxins family to be discovered and indeed was the most conserved in the vertebrate evolution. Nearly all animal clades have orthologs of the insulin and relaxin peptide genes and its receptors. The superfamily of the insulin and relaxin peptides is composed of three groups, i.e. insulins, insulin-like growth factors (IGF) and relaxins. All these peptides are synthetized as a pre-propeptide containing the N-terminal signal peptide and the propeptide which consists of chains B and A separated by the C-peptide. In the insulin-like growth factors, the short C-peptide is retained. By contrast, the C-peptide is removed in insulins and relaxins thus producing the basic structure of two chains joined by disulphide bonds. In vertebrates, insulin and insulin-growth factor signaling pathways are involved in metabolism regulation, growth and ageing while relaxin family peptides deal with a wide variety of functions ranging from vascular modulation, cardiac regulation reproduction and neural function. Insulins and IGFs bind to receptors tyrosine kinase (RTKs) while relaxins bind to G-protein coupled receptors (GPCR). There are two types of GPCR, leucine rich GPCR (LGR) and small peptide GPCR (spGPCR). This same basic scheme is also followed in non vertebrates. Drosophila contains at least eight Drosophila insulin liker peptides (DILP1-8). DILP8 is mainly expressed in neural tissue and regulates lifespan, growing and molting. In addition, DILP8 activates LGR3 and thus could be considered as a relaxin orthologue. Cells expressing Relaxin3 in teleosts and mammals occupy similar brain locationswhich is mainly the nucleus incertus in the brainstem. While different fish taxa have duplicated both relaxin3 and its receptor rxfp3, in mammals only one relaxin3 and rxfp3 are retained. The mammalian nucleus incertus provides a widespread system of connections over hypothalamic and telencephalic areas involved in cognitive and emotional functions. The strong projections over the interpeduncular, supramammillary, medial septum, hippocampus, and retrosplenial cortex led to an involvement of this system in a modulatory effect over memory formation. A direct connection over the medial amygdala and prefrontal cortex also provides new vistas on an important effect on social and emotional behavior.

Symposia

SP14_2

Title

Peptidergic systems involved in aggressive processes after alcohol intoxication

Authors

Mohamed Aly Zahran, Mónica Navarro-Sánchez, Esther Castillo-Gómez, Francisco E Olucha-Bordonau. School of Medical Sciences, Spain. Universitat Jaume I

Abstract

Alcoholism has been linked extensively to violence and aggressive behavior. About half of all violent crimes and sexual assaults are committed under the influence of alcohol. However, the underlying neuroanatomical mechanism of this connection remains elusive. The role of the Nucleus Incertus in addiction and relapse of alcoholism has already been demonstrated in animal models. Its Relaxin 3 expressing cells project towards telencephalic centers that control important aspects of motivation and emotions, including the Amygdala and Septum. Relaxin3 signaling was shown to regulate alcohol intake and relapse-like behaviors. Moreover, previous research in our group points to the somatostatin neurons in the medial amygdala as a relevant regulator of this behavior. In this work, we have investigated sex-specific behavioral alterations after chronic alcohol intoxication. Also, we have considered neuronal activity changes, Relaxin 3 levels, and introduced an emerging therapeutic target. Only male mice showed increased aggressive and dominant behavior after chronic alcohol intoxication during acute but not during protracted withdrawal periods. This transient increase was parallel to significantly lower levels of c-Fos in several brain areas of alcohol-intoxicated males, and these levels were restored during protracted withdrawal. Additionally, Relaxin 3 expression showed an increase during females' acute withdrawal and males' protracted withdrawal, which indicates its potential involvement in mitigating withdrawal-induced aggression and restoring behavioral homeostasis. The observed correlations between Relaxin-3 levels, cFos activity, and offensive behaviors imply that these effects may be mediated by interactions within the medial amygdala. Furthermore, medial amygdala somatostatin interneurons showed significantly lower expression of c-Fos in alcohol-intoxicated males during the acute withdrawal period. This c-Fos downregulation in somatostatin interneurons was restored during the protracted withdrawal. Interestingly, Pharmacogenetic manipulation of somatostatin interneurons in the medial amygdala modulates aggressive behavior after chronic alcohol intoxication. Inhibition of somatostatin interneurons through chemogenetically expressed hM4Di receptors promoted the aggressiveness, while activation through chemogenetically expressed hM3Dq receptors significantly blocked the aggressiveness during both acute and protracted withdrawal. These results introduce somatostatin interneurons in the medial amygdala as an emerging therapeutic target for aggressive behavior associated with alcohol withdrawal syndrome.

Symposia

SP14_3

Title

Neuroanatomical basis for the anti-nociceptive role of the relaxin-3/RXFP3 peptidergic system

Authors

Solène Escoffier*[1], Thibault Dhellemmes[1]; Akhter Hossain[2]; Andrew Gundlach[2]; Marc Landry[1].

1: Neurodegenerative Diseases Institute of Bordeaux, France;
2: Florey Institute, Australia ; , Florey Institute, Australia,

Abstract

Affecting around 10% of the world population, chronic pain and its related psychiatric comorbidities (e.g. anxiety and depression) are major health issues. The implication of neuropeptides in the modulation of pain remains poorly described in the brain. The relaxin-3 (RLN3) neuropeptide displays antidepressant and anxiolytic effects, and our preliminary results indicate an analgesic role in rodents. RLN3 is expressed by nucleus incertus (NI) neurons that project to different cortical (e.g. anterior cingulate cortex (ACC)) and subcortical (e.g. amygdala) areas of the pain matrix. We aim at studying the pain modulatory effects of RLN3/RXFP3 system by using pharmacological, behavioral, and anatomical approaches in a mouse model of persistent inflammatory pain. Persistent inflammation has been induced by the injection of Complete Freund's Adjuvant (CFA) in the paw of the animal. Intra-amygdalar injection of RXFP3 agonists alleviated both mechanical and thermal pain, while intra-ACC injection affected only on mechanical sensitization. The effect of AAV-mediated chronic release of another RXFP3 agonist (R3/I5) confirmed these effects in the ACC and amygdala. Tracing experiments (eGFP) of NI RLN3 neurons showed a dense but heterogeneous network. In situ hybridization experiments demonstrated RXFP3 mRNA expression in somatostatin interneurons both in the ACC and amygdala, with an increase of RXFP3 expression in the ACC in the pain condition. 3D quantification in the ACC indicated an increase in the number of RLN3 profiles, but a decrease in their volume under inflammatory conditions. Our data highlight the plasticity of the RLN3/RXFP3 system and a novel antinociceptive role for this peptide family, suggesting its therapeutic potential in persistent pain conditions.

Keywords: Pain, Neuropeptide, Pharmacology, Neuroanatomy.

SP14_4

Title

Neuropeptidergic Modulation and Neural Pathways in Contextual Fear Conditioning and Extinction: Insights from the Retrosplenial Cortex and Nucleus Incertus

Authors

Navarro-Sánchez M [1,2]; Gil-Miravet I [1,2]; Zahran MAE [1,2]; Hidalgo-Cortes J [1,2]; Mañas, Ojeda A [1,2]; Castillo-Gómez E. [1,2] Olucha-Bordonau F E [1,2]. 1 Department of Medicine, School of Medical Sciences, Universitat Jaume I, 12071 Castelló de la Plana, Spain. 2 Spanish National Network for Research in Mental Health (CIBERSAM), Instituto de Salud Carlos III, Madrid, Spain

Abstract

This work examines the mechanisms underlying contextual fear conditioning and the role of neural networks in memory processing. We assessed how different procedural factors influence the acquisition and extinction of fear in rats, revealing that spaced sessions of conditioning enhance extinction, while minimal shocks produce context-specific fear. More intense conditioning, however, leads to fear generalization across contexts, which can be extinguished similarly to the conditioned context. Additionally, we explored the role of the retrosplenial cortex (RSC) and its interaction with the neuropeptide relaxin-3 (RLN3) via the RXFP3 receptor. Modulation of RXFP3 in the RSC delayed fear extinction without affecting acquisition, suggesting that RLN3/RXFP3 signaling enhances memory strength, making extinction more resistant. Anatomical studies confirmed a strong presence of RLN3-immunoreactive fibers in the RSC, particularly co-localizing with inhibitory circuits. Finally, we investigated the connectivity between the RSC and other brain regions, particularly the bidirectional projections between the RSC, prefrontal cortex, and nucleus incertus. These connections were confirmed using anterograde and retrograde tracers, highlighting a broader cortical and subcortical network that involves key regions such as the prelimbic and orbitofrontal cortices, the medial septum, and the supramammillary nucleus. Together, these findings provide a more integrated understanding of the circuitry involved in contextual fear and memory processing, emphasizing the long-loop interactions between the retrosplenialcortex, prefrontal areas, and subcortical structures in regulating fear acquisition and extinction processes.

KEYWORDS: Emotion, fear memory, RLN3, nucleus incertus, RSC, excitation/inhibition balance.

Symposia

Symposium 15

Neuroimmune dysfunction and mental health outcomes: advances in immunopsychiatry in Africa

Organizer: **Arish Mudra Rakshasa-Loots**

University of Edinburgh, Edinburgh, UK
email: **arish.mrl@ed.ac.uk**

Abstract:

Neuroinflammation is increasingly being recognised as a notable predictor of neuropsychiatric conditions. The goal of immunopsychiatry is to enable effective prediction and intervention for these conditions using neuroimmune markers. This symposium seeks to highlight diverse approaches to investigating the contribution of neuroimmune dysfunction to mental health outcomes in African populations.

Beginning with a psychiatric genetics approach, Dr Allan Kalungi will first discuss the overlapping genetic variants implicated in neuroinflammation and depression. Next, we will reflect on the role of peripheral biomarkers of inflammation in psychiatric outcomes. Using data from young people with perinatally-acquired HIV, Dr Arish Mudra Rakshasa-Loots will demonstrate that early-life inflammation may be associated with a higher risk for experiencing symptoms of depression in adolescence. Dr Lindokuhle Thela will then examine the utility of neuroinflammatory biomarkers in informing diagnosis and clinical outcomes in people with new-onset psychosis. Finally, Prof Sian Hemmings will discuss the role of the gut microbiome in facilitating pro-inflammatory cascades and increasing susceptibility to depression, schizophrenia, and post-traumatic stress disorder.

All talks will involve data from African cohorts, and the determinants of mental health outcomes that may be unique to the African context will be a cross-cutting theme of discussion. Together, these innovative studies seek to identify the neurobiological mechanisms that may help predict the development of neuropsychiatric conditions or inform the delivery of targeted interventions to reduce their burden.

This session will thus feature recent advances in immunopsychiatry in Africa across multiple neuropsychiatric conditions and using a wide range of neuroimmune markers. These talks will be accessible to non-specialist neuroscientists to encourage attendees to collaborate on immunopsychiatry research. Our speakers have published widely in immunopsychiatry and represent various genders, ethnicities, countries, and career stages. The diversity of speakers and approaches in this symposium, combined with its emphasis on African neuroscience, will bolster its relevance to a wide range of neuroscientists at SONA 2025.

Speakers

Number	Speaker	e-mail	Title of the communication
SP15_1	Allan Kalungi	allankalungi1 @gmail.com	Genetic risk for depression among Continental Africans: establishing a genetic database for depression in Africa
SP15_2	Arish Mudra Rakshasa-Loots	arish.mrl @ed.ac.uk	Depression in people with HIV: Intersections with neuroimmune and metabolic dysfunction
SP15_3	Lindokuhle Thela	Thelal @ukzn.ac.za	The utility of neuroinflammatory biomarkers in informing diagnosis and clinical outcomes in people with new-onset psychosis

Symposia

SP15_1

Title

Genetic risk for depression among Continental Africans: establishing a genetic database for depression in Africa

Authors

Allan Kalungi[1], Dickens H Akena[2], Eugene Kinyanda[1,2], Cathryn M Lewis[3], Segun Fatumo[1,4], Karoline Kuchenbaecker[5]

1-MRC/UVRI & LSHTM Uganda Research Unit,
2-Department of Psychiatry, Makerere University, 3-Social, Genetic & Developmental Psychiatry Centre, Institute of Psychiatry, Psychology & Neuroscience, King's College London, 4-Precision Health Care University Research Institute, Queen Mary University of London, 5-Division of Psychiatry, UCL Genetics Institute, University College London.

Abstract

Depression is a significant global health issue, with genetic factors playing role in its etiology. Current genetic research on depression largely includes African Americans and Africans in Western countries, excluding continental Africans. This gap limits understanding of gene-environment interactions unique to Africa, perpetuating health disparities as indigenous Africans may miss out on tailored treatments. Our study addresses this gap by creating a genetic database for depression in Africa. The database combines data from four cohorts: (i) MRC/UVRI & LSHTM Uganda Research Unit's general population cohort (GPC, n=10,560), (ii) the NeuroGAP study (n=7,073), (iii) Malawi Epidemiology & Interventions Research Unit (MEIRU, n=1,700), and (iv) DepGenAfrica (n=14,000). The GPC is on-going and has so far enrolled 4,200 participants, assessing depression with the MINI (version 7.0.2) and to sequence participants? DNA using the blended genome-exome (BGE) technology. Preliminary data from 1,066 participants show a 23.3% lifetime depression prevalence. NeuroGAP is complete and has MDD and BGE sequence data on 7,073 participants (360 MDD cases) across Uganda, Kenya, Ethiopia, and South Africa. MEIRU includes genetic and depression data on 1,700 participants from Malawi. DepGenAfrica aims to recruit 10,000 cases and 4,000 controls across Nigeria, Malawi, and Ethiopia, using PHQ-9 for MDD assessment and 4xwhole-genome sequencing. Collectively, this database will encompass ~33K participants (~11K cases). The database will be used to investigate genetic risk factors for depression in continental Africa through genome-wide association study (GWAS) followed by post-GWAS analyses, develop a machine learning model for prediction and test causal inferences to MDD using Mendelian randomisation. Portability of genetic markers and polygenic risk scores across diverse ethnic backgrounds will also be investigated. This groundbreaking study has potential to illuminate the genetic underpinnings of depression within African populations, potentially uncovering novel loci and elucidating the transferability of genetic risk factors. Preliminary GWAS analysis among 1,000 participants from GPC has identified four novel single nucleotide polymorphisms associated with depression, of which one (rs1513848, near FHOD3 gene) has been replicated in a large GWAS study from the Psychiatric Genomics Consortium. Depression genetic research in Africa has potential to enhance global discovery and will potentially add to global efforts.

Symposia

SP15_2

Title

Depression in people with HIV: Intersections with neuro-immune and metabolic dysfunction

Authors

Arish Mudra Rakshasa-Loots, Division of Psychiatry, University of Edinburgh, Edinburgh, UK.

Abstract

Inflammation and HIV status have been independently linked to an increased risk for depression, but few studies have explored the combined effects of these risk factors. Given that HIV results in persistent and chronic inflammation despite antiretroviral therapy, it is possible that the increased prevalence of depression among people with HIV may be partly attributed to inflammation. This talk will focus on the possible contribution of inflammation to the risk for depressive symptoms, using data from African cohorts of young people living with and without HIV. In a small sample (N = 36) of young people living with HIV in South Africa, we first show that early-life systemic inflammation measured using high-sensitivity C-reactive protein (CRP) concentrations was correlated with depressive symptom severity in adolescence (? = 0.56, p = 0.008). Next, in a large cohort (N = 862) of children in Uganda followed longitudinally for up to 24 months, we find that the longitudinal trajectories of depressive symptoms were influenced by a significant interaction of HIV status and baseline CRP concentrations. At higher CRP concentrations, children with HIV showed lower baseline depressive symptoms (? = ?0.14, p = 0.003), but their symptoms decreased less sharply over time compared to children without HIV (? = 0.08, p < 0.001). Therefore, the relationship between inflammation and depressive symptoms varied significantly according to HIV status. Finally, this talk will consider emerging data on possible interactions of immune and metabolic dysfunction to the increased risk for depression in people living with HIV. We will discuss the value of investigating neuroimmune mechanisms of the risk for depression in people with HIV, both as a means to positively impact the mental health of a large population group that is often underserved, and as a model for immunopsychiatry research more generally.

Symposia

SP15_3

Title

The utility of neuroinflammatory biomarkers in informing diagnosis and clinical outcomes in people with new-onset psychosis

Authors

Lindokuhle Thela, V Ntlatsana, B Nkambule, S Paruk, A Tomita, U Chhagan, B Chiliza, N Abbai.
Nelson R Mandela School of Clinical Medicine, 719 Umbilo Road, Durban South Africa

Abstract

Background: Psychotic disorders have a median age of manifestation in early adulthood, and approximately 30% of individuals who receive the diagnosis will have schizophrenia, a progressive condition leading to a significant burden of years lived with disability and a high cost of care. Neuroinflammation is increasingly recognised as one of the mechanisms of schizophrenia. African populations have a high burden of pro-inflammatory conditions, for example, a high burden of infections during pregnancy and also a high prevalence of trauma; these may be linked to neuroinflammation. However, little is known regarding inflammatory markers in persons living with schizophrenia in the African continent. The study aimed to review the current literature on inflammatory markers of schizophrenia in African populations and the utility and practical implications of research on inflammatory markers in the population. Methods: A literature review of studies conducted in the African setting on inflammatory markers was performed. Based on preliminary findings of an ongoing cohort study of patients with FEP in South Africa (KwaZulu-Natal) as a case study, the utility and practical implications of research on serum and cerebrospinal fluid inflammatory biomarkers in schizophrenia in the African population are described. Results: XX. The study on biomarkers had many challenges, and these included individual and contextual factors. Individual factors included the fact that biomarkers currently used to date are non-specific and may be raised in a variety of conditions; therefore, association with clinical symptoms was difficult to establish. Contextual factors included financial and human resource limitations and systemic barriers to conducting new research techniques. Conclusion: Existing data shows that psychosis, particularly schizophrenia, is more than just a psychological condition; more research is needed on African populations to establish specific neuro-inflammatory markers as the first step towards the management of this condition. Additionally, the existing system barriers in the emerging field must be countered to facilitate progress in research.

Symposia

IBRO-sponsored
Symposium 16
" Neuroinfections"

Organizer: **Francesca Cirulli & Rachael Dangarembizi**

Neuroscience Institute, University of Cape Town, South Africa
Center for Behavioral Sciences and Mental Health, Instituto Superiori di Sanita, Italy
email: **rachael.dangarembizi@uct.ac.za; francesca.cirulli@iss.it**

Abstract:

Neuroinfections from bacterial, viral, fungal and parasitic pathogens pose a significant burden on Africa's health systems, particularly affecting vulnerable populations like children, pregnant women, and immunosuppressed individuals living with HIV/AIDS. By far the most debilitating and fatal opportunistic infectious diseases are those that cause damage to the nervous system and each year Africa loses millions of potentially productive brains to these infections. Despite this, neuroinfections have been neglected in research and policy and there is very little data on how brain injury occurs in these diseases to guide the development of diagnostic tools and to aid in drug development. We have curated a panel of leading neuroinfections researchers studying bacterial, viral, fungal and parasitic diseases to share their research with the rest of the neuroscience community at SONA.

The Symposium will help showcase the work currently occurring in the neuroinfections field in Africa and hopefully open the much-needed discussion around scaling up neuroinfections research and reduce their impact of African brain health. IBRO fully funds this Symposium and will be held in collaboration with the journal Neuroscience.

Speakers

Number	Speaker	e-mail	Title of the communication
SP16_1	Muazzam Jacobs	muazzam.jacobs @uct.ac.za	The transcriptomic profile of Astrocytes during Mycobacterium tuberculosis infection.
SP16_2	Anja de Lange	anja.delange @uct.ac.za	Brain injury, inflammation, and immunometabolic dysregulation in a murine model of cryptococcal meningitis
SP16_3	Duyilemi Ajonijebu	Chris.Ajonijebu @mandela.ac.za	IL-4R Inhibition and Prostacyclin Treatment in Trypanosome-Induced Neurological Disorders
SP16_4	Ashmita Manillal	ashmita.manillal @wits.ac.za	Cytokine-mediated inflammatory responses in neonatal Group B Streptococcus disease and their role in neurodevelopmental impairment
SP16_5	Lumbuka Kaunda	Lkaunda @unza.zm	HIV-1 Clade C and Coinfections in the ART Era in Zambian Adults Living with HIV

Symposia

SP16_1

Title

The transcriptomic profile of Astrocytes during Mycobacterium tuberculosis infection.

Authors

Muazzam Jacobs, Sohair Geyer[1], Conchita Kamanzi[1], Natalie Nieuwenhuizen[2], Avril Walters[1], Stefan H. E., Kaufmann[2], Hans-Joachim Mollenkopf[3], Nai-Jen Hsu*[1] and Muazzam Jacobs [1,4,5]

1-Division of Immunology, Department of Pathology and Institute of Infectious Diseases and Molecular Medicine, Faculty of Health Sciences, University of Cape Town, South Africa. 2-Department of Immunology, Max Planck Institute for Infection Biology, Berlin, Germany. 3- Core Facility Genomics/Microarray, Max Planck Institute for Infection Biology, Berlin, Germany. 4-National Health Laboratory Service, Johannesburg, South Africa. 5-Neuroscience Institute, University of Cape Town, Cape Town, South Africa; Current Address: Natalie Nieuwenhuizen: Institute of Hygiene and Microbiology, University of Würzburg, Germany

Abstract

One to five percent of all tuberculosis cases are classified as Central Nervous System tuberculosis (CNS-TB) which is associated with significant morbidity and mortality rates. neuroinflammation Currently, little is known about the cells that regulate immune responses during CNS-TB infection. Astrocytes is an important cell type demonstrated to regulate innate and adaptive immunity in CNS disease. Astrocytes display morphological and functional heterogeneity and maintain neurological homeostasis during infection. Astrocytes are responsive to pathogenic neuroinvasion and increasing evidence suggest that they can direct immune responses in the brain. In this study we used transcriptional profiling to explore astrocytic responses during mycobacterial infection. We unequivocally demonstrated that M. tuberculosis bacilli are indeed internalized by astrocytes, although the mechanism by which this occurs remains unclear. Our transcriptomic analysis demonstrates that M. tuberculosis triggers highly selective astrocyte gene expression relative to non-infected cells and engage a transcription profile that typifies that of a classical immune cell. providing support of its immune regulatory potential during neuroinfection. Gene ontology analysis identified gene modules involved in immune responses to bacteria, and the ability of astrocytes to regulate CNS inflammation. Furthermore, the data shows astrocytes to be responsive to IFNy, a key cytokine necessary for control M. tuberculosis infection. Corresponding protein expression of selected genes validated astrocyte transcription during M. tuberculosis infection. In summary astrocytes engages unique gene modules that contributes to the regulation of immune protection during M. tuberculosis infection.

Symposia

SP16_2

Title

Brain injury, inflammation, and immunometabolic dysregulation in a murine model of cryptococcal meningitis

Authors

de Lange A., Higgitt E.R., Awala A.N., Kauchali M., Pato Y, B., Raimondo J.V., Gifford, A., Hoving, J.C., Ross, P., Lai, R., and Dangarembizi R. The Crypto Lab, University of Cape Town Faculty of Health Sciences, Anzio Road, Cape Town, South Africa.

Abstract

Cryptococcal meningitis is a lethal fungal brain infection caused by the yeast Cryptococcus neoformans. Cryptococcal meningitis has been classified as a neglected tropical disease that disproportionately affects people living with HIV in developing countries. Due to its neglect in research and policy, little is known about the pathogenesis of the disease, especially at the level of the brain. More specifically, the mechanisms underlying the neurological damage associated with death in cryptococcal meningitis patients remain uncharacterized. This study aimed to characterise the neuroimmune response to C. neoformans infection by dissecting host fungal interactions at the cellular and molecular levels. Male C57BL/6 mice were intravenously injected with 5x104 CFU C. neoformans. At one, three, and six-days post-infection, brains were collected for fungal load determination, histopathology, and immunofluorescence analyses. Cellular and molecular responses were characterised using cultured organotypic brain slices treated with 1x10? CFU of a fluorescent reporter strain of C. neoformans. Neuroimmune signalling was determined by tracking nuclear translocation of the pro-inflammatory transcription factor, nuclear factor for interleukin 6 (NF-IL6), and the anti-inflammatory transcription factor, nuclear factor erythroid 2-related factor 2 (NRF2). Multiplex assays for pro- and anti-inflammatory cytokines were performed on blood and brains collected from mice and brain slice culture media. Single-cell RNA sequencing was used for determining cell-type specific transcriptomic responses while bulk metabolomic analyses were carried out to measure metabolic profiles in infected vs uninfected brains. Our results showed that brain invasion by C. neoformans occurred via the vascular route, followed by aggregation of the fungus within perivascular spaces before infection of the brain parenchyma and meninges. We observed severe parenchymal lesions (cryptococcomas) formed by yeast aggregates in the cortex, thalamus, midbrain, and cerebellum. Cryptococcal infection was initially associated with limited peripheral and central inflammation, followed by a delayed, injury-related hyperinflammatory response that was accompanied by extensive metabolic dysregulation later during infection.

Symposia

SP16_3

Title

IL-4R Inhibition and Prostacyclin Treatment in Trypanosome-Induced Neurological Disorders

Authors

Duyilemi Chris Ajonijebu, Oluwashola Olaolu, Hajierah Davids, Gill Dealtry. Nelson Mandela University, South Africa.

Abstract

Background and Objectives: Neurological disorders caused by trypanosome brain infection involve complex and poorly understood pathological mechanisms, while current therapies are focusing on activating type-2 immune responses. Given the pharmacological significance of prostacyclin in neuropsychiatric disorders and the limited experimental data on its role in trypanosome-induced CNS disease pathology, we hypothesized that enhanced IL-13/IL-4 signaling combined with Iloprost (prostacyclin analogue) treatment could effectively combat trypanosome-induced neurocognitive changes and potentially reverse associated pathological alterations. Additionally, we explored the epigenetic and transcriptional modifications associated with trypanosome CNS infection. Methods: Sixty-four male albino Swiss mice (8-10 weeks old) were divided into two groups (n=32 per group); mice with pharmacologically inhibited interleukin 4 receptor (IL-4Rα-/-) and their wild-type (WT) littermates. At day 4 post-infection with Trypanosoma brucei brucei, both WT and IL-4Rα-/- infected mice received Diminazine (4mg/kg) or Iloprost (200 µg/kg) injections for 3 days. Subsequently, the animals were assessed for locomotion and anxiety-like behaviour, with blood samples collected for parasitaemia and hemogram tests. The expression of target gene transcripts in the prefrontal cortices (PFC) and hippocampal (HPC) tissues was quantified using RT-qPCR and DNA gel electrophoresis. Also, pathological changes, cytokine expression, and methylation profiling were assessed using histological techniques, ELISA and EZ DNA methylation kits, respectively. Results: IL-4R inhibition induced systemic inflammation, while treatments reversed decreased vertical exploration and anxiety-like behaviour. Molecular analysis indicated IL-13 downregulation in both the PFC and HPC, with IL-4 and IL-4R downregulated specifically in the HPC, suggesting region-specific infection responses. Iloprost and Diminazine treatments restored BDNF expression in the PFC, reduced TNF-α levels in both regions, and suppressed CNS trypanosome burden by downregulating Pfr expression. Differential DNA methylation changes in IL-4, IL-13, IL-4R, and BDNF promoters were observed in both brain regions of IL-4R-/- mice. Discussion/Conclusion: Overall, Iloprost effectively alleviated neuroinflammation and anxiety-like behaviour while reducing CNS parasite burden, offering potential therapeutic strategies for managing trypanosome-induced neurological disorders through immune modulation and pharmacological intervention.

Symposia

SP16_4

Title

Cytokine-mediated inflammatory responses in neonatal Group B Streptococcus disease and their role in neurodevelopmental impairment

Authors

Ashmeetha Manilall[1], Khaalid Khan[1], Marié Landsberg[1], Gaurav Kwatra[2], Ziyaad Dangor[2], Shabir A. Madhi[2], Lois Harden[1]

1- Brain Function Research Group, School of Physiology, Faculty of Health Sciences, University of the Witwatersrand, Johannesburg, South Africa. 2-South African Medical Research Council Vaccines and Infectious Diseases Analytics Research Unit, University of the Witwatersrand, Johannesburg, South Africa

Abstract

Background and Objectives: Streptococcus agalactiae, known as Group B Streptococcus (GBS), a Gram-positive bacterium, causes invasive GBS (iGBS) disease in neonates, often resulting in neurodevelopmental impairment (NDI). Clinical presentations include pneumonia, meningitis, and sepsis, with sepsis being most common. While meningitis is strongly associated with NDI, ~18% of neonates with iGBS-related sepsis also develop NDI. We proposed that cytokines may be one of the role players involved in iGBS-induced NDI. Therefore, this study investigated cytokine responses using a neonatal rat model of iGBS disease and human neonates presenting with iGBS disease. Neurodevelopmental assessments were also performed on human iGBS survivors. Methods Neonatal rats were injected intraperitoneally with GBS or saline. Plasma and hippocampal samples were analyzed for cytokine levels using RT-PCR and Luminex protein assays. Plasma concentrations and hippocampal mRNA levels of tumour necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6) were measured. Protein levels of IL-6 and interleukin-10 (IL-10) in the hippocampus were assessed. Human serum TNF-α, IL-6, and IL-10 levels were measured in neonates without iGBS disease (non-iGBS) (n=152) and neonates who survived iGBS disease (iGBS) (n=54) within 24-72 hours of onset. Neurodevelopmental outcomes in iGBS survivors were assessed using the Griffiths Mental Development Scales-Extended Revised (GMDS-ER). Results Compared to neonatal saline rats, infected pups showed elevated plasma levels and increased hippocampal mRNA expression of TNF-α, IL-1β, IL-6 and IL-10 (all $p<0.05$). Hippocampal protein levels of IL-1β were increased in infected pups ($p<0.05$). Human neonates with iGBS-meningitis and iGBS-sepsis had increased levels of serum IL-6 versus non-iGBS neonates ($p<0.001$). IL-10 levels were higher in iGBS-meningitis neonates versus iGBS-sepsis neonates ($p=0.003$) and non-iGBS neonates ($p<0.001$). Neurodevelopmental outcomes showed that iGBS survivors had impairments versus non-iGBS children, with meningitis survivors more notably affected than sepsis survivors. Discussion GBS infection induces systemic and central inflammatory responses characterized by elevated pro- and anti-inflammatory cytokines. These responses likely contribute to neuronal damage and subsequent NDI. Children surviving iGBS disease, particularly meningitis, are more likely to have increased cytokine concentrations and NDI compared to non-iGBS children. This study highlights the need for further research to elucidate how specific cytokine responses drive neurodevelopmental impairments in survivors of iGBS disease.

Symposia

SP16_5

Title

HIV-1 Clade C and Coinfections in the ART Era in Zambian Adults Living with HIV

Authors

Lumbuka Kaunda[1] · Mary S. Ngoma[2] · J. Anitha Menon[3] · Robert K. Heaton[4] · Sara Gianella[5]· Ajay R. Bharti[5] · Scott Letendre[5] · Michelli Faria de Oliveira[5] · Knut A. Hestad[6]

1-Department of Physiological Sciences, School of Medicine, University of Zambia, Lusaka, Zambia 2Department of Pediatrics, and Child Health, University of Zambia, Lusaka, Zambia 3Department of Psychology, University of Zambia, Lusaka, Zambia 4 Department of Psychiatry, University of California, San Diego

Abstract

Despite the fact that many coinfections in people with HIV (PWH) are treatable or suppressible, they may still impact neurocognitive (NC) functioning. In this study, we aimed to evaluate the presence of latent/treated coinfections and their association with NC functioning in a cohort of PWH in Zambia. We carried out a cross-sectional, nested study involving 151 PWH with viral suppression and a normative sample of 324 adults without HIV. Plasma samples from PWH who underwent a comprehensive NC assessment were evaluated for the presence of treated/latent coinfections that are common in Zambia. Information about treated pulmonary tuberculosis (TB) was obtained from participants. Clinical charts. Overall, PWH differed significantly from the HIV seronegatives on all neuropsychological domains except for fine motor control. ANOVA comparisons of all 3 HIV+ groups? demographically corrected mean NC T-scores showed that the HIV+/TB+ group had the poorest NC functioning in the following domains: executive functioning ($F=4.23$, $p=0.02$), working memory ($F=5.05$, $p=0.002$), verbal fluency ($F=4.24$, $p=0.006$), learning ($F=11.26$, $p<0.001$), delayed recall ($F=4.56$, $p=0.01$), andspeed of information processing ($F=5.16$, $p=0.005$); this group also was substantially worse on the total battery (global mean T-scores; $F=8.02$, $p<0.001$). We concluded that treated TB coinfection in PWH was associated with worse NC performance compared to both those with antibodies against other coinfections and without. PWH with antibodies for other coinfections (HIV+/CI+) showed somewhat better NC performance compared to those without (HIV+/CI+), which was not expected, although comparisons with the HIV+/CI+ group are limited by its lack of specificity regarding the type of coinfection being represented. Our study also showed that during ART, an increase in CD4+ T-cells was significantly associated with a better Global Mean T-score ($p=.002$). Neither nadir CD4+ T-cell count nor plasma HIV RNA during ART were associated with cognitive outcomes.

Symposia

MDS-Sponsored
Symposium 17

The Pathophysiology and Challenges in therapy for Neurogenerative disease

Organizer: **Abdelhamid Benazzouz**

Department of Anatomy.University of Port Harcourt.Rivers State
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Abstract:

Neurodegenerative diseases, marked by progressive neuronal degeneration, represent an escalating global public health crisis. Emerging evidence strongly links exposure to heavy metals like lead, mercury, cadmium, and aluminum with the onset and progression of these disorders. This symposium will examine the molecular mechanisms by which heavy metals induce neurotoxicity, emphasizing their role in the pathogenesis of neurodegenerative diseases. Heavy metal toxicity initiates a cascade of detrimental molecular events, including oxidative stress, chronic inflammation, mitochondrial dysfunction, and disrupted metal homeostasis. These processes contribute to misfolded protein accumulation, synaptic dysfunction, and neuronal cell death hallmarks of conditions such as Alzheimer's, Parkinson's, and ALS. A thorough understanding of these pathways is critical for the development of targeted therapeutic interventions. Translational medicine is essential in transforming laboratory research into clinical applications, offering new hope for those affected by neurodegenerative diseases. Advances in understanding heavy metal-induced neurotoxicity have led to innovative diagnostic tools, biomarkers, and therapeutic strategies.

Approaches like nanotechnology, gene therapy, and pharmacological interventions show promise in counteracting the neurotoxic effects of heavy metals, potentially slowing or halting disease progression ting agents exhibit neuroprotective properties by scavenging free radicals, modulating inflammation, and enhancing metal excretion. These natural compounds provide valuable insights for developing safe, adjunctive therapies. This presentation will synthesize the current understanding of the molecular mechanisms underlying heavy metal toxicity in neurodegenerative diseases. It will also highlight the translational potential of recent scientific advancements and the role of natural antidotes in creating effective treatment strategies. By elucidating these complex interactions, this symposium aims to contribute to the development of targeted interventions that could significantly alleviate the global burden of neurodegenerative diseases.

Speakers

Number	Speaker	e-mail	Title of the communication
SP17_1	Abdelhamid Benazzouz	abdelhamid.benazzouz@u-bordeaux.fr	Deep Brain stimulation in Africa
SP17_2	Houyam Tibar	tibarhouyam@gmail.com	Deep Brain stimulation: the experience of Rabat University Hospital
SP17_3	Toshihide Yamashita	yamashita@molneu.med.osaka-u.ac.jp	Repulsive guidance molecule regulates glial and immune function under neurological diseases

Symposia

SP17_1

Title

SP17_2

Title

SP17_3

Title

Repulsive guidance molecule regulates glial and immune function under neurological diseases

Authors

Toshihide Yamashita, Department of Molecular Neuroscience, Graduate School of Medicine, Osaka University

Abstract

Repulsive guidance molecule-a (RGMa), which is a glycosylphosphatidylinositol-linked glycoprotein, is expressed in glial cells and immune cells. RGMa was previously recognized as the protein that regulates axon growth negatively in the adult central nervous system (CNS). Enhanced recovery of skilled forelimb movement as well as neural rewiring was observed after spinal cord injury (SCI) in adult macaque monkey following anti-RGMa antibody treatment. Based on the findings by the preclinical studies, the international clinical trials of humanized anti-RGMa monoclonal antibody (Unasnemab) for SCI is ongoing currently. Furthermore, RGMa was shown to be involved in immune regulation. RGMa expressed in dendritic cells promotes activation of T cells, leading to deterioration of autoimmune encephalomyelitis. Further, under the condition of neuromyelitis optica (NMO), anti-RGMa antibody treatment significantly suppressed neutrophil infiltration, and decreased the expression of neutrophil chemoattractants. The multiple modes of actions of anti-RGMa antibody may explain the potent effects on the neurodegenerative and neuroimmune diseases as well as the CNS injuries. The clinical trial of Unasnemab for HTLV-1-associated myelopathy is also ongoing. We recently reported that RGMa regulates blood-brain barrier integrity and cell survival in the CNS. Intravenous, administration of anti-RGMa antibodies reduced the loss of tyrosine hydroxylase (TH)-positive neurons and accumulation of Iba1-positive microglia/macrophages in the substantia nigra (SN) in a mouse model of Parkinson's disease (PD). Selective expression of RGMa in TH-positive neurons in the SN induced neuronal loss/degeneration and inflammation, resulting in a progressive movement disorder. Increased RGMa expression upregulated pro-inflammatory cytokine expression in microglia. Our observations suggest thatthe upregulation of RGMa is associated with the PD pathology; furthermore, inhibitory RGMa antibodies are a potential therapeutic option.

Symposia

Symposia

IBRO-ARC sponsored

Symposium 18

The rising Star symposium: Neuroimmunology Schools in Africa Alumni Research and Progress

Organizer: **Willias Masocha & Roberto Furlan**

Department of Pharmacology and Therapeutics, College of Pharmacy, Kuwait University, Safat, Kuwait
Clinical Neuroimmunology Unit, Institute of Experimental Neurology. INSpe, San Raffaele Scientific Institute, Milan, Italy
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Abstract:

Neuroimmunology is at the core of the pathophysiology of both communicable and non-communicable diseases affecting African populations. Our two previous symposiums held during the bi-annual meetings of SONA, in Accra 2021 and in Johannesburg 2023 covered neuroimmunology of neuroinfections and non-communicable diseases, respectively. The presenters were instructors, organizers of the Neuroimmunology schools or authors of articles in the Frontiers E-book Neuroimmunology in Africa. This symposium to be held during the bi-annual meeting of SONA, in Marrakesh 2025 will have four students who passed through the Neuroimmunology schools present their research in neuroimmunology and their trajectory in research after attending the IBRO Neuroimmunology schools. The presenters are at different stages of their career i.e., PhD Student, Post-doctoral Researcher, Lecturer, and Senior Lecturer and PI. The presenters, four former students of the Neuroimmunology Schools held in Rabat, Morocco will share their data and experience, and will interact with attendants, including younger African students, fully in line with the conference's aim to foster knowledge exchange and education in Africa. Information about ISNI & the African School of Neuroimmunology will also be availed to the participants.

Speakers

Number	Speaker	e-mail	Title of the communication
SP18_1	Hind Ibork	hindibork88 @gmail.com	CBD Effect on Neuroinflammation-Induced Metabolic Changes in Astrocytes: Focus on Cannabinoid Receptor Type 1
SP18_2	Ahmed Olalekan Bakare	abakare6 @jh.edu	An assessment of T-cell involvement in CIPN
SP18_3	Adaeze N Adebesin	Adaezeadebesin @yahoo.co.uk	Jobelyn® ameliorates anxiety-like behaviour, thermal hyperalgesia and neuroinflammation in formaldehyde- induced arthritis in mice
SP18_4	Rachael Dangarembizi	rachael.dangarembizi @uct.ac.za	Decrypting the complex neuroimmune interactions underlying brain injury during cryptococcal infection

Symposia

SP18_1

Title

CBD Effect on Neuroinflammation-Induced Metabolic Changes in Astrocytes: Focus on Cannabinoid Receptor Type 1

Authors

Hind Ibork [1], Oualid Abboussi [1], Khalid Taghzouti [1], Ignacio Fernandez Moncada [2] and Giovanni Marsicano [2]. 1-Physiology and Physiopathology Team, Faculty of Sciences, Genomic of Human Pathologies Research Centre, Mohammed V University in Rabat, Rabat, Morocco. 2-Univ. Bordeaux, Institut national de la santé et de la recherche médicale (INSERM), Neurocentre Magendie, U1215, F-33000 Bordeaux, France

Abstract

Neuroinflammation is implicated in various neurodegenerative disorders. During neuroinflammation, immune cells like microglia and macrophages release cytokines that cause neurotoxicity and neuronal death. These cytokines also affect astrocytes, the main glial cells of the brain; astrocytes thereby alter their metabolic function. Astrocytes release lactate, which in turn is used by neurons to fuel their mitochondrial metabolism. However, this metabolic coupling can become defective during neuroinflammation. In this context, the present work aimed to explore: (i) the impact of lipopolysaccharides (LPS)-induced neuroinflammation on the lactate and ATP dynamics of astrocytes, (ii) the potential rescuing effect of cannabidiol (CBD) treatment on the neuroinflammation-mediated metabolic failure, and (iii) the involvement of Cannabinoid type 1 Receptors (CB1R), on the LPS and CBD effects. These objectives were studied in cortical glia-neuron co-cultures derived from wild-type mice, CB1R-Knockout mice, and DN22-mice (lacking CB1 receptor expression on mitochondria). To study the lactate and ATP dynamics, state-of-the-art genetically encoded fluorescent biosensors for lactate and ATP were expressed in astrocyte via a viral approach, and their activity was imaged in real-time with fluorescent microscopy. In parallel to this, immunofluorescence analyses of GFAP, STAT3, p-STAT3, and GAP43 levels were performed as proxy markers of neuroinflammation. Astrocyte were treated with Vehicles, LPS, CBD, or LPS + CBD for 24 hours before experiments. Our results showed that LPS treatment induced an increase in intracellular lactate that was reversed in CB1R-KO astrocytes, where lactate production significantly decreased following LPS exposure. Interestingly, CBD significantly rescued the metabolic abnormalities induced by LPS, which required CB1 receptor expression. LPS treatment also sensitizes astrocytes to mitochondrial inhibition and increases the depletion of ATP by Complex IV inhibition in CB1R-WT astrocytes. Our immunofluorescence assays showed that GFAP, STAT3, p-STAT3, and GAP43 levels were significantly upregulated in astrocytes following LPS exposure, while CBD treatment was able to revert this LPS-mediated effect. Our data is consistent with a rewiring of astrocyte metabolism induced by inflammatory conditions, and propose CB1R as key controller of the CBD-mediated effects on the metabolic activity of astrocytes. This suggests a promising strategy for downregulating neuroinflammation and consequently diminishing negative neurological outcomes.

SP18_2

Title

An assessment of T-cell involvement in CIPN

Authors

Ahmed Olalekan Bakare, Eellan Sivanesan, Department of Anesthesiology and Critical Care Medicine, Johns Hopkins University, School of Medicine; Baltimore, MD, 21205, USA

Abstract

Background and Objectives: Conflicting reports on the role of T cells in chemotherapy-induced peripheral neuropathy (CIPN) create uncertainty regarding their influence on neuropathic pain, hindering progress in CIPN treatment and prevention. This study compares T-cell competent (RNU+/-) and T-cell deficient (RNU-/-) rats to explore the role of T cells in painful paclitaxel (PTX)-induced peripheral neuropathy (PIPN). Methods: Adult male, T-cell competent and T-cell deficient rats were treated with paclitaxel (8 mg/kg i.p. total dose) in a model of non-squamous cell lung carcinoma. Reflexive (mechanical, heat, cold) and spontaneous (burrowing, gait, open field) pain behaviors were evaluated before and after the induction of PIPN. Immunohistochemistry (CD206, CD68, CX3CL1, CX3CR1) and flow cytometry (CD3, CD161a, CD45RA, CD163, CD86) were employed to assess macrophages and lymphocytes in the dorsal root ganglia (DRG), sciatic nerves, and spleen. Results: Our findings demonstrate that T cells are essential for the development of PTX-induced cold hypersensitivity. T cells delayed the onset of mechanical hypersensitivity and decreased burrowing activity. Flow cytometry revealed an increased CD4+/CD8+ T-cell ratio in paclitaxel-treated T-cell competent rats in the DRG, sciatic nerve, and spleen. Additionally, paclitaxel treatment led to a reduction in B cells and a shift in macrophage polarization towards the M1 phenotype, reducing the M2/M1 ratio, which was independent of T cells. However, an increase in M2 macrophages (M2? and M2a) in sciatic nerves was dependent on the absence of T cells. NK cell levels were reduced in paclitaxel-treated T-cell-deficient rats but remained unchanged in T-cell-competent rats. Discussion: T cells are essential for the development of cold hypersensitivity and reduce mechanical hypersensitivity and spontaneous pain at the onset of CIPN. These T-cell dependent effects may be mediated by an increased CD4+/CD8+ T-cell ratio, decreased M2? macrophages in sciatic nerves, and maintenance of NK cells in T-cell competent rats.

Symposia

SP18_3

Title

Jobelyn® ameliorates anxiety-like behaviour, thermal hyperalgesia and neuroinflammation in formaldehyde-induced arthritis in mice

Authors

Adaeze N. Adebesin[1*], Gbemisayo A. Abbas, Anjolajesu M. Ogunsola, Oluwafolaranmi O. Mabekoje, Damilola J. Adebambo, Joy M. Oni1, Habeeb A. Adeogun, Opemipo Akinyemi, Itunu R. Ayoade, Adetoke M. Ifesanwo, Abayomi M. Ajayi

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Abstract

Arthritis describes a chronic inflammatory disease characterized by joint pain, stiffness, and swelling. Anxiety and depression are common comorbidities in RA patients, which can worsen disease outcomes. Jobelyn® (JB), a polyphenol-rich extract, has been shown to possess anti-inflammatory and antioxidant properties. This study investigated the effects of Jobelyn® on anxiety-like behaviour, thermal hyperalgesia, and neuroinflammation in a mouse model of formaldehyde-induced arthritis. Swiss male mice were injected with formaldehyde (2.5%) to induce arthritis, and then treated orally with JB (50, 100, or 200 mg/kg) or vehicle for 7 days. Anxiety-like behaviour was assessed using the elevated plus maze and open field tests. Thermal hyperalgesia was evaluated using the hot plate test. Oxidative stress and pro-inflammatory cytokines were assessed in brain and paw tissues. JB treatment significantly reduced anxiety-like behaviour in EPM and OFT, and thermal hyperalgesia in hot plate in arthritic mice. The JB decreased brain and paw tissues malondialdehyde, nitrites and increased reduced glutathione, catalase, superoxide dismutase and glutathione-s-transferase in arthritic mice. Also, JB caused significant reduction in brain TNF-α and IL-6 as well as paw TNF-α. This study demonstrates the potential of Jobelyn® to ameliorate anxiety-like behaviour, thermal hyperalgesia, and neuroinflammation in a mouse model of formaldehyde-induced arthritis. The findings suggest that Jobelyn® may be a useful adjunctive therapy for managing RA-related comorbidities. Keywords: Jobelyn®, Arthritis, hyperalgesia, anxiety, neuroinflammation.

Symposia

SP18_4

Title

Decrypting the complex neuroimmune interactions underlying brain injury during cryptococcal infection

Authors

Emily Higgitt, Amalia Awala, Anja De Lange, Maahir Kauchali, Lucian Duvenage, Sumaya Salie, Rachel Lai, Peter Rossi-Smith, Joseph Raimondo and Jennifer Hoving. Neuroscience Institute, University of Cape Town, South Africa.

Abstract

Fungi have been allies to humanity since the beginning of our existence, aiding in the production of food, beverages, and even treatments for human ailments. However, in recent times, invasive fungal infections have emerged as a significant threat to human health, an adversary for which we currently lack adequate defenses. We study the pathobiology of *Cryptococci meningitis*; a highly invasive and deadly fungal infection of the central nervous system (CNS) caused by the ubiquitous fungus *Cryptococcus neoformans*. *C. neoformans* is a neuroinvasive and neurotropic yeast that is associated with a lethal form of meningoencephalitis (inflammation of the meninges and neural tissue). Patients with CM develop severe neurological damage but the mechanisms driving brain injury in this fatal disease are poorly described. Using both mouse and human brain tissue models, we have characterized host-fungus interactions at the cellular and molecular levels. We employed single-nucleus RNA-sequencing, multiplex assays, and metabolomics analyses to determine the transcriptomic, proteomic, and metabolic changes that occur in the brain following infection with *C. neoformans*. We further investigated neuro-immune signaling in cultured murine and human brain slices and measured cytokine release from infected slices using multiplex cytokine assays. Our findings reveal that fungi can evade immune destruction in the peripheral system, penetrate the brain via its extensive vascular network, and disrupt neuro-immune balance, resulting in both hypo- and hyper-inflammatory responses. This dysregulation leads to injury and neurological damage. We further demonstrate how fungal invasion impairs fluid and waste homeostasis in the brain, exacerbating inflammation and tissue damage.

Symposia

PsyCoMed-soposored

Symposium 19

Neurological disorders caused by Mediterranean pollutants

Organizer: **Marc Landry**

PsyCoMed Project: University of Bordeaux,
IMN CNRS UMR 5293, 146 Rue Leo Saignat, 33076 Bordeaux, France
email: **marc.landry@u-bordeaux.fr**

Abstract:

Pollutants in African countries and the Mediterranean area are increasing threats for health. Their neurological consequences need to be described and their mechanisms of action deciphered to objectively determine their potential risks. This symposium will consider the effects on the nervous system of two types of very common pollutants which can persist in the environment for months or years. Glyphosate is the most widely common herbicide in the world, making its intensive use a major environmental and health problem. Nanoplastics are accumulating in different organs and may interfere with their normal biological functions.
Prenatal exposure to glyphosate provokes neurotoxic effects due to oxidative stress and massive neuronal apoptosis in the developing brain, causing lifelong behavioral abnormalities. Pituitary adenylate cyclase-activating polypeptide (PACAP) is a neuropeptide with important neurotrophic and neuroprotective functions. Dr Said Galai's presentation focuses on the capacity of intra-nasally injected PACAP in rats to protect the brain of offspring exposed in utero to glyphosate against oxidative damage and cellular neurotoxicity. Dr Ait Bali's research in rats revealed that glyphosate increases anxiety, reduces serotonergic fibers, and causes neuronal hyperactivation in the medial prefrontal cortex and amygdala. Additionally, glyphosate exposure leads to gut microbiome dysbiosis, which coincides with anxiety-like behaviors. These findings suggest a link between GBH neurotoxicity and both gut-brain axis disruption and anxiety. Zineb Bouargane was interested in the capacity of nanoplastics to cross the blood brain barrier while it is not completely closed, during gestation. Their results in rats indicate that nanoplastics intake induces changes in the pups that results in impairment of normal behavior during adulthood. The recent high rise in the prevalence of Attention Deficit/Hyperactivity Disorders (ADHD) has been linked to possible pollutant effects. Prof Landry's team worked on the mechanisms underlying ADHD and its comorbid pain sensitization. They have used high-throughput methods to highlight signaling pathways dysregulated in these interacting neuropsychiatric disorders.

Speakers

Number	Speaker	e-mail	Title of the communication
SP19_1	Said Galai	said.galai @fmt.utm.tn	Implementation of new protocols for Glyphosate and its byproduct detection. Application for demonstration of neurotoxicity by in vitro N2a cells culture.
SP19_2	Yassine Ait Bali	yassine.aitbali @gmail.com	Insight on the neurotoxicity of glyphosate: Anxiogenic effect and underlying mechanisms
SP19_3	Zineb Bouargane	Bouargan @uji.es	The alterations in normal behavior in adult rats resulting from nanoplastic intoxication of their mothers during pregnancy and/or lactation
SP19_4	Marc Landry	marc.landry @u-bordeaux.fr	Neuroinflammatory Mechanisms of Pain Hypersensitization in a Mouse Model of ADHD

Symposia

SP19_1

Title

Implementation of new protocols for Glyphosate and its byproduct detection. Application for demonstration of neurotoxicity by in vitro N2a cells culture.

Authors

Said Galai[1], Amine Aladnani[1,2] , Yasmine Limam[1,2], Sihem Haj Kacem1, Taoufik Ghrairi[2], Rafaella Silva[4] , Andreia Rosatella[3,4] , Carlos Afonso[3], Souheil Omar[1], Olfa Masmoudi[2].

1-Laboratory of Clinical Biology, National Institute of Neurology Mongi Ben Hmida, La Rabta, Tunis, Tunisia. 2-Department of Biology, Faculty of Sciences of Tunis, University of Tunis El Manar, Tunis, Tunisia. 3-Research Institute for Medicines (iMed.ULisboa), Faculty of Pharmacy, University of Lisbon, Lisbon, Portugal. 4 CBIOS-Universidade

Abstract

its byproducts, particularly the AMPA (Amino-Methyl-Phosphonic Acid), have been found to exhibit significant neurotoxic effects. Its detection is difficult to implement due to its chemical properties and its low prevalence; there is a strong need to develop sensitive analytical methods for glyphosate (GLP) monitoring to study its prevalence and its toxicity. For this purpose, a new specific biosystem based on enzyme reaction was implemented by laccase, redox mediator (Acetosyringone: ASGN), and ionic liquid (IL, as conservator and activator) to catalyze GLP. This enzymatic reaction revealed a progressive catalysis of glyphosate by generating toxic byproducts accompanied by a gradual decrease of the glyphosate concentration, indicating its active degradation. This fact was demonstrated, for first time, by HPLC. Then, laccase-catalytic system has been investigated by two analytical methods: spectrophotometric and electrochemical one. To investigate the toxicity of glyphosate and its byproducts, it was evaluated via N2A cells culture, using cellular toxicity assays (FDA and LDH). It was demonstrated that the degradation byproducts of glyphosate induced significant cellular damage in the N2A neuronal cells causing essentially by ROS production proportionally with the reaction time. Prolonged exposure of N2A cells to glyphosate and its byproducts lead to severe cellular damage, highlighting the neurotoxic potential of these compounds. Regarding the implementation of new method for glyphosate detection, it was successfully set up two methods with different LOD, the detection limits for spectrophotometric method was 25 µM GLP while electrochemical method was even lowest around 5 µM GLP. The developing biosensor based on this enzymatic system has been carried out using gold-plated screen-printed electrode and Nafion polymer for laccase, redox mediator and ionic liquid complexes immobilization. GLP samples were successfully analyzed using cyclic voltammetry (CV) measurement at scan rate of 100 mV/s. The concentration of GLP was accurately determined in the range of 5 µM to 15 µM GLP, and high correlation rate (98%) between current density and GLP concentration was determined using the laccase-based-biosensor, which been used for GLP assays in biological samples (cell lysate and culture medium) to demonstrate the prevalence of glyphosate into the medium and cells.

Symposia

SP19_2

Title

Insight on the neurotoxicity of glyphosate: Anxiogenic effect and underlying mechanisms

Authors

Yassine Ait-Bali, Nour-eddine Kaikai, Saadia Ba-M-hamed, Marco Sassoe-Pognetto, Maurizio Giustetto and Mohamed Bennis. Higher Normal School, Mohammed V University in Rabat, Morocco, and Cadi Ayyad University, Marrakech, Morocco

Abstract

Glyphosate, the active ingredient in many herbicides, has been shown to target the nervous system in various models. Our research revealed that glyphosate-based herbicides (GBH) increase anxiety, produced a reduction of 5-HT-immunoreactivity in the dorsal raphe nucleus, basolateral amygdala and ventral medial prefrontal cortex (mPFC) in treated mice. Furthermore, confocal microscopy investigations into the prelimbic/infralimbic regions of the mPFC and in basolateral/central nuclei of the amygdala disclosed that the behavioral alterations are paralleled by a robust increase in the density and labelling intensity of c-Fos- and pCREB-positive cells proving that GBH exposure caused neuronal hyperactivation in these regions. Additionally, GBH significantly altered the gut microbiome (GM) composition in terms of relative abundance and phylogenic diversity of the key microbes which coincided with the anxiety-like behaviors. Indeed, it decreased more specifically, Corynebacterium, Firmicutes, Bacteroidetes and Lactobacillus in treated mice. Taken together, these data highlight the essential link between GM dysbiosis and GBH toxicity and reinforce the link between GBH neurotoxicity and both gut-brain axis disruptions and anxiety.

Symposia

SP19_3

Title

The alterations in normal behavior in adult rats resulting from nanoplastic intoxication of their mothers during pregnancy and/or lactation

Authors

Zineb Bouargane[1,2], Mónica Navarro-Sánchez[1], Francisco E. Olucha Bordonau[1], Mohamed aly Zahran[1], Esther Castillo-Gómez[1], Liana Fattore[3].

1-Universitat Jaume I; 2-Cadi Ayaad University ; 3-CNR, IN, Cagliari

Abstract

Exposure to nanoplastics represents a significant health concern, particularly regarding their potential effects on neurological function. Previous studies have highlighted the adverse impacts of nanoplastic accumulation in various organs. Still, the effects on neural activity remain unclear, largely due to the protective role of the blood-brain barrier (BBB). However, under certain conditions, such as during development or in specific brain regions like the circumventricular organs, the BBB can become permeable. This study aimed to examine whether maternal exposure to nanoplastics during critical developmental periods of pregnancy, lactation, or both could impact behavioral outcomes in offspring. In this study, pregnant rats were exposed to either nanoplastics or saline during pregnancy, lactation, or both periods. When the offspring reached adulthood, a battery of behavioral tests was conducted, including the open field test, elevated plus maze, 3-chambers test, and contextual fear conditioning. Statistical analysis revealed that nanoplastic exposure significantly affected behavior and showed sex-dependent outcomes. Specifically, male offspring exposed to nanoplastics during pregnancy or lactation displayed increased locomotor activity, while no such effect was observed in the combined exposure group. Results from the elevated plus maze indicated heightened anxiety levels in both sexes for the pregnancy group compared to controls. Social behavior assessments in the 3-chamber test indicated that offspring exposed to nanoplastics during lactation or both pregnancy and lactation lacked discrimination between social and non-social stimuli, suggesting impaired sociability. This impairment was more pronounced in the social novelty test, where offspring exposed during pregnancy or both periods failed to recognize novel social targets. Finally, contextual fear conditioning showed that nanoplastics exposure disrupted offspring's ability to acquire, retain, and extinguish fear memories. Male offspring from mothers exposed during pregnancy failed to learn context-fear associations, while those exposed during lactation could learn but not extinguish fear responses. Females exhibited similar learning impairments, though with slight variations. Overall, these results indicate that maternal exposure to nanoplastics during pregnancy and/or lactation induces significant behavioral changes in offspring, highlighting the potential long-term neurodevelopmental risks of nanoplastics exposure.

SP19_4

Title

Neuroinflammatory Mechanisms of Pain Hypersensitization in a Mouse Model of ADHD

Authors

sARAh Bou Sader Nehme, Sandra Sanchez-Sarasua, Walid Hleihel, Marc Landry, University of Bordeaux, Institute of Neurodegenerative Diseases, UMR CNRS 5293, 146, rue Leo Saignat, 33000 Bordeaux, France

Abstract

Attention-deficit/hyperactivity disorder (ADHD) is a common multifactorial neurodevelopmental disorder, with a prevalence of around 8% in children worldwide. This prevalence is increasing upon pollutant, especially plastics, contamination worldwide, making it a growing public health issue in the Mediterranean area. ADHD is characterized by symptoms of inattention, hyperactivity, and impulsivity and is usually associated with cognitive, emotional, and behavioral deficits. Clinical studies suggest that pain hypersensitivity develops in subjects with ADHD. However, the mechanisms and neural circuits involved in these interactions remain unknown. Our team has previously validated a mouse model of ADHD obtained by neonatal 6-hydroxydopamine (6-OHDA) intracerebroventricular injection. We showed that 6-OHDA mice exhibited a marked sensitization to thermal and mechanical stimuli, suggesting that ADHD conditions increase nociception. Moreover, by combining different approaches, we also demonstrated that the anterior cingulate cortex (ACC) hyperactivity alters the “ACC” posterior insula (PI)? circuit, and triggers changes in spinal networks that underlie pain sensitization. We make the hypothesis that neuroinflammation is a major factor triggering ACC hyperactivity and the comorbidity between ADHD and pain. By using immunofluorescence staining, we demonstrated changes in the morphology of microglia and astrocytes, indicative of their activation. With RT-qPCR, we have identified markers of inflammation and oxidative stress, in the cingulate and insular cortex. Through mass spectrometry and high-throughput phosphoproteomic assays, we characterized deregulated kinase activity and signaling pathways under ADHD conditions. All these changes lead to altered inflammatory pathways, which may underlie ADHD and its comorbid pain. The identification of shared mechanisms, engaging overlapping neuronal circuits and inflammation, and underlying both disorders, is key to better treatments.

Symposia

Symposium 20

Environmental toxins and brain alterations: from mild cognitive effects to severe consequences on neuronal cell death

Organizer: **Samir Ahboucha**

FPK, USMS, P.B :145, code postal 25000 Khouribga, Morocco
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Abstract:

Chronic exposure to environmental toxins represents a high threat to humans by inducing biological damage including brain alterations that cause significant cognitive decline and lifestyle of a large population in Africa and worldwide. Examples of these multiple toxins include toxic nutriments, pesticides or heavy metals contained in several products to which humans are daily exposed. These toxins may cause a range of alterations from subtitle effects on metabolism, neurotransmission, inflammation to more severe effects that lead to neuronal cell death. The symposium will address the consequence of dietary cyanogens that causes the Konzo disease in Africa, the effect of pesticides that belong to either pyrethroids such as lambda cyhalotrin or the organophosphates such as the Malathion and glyphosate on specific neurobehavioral and biochemical effects in adults and during development. The symposium will also shed light on examples of the consequences of metal neurotoxicity such as lead, aluminum and other metals on neurodegeneration. Overall, the symposium will shed light on how environmental toxins affect brain function, and will discuss, in addition to obvious prevention strategies, new therapeutic aspects that may help alleviate their consequence on brain function.

Speakers

Number	Speaker	e-mail	Title of the communication
SP20_1	Desire Tshala Katumbay	Tshalad @ohsu.edu	Nutritional Neurotoxic Disease (Lathyrism, Cassavism) in Changing Climates.
SP20_2	Assmaa Tali	asmaa.tali @yahoo.com	Neurotoxic Effects of Lambda-Cyhalothrin: Behavioural Alterations and Protein Interaction Insights.
SP20_3	Laila Berroug	Berrouglaila @gmail.com	Sex-dependt effects of developmental exposure to Malathion on behavior and brain biochemistry
SP20_4	Hammou Anarghou	h.anarghou @gmail.com	Chronic Glyphosate Exposure Associated with Neurobehavioral and Cognitive Impairments
SP20_5	Abdeljalil El Got	abdeljalil.elgot @uhp.ac.ma	The hallmark of Docosahexaenoic acid against the Manganese intoxication in mice: links with Parkinsonism

Symposia

SP20_1

Title

Nutritional Neurotoxic Disease (Lathyrism, Cassavism) in Changing Climates. Desire

Authors

Tshala-Katumbay, HILAIRE MUSASA, OKITUNDU LUWA, Valerie S. Palmer, Matthew Bramble, mumba Ngoyi, Peter S. Oregon. Health & Science University, Portland, OR, USA.

Abstract

Lathyrism and cassavism are self-limiting upper-motor-neuron disorders that impact poor children and adults in Africa and elsewhere who depend for food on seed of grasspea (*Lathyrus sativus* L.) and the root and leaves of the tuberous cassava plant (*Manihot esculenta* Crantz), respectively. Both plants grow without chemical inputs and tolerate environmental extremes (notably drought), which make them well suited to future climate change. Cassava is a staple and major caloric source for ~800 million people in tropical areas worldwide, with pockets of cassavism (up to 20% prevalence) in Africa from the DRC to Mozambique. Grasspea is a staple food in parts of northern Ethiopia where lathyrism prevalence is high (circa 5%). Both plant products must be detoxified for use as a staple food because protein-rich grasspea harbors the excitotoxin Beta-N-oxalylamino-L-alanine, while carbohydrate-rich cassava root contains the cyanogenic glucosides linamarin and lotaustralin. Neurological burden of these food-associated insults includes but not limited to distinct motor neuron disease and possibly, cognitive impairments. Food use of low-toxin strains of these valuable plants may reduce the risk of nutritional neurotoxic disease. Recent advances indicate that use of antioxidants and/or probiotics may be explored to unveil novel therapeutics.

Symposia

SP20_2

Title

Neurotoxic Effects of Lambda-Cyhalothrin: Behavioural Alterations and Protein Interaction Insights

Authors

Assmaa TALI[1], Nadra LEKOUCH[2], Samir AHBOUCHA[1]. 1- Polydisciplinary Faculty of Khouribga, Sultan Moulay Slimane University, Morocco. 2-Faculty of Sciences - Semlalia, Cadi Ayyad University, Morocco.

Abstract

Lambda-cyhalothrin (LCT) is a type-II pyrethroid widely used in agriculture to protect crops from pests. Despite being considered safer for non-target organisms compared to other pesticide families, such as organophosphates, LCT poses potential risks for rural female farmworkers. The present study investigates the behavioral effects of LCT in 8-week-old female Swiss mice subjected to daily oral gavage for 21 consecutive days. Mice were divided into three groups: a control group treated with corn oil (vehicle), and two treated groups receiving LCT at doses of 0.5 mg/kg and 2 mg/kg b.w. Behavioral tests were conducted to evaluate locomotor activity (open field test), anxiety (dark-light box test), learning memory (novel object recognition test), memory retention (elevated plus maze test), and spatial working memory (Y-maze test). LCT-treated mice exhibited a decrease in locomotor activity, an anxiogenic effect characterized by reduced time in the enlightened compartment, impaired learning memory with a lower recognition index, and altered memory retention with increased latency time. Spatial working memory, however, remained unaffected. Emerging bioinformatics analyses suggest that LCT may interact with proteins involved in memory, such as retinoblastoma-binding protein (RbAp48), which is highly expressed in the dentate gyrus of the hippocampus. These findings indicate that LCT's neurotoxic effects on behavior, might be involved in disruptions in neuronal circuits, potentially mediated through oxidative stress and altered neurotransmission. Thus, preliminary bioinformatics analyses suggest that LCT may interact with proteins related to memory, such as RbAp48. Molecular docking studies reveal potential binding sites for LCT on RbAp48, highlighting its role as a possible molecular target contributing to LCT-induced memory impairments.

Keywords: Lambda-cyhalothrin, neurotoxicity, behaviour, Locomotion, anxiety, memory, RbAp48, Swiss mice.

Symposia

SP20_3

Title

Sex-dependent effects of developmental exposure to Malathion on behavior and brain biochemistry

Authors

Laila Berroug; Meriem Laaroussi; Oumaima Essaidi; Hafsa Malqui; Hammou Anarghou· Ahmed Ait Chaoui; Mohamed Najimi; Fatiha Chigr. Biological Engineering Laboratory, Faculty of Science and Technology, Sultan Moulay Slimane University, Beni Mellal, Morocco.

Abstract

Malathion is an organophosphate pesticide (OP) commonly used in agriculture, industry, and veterinary medicine. Sex is a crucial factor in responding to neurotoxicants, yet the sex-species effects of OP exposure, particularly neurological impairments following chronic low-level exposure, remains limited. Our study aims to evaluate the neurobehavioral and biochemical effects of developmental exposure to Malathion across sexes. Pregnant mice were exposed to a low oral dose of Malathion from gestation up to the weaning of the pups, which were individually gavaged with a similar dose regimen until postnatal day 70. Our results show that Malathion decreased body weight and food intake, reduced locomotor activity and recognition memory. Motor coordination and special memory were only altered in females, whereas we found a male-species effect of Malathion on social behavior and marble burying. These alterations were accompanied by increased malondialdehyde (MDA), decreased brain acetylcholinesterase activity (AChE), and disrupted brain redox homeostasis. Our findings about the effects of Malathion exposure across sexes may, in part, contribute to understanding the dimorphic susceptibilities observed in neurological disorders.

Symposia

SP20_4

Title

Chronic Glyphosate Exposure Associated with Neurobehavioral and Cognitive Impairments

Authors

Hammou Anarghou[1,2], Abderrahamne Lamiri[3,4] Said Ihbour[1] Hafsa Malqui[1], Oumaima Essaidi[1], Meriem Laaroussi[1], Mohamed Najimi[1] · Fatiha Chigr[1]

1: Biological Engineering Laboratory, Faculty of Sciences and Techniques, Sultan Moulay Slimane University, Beni Mellal, Morocco. 2: High Institute of Nursing Professions and Health Techniques Dakhla, Morocco. 3: Care, Health, and Sustainable Development Laboratory, Care and Biology-Health Research Team. High institute of nursing professions and health techniques Casablanca, Morocco. 4: Applied Materials Physics and Chemistry Laboratory (LCPMA) FSBM.

Abstract

Glyphosate-based Herbicide (GBH) is a widely used pesticide that functions as a broad-spectrum, non-selective herbicide. Despite advanced research to describe the neurotoxic potential of GBH, the harmful effects on maternal behavior and neurodevelopment of offspring remain unclear. This study was conducted to highlight the effects of GBH on the antioxidant system, anxiety traits, social interaction, and cognitive and sensorimotor functions in pups exposed to 25 or 50 mg/l daily via their mother's milk. Concerning the biochemical biomarkers, GBH administered during the early stages of development negatively affected the status of antioxidant enzymes and lipid peroxidation in the brain structures of the pups. Furthermore, our results showed a significant decrease in acetylcholinesterase (AChE) specific activity within the brains of treated pups. The results of the behavioral tests indicated that the treated offspring developed anxiety, memory, and sociability disorders, as evidenced by the Open Field, Y-maze, object recognition task, and social interaction tests. Through neurodevelopmental testing, we also showed sensorimotor impairment (righting reflex and negative geotaxis) and abnormal maternal behavior. Altogether, our study clearly demonstrates that the developing brain is sensitive to GBH.

Symposia

SP20_5

Title

The hallmark of Docosahexaenoic acid against the Manganese intoxication in mice: links with Parkinsonism

Authors

Elgot Abdeljalil, Elfari Radouane, Gamrani Halima. Higher Institute of Health Sciences, Settat

Abstract

Manganese (Mn) is an essential metallic trace element involved in several vital biological functions. Conversely, exposure to excessive levels of Mn induces manganism, causing neurodegeneration and symptoms similar to Parkinson's disease. Docosahexaenoic acid (DHA) is a long-chain polyunsaturated fatty acid exhibiting neuroprotective properties against neurodegenerative diseases and brain injuries. In the present study, mice were used for a sub-acute Mn intoxication model to investigate DHA neuroprotective potential against Mn neurotoxicity. We also seek to understand the mechanism by which Mn intoxication induces these motor impairments at 30 mg/kg, by pretreatment with DHA at 300 mg/kg and assessment of changes in spontaneous locomotor behavior by open field test (OF), motor coordination using the rotarod test (RR) and strength by mean of weights test (WT). To highlight these effects on brain neurotransmission, we evaluated the tyrosine hydroxylase immunoreactivity (TH-IR) within substantia nigra compacta and striatum. Results showed that Mn intoxication significantly ($p<0.001$) altered motor behavior parameters including, decreased of traveled distance by 46%, decreased mean speed by 36%, reduced the ability to sustain the rotarod test to 42%. Pretreatment by DHA prevents mice from Mn toxicity and maintain normal spontaneous activity, motor coordination and strength. Data also showed the ability of Mn to disrupt dopamine neurotransmission by altering tyrosine hydroxylase activity, DHA prevented this disruption. Data approved the potential neurotoxic effect of Mn as a risk factor of the Parkinsonism onset, and then demonstrated for the first time the neuroprotective and nutraceutical outcomes of DHA in the sub-acute Mn-intoxication animal model.

Keywords: Manganese; DHA; Parkinsonism; Locomotor activity; Dopamine; substantia nigra, striatum, mice.

Symposia

Symposium 21

Artisanal mining and its impact on the brain in the eastern region of Cameroon

Organizer: **Ayissi Rigobert Espoir & Eric Bila Lamu (Cameroon)**

Université de Douala
Faculté de Médecine et des Sciences pharmaceutiques
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Abstract:

Mining operations, especially in gold extraction, heavily rely on water, which serves as a primary medium for transporting and disseminating mining-related pollutants. Consequently, intensified mining activities in these regions have led to significant alterations in the composition of surface water, raising concerns about downstream water quality and potential health risks for local populations. So, the main objective of this symposium is to highlight the correlation or causal relationship between the mining environment and gold mining and the incidence of neurological pathologies; and then propose natural medicinal solutions as a promising direction in the management of this cerebral ecotoxicity.

Cancelled

Symposia

Symposium 22

The Brain, Environment and Nutraceuticals; Combating Neurodegeneration through Neuroprotection

Organizer: **Abel N. Agbon**

Neuroanatomy and Neuroscience Research Unit, Department of Human Anatomy, Faculty of Basic Medical Sciences, College of Medical Sciences, Ahmadu Bello University, Zaria, Nigeria
email: **bellulu@gmail.com**

Abstract:

The brain is an integral part of a biological system involved in the homeostasis of several vital functions. Exposure to environmental neurotoxins have been associated with neurodegenerative disease conditions including Alzheimer's and Parkinson's disease that are characterized by progressive structural, biochemical and physiological alterations in different brain parts critically associated with motor and cognitive functions. Nutraceuticals, especially plants-derived, have been reported as potential therapies for the management or treatment of neurological disease conditions. A promising strategy for the management and therapy of neurodegenerative diseases is the targeting of multiple mechanisms of actions associated with the pathologies of neurodegeneration through neuroprotective approach.
A.N. Agbon will focus his talk on the neuroprotective activity of Phoenix dactylifera (date palm) on mercury-triggered neurodegenerative changes in animal model. S.B. Mesole will explain the neuroprotective effects of Eugenol following aluminium-induced neurotoxicity on caspase-3, apoptotic proteins (Bcl-2 and Bax), oxidative stress biomarkers and levels of 8-hydroxy -2-deoxyguanosine (mitochondrial DNA stress marker) in animal model. Then, FSA Yadang will focus her talk on the neuroprotective activity of Carissa edulis against L-glutamic acid-induced oxidative stress, inflammation, and memory impairment in animal model and, Rachael Henry will discuss the neuroprotective properties of flavonoid fraction of P. dactylifera on rotenone model of Parkinson's disease in animal model.

Speakers

Number	Speaker	e-mail	Title of the communication
SP22_1	Abel N. Agbon	Bellulu @gmail.com	Phoenix dactylifera (date palm) has Neuroprotective Activity on Mercury-triggered Neurodegenerative Changes in Wistar rats
SP22_2	Samuel B. Mesole	ms361450 @gmail.com	Apoptotic Inducement of Neuronal Cells by Aluminium Chloride and The Neuroprotective Effects of Eugenol in Wistar rats.
SP22_3	Fanta S.A. Yadang	Fantayadang @gmail.com	Protective effect of Carissa edulis aqueous extract against L-glutamic acid-induced neurotoxicity in mice by regulating oxidative stress and neuroinflammation
SP22_4	Rachael Henry	henryrachel99 @gmail.com	Neuroprotective Efficacy of Phoenix dactylifera L. in rotenone rat model of Parkinson's Disease

Symposia

SP22_1

Title

Phoenix dactylifera (date palm) has Neuroprotective Activity on Mercury-triggered Neurodegenerative Changes in Wistar rats

Authors

Abel Agbon[1], Yasir M. Shuaib[1,2]; Rachael Henry[1]; Mojisola Abiodun[1,3]; Stephen S. Lazus [4]; Musa G. Abubakar[1]:

1: Ahmadu Bello University (ABU), Zaria, Nigeria; 2 Federal University, Wukari, Nigeria; 3 New Gates University, Minna, Nigeria; 4 Nigerian Defence Academy, Nigeria

Abstract

Background: Heavy metals including mercury are established environmental neurotoxicants, reported to induce structural, biochemical and physiological alterations in different brain parts by eliciting oxidative stress which results in neurological deficits. Brain regions including cerebral M1 and M2, cerebellar cortex, and subcortical regions such as the hippocampus are critically associated with motor functions, learning and memory. Alterations or injury to these brain regions may lead to motor deficits, behavioural and cognitive changes that mimic neurological conditions including Parkinson's and Alzheimer's diseases. Phoenix dactilyfera (date palm) has been scientifically demonstrated to possess various pharmacological activities. Objectives and Methods: This study empirically demonstrated the neuroprotective properties of certain solvent extract forms of P. dactilyfera fruit pulp against mercury in different brain regions of Wistar rats using microscopic (histology and histochemistry), neurochemical (AchE activity and neurotrace elements: Mg, Zn, Cu and Fe)/ biochemical (oxidative stress biomarkers: MDA, SOD, CAT and GPx) and neurobehavioural (cognition, anxiety, sensori-motor and motor coordination) assessments. Rats obtained were categorized as control, mercury-treated and extract + mercury-treated groups. All administration was orally and lasted for two weeks. Results and Discussion: revealed pathological changes in the histoarchitectural features of assessed brain regions, and alternations in biochemical and neurobehavioural parameters. However, the administration of extracts of P. dactylifera preserved the histology of the brain regions and ameliorated mercury-induced biochemical and neurobehavioural alterations. In conclusion, findings suggest that fruit pulp extracts of P. dactylifera may prove efficacious in ameliorating mercury-triggered alterations in different brain parts of Wistar rats. Neuroprotective properties could be attributed to the potent antioxidant activities of constituent phytochemicals. Thus, fruit pulp extracts of P. dactylifera are potential candidates for application in managing and treating mercury-induced neurodegenerative changes and related disease conditions.

Keywords: Histology, Neurochemistry, Neuroprotection, Neurobehaviour, Oxidative stress.

Symposia

SP22_2

Title

Apoptotic Inducement of Neuronal Cells by Aluminium Chloride and the Neuroprotective Effects of Eugenol in Wistar Rats.

Authors

Samuel Bolaji Mesole[1*], Okpanachi Omachonu Alfred[1], Uthman Ademola Yusuf[2], Lwiindi Lukubi[3], and Dailesi Ndhlovu[4]

1: Department of Medicine Eden University School of Medicine Lusaka, Zambia. 2: Department of Human Physiology, Kampala International University, Uganda. 3: Department of Human Anatomy, Mulungushi University, Zambia. 4: Department of Physiological Sciences, University of Zambia, Zambia & Department of Human Anatomy, Levy Mwanawasa Medical University, Zambia

Abstract

Aluminium is known to accelerate oxidative stress, amyloid beta (Aβ) deposition, and plaque formation in the brain of rats. The present study is aimed at studying the neuroprotective effects of eugenol following aluminium-induced neurotoxicity on caspase-3, apoptotic proteins (Bcl-2 and Bax), and oxidative stress markers in Wistar rats such as superoxide dismutase (SOD), glutathione peroxidase (GPx), nitric oxide (NO), and assay oxidative stress to mitochondrial DNA (mtDNA) by measuring the levels of 8-hydroxy-2-deoxyguanosine (8-OHdG). Materials and methods. Twenty (20) adult Wistar rats were randomly divided into four (4) groups with five animals in each group. The route of administration was oral throughout the duration of this study and this study lasted for 21 days. Rats were sacrificed 24 hours after administration of the last dose (i.e., day 22) with 0.8 mg/kg ketamine as an anaesthetic agent. Results. Exposure to AlCl3 resulted in a significant ($p<0.01$) elevation in the levels of nitric oxide and 8-hydroxy-2-deoxyguanosine (8-OHdG), enhanced the activity of caspase-3, increased the level of proapoptotic protein Bax and reduced the levels of antiapoptotic protein Bcl-2, and significantly ($p<0.01$) reduced the levels of SOD and GPx. However, treatment with eugenol resulted in a significant reduction ($p<0.01$) in the levels of nitric oxide (NO) and 8-hydroxy-2-deoxyguanosine (8-OHdG) levels, inhibited the activity of caspase-3, increasedlevels of Bcl-2 and significantly ($p<0.05$) reduced levels of Bax protein, respectively, and also significantly ($p<0.05$) increased the levels of SOD and GPx. Conclusion. Our results would hereby suggest that eugenol would provide a therapeutic value against aluminium-induced oxidative stress as related to antioxidant and antiapoptotic activities. Keywords: Aluminium Chloride, Neurotoxicity, Antipapoptotic Protein, Antioxidant, 8-hydroxy-2-deoxyguanosine.

Symposia

SP22_3

Title

Protective effect of Carissa edulis aqueous extract against L-glutamic acid-induced neurotoxicity in mice by regulating oxidative stress and neuroinflammation

Authors

Fanta SA Yadang[1,2*], Yvette Nguezeye[1,2], Germain Sotoing Taiwe[3], Gabriel A. Agbor[1], Nisar Ur-Rahman[4], Elisabeth Ngo Bum[2]

1 Centre for Research on Medicinal Plants and Traditional Medicine, Institute of Medical Research and Medicinal Plants Studies, Cameroon. 2: Department of Biological Sciences, Faculty of Science, University of Ngaoundere, Cameroon. 3: Department of Zoology and Animal Physiology, Faculty of Science, University of Buea, Cameroon. 4: Department of Pharmacy, Comsats Institute of Information Technology, Abbottabad, Pakistan

Abstract

Background and objective: Glutamate is the principal excitatory neurotransmitter in the central nervous system. It has been previously reported that the over-activation of NMDA receptors with glutamate contributes to mitochondrial dysfunction, oxidative stress, and inflammation. These pathological hallmarks play a crucial role in many neurological conditions. Carissa edulis has been shown to possess antioxidant and anti-inflammatory properties. This study investigated the protective effect of C. edulis aqueous extract on L-glutamic acid-induced neurotoxic impairment in mice. Methods: Two-month-old mice were intraperitoneally injected with L-glutamic acid (2 g/kg) for seven consecutive days and subsequently treated with an aqueous extract of C. edulis. A behavioural study was conducted using an elevated plus-maze to assess spatial memory. Oxidative stress, induced by reactive oxygen species, was evaluated by measuring lipid peroxidation and antioxidant enzyme levels (MDA, catalase, and glutathione). Neuroinflammation was monitored using IL-1? and TNF-? ELISA kits, and the acetylcholinesterase activity was evaluated using the Ellman method. Additionally, brain histopathology was examined using cresyl violet staining. Results and discussion: C. edulis has decreased the latency time for mice to enter the enclosed arm when compared to the L-glutamic acid group. This suggests an improvement in spatial memory. A reduction in latency indicates better memory performance in the mice. Our data suggest that the memory-enhancing effects of C. edulis can be attributed to the inhibition of acetylcholinesterase activity. The treatment with C. edulis has decreased the MDA levels and increased antioxidant enzyme activity, suggesting that C. edulis offers a protective effect against the oxidative stress induced by L-glutamic acid. Furthermore, this property can be attributed to its high levels of bioactive compounds, which significantly contribute to its antioxidant activity. Additionally, there was a notable decrease in the cytokine levels (IL-1? and TNF-?) in the mice treated with C. edulis, showing its anti-inflammatory properties. No pathological changes were observed in the brain sections. In conclusion, C. edulis aqueous extract has therapeutic effects by alleviating memory impairment and reducing oxidative stress and neuroinflammation. Therefore, Carissa edulis may be a potential pharmacological agent for neuroprotection in neurodegenerative diseases.

Keywords: Carissa edulis, L-glutamic acid, neurotoxicity, oxidative stress, inflammation.

SP22_4

Title

Neuroprotective Efficacy of Phoenix dactylifera L. in rotenone rat model of Parkinson's Disease

Authors

Rachael Henry* [1,2], **Fatimah Abisola Abdulmojeed**[1], **Abel Nosereme Agbon**[1]. 1-Sunday Abraham Musa Ahmadu Bello[1] University, Zaria, Nigeria. 2-Federal University Wukari, Taraba, Nigeria

Abstract

Background: Parkinson's disease is a neurodegenerative disorder characterize by motor and non-motor symptoms. Several plants have been reported to be beneficial in the management of Parkinson's disease symptoms. Phoenix dactylifera L. has been used in the management of various ailments such as memory disturbances, fever and nervous disorders. It has been reported to have beneficial properties such as antioxidant activity, anti-inflammatory and neuroprotective activity. Therefore, we aimed to assess the potential benefits of Phoenix dactylifera L. in the rotenone rat model of Parkinson's disease. Methods: In this study, thirty-five (35) male Wistar rats were divided into five groups (I-V) consisting of seven (7) rats each. Group I, which served as control, was administered distilled water (1ml/kg, orally), Group II received Olive Oil (1ml/kg vehicle, intraperitoneally), and groups III-V were the treatment groups. Group III received rotenone (3 mg/kg, i.p) only. Groups IV and V were administered Rotenone (3 mg/kg, i.p) followed concurrently by the n-butanol fraction of Phoenix dactylifera (500 mg/kg and 1000 mg/kg, respectively, orally). All administration lasted for 21 days. Effect of n-butanol fraction of Phoenix dactylifera was assessed was assessed by neurobehavioural tests using Open field test for locomotion, footprint analysis for gait disorder and beam walk test for motor coordination and balance, microscopic assessment of the substantia nigra and cerebellar cortex and brain tissue concentration level of dopamine, glutamate, acetylcholinesterase. Results and Discussion: Results revealed administration of rotenone induced remarkable alterations in locomotor activity and footprint parameters, histoarchitectural distortions in substantia nigra, decreased dopamine level and increased endogenous acetylcholinesterase level. However, Phoenix dactylifera L. was able to remarkably ameliorate motor deficits and confer some preservation to the substantia nigra thereby increasing the dopamine level. Findings from this study suggest the potential of Phoenix dactylifera L. in attenuating rotenone-induced neurotoxicity and could be beneficial in the management of Parkinson's-like symptoms.

Keywords: Dopamine, Acetylcholinesterase, Microscopy, Motor Deficit.

Symposia

Symposium 23

Epidemiology, physiopathological and therapeutic aspects of multiple sclerosis: an emerging disease in Africa

Organizer: **Fatiha Chigr & Samir Ahboucha**

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Abstract:

Multiple sclerosis (MS) is a chronic inflammatory and demyelinating disease of the central nervous system that typically affect young adults, especially women. Recently several body of evidence suggests that many neuro-inflammatory and neurodegenerative diseases are increasing in Africa and middle Est including MS. The aim the proposed symposium is to shed light on MS disease and address an update of the epidemiological and clinical aspects of MS in Morocco and Africa. The symposium will address particular symptoms that affect MS patients including fatigue and depression and their relationship with disease severity. So far, the pathophysiologic mechanisms for MS were not fully elucidated although inflammatory, systemic, blood brain barrier, immune system, vascular, neuromodulatory, and demyelinating mechanisms where suggested to play a role. The symposium will also address potential therapeutic aspects and possible pathophysiological components involving the role of adenosinergic and serotoninergic systems in experimental model of chronic MS.

Speakers

Number	Speaker	e-mail	Title of the communication
SP23_1	Fatiha Chigr	f.chigr@usms.ma	Multiple sclerosis in Morocco: Epidemiological, clinical, and therapeutic profile.
SP23_2	Rachid Lotfi	r.lotfi@usms.ma	Impact of multiple sclerosis on patients' quality of life in Morocco
SP23_3	Olamide E Adebiyi	olamideadebiyi24@gmail.com	Rescuing cognitive dysfunction in a mouse model of hippocampal demyelination
SP23_4	Samir Ahboucha	s.ahboucha@usms.ma	Alterations in the serotonergic system and mood behaviors in a cuprizone-induced mouse model of multiple sclerosis

Symposia

SP23_1

Title

Multiple sclerosis in Morocco: Epidemiological, clinical, and therapeutic profile

Authors

Fatiha Chigr, Lotfi R 1, Bel Amgharia H 1, Ennaciri S 1, El Kardoudi A 1 1 Biological Engineering
Laboratory, Faculty of Science and Technology, Sultan Moulay Slimane University, Beni Mellal, Morocco.

Abstract

Multiple sclerosis (MS) is a neurodegenerative disease and the leading cause of non-traumatic neurological disability in young adults, with significant social and economic impact. In Morocco, studies on MS remain scarce. This research aims to improve understanding of the factors affecting MS care and to define its epidemiological, clinical, and therapeutic profiles in the country. The study surveyed 520 MS patients, with 69.4% female and an average age of 35.36 years. Most participants had a university-level education. Recruitment used snowball sampling due to accessibility challenges. Clinically, 80% presented with RRMS, 7% had familial MS, and the average disease duration was 10.9 years. First-line treatments were used by 66.5%, with 41.5% on immunomodulators. Additionally, 60.5% used traditional and complementary medicine. Sociodemographic factors influenced access to care. Vitamin D deficiency affected 77%, with a higher prevalence in women ($p = 0.0454$). Men were more prone to stress, hypertension, and overweight ($p < 0.0001$). Physical inactivity was reported in 28%, with men more affected ($p = 0.0104$). Quality of life scores averaged 47.62/100, with significant impacts on physical (47/100) and mental (43.14/100) health, worsening with age and disease duration. Fatigue, measured by EMIF-SEP, averaged 65.52, affecting physical, cognitive, and psychosocial states. In conclusion, the results of this study will help fill, at least in part, the large data gap on MS and will contribute to strengthening the quality of care for this disease in Morocco in general.

SP23_2

Title

Impact of multiple sclerosis on patients' quality of life in Morocco

Authors

Rachid Lotfi, Hind Bel Amgharia, Fatiha Chigr University Sultan Moulay Slimane, Faculty of Sciences and Techniques, Department of Biology, Beni Mellal, Morocco.

Abstract

Background and objectives: Multiple sclerosis (MS) is a chronic neurodegenerative disease that affects the central nervous system, primarily impacting young adult women. The disease can progress in a relapsing-remitting or progressive manner, often leading to physical disability and emotional distress, which results in a significant reduction in patients' quality of life. Methods: The primary objective of our study is to assess the impact of MS on the quality of life of affected patients. This study was conducted using the Arabic version of the MSQOL-54 and EMIF-SEP scales, as well as a series of questions relating to the socio-demographic characteristics of the participants. The target population comprised MS patients in Morocco, with a sample size of 170 participants. Results: The results of our study indicate that females are the most affected by MS, representing 67% of the cases. The disease primarily impacts young individuals, with a mean age of 33.25 years. Moreover, the study revealed that the quality of life scores, as measured by the MSQOL-54, averaged 47.62/100, indicating significant deterioration in the physical (47/100) and mental (43.14/100) dimensions. Quality of life showed severe impairment in men and patients with PPMS. Specifically, the study highlighted a marked deterioration in the three dimensions of the physical score: activity limitations (25.75), energy levels (39.04), and perceived health (41.63). Furthermore, the mental score was found to be lower compared to other studies. This decline is significantly influenced by factors such as gender, age, and the type of MS. The mean pathological fatigue score, assessed using the EMIF-SEP, was 65.52 (± 18.00). This symptom had a significant impact on the physical (70.38), cognitive (62.28), and psychosocial (73.91) abilities. Discussion: Multiple sclerosis profoundly affects quality of life by impairing physical, cognitive, and psychosocial functioning. Its complex and variable nature requires multidisciplinary management, including medical, psychological, and rehabilitative interventions.

Keywords: Multiple Sclerosis, Quality of Life, Physical Score, Mental Score, fatigue.

Symposia

SP23_3

Title

Rescuing cognitive dysfunction in a mouse model of hippocampal demyelination

Authors

Olamide Adebiyi; Fatiha CHIGR; LOTFI; Samir AHBOUCHA. Sultan Moulay Slimane University, Beni Mellal, Morocco.

Abstract

Demyelinating diseases such as multiple sclerosis are characterized by loss of myelin, disruption of nerve conduction, and impairments in cognitive functions, including working memory. Presently, there is a heightened search for effective therapies for treating these disorders and the accompanying cognitive dysfunctions. Recent findings indicate that myelination can be stimulated by artificially driven neuronal activity. Hence, we posited that the activation of excitatory (Gq) Designer Receptors Exclusively Activated by Designer Drugs by its designer ligand, clozapine-N-oxide (CNO), would increase the excitability of neurons and accelerate remyelination. In a 2x2 longitudinal study, we bilaterally injected the hippocampus with AAV9-CAMKIIα- hM3Dq-mCherry following focal demyelination induced by lysophosphatidylcholine (LPC). Thereafter, we activated CAMKIIα-neurons by administering CNO 3?10-day post-LPC injection, while a separate cohort of mice was given saline. In a touchscreen operant chamber, we used the trial-unique nonmatching-to-location (TUNL) task to investigate spatial working memory in the experimental mice at different time points (before and after LPC injection). We also used magnetic resonance imaging to monitor myelin dynamics at these time points. We observed that activation of CAMKIIα-expressing neurons in the demyelinated hippocampus significantly increased proliferating oligodendrocyte precursor cells and myelin basic protein. We found that the mice given saline displayed impairment in working memory and showed a significant decrease in the magnetic transfer ratio. Furthermore, activation of CAMKIIα- expressing hippocampal neurons rescued this cognitive impairment. Taken together, our results show that selective activation of CAMKIIα-expressing neurons in the hippocampus accelerates the speed of remyelination, suggesting that activity-induced repair of damaged myelin sheaths may be an attractive strategy for rescuing cognitive deficits in demyelinating diseases.

Symposia

SP23_4

Title

Alterations in the serotonergic system and mood behaviors in a cuprizone-induced mouse model of multiple sclerosis model of multiple sclerosis

Authors

Samir AHBOUCHA, Themoi Demsou SOUHOUDJI, Nahla OUARD, Alpha Amadou BAH, Assmaa TALI, Rajaâ JEBBOUJ, Samir AHBOUCHA

Multidisciplinary Laboratory of Research and Innovation (MLRI); Research Team: Technological Applications, Environmental Resources and Health, Sultan Moulay Slimane University, Polydisciplinary Faculty Khouribga, Morocco.

Abstract

Multiple sclerosis (MS) is a common demyelinating disease, with mood changes such as anxiety and depression being prominent symptoms. Serotonin (5-hydroxytryptamine; 5-HT) is a neurotransmitter involved in mood regulation. This study investigates the role of the 5-HT system in chronic MS, specifically its potential alteration in an animal model of MS. We hypothesize that the 5-HT system may be modified in MS, and we aim to explore its relationship with anxiety and depressive behaviors in a cuprizone (CPZ)-induced mouse model of MS. C57BL/6 mice were treated with 0.2% cuprizone in their diet for 5 weeks to induce demyelination. Control mice were given a standard diet. Demyelination was assessed by Luxol fast blue (LFB) staining and immunohistochemistry (IHC) targeting myelin proteolipid protein (PLP). 5-HT and its transporter protein (5-HTT) were evaluated by IHC using specific antibodies. Anxiety and depressive behaviors were assessed using the dark/light box and forced swim tests, respectively. CPZ-treated mice showed significant demyelination, confirmed by LFB staining and PLP IHC. CPZ-treated mice spent more time in the light compartment of the dark/light box, indicating heightened anxiety, and showed increased immobility in the forced swim test, suggesting depressive behavior. Additionally, there was increased 5-HT immunostaining in the dorsal and median raphe nuclei neurons and their cortical projections. This increase in 5-HT was accompanied by a reduction in 5-HTT fiber density. These findingsindicate that cuprizone treatment for 5 weeks increases 5-HT levels and cortical projections, along with a reduction in 5-HTT expression. This suggests enhanced serotonin production and availability, potentially contributing to the mood changes observed in CPZ-treated mice. These results support the involvement of the 5-HT system in behavioral alterations, demyelination, and potentially in the inflammatory processes related to MS.

Symposia

Symposium 24

Management of Neurodegenerative Disorders by Potential Bioactive Compounds

Organizer: **Olfa Masmoudi & Taoufik Ghrairi**

University Tunis el Manar, Tunisia
email: **taoufik.ghrairi@fst.utm.tn; olfa.masmoudi@fst.utm.tn**

Abstract:

Neurodegenerative disorders, including Alzheimer's disease (AD), Parkinson's disease (PD), Huntington's disease (HD), and Lewy body dementia (LBD), affect millions worldwide, leading to significant mental and physical disabilities as well as and socio-economic burdens on the population. While most of these diseases are closely linked to aging and share common aging-related biological features, they are also driven by cellular stress, resulting in biochemical alterations like abnormal protein aggregation, excessive production of reactive oxygen and nitrogen species, mitochondrial dysfunction, and neuroinflammation, all of which can damage brain cells.
Neuroprotective strategies typically focus on therapies that promote cell survival pathways and rehabilitation methods aimed at enhancing the brainâ€™s repair mechanisms or inhibiting cell death processes, thereby strengthening neurons' ability to resist disease. Recent advances have identified critical pathways and targets associated with neurodegenerative diseases and neuroprotection. This symposium highlights groundbreaking research for neurodegenerative disorders and offers various perspectives for advancing neuroprotective therapies.

- Speakers

Number	Speaker	e-mail	Title of the communication
SP24_1	Taoufik Ghrairi	taoufik.ghrairi @fst.utm.tn	A Proteomic Analysis to Study Neuroprotective Bioactivity of Essential Oil
SP24_2	Olfa Masmoudi	olfa.masmoudi @fst.utm.tn	Protective and Neurotrophic Effects of OctaDeca Neuropeptide (ODN) in in vitro and in vivo models of neurodegenerative diseases
SP24_3	Amira Zaky	Amzakyha @yahoo.com	Implication of differential targeting of key cellular modulators in pain management.
SP24_4	Gérard Lizard	Gerard.Lizard @u-bourgogne.fr	Implication of cholesterol oxidation products (oxysterols) in neurodegeneration: cytoprotective activities of polyphenols, tocopherols and fatty acids, representative nutrients of the Mediterranean diet

Symposia

SP24_1

Title

A Proteomic Analysis to Study Neuroprotective Bioactivity of Essential Oil

Authors

Taoufik GHRAIRI[1], Amel ABIDI[1], achwek meftahi[1], OLFA MASMOUDI[1], CLAUDIA LANDI[2]. (1)
Laboratory of Neurophysioloy Cellular Physiopathology and Biomolecules Valorisation - Faculty of Sciences of Tunis University Tunis El Manar, TUNSIA; (2) Siena university, italy

Abstract

Parkinson's disease (PD) is the second most prevalent neurodegenerative disorder that primarily affects the motor system, and occurs mostly in individuals aged 60 and over. PD is characterized pathologically by the progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta accompanied by the presence of α-synuclein containing Lewy bodies. Emerging research on the effects of aromatic plants and their essential oils (EO) has shown promising potential in delaying neurodegenerative processes with fewer or no side effects compared to certain pharmaceuticals. In this study, we analyzed the chemical composition of ECEO by GC/MS analysis. We then investigated its neuroprotective effect against 6-hydroxydopamine-induced neurotoxicity in murine neuroblastoma cells N2A and we provided new insight into the underlying molecular mechanism through proteomic analysis. GC and GC/MS analysis of the Eucalyptus Citriodora essential oil led to identifying and quantifying components, which accounted for 98%of the total oil. N2a cells were pre-treated with various dilutions of EO (1:1000, 1:2000, 1:3000, 1:4000, and 1:5000) for 24 hours, followed by exposure to 50 µM 6-OHDA for an additional 24 hours. Under our experimental conditions, ECEO alone had no toxic effects on N2a cells regardless of the dilution and time. ECEO significantly attenuated the production of both ROS and NO to approximately 95% and 100% respectively, suggesting that its neuroprotective effects may be mediated, at least in part, by reducing oxidative stress and nitric oxide production. Emerging research on the effects of aromatic plants and their essential oils (EO) has shown promising potential in delaying neurodegenerative processes with fewer or no side effects compared to certain pharmaceuticals. The functional analysis of differential proteins identified by MALDI-ToF mass spectrometry reveals a pronounced involvement in metabolic processes highlighting their essential roles in cellular energy regulation and metabolic homeostasis under neurotoxic conditions. Analysis of 6-OHDA-treated cells showed that the most significantly implicated pathways included glycolysis and gluconeogenesis, transcription HIF-1 targets and apoptosis. in contrast, cells pretreated with ECEO showed enrichment in pathways associated with neuronal structure, and energy metabolism.

Symposia

SP24_2

Title

Protective and Neurotrophic Effects of OctaDecaNeuropeptide (ODN) in in vitro and in vivo models of neurodegenerative diseases

Authors

Yosra Hamdi[1], Sada Mashadani[1], Ikram Ghouili[1], Amine Bourzam[1,2], Amira Namsi[1,5], Taoufik Ghrairi[1], Zekri Sami[1,3], Jérôme Leprince[2], David Vaudry[4], Gérard Lizard[5] and Olfa Masmoudi-Kouki1 1Laboratory of Neurophysiology Cellular Physiopathology and Biomolecule Valorisation, LR18ES03, Faculty of Sciences of Tunis, University Tunis El Manar, 2092 Tunis, Tunisia 2Normandy University, Neuronal and Neuroendocrine Differentiation and Communication, Inserm U1239, Rouen, France, 3Confocal Microscopy Unit. Faculty of Medicine of Tunis, University Tunis El Manar, Tunis, Tunisia 4Genetics and Pathophysiology of Neurodevelopmental.

Abstract

Octadecaneuropeptide (ODN) is a peptide belonging to the family of endozepines, which are exclusively produced by astroglial cells. There is now compelling evidence that the gliopeptide ODN rescues cultured neurons and astrocytes from apoptotis induced by various neurotoxic agents. ODN, at the subpicomolar range, has been shown to protect neurons and glial cells from neurotoxicity induced by several substances such as H2O2, 6-OHDA and MPTP. ODN also exerts a strong protective effect against oxidative stress-induced apoptosis on cultured neurons and astrocytes. ODN acts by preventing i) the accumulation and overproduction of intracellular ROS, ii) the depletion of GSH levels, and iii) the decrease of the expression and activity of the antioxidant enzymes provoked by oxidative stress. Furthermore, ODN down-regulated the expression of miR-34b and miR-29a and rescued the 6-OHDA-associated reduced expression of miR21. In vivo, ODN prevents degeneration of nigrostriatal DA neurons in a mouse model of Parkinson's disease, through mechanisms involving downregulation of neuroinflammatory, oxidative and apoptotic processes. The gliopeptide ODN exerts its cytoprotective effects by activation of ODN metabotropic receptor positively coupled to PKA, PKC and MAPK/ERK transduction pathway, which ultimately reduces the pro-apoptotic geneBax and stimulates Bcl-2 expressions, and inhibits the mitochondrial apoptotic pathway. Moreover ODN could promote scratch-wound closure of primary astrocytes through activation of ODN-metabotropic receptor and calcium/mTOR signaling pathway, which stimulates astrocyte proliferation The antiapoptotic and neurotrophic properties of ODN, including its antioxidant, antiapoptotic, and neurotrophic actions, suggesting that ODN derivatives could potentially be useful for treatment of cerebral injuries involving oxidative stress and neurodegeneration.

Symposia

SP24_3

Title

Implication of differential targeting of key cellular modulators in pain management

Authors

Amira Zaky, Samar Harmoush, Mohammad Abdelkader, Ahmad Bassiouny. Alexandria University, Egypt.

Abstract

Pain is a complex and multifactorial condition marked by dysregulated signaling pathways. It is generally classified into two categories: neuropathic pain and inflammatory pain. Inflammatory pain specifically results from the activation and alteration of nociceptor function, influenced by immune signals and signals from damaged cells. Inflammatory pain mechanisms are driven by several pro-inflammatory mediators, including cytokines such as interleukin-1β (IL-1β), interleukin-6 (IL-6), and trigger inflammatory signaling pathways, most commonly the NF-kB, and tumor necrosis factor-alpha (TNF-α). The AP endonuclease 1 (APE1) plays a critical role in cellular homeostasis, serving as a DNA repair enzyme and a modulator of transcription factors, especially NF-kB. Changes in APE1 expression and sub-cellular localization are closely linked to inflammatory pain, particularly through its redox functions, with redox activity and nuclear accumulation in neurons and astrocytes being vital for the onset and persistence of pain. This study uses a formalin-induced pain model in rats to investigate the effects of E3330 (APX-3330), a selective APE1/Ref-1 redox activity inhibitor, on inflammatory pain pathways in the spinal cord and brain. We hypothesize that E3330 will reduce inflammatory cytokines, oxidative stress biomarkers, and pain comorbidity disorder. Our experimental design included four groups: Group 1 (control, n=6) received saline; Group 2 (induced, n=10) was given 5% formalin; Group 3 (co-treated, n=10) was pre-treated with E3330 before formalin injection; and Group 4 (positive control, n=6) received E3330 alone. We evaluated oxidative stress biomarkers (Glutathione [GSH], Malondialdehyde [MDA], Nitric Oxide [NO]), pro-inflammatory cytokines (IL-1β, IL-6, TNF-α), inflammatory enzymes (APE1, cyclooxygenase-2 [COX-2]), and neurotransmitter levels (Dopamine) in hippocampus samples from these groups. We alsoconducted electron microscopy imaging of brain samples to assess structural changes associated with biochemical alterations. Preliminary results showed significant differences in key biomarkers, with the co-treated group displaying substantial reductions in pro-inflammatory cytokines and oxidative stress markers, indicating that E3330 effectively modulates the inflammatory response in this pain model. These findings suggest the potential for targeting APE1 and related pathways in developing novel pain management strategies, enhancing our understanding of the mechanisms underlying inflammatory pain.

Symposia

SP24_4

Title

Implication of cholesterol oxidation products (oxysterols) in neurodegeneration: cytoprotective activities of polyphenols, tocopherols and fatty acids, representative nutrients of the Mediterranean diet

Authors

Gérard LIZARD[3] ; **Imen GHZAIEL**[1-2]**.** 1-University Clermont Auvergne, Clermont Auvergne INP, CNRS, Institut Pascal, 63000 Clermont-Ferrand, France. 2-Lab-NAFS 'Nutrition-Functional Food & Vascular Health', Faculty of Medicine, University of Monastir, LR12ES05, Monastir 5000, Tunisia, 3-Université de Bourgogne, Inserm, 21000 Dijon, France, and PHYNOHA Consulting, 21121 Fontaine-lès-Dijon, France

Abstract

The most abundant lipid identified in the central nervous system (brain + spinal cord) is cholesterol, and there are now several evidences that cholesterol and its derivatives, such as those formed by cholesterol oxidation (oxysterols), have important roles in brain function. Studying cholesterol and oxysterols at the cerebral level is therefore a subject worthy of interest for addressing neurodegeneration which can result from aging and which is also observed in some neurodegenerative diseases. Noteworthy, neurodegeneration is often associated with a rupture of the RedOx homeostasis and with inflammation. Oxidative stress favors an increase of cholesterol oxidation products (oxysterols) leading in brain tissue to an accumulation of lipid peroxidation products including 7-ketocholesterol (7KC) and 7α-hydroxycholesterol (7α-OHC) formed by cholesterol autoxidation. In addition, modifications of cholesterol metabolism in the brain of patients with AD are associated with a successive increase and decrease of 24S-hydroxycholesterol (24S-OHC) formed from cholesterol by CYP46-A1 in neuronal cells. At advanced stages of AD, the rupture of the blood brain barrier (BBB), in part due to inflammatory processes, favors the entrance in the brain of 27-hydroxycholesterol (27-OHC) formed from cholesterol by CYP27-A1. 25-hydroxycholesterol (25-OHC), produced by cholesterol 25-hydroxylase (CH25H), is a known modulator of both inflammation and lipid metabolism in microglial cells. However, as 7KC, 7α-OHC, 24S-OHCand 25-OHC have pro-oxydant and pro-inflammatory activities and can trigger a hybrid type of cell death defined as oxiapoptophagy (involving OXIdative stress + APOPTOsis + autoPHAGY), they are supposed to play major roles in neurodegeneration and brain damages. Currently, several molecules widely represented in the Mediterranean diet, such as polyphenols (resveratrol, quercetin), tocopherols (tocopherol), and fatty acids (α-linolenic acid, eicosapentaenoic acid, docosahexaenoic acid, oleic acid) strongly attenuate 7KC- and 7α-OHC-induced oxiapoptophagy. Noteworthy, similar cytoprotective activities than oleic acid, were observed with the synthetic oleate derivative, sulfo-N-succinimidyl oleate (SSO), without lipid droplets accumulation. Among these different cytoprotective molecules, the most efficient and the less cytotoxic, is tocopherol which has the ability to cross the BBB. A better knowledge of the roles of oxysterols in the brain should contribute to better know the physiopathology of several neurodegenerative diseases and open new therapeutic perspectives.

Symposia

ISN-UM5-sponsored

Symposium 25

ISN sponsored UM5 School Alumni symposium

Organizer: **Nouria Lakhdar-Ghazal**

African Center for Advanced Training in Neuroscience,
Mohammed V University, Rabat, Morocco
email: **nlakhdarghazal@gmail.com**

Abstract:

African neuroscience developed with the establishment in 2000 of the African Neuroscience Schools, supported and funded by the IBRO. ISN quickly joined this training programme by co-funding these schools or by being the main sponsor. In this way, Africa in general and Morocco in particular has seen the gradual emergence of IBRO schools, IBRO-ISN schools and or ISN schools. In 2015, the IBRO created two centres of excellence in neuroscience, which it named African Centres for Advanced Training in Neuroscience, one in Cape Town and the other in Rabat. These two centres have set up very high-level teaching programmes in basic and clinical neuroscience. Many young neuroscientists, doctoral students, post-doctoral students, neurologists and teachers at the start of their careers have benefited from this training and have been injected into the neuroscience community. Many of them are now established researchers, in charge of research structures in Africa or outside Africa, and have taken over as trainers. Today, the centres continue to operate with the same scientific and pedagogical quality, and the alumni of these training courses have attained qualifying neuroscientific levels, enabling them to participate in all scientific communication activities in Africa and throughout the world.

The Rabat centre, attached to Mohammed V University, has been supported and co-sponsored by IBRO and ISN since 2021. On the occasion of the organisation of the International Conference of the Society of Neuroscientists of Africa, the International Society for Neurochemistry accepted to financially support a symposium of the alumni ISN supported schools. Khadija Boualam from Morocco will participate with a work on "Cognitive decline during Insuline resistance and protective effect of Brocchia cinerea", Maahir Kauchali from South Africa will present his work on "the pathogenesis of C. gattii-induced meningitis", Mundih Njohjam from Cameroon but leaving in Senegal will talk about "the Cognitive Impairment induced by Onchocerciasis-associated Epilepsy" and Leviticus Arietahire from Nigeria will participate with a work on the "Exposure to Ibuprofen Differentially Alters the Morphology and Synaptic Integrity of the Prefrontal Cortex and Amygdala".

Khadija Boualam and Maahir Kauchali will represent the school 2023 on "Neuroimmunology, Neuroinflammation and Neuroinfectio" while Mundih Njohjam and Leviticus Arietahrire will represent the school on Basal Ganglia and Motor Disorders organized in 2024.

Symposia

Speakers

Number	Speaker	e-mail	Title of the communication
SP25_1	Mundih Njohjam	Njohjammmundih@yahoo.com	NeuroOnchocerciasis: A Case-Control Study Assessing the Cognitive Impairment induced by Onchocerciasis-associated Epilepsy
SP25_2	Maahir Kauch ali	KCHMAA001 @myuct.ac.za	Characterising the pathogenesis of C. gattii-induced meningitis
SP25_3	Khadija Boualam	khadijaboualam94 @gmail.com	Targeting cognitive decline in Insulin resistance: The neuroprotective role of Brocchia cinerea essential oil abdominal massage.
SP25_4	Leviticus Arietarhire	Arietarhirel @babcock.edu.ng	Exposure to Ibuprofen Differentially Alters the Morphology and Synaptic Integrity of the Prefrontal Cortex and Amygdala of Adult Male Wistar Rat

Symposia

SP25_1

Title

Neuro-Onchocerciasis: A Case-Control Study Assessing the Cognitive Impairment Induced by Onchocerciasis-associated Epilepsy

Authors

Mundih Njohjam[1], Leonard Ngarka[1], Leonard Nfor[2], Cheikh Anta Diop[2], Alfred Njamnshi[1],

1: University of Yaounde I, 2: University, Yaounde Central Hospital.

Abstract

Onchocerciasis-associated epilepsy (OAE) is associated with significant cognitive impairment. Data on the prevalence and pattern of neurocognitive impairment caused by OAE is sparse in Africa. The aim of this study was to determine the prevalence, spectrum, and severity of neurocognitive impairment among patients with OAE in two onchocerciasis-endemic communities. Methods A case-control study was conducted in two rural villages in an onchocerciasis-endemic zone in the central region of Cameroon. Based on diagnostic criteria established in other studies, we diagnosed patients with OAE and matched by age, sex, and educational level to controls. Neurocognitive functions were assessed using the Montreal Cognitive Assessment Test, Frontal Assessment Battery, International HIV Dementia Scale, Dubois? Five Words Tests, and Isaac Set's tests. The threshold for statistical significance was set at P<0.05. Logistic regression analysis was done to identify factors associated with cognitive impairment. Results: The study included fifty people with OAE and fifty healthy controls. The mean age for the cases was 26.3 years, and for the controls, it was 26.4 years. Cases were more impaired cognitively than the controls, 96% for the cases vs. 78% for the controls (P=0.007). The main cognitive functions affected were memory (86% of cases vs 30% of controls, P<0.001, verbal fluency (80% of cases Vs 42% of controls, P<0.001, OR = 15.6), and executive functions (80% of cases as against 42% of controls, P<0.001). The main factors associated with poorer neurocognitive outcomes were longer duration of epilepsy (P<0.001), frequent seizures(P<0.001), low educational level (P=0.027), generalized tonic-clonic seizures (P<0.001) and early age of onset of epilepsy (0.001). Conclusion: Onchocerciasis-associated epilepsy causes severe neurocognitive impairment in those affected. The need to eliminate onchocerciasis and, hence, reduce the prevalence of OAE has never been more imperative. Keywords: Onchocerciasis-associated epilepsy, neurocognitive impairment, Onchocerciasis

Symposia

SP25_2

Title

Characterising the pathogenesis of C. gattii-induced meningitis

Authors

Maahir kauchali, Lilitha cengani and Dr Rachael Dangarembizi.

136 second avenue rondebosch east Cape town South africa

Abstract

Cryptococcal meningitis (CM) is an opportunistic fungal infection of the brain that is responsible for approximately 181,000 annual deaths, 75% of which are from Sub-Saharan Africa. CM has historically been associated with people with weakened immunity, but as global temperatures rise, there is an increased incidence of CM infections in immunocompetent individuals, caused by newly emergent species of Cryptococcus. Recent research has shown that this new type of CM is caused by Cryptococcus gattii but the pathogenesis of the disease is not fully described. To address this gap, we investigated C. gattii-induced meningitis using a mouse model. Mice were intravenously infected with C. gattii and euthanised at days 1, 3 and 6 post-infections. Fungal burden and cytokine analyses were performed on tissue homogenates obtained from one hemisphere of the brain, lungs, spleens, and blood. We used histology and immunohistochemistry to determine the spatial and temporal distribution of the fungus in the brain through the course of the disease. We additionally used organotypic brain slice cultures to characterise the neuroimmune response to C. gattii at the cellular and molecular level. Additionally, we compared the disease progression to that caused by C. neoformans to determine if there were any species differences between C. gattii- and C. neoformans-induced CM. Preliminary data from this study shows that the C. gattii, like C. neoformans, invades the brain via the vascular route but fungal loads of C. gattii (per unit of organ mass) were lesser than those observed in C. neoformans-induced CM. We anticipate that the data from our characterisation of the neuroimmune response will enable us to better understand the mechanisms of brain injury during C. gattii-induced CM.

Symposia

SP25_3

Title

Targeting cognitive decline in Insulin resistance: The neuroprotective role of Brocchia cinerea essential oil abdominal massage

Authors

Khadija Boualam[1,2], Afaf Allaoui[3], Ahmet Buğra Ortaakarsu[4], Nidal Fahsi[1], Khalid Taghzouti[2].

1-AgroBioSciences Program, College of Agriculture and Environmental Sciences, University Mohammed VI Polytechnic, Ben-Guerir, Morocco. 2-Physiology and Physiopathology Team, Genomics of Human Pathologies Research Center, Faculty of Sciences, Mohammed V University in Rabat, Rabat, Morocco. 3-Department of Biology, Faculty of Sciences, Mohammed V University, Rabat, Morocco. 4-Department of Chemistry, Faculty of Science, Gazi University, Turkey Mansour Sobeh1.

Abstract

Background: Cognitive decline is increasingly recognized as a complication of insulin resistance (IR), often preceding more overt metabolic disorders like type 2 diabetes. Insulin resistance negatively impacts brain function, particularly in regions such as the hippocampus and cortex, which are critical for memory and learning. Conventional therapies for insulin resistance may help mitigate cognitive decline, but complementary approaches like aromatherapy using essential oils have shown potential neuroprotective effects. Objective: This study aims to assess the impact of Brocchia cinerea (B.cinerea) essential oil (EO), applied in abdominal massage, on cognitive function in a rat model of high-fat, mono-carbohydrate diet (HFCD)-induced insulin resistance. Comparisons were made with metformin, a standard insulin-sensitizing agent, to evaluate the cognitive protective potential of this alternative approach. Methods: Wistar Rats were divided into seven groups (n=6 per group): normal diet (ND), HFCD (negative control), HFCD with coconut oil (vehicle), HFCD with 2%, 5%, and 10% B. cinerea EO, and HFCD with metformin (positive control). Behavioral tests including the Open Field Test (OF), Novel Object Recognition (NOR), and Y-Maze were employed to assess exploratory behavior, memory, and spatial cognition. Inflammatory (IL-6, TNF-α, and IL-10) and oxidative stress markers (glutathione, GPx, and catalase) were measured in brain regions, specifically the hippocampus and cortex. Results: The HFCD group exhibited significant cognitive impairments, characterized by reduced exploration in the Open Field Test, diminished recognition memory in NOR, and impaired spatial memory in the Y-Maze. These effects were accompanied by elevated inflammatory markers and oxidative stress in the hippocampus and cortex. Treatment with B. cinerea EO, particularly at the 10% dose, significantly improved cognitive performance, showing comparable effects to metformin. The essential oil reduced neuroinflammation and oxidative stress, suggesting its neuroprotective potential.

Symposia

SP25_4

Title

Exposure to Ibuprofen Differentially Alters the Morphology and Synaptic Integrity of the Prefrontal Cortex and Amygdala of Adult Male Wistar Rat.

Authors

Leviticus Oghenevurinrin Arietarhire, Precious Agnes Oduh, Toluwanimi Omolara Afolabi, Oladimeji Emmanuel Soremekun, Pelumi Ezekiel Alege, Stephen Opeyemi Adeleke, Abayomi Sodipo, Joseph Igbo Enya, John Afees Olanrewaju

Abstract

Background: Ibuprofen, a widely used NSAID with analgesic and anti-inflammatory properties, has shown conflicting effects on the nervous system; some studies suggest neuroprotective effects, while others indicate potential harm. Aim: This research aimed to investigate the specific effects of long-term ibuprofen exposure on the amygdala and prefrontal cortex. Methods: Twenty-four male Wistar rats were divided into three groups of eight animals each and treated orally as follows: Group 1 received distilled water for 28 days, Group 2 received 50 mg/kg of ibuprofen for 28 days, and Group 3 received 200 mg/kg of ibuprofen for 28 days. Body weight was recorded, and behavioral assays (open field, tail suspension, and tail flick tests) were conducted. We assessed oxidative redox parameters, acetylcholinesterase levels, and total protein profiles in the brain and amygdala using biochemical assay kits. Morphological examinations of the prefrontal cortex and amygdala were conducted using H&E and Cresyl Violet staining, along with assessments of astrocytic morphology, synaptic integrity, and neuronal markers using anti-GFAP, anti-Synaptophysin, and anti-NeuN antibodies. Molecular docking analyses were also performed to examine interactions between ibuprofen and specific target proteins (MAOA, oxytocin, and BCHE). Results: Molecular docking indicated that ibuprofen strongly interacted with MAOA, oxytocin, and BCHE in their inhibitory conformations. In the animal study, ibuprofen had minimal impact on the prefrontal cortex; however, effects on the amygdala were pronounced, resulting in behavioral deficits, oxidative stress, neuronal loss, reduced Nissl bodies, astrogliosis, and impaired synaptic plasticity.Conclusion: This study reveals that prolonged use of ibuprofen negatively affects amygdala-related functions, potentially through oxidative stress and oxytocin inhibition, underscoring the need for further research to understand the mechanisms and clinical implications of long-term ibuprofen use.

Keywords : Empathy, Ibuprofen, Amygdala, Prefrontal cortex, Neurons

Symposia

Symposium 26

Recent advances at the interface of neuroscience and AI (NeuroAI)

Organizer: **Srikanth Ramaswamy** (UK)
co-organizer: **Jie Mei** (Austria)

Biosciences Institute, Newcastle University Framlington Place,
NE2 4HH Newcastle upon Tyne, United Kingdom
Altenberger Str. 66c/Science Park 4, 4040 Linz, Austria
email: srikanth.ramaswamy@newcastle.ac.uk & jie.mei@it-u.at

Abstract:

Biological neural networks adapt and learn in diverse behavioral contexts. Artificial neural networks (ANNs) have leveraged principles from biology to solve complex problems. However, despite their success in specific tasks, ANNs still fall short of matching the flexibility and adaptability of biological cognition. This symposium will highlight recent advances in neuroscience that deepen our understanding of both biological and artificial intelligence. Neuroscience-inspired artificial intelligence (NeuroAI) has produced powerful tools for solving complex tasks. Efforts such as the US BRAIN Initiative, the NSF National AI Research Institutes, and the EU Human Brain Project are laying the neural and cognitive foundations for future AI systems.

This symposium will bring together researchers to synthesize an integrative view of how insights from biological intelligence can catalyze the development of next-generation AI. The symposium is designed for doctoral students, postdocs, early and mid-career researchers, industry experts, and clinicians. An international panel will present short talks on how neural networks regulate learning and higher-order cognition, fostering dialogue aimed at identifying common organizing principles shared between biological and artificial intelligence. A concluding panel discussion will explore how advances in brain research can inspire the next generation of AI.

Speakers

Number	Speaker	e-mail	Title of the communication
SP26_1	Maryeme Ouafoudi	Omaryeme @gmail.com	Evolving Learning Rules on and for Neuromorphic Hardware
SP26_2	Sadiq ADEDAYO	sadiq.adedayo @univie.ac.at	Causal Discovery to link Neuronal Activities to Behaviour
SP26_3	Jie Mei	jie.mei @it-u.at	Improving the adaptive and continuous learning capabilities of artificial neural networks using multi-scale, neuromodulation-aware rules
SP26_4	Nina Hubig	nina.hubig @it-u.at	Explainability and Interpretability in the Neurosciences

Symposia

SP26_1

Title

Evolving Learning Rules on and for Neuromorphic Hardware

Author

Maryeme Ouafoudi

Abstract

SP26_2

Title

Causal Discovery to link Neuronal Activities to Behaviour

Author

Sadiq Adedayo

Abstract

SP26_3

Title

Improving the adaptive and continuous learning capabilities of artificial neural networks using multi-scale, neuromodulation-aware rules

Authors

Jie Mei,
IT: U Interdisciplinary Transformation University Austria, Linz, Austria

Abstract

Continuous, adaptive learning, the ability to constantly adjust to changing environments and improve performance is a defining feature of both natural and artificial intelligence. Biological organisms excel in acquiring, transferring, and retaining knowledge while adapting to dynamic conditions, making them a rich source of inspiration for artificial neural networks (ANNs). Although the underlying principles of the biological brain have inspired the development of learning algorithms for ANNs, physiological processes such as neuromodulation are typically oversimplified in these models. Neuromodulators like dopamine (DA), acetylcholine (ACh), serotonin (5-HT), and noradrenaline (NA) play a critical role in brain function, operating at multiple scales to facilitate adaptive responses to environmental changes. These neuromodulatory processes range from local synaptic plasticity to global network-wide adaptations, allowing for flexible, context-dependent learning. Notably, the relationships between neuromodulators and their roles in modulating sensory and cognitive functions are more complex than previously thought, involving intricate "many-to-one" mappings between neuromodulators and tasks. This talk will focus on the following key areas: 1. How multi-neuromodulatory interactions enrich single-neuromodulator-driven learning. 2. The impact of neuromodulators across different spatial and temporal scales. 3. Strategies for integrating or approximating neuromodulatory learning processes in ANNs.I will begin with a brief overview of past and current efforts to incorporate multi-scale neuromodulatory components into computational models, highlighting how these efforts have enabled more flexible and adaptive learning in behavioral tasks. Next, I will present my research on integrating multi-scale, parallel neuromodulatory mechanisms into ANNs, with insights drawn from both behavioral experiments and single-unit recordings. I will then illustrate how neuromodulation-inspired mechanisms, such as DA-driven reward processing and NA-driven cognitive flexibility, can enhance ANN performance in a Go/No-Go

task. Finally, I will outline key directions for future research. In conclusion, by incorporating multi-scale neuromodulation, we aim to bridge the gap between biological learning processes and artificial systems, ultimately enabling ANNs with greater flexibility, robustness, and adaptability.

Symposia

SP26_4

Title

Explainability and Interpretability in the Neurosciences

Authors

Nina Nubig. Altenbergerstrasse 66c, 4040 Linz, Austria

Abstract

In this talk, we will introduce and discuss the fundamentals of explainability in neuroscience, emphasizing how computational models process neural and behavioral data. We will explore foundational explainable artificial intelligence (XAI) techniques, such as saliency maps and SHAP values, which identify influential features, and advanced approaches like GraphLIME, GNNExplainer, and topological data analysis, tailored for graph and network structures like the connectome. By the end, participants will understand how these methods foster trust, validation, and deeper insights in neuroscience research.

Symposia

Symposium 27

Advancing Neuroscience Education in Africa through Capacity Building

Organizer: **Sharon L. Juliano**

USUHS, Bethesda, MD USA
email: **sharon.juliano@usuhs.edu**

Abstract:

Over the last decade, opportunities to study Neuroscience in Africa have improved. Several endeavors emerged on the continent, expanding the ability of African students to investigate this discipline. 1. As Neuroscience becomes more visible, very few programs in Africa offer comprehensive Neuroscience degrees. Dr Sadiq Yusuf established multiple University level curricula that offer such programs. He found that critical factors to establish these platforms include support from individual departments, faculty, and university leaders. Also crucial for success is an understanding of University regulations and the rationale underlying course content and objectives, principles of staffing, facilities, and finance. The advent of these programs represents an important resource to cement African Neuroscience excellence. 2. Dr. Mahmoud Bukar Maina and colleagues created BioRTC, a scientific institute with a Neuroscience emphasis. Through community engagement, stakeholder involvement, and strong collaborations, including significant contributions from TReND in Africa, Dr. Maina established a state-of-the-art facility. The support of TReND in acquiring equipment was pivotal, creating a hub capable of supporting a world-class Neuroscience research community. 3. Dr. Sharon Juliano and colleagues initiated the Teaching Tools Workshops in Africa (TTW), providing teaching skills and Neuroscience content to young faculty. The TTW offers modules that integrate seamlessly into various curricula, in part by reducing the overall complexity of this vital subject. Presented in 12 African countries, the TTW has reached over 400 attendees from 30 countries, indirectly benefiting over 10,000 students across Africa. 4. Dr. Royhaan Folarin emphasizes the role of SciComNigeria in bridging the gap between science and society in Nigeria. Established in 2018 by two Nigerians, SciComNigeria is a dynamic platform that promotes science communication and public engagement. Through workshops, webinars, competitions and outreach initiatives, SciComNigeria enhances scientific literacy and inspires the next generation of scientists. Its efforts are crucial in fostering a culture of science communication that empowers researchers and engages the public with the importance of scientific inquiry.

Speakers

Number	Speaker	e-mail	Title of the communication
SP27_1	Sharon L. Juliano	sharon.juliano @ usuhs.edu	Advancing Neuroscience Education in Africa through Capacity Building
SP27_2	Sadiq Yusuf	sadiqyus@ gmail.com	Neuroscience Education and Research Capacity Building in Africa
SP27_3	Mahmoud Bukar Maina	M.Bukar-Maina@ sussex.ac.uk	Advancing Neuroscience in Africa: The Biomedical Science Research and Training Centre (BioRTC) as a Model
SP27_4	Royhaan Folarin	royhaan.folarin@ oouagoiwoye.edu.ng	SciComNigeria: Advancing Scientific Literacy and Empowering Future Generations in Nigeria.

Symposia

SP27_1

Title

Advancing Neuroscience Education in Africa through Capacity Building

Authors

Sharon L Juliano,
USUHS, Bethesda, MD USA

Abstract

The Teaching Tools Workshops in Africa (TTW) have been held for nearly 20 years in over 10 African countries. Each Workshop hosts 15-25 applicants and presents tools to assist in teaching Neuroscience subjects. Because we hosted over 300 students and each of those students teaches others, we estimate that over the years we reached many thousands of African graduate, medical students, and residents. Each Workshop delivers elements of Neuroscience teaching including samples of lectures on key subjects necessary for any course in Neuroscience, pedogological tools, interactive laboratories, curriculum and test design. We include applicants from all corners of the continent and involved students from at least 28 African countries. The attendees include young faculty that have been teaching an aspect of Neuroscience for a few years. We introduce multiple techniques that faculty can use to engage students in large and small classrooms. The Workshop encourages group interaction and includes small groups to discuss how to integrate Neuroscience courses into a curriculum and introduce novel teaching methods and tools into their programs. We also work on presentation techniques and each attendee delivers a short lecture at the end of each session. It is essential to educate both teachers and the public about tools to simplify and make understandable diseases and structure of the brain, since so many are fearful about the subject. This is in part because the material is difficult and students are often insufficiently prepared for the subject. In addition, Neuroscience is not taught consistently, which does not prepare graduates to contradict the stigma of diseases that affect the nervous system. By learning methods to present material in a manner more conducive to learning and retaining the material, we hope to ultimately enhance diagnosis and treatment of neurologic and psychiatric diseases.

Symposia

SP27_2

Title

Neuroscience Education and Research Capacity Building in Africa

Authors

Sadiq Yusuf, Mahmoud Bukar Maina, Sussex Neuroscience, School of Life Sciences, University of Sussex, Brighton, UK. **Royhaan Folarin**, Department of Anatomy. **Olabisi Onabanjo** University, Nigeria **Sharon L. Juliano**, USUHS, Bethesda, MD USA. Newgate University Minna, Nigeria

Abstract

Neuroscience, a cornerstone of understanding the brain and neurological health, remains underdeveloped in Africa despite the continent's significant burden of neurological disorders, including stroke, epilepsy, neuroinfectious diseases, and neurodegenerative conditions. This paper explores the current challenges in neuroscience education and research in Africa. It proposes actionable strategies for capacity building, emphasizing the establishment of neuroscience programs, creation of research centers of excellence, interdisciplinary collaboration, and the integration of emerging technologies. The importance of advocacy, policy support, and regional and international partnerships is underscored as essential enablers of progress. Opportunities such as Africa's youthful demographic, global interest in brain research, and technological advancements are highlighted as avenues for growth. The paper concludes by recommending targeted investments in education, research funding, mentorship programs, and digital tools to enhance neuroscience capacity across the continent. By addressing these priorities, Africa can overcome systemic barriers, advance neurological health, and contribute significantly to global neuroscience research and innovation.

Symposia

SP27_3

Title

Advancing Neuroscience in Africa: The Biomedical Science Research and Training Centre (BioRTC) as a Model

Authors

Mahmoud Bukar Maina, Biomedical Science Research and Training Centre, Yobe State University, Damaturu, Yobe State, Nigeria & Sussex Neuroscience, School of Life Sciences, University of Sussex, Brighton, UK

Abstract

Established in 2021 through strategic stakeholder engagement, community involvement, and international collaborations, the Biomedical Science Research and Training Centre (BioRTC), located in Yobe State, Northeast Nigeria, is an effort to advance neuroscience research and capacity building in Africa. BioRTC is Nigeria's first open-access core facility equipped with state-of-the-art bioscience tools, including two laser scanning confocal microscopes. Here, I will highlight BioRTC's commitment to fostering on-continent research excellence, its achievements in hosting impactful summer schools and workshops, and its dedication to training the next generation of African scientists. I will discuss our open-access model, inclusivity, and resource optimisation strategies, which have enabled us to overcome challenges commonly faced in resource-limited settings. We invite neuroscientists, institutions, and funding bodies to collaborate with BioRTC by utilising its cutting-edge facilities to help advance research, capacity building, and innovation, helping to drive forward the global neuroscience agenda while strengthening Africa's scientific ecosystem.

Symposia

SP27_4

Title

SciCom Nigeria: Advancing Scientific Literacy and Empowering Future Generations in Nigeria

Authors

Royhaan Folarin[1], Mahmoud Bukar Maina[1], Olabisi Onabanjo[2].

1: Sussex Neuroscience, University of Sussex, UK**, 2:** Department of Anatomy**,** University, Nigeria

Abstract

Science communication plays a pivotal role in bridging the gap between scientific research and society, fostering public understanding and engagement. SciComNigeria, established in 2018, stands as a beacon in Nigeria's science communication landscape, championing innovative approaches to empower researchers and inspire the public. The organization has implemented several impactful initiatives, including the National Life Sciences Competitions (NLSC), which have, over three consecutive editions, awarded cash prizes, delivered inspiring lectures by erudite international faculty, and established mentorship opportunities for winners across secondary schools and universities. These programs have profoundly impacted participants, many of whom continue to excel in life sciences, STEMM, and research. Their ongoing engagement with SciComNigeria as they progress in their studies highlights the program's enduring influence. During the COVID-19 pandemic, SciComNigeria actively dispelled myths and misconceptions about the virus's pathogenesis and therapeutic interventions by publishing scientifically accurate articles through its website. The organization also organized periodic webinars on topics central to advancing biomedical research in low-resource settings. Notably, collaborations with international bodies enriched these webinars, addressing essential topics such as realistic resumes which uncovered the paths of accomplished scientists. Building on its successes, SciComNigeria's competitions have evolved to include innovative trends such as translating scientific content into local Nigerian languages to enhance accessibility and creating video-based content to illustrate these translations. This shift focus from essays to rewarding creative engagement, reflecting the changing dynamics of learning ina technology-driven world. Since its inception, SciComNigeria has continually refined its mission to address the evolving needs of society. By fostering collaborations, spearheading impactful programs, and adapting to emerging trends, the organization remains at the forefront of science communication in Nigeria. These efforts empower researchers and engage the public, driving scientific literacy and inspiring future generations to contribute meaningfully to local and global challenges.

Symposia

Symposium 28

The Brain Research International Data Governance & Exchange (BRIDGE): African perspectives

Organizer: **Amadi Ihunwo (South Africa)**

School of Anatomical Sciences, Faculty of Health Sciences,
University of the Witwatersrand, Johannesburg, South Africa
email: **amadi.ihunwo@wits.ac.za**

Abstract:

The Brain Research International Data Governance and Exchange (BRIDGE) project aims to create responsible and sustainable governance frameworks for data sharing, building toward a long-term goal of forming a sustainable global consortium to develop, operate, update and disseminate a robust brain and mental health international data governance framework (IDGF). During the symposium the current global scenario on international data governance will be presented in addition to perceptions from African on the neurodata governance from the Ethics, People with Lived Experience (PWLE) and Technology domains. A discussion on the potential role of the Society of Neuroscientists of Africa (SONA) as an organization partner within the BRIDGE project will follow.

Speakers

Number	Speaker	e-mail	Title of the communication
SP28_1	Ebere Wogu	eberechi.wogu @uniport.edu.ng	Brain Data Governance in Africa: Ethical Considerations, Gaps and Best Practices.
SP28_2	Uchenna B. Amadi-Ihunwo	uchey4adi @gmail.com	People With Lived Experiences and Data Governance in Africa
SP28_3	Damian Eke	Damian.Eke @nottingham.ac.uk	Technical Challenges and opportunities of Brain Data Governance in Africa.
SP28_4	Franco Pestilli	Pestilli @utexas.edu	Bridging the gap in data sharing: Developing International Data Governance Frameworks for Brain Health Research.

Symposia

SP28_1

Title

Brain Data Governance in Africa: Ethical Considerations, Gaps and Best Practices

Authors

Eberechi Wogu[1], Franco Pestilli[1], Amadi Ogonda Ihunwo2, Damian Eke[3], Amadi-Ihunwo, Uchenna[2].

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2: University of the Witwatersrand, Johannesburg, South Africa. 3 : Nottingham University, Nottingham, UK.

Abstract

The emerging advancement in neuro-technology and its ability to collect, analyze, and utilize brain data presents exceptional opportunities and also raises significant ethical concerns, increasing the relevance of brain data governance in today's society. In Africa, the rich and diverse cultures undoubtedly shape the beliefs and ethical principles of Africans. The ethical implications that shape existing brain data protection in Africa have remained underexplored. This study presents a comprehensive, structured view of the ethical considerations, policy gaps, and best practices in brain data governance in Africa. Method: We conducted a Multi-vocal Literature Review (MLR) by systematically collecting an initial pool of 46 academic studies and 37 grey studies from popular databases by following a well-known MLR guideline. We also carried out a survey across the 5 geopolitical regions of Africa. Results: We identified 21 ethical considerations and 9 policy gaps some of which include language, literacy and cultural barriers, trust issues, ethics dumping, data colonialism, fear of data misuse, ambiguity about data ownership and data subject rights, commercial exploitations, mental health stigma, access to benefits, anonymization limitations, unauthorized access, lack of motivation to share data, cross-border issues, lack of clear ethical guiding frameworks. Some of the best practices observed were the mandatory institutional research ethics reviews, informed consent use and penalties for breaching data protection guidelines. Discussion: Our findings point out the need for increased sensitization on data subject rights and data ownership through community engagements. There is also a need for increased awareness on the relevance of FAIR brain data among African neuroscience research communities, clinicians, and patients. Our findings also show the need to modify existing ethical frameworks and create clear ethical guidelines for brain data anonymization, reporting of incidental findings and ensuring equity in sharing of benefits. Of great importance is the involvement of stakeholders in Africa in the creation of a brain data governance framework that is culturally sensitive and inclusive. Conclusion: These strategic roadmaps for brain data governance in Africa would ensure acceptable and responsible data governance at each stage of the data life cycle, from collection, storage, processing, sharing, use to deletion.

Symposia

SP28_2

Title

People With Lived Experiences and Data Governance in Africa

Authors

Uchenna Beatrice Amadi-Ihunwo, damina eke, University of Nottingham, United Kingdom, franco pestilli, University of Texas, Austin, TX, USA; Amadi ogonda ihunwo, university of the Witwatersrand, Johannesburg, south Africa

Abstract

Mental health data governance centers around the generation and sharing of “human” data amongst researchers, management and strategic interrogations worldwide. This suggests that the said data is about people and should aim to represent people. Therefore, mental health data is expected to speak to people living with a mental health diagnosis and related challenges. Unfortunately, mental health is not a consciously and intentionally focused health issue in Africa, as it is scantly represented in research studies or policies. Likewise, the voices of the people living with and those affected by mental health challenges are exclusive in the available studies. Methods: A critical and integrative literature review for the study. There are extensive research done on mental health but the paucity of studies around the people with lived experience (PWLE) of mental health challenges and their data management and sharing. Results Evident in the findings from the literature showed that African leaders are yet to consider mental health challenges as critical medical challenges, such as lack of finances to mental health, professional training, diagnostic equipment, advocacy and research funding. The format of data collection and storage on mental health at most mental health facilities are through hard copy folders. More PWLE data get lost from the indigenous treatments through the faith-based and traditional healers based on the concept of unknown causes of mental health challenges. Discussion The use of vignette as research tool to collect empirical data is designed due to the sensitivity nature of this study. Participants for data collection will involve PWLE and their caregivers, psychiatric nurses and psychiatrists in four African countries. Data will also be collected from men and women using focus group discussion (FGD) and direct one-on-one interviews. Contextual data analysis will adopt descriptive and narrative approaches to represent verbatim data collected.

Symposia

SP28_3

Title

Technical Challenges and Opportunities of Brain Data Governance in Africa

Authors

Damian Eke[1], Franco Pestilli[1], Amadi Ogonda Ihunwo[2]; Wogu Eberechi3; Amadi-Ihunwo, Uchenna[2].

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Abstract

Brain data governance includes different elements that include ethics, law, technology and people with lived experience. These critical elements are important in the development and implementation of data governance frameworks. In Africa, there are many technical or technology-related challenges and opportunities for brain data governance. This presentation will highlight the results of our research and make recommendations for key stakeholders including policy makers, academia, industry and citizens. Methods This research combined narrative literature review and an empirical qualitative survey to provide a comprehensive and contextually informed analysis of the technical elements of brain data governance in Africa. This methodological approach combines theoretical insights with real-world perspectives, offering a well-rounded understanding of the subject. Results The findings showed that lack of regulatory oversight on African brain data, lack of technical expertise to generate, process and share data, lack of data and insufficient technical infrastructure for brain data are some of the technical challenges to brain data governance in Africa. It also showed that there is a critical lack of technical expertise for brain data governance on the continent. But despite all these challenges, there are great opportunities including funding. DiscussionTherefore, there is a need for brain data awareness at all levels of education and research. A case must be made to researchers as individuals as well as government institutions to ensure early buy-in for sufficient generation, application and responsible sharing of brain data. Policy makers who may likely shape the emerging ecosystem of data nationalism or data protectionism need to be educated on the technical side of brain data to ensure that they understand implications of preventing data sharing. Rather the focus should be on developing mechanisms for responsible data governance.

Symposia

SP28_4

Title

Bridging the gap in data sharing: Developing International Data Governance Frameworks for Brain Health Research

Authors

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1-The University of Texas at Austin. 2-D'Or Institute for Research and Education (IDOR), Brazil. 3-The Federal University of Rio de Janeiro (UFRJ), Brazil. 4-University of the Witwatersrand, Johannesburg, South Africa 5-University of Nottingham, UK. 6-Zurich University of Applied Sciences, Switzerland. 7-University of Oxford, Oxford, UK

Abstract

As brain and mental health research generates increasingly large, multifunctional, multidimensional, and multi-jurisdictional datasets, data governance has become a critical component in ensuring the usability, integrity, and security of data. Governance not only safeguards against management errors, compliance issues, and ethical breaches but also facilitates the coordination necessary for international collaborations that span institutional regulations, national laws, and cultural norms. However, the current lack of an agreed-upon International Data Governance Framework (IDGF) poses significant challenges to researchers, institutions, and stakeholders from academia, industry, and policy domains, hindering the ability to address global challenges in neuroscience data sharing effectively. The BRIDGE (braindatagovernance.org) project, aims to address this critical gap by developing an IDGF that advances responsible international brain research and innovation. Stemming from early efforts at the internationalbraininitiaitve.org and supported by The Wellcome Trust, this initiative exemplifies a global and interdisciplinary approach to tackling the complexities of international data governance. A key goal of the BRIDGE project is the co-creation of governance processes, tools, and resources tailored to the needs of brain and mental health research. Central to this effort will be the development of a comprehensive handbook that compiles laws and regulations on datagovernance across high-, medium-, and low-income countries. This resource will include actionable methods for data sharing, guidelines for ethical practices, and a detailed collection of "do's and don'ts" for cross-border data management. By addressing these critical issues, the handbook will serve as a foundational resource for researchers, institutions, funders, and industry stakeholders, providing clarity and direction for planning and executing large-scale, international collaborations. Through the collective efforts of a consortium of 15 international organizations, the BRIDGE project aspires to establish the groundwork for a global data governance consortium. This effort is not only about creating practical tools and guidelines but also about fostering a culture of ethical, transparent, and equitable data practices across the neuroscience research community. By laying the foundation for an effective IDGF, the BRIDGE project seeks to unlock the potential of global data sharing, ultimately accelerating progress in brain and mental health research and innovation.

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The SONA 2025 Conference

Oral Presentations Abstracts

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Oral Presentations

Session 1 thematic:
Clinical Neurosciences and Epidemiology of Neurological and Psychiatric Disorders

Chair of the session: **Fatima Zahra Lamghari-Moubarrad**,
Cadi Ayyad University, Marrakech, Morocco

S1-0P1

Plasticity as a clinical marker: implications for resilience and vulnerability to psychopathology

Igor Branchi

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Abstract:

Plasticity is the ability to modify brain functioning and behavior. It is increasingly recognized as a key process in psychiatry and applied neuroscience because it is fundamental for the reorganization of the mental state and behavioral outcome during the transition from psychopathology to well-being. High plasticity has been shown to be associated with high susceptibility to contextual factors, e.g., quality of life, which ultimately drives plasticity outcome. Thus, plasticity is not beneficial per se, but its value depends on context, and resilience and vulnerability are not univocally associated to high or low plasticity. This implies that preventive and therapeutic strategies for psychiatric disorders, such as depression, should be tailored to both an individual's plasticity levels and their quality of life. Despite the existence of effective strategies to assess the quality of life, such as questionnaires, there is currently no standardized approach to measure plasticity levels. We have recently developed and validated a method for operationalizing plasticity using network theory. Specifically, we have exploited network analysis to demonstrate that plasticity? Defined as the susceptibility to modify depression scores? Can be measured at baseline by evaluating symptom network connectivity: the weaker the connectivity, the higher the plasticity, resulting in a greater modification in mood symptoms. We analyzed the STAR*D dataset and found that baseline connectivity strength was weaker in responder compared to non-responder patients. Moreover, connectivity strength was inversely correlated with improvement in depression score and susceptibility to change mood according to context. Overall, this operationalization of plasticity provides a mathematical tool to predict resilience, vulnerability and recovery, and to develop novel approaches for the prevention and treatment of major depressive disorder.

Oral Presentations

Session 1 thematic:
Clinical Neurosciences and Epidemiology of Neurological and Psychiatric Disorders

S1-0P2

Ocular microtremor in assessing the dynamics of the condition in schizophrenia

Irina Shoshina1; Sergey Lyapunov[2]; Alisa Kosikova[1]; Anna Fedorova[1]; Arslan Moritz[1]; Anton Torgovtsev[3]; Alexander Khanko[3]; Oleg Limankin[3]

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Abstract:

Introduction: Ocular micro tremor (OMT) controlling with nucleus of the reticular formation of the brain stem, has a frequency of about 87hz and an amplitude of 20-40 arcs/s. There is no information in the literature on the parameters of OMT in psychopathology. The purpose of the study was to assess the features of OMT in dynamic of state of patients in schizophrenia. Subjects and methods. The study involved 30 people without a history of neurological and psychiatric diseases; 37 patients with paranoid schizophrenia. OMT was recorded using high-speed video recording with an original optical setup.
Results: In schizophrenia the average OMT frequency in the subacute state was lower than in a stable mental state. The average values of the OMT frequency in a relatively stable state corresponded to the values of the healthy control. The data on the assessment of the OMT dynamics, taking into account the spectral approach, indicated a significantly higher frequency of OMT frequency falling into the range of 0-50hz in the subacute state, compared with a relatively stable state. Whereas the frequency of falling into the high-frequency range from 100 to 110hz in the subacute state was lower than in a relatively stable state. With relative stabilization of the state, the high-frequency component of the OMT becomes more pronounced, but does not reach normal values. The OMT amplitude in dynamics differed only in the low-frequency range of the OMT from 40 to 50hz and was lower at the first measurement in the subacute state, compared with the second measurement with relative stabilization of the state.
Conclusions: In schizophrenia in the subacute state, the OMT amplitude in the low-frequency range is significantly lower, compared with a more stable state. Thus, evidence was obtained of the informativeness of the OMT indicators for assessing the dynamics of the condition in schizophrenia.

Support by the Russian Science Foundation (No.24-25-00494).

Oral Presentations

Session 1 thematic:
Clinical Neurosciences and Epidemiology of Neurological and Psychiatric Disorders

S1-0P3

Clinical, perceptual, and neural features associated withhallucinations in clinical and nonclinical voicehearers

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Abstract:

Background: Auditory verbal hallucinations (AVH) are common in people with schizophrenia(PSZ) but progress in understanding the underlying mechanisms has been difficult. One major challenge is that (PSZ) endorse varying combinations of psychotic, negative, and disorganization symptoms making it problematic to isolate variance that is unique to hallucinations. Here we study nonclinical voice-hearers(NCVH), offering the potential to illuminate mechanisms specific to AVH, untainted by factors associated with disability and consequences of illness. Our aim is to determine what the similarities (and differences) in phenomenology of AVH in PSZ and NCVH suggest about underlying mechanisms.

Methods: We studied 76 PSZ, 51healthy volunteers(HCs) and 40 NCVH. None of the NCVH received a psychotic or current mood disorder diagnosis. Ratings included the BPRS, Chicago hallucination Assessment Tool, Peters Delusion Inventory(PDI). We also conducted behavioral experiments assaying the use of priors in perception, along with obtaining structural neuroimaging measures.

Findings: We show that AVH of PSZ and NCVH have similar sensory features (loudness, duration etc.) but PSZ experience less control over AVH and greater distress. In behavioral tasks, NCVH and PSZ show an over-reliance on priors relative to sensory inputs, consistent with predictive coding models. PSZ show greater cortical thinning and volume reduction relative to hCs than do NCVH, but the profile of group differences between NCVH and hCs resembles that seen in the contrast of PSZ and hCs.

Discussion: Our results suggest that NCVH share some, but not all, features associated with AVH in PSZ. By focusing on what is unique to PSZ relative to NCVH, we have the opportunity to address origins of AVH in PSZ, and isolate features associated with need for care.

Oral Presentations

Session 1 thematic:
Clinical Neurosciences and Epidemiology of Neurological and Psychiatric Disorders

S1-0P4

Relationship between thyroid dysfunction in a Moroccan population and behavioral status

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Multidisciplinary Laboratory of Research and Innovation (MLRI); Research Team: Technological Applications, Environmental Resources and health, Sultan Moulay Slimane University, Polydisciplinary Faculty Khouribga, Morocco.

Abstract:

Thyroid hormones are essential for every tissue in the body, including the central nervous system. Thyroid disorders have been associated with brain function including neurogenesis and myelination, and consequence on cognitive and behavioral functions were reported. Fatigue and depression are common symptoms in patients suffering from thyroid dysfunction. To evaluate thyroid functioning on behavioral performances, we performed a cross-sectional, either by self-administered patient survey or questionnaire assisted by health professionals within Hassan II provincial hospital in Khouribga province, Morocco. The primary outcome measures were the fatigue severity scale (FSS), the Patient Health Questionnaire-9 (PHQ-9), and the ascertain dementia 8 (AD8), to evaluate respectively fatigue scores, depression/anxiety symptoms, and the memory disabilities in these patients. Our case-control study concerns patients with thyroid dysfunction in our region with a total of 115 with 102 females (88%) and 13 males (12%) and age and gender controls. Our data demonstrate the existence of fatigue, depression and anxiety symptoms in patients with thyroid dysfunction, and showed a highly correlation between mood changes and fatigue scores (R2: 0, 24; p0, 0001). Particularly, fatigue was the hall mark symptom in the studied population as compared to controls. In patients with thyroid dysfunction, these behavioral alterations seem to be highly correlated with T4 plasma levels with fatigue scores (R2: 0, 33; p0.01) and T3 plasma levels particularly with anxiety and depressive scores (R2: 0, 4; p0.01). Our preliminary data provide evidence of altered behavioral components in patients with thyroid dysfunctions probably due to altered thyroid hormones. Further studies to verify such relationship and the mechanisms which trigger these changes in animal model of drug-induced thyroid dysfunction are warranted.

Key words: thyroid hormone, nervous system, fatigue, anxiety, memory

Oral Presentations

Session 2 thematic:
Ethics, Sleep, and Neuroscientific Insights from Animal and Computational Models

Chair of the session: **Rachida Roky**
Hassan II University, Casablanca, Morocco

S2-OP1

Brain and environment: can neuroethics meet the challenge of the future?

Tom Buller,
Department of philosophy, Illinois State university

Abstract:

Brain and environment: can neuroethics meet the challenge of the future?

The field of neuroethics has examined the ethical implications of advances in neuroscience, and the novel ways in which the brain can be monitored and manipulated through neuro-technology and neuropharmacology, for example, through "brain-reading", brain-computer interfaces, and cognitive enhancement.

This focus on emergent neuro-technologies, and the underlying materialist theory of mind has led to critics accusing neuroethics of being speculative, futuristic, and exclusive, and thereby ignoring many of the neurological conditions and ethical and practical issues that are of critical importance to the Global South. Furthermore, neuroethics has also been too narrow in emphasizing individual agency and identity and ignoring the relevance of culture, family, environment, and religion.

The challenge facing neuroethics is to make the field "braver" and to matter to those outside the Global North. This means refocusing our attention towards practical concerns such as the high incidence of traumatic brain injury and neuro-infectious diseases in Africa. It also means working to make the distribution of resources and collaborations to support research and research activity more inclusive.

A deeper challenge is for neuroethics to revise its materialist, monistic, and secular view of mind. This would mean moving away from understanding clinical depression, for example, in terms of neurotransmitters, and embracing a more pluralistic perspective that includes greater awareness of communal, religious, and spiritual factors. This, however, will require neuroethics to rebuild its foundations.

Oral Presentations

S2-OP2

Heartificial intelligence: exploring empathy in language models'

Victoria Williams, Benjamin Rossman
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Abstract:

Background and objectives: language models are becoming increasingly important in our daily lives as virtual assistants and digital companions. For effective human-machine interactions, language models need both cognitive empathy (to understand others? thoughts and emotions) and affective empathy (to share others? feelings) but their capabilities have been understudied. We thus examined cognitive and affective empathy in small (SLMs) and large language models (LLMs), comparing their performance to human data from diverse cultures, education and age groups.

Methods: we employed established psychological assessments, commonly tested inhuman, to measure cognitive empathy (e.g., the Strange Stories Revised [SSR] test; the Situational Test of Emotional Understanding [STEU]; Seeing Emotions in the Eyes (SEE-48) and affective empathy (the Toronto Empathy Questionnaire [TEQ]) in SLMs and LLMs. The language models varied in size to evaluate how model size influences empathy performance.

Results: the STEU, STEU-B, and SSR tests showed that SLMs (Tiny Llama 1.1B and Phi-2) struggled with perspective-taking and understanding complex social scenarios, while mid-tier models (Mistral-7B) performed moderately, approaching human-level performance. Advanced LLMs (GPT-4o, Gemini 1.5 Pro, and Claude 3.5 Sonnet) outperformed graduate students and employees from culturally diverse populations. In emotion recognition tasks (SEE-48), GPT-4o excelled at identifying basic emotions, but humans, particularly Master's students, were better at detecting complex negative emotions. For affective empathy, language models scored lower than humans on the TEQ.

Discussion: by incorporating a range of experimental tests in psychology, we provide a more nuanced understanding of how empathy in language models compares to humans? empathy from diverse cultures, education and age groups. This novel approach to assessing machine intelligence uncovers key similarities and differences between SLMs, LLMs and humans that might have remained hidden. Our study reveals that LLMs rival humans in cognitive empathy tasks that require perspective-taking and theory of mind, but struggle with affective empathy, i.e., sharing or experiencing emotions themselves. This highlights gaps in empathy capabilities between humans and AI and proposes a unique framework for future research in machine intelligence and human-AI interactions.

Keywords: cognitive and affective empathy, language models, machine-human interactions.

Oral Presentations

S2-OP3

Sleep, Chronotype, and Stress Among Students: A Sex-Based Analysis

Rachida Roky, Asma Laafram

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Abstract:

Background and Objectives: Sleep quality, chronotype preferences, and stress levels significantly impact students' well-being and academic performance. This study aimed to explore sex-based differences in these parameters among university students at the Faculty of Sciences Aïn Chock, Casablanca, during the autumn semester of 2024. The primary objective was to assess whether men and women exhibit significant variations in sleep duration, sleep-hygiene, chronotype, and stress levels.

Methods: A total of 373 participants (189 women and 184 men), with a mean age of 19.86 ± 2.14 years, were included in this cross-sectional descriptive study. Informed written consent was obtained from all participants. Data collection utilized three validated tools administered anonymously: The Perceived Stress Scale (PSS), the Sleep-hygiene Index (SHI), and the reduced Morningness-Eveningness Questionnaire (rMEQ). Demographic characteristics were also gathered. These self-reported questionnaires were administered face-to-face. Statistical analyses were performed to identify mean differences and significant trends between men and women.

Results: Significant sex-based differences were observed across multiple parameters. Women demonstrated poorer sleep-hygiene scores (mean SHI: 37.7 vs. 36.20, p= 0.0483), with only 6% of women reporting good sleep-hygiene compared to 12% of men. Women also reported higher perceived stress levels (mean PSS: 23.09 vs. 18.62, p= 0.0001). However, no significant differences were observed in chronotype preferences, as both genders displayed an eveningness preference (mean MEQr: 14.44 for women vs. 14.42 for men). Sleep duration and preferred sleep duration were longer in women than in men (mean: 7h17min, 8h47min vs. 6h54min, 7h59min; p= 0.0231, respectively).

Discussion and Conclusion: These findings suggest that female students experience higher stress levels and poorer sleep-hygiene than their male counterparts, possibly due to gender-specific academic and social pressures. Despite these differences, both genders showed a shared evening chronotype preference. The results underline the importance of implementing targeted interventions, such as stress management workshops and sleep-hygiene education, to promote students' mental and physical health.

Keywords: sex Differences, Sleep-hygiene index, Perceived Stress scale, university students

Oral Presentations

S2-OP4

Circadian Regulation of Rumination in Desert Black Goats (*Caprahircus*): Evidence for an Endogenous Rhythm

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3: Institute of Cellular and Integrative Neurosciences, CNRS and University of Strasbourg, Strasbourg, France

Abstract:

Rumination exhibits a clear daily rhythm in desert black goats, a species well adapted toharsh environmental conditions. This study aimed to further investigate the endogenous nature of the rumination rhythm, building upon previous findings suggesting that this rhythm is not solely driven by feeding behavior. The experiment was conducted under constant temperature (23°C) and total darkness (DD) conditions, across four stages: feeding once daily at 10:00 (stage 1), feeding four times per day (stage 2), ad libitum feeding (Stage 3), and total fasting for four days (Stage 4). The results showed that in Stage 1, rumination exhibited a distinct daily rhythm with a period of 24.0h. In Stage 2, the rhythm has changed, displaying four peaks per 24h, with a period ranging from 23.5 to 24.0h. During Stage 3, a free running rhythm of rumination was observed with a daily progressive delay and a period of 24.7h, indicating an endogenous origin of this rhythm. In Stage 4, fasting did not result in the complete elimination of ruminating behavior but has induced a substantial decrease in its total duration. This study demonstrates that, unlike previously thought, the rhythm of rumination is not driven by food and instead it is endogenously generated by the central circadian clock.

Key-Words: Desert black goat; Rumination rhythm; Circadian clock; Endogenous rhythm

Oral Presentations

S2-OP5

Entrainment of the master circadian clock by the timing of food distribution in goats (Caprahircus)

M'HANI Mohammed El Mehdi[1], BENIAICH Younes[1], FARSI Hicham[1], PIRO Mohammed[2], ACHAABAN Mohamed Rachid[1], DADEN Réda[1], RAVEROT Véronique[3], CHALLET Etienne[4], PEVET Paul[4] and EL ALLALI Khalid[1]

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3: Institute of Cellular and Integrative Neurosciences, CNRS.

Abstract:

In desert areas, mammals are subjected to extreme environmental conditions, including heat stress and limited food resources. Ambient temperature (Ta) cycles were shown to entrain the circadian rhythm in two desert mammals: the dromedary camel and the goat. In the present study, we assumed that in the desert goat, an additional non-photic factor, the timing of food availability, may have a comparable synchronizing effect on the central clock. The effect of the phase shift in the timing of food distribution on three outputs of the master circadian clock, the rhythms of body temperature (Tb), locomotor activity (LA) and plasma melatonin secretion (Mel), has been studied in ten bucks maintained under continuous total darkness (DD) and constant ambient temperature (CTa). First, food was given once daily at 10h am (stage 1*);* then, it was delayed by 12hours (stage 2), with feeding during the subjective night at 10h pm, and finally, in "stage 3", the previous conditions of "stage 1" were restored, with daily single food distribution at 10h am. The obtained results demonstrated that under the different stages of the experiment, the Tb and LA had a clear rhythmic expression, with a period of exactly 24.0hours. The inversion of the moment of food distribution resulted in a phase shift of the acrophases of Tb and LA rhythms of 12.47 and 11.55hours respectively. To make sure that the induced shifting of LA and Tb by the timing of food distribution is a real entrainment of the central circadian clock and is not a result of a masking effect, the melatonin secretion, the most robust circadian output of the clock, was then monitored. The corresponding results confirmed Mel rhythm entrainment, inducing either 2.02 to 11.07 hours-phase delay of the rhythm in six animals or 6.2 to 8.9hours-phase advance in four animals. Changing the time of feeding affects the Tb and LA rhythms as well as melatonin secretion rhythm. Hence, timing of food distribution is able to entrain the master circadian clock of the desert goat in the absence of the LD and Ta cycles.

Keywords: Food, circadian

Oral Presentations

Session 3 thematic:
Neuroprotection and Neurobiological Disruption in Neuro-degenerative Diseases

Chair of the session: **Samir Ahboucha**,
FPK, USMS, Khouribga, Morocco

S3-OP1

Protective Effects of Lutein-20 Against Cadmium-Induced Oxidative Stress and Neuroinflammation in the Prefrontal Cortex and hippocampus of Adult Male Wistar Rats

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Abstract:

Background: Environmental pollutants, such as cadmium (Cd), pose significant threats to brain health because of its vulnerability and sensitivity to oxidative stress resulting in neurodegenerative diseases such as Alzheimer's disease. There is an interplay between the brain and the environment providing plant based dietary compounds with strong antioxidant activity averting environmental toxins mediated oxidative threats to brain health. Lutein-20 is a plant based dietary carotenoid from dark green leafy vegetables with potent antioxidant and anti-inflammatory properties.
Objectives: To investigate the interplay between Lutein-20 on cadmium-induced neurotoxicity in the prefrontal cortex and hippocampus by assessing the antioxidant and inflammatory indexes., in addition to histological changes in the prefrontal cortex (PFC) and hippocampus (HP).
Methodology: Twenty (20) adult male Wistar rats were grouped into four (n=5): The control, Lutein-20 only treated group receive 20 mg/kg of Lutein-20 for 10 days. Cadmium-exposed group received 2 mg/kg of cadmium for 5 days. The fourth group was pre-treated with 2 mg/kg Cadmium Chloride for five days, then 20 mg/kg of Lutein-20 for 10 days. Administration was done orally. Brain samples were fixed in 10% formol calcium and processed for haematoxylin and Eosin staining while enzyme assayed lipid peroxidation enyme-malondialdehyde (MDA), inflammatory marker total protein, pro-inflammatory cytokines marker- tumour necrosis factor alpha (TNF alpha); antioxidant status of superoxidase dismutase (SOD), Catalase (CAT) was undertaken using spectrophotometric method. Statistical analysis was undertaken using one-way ANOVA with significance set at p0.05.
Results: Our results demonstrated that cadmium significantly elevated markers of oxidative stress and pro-inflammatory cytokines (MDA and TNF-α), declined antioxidant enzyme activities (SOD, CAT, GSH), leading to neurodegeneration in the prefrontal cortex and hippocampus. however, administration of Lutein-20 markedly reduced oxidative damage and suppressed neuroinflammatory responses through elevation of antioxidant enzyme activities resulting in a reduced disruption of the neuropil in the PFC and hP.
Discussion: These findings suggest that Lutein-20has a protective role against cadmium-induced neuroinflammation and neurodegeneration, hence underscores its potential as a therapeutic agent for mitigating environmental neurotoxicity and potentials in future interventions for neurodegenerative conditions linked to environmental pollutants.

Keywords: Lutein-20, Cadmium Chloride Neurodegeneration, Environmental Pollutants, Neurotoxicity, pro-inflammatory cytokines.

Oral Presentations

S3-OP2

Parkinson's Disease in Mauritania: Epidemiological Profile and contribution to the genetic analysis (the G2019S mutation).

Elhafedh El Mouhab, Sidati Mohamed Lemine[1-2], Fatimetou Veten[1], Mohamed Yehdih Amar Abdi[1], Maimouna Ishagh[1-2], Mohamed Sideleoua, Samy Daddah, Mohamed Vall Ould Kebir[2], Ahmed Houmeida[1]

Abstract:

Background: Parkinson's disease (PD) is the second most common neurodegenerative disorder after Alzheimer's disease, characterized by the degeneration of dopaminergic neurons and the presence of Lewy bodies. In Mauritania, the growing elderly population has led to an increase in PD cases. This study aims to assess the epidemiological profile of PD in Mauritania and prepare samples for future genetic analysis, including the investigation of the G2019S mutation in the LRRK2 gene, which is prevalent in North African populations.

Materials/Methods: Epidemiological data were collected from Parkinson's patients in Mauritania, focusing on age, sex, and ethnic distribution. Blood samples were obtained, with a particular focus on patients with early onset or a family history of the disease. DNA extraction was performed on the collected samples, and both quantitative and qualitative assessments were conducted using spectrophotometry and gel electrophoresis to ensure the samples' suitability for subsequent genetic studies.

Results: The average age of PD onset in Mauritania was 63.35 years, with a higher prevalence among men (sex ratio of 1.63). Ethnic distribution revealed that 82% of the patients were Moors, while 18% were Black Africans. Approximately 42% of cases exhibited early onset of the disease. DNA extraction yielded sufficient and high-quality samples, ready for further genetic analysis, including PCR and sequencing.

Conclusion: The study provides a clear epidemiological profile of PD in Mauritania, with a male predominance and a significant representation of the Moor population. The successful extraction of high-quality DNA samples marks a crucial step toward future genetic studies, which will explore the role of genetic mutations such as G2019S in the onset and progression of Parkinson's disease in this population.

Oral Presentations

S3-OP3

In Silico Modeling of LRRK2 G2019S Protein-Protein Interactions in Parkinson's disease: A Machine Learning Approach

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Abstract:

Parkinson's disease (PD) is a debilitating neurodegenerative disorder affecting millions globally. Among the genetic factors linked to PD, the LRRK2 G2019S mutation stands out due to its high prevalence in certain populations, including Moroccans. This mutation disrupts various cellular processes, yet its precise molecular role in neurodegeneration remains unclear.

The primary aim of this project is to elucidate how the LRRK2 G2019S mutation contributes to PD through its protein-protein interactions (PPIs) across different subcellular localizations. Using machine learning techniques, we predicted PPIs of LRRK2 in PD. Our computational pipeline identified 716 LRRK2 interactors, offering unprecedented insights into the complex interaction networks linked to PD. Notably, 49% of these interactions are novel, shedding new light on its role in PD. These PPIs were further mapped to LRRK2 G2019S expression profiles, enhancing the biological relevance of these interactions to the mutation's role in disease mechanisms.

These computational analyses enabled us to uncover novel interactions that highlight key proteins potentially involved in PD pathology. The PPI network generated not only advances our understanding of the molecular mechanisms driving PD onset and progression but also provides a strong foundation for identifying potential therapeutic targets. This research goes beyond neurodegeneration, contributing to broader insights into protein dynamics within subcellular processes, with implications for developing novel therapeutic strategies for PD and related disorders.

Keywords: LRRK2 G2019S Mutation, Protein-Protein Interactions (PPIs), Parkinson's Disease (PD), *In Silico* Modeling, Machine Learning.

Oral Presentations

S3-OP4

Neonatal microglia enhance aged OPC differentiation via autophagy, and metformin restores differentiation capacity in aged opcs: insights from a mouse co-culture model

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Abstract:

Aging profoundly affects neurodegenerative disease mechanisms by altering essential cellular processes such as myelination and remyelination. Microglia, the immune cells of the central nervous system (CNS), regulate oligodendrocyte precursor cell (OPC) differentiation and myelin repair. however, aging disrupts microglial functions, including autophagic activity, which is critical for maintaining cellular homeostasis and supporting OPC maturation. This study aimed to investigate (1) the impact of aging microglia on OPC survival and differentiation in a co-culture model and (2) the potential of metformin, a metabolic modulator, to enhance the differentiation capacity of aged OPCs. Microglia and OPCs were isolated from neonatal (postnatal day 6, P6) and aged (13-month-old) CD1 mice. Co-culture experiments were performed with all combinations of neonatal and aged cells. OPC differentiation was assessed using immunocytochemistry for myelin basic protein (MBP) and Olig2 markers. Microglial autophagic activity was evaluated through LC3-II Western blot analysis. Aged microglia exhibited impaired autophagic activity, demonstrated by the absence of LC3-II expression, which correlated with reduced support for OPC differentiation. In contrast, neonatal microglia retained autophagic functionality, as indicated by LC3-II expression, and significantly enhanced the survival and differentiation of aged OPCs. Metformin treatment substantially improved the differentiation potential of aged OPCs, as shown by increased MBP+/Olig2+ cells, but had no notable effect on neonatal OPCs. The findings highlight that impaired autophagy in aging microglia diminishes their ability to support OPC differentiation, representing a significant barrier to effective myelination. Metformin's capacity to restore the differentiation potential of aged OPCs underscores its therapeutic potential in addressing age-related deficits in myelin repair. Interventions targeting microglial autophagy and metabolic modulation offer promising strategies to enhance remyelination in aging and neurodegenerative diseases.

Keywords: Aging, differentiation, metformin, microglia, oligodendrocyte precursor cells

Oral Presentations

S3-OP5

Systematic review of the effect of environmental enrichment on brain-derived neurotrophic factor (BDNF) activity for mitigating behavioral manifestations in an autism spectrum disorder (ASD) model.

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Abstract:

Background: Brain-derived neurotrophic factor (BDNF) plays a crucial role in nervous system development, and dysregulation in its function is implicated in neurodevelopmental disorders, notably Autism Spectrum Disorder (ASD). Currently, no definitive treatment exists for addressing the core symptoms of ASD, save for behavioral interventions that mitigate its manifestations. Previous animal studies suggest BDNF as a mediator through which Environmental Enrichment (EE) can enhance memory and learning. This systematic review aimed to examine BDNF as a potential target through which EE may ameliorate core symptoms of ASD.

Methods: A systematic search was conducted in PubMed, Web of Science, and Scopus in April 2024. Inclusion criteria were primary animal intervention studies investigating the effects of EE on BDNF in ASD models. The review followed the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) statement and checklist, and the SYRCLE Risk of Bias tool was used for quality assessment. Data on molecular BDNF expression and behavioral outcomes were extracted and synthesized. The systematic review protocol was registered in PROSPERO (registration number: CRD42024517384). Results: Out of 1588 unique records, 7 studies met the inclusion criteria, examining various ASD models, including VPA exposure, maternal separation, Caps2, Fmr1, MeCP2 knockouts, and BTBR mice, across different rodent strains (e.g., Wistar rats, FVB, ICR, and CD1 mice). EE protocols varied by starting age, duration, and components. Of the seven studies, six reported a positive effect of EE on BDNF levels in at least one brain structure, in addition EE ameliorated ASD manifestations. However, the findings varied across studies. Risk of bias assessment revealed that all seven studies had a moderate risk of bias.

Conclusions: This systematic review provides evidence that EE can positively influence BDNF levels and behavioral outcomes in ASD animal models. However, the variability observed across studies highlights the need for further research and a standardized EE protocol to achieve comparable results.

Keywords: Autism Spectrum Disorder, Brain-derived neurotrophic factor, Animal model, Environmental enrichment

Oral Presentations

S3-OP6

Longitudinal brain ageing after stroke: a marker for neurodegeneration and its relevance for motor outcome

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Abstract:

Brain age, as distinct from chronological age, may increase post-stroke and relate to poor sensorimotor outcomes. However, longitudinal studies investigating brain age post-stroke are lacking. We hypothesized that brain age would increase over the first month's post-stroke, reflecting secondary neurodegeneration, and that this would relate to upper limb sensorimotor outcome.

We retrospectively analyzed T1-weighted MRI at two time-points: at baseline (3 weeks) and follow up (3-7 months) post-stroke and clinical sensorimotor measures at follow up. The primary lesion site was masked using enantiomorphic normalization on MRI scans by replacing it with healthy tissue prior to brain age estimation. Brain age was computed and the difference to chronological age was calculated as brain age gap (BAG). We then calculated BAG change as the difference in BAG over the two time points. Voxel-based morphometry was used to investigate brain structures undergoing secondary post-stroke degeneration. Voxel-based lesion symptom mapping was used to identify lesion location related to accelerated brain age. Linear mixed effect regression analysis was used to study the relationship between BAG change and sensorimotor outcome.

114 patients with first-ever stroke and arm/hand hemiparesis were pooled from three studies. There was significant difference between BAG at baseline and follow-up (mean difference= 3.62 years; t= -7.31; P0.001). Patients with increased BAG change had reduced grey and white matter volume distant to the stroke lesion, specifically in the medio-dorsal nucleus of the thalamus and internal capsule respectively. BAG change, but not chronological age, was associated with motor outcomes in the sub-acute to chronic phase, as expressed by FMA_UE ((t=-6.16, SE=2.43, t=-2.54, P=0.01)., maximum grip strength (t=-0.12, SE=0.04, t=-3.24, P=0.002), and dexterity assessment t=-0.08, SE=0.04, t=-2.29, P=0.03).

We demonstrate that there is increased brain ageing within the first few months after stroke. This secondary neurodegeneration negatively related to motor outcome. Brain age may be a valid whole-brain probe of individual secondary post-stroke degeneration, relevant for predicting recovery and identifying targets of neural plasticity.

Oral Presentations

S3-OP7

Investigating neuronal cell cycle re-entry in C9ORF72 frontotemporal dementia

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Abstract:

Background & Objectives: Most cells grow and divide in a continuous cycle of 4 stages: cell division by mitosis; a growth stage; DNA replication; and a second growth stage. Mature adult neurons stop dividing and permanently exit the cell cycle. However, new evidence from Alzheimer's disease (AD) post-mortem brain tissue and cell models suggests that neurons re-enter the cell cycle in response to damage in disease, and that this response contributes to cell death. Little is understood about neuronal cell cycle re-entry, and it is unknown whether this phenomenon occurs in other neurodegenerative diseases.

We explore for the first time whether cell cycle re-entry occurs in neurons in frontotemporal dementia (FTD) caused by a repeat expansion mutation in the C9ORF72 gene, using post-mortem brain tissue and induced pluripotent stem cell (iPSC) derived neurons.

Methods: Proteins associated with active cell cycle stages (CyclinB1, CyclinD1, PCNA, and pRb) were probed via immunofluorescence in post-mortem frontal cortex tissue, in conjunction with immunofluorescence targeting pathological protein aggregates associated with FTD. Brains of neurologically healthy controls and individuals with C9ORF72 FTD or AD were examined.

Wildtype iPSC-derived cortical neurons were treated with poly-proline-arginine (poly-PR), one of several toxic proteins produced by the C9ORF72 mutation. The same cell cycle proteins were visualized via immunofluorescence.

Results: We replicate independent observations of increased proteins associated with cell cycle activity in AD post mortem brain tissue. We also observe the same phenomenon for the first time in C9ORF72 FTD post-mortem brain tissue.

In wildtype iPSC-derived cortical neurons, we find that poly-PR protein toxicity alone is not sufficient to trigger cell cycle re-entry.

Discussion: This study demonstrates that neuronal cell cycle re-entry occurs in degenerating neurons in C9ORF72 FTD and is likely to be triggered by a combination of multiple pathological components derived from the mutation. We identify an under-explored area of neuronal function which appears to impact cell survival in multiple neurodegenerative diseases. Furthermore, many drugs targeting cell cycle regulation already exist in cancer medicine, highlighting future exploration of drug re-purposing as a possible cost-effective therapeutic strategy for preserving neurons in FTD by targeting neuronal cell cycle re-entry.

Oral Presentations

S3-OP8

Neurosteroids and Brain Injuries: Are they protective or deleterious?

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Abstract:

Neurosteroids (NS) are found in the central nervous system (CNS) at high levels long after ablation of gonads and adrenals, thus leading to the concept of novo brain synthesis of steroids. Since the 1980s, this concept has been proposed and then established based mainly on the evidence of expression of steroidogenic enzymes in the brain. NS enzymes, after translocation of cholesterol within the mitochondria by the translocator protein (TSPO), lead to the synthesis of the parent NS pregnenolone and then a variety of NS. Among NS targets are membrane receptors with an impact on the excitability of neurons known as the "non-genomic effect". However, like the classical steroids, these neuroactive compounds were also shown to affect expression of cell components "genomic effect", which triggers deep changes in the morphology, size and number, as well as the function of neurons and glial cells. There is substantial evidence to suggest that NS changes occur in most brain injuries, and that NS seems to contribute to neuroprotection. However, in some pathologies NS may induce deleterious effects. In fact, brain injuries seem to induce activation of the TSPO notably within glial cells. Both in vitro and in vivo studies suggest the involvement of TSPO changes in brain injuries with subsequent production of NS that trigger beneficial effect by reducing excito-toxicity, neurons loss, and neuro-inflammation or by promoting brain repair. Deleterious effects could be due to neurotransmission exaggerating effects and to glial and neuronal effects components with consequences on cell swelling. The presentation will shed light on the effects of steroids on brain injuries, with focus on neurodegenerative and metabolic diseases including Traumatic brain injury, Spinal cord and nerve injuries, ischemia, stroke, and hepatic encephalopathy.

Oral Presentations

Session 4 thematic:
Neuropharmacology and Traditional Ethnobotanical Agents in Modulating Cognitive and Behavioral Disorders

Chair of the session: **Nikolaos Pitsikas,**
University of Thessaly, Larissa, Greece

S4-OP1

Manipulating endogenous nicotinic receptors and associated behaviors with photoactivatable agonists

Nicolas Guyon

ESPCI Paris, PSL University, France

Abstract:

Nicotinic acetylcholine receptors (nAChRs) are ligand-gated ion channels activated by acetylcholine, a neurotransmitter essential for processes like learning, memory, attention, and pain regulation, as well as by nicotine. These receptors are widely distributed throughout the body and across various brain regions. However, distinguishing the specific effects of acetylcholine and nicotine in different parts of the brain remains a significant challenge. We have developed two novel photoactivatable agonists for nicotinic acetylcholine receptors: NV-epibatidine and NV-ABT594. These "caged" compounds can be systemically administered to mice in their inactive forms and subsequently activated with high spatial and temporal precision in specific brain regions using implanted optical fibers. Photoactivation in the ventral tegmental area (VTA) a midbrain dopaminergic nucleus involved in reward-related and motivational behaviors, caused a sharp increase in extracellular dopamine concentration in the nucleus accumbens (NAc). Additionally, activation in the periaqueductal gray (PAG), a primary center for descending pain modulation, led to reduced mechanical pain sensitivity. Importantly, exposure to light alone did not produce these effects, confirming the specificity of the caged compounds. These findings demonstrate that our caged drugs can effectively cross the blood-brain barrier, and that targeted photoactivation allows precise modulation of endogenous nAChR activity in brain circuits, affecting behaviors related to reward-seeking and pain. The methodologies developed in this study could be adapted to other drug candidates and neurotransmitter receptors, holding promise not only for advancing our understanding of nAChR physiology in the mouse brain but also for creating targeted therapies with fewer side effects for human applications.

Keywords: Nicotine, Photopharmacology, Dopamine, Reinforcement, Pain

Oral Presentations

S4-OP2

The nitric oxide synthase inhibitor 7-nitroindazole counteract social withdrawal and cognition deficits induced by blockade of the NMDA receptor in the rat.

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Abstract:

Background and objectives: Nitric oxide (NO), an intra- and inter-cellular messenger in the brain, is involved in the pathogenesis of schizophrenia, so excessive NO production might contribute to the pathology. This, implies that it might be useful to reduce nitrergic activity, so molecules aiming to decrease NO production such as NO synthase (NOS) inhibitors might be candidates. The aim of the present study was to detect the ability of the NOS inhibitor 7-nitroindazole (7-NI) to reduce schizophrenia-like impairments produced by the blockade of the N-Methyl-D-Aspartate (NMDA) receptor in rats.

Methods: To this end, the social interaction test (SIT), the object recognition test (ORT), the object location test (OLT) and the step-through passive avoidance test (STPAT) were used.

Results: 7-NI counteracted ketamine-induced social withdrawal evidenced in the SIT, a procedure resembling negative symptoms of schizophrenia in rodents. Further, 7-NI attenuated cognitive deficits induced either by ketamine or MK-801 in the ORT, OLT and STPAT. Finally, the joint treatment of sub-effective doses of 7-NI with those of the atypical neuroleptics clozapine and risperidone counteracted recognition memory deficits caused by ketamine.

Discussion: The current findings suggest that 7-NI is sensitive to glutamate hypo-function since it attenuated behavioral impairments in animal models mimicking the negative symptoms and cognitive deficits of schizophrenia. Additionally, the present results support the potential of 7-NI as an adjunctive drug for the therapy of schizophrenia.

Keywords: Schizophrenia, nitric oxide, 7-nitrondazole, ketamine, rat.

Oral Presentations

S4-OP3

A Novel Benzodiazepine Derivative: Exploring Anxiolytic-like effect and potential side effect

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Abstract:

Benzodiazepines (BZDs) are widely prescribed for the treatment of conditions such as anxiety, depression, insomnia, and seizures. However, their use is associated with side effects such as dependence, withdrawal symptoms, cognitive and motor impairments, which limit their therapeutic applications. The aim of the current study was to investigate the potential in vivo anxiolytic effect of a novel semi-synthetic benzodiazepine derivative. Therefore, we explored the pharmacological profile of this new molecule through various neurobehavioral experiments in mice, including open field, elevated plus maze, dark light box, social interaction, hole board, and marble burying tests for anxiety-like behaviors. We also assessed motor function and coordination using open field, static bars, and rotaroad tests, as well as short working memory via Y-maze, a novel recognition object. Our results showed that our moleculehas a strong anxiolytic effect typified by a significant reduction in anxiety-like behaviors. While, no signs of motor dysfunction, sedation, or short-term memory were observed. Hence, our benzodiazepine derivative administration in mice demonstrated anxiolytic effects. These findings could suggest that our molecule can be effective in reducing anxiety without alteration in motor skills, supporting more investigation for further therapeutic development.
Keywords: BZD, BZD-derivative, Adverse effects, Anxiolytic effect.

Oral Presentations

S4-OP4

Bioassay-guided Identification of Potential Alzheimer's Disease Therapeutic Agents from Kaempferol-Enriched Fraction of *Aframomum melegueta* Seeds using in Vitro and Chemoinformatics Approaches

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2: Federal University of Allied Health Sciences Enugu, Nigeria

Abstract:

Background and objectives: Alzheimer's disease (AD) has become a major public health concern and the fifth major cause of death among the aging population globally.

Methods: In this study, the total phenols and flavonoids contents (TPC and TFC) and in vitro antioxidant actions of the methanol extract and the various fractions of *Aframomum melegueta* were evaluated using 2, 2-diphenyl-2-picrylhydrazyl (DPPH) radical scavenging activity, nitric oxide scavenging activity (NO), lipid peroxidation (TBARS) activity and ferric reducing power assay (FRAP). Furthermore, acetylcholinesterase (AChE) and butyryl-cholinesterase (BuChE) inhibitory activities of the two most potent fractions were investigated, and the phytochemicals identified in the ethyl acetate fraction, which had the best antioxidant and cholinesterase inhibitory effects were subjected to chemoinformatics studies.

Results and Discussion: The extract and its fraction had high amounts of TPC and TFC. The ethyl acetate fraction exerted the best DPPH, NO, TBARS, and FRAP inhibition with IC50 values of 5.06, 6.58, 2.12, and 88.73µg/mL, respectively. Interestingly, n-hexane and ethyl acetate fractions inhibited AChE (IC5016.83 and 11.67µg/mL) and BuChE (IC50 7.54 and5.21
µg/mL) enzymatic activities more than the standard inhibitor, rivastigmine which had 11.99 and 11.40µg/mL IC50 values, respectively. A total of 18 compounds were identified, and kaempferol was the major component, with 40.01µg/g (30%). More strikingly, the top-scoring compounds (catechin, and kaempferol) exhibited good binding affinity, and interacted favorably with amino acids residues around and within the active sites of AChE and BuChE and also obeyed drug-likeness rules, and did not show a tendency towards toxicity when placed side by side with rivastigmine which is immunogenic. Thus, A. melegueta seeds contain safe bioactive chemicals, which could be a veritable remedy for managing Alzheimer's and other neurodegenerative diseases.

Keywords: *Aframomum melegueta*, acetylcholinesterase, antioxidants, butyryl-cholinesterase, drug-likeness, antioxidants.

Oral Presentations

S4-OP5

Evaluating the Potential of Stigmasterol as a Scaffold for Novel Antidepressants

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Abstract:

Depression affects over 9% of the total African population. Despite strong evidence of actions to lessen the global burden of depression, no significant decline in global prevalence has been recorded since 1990. The complexities of depression and inadequacies of current antidepressants requires the continued search for alternative treatment options. The unsaturated phytosterol, stigmasterol, a tetracyclic triterpene, is one of the most prevalent plant sterols andhas been found to possess acute antidepressant-like actions in preclinical studies. We evaluated the antidepressant potential of stigmasterol (STIG) in chronic models of depression and also assessed mechanisms that may be involved in its actions. We used the corticosteroid-induced depression (CID) and open space swim test (OSST) to model chronic depression-like behaviors and delineate the time-course of antidepressant-like effect of STIG at doses 1, 3, 10, 30 and 100 mg/kg in mice. To elucidate involvement of monoaminergic mechanism(s) in STIG's action, mice were pretreated with selective inhibitors of monoamine synthesis and storage (p-chlorophenylalanine (pCPA), alpha methyl para tyrosine (AMPT) and reserpine) after which the antidepressant-like effects of STIG was re-evaluated in the forced swim test (FST). Additionally, whole brain antioxidant levels and hippocampal BDNF levels were assayed while hippocampal neuronal density was determined by crystal violet staining of the hippocampus. STIG demonstrated significant antidepressant-like effects similar to fluoxetine and onset of antidepressant action was slightly faster than in fluoxetine-treated mice. This antidepressant action was significantly influenced by serotonin neurotransmission. Antidepressant-like activity was also observed in CID. STIG additionally, elevated brain glutathione levels and suppressed catalase enzyme activity. It further reversed depression-induced low neuronal density and increased hippocampal BDNF levels. Overall, we confirm STIG as a good scaffold for novel antidepressants discovery.

Keywords
Mood disorders, Neuropsychiatry, Drug discovery, Neurotransmitters

Oral Presentations

S4-OP6

Neuroprotective effects of *Schinus terebinthifolius* Raddi on streptozotocin-induced Alzheimer's disease-like symptoms in rats

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Abstract:

Streptozotocin (STZ) is well recognized for its ability to induce Alzheimer's disease (AD) models in rodents through direct administration into the brain. This administration leads to changes in brain function, manifesting symptoms characteristic of AD, such as metabolic disturbances and cognitive deficits. *Schinus terebinthifolius* Raddi (ST) is known for its abundance of secondary metabolites, including phenolic compounds and flavonoids, which possess antioxidant and anti-inflammatory properties. This study aimed to evaluate the neuroprotective effects of the ethanolic extract of ST fruits in mitigating key features of AD induced by STZ. Specifically, this investigation sought to examine the anti-amnesic, antioxidant, and cholinergic regulatory properties of ST.

24 adult Wistar rats were divided into three groups (n= 8). The control group received an intra-cerebroventricular (ICV) injection of sterile saline. The disease group was administered STZ (3mg/kg) using the same ICV method. The treated group received STZ and a daily oral dose of the ethanolic extract of ST (2.5mg/kg) for two months. At the end of the experiment, all rats were assessed for memory deficits and neurochemical changes.

Our results demonstrated that the extract significantly enhanced memory in the novel object recognition test, as indicated by an increase in the recognition index for both short-term (p0.05) and long-term memory (p0.01). Additionally, in the Morris water maze test, time was increased in the correct quadrant (p0.05) compared to the disease group. Furthermore, ST significantly elevated acetylcholine levels (p0.001) while concurrently reducing acetylcholinesterase activity (p0.001) in the hippocampus. It also led to a significant decrease in nitric oxide levels (p0.05), along with increases in superoxide dismutase and catalase activity (p> 0.05), as well as non-protein thiol levels (p0.05).

ST enhances memory through multiple mechanisms. Primarily, it inhibits acetylcholinesterase activity due to its anti-acetylcholinesterase properties, resulting in elevated acetylcholine release. Additionally, the extract restores brain antioxidant status through its antioxidant activities by scavenging free radicals and enhancing the activity of endogenous antioxidant enzymes. Collectively, these effects contribute to improved memory function.

Keywords: Streptozotocin, Alzheimer's disease, memory impairments, cholinergic dysfunction, oxidative stress.

Oral Presentations

S4-OP7

Preventive effect of *Graptophyllum grandulosum* on Conditioned place preference-induced nicotine addiction on pubescent adolescent rats

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Abstract:

Graptophyllum grandulosum (*G. grandulosum*) is traditionally used by the local population in Cameroon for its various medicinal properties. *G. grandulosum* contains multiple bioactive compounds such as chrysoeriol and luteolin. This study aimed to evaluate the preventive effect of the aqueous extract of *G. grandulosum* on nicotine addiction in pubescent rats. 42 Wistar pubescent adolescent male rats (divided into 6 groups of 7 animals each) were used during this experiment. Nicotine addiction was induced through 5 days of injections of nicotine tartrate (0.4 mg/kg). The behavioural assessment was done using the Conditioned Place Preference (CPP) test and the Elevated plus maze (EPM) test. The animals were sacrificed after the last behavioural test by cervical dislocation; the hippocampus and striatum were collected for biochemical assays (dopamine, acetylcholine, BDNF and pro-inflammatory cytokines). *G. grandulosum* inhibited CPP preference change (p 0.001) induced by nicotine injection and reduced hyperactivity and risk taking (p 0.001) in EPM test. G. grandulosum reduced dopamine levels (p 0.001) and increased acetylcholine levels (p 0.01). Additionally,
G. grandulosum modulated BDNF levels (p 0.001) and reduced pro-inflammatory cytokines (Il-1β and TNFα) (p 0.01). These results suggested that G. grandulosum had anti- addictive properties via modulation of dopaminergic, cholinergic, BDNF and neuroinflammatory pathways.

Keywords: *Graptophyllum grandulosum*, nicotine, dopamine, acetylcholine, BDNF.

S4-OP8

Protective effect of *Leptadenia hastata* (Pers.) Decne (Asclepiadaceae) in a mouse model of monosodium glutamate-induced neurotoxicity

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Abstract:

Background: brain degeneration during aging has become a major health problem. Glutamate toxicity has been implicated in numerous diseases such as Alzheimer's and schizophrenia. Plants with antioxidant properties protect the brain against glutamate toxicity. The aim of this study was to determine the therapeutic efficacy of *Leptadenia hastata* (*L. hastata*) against glutamate-induced neurotoxicity.

Methodology: Different doses (19, 38, and 76 mg/kg) of *L. hastata* decoctate were administered orally to mice 1 hour before glutamate (4 mg/kg p.o.). Behavioral tests consisted of Morris and Y-maze tests. After the behavioral tests, the animals were sacrificed, and the brains were removed for analysis of oxidative stress parameters.

Results: *L. hastata* decoctate reversed the glutamate-induced alteration in behavior by significantly ($P<0.001$) reducing the latency to reach the platform in the Morris maze, significantly increased the percentage of spontaneous alternation in the Y maze. Decocting significantly counteracted ($P < 0.001$) glutamate-induced oxidative stress parameters.

Conclusion: Our results show that *L. hastata* decoctate protects the brain against glutamate-induced neurotoxicity.

Key words: neuroprotection; monosodium glutamate; *Leptadenia hastata*; oxidative stress

Oral Presentations

Session 5 thematic:
Neurobiological and Psychosocial Impacts of Stress, Trauma, and Therapeutic Interventions

Chair of the session: **Hassan Ainani**,
Mohamed VI University Benguerir

S5-OP1

Vestibular perceptual training enhances vestibular perception, posture, and gait in older adults

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Abstract:

Background: Vestibular function declines with age, which is associated with the risk of falling. The highest rate of fall-related deaths or serious injuries is among people over the age of 60. We investigated the effects of vestibular perceptual training in older adults to counteract this sensory impairment, improve self-motion perception, posture, and gait.
Methods: Forty adults aged 70-88 took part in a two-week motion direction discrimination training with 2800 training trials per person. Vestibular thresholds, post-urography and gait parameters were measured before, during, and after training. Psychometric functions were fitted for the threshold comparison. For posture and gait, the pre- / post-difference was predicted.
Results: Post-training, participants exhibited improved angular roll tilt and linear inter-aural translation vestibular thresholds. This indicates improved perceptual sensitivity after specific perceptual training. The perception of the untrained motion was not affected by the training. The perceptual roll-tilt training reduced body sway. Gait was mainly influenced by the inter-aural translation training, leading to an increased step size and walking speed. These training effects only manifested after the completion of the full two-week training period.
Conclusions: We demonstrate that vestibular perceptual training lowers perceptual thresholds and improves posture and gait parameters in older adults. These findings suggest that targeted vestibular training can provide a unique and novel intervention preventing falls, improving overall quality of life, and counteracting age-related sensory decline in aging populations. Vestibular perceptual learning can complement traditional approaches to fall prevention.

Oral Presentations

S5-OP2

Potential role of Piezo1 in mechanical responses of neurons and glial cells in dorsal root ganglia

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Abstract:

Dorsal root ganglia (DRGs), located at the junction of the central and peripheral nervous system, are critical units involved in sensory signal transmission and processing mediating pain, touch and proprioception. These structures composed mainly of primary sensory neurons surrounded by satellite glial cells (SGCs) are subjected to diverse mechanical forces as a result of fluid flow (cerebrospinal fluid and vascular dynamics), tissue deformation or injuries. These mechanical forces are detected by mechanosome complexes of ion channels and signaling components. The Piezo mechanosensitive ion channels are believed to play specific roles in DRG neurons: Piezo2 is the touch receptor, while Piezo1 may contribute to itch. However, Piezo1 in other systems is crucial in detecting very low levels of shear stress. Its activation leads to an increase in Ca2+ influx and activation of downstream signaling pathways.

Our preliminary results show that Piezo1 activation with ahighly selective agonist greatly enhances neuron and SGC sensitivity to shear stress, as evidenced by a massive increase in Ca2+ signaling. In contrast to the apparent enhancement of neuronal responses to mechanical stimulation, Piezo1 activation depressed parameters of neuronal excitability, possibly through activation of Ca2+ sensitive K+ channels. TRPV4, a mechanosome component in some systems, has been previously shown to be activated downstream of Piezo1 signaling. Its activation in DRG neurons and SGCs enhances Piezo1 response to shear stress.

Activation of Piezo1 and its downstream signaling by shear stress levels experienced during physiological and pathological conditions encountered within the ganglia likely impact DRG neuron excitability and SGC Ca2+ levels. Studies are underway to evaluate whether targeting mechanosensitive ion channels in DRG neurons or SGCs may provide novel therapy in relief of hyper-excitability as occurs in neuropathic pain.

Keywords: DRGs, mechanosome, sensory neurons, SGCs

This project is supported in part by the NIH grant #R01DK138832-01 and Montefiore Orthopaedic Research Seed Grant

Oral Presentations

S5-OP3

Aspirin as A Modifier of Epigenetic Responses: DNA Methylation Changes in a Social Instability Stress Model of Depression in Female Wistar Rats

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Abstract:

Depression is the most common psychiatric disorder that poses a significant public health concern. Recent studies have implicated DNA methylation, an epigenetic modification, as a potential mechanism for increased susceptibility to depression. Aspirin [acetylsalicylic acid (ASA)] has been reported to have a possible antidepressant effect. This study aimed to determine the mechanisms of the antidepressant effect of aspirin in a social instability stress (SIS) model of depression. Eighty adult female Wistar rats (180?220 g) were acclimatised for twenty-one days. Ten were in the control group, and 70 were exposed to SIS to induce depression-like behaviours. Before and after induction, rats were subjected to behavioural tests to determine resilient and susceptible ones. Rats were divided into nine groups (n=7), and treated as follows: (i) control+distilled water (DW) (1 ml/kg) (ii) resilient+DW (1 ml/kg) (iii) susceptible+DW (1 ml/kg) (iv) susceptible+escitalopram (ESC) (10 mg/kg) (v) susceptible+RG108 (0.4 mg/kg) (vi) susceptible+ASA (10 mg/kg*);* (vii) susceptible+ASA (10 mg/kg) +ESC (10 mg/kg*);* (viii) susceptible+ASA (100 mg/kg*);* and (ix) susceptible+ASA (100 mg/kg) +ESC (10 mg/kg). ASA (oral), ESC (oral), and RG108 (intraperitoneal) were administered once daily for twenty-one days. Rats were euthanised, and brain samples were collected for gene expression and whole genome methylation studies. Data were analysed using one-way analysis of variance, Tukey post-hoc test with a significance level of p0.05 using GraphPad Prism 8.02. The result showed: (i) reversal of depression-like behaviours on behavioural tests; (ii) upregulation and downregulation of DNMT3L gene expression in the susceptible+ASA (100 mg/kg) and susceptible+ESC groups respectively, compared to the control; (iii) knockout of methylated depression susceptibility genes in the susceptible+ASA (100 mg/kg) and susceptible+RG108 (0.4 mg/kg) groups. However, all other groups showed differential methylation of several depression susceptibility genes. In conclusion, aspirin had DNA methylation inhibitory properties comparable to the standard DNA methyl-transferase inhibitor - RG108, and exerted its antidepressant effect through this epigenetic mechanism. This study recommends that aspirin should be used as an epigenetic-targeted (adjunct) antidepressant.

Keywords: Acetylsalicylic acid (ASA*);* Aspirin; Depression; DNA methylation; Gene expression; Whole genome methylation sequencing

Oral Presentations

S5-OP4

Effects of early-life stress on reproductive behavior in female mice

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Abstract:

Recent evidence suggests that puberty might be a particularly sensitive period to stress, potentially leading to lasting alterations in female reproductive behavior. In this study, we investigated the effects of chronic stress over puberty on female reproductive behavior and the neural circuits involved in its modulation.

Our findings reveal that stress during this critical developmental window disrupts lordosis behavior without affecting mate preference. This reduction in lordosis was associated with a specific reduction in the number of neurons expressing the neuronal form of nitric oxide synthase (nNOS) in the ventrolateral region of the ventromedial hypothalamus (VMHvl). Utilizing fiber photometry, we demonstrated that VMHvl nNOS neurons are responsive to olfactory social cues, showing the highest activation in response to male urine. However, this activation was significantly reduced in females subjected to stress during puberty.

Moreover, our results indicated that treatment with SNAP, a nitric oxide donor, into the VMHvl significantly enhanced lordosis behavior in pubertally stressed female mice.

Together, these findings provide novel insights into the role of VMHvl nNOS neurons in processing olfactory cues essential for the expression of female sexual behavior. Furthermore, we confirm that puberty is a critical stage for the development and organization of female sexual behavior. Stress during this period can induce permanent changes in sexual performance, due to disrupted integration of male olfactory cues by VMHvl nNOS neurons.

Keywords: Pubertal stress, Reproductive behavior, Lordosis, nNOS, VMHvl

Oral Presentations

S5-OP5

Evaluation of the anxiolytic potential of passionflower, valerian and lemon balm on physiological parameters and neurobehavioral effects in mice subjected to stress

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Abstract:

Background and objectives: Anxiety disorders are complex mental conditions characterized by intense anxiety that disrupts daily functioning. Anxiolytic drugs are effective, but their use is accompanied by side effects. Approaches such as phytotherapy emerge as good alternatives. This study aims to evaluate of plant extracts: *Passiflora Incarnata*, *Valeriana Officinalis* and *Melissa Officinalis*, compared to standard benzodiazepine treatment in a mouse model of constrained induced stress.

Methods: We utilized BALB/c female mice subjected to constraint induced stress, in order to assess the effect of stress on various behavioural and physiological parameters. The anxiolytic efficacy of the plant extracts was evaluated and compared to benzodiazepine treatment.

Result: We found that constrained induced stress effects various parameters such as food intake, body weight, anxiety behaviours, locomotor activity, motor coordination, and memory. Demonstrating the complexity of stress and anxiety regulation. Benzodiazepine showed effective anxiolytic properties, but did not address all stress consequences. Phytotherapy treatments showed notable anxiolytic effects. Each plant demonstrated specific targeting of different stress affected aspects.

Discussion: The findings suggest that *Passiflora Incarnata*, *Valeriana Officinalis* and *Melissa Officinalis* offer promising applications for phytotherapy in stress and anxiety management. The natural alternatives showed lower potency compared to conventional treatments, their lower risk profiles make them valuable therapeutic options.

Keywords: Anxiety, anxiolytic effects, phytotherapy, neurobehavior

Oral Presentations

Session 6 thematic:

Pollutants, Neurotoxicity, and Brain Disorders

Chair of the session: **Said Galai**,

National Neurological Institute, Tunis, Tunisia

S6-OP1

High-fat diet and Bisphenol A interactions sustained the physiological state of microglia and elicited expression of alpha-synuclein and parvalbumin proteins in the cerebellum of male Wistar rats

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Abstract:

Industrialization h as contributed to diet modifications and growing amount of toxic chemicals that seeps into these diets and poses threat to human health upon consumption. This study examined the interactional effects ofhigh-fat diet (HFD) and Bisphenol-A (BPA) on cerebellar expression of microglia and alpha-synuclein and parvalbumin proteins of male Wistar rats. The rats were divided into 4 groups each containing 7 rats. Groups A, B1, B2 and B3 received feed and water, 12.6 mg/kg body weight (b.w) of BPA only, hFD only (with 47.82% of fat), hFD and 12.6 mg/kg b.w of BPA respectively for 84 days. On 85th day, brains were excised and fixed 10% neutral buffered formalin for routinehistological and immunohistochemical procedures for the expression microglia, alpha synuclein and parvalbumin proteins of the cerebellum. Image J and Statistical Package for Social Science version 25 were used for numerical assessment with significant level set at P0.05. The nuclear areas expressing (NAE) alpha-synuclein in cerebellum of group B3 showed significant difference (P0.05; P= 0.013) when compared to group A. The comparison of NAE parvalbumin in cerebellum of group B3 showed significant difference (P0.05; P= 0.001) when compared to group A. No significance difference (P0.05; P= 0.566) was observed in NAE microglia in cerebellum of group B3 when compare to group A. Indigestion ofhFD and BPA combination, stimulated cerebellar expression of alpha-synuclein and parvalbumin proteins in male Wistar rats.

Keywords: Microglia, Alpha-synuclein, Parvalbumin, Cerebellum, Bisphenol-A, High-Fat

Oral Presentations

S6-OP2

Effects of exposure to micro/nanoplastics of polystyrene on neuronal oxidative stress, neuroinflammation, and anxiety-like behavior in mice

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Abstract:

Polystyrene is a polymer widely used across various industrial and commercial sectors. Upon degradation, it fragments into microplastics (MP) and nanoplastics (NP), whose accumulation in the environment, raises significant ecological and health concerns. These particles can disrupt digestive, reproductive, and other functions in exposed organisms. In this systematic review, the effects of mice exposure to PS-NP or PS-MP (PS-MP/NP) were systematically examined, focusing on neuronal oxidative stress, neuroinflammation, and anxiety-like behavior. Three databases (PubMed, Web of Science, and Scopus) were searched without any time filters until July 20, 2024. The study followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement. Two independent reviewers assessed the quality of studies using the Systematic Review Centre for Laboratory Animals Experimentation tool (SYRCLE). A total of 24 original articles were included of 332 citations. Articles were published between 2021 and 2024. Out of the studies reviewed, 12 used PS-NP, 10 used PS-MP, and two used both PS-NP and PS-MP separately. The particle sizes ranged from 0.023 to 50 µm, with the majority exhibiting a spherical shape. Seven studies reported results that the exposure to PS-MP/NP elevated reactive oxygen species (ROS) levels, and/or increased lipid peroxidation-Malondialdehyde (LPO-MDA), and/or decreased antioxidants, especially superoxide dismutase (SOD), catalase (CAT), and glutathione (GSH). Eight studies demonstrated increased neuroinflammation markers, including TNF-α, IL-1β, IL-6, GFAP, and Iba1. Regarding anxiety-like behavior, 10 studies confirmed its induction. Furthermore, maternal exposure induced neurotoxic responses in offspring. Overall, exposure to PS-MP/NP induced oxidative stress, neuroinflammation, and anxiety-like behavior in mice. These results highlighted complex interactions between PS-MP/NP and the nervous system, emphasizing the need to thoroughly explore involved mechanisms.

Keywords: Microplastics; Nanoplastics; Neurotoxicity; Mice.

Oral Presentations

S6-OP3

Maternal Exposure to Metam Sodium-Based Pesticides Induces Long-Term Neurodevelopmental Disorders Linked to Oxidative Stress and Neuroinflammation

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Abstract:

Maternal exposure to environmental toxicants is known to negatively affect the developing nervous system, with some effects persisting long after exposure and potentially resulting in permanent developmental neurotoxicity. Metam sodium-based pesticides (MS-BPs) are widely used for their economic efficiency, resulting in exposure levels that often exceed EPA guidelines, yet they remain notably understudied within this context.

The present study aimed to investigate the combined effects of prenatal and lactational exposure to MS-BP on postnatal neurodevelopment and behavior in mice. It also sought to elucidate potential mechanisms of MS-BP-induced neurotoxicity by examining oxidative stress and neuroinflammation. Sensorimotor maturation was assessed in offspring shortly after birth, and long-term behavioral outcomes evaluated in adulthood. Subsequent analyses measured oxidative stress markers and glial activation following behavioral assessment.

Our findings demonstrated that MS-BP exposure leads to significant delays in sensorimotor development and induces persistent anxiety-like, depressive-like, and cognitive deficits in adulthood. These behavioral alterations are accompanied by pronounced oxidative stress characterized by 300% increase in superoxide dismutase activity, a 100% rise in malondialdehyde levels, and a 50% reduction in catalase activity in the whole brain as well as in specific brain regions of exposed animals. Additionally, offspring exhibited a neuroinflammatory response, as evidenced by elevated expression of GFAP by 150% and Iba-1 markers by 20% relative to controls.

Together, these findings demonstrate that MS-BP exposure disrupts neurodevelopment through mechanisms involving oxidative stress and neuroinflammation, shedding light on the long-lasting neurotoxic effects of maternal MS-BP exposure. This underscores the urgent need for stricter regulations on MS-BP use to protect vulnerable populations and highlights the importance of further research to unravel the links between environmental toxicants and neurodevelopmental disorders.

Keywords: metam sodium, development, behavior, neuroinflammation, oxidative stress.

Oral Presentations

S6-OP4

Understanding the neurotoxic impact of pesticides on mammalian brain nicotinic acetylcholine receptors

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Abstract:

Neonicotinoid insecticides have become the fastest growing class of insecticides over the past few decades and have been banned in the European Union due to their potential adverse effects on humans and the environment. Recent derivatives, including a sulfoximine insecticides, sulfoxaflor, and a butanolide insecticide, flupyradifurone, were designed to replace neonicotinoids on the market, leading to new polemics concerning their use. They share their mode of action with neonicotinoids as selective agonists of nicotinic acetylcholine (ACh) receptors (nAChRs). Several studies have demonstrated that non-target species, including humans, could be exposed to these new compounds, and subsequently adversely impacted by them.

We conducted electrophysiological and molecular docking studies using human homomeric α7 and mammalian **α**4β2 neuronal nAChRs, to compare the effects of four neonicotinoids with recently introduced sulfoximine and butenolide insecticides. All neonicotinoids (except thiamethoxam), as well as the recently introduced flupyradifurone and sulfoxaflor, appear to be weaker agonists than acetylcholine on human α7 nAChR. The heteromeric α4β2 nAChR can be expressed in two different stoichiometries in the mammalian brain, high sensitivity and low sensitivity α4β2 receptors. Interestingly, we found that the low sensitivity α4β2 nAChR was activated by all tested insecticides, whereas the high sensitivity α4β2 receptor was only activated by ACh. Our research highlights the importance of receptor stoichiometry in determining the pharmacological properties of neonicotinoid insecticides. This insight is crucial for understanding their interactions with neuronal nAChRs and their safety for humans.

Oral Presentations

S6-OP5

Implementation of new protocols for Glyphosate and its byproduct detection. Application for demonstration of neurotoxicity by in vitro N2a cell culture.

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4 : CBIOS – Research Center for Biosciences & Health Technologies, Universidade Lusófona de Humanidades e Tecnologias Campo Grande, Lisbon, Portugal.

Abstract:

Glyphosate and its byproducts, particularly the AMPA (Amino-Methyl-Phosphonic Acid), have been found to exhibit significant neurotoxic effects. Its detection is difficult to implement due to its chemical properties and its low prevalence; there is a strong need to develop sensitive analytical methods for glyphosate (GLP) monitoring to study its prevalence and its toxicity. For this purpose, a new specific biosystem based on enzyme reaction was implemented by laccase, redox mediator (Acetosyringone: ASGN), and ionic liquid (IL, as conservator and activator) to catalyze GLP. This enzymatic reaction revealed a progressive catalysis of glyphosate by generating toxic byproducts accompanied by a gradual decrease of the glyphosate concentration, indicating its active degradation. This fact was demonstrated, for first time, by hPLC. Then, laccase-catalytic system has been investigated by two analytical methods: spectrophotometric and electrochemical one. To investigate the toxicity of glyphosate and its byproducts, it was evaluated via N2A cells culture, using cellular toxicity assays (FDA and LDH). It was demonstrated that the degradation byproducts of glyphosate induced significant cellular damage in the N2A neuronal cells causing essentially by ROS production proportionally with the reaction time. Prolonged exposure of N2A cells to glyphosate and its byproducts lead to severe cellular damage, highlighting the neurotoxic potential of these compounds. Regarding the implementation of new method for glyphosate detection, it was successfully set up two methods with different LOD, the detection limits for spectrophotometric method was 25µM GLP while electrochemical method was even lowest around 5µM GLP. The developing biosensor based on this enzymatic system has been carried out using gold-plated screen-printed electrode and Nafion polymer for laccase, redox mediator and ionic liquid complexes immobilization. GLP samples were successfully analyzed using cyclic voltammetry (CV) measurement at scan rate of 100 mV/s. The concentration of GLP was accurately determined in the range of 5µM to 15µM GLP, and high correlation rate (98%) between current density and GLP concentration was determined using the laccase-based-biosensor, which been used for GLP assays in biological samples (cell lysate and culture medium) to demonstrate the prevalence of glyphosate into the medium and cells.

Oral Presentations

S6-OP6

Multigenerational Reproductive and Neuroendocrine Effects of Chronic Mercury Chloride Exposure in Female Mice

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3: high Institute of Nursing Professions and Health Techniques Dakhla Annex, Dakhla, Morocco

Abstract:

Chronic exposure to mercury, recognized as one of the ten most hazardous chemicals for Public health poses significant risks to reproductive health. Our study investigates the multigenerational effects of low-dose of mercury chloride exposure on female reproductive health, focusing on the maternal lineage across F1, F2, and germline-exposed F2' generations. Phenotypic analyses revealed that mercury exposure delayed vaginal opening and first estrus, disrupted estrous cyclicity, and reduced mating behavior, as confirmed by a two-choice preference test. These phenotypic changes were associated with decreased mRNA expression of kiss1 and Gnrh1 in the hypothalamus, critical regulators of the hPG axis, alongside reduced Bdnf expression, suggesting impaired neuronal plasticity and communication within the reproductive neural circuits. The inflammatory response, a potential contributor to these observed phenotypes, was evident through increased TNF-α and Il-6 mRNA expression across all exposed generations. Importantly, these outcomes were observed not only in directly exposed F1 and F2 generations but also in germline-exposed F2', indicating the transgenerational impact of mercury on reproduction.
Our findings highlight mercury's potential to induce heritable reproductive deficits through neuroendocrine and inflammatory pathways, underscoring the need for stricter environmental regulations.

Oral Presentations

S6-OP7

Roles of oxidative stress and pro-inflammatory cytokines in copper sulfate-induced depression-like disorders and abnormal neuronal morphology in mice.

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Abstract:

Epidemiological studies have implicated copper as one of the key environmental risk factors for the pathogenesis of major depression. However, the precise mechanism by which copper contribute to the genesis of depression particularly the involvement of oxidative stress-driven neuro-inflammation is yet to be fully elucidated. Thus, this study was designed to evaluate the effects of copper sulfate (CuSO4) on depression-like behaviors and the role of oxidative stress and pro-inflammatory cytokines in mice. Forty male Swiss mice were distributed into control and three test groups (n= 10), and were treated orally with distilled water (10 mL/kg) or CuSO4 (25, 50 and 100 mg/kg) daily for 28 days. Afterwards, the tail suspension, forced swim, and sucrose splash tests were used for the detection of depression-like behaviors. The animals were then euthanized and the brains were processed for the estimation of biomarkers of oxidative stress and pro-inflammatory cytokines (tumor necrosis factor-alpha and interleukin-6). The histo-morphological derangements and neuronal viability of the prefrontal cortex, hippocampus and striatum were also evaluated. Mice exposed to CuSO4 displayed depression-like features when compared with controls. The brain concentrations of malondialdehyde, nitrite and pro-inflammatory cytokines were elevated in mice exposed to CuSO4. Mice exposed to CuSO4 also had reduced brain antioxidant status (glutathione, glutathione-s-transferase, total thiols, superoxide-dismutase and catalase), as well as altered histo-morphological features, and decreased population of viable neuronal cells. These findings suggest that CuSO4 induces oxidative stress and pro-inflammatory cytokines, and abnormal neuronal morphology to elicit depression-like effects in mice.

Keywords: Copper sulfate, Depression, Oxidative stress, Pro-inflammatory cytokines.

Oral Presentations

S6-OP8

Effect of Aging and mercury chloride intoxication interaction in mice

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Abstract:

Exposure to mercury chloride (HgCl2), a deleterious environmental toxic, thath as significanthealth implications, such as neurological abnormalities, particularly in older adults. Aging naturally induces physiological changes that may increase susceptibility to mercury's toxic effects.The aim of this study was to evaluate the interaction of age and mercury exposure and their combined effects on the nervous system of aging mice, particularly in relation to memory, motor activity, and anxiety. To achieve this, we employed behavioral tests, including the Y-maze, Open field, and object recognition (TRO) tests on mice treated withhgCl2 from fetal life through postnatal stages and into aging. Results indicate that the association between mercury and aging decrease memory in TRO and Y-maze, motor activity in open filed and increases anxiety in aging mice treated with mercury compared with controls.

Key words: Mercury chloride (HgCl2), Aging, Neurotoxicity

Oral Presentations

S6-OP9

Lambda-cyhalothrin sex-dependently alters behavioural abilities in Swiss mice

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Abstract:

Lambda-cyhalothrin (LCT) is a type-II pyrethroid extensively used in agriculture for plant protection against pests. However, few studies explored the effect of LCT on mice, and especially the sex-based differences. The present study evaluates the effect of LCT on behavioural aspects of eight weeks aged female (F) and male (M) Swiss mice. Mice were divided into six groups including LCT-treated mice that received through gavage (i) 0.5 mg/kg bw F, (ii) 0.5 mg/kg bw M, (iii) 2 mg/kg F, (iv) 2 mg/kg M, and two vehicle control groups (v) F and (vi) M. Behavioural aspects are assessed for locomotor activity by the open field test, anxiety using the dark-light box test, learning memory with the novel object recognition test, memory retention by the elevated plus maze test, spatial working memory using the Y-maze test, and social memory via the three-chambered social interaction test. Subacute treatment for three weeks with low doses of LCT shows a decrease in the locomotor activity in both male and female mice by reducing the number of line crossings. Yet, in females specifically but noy in males, LCT exhibits an anxiogenic effect; by increasing the time spent in the dark compartment, impairs memory retention; by raising the latency time, and affects learning memory by reducing the recognition index parameter. Notably, social memory shows an alteration in males at the expanse of females indicated by reducing the novelty percentage. However, LCT does not significantly alter spatial working memory in both genders. In conclusion, LCT-treated male and female mice exhibit distinct alterations influenced by both sex and behaviour. LCT-differential effects could either be associated with sex-based differences of LCT-CNS accessibility or may reflect different actions on steroid components within the CNS.

Keywords: Lambda-cyhalothrin, sex-based difference, locomotion, anxiety, memory, behavioural tests.

Oral Presentations

Session 7 thematic:

Neurobiological mechanisms of behavior, emotion, and addiction across diverse models

Chair of the session: **Abderrazzak Ghanima**,
Cadi Ayyad University, Marrakech Morocco

S7-OP1

Peptidergic systems involved in aggressive processes after alcohol intoxication

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Abstract:

Alcoholism has been linked extensively to violence and aggressive behavior. About half of all violent crimes and sexual assaults are committed under the influence of alcohol. However, the underlying neuroanatomical mechanism of this connection remains elusive. The role of the Nucleus Incertus in addiction and relapse of alcoholismhas already been demonstrated in animal models. Its Relaxin 3 expressing cells projects towards telencephalic centers that control important aspects of motivation and emotions including the Amygdala and Septum. Relaxin3 signaling was shown to regulate alcohol intake and relapse-like behaviors. Moreover, previous research in our group points at the somatostatin neurons in the medial amygdala as a relevant regulator of this behavior. In this work, we have investigated sex-specific behavioral alterations after chronic alcohol intoxication. Also, we have considered neuronal activity changes, Relaxin 3 levels and introduced an emerging therapeutic target. Only male mice showed increased aggressive and dominant behavior after chronic alcohol intoxication during acute but not during protracted withdrawal periods. This transient increase was parallel to significantly lower levels of c-Fos in several brain areas of alcohol-intoxicated males and these levels were restored during protracted withdrawal. Additionally, Relaxin 3 expression showed an increase during females- acute withdrawal and males protracted withdrawal. Both c-Fos and Relaxin 3 levels in Medial amygdala showed negative correlation with aggressive behavior. Furthermore, medial amygdala somatostatin interneurons showed significant lower expression of c-Fos in alcohol-intoxicated males during the acute withdrawal period. This c-Fos downregulation in somatostatin interneurons was restored during the protracted withdrawal. Interestingly, Pharmacogenetic manipulation of somatostatin interneurons in medial amygdala modulates aggressive behavior after chronic alcohol intoxication. Inhibition of somatostatin interneurons through chemogenetically expressedhM4Di receptors promoted the aggressiveness while activation through chemogenetically expressedhM3Dq receptors significantly blocked the aggressiveness during both acute and protracted withdrawal. These results introduce somatostatin interneurons in medial amygdala as an emerging therapeutic target for aggressive behavior associated with alcohol withdrawal syndrome.

Oral Presentations

S7-OP2

Effects of maternal separation with or without social isolation on aggressive behavior in mice

Cathy Perrina Ranaivomanana, Ilias Chaibi, Ihsane Ait-Mansour, Fatima-Zahra Lamghari Moubarrad, Saadia Ba-M'hamed, Mohamed Bennis

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Abstract:

Aggression is vital for survival but becomes problematic when excessive, manifesting as pathological violence. Social isolation (SI) is a recognized factor that exacerbates aggressive and antisocial behaviors in both humans and adult rodents. Previous research has demonstrated that SI induces abnormalities in the anterior cingulate cortex (ACC), a key brain region regulating social and emotional behaviors. These disruptions influence aggression from childhood onward, with early-life stressors such as parental abuse or neglect leaving lasting impacts on social development and psychiatric vulnerability. Maternal separation (MS), a widely used model of early-life stress in rodents, is associated with increased aggression, although the underlying mechanisms remain unclear.

This study investigates the effects of MS, with or without SI, on comorbid aggression with anxiety and pain sensitivity, as well as neural changes in the ACC of adult mice. Swiss albino pups were subjected to MS for four hours daily from postnatal day (PND) 2 to PND 20. During adolescence (PND 46-60), some groups also underwent two weeks of SI. The mice were divided into four groups for each sex: a control group (MS-/SI-), a group exposed to MS alone (MS+/SI), a group exposed to SI alone (MS-/SI+), and a group subjected to both stressors (MS+/SI+).

The results revealed a significant increase in aggression exclusively in males of the MS+/SI+ group. Reduced social behavior was observed in both sexes in the MS+/SI- and MS+/SI+ groups. Elevated anxiety was detected solely in the MS+/SI+ group, while mechanical allodynia occurred in the MS+/SI- and MS+/SI+ groups. No alterations in thermal sensitivity were observed. Neuronal analysis revealed morphological changes in the ACC in the MS+/SI- and MS+/SI+ groups, regardless of sex. These changes included reduced dendritic length, branching, spine density, and arborization complexity in pyramidal neurons.

This study demonstrates that MS increases susceptibility to SI-induced behavioral disorders, while SI amplifies MS-related behavioral and structural effects. Together, these findings highlight the synergistic impact of MS and SI on aggression, anxiety, pain sensitivity, and ACC alterations, providing insights into pathological aggression mechanisms.

Keywords: Aggression, maternal separation, social isolation, and anterior cingulate cortex.

Oral Presentations

S7-OP3

Topiramate inhibits aggressive behavior through targeting morphological and functional alterations of the anterior cingulate cortex in socially isolated mice

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Abstract:

Topiramate, an approved antiepileptic drug, was found effective in treating aggressive symptoms in humans and rodents. However, the effects and mechanisms of Topiramate on aggressive behavior are still unclear. Our previous study indicated that intraperitoneal administration of Topiramate successfully decreased aggression and reinforced sociability in socially aggressive mice, and increased cFos-expressing neurons in the anterior cingulate cortex (ACC). In addition to its pharmacological properties, previous studies have approved the neuroprotective effects of Topiramate. These suggest a potential effect of Topiramate on ACC's structure and function. In the present study, we first investigated the structural characteristics of ACC in the social isolation- induced aggression paradigm. The results showed that hyper-aggressive behavior in socially aggressive mice was associated with several structural alterations in ACC: increased neuron death combined with decreased neuron density, increased damaged neuronal morphology and increased neuroinflammation markers. Moreover, in vivo extracellular recordings indicated that ACC neurons were hypoactive in aggressive mice compared to the control group. Based on these observations, we next investigated the potential neuroprotective effect of Topiramate against structural alterations of ACC observed in socially aggressive mice. Results indicated that intraperitoneal administration of Topiramate (30 mg/kg) decreased aggression and enhanced sociability without affecting locomotor activity. Interestingly, these effects were associated with decreased neuronal death, ameliorated damaged neuronal morphology, decreased reactive microglia markers in ACC and ameliorated neuronal activity.

Our results provide insights into the structural alterations of ACC in aggressive socially aggressive mice. Moreover, the present study suggested that the anti-aggressive effect of Topiramate could be related to its neuroprotective effects against the structural alterations of ACC.

Oral Presentations

S7-OP4

Neuropeptidergic Modulation and Neural Pathways in Contextual Fear Conditioning and Extinction: Insights from the Retrosplenial Cortex and Nucleus Incertus

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Abstract:

This work examines the mechanisms underlying contextual fear conditioning and the role of neural networks in memory processing. We assessed how different procedural factors influence the acquisition and extinction of fear in rats, revealing that spaced sessions of conditioning enhance extinction, while minimal shocks produce context-specific fear. More intense conditioning, however, leads to fear generalization across contexts, which can be extinguished similarly to the conditioned context. Additionally, we explored the role of the retrosplenial cortex (RSC) and its interaction with the neuropeptide relaxin-3 (RLN3) via the RXFP3 receptor. Modulation of RXFP3 in the RSC delayed fear extinction without affecting acquisition, suggesting that RLN3/RXFP3 signaling enhances memory strength, making extinction more resistant. Anatomical studies confirmed a strong presence of RLN3-immunoreactive fibers in the RSC, particularly co-localizing with inhibitory circuits. Finally, we investigated the connectivity between the RSC and other brain regions, particularly the bidirectional projections between the RSC, prefrontal cortex, and nucleus incertus. These connections were confirmed using anterograde and retrograde tracers, highlighting a broader cortical and subcortical network that involves key regions such as the prelimbic and orbitofrontal cortices, the medial septum, and the supra mammillary nucleus. Together, these findings provide a more integrated understanding of the circuitry involved in contextual fear and memory processing, emphasizing the long-loop interactions between the retrosplenial cortex, prefrontal areas, and subcortical structures in regulating fear acquisition and extinction processes.

Oral Presentations

S7-OP5

Predictive processing and autistic traits: Neurophysiological evidence across sensory modalities and task demands

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Abstract:

Background: Predictive Processing is a framework that suggests the brain constantly generates and updates predictions about sensory input, minimizing the difference (or "prediction error") between expectations and actual experiences. This framework has recently gained attention as a promising model for understanding Autism Spectrum Disorder (ASD) manifestations. However, neurophysiological findings remain inconsistent, suggesting that factors such as sensory modality, task complexity, and age may influence how these mechanisms work. Furthermore, recent studies indicate that autistic traits exist on a spectrum within the general population. Individuals with high autistic traits in non-clinical groups often exhibit certain neurocognitive and behavioral similarities to those diagnosed with ASD. If individuals with high autistic-like traits demonstrate neural patterns similar, though milder, to those seen in clinical ASD, examining these subclinical populations could yield valuable insights into ASD-related mechanisms.
Methods: 122 participants (ages 18-60; 62 females) completed visual and auditory oddball tasks with two difficulty levels while undergoing EEG. Autistic traits were assessed using the Autism Quotient, and event-related potentials were averaged from target/ unexpected trials.
Results: In the visual tasks, participants with higher communication difficulties (based on their AQ Communication scores) showed a trend-level reduction in P300 amplitude. In contrast, those with higher levels of Restricted Interests and Detail Orientation (RIDO) showed increased N2 amplitude (but not P300). In the auditory tasks, we also found changes in the N1-P2 brain response among participants with higher communication difficulties. Notably, the RIDO trait did not strongly correlate with other autism-related traits like Social Skills or Communication. These findings, alongside neurophysiological data, underscore the presence of distinct social and non-social dimensions within ASD, detectable even at the subclinical level, particularly for visual stimuli.
Conclusion: Our findings partially support the Predictive Processing framework in ASD, showing that increased prediction errors (enhanced N2 amplitudes) depend on task demands and sensory modality. Trait-specific dimensions (social vs. non-social) influence prediction errors differently, with significant effects for visual stimuli evident even at subclinical levels. This contributes to dimensional systems focusing on symptom-specific neurobiological markers, refining perspectives on ASD beyond traditional diagnoses.

Keywords: Autism, EEG/ERP, Predictive Processing, Visual, Auditory

Oral Presentations

Session 8 thematic:

Neurodevelopmental, Neuroinflammatory, and Neurodegenerative Mechanisms in Disease Models and Behavioral Outcomes

Chair of the session: **Silvestre Sampino**
Institute of Genetics and Animal Biotechnology, Poland

S8-OP1

Dysfunction of the Nfe2l1 Gene Disrupts Parkinson's Disease-Related Gene Expression Under Ubiquitin Stress and Impairs Neuronal Differentiation in P19 Cells

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Abstract:

Proteostasis is essential for neuronal health, and its dysregulation is implicated in neurodegenerative diseases like Parkinson's disease (PD). Nfe2l1, a key regulator of proteostasis and ubiquitination, has emerged as a critical factor in neuronal health, though its precise molecular functions in neuronal cells remain poorly understood. Upon proteasome inhibition using MG132, both Nfe2l1 protein and RNA levels were significantly elevated in wild-type (WT) and differentiated P19 cells. RNA sequencing of differentiated cells under proteasome inhibition revealed an enrichment of neurodegenerative pathways, particularly those linked to PD, emphasizing the relevance of Nfe2l1 in these processes. Proteasome inhibition also upregulated several PD-related genes, including Atf6, Camk2d, and Sod1. Importantly, Nfe2l1 knockdown in differentiated cells significantly reduced the expression of these genes, under proteasome inhibition, indicating that Nfe2l1 is a key regulator of PD-related pathways. Interestingly, Nfe2l1 knockdown in differentiated cells caused a marked reduction in the expression of the long non-coding RNA Neat1, which was initially upregulated in response to proteasome inhibition. Given the emerging role of Neat1 in PD pathology, its dysregulation further implicates Nfe2l1 in neurodegenerative processes. Additionally, the neuronal marker Map2 was notably downregulated in Nfe2l1 knockdown cells, suggesting impaired neuronal differentiation. Finally, under proteasome inhibition in differentiated cells, Nfe2l1 knockdown resulted in increased ubiquitination levels compared to controls, underscoring Nfe2l1's critical role in protein degradation and neuronalhomeostasis under stress conditions. Our findingshighlight Nfe2l1 as a vital regulator in neuronal cells, with essential roles in maintaining proteostasis and modulating neurodegenerative disease mechanisms, including those associated with PD.

Oral Presentations

S8-OP2

Prenatal determinants of neurodevelopmental programming in the BTBR mouse model of autism

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Abstract:

The etiology and pathogenesis of neurodevelopmental disorders remain poorly understood, with evidence suggesting a complex interplay of genetic factors and environmental influences disrupting neurodevelopmental trajectories during fetal life. Using the BTBR T+ Itpr3tf/J (BTBR) mouse model of idiopathic autism and corpus callosum agenesis, we investigated maternal and embryonic contributions to offspring neurodevelopment and the programming of autism-like behaviors through reciprocal embryo transfers between BTBR and the C57BL/6J (B6) control strain. Furthermore, we generated BTBR-B6 chimeric mice by aggregating TAU-GFP-labeled BTBR embryonic stem cells (ESCs) with B6 preimplantation embryos to assess the contributions of each strain's cells to chimeric brain structures.

BTBR conceptuses exhibited fetal and placental growth restriction, accompanied by altered placental histology, transcriptome profiles, and metabolic function compared to B6 controls. Notably, the autism-like behavioral phenotype characteristic of BTBR mice persisted after embryo transfer into B6 female recipients, highlighting its intrinsic genetic basis. In chimeras, BTBR ESCs successfully integrated into B6 preimplantation embryos, contributing to multiple organs and tissues, including the brain. Remarkably, most chimeric brains displayed normal neuroanatomy, including the presence of a corpus callosum derived from BTBR cells, indicating that BTBR neurons retain the intrinsic capability to form callosal projections when engrafted into a B6host environment.

These findings demonstrate that the autism-like behavior of BTBR mice is innate and independent of the maternal environment during gestation. They also highlight the critical role of prenatal metabolic adaptations mediated through the placenta in shaping neurodevelopmental outcomes. Additionally, our results suggest that non-neuronal cells or tissues may play a pivotal role in the neuropathogenesis of corpus callosum agenesis, as BTBR neurons inherently possess the ability to form callosal projections in chimeras.

Oral Presentations

S8-OP3

Modeling Schizophrenia Endophenotypes in *Drosophila Melanogaster*: Effects of Ketamine on Anxiety, Aggression, Locomotion, Neuroinflammatory and Neurodegenerative Responses

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Abstract:

Schizophrenia presents a significant challenge in mental health, characterized by a profound distortion of reality, often accompanied by hallucinations, delusions, cognitive deficits and neuroinflammatory processes. Ketamine has been widely used as a pharmacological agent to model schizophrenia symptoms in both human and animal studies. However, the potential of ketamine to induce schizophrenia-like phenotypes in Drosophila melanogaster remains under-investigated. This study therefore investigated the effects of ketamine on anxiety, aggression, locomotor activities, neuroinflammatory and neurodegeneration responses as a preliminary step toward developing a pharmacological model of schizophrenia in this organism.

Virgin male and female Oregon-R flies were collected after eclosion and exposed to four different concentrations of ketamine (100, 250, 500, 1000µg/mL) for 1 week under standard laboratory conditions (22-25°C, 50-60%humidity). Experimental groups consisted of 10 vials each containing 10 flies each. Anxiety, aggression and locomotory functions were assessed behaviorally through the open field, aggression, and rapid iterative negative geotaxis (RING) assays. Pro-inflammatory and astrogliotic responses were measured immunohistochemically using Tumor necrosis factor-alpha (TNF-A) and Glial fibrillary acidic protein (GFAP) antibodies. General neuronal architecture was evaluated using the H&E histological staining techniques.

The results showed a dose-dependent induction of aggressive behavior. Improved climbing ability in the RING assay demonstrated that ketamine enhanced Motor function in a dose-dependent manner. However, at the highest dose, it impaired locomotion. Survival assays indicated that higher doses of ketamine reduced survival rates. Immunohistochemical analysis revealed a dose-dependent increase in TNF-A and GFAP mean fluorescence intensity across the treatment groups, indicating upregulation of TNF and GFAP expressions. This suggests a robust pro-inflammatory and astrogliotic response to ketamine administration, aligning with the emerging neuroinflammatory endophenotype theory of schizophrenia aetiology and its experimental modeling. Histological analysis displayed significant dose-dependent histopathological changes, including increased cell loss and vacuolization at higher ketamine concentrations.

In conclusion, the findings contribute to our understanding of how ketamine influence key behavioral and neurobiological parameters offering insights into it's potential as a pharmacological model of schizophrenia in Drosophila as the findings are consistent with those seen in mammalian models.

Keywords: Ketamine, Drosophila melanogaster, Neuroinflammation, TNF-A, Schizophrenia, GFAP.

Oral Presentations

S8-OP4

Different cortical and subcortical astroglial responsiveness in rats with acute liver failure

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Abstract:

Hepatic encephalopathy (HE) is a neuropsychiatric complication of liver failure. Previous studies described astroglia alterations in hE, but regional changes were not well investigated and available data were divergent. The present study aims to investigate the regional astroglial responsiveness in an experimental model of acute liver failure (ALF) in rats. We used a surgical model of ALF induced by a portocaval anastomosis (PCA) followed byhepatic artery ligation (HAL). At the coma stage, regional astroglial responsiveness was assessed using glial fibrillary acidic protein (GFAP) immunohistochemistry with a morphometric analysis using ImageJ. The brain regions selected for analysis included four cortical regions: somatosensory (S1Tr and S1BF), piriform (Pir), and perirhinal (PRh) cortices and four subcortical regions including the corpus callosum (CC), ventromedial thalamus (VMT), mammillothalamic tract (MTT), and the dorsomedial hypothalamic nucleus (DHN). Our data showed a decreased number of astrocytes in S1Tr, Pir, and CC in ALF rats. GFAP-immunoreactive cells are increased within other regions including PRh, VMT, MTT, and DHN. Cell morphometric analysis showed a significant increase in GFAP-immunoreactive astrocyte processes and cell bodies in cortical and subcortical regions but not in CC and DHN. However, astrocyte perimeter showed a high increase, particularly in S1Tr and VMT. Our work demonstrates regional specificity including (1) regions with astrocyte activation associated with increase of GFAP-immunostaining and astrocyte cell counts, together with (2) unaltered GFAP components, and (3) regions characterized by supposed inactive astrocyte with a reduced GFAP-immunostaining. Our findings may reflect either (a) alterations regionally different inhE, or (b) stages of an alteration progressing differently within brain regions.

Keywords: Astrocytes, ALF, immunohistochemistry, hepatic encephalopathy, GFAP, regional responsiveness.

Oral Presentations

Session 9 thematic:

Neurobiological Impacts of Environmental, Genetic, and Nutritional Factors on Brain Structure and Function

Chair of the session: Toluwalope AJONIJEBU,

Northwest University South Africa

S9-OP1

The impact of Preterm Birth on Gyri and Sulci' Structural and Functional Connectivity

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Abstract:

Preterm birth is increasingly prevalent worldwide and is associated with severe consequences, including motor, cognitive, and psychiatric abnormalities. It particularly affects the gyrification process during the third trimester. Meanwhile, growing evidence has identified significant structural and functional differences between gyri and sulci regions, supporting the hypothesis that these regions might play distinct roles in cortical function. This study aimed to investigate how preterm birth impacts the structural and functional patterns of gyri and sulci regions. We used a dHCP open dataset of full-term and preterm-born neonates (207 subjects) and further parcellated brain regions into gyri and sulci based on the vertex curvature values. Using DTI images, structural connectivity between these region types was calculated, and functional differences were analyzed from fMRI BOLD signals using synchronization measures, nodal degree, and network-based statistics (NBS). Our findings revealed that preterm birth reduces the average structural connectivity strength between gyri and decreases the ratio of intra-gyri to gyri-sulci connections. This ratio was significantly associated with gestational age, birth weight, and global mean synchronization. Additionally, gyri were less involved as functional hubs in preterm neonates, while decreased synchronization disproportionately affected sulci regions. Using NBS, we identified a cluster of hypo-connections, with most links connecting gyri and sulci regions. Our results suggest that preterm birth affects gyri and sulci differently, and that the possible distinct functional roles of both region types may be disrupted, providing new insights into the underlying mechanisms of its impact on brain function.

Key words: Preterm birth, neonates, cortical folding, gyri, sulci, synchronization

Oral Presentations

S9-OP2

Dissociating sensory-discriminative and affective-emotional pain processing in the mediodorsal thalamus

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Abstract:

The mediodorsal thalamus (MD) is a key structure in several neurological and psychiatric diseases. In rats, MD has three subdivisions (Central, medial, and lateral). Each subdivision projects to a single and specific region of the PFC, suggesting potential differential involvement in pain processing. Prior research suggests distinct roles for MD subdivisions in cognitive processes, but their contributions to pain processing remain unexplored. Therefore, the present study was designed to explore the role of the MD thalamus in pain processing, with a specific focus on the contribution of each subdivision to the transmission and processing of the sensory-discriminative and affective-emotional components of pain. Bilateral excitotoxic lesions were performed in adult rats targeting the medial-central (MDmc) section, or its lateral (MDL) subdivision. Pain sensitivity was assessed through sensory (Von Frey filaments, hot/cold plate) and affective (place escape avoidance) tests. The prefrontal cortex was also analyzed for neuronal morphology (Golgi-Cox) and glial cell activity (GFAP, iba1 markers). To investigate the role of MD projections to the prefrontal cortex in pain dimensions, optogenetic modulation of these projections was performed in vivo, using the same behavioral pain tests performed in the lesioned rats. Our findings revealed a significant increase in both mechanical and thermal nociception following MD lesions, demonstrating a filtering role for the MD in the sensory-discriminative component of pain. However, only lateral MD lesions reduced the aversive response to pain, implicating this region in emotional processing. Morphological analysis showed disrupted neuronal structures and increased glial activation in the PFC. Optogenetic manipulation of MD projections to the PFC either mimicked or reversed the effects observed following MD lesions in the lateral subdivision, confirming the role of these projections in processing the emotional component of pain. Conversely, stimulation or inhibition of the medial-central MD consistently elicited hyperalgesia, suggesting a more complex role for this part in sensory pain treatment. Our findings underscore the MD's critical role in pain processing, emphasizing the functional specialization of its subdivisions and their modulatory influence on the PFC. Considering each subdivision as distinct opens avenues for targeted interventions in chronic pain management. Mediodorsal thalamus, pain, prefrontal cortex, affective component, sensory-discriminative component.

Oral Presentations

S9-OP3

Preventive effects of environmental enrichment on behavioral impairment and morphological alterations of the mid-cingulate cortex in aggressive socially isolated mice

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Abstract:

Social and environmental factors influence behavior by modulating brain structure and function. Additionally, the stress response in both rodents and humans is profoundly dependent on the environmental context. Recently, accumulating evidence have demonstrated that enriched environment (EE) -housing conditions that enhance sensory, cognitive, and motor stimulation-has preventive and therapeutic effects against stress. Besides, many studies have implicated the mid-cingulate cortex (MCC) in the control of aggressive behavior. Therefore, in this study, we aimed examining whether EE can prevent social isolation-induced behavioral outcomes. We also investigated the neuronal morphology of the MCC as a potential mechanism by which EE exerts its protective effects. For this purpose, adult male Swiss mice were individually housed for six weeks, either in standard cages (SC-isolated) or in an enriched environment (EE-isolated) consisting of a larger cage with a running wheel, regularly changed toys, tunnels, and treats. Behavioral tests were then performed to evaluate aggressive behavior, social behavior, and anxiety-like behavior. Subsequently, a histological analysis of the MCC was conducted using Golgi-Cox staining at the end of the behavioral testing. We found that, compared to SC-isolated mice, those in the enriched environment showed reduced aggressiveness, better social interactions, and reduced anxiety-like behavior. Moreover, MCC neurons of EE-isolated mice exhibited more complex dendritic arborization. Taken together, our data suggest that environmental enrichment during social isolation can prevent behavioral impairment and that unimpaired neuronal morphology in the MCC is essential for this resilience. In summary, our results support the use of EE as a non-pharmacological alternative for confronting stress, particularly in situations where socialisolation is imposed (e.g., pandemic confinement, solitary confinement).

Keywords: Enriched environment, social isolation, aggressive behavior, mid-cingulate cortex

Oral Presentations

S9-OP4

Neuroanatomical adaptations in the ocular and visual systems of African tree squirrels, crucial for their arboreal lifestyle.

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Abstract:

Background and Objectives: African tree-dwelling squirrels are diurnal rodents known for their exceptional visual acuity; an adaptation crucial for survival in their arboreal habitats. This study aimed to investigate the neuroanatomical adaptations of these squirrels by conducting a detailed histological and immunohistochemical analysis of their ocular structures and key visual brain regions, providing insight into the mechanisms supporting their tree-dwelling lifestyle.

Methods: We examined ocular and brain tissues from five squirrels captured at the University of Ibadan, Nigeria. Paraffin-embedded samples were subjected to routine histological processing, special staining, and immunohistochemical analyses.

Results: Histological analysis revealed densely packed stromal fibers in the cornea and undulations in the innermost corneal epithelial layer. The basement membrane of the corneal epithelium was strongly PAS-positive, and pronounced pigmentation was noted in the choroid, iris, and ciliary epithelia. The retinal architecture was multilayered with densely packed ganglion cells. Immunohistochemistry showed strong glial fibrillary acidic protein expression in the retinal nerve fiber layer and optic nerve.

In the brain, neuroglial cells resembling astrocytes were found in association with pinealocytes and capillaries in the pineal gland. The rostral colliculus exhibited an enlarged structure with densely packed neurons of varying sizes, organized into two laminae.

Discussion: The immunohistochemical findings demonstrate robust neural support, particularly in the retinal and optic nerve regions. Additionally, the analysis of key visual brain structures reveals a complex neural organization, comparable to that observed in primates. These results highlight significant neuroanatomical adaptations in the ocular and visual brain regions of African tree squirrels, which are critical for their exceptional visual acuity and sensorimotor coordination-key factors for thriving in arboreal environments. This study offers the first in-depth characterization of these adaptations, paving the way for future research into the neurobiological mechanisms that support arboreal lifestyles.

Keywords: Ocular adaptations, Neuroanatomy, Arboreal rodents, histology, Visual processing.

Oral Presentations

S9-OP5

Morphological alterations of the pyramidal cells in the anterior cingulate cortex in Monosodium Iodo-acetate-Induced Osteoarthritis in Mice

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Abstract:

Osteoarthritis (OA) is a degenerative joint disease characterized by chronic pain, which leads to various comorbidities, including emotional dysregulation and cognitive impairment. Patients with OA, exhibit alterations in brain connectivity within regions comprising the pain matrix. The objective of our study is to ascertain whether monosodium iodo-acetate (MIA) -induced OA affects neuronal morphology in the anterior cingulate cortex (ACC), given the critical role of this region in pain perception and affective processing. To this end, mice were given MIA injections and tested for pain sensitivity, anxious-depressive-like behaviors, and cognitive dysfunction. Results showed that MIA-treated mice developed mechanical allodynia and thermal hypersensitivity. Moreover, gender differences were observed, with female mice showing heightened sensitivity to painful stimuli. Pain-induced anxious-depressive-like behavior and cognitive impairment were found in mice with late-stage OA compared to the sham group. These behavioral changes were associated with significant structural alterations in the pyramidal neurons of the ACC, characterized by reduced dendritic branching complexity and decreased dendritic spines in both male and female OA mice compared to sham controls. These findings are the first to demonstrate abnormal dendritic morphology in pyramidal neurons of the ACC in a MIA-induced OA mouse model, aligning with similar structural abnormalities reported in OA patients.

Oral Presentations

S9-OP6

Protective effects of prenatal Omega-3 supplementation on brain and behavioral changes in mouse offspring exposed to a maternal high-fat diet

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Abstract:

A maternalhigh-fat diet (mHFD), characterized by a high Omega-6/Omega-3 (n-6/n-3) polyunsaturated fatty acids (PUFA), is associated with increased systemic inflammation and oxidative stress, leading to a sub-optimal intrauterine environment and increased risks for neuropsychiatric disorders in the offspring. We hypothesize that prenatal Omega-3 supplementation, by buffering inflammation, oxidative stress and re-balancing n-6/n-3 PUFA, might prevent the negative effects of mHFD.

Female C57Bl6 mice received either ahFD (26% carbohydrate, 16% protein, 58% fat; n-6/n-3 ratio=120) or a control diet (CD: 73% carbohydrate, 16% protein, 11% fat; n-6/n-3 ratio=7) for 4 weeks. Half of the females were then supplemented with Omega-3 (n-6/n-3=1) for 6 weeks, while the other half of the females were kept on the assigned diets until delivery. Maternal anxiety-like behaviors were tested through the marble burying test on gestational day (GD) 15. A subset of pregnant females was sacrificed at GD 17.5 to assess fetal brain fatty acid composition, while the remaining dams delivered and their pups underwent the homing test to evaluate early sensory-motor and associative capabilities.

Preliminary results indicate that mHFD increased maternal anxiety-like behaviors, as shown by a higher number of marbles buried. Fatty acids analysis on fetal brains revealed increased n-6 PUFA and decreased n-3 PUFA in fetuses exposed to the mHFD, while prenatal Omega-3 supplementation prevented this imbalance. In the homing test, Omega-3 supplementation increased the time spent in the nest in female offspring exposed to mHFD.

Overall, data indicate negative effects of a dysbalanced mHFD: changes in PUFA ratio in the maternal diet led to a disruption in the behavior of both mother and offspring and were reflected in fetal brain lipid composition. These findings underscore the critical impact of unbalanced maternal diets during pregnancy on the neurobehavioral development of the offspring, highlighting the importance of nutritional interventions to promote brain health.

Keywords: nutrition, maternal obesity, brain development, omega-3 PUFA

Oral Presentations

S9-OP7

Brain-specific and time-dependent KCNQ1 knockout leads to cognitive and metabolic alterations in mice

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Abstract:

Background and objectives: Insulin signaling, a well-established pathway involved in maintaining metabolic homeostasis through its glucose-lowering activity, has recently been implicated in brain function. This emerging role is supported by the high expression of insulin receptors in the central nervous system, including the Insulin Receptor and Insulin-like Growth Factor 1 Receptor. Recent research highlights insulin's multifaceted role in the brain, affecting neuronal patterns, memory formation, and neurogenesis. Abnormal insulin levels are observed in neurodegenerative conditions like Alzheimer's Disease and psychiatric disorders such as obsessive-compulsive disorder. Among several factors, KCNQ1 is emerging as a key modulator of insulin signaling cascades in the central nervous system. KCNQ1 is a voltage-dependent potassium channel capable of modulating local insulin secretion by neurons. Kcnq1 constitutive knock-out (KO) mice exhibited a diabetic phenotype accompanied by repetitive behaviors and reduced cognitive flexibility. Dysregulated central insulin signaling may underlie obsessive-compulsive traits, behavioral rigidity, and deficits in memory and learning. Our aim is to distinguish the mechanisms of central and peripheral insulin dysregulation by investigating the role of KCNQ1 in the brain as a candidate molecule influencing insulin signaling. To achieve this, we used a conditional KO mice model for kcnq1 in the brain, inhibiting KCNQ1 expression only in the brain and during specific time windows (adolescence and adulthood).
Methods: Male and female KCNQ1fl/fl NesCreERT2+ and KCNQ1fl/fl NesCreERT2- mice received Tamoxifen (62 mg/Kg) or Vehicle injections. We assessed metabolicparameters (hyperglycemia and insulin resistance, fluid intake, lipid vs. carbohydrate metabolism, and energy expenditure via automated metabolic cages). Cognitive domains were evaluated through T-maze (spontaneous alternation), attentional set-shifting task (asst, cognitive flexibility), Barnes maze (spatial memory), and marble burying (repetitive and compulsive-like behavior).
Results: The absence of brain KCNQ1 impacted lipid metabolism during adolescence but did not affect metabolism in adulthood. Notably, conditional knockout mice exhibited attention deficits and increased compulsive behavior.
Discussion: Brain-specific deficiency of KCNQ1 impacted metabolic regulation solely during adolescence, yet manifested impaired attention and increased compulsive behavior across adolescence and adulthood. These findings underscore KCNQ1's pivotal role in central insulin signaling modulation, shedding light on potential mechanisms underlying neurobehavioral disorders.

Keywords: Insulin, Cognition, Metabolism, Mice, KCNQ1

Oral Presentations

S9-OP8

Role of the glucose transporter GLUT3 in neuronal energy metabolism and somatosensory cortex cognitive functions

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Abstract:

Background and Objectives Brain energy metabolism has gained renewed interest due to its crucial role in normal brain function (learning and memory) and in neuropathological processes (e.g. Alzheimer's disease). Understanding how neuronal energy demands are met is essential for deciphering physiological and pathophysiological processes. Energy substrate transporters, such as mono-carboxylate and glucose transporters, are critical components of neuronal energy supply. Our study aimed to evaluate the impact of reducing the expression of the neuronal glucose transporter GLUT3 on metabolic parameters in vitro, and on certain cognitive functions in vivo.

Methods: A first step was conducted in vitro using primary cultured neurons. We selected the most effective shRNA sequence (out of two tested) to reduce GLUT3 expression while preserving neuronal lactate transporter MCT2 expression. We also assessed the vector's toxicity to determine the optimal dose-response combination. Finally, we evaluated the impact of GLUT3 downregulation on several neuronal energy parameters in vitro. The second step was carried out in vivo to assess the effect of reduced GLUT3 expression on cognitive functions associated with the primary somatosensory cortex (S1BF). For this purpose, we used a textured novel object recognition (tNOR), using the whisker-to-barrel system, to evaluate the role of the neuronal GLUT3 in memory formation within S1BF. Memory formation independent of the S1BF was evaluated using a visual task.

Results: In vitro results show that reducing GLUT3 by approximately 80% at the protein levelhad no significant effect on several key parameters of neuronal energy metabolism (ATP production, mitochondrial respiration, glycolysis) or on glutathione implicated in the pentose phosphate pathway. In vivo, reducing GLUT3 by approximately 38% at the protein level in the S1BF region had no impact on memory task involving the S1BF. This result contrasts with the memory impairment observed when MCT2 was downregulated with a similar approach in the same cortical region.

Discussion: Our work suggests that i) GLUT3 expression in vitro is not a limiting factor for sustaining neuronal energy metabolism; and ii) in vivo, neuronal GLUT3 expression is less critical than MCT2 for supporting cognition and brain activation during tasks involving the whisker-to-barrel system and the somatosensory cortex.

Oral Presentations

S9-OP9

Increased activation of glucagon-like peptide-1 signaling mediates maternal high-fat induced oxidative and inflammatory response changes in diet naïve male offspring.

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Abstract:

The mechanistic link between maternal high-fat feeding and post-natally induced sex-dependent neurochemical alterations in the diet-naïve offspring's brain is not completely understood. In this study, we examined the molecular underpinnings of quercetin-3-o-rutinoside (QR) and glucagon-like peptide 1 (GLP-1) regulatory effects on high-fat diet (HFD) induced neuroinflammatory and/or biochemical changes in direct consumers and diet-naïve descendants. Pregnant rats (previously fed a normal diet (ND) or 45% hFD) were maintained on supplemented chow (plus 150mg/kg QR) - ND/QR, hFD/QR throughout gestation. Subsequently, the animals were sacrificed, and brain samples were processed for expression of TNF-A, GLP-1, MDA and GSH/NO content. The data show that chronic consumption of hFD by dams failed to alter significantly the brain TNF-A levels in dams and offspring at postnatal day (PND) 21. However, a non-significant increase in TNF-A levels that appears suppressed by QR was observed in the female offspring rats ofhFD-fed dams at PND 28. Surprisingly, hFD-fed dams exhibited non-significant increase in brain MDA levels accompanied by time and sex-dependent changes in lipid peroxidation in their progenies. NO and GSH levels were not directly affected by maternal hFD treatment, whereas brain GSH concentration increased significantly in the female offspring of hFD-fed dams at PND 28. Moreover, GLP-1 expression significantly increased in dams that received QR supplemented chow, but not in the offspring. In conclusion, the current findings indicate that maternal consumption of 45%hFD only produced low-grade inflammatory and biochemical changes in the brain which can potentially trigger neuroinflammatory changes in the progenies

Key Words: high fat diet, Neuroinflammation, Glucagon-like peptide-1, oxidative stress, Sex dependent

Oral Presentations

S9-OP10

Anxiety, depression and diet in adolescents with type 1 diabetes in Rabat city-Morocco.

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Abstract:

Background: Type 1 diabetes (T1D) is a chronic condition marked by insulin deficiency due to pancreatic beta cell destruction. It accounts for 5-10% of global diabetes cases, primarily affecting children and adolescents. T1D is associated with risks of acute and long-term complications, as well as mental disorders such as depression and anxiety. Links between diet, mental disorders and T1D are consequently gaining interest in nutritional psychiatry.

Objective: This study aims to describe the diet of adolescents with T1D, to examine the existence of anxiety and depression among them, and to study the links between these different concepts.

Methods: This is a descriptive cross-sectional survey of 104 adolescents with type 1 diabetes, aged 10 to 19 years old and treated in health care establishments in the city of Rabat in Morocco. Data was collected using a questionnaire including general patient questions, anthropometric measurements, assessment of nutritional intake, and of anxiety and depression using adapted psychological tests. Data analysis was carried out using Excel and SPSS software.

Results: The results of the study highlight that only 8% of adolescents had an HbA1c level below 7.5%, indicating inadequate glycemic control among the majority of participants. High to very high anxiety was found in 60% of participants. Additionally, the prevalence of anxiety was found to be similar in both sexes. When it comes to depression, the study found that 31% of adolescents had mild levels of depression, while 5% had very severe depression. The results showed that boys were more likely to suffer from depression than girls were. In addition, in our study, anxiety and depression were found inversely correlated with dietary intake of certain vitamins and minerals such as vitamin B1 ($r=-0.93$; $p<0.05$) and zinc ($r=-0.70$; $p<0.05$), respectively.

Discussion: In summary, these findings underscore the need for comprehensive mental health support for children and adolescents with T1D. Additionally, incorporating nutritional considerations is crucial for preventing and managing these disorders effectively.

Key words: Type 1 diabetes, Nutrition, Anxiety, Depression, Adolescents.

Oral Presentations

S9-OP11

Impact of Vitamin a Modulation on Biochemical and Behavioral Abnormalities in a Valproic Acid-Induced Autism Model in Wistar Rat offspring

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Abstract:

Autism Spectrum Disorder (ASD) is a neurodevelopmental disorder that could be caused by genetic and environmental factors, like prenatal nutrition. This study aims to investigate the influence of Vitamin A deficiency (VAD) on ASD-like behaviors and the effect of Vitamin A supplementation. To this end, twenty-five female Wistar rats were divided into five groups: controls, VPA, VAD, VPA+VAD, and VPA+Vitamin A Supplementation (VAS). Neurodevelopmental and behavioral assessments were conducted on the offspring two months after birth, using tests such as Rollover, Grip, Negative Geotaxis, Gait, Three Chamber, and Open Field Tests. VPA exposure increases muscular endurance and ASD-like behaviors, such as social deficits and hyperactivity. VAD was found to significantly impair motor coordination and further exacerbate the behavioral symptoms induced by VPA exposure. In contrast, Vitamin A supplementation ameliorated several of the neurodevelopmental and behavioral deficits observed in the VPA and VAD groups. In the same hand, dosages of acetylcholine, acetylcholine esterase, malondialdehyde (MDA), nitric oxide (NO), catalase, superoxide dismutase (SOD), and glutathione were performed. The results showed that VPA and VAD significantly decreased acetylcholine levels and increased oxidative stress markers (MDA and NO), while reducing the activity of antioxidant enzymes (catalase, SOD, and glutathione). Vitamin A supplementation helped restore acetylcholine levels and antioxidant enzyme activities, while also reducing oxidative stress markers. These findings suggest that Vitamin A deficiency during pregnancy exacerbates both the behavioral and biochemical abnormalities associated with ASD, while Vitamin A supplementation offers a potential therapeutic strategy to mitigate these effects. This study highlights the critical role of adequate prenatal nutrition in reducing ASD risk and improving neurodevelopmental outcomes in offspring.

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The SONA 2025 Conference

Poster Abstracts

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Poster Presentations

Session 1

S1-01

Generation of Induced Pluripotent Stem Cell Model from Peripheral Blood Mononuclear Cells of Centenarians of Northern Nigerian Origin

Abdulrahman Alkhamis Idris, *Mahmoud Bukar Maina[1,2], Zaid Muhammad[1,2], Larema Babazau[2,4], I.G. Bako[3], UmarAdam[3], Dawoud Usman[2], Abdulrahman Alkhamis[1,2,3]*
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Abstract
Centenarians represent a unique population with exceptional longevity and resistance to age-related diseases, offering a valuable lens through which to explore the molecular drivers of aging and health span. Despite the critical need for diversity in biomedical research, induced pluripotent stem cell (iPSC) models from African populations, particularly centenarians, remain nonexistent. To address this gap, our work aims to generate iPSC lines from the peripheral blood of centenarians of Nigerian origin. Peripheral blood mononuclear cells (PBMCs) were isolated and enriched for erythroid progenitor cells (EPCs), which were expanded and characterized using differentiation markers CD71 and Glycophorin A using fluorescence microscopy and qPCR. Rigorous mycoplasma testing validated the health and reliability of the cultures. The EPCs are currently being used to generate the iPSCs. These iPSC models derived from an underrepresented population offer a promising platform for exploring genetic and cellular factors contributing to longevity and resistance to disease, advancing personalized medicine and aging research.
Keywords: Induced Pluripotent Stem Cells (iPSCs), Peripheral Blood Mononuclear Cells (PBMCs), Centenarians, Erythroid Progenitor Cells (EPCs), Northern Nigerian Population.

S1-02

Simvastatin Had a Barrel-Specific Neuroprotective Effect Against Alcohol-Induced Damage of the Somatosensory Posterior Medial Sub-Field Barrels of Adolescent Mice

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Abstract
Alcohol is commonly used and abused by adolescents. The rising prevalence of alcohol-related diseases and disabilities and governmental costs necessitate investigating interventions that could

protect neurons against alcohol-related damage. Alcohol causes neurodevelopmental, neurobehavioral, neurocognitive, and social problems due to the negative effects of chronic use on the structure and function of neurons. The antioxidant properties of Simvastatin, an FDA-approved drug for lowering blood cholesterol levels, seem promising in preventing neurodegeneration. We evaluated the effect of Simvastatin on the sizes of the posterior medial barrel sub-field (PMBSF) of the somatosensory cortex and on the morphometry of the sciatic nerve of adolescent mice that were FDA-approved administered alcohol. Four-28-day-old C57BL/6J male and female mice were administered 20% alcohol (i.p.), 5 or 10mg/kg Simvastatin orally followed by 20% alcohol (i.p.) or the controls (i.e. 5 mg/kg Simvastatin only or non-treated) for 28 consecutive days. The sizes of the 27-PMSBF barrels and the morphometry of the sciatic nerve were assessed. Alcohol did not significantly reduce the mean areas of the PMBSF barrels, enclosure, or septal portion in both sexes. However, the barrel-to-barrel comparison revealed alcohol toxicity on specific barrels in specific rows and arcs of the PMBSF barrels. Both concentrations of Simvastatin were effective against alcohol-induced damage on those specific barrels. For the sciatic nerves, the axonal density and myelination were significantly affected by alcohol in both sexes and Simvastatin was also effective against the onset of alcohol nerve damage. These observations may explain the reasons for the sensory-motor delays often seen in alcoholics owing to the delay in sensing, relaying, processing, and interpreting sensory information. Simvastatin seems to have the ability to protect against the damaging effect of alcohol and this may be beneficial in reducing the prevalence of alcohol-related diseases or disabilities, especially in adolescents that are prone to abusing alcohol.

Poster Presentations

S1-03

Neuroscience Education as a Catalyst for Mental Health Awareness and Societal Well-being in Nigeria

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Abstract

Neuroscience education plays a crucial role in shaping societal understanding of brain function and mental health. Our teaching and public awareness initiatives focus on equipping high school students and teachers in Nigeria with fundamental neuroscience knowledge, covering key cognitive processes such as learning, memory, cognitive control, motivation, and social-emotional regulation. By deepening public understanding of how the nervous system governs behavior, decision-making, and overall well-being, neuroscience education serves as a powerful tool for fostering cognitive resilience and promoting healthier life choices. Mental health challenges, including rising substance abuse, increasing crime rates, and youth-related psychiatric disorders, have become major public health concerns in Nigeria. A strong foundation in neuroscience can empower students with the knowledge to understand addiction, emotional regulation, and self-control factors that are closely linked to academic success and social stability. Additionally, educating teachers, parents, and policymakers on brain function and its impact on mental well-being will contribute to a more informed and proactive approach to mental health challenges. Crucially, neuroscience education should be integrated into educational policy, emphasizing the role of physical health, exercise, sleep, and nutrition in maintaining cognitive and emotional stability. Public engagement between neuroscience researchers, educators, parents, and policymakers is essential to harnessing neuroscience knowledge for societal progress. From 2017 till date, we have been involved in outreach and awareness programs on brain health, with support from IBRO/DANA, IBRO, MDS, and TReND. A well-informed society, grounded in neuroscience education, will be better equipped to address mental health challenges and foster a healthier, more productive population.

Keywords: Neuroscience Education, Mental Health, High School Students, Public Awareness, Policy Integration.

Poster Presentations

S1-04

Sexual Dimorphism in Sleep Disturbances in Parkinson's Disease: A Systematic Review and Meta-Analysis

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Abstract
Sleep disturbances such as insomnia, REM sleep behavior disorder (RBD), and excessive daytime sleepiness (EDS) are debilitating non-motor symptoms of Parkinson's Disease (PD), often emerging early and worsening over disease progression. Evidence suggests sex differences influence the prevalence and severity of these disorders, potentially through variations in sleep regulatory mechanisms, homeostatic, circadian, and motivational processes. This systematic review and meta-analysis synthesize findings on sex-specific sleep regulation in PD, examining the biological and clinical implications of sexual dimorphism. A systematic search across PubMed, Web of Science, and Scopus up to 2024 was conducted, with data extracted and quality-assessed by two reviewers. Where possible, a meta-analysis using a random-effects model was performed to assess sex-based prevalence rates. Preliminary findings highlight the need for sex-specific approaches in managing PD-related sleep disorders and showcase gaps for further research to enhance therapeutic strategies.
Keywords: Parkinson's Disease, sleep disorders, sex differences, systematic review, meta-analysis, circadian regulation, homeostasis, motivation.

S1-05

Elucidating the complex protein-protein interactions in Huntington's disease

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Abstract
Huntington's disease (HD) is a polyglutamine neurodegenerative disorder characterized by a progressive loss of a selective vulnerable neuronal network in the Medium Spiny Neurons (MSNs) of the striatum, due to an expansion of the CAG trinucleotide repeat (CAG >36) in exon 1 of the huntingtin gene which codes for a mutant huntingtin protein (mHTT). Recruitment of mHTT into aberrant Protein-Protein Interactions (PPIs) disrupts its normal functions and forms toxic aggregates that interfere with critical cellular processes, exacerbating neurodegeneration. The altered PPIs affect pathways involved in transcriptional regulation, proteostasis, and mitochondrial function. By integrating single-cell RNA sequencing (scRNAseq) and PPIs network analyses from striatal tissues

we identified thousands of differentially expressed genes across various striatal cell types, including MSNs, astrocytes, and oligodendrocytes. Notably, many cell type-specific genes exhibited bidirectional dysregulation: repression in their primary cell type and de-repression in other striatal cell types. These findings highlight the interplay between dysregulated gene expression and perturbed PPIs in HD pathogenesis, offering novel insights into potential therapeutic targets.

Keywords: Huntington's disease, Protein-protein interactions, scRNAseq, Striatum, Neurodegeneration.

S1-06

Inhibited FSH-induced Alzheimer's disease in rodent models down-regulated il-6 gene expression

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Abstract

Background and objectives: Alzheimer's disease (AD) pathogenesis is multifactorial comprising genetic, hormonal, and environmental factors. Studies have shown that increased luteinizing hormone levels contribute to the occurrence of AD pathologies. Interleukin-6 (IL-6) gene-expressed protein is one of the pro-inflammatory cytokines that create a concourse between cognitive dysfunction and hippocampal neurodegeneration in AD. This study was aimed at investigating IL-6 gene expression in the hippocampus by inhibiting follicle stimulating hormone in the Alzheimer's disease state.

Methods: A total of 40 Albino Wistar rats (250- 300 grams) were used for this study. The rats were randomly assigned to 4 groups with a total of 10 rats per group. Group 1 served as control (water and feed ad libitum) Group 2 (AlCl3 only 100 mg/ kg body weight for 28 days); Group 3 (AlCl3 100 mg/kg body weight for 28 days and post-treated with Estradiol 3 mg/kg for 21 days); Group 4 (Estradiol only 3 mg/kg body weight for 21 days). AD was confirmed in groups 2 and 3 with neuro-behavioral assessment standards. Thereafter, the hippocampi were extracted and homogenized for the analysis of IL-6 gene using reverse-transcriptase polymerase chain reaction (RT-PCR). Results: Inhibiting FSH levels by estradiol administration downregulated hippocampal IL-6 gene expressed protein. This was demonstrated in the AD experimental group post-treated with estradiol (3 mg/kg) which showed a statistically significant decrease ($P<0.05$) in hippocampal IL-6 gene expression when compared to the AD-only group.

Discussion: LH levels have been reported to be increased in women with AD. Estradiol, a hormone produced during reproductive years, was employed to inhibit FSH, and this was to highlight the significance of LH alterations in the management of AD by down-regulating FSH levels in the AD-induced rats. IL-6 gene-expressed protein is upregulated in AD pathology and from the findings in

this study, there was downregulation of IL-6 gene-expressed protein, suggesting the importance of investigating FSH levels in AD management.
Keywords: AlCl3: Aluminum Chloride, AD: Alzheimer's disease, IL-6: Interleukin 6, RT-PCR: Reverse transcriptase polymerase chain reaction

S1-07

Adaptation and Evolution of Pain Mechanisms: Insights from Testudines

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Abstract
Background: Nociception is an important sensory process for the detection of potentially damaging stimuli. Studies on the evolution and adaptation of nociceptive systems can identify potential targets for analgesic drugs and pain management strategies. Phylogenetically, testudines are ancient animals and therefore ideal models for studying the evolution and adaptation of pain mechanisms. The aim of this study was to investigate the involvement of monoamine neurotransmitters and voltage-gated sodium ion channels on nociception in Speke's hinge-back tortoises (Kiniskys spekii) and marsh terrapins (Pelomedusa subrufa). Methods: Twenty-four male and female Speke's hinge-back tortoises weighing between 600-1000 g were used for the experiments. Clonidine, yohimbine, morphine, and pethidine were used to investigate the involvement of monoaminergic neurotransmitters. ICA 121341 (selective blocker for Nav1.1/Nav1.3), NAV 26 (selective blocker for Nav1.7), and A803467 (selective blocker for Nav1.8) were used to investigate the involvement of Nav1.3, Nav1.7, and Nav1.8 respectively. The analgesiometric tests used were the formalin, capsaicin, and hot plate tests. Pain-related behaviour was recorded and analysed. Results: The agonists and ion channel blockers used except ICA 121341 have significant antinociceptive effects in the testudines like most animals studied. There are differences in behavioural responses shown by testudines compared to other animal models of pain in the nociceptive tests used. In the formalin test, testudines express monophasic pain behaviour while most animals studied show biphasic pain behaviour. In addition, the animals undergo desensitisation in the capsaicin test. They respond to higher temperatures in hot plate test compared to other animal models. Conclusion: Overall, the results show that ion channels and monoamine neurotransmitters involved in nociception seem to be functionally conserved through the evolution of vertebrates. The differences in behavioural responses to the nociceptive tests could be attributed to the adaptation of the animals to their ecosystems.
Keywords: Pain, Nociception, Evolution, Sensory, Testudines

Poster Presentations

S1-08

The Role of CHCHD2 in Mitochondrial Dysfunction in Parkinson's Disease Pathology: A multi-Omics approach

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Abstract
Mitochondria are essential organelles responsible for producing the majority of cellular ATP through oxidative phosphorylation, a process crucial for neurons to maintain membrane potential and facilitate synaptic transmission. Mitochondrial dysfunction is a hallmark of numerous diseases, including neurodegenerative disorders, including Parkinson's disease (PD), emphasizing the urgent need for novel therapeutic strategies targeting mitochondrial pathways. Disruption of protein-protein interactions (PPIs) within mitochondria can lead to impaired function and contribute to disease progression. Recent studies have implicated the inner mitochondrial membrane protein, CHCHD2, in critical processes such as oxidative phosphorylation and mitochondrial dynamics. Mutations in CHCHD2, particularly the T61I variant, have been linked to familial forms of PD and other neurodegenerative pathologies, yet the mechanisms by which these mutations affect mitochondrial function remain unclear. In this study, we explored the role of CHCHD2 in mitochondrial function at the single-cell level by utilizing RNA sequencing (ScRNA-seq), along with proteomic analyses conducted in our laboratory. Our results revealed altered PPIs involving CHCHD2, suggesting its role in safeguarding neuronal cells against oxidative stress and bioenergetic failure. Specifically, we identified interactions between CHCHD2 and proteins involved in the electron transport chain, reactive oxygen species detoxification, and mitochondrial fission/fusion dynamics. In addition, gene expression analysis indicated that CHCHD2 mutations lead to dysregulation of genes associated with mitochondrial respiration, apoptosis, and oxidative stress response. This Study provides an integrative approach for a better understanding of the molecular mechanisms driving PD and highlights novel targets for therapeutic intervention, facilitating future drug discovery efforts aimed at neuroprotection in PD and related neurodegenerative diseases.

S1-09

Glutathione peroxidase 3 is a potential biomarker for konzo

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Abstract

Konzo is a neglected paralytic neurological disease associated with food (cassava) poisoning that affects the world's poorest children and women of childbearing ages across regions of sub-Saharan Africa. Despite understanding the dietary factors that lead to konzo, the molecular markers and mechanisms that trigger this disease remain unknown. To identify potential protein biomarkers associated with a disease status, plasma was collected from two independent Congolese cohorts, a discovery cohort (n = 60) and a validation cohort (n = 204), sampled 10 years apart and subjected to multiple high throughput assays. We identified that Glutathione Peroxidase 3 (GPx3), a critical plasma-based antioxidant enzyme, was the sole protein examined that was both significantly and differentially abundant between affected and nonaffected participants in both cohorts, with large reductions observed in those affected with konzo. Our findings raise the notion that reductions in key antioxidant mechanisms may be the biological risk factor for the development of konzo, particularly those mediated through pathways involving the glutathione peroxidase family.

S1-10

The Effects of Argan Oil and Cannabidiol in 6-OHDA Animal Model of Parkinson's Disease as a Future Perspective Treatment

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Abstract

The Argania spinosa from southwestern Morocco has fruit surrounding tough nuts containing argan oil seeds. Parkinson's disease is a progressive neurodegenerative disease characterized by motor and nonmotor symptoms. The symptoms are result of the death of dopaminergic neurons in the substantia nigra pars compacta (SNc). Evidence suggests the involvement of oxidative stress and the overproduction of reactive oxygen species (ROS). Until the present project, no evidence has been correlating argan oil to Parkinson's disease. However, evidence suggests the walnut in its aqueous form could decrease ROS and NOS generation, limit the reduction of striatal dopamine, limit the MPTP-induced movement problems in Parkinson's disease mouse models, and also improve memory

functions. The results suggest a significant increase of live cells in the CA1-CA3 mice hippocampus. The cannabidiol (CBD) reduces ROS production. The primary astrocytes exposed to lipopolysaccharide (LPS) treatment with CBD could decrease the TNF-α, IL-6, and ROS levels. However, there was no evidence of an effect of ROS levels in primary astrocytes exposed to LPS treated with CBD after the co-treatment SR141716A, suggesting the CBD could not be mediated directly through CB1 receptors. The project's central aim is to investigate the possible benefits of argan oil and CBD in Parkinson's disease-like animal models for a potential Parkinson's disease treatment. Through the Parkinson's disease-like models, injected with 6-hydroxydopamine (6-OHDA) into the left MFB, it will investigate the effects of treatment of argan oil and CBD in different concentrations investigating the ROS, TNF-α, and IL-6 levels, the limit the reduction of striatal dopamine, limit of movements and memory impairment through Novel Object Recognition Test, The Beam-Walking Test, Rotarod Test, Morris Water Maze, morphological examination, striatal dopamine analyze, measuring and quantification ROS levels and cytokine concentration and Western Blotting. Through CB1 knock-out mice, the molecular mechanisms underlying CBD effects will be investigated by measuring and quantifying ROS levels and cytokine concentration. This project's results will not only elucidate the molecular mechanisms underlying CBD effects through CB1 receptors, but it could also increase the argan oil and CBD knowledge in Parkinson's disease and be a potential treatment perspective.

Keywords: argan oil, cannabidiol, Parkinson's disease.

S1-11

The Role of Apolipoprotein E (APOE) in Alzheimer's disease

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Abstract

Alzheimer's disease (AD), a degenerative neurological condition, is the most common type of dementia affecting people over 65 years of age. The prevalence of AD is rising quickly because of extended lifespans, and by 2050, there will be roughly 115 million AD patients worldwide. The current therapies can only delay the worsening of symptoms and slow down the course of AD. Findings have initiated the future development of apolipoprotein E (APOE)-targeted AD therapeutics. The present study aimed to review the latest developments in APOE biology and its mechanisms in AD pathogenesis through pathways dependent on Aβ. The three APOE isoforms are APOE4, APOE3, and APOE2. The APOE4 raises the risk of AD by causing earlier and more abundant amyloid pathology and impairs several aspects of normal brain functions. APOE4 affects the production, clearance, and aggregation of Aβ, leading to amyloid plaque formation. It also has other effects, like disruption of the

blood-brain barrier, increased tau hyperphosphorylation to neurofibrillary tangle, neuroinflammation, and mitochondrial and synaptic dysfunctions. In summary, APOE4 contributes to AD pathogenesis, mainly through pathways dependent on amyloid beta. Therefore, APOE4-targeted therapy could be used for Alzheimer's disease treatment.

Keywords: Alzheimer's disease, Pathogenesis, Apolipoprotein E, Amyloid beta, and Mechanisms.

S1-12

SLC44A1 expression in oligodendroglial cells during aging and in Alzheimer's Disease

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Abstract

Background: Alzheimer's disease (AD) is a progressive neurodegenerative disorder that mainly affects people over age 65. AD is characterized by the accumulation of amyloid plaques and neurofibrillary tangles, associated with the degeneration of cholinergic neurons. Recently, several evidence have also highlighted a massive loss of white matter and myelin degeneration in AD. Myelin is formed by the membrane compaction of a specific type of glial cells: the oligodendrocytes. With age, oligodendrocytes also show a decline in myelin formation and remodeling. Furthermore, transcriptomic analysis of AD brain patients and animal models has shown a dysregulation within the oligodendroglial cell lineage, including in their metabolic pathways (such as lactate and choline transports). Of interest, we identified that SLC44A1, a specific choline transporter highly enriched in the oligodendroglial cell lineage and dysregulated in aging and in AD at the transcriptomic level. Objectives: The main objective of our study is to check whether SLC44A1 is expressed within the oligodendroglial cell lineage and to determine if it is dysregulated in aging and in AD. Methods and results: The results of our immunohistochemistry study show that SLC44A1 is highly expressed in oligodendroglial cells in young spinal cord and brain sections, including in the hippocampus a crucial area involved in memory. Western blot analysis on brain and spinal cord tissues of wild-type mice at three different ages (P6, P21, and P60), did not show any significant difference in SLC44A1 expression levels during developmental myelination. Discussion: Other studies and ours indicate that SLC44A1 is expressed in oligodendrocytes and in myelinated fiber tracts. Due to its high expression in oligodendrocytes in the hippocampus, SLC44A1 could play a role in cognitive mechanisms and could be involved in the progression of AD. Additional investigation, using older tissues and AD model tissues, is required to check whether SLC44A1 expression changes with age and in AD.

Keywords: Oligodendroglial cells, SLC44A1, Myelin, Choline, Alzheimer's Disease.

Poster Presentations

S1-13

Bi-allelic PRRT2 variants may predispose to Self-limited Familial Infantile Epilepsy

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Abstract
Heterozygous PRRT2 variants are frequently implicated in Self-limited Infantile Epilepsy, whereas homozygous variants are so far linked to severe presentations including developmental and epileptic encephalopathy, movement disorders, and intellectual disability. In a study aiming to explore the genetics of epilepsy in the Sudanese population, we investigated several families including a consanguineous family with three siblings diagnosed with self-limited infantile epilepsy. We evaluated both dominant and recessive inheritance using whole exome sequencing and genomic arrays. We identified a pathogenic homozygous splice-site variant in the first intron of PRRT2 [NC_000016.10(NM_145239.3): c.-65-1G > A] that segregated with the phenotype in this family. This work taps into the genetics of epilepsy in an underrepresented African population and suggests that the phenotypes of homozygous PRRT2 variants may include milder epilepsy presentations without movement disorders.

S1-14

Octadecaneuropeptide promotes cell migration in cultured rat astrocyte via ODN metabotropic receptor and calcium signaling pathway

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Abstract
Astrocytes specifically synthesize and release endozepines, a family of regulatory peptides, including the octa-deca-neuropeptide (ODN). ODN promotes proliferation and prevents oxidative damage-

induced cell death apoptosis on both neurons and astrocytes. However, little is known regarding the effects of ODN on neuronal cell migration. Migration is an essential characteristic of cells that occurs during many physiological and pathological processes. The purpose of the present study was to investigate the potential effect of ODN in the induction of astrocytes migration, which is critical for the formation of neurons during development and maintaining brain homeostasis. To investigate the effect of ODN on cell migration, we used the wound healing technique. Then the wound edges were remicrographed after treatment. Images of migratory cells from the scratched boundary were observed at 48h with different concentrations of ODN and using an inverted microscope. We measured astrocyte cell viability following injury with the fluorescein diacetate (FDA). We therefore investigated the effect of ODN on intracellular Ca^2+ levels. We studied the metabotropic receptor and the signaling pathway involved in the action of ODN on the induction of astrocyte cell migration. Finally, we tried to provide a new insight into the effect of ODN on mTOR gene expression for cell migration through RT-qPCR. The result in the wound healing assay, the representative photomicrograph images of cultured astrocytes showed that administration of graded concentrations of ODN increased cell migration in a dose-dependent manner and adhesion. We have also observed that the effect of ODN that was abrogated by the metabotropic ODN receptor antagonist cyclo1?8[DLeu5] OP and agonist, we have also found that the metabotropic ODN receptor, which is positively coupled to adenylyl cyclase in astrocytes, activates calcium-signaling pathways. Downstream of calcium signaling pathways, ODN induced ERK phosphorylation, which in turn activated the expression of the anti-apoptotic TOR gene. This effect of ODN involves the activation of its metabotropic receptor associated with intracellular transduction pathways PKA, PKC and MAPK / ERKs; The study of signaling pathways demonstrates that the effects of ODN involve the activation of PKC, PKA and MAPK / ERK transduction pathways.

S1-15

Olax subscorpioidea prevented scopolamine-induced memory impairment through the prevention of oxido-inflammatory damage and modulation of cholinergic transmission

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Abstract

Background and objectives: Olax subscorpioidea oliv. is a shrub plant of the family: Olacaceae with reported usage in ethnomedicine as a nootropic agent for the management of Alzheimer's-like dementia. The aim of this study is to investigate the nootropic potential of Methanol Extract of Olax subscorpioidea (MEOS) in scopolamine-induced Alzheimer's-like dementia. Methods: Thirty male

Swiss mice, assigned into six groups (n = 8), were used for this study. Group I received distilled water, Group II received scopolamine (1 mg/kg, i.p.), Groups III-V received 25, 50, and 100 mg/kg (p.o.) of MEOS and scopolamine (1 mg/kg, i.p.), and group vi received donepezil 5 mg/kg (p.o. and scopolamine (1 mg/kg, i.p.). The animals were pre-treated with MEOS and donepezil for 14 days, and scopolamine from the 8th to 14th day. Then, it is followed by cognitive, oxidative stress, neuro-inflammation, and histology assessments. Results: Methanol Extract of Olax subscorpioidea (100 mg/kg) significantly reduced transfer latency and increased discrimination index in the elevated plus maze and novel object recognition test cognitive assessments. The same dose of MEOS, significantly reduced oxidative stress, protected endogenous antioxidants, suppressed neuroinflammation, and acetylcholinesterase (ACHE) activity. The histomorphometry study of the hippocampus revealed that MEOS prevented extensive pyknosis, karyolysis, chromatolysis, and loss of hippocampal neurons that accompanied scopolamine treatment. Conclusion: The MEOS protects against Alzheimer's-like conditions via the suppression of neuroinflammation and oxidative stress associated with scopolamine-induced amnesic behavior.

Keywords: Acetylcholine; Dementia; Neuroinflammation; Nootropics; Oxidative stress.

S1-16

Restraint Stress Exacerbates Apoptosis in a 6-OHDA Animal Model of Parkinson's disease

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Abstract

Activation of the apoptotic pathway has been associated with promoting neuronal cell death in the pathophysiology of Parkinson's disease (PD). Nonetheless, the mechanisms by which it may occur remain unclear. It has been suggested that stress-induced oxidation and potential apoptosis may play a major role in the progression of PD. Thus, in this study, we aimed to investigate the effect of subchronic restraint stress on striatal dopaminergic activity, iron, p53, caspase-3, and plasmatic acetylcholinesterase (AChE) levels in male Wistar rat model of PD induced by administration of 6-hydroxydopamine (6-OHDA) in the medial forebrain bundle (MFB). The obtained results showed that restraint stress exacerbates motor coordination deficits and anxiety in animals treated with 6-OHDA in comparison to animals receiving saline, and it had no effect on object recognition memory. On another hand, 6-OHDA decreased dopamine (DA) levels, increased iron accumulation, and induced overexpression of the pro-apoptotic factors' caspase-3, p53, and AChE. More interestingly, post-lesion restraint stress exacerbated the expression of caspase-3 and AChE without affecting p53 expression. These findings suggest that subchronic stress may accentuate apoptosis and may contribute to DA neuronal loss in the striatal regions and possibly exacerbate the progression of PD.

Poster Presentations

S1-17

Maternal Depression, Exclusive Breastfeeding and Psychomotor Development in Children 6 to 12 Months of Age at the N'DJILI General Hospital in Kinshasa, Democratic Republic of Congo

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Abstract

Background and objective: Exclusive breastfeeding has positive effects on psychomotor development while maternal anxiety and depression have negative effects. The research on the opposite effects of these conditions is insufficient in our environment or even in the general literature. The objective was to determine the links between postpartum maternal depression, exclusive breastfeeding and psychomotor development in children aged 6 to 12 months. Methods: This is a prospective cross-sectional study carried out in a preschool consultation at N'DJILI General Hospital in Kinshasa, involving 40 mother-child dyads recruited from March 21 to April 21, 2023. Maternal depression and anxiety were assessed using the Goldberg Anxiety and Depression Scales, and exclusive breastfeeding for the first 6 months of life was assessed by history-taking. Global psychomotor development quotient (GPDQ), perception and communication (PCDQ), motor (MDQ), and adaptive (ADQ) development quotients were measured using the Gensini-Gavito Infant Development Scale. Results: Exclusive breastfeeding accounted for 42.5% of subjects. Maternal depression and anxiety accounted for 37.5% and 20% of subjects respectively. G insufficient or <100 accounted for 42.5%. Mothers who had exclusively breastfed for the first six (6) months had lower anxiety and depression scores compared with those who had not exclusively breastfed p= 0.066 for anxiety and 0.149 for depression. GPDQ and ADQ were negatively correlated with child age, r=-0.382 (p=0.015) and r=-0.331(p=0.037), respectively. Maternal depression scores were negatively correlated with ADQ, PDQ, and GPDQ respectively r=-0.474 (p=0.002), r=-0.402 (p=0.010), and r=-0.348(p=0.028), and those of maternal anxiety too, with respectively r = -0.509(p=0.001), r=-0.479(p=0.002) and r= -0.354(p=0.025). In the case of exclusive breastfeeding, GPDQ<100 accounted for 35.3% (6/17) versus 47.8% (11/23) for non-exclusive breastfeeding (p=0.428). In the case of the combination of maternal depression and non-exclusive breastfeeding, GPDQ-100 reached 63.6% (7/11) versus 33% (4/12) in the case of non-exclusive breastfeeding without maternal depression and 23.1% (3/13) in the case of exclusive breastfeeding without maternal depression. Conclusions The results of this study show that exclusive breastfeeding has positive effects on early psychosocial development opposite to those of maternal depression. These results should be taken into account in practice and research related to exclusive breastfeeding in our environment.

Poster Presentations

S1-18

A cross-cultural study of affective touch in South African and United Kingdom women, using self-report and EEG/ERP methodology

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Abstract

Background and objectives: An affective touch is vital for human social development, communication, bonding, and well-being. Despite cultural differences, most research focuses on Western populations, often overlooking cross-cultural nuances. This pioneering pre-registered study explored, for the first time, how women from South Africa (SA) and the United Kingdom (UK) respond to affective touch both subjectively and neurophysiologically. We investigated cross-cultural differences in (1) subjective touch evaluations and (2) neural oscillations in response to slow (3cm/s) and fast (18cm/s) touch on C-tactile (CT) innervated skin (dorsal forearm) versus non-CT-innervated skin (palm), accounting for individual differences in touch experiences and attitudes. Methods: Thirty-six women (18 UK, 18 SA) participated. They received slow (3cm/s) and fast (18cm/s) touch on their dorsal forearm and palm. Participants rated each type of touch according to positivity and intensity. EEG recorded neural oscillations: theta, alpha, beta ERD/ERS. Questionnaires on touch experiences, attitudes, and attachment styles controlled for individual differences. Results: SA participants rated touch as more positive and less intense than UK participants. EEG data revealed stronger alpha and beta ERD in response to faster touch and palm touch across all participants. In SA participants, palm touch elicited greater beta and theta ERD and greater ERS for slow palm touch. UK participants showed effects primarily in frontal theta oscillations. Discussion: our findings reveal that cultural context significantly shapes behavioural and neural responses to affective touch. South African participants rated touch as more positive and less intense than UK participants, suggesting cultural differences in touch perception, possibly due to greater touch familiarity. Unique neural oscillatory patterns in SA participants, particularly distinct ERD/ERS responses to palm touch, indicate that sensory processing of touch varies across cultures. This research highlights that some aspects of touch are shaped by cultural norms and practices, which has important implications for psychology, anthropology, and healthcare, informing culturally sensitive approaches to therapy, caregiving, and interpersonal communication.

Keywords: affective touch; cross-cultural research; electroencephalography; neural oscillations; attachment style.

Poster Presentations

S1-19

The Impact of Depression on Cognitive Function Among Women in a Psychiatric Hospital: Analysis via the Beck Scale Short Version and the Moroccan Version of the MoCA Test

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Abstract

Background: This study sought to assess the impact of depression on cognitive function in newly diagnosed women who presented to psychiatric emergency services through the cold track for a consultation. Methods: A cross-sectional study was conducted at the Ar-Razi Psychiatric Hospital in Salé, Morocco, on 86 women newly diagnosed with depression, aged between 18 and 58 years. Depression was evaluated using the 13-item Beck Depression Inventory Short Version, while cognitive functions were assessed using the Moroccan version of the MoCA test. Data analysis was performed using SPSS v.26, employing correlation tests to explore relationships between depression and cognitive function, and one-way ANOVA to assess group differences. A confidence level of 95% and a significance threshold of 0.05 were applied. Results: Among the 86 women studied, aged 18 to 58 years, severe depression was prevalent. Women aged 38-48 years exhibited the highest prevalence of severe depression, while those with only primary education had significantly higher depression scores. Widowed women had the highest overall depression scores, and students and unemployed participants were the most severely affected groups. The correlation test revealed a negative relationship between depression symptoms and cognitive function, with higher depression levels associated with lower cognitive scores. Specifically, feelings of sadness and concentration deficits were negatively correlated, as were feelings of discouragement and executive functions. Positive correlations were observed between feelings of guilt and verbal fluency. Conclusion: The findings of this study contribute to the understanding of cognitive impairment associated with depression in women. Additionally, this research highlights the importance of assessing and addressing cognitive disorders related to depression, while promoting the development of tailored support strategies for affected individuals.

Poster Presentations

S1-20

Generation of an induced pluripotent stem cell line (BIORTCi001-A) from a healthy adult indigenous Nigerian participant

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Abstract
Genetic backgrounds influence cellular phenotypes, drug responses, and health outcomes, yet most human iPSC lines are derived from individuals of European descent, with lines from indigenous Africans particularly scarce. Addressing this gap, we generated iPSCs from dermal fibroblasts of a healthy 60-year-old indigenous Nigerian male of the Babur ethnic group using Sendai virus. The iPSC line displayed a normal karyotype, was characterized for pluripotency markers and differentiated into neural progenitor cells and astrocytes. To enhance African representation in research, this iPSC line will be available to the scientific community, with ongoing efforts focused on creating an open-access African iPSC biobank.

S1-21

Intranasal Administration of Octadeca-neuropeptide Analog Protects Dopaminergic Neurons from MPTP-Induced Neurotoxicity in a Mouse Model of Parkinson's Disease

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Abstract
Parkinson's disease (PD) is a severe neurodegenerative disorder for which existing are primarily palliative and lack curative efficacy. The development of neuroprotective agents capable of slowing or halting disease progression is crucial for advancing therapeutic strategies. Astrocytes release endozepines, such as octadecaneuropeptide (ODN), which exhibit significant neuroprotective properties. Prior research has shown that ODN protects nigrostriatal dopaminergic neurons from 1-

methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP)-induced degeneration in mice. However, its clinical application is limited by the necessity of intracerebroventricular administration. This study investigates the efficacy of CycloOP, an ODN analog, delivered via intranasal administration, to bypass the restrictive blood-brain barrier and provide neuroprotection in a Parkinsonian mouse model. A single intranasal injection of CycloOP (10ng/10ul), administered one hour after the final MPTP dose (3x 20mg/kg), significantly suppressed oxidative stress, as evidenced by reduced levels of reactive oxygen species (ROS), malondialdehyde (MDA) and protein carbonylation. CycloOP-mediated neuroprotection was associated with the restoration of key antioxidant defense enzymes, superoxide dismutase (SOD) and catalase (CAT), while blocking the activation of pro-apoptotic genes, Bax and caspase-3, in the striatum. These findings suggest that intranasal CycloOP offers potent antioxidant and anti-apoptotic neuroprotection, with efficacy comparable to its parent compound, ODN, but with the advantage of a more feasible administration route. CycloOP holds potential as a novel therapeutic candidate for PD, addressing the limitations of ODN's delivery method and opening avenues for further clinical development in neurodegenerative disease therapy.
This work was supported by a France-Tunisia CMCU-Campus France/PHC Utique 20G0826/44306YD and 24G0807/ 50283RD exchange program

S1-22

From anxiety to depression in the PTZ-induced kindling model of epilepsy: an assessment of comorbidity versus exposure to seizures

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Abstract
About 30-40% of patients affected by epilepsy have an associated psychiatric disorder. The present study was designed to characterise the sequence of occurrence of the two most prevalent comorbidities of epilepsy, anxiety and depression following kindling. Animals were divided into groups as follow: control groups, a group subjected to kindling (CKEOD+0), a group subjected to kindling which received 7 extra injections following kindled state (CKEOD+7), and a group subjected to kindling which received 14 extra injections following the kindling state (CKEOD+14). Animals were then subjected to EPM, OFT to evaluate anxiety, to SPT and FST to evaluate depression. 24h following the last behavioural test, animals were decapitated and their blood, hippocampi, prefrontal cortices (PFC) were collected to assess oxidative stress (GSH, CAT, MDA), inhibitory signalisation (GABA and GABA-T), excitatory signalisation (glutamate and EAAT-2), neuroinflammatory signalisation (IL-1β, TNF-α and TGF-1β), and HPA-axis (corticosterone and CRH) and histologi cal changes were also assessed. Results revealed that anxiety manifests before depression. The expression of the anxious

phenotype is maximum and optimum in CKEOD+7, and the depressive phenotype was maximum and optimum in CKEOD+14. Biochemical analysis demonstrated that oxidative stress was enhanced in all kindled groups both in the hippocampus and the PFC. Depressed inhibitory signalling coupled to an increased excitatory signalling pathway as well as increased CRF in hippocampi of both anxious and depressed animals and increased plasma corticosterone levels in depressed animals was shown. In addition, there was an enhancement of pro-inflammatory and a dampening of anti-inflammatory mechanisms in the hippocampus of kindled animals. Histological analysis revealed an altered hippocampal structure. These changes were, however, heightened in CKEOD+7 and CKEOD+14. Taken together, these results demonstrate that on a sequential basis, anxiety occurs before depression, thus preclinical screening of anxiolytic and anti-depressive drugs in epilepsy animal models should consider this time scale for optimum response.

Keywords: Epilepsy, comorbidities, neuroinflammation, glutamate, GABA

S1-23

An investigation on the role of oxytocin in chronic neuropathic pain in a Wistar rat model

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Abstract

Introduction: Chemotherapy-induced peripheral neuropathy (CIPN) is a dose-limiting side effect with ineffective preventative and curative treatment. Currently, only Duloxetine has been recommended as an effective treatment for CIPN, which has shown individual-dependent, short-term analgesic effects, with limiting adverse effects and poor bioavailability. The neuropeptide, oxytocin, may offer significant analgesic and anxiolytic potential, as it exerts central and peripheral attenuating effects on nociception. However, it is unknown whether the intervention administered in a model of CIPN is an effective therapeutic alternative or adjuvant.

Materials and Methods: The intervention was divided into two phases. Phase 1 aimed to induce CIPN in adult Wistar rats using the chemotherapeutic agent Paclitaxel. Mechanical (electronic von Frey filament) and thermal (acetone evaporation test and Hargreaves test) hypersensitivity testing were used to evaluate changes due to the neuropathic induction. Phase 2 consisted of a 14-day intervention period with saline (o.g.), duloextine (o.g.), or oxytocin (i.n.) administered as treatment. Following the intervention, anxiety-like behaviour was assessed using the elevated plus maze (EPM) and light-dark box protocols. Analysis of peripheral plasma corticosterone, peripheral plasma oxytocin, and hypothalamic oxytocin concentrations were assessed using ELISA assays. Results: The findings showed that we were able to successfully establish a model of chemotherapy-induced peripheral neuropathy during Phase 1, determined by the increase in mechanical and thermal nociceptive

responses following Paclitaxel administration. Furthermore, the animals treated with oxytocin displayed a significant improvement in mechanical sensitivity over the intervention phase, indicative of an improvement in nociceptive sensitivity in the presence of neuropathic pain. Animals that received Paclitaxel and treated with oxytocin also displayed significantly greater explorative behaviour during the EPM, indicative of a reduced presence of anxiety-like behaviour.
Conclusion: Our results support the hypothesis that intranasally administered oxytocin may augment the analgesic and anxiolytic effects of duloxetine in a chemotherapy induced peripheral neuropathy model in a Wistar rat. Future studies should consider administering the treatments in combination to observe the potential synergistic effects.
Keywords: Analgesia; Anxiolytic; Chemotherapy-induced peripheral neuropathy; Chronic pain; Oxytocin.

S1-24

Potential therapeutic effect of Nigella sativa oil on an animal model of Parkinson

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Abstract
Parkinson's disease (PD) is a neurodegenerative disorder marked by the progressive loss of dopaminergic neurons, with apoptosis and oxidative stress playing key roles in its pathogenesis. This study focuses on the effects of subchronic restraint stress on dopaminergic activity, iron accumulation, and the expression of pro-apoptotic markers, including p53, caspase-3, and acetylcholinesterase (AChE), in a 6-hydroxydopamine (6-OHDA)-induced animal model of PD. Findings reveal that restraint stress worsens motor coordination impairments and anxiety in 6-OHDA-treated animals while leaving object recognition memory unaffected. Additionally, 6-OHDA reduces dopamine levels, increases iron deposition, and elevates caspase-3, p53, and AChE expression. Restraint stress further amplifies caspase-3 and AChE expression, suggesting an exacerbation of apoptosis and dopaminergic neuronal loss. To identify potential therapeutic options, this study will explore the neuroprotective properties of Nigella sativa, a medicinal plant known for its antioxidant and anti-inflammatory effects. Given its reported ability to counteract oxidative stress and reduce apoptosis, Nigella sativa will be evaluated for its capacity to mitigate stress-induced exacerbation of PD pathology. The investigation will focus on its impact on apoptotic pathways and dopaminergic SIGNALLING, aiming to provide insights into novel strategies for managing PD progression.
Keywords: Parkinson disease; 6-OHDA; Nigella sativa; restraint stress; oxidative stress.

Session 2

S2-01

Cannabidiol-Mediated Neuroprotection in Amyloid Beta-Induced Alzheimer's Model of Drosophila: Behavioral and Morphological Evidence

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Abstract

Background and objectives Alzheimer's disease (AD) is a progressive and irreversible neurodegenerative disorder characterized by cognitive decline and neuropathological transformations, imposing a significant burden on individuals and healthcare systems globally. Despite ongoing research endeavors, effective treatments to halt AD progression remain elusive. Cannabidiol (CBD) is a natural compound derived from cannabis renowned for its anti-inflammatory, neuroprotective, and antioxidant properties. This study investigated the neuroprotective potential of CBD in mediating neurobehavioral and morphological changes in the Amyloid beta 42 transgenic model of AD. Methods 150 flies were grouped into five. Group 1 & 2 are negative and positive control and were exposed to 10 g of diet only, group 3 is an experimental control and was exposed to 1 mM Donepezil. Group 4 & 5 were subjected to 2 mg and 4 mg of CBD respectively for 2 weeks. Motor function, memory abilities, social interactions, and expression of amyloid beta and glial fibrillary acidic protein (GFAP) were evaluated using climbing, aversive phototaxis suppression, social space assay, and immunostaining respectively. ResultsFindings revealed a significant decrease in motor coordination (0.31 ± 0.08, P = 0.007), memory function (7.00 ± 8.52, P = 0.008), and social behavior (3.09 ± 0.51, P = 0.0008) in the positive control compared to the negative control group, accompanied by elevated Amyloid beta 42 and GFAP expression (58.50 ± 8.000, P = 0.03). However, treatment with CBD effectively mitigated these deficits. Motor function was restored in the 4 mg CBD (0.69 ± 0.08, P = 0.028), memory abilities were improved in the 4 mg CBD (63.00 ± 7.35, P = 0.007), social interaction was increased in the 4 mg CBD group (1.19 ± 0.53, P = 0.0071). Furthermore, CBD treatment reduced Amyloid beta 42 and GFAP immunoreactivity (58.50 ± 8.000, P = 0.03). Discussion This study provides compelling evidence for the therapeutic potential of CBD oil in mitigating motor and cognitive deficits and neuropathological changes associated with AD, underscoring the importance of further research into the mechanisms of action and optimization of treatment regimens for AD.
Keywords Alzheimer's disease, Neurobehavioral changes, Morphological alterations, Cannabidiol, Drosophila melanogaster.

S2-02

Neuroprotective Effect of Ononis natrix Against Lead-Induced Brain Dysfunction in Mice

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Abstract
Lead (Pb) exposure is a major public health issue, causing severe neurological damage. Available pharmaceutical treatments often have undesirable side effects, prompting increasing interest in phytotherapy solutions. This study aims to evaluate the neuroprotective effects of Ononis natrix, a plant rich in phenolic compounds with antioxidant and anti-inflammatory properties, against lead-induced brain dysfunction. Mice were divided into four groups: a control group (water only), a lead-exposed group (Pb 1 g/L via drinking water), and two lead-exposed groups treated orally with Ononis natrix extract (100 mg/kg and 500 mg/kg) starting from day 21. Behavioral tests were performed to assess memory, anxiety, and depression-like behavior. Additionally, oxidative stress analysis was conducted to measure GPx and MDA activity. The results showed that Ononis natrix administration significantly alleviated behavioral disorders and decreased oxidative stress levels. These findings suggest that Ononis natrix could be a potential source of active compounds for the development of therapeutic agents to treat neurological effects of lead intoxication.

S2-03

TeMacTM Modulates amyloid beta-fibrillation, tau proteostasis, glucose utilization and membrane fluidity: animal and cellular model

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Abstract
Background: Recent studies have proposed a new conceptualization of Alzheimer's disease (ad) as "type 3 diabetes" emphasizing the critical roles of insulin resistance, impaired glucose metabolism,

and decreased membrane fluidity in the pathogenesis as these are closely linked to the formation of senile plaques and dysregulation of tau proteostasis. Therapies that modulate these are currently being investigated for their potential role in the prevention of ad. For this purpose, medicinal plants with known antidiabetic properties are potential and promising candidates. Objectives: This study investigates the effects of a tannin-rich fraction of Terminalia macroptera (temactm) (antidiabetic) on the biochemical alterations of ad. Method: In an animal experiment, we induced ad-like alterations in rats by a high-fat diet (hfd) and exposure to alcl3 (75mg/kg bw). All animals except the cn group were fed hfd. Animals received alcl3 (75mg/kg bw) alone (ad control) or concomitantly with 400 mg/kg bw temactm or 10 mg/kg bw atorvastatin by daily gavage for 56 days. At the end of the experiment, rats were sacrificed, blood and brains were collected. The levels of amyloid fibrils, glucose, albumin, and the activities of dpp4, α-secretase, as well as markers of oxidative stress and lipid levels (cholesterol, phospholipids, and glycerophospholipids) in the brain were assessed. Furthermore, using the sk-n-sh neuronal cell line, we evaluated the effect of temactm in normalizing tau protein homeostasis that was disrupted by H2O2 treatment (250'M). Results: Our results demonstrated that temactm improves glucose utilization, increases membrane fluidity, and inhibits the formation of senile plaques. temactm not only alleviates oxidative stress but also restores crucial cellular functions associated with tau proteostasis dysregulation. Conclusion: Overall, our results support the potential of temactm as a candidate for further research in the treatment of ad.

Keywords: type 3 diabetes, temactm, amyloid fibrils, tau proteostasis and impaired glucose metabolism

S2-04

Neurotoxic Impact of Mercury Chloride in Diabetic Rats: Cognitive Deficits, Neuroinflammation, and Reduced BDNF Signaling

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Abstract

Diabetes, a metabolic disorder, is frequently associated with cognitive impairments, increased neuroinflammation, and reduced levels of brain-derived neurotrophic factor (BDNF). Concurrently, mercury chloride ($HgCl_2$), an environmental pollutant, is recognized for its neurotoxic effects, particularly through the induction of neuroinflammatory processes. The interaction between chronic hyperglycemia and prolonged exposure to $HgCl_2$ may exacerbate cognitive and neuroinflammatory dysfunctions. Therefore, the primary objective of this study was to evaluate the effect of $HgCl_2$ administration on learning, memory, and neuroinflammation, as well as on key markers such as acetylcholinesterase activity and BDNF levels in the hippocampus and prefrontal cortex.

The experimental groups included non-diabetic and diabetic rats, treated or not with $HgCl_2$ for 45

days. Cognitive functions were assessed using the Morris Water Maze (MWM), Y-maze, and novel object recognition tests. Biochemical markers of neuroinflammation (TNF-alpha and IL-6), acetylcholinesterase activity, and BDNF levels were also analyzed in the aforementioned brain regions.

The results revealed significant cognitive impairments in diabetic rats and diabetic rats treated with $HgCl_2$, with the latter showing the most pronounced deficits in spatial learning, reference memory, and working memory. Biochemical analyses demonstrated elevated levels of TNF-alpha and IL-6 in the hippocampus and prefrontal cortex of $HgCl_2$-treated diabetic rats compared to untreated diabetic rats ($P<0.05$). Additionally, acetylcholinesterase activity was significantly reduced ($P<0.05$) in these two regions, particularly in diabetic rats exposed to $HgCl_2$. Furthermore, a marked decrease in BDNF levels was observed in the hippocampus and prefrontal cortex, exacerbated by the combination of hyperglycemia and $HgCl_2$ exposure.

Chronic exposure to $HgCl_2$, in synergy with hyperglycemia, worsens cognitive impairments, neuroinflammation, and BDNF deficits, while reducing acetylcholinesterase activity. This interaction exacerbates neuronal and cognitive dysfunctions associated with diabetes.

Keywords: Microglia, Alpha-synuclein, Parvalbumin, Cerebellum, Bisphenol-A, High-Fat

S2-05

Attenuation of Scopolamine-Induced Amnesia in Mice by the Waste of the Essential Oil Extraction of Rosa damascena Flowers

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Abstract

Memory loss is an increasing global issue, particularly due to aging populations and the rise of neurodegenerative diseases. Medicinal plants, rich in bioactive compounds, are being explored as potential natural remedies for cognitive decline. Rosa damascena, a fragrant flower valued in traditional medicine, cosmetics, and perfumes, also generates waste biomass from essential oil extraction. This by-product, often discarded, is rich in bioactive compounds with untapped potential. This study aimed to evaluate the preventive effects of a hydro-ethanolic extract of Rosa damascena essential oil extraction waste (RDFORE) on scopolamine-induced amnesia in mice. To accomplish our objective, an in vivo study was conducted where RDFORE, donepezil, and memantine were

administered orally to mice one week before scopolamine injection (1mg/kg) and lasted until the end of the experiment. The effects on long-term and working memory were assessed using the Novel Recognition and Spontaneous Alternation (Y-maze) tests. The results showed that RDFORE decreased memory deficits in scopolamine-treated mice, though not as effectively as donepezil and memantine. These findings suggest that RDFORE's antioxidant properties may contribute to memory enhancement, highlighting Rosa damascena as a promising natural approach for managing cognitive decline associated with neurodegenerative conditions. Additionally, this study underscores the potential of Rosa damascena's essential oil waste as a sustainable bio-resource. Keywords: Rosa damascena, valorization, essential oil production waste, memory, scopolamine, amnesia, novel recognition test, Spontaneous Alternation test. Acknowledgment: This work was supported by the 4th Project on the Valorization of Medicinal and Aromatic Plants (VPMA4-2022/12) co-financed by the National Center for Scientific and Technical Research (CNRST) of the Kingdom of Morocco, the National Agency for Medicinal and Aromatic Plants (ANPMA) and Cadi Ayyad University of Marrakech. (2022-2025).

S2-06

Anti-inflammatory and anti-oxidative effects of vanadium on motor and cerebellar cortices of juvenile hydrocephalic mice

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Abstract
Hydrocephalus presents a significant clinical neurology challenge, manifesting complications such as neuronal degeneration, cognitive impairments and motor deficits. Ventricular shunting is the primary recourse for treatment, but it is fraught with complications such as infection and obstruction. Due to the need for alternative therapeutic modalities, vanadium, a ubiquitous transition metal known for its promising therapeutic potential in neurological conditions, has recently been studied. This study investigates the anti-inflammatory and anti-oxidative effects of vanadium on the motor and cerebellar cortices in juvenile hydrocephalic mice following treatment with two doses of vanadium. Forty juvenile mice were divided into four groups (n=10 each): control, hydrocephalus-only, low dose- (0.3mg/kg) and high dose- (3mg/kg) vanadium groups. Hydrocephalus was induced by intra-cisternal injection of kaolin, and sodium metavanadate was administered daily by intraperitoneal injection for 14 days. Neurobehavioral assessments: Inverted square grid test, pole test, Y-maze, and

Novel Object Recognition test were conducted to evaluate muscular strength, motor coordination/balance, learning and memory respectively. The cerebral motor and cerebellar cortices underwent cresyl violet staining and immunohistochemistry targeting inflammation markers, Tumor Necrosis Factor-α (TNF-α), the astrocytic marker-Glial Fibrillary Acidic Protein (GFAP) and Aquaporin-4 (AQP-4). Additionally, biochemical assays measuring TNF-α, Interleukin-1 (IL-1), Superoxide Dismutase (SOD), and Glutathione-S-Transferase (GST) activities were performed. Hydrocephalic mice exhibited significant weight loss and pronounced neurobehavioral deficits, characterized by diminished motor function and impaired cognitive function. Histological staining revealed pyknotic cells, while immunostaining revealed reactive astrocytes, increased AQP-4 and TNF-α expression. Biochemical analyses revealed increased TNF-α and IL-1 levels, accompanied by reduced SOD and GST activities in hydrocephalus-only group. These were all ameliorated in both vanadium-treated groups. In conclusion, this study highlights the promising anti-inflammatory and anti-oxidative effects of vanadium treatment in mitigating neuroinflammation and oxidative stress associated with hydrocephalus.

S2-07

Exploring the Impact of Substance Abuse on Personality and the Role of Rehabilitation in Recovery

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Abstract

Substance abuse represents a critical public health issue, deeply affecting individuals' physical health, psychological well-being, and personality traits. This study seeks to investigate how substance abuse alters personality dimensions and evaluate the effectiveness of rehabilitation in restoring affected traits. The research is divided into two parts. The first is a quantitative study assessing the impact of substance abuse on personality traits in 50 patients from the Marrakech Addiction Center. Multiple standardized tools were employed: the Drug Abuse Screening Test (DAST) to measure addiction severity, the Early Maladaptive Schema Inventory (SPI 13) to evaluate personality traits, the DEBA A/D scale for addiction severity, and the Rosenberg Self-Esteem Scale to measure self-esteem. Data were analyzed using SPSS, with mean calculations and ANOVA tests to identify significant correlations. Results revealed that substance abuse significantly impacts various personality traits, including a noticeable decline in self-esteem. The second part is a case study following a single patient through a three-month rehabilitation program at the same center. The ASSIST test (Alcohol, Smoking, and Substance Involvement Screening Test) measured addiction levels before and after rehabilitation. The patient's results showed significant improvements, including reduced dependency levels and enhanced psychological well-being. The findings highlight the detrimental effects of

substance abuse on personality traits, exacerbating conditions such as anxiety, depression, and low self-esteem. Rehabilitation, however, proves effective in mitigating addiction and restoring affected personality dimensions. The study emphasizes the importance of incorporating psychological interventions targeting personality traits into rehabilitation programs for comprehensive recovery. Despite its contributions, the study has limitations, including a small sample size for the quantitative analysis and the lack of generalizability from a single-case study. Future research should explore larger and more diverse populations to validate findings. Overall, this study underscores the profound interplay between substance abuse and personality and the transformative potential of targeted rehabilitation programs.

S2-08

Cannabidiol Alleviates Pain and Inflammation: Insights from Acute Pain Models

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Abstract
Background and objectives Cannabidiol (CBD), a non-psychoactive phytocannabinoid, has recently attracted interest due to its potential therapeutic applications in several health conditions. This study evaluated the effects of CBD in two acute pain models in mice. Methods CBD (30 mg/kg) was administered orally and compared to diclofenac sodium and a vehicle group in the writhing and formalin tests. Paw weight, paw edema, and histological analysis of paw tissues were also assessed to evaluate inflammation. Results In the writhing test, CBD significantly reduced the number of abdominal constrictions compared to the vehicle group. In the formalin test, CBD attenuated pain behaviors during both the neurogenic (phase 1) and inflammatory (phase 2) phases. These effects were consistent with reduced paw weight and paw edema. Histological analysis further confirmed CBD's anti-inflammatory action, showing decreased inflammatory cell infiltration in paw tissues. Conclusions CBD exhibited antinociceptive and anti-inflammatory effects in acute pain models, comparable to diclofenac sodium. These findings support the hypothesis that CBD has potential as a therapeutic agent for the management of acute pain and inflammation, but further investigation is warranted in the context of chronic conditions.
Keywords: cannabidiol, acute pain, writhing test, formalin test, mice

S2-09

Poster Presentations

Impact of water insecurity on neurocognition in children with konzo, a paralytic disorder associated with food toxicity

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Abstract

Background: Konzo is an irreversible paralysis associated with food insecurity and toxicity of insufficiently processed cassava, sometimes because of water shortage. However, the exact impact of water insecurity on child health in konzo affected areas has remained unknown. Objective: To elucidate the links between household water insecurity and evolution of neurocognitive performances of children in a konzo affected area. Method: We carried out a longitudinal study of 280 children (aged 60-180 months) from 153 households in Kahemba, a konzo affected area in the Democratic Republic of Congo (From June 2017 to April 2021). This prospective study used the HWISE-Scale to assess level of water insecurity. Neurocognition performances of children were assessed using the KABC-II for cognition performances. Neurocognitive performance scores were analyzed across levels of water insecurity using generalized linear models at the 0.05 significance level. Results: Of the 280 children, 63.6% (178/280) were males and 36.4% (102/280) females; 54.6% (153/280) had Konzo and 45.5% (127/280) were healthy. The median age was 12 years and 10 years for konzo children vs. healthy children, respectively (p= 0.001). Food insecurity was judged as severe in 97.4% (149/153) of households. Water insecurity was high in 82.4% (126/153) of households. Level of food insecurity significantly and positively correlated with that of water insecurity (r=0.406; p=0.001). After adjusting for socioeconomic status, higher level of water insecurity was significantly and negatively associated with lower learning capability score (B=-0.357: p=0.04). Conclusion: Our study brings the first evidence of an association between water insecurity, food insecurity, and poor learning (cognition) in children from households affected by konzo.
Acknowledgment Funded by NIH Grant NIHES/FIC RO1ES019841
Keywords: cassava, water insecurity, cognition, konzo, food security.

S2-10

Bioassay-guided Identification of Potential Alzheimer's Disease Therapeutic Agents from Kaempferol-Enriched Fraction of Aframomum melegueta Seeds using in Vitro and Chemoinformatics Approaches

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Abstract

Background and objectives: Alzheimer's disease (AD) has become a major public health concern and the fifth major cause of death among the aging population globally. Methods: In this study, the total phenols and flavonoids contents (TPC and TFC) and in vitro antioxidant actions of the methanol extract and the various fractions of Aframomum melegueta were evaluated using 2,2-diphenyl-2-picrylhydrazyl (DPPH) radical scavenging activity, nitric oxide scavenging activity (NO), lipid peroxidation (TBARS) activity and ferric reducing power assay (FRAP). Furthermore, acetylcholinesterase (AChE) and butyrylcholinesterase (BuChE) inhibitory activities of the two most potent fractions were investigated, and the phytochemicals identified in the ethyl acetate fraction, which had the best antioxidant and cholinesterase inhibitory effects were subjected to chemoinformatics studies. Results and discussion: The extract and its fraction had high amounts of TPC and TFC. The ethyl acetate fraction exerted the best DPPH, NO, TBARS, and FRAP inhibition with IC50 values of 5.06, 6.58, 2.12, and 88.73 µg/mL, respectively. Interestingly, n-hexane and ethyl acetate fractions inhibited AChE (IC5016.83 and 11.67 µg/mL) and BuChE (IC50 7.54 and5.21 µg/mL) enzymatic activities more than the standard inhibitor, rivastigmine which had 11.99 and 11.40 µg/mL IC50 values, respectively. A total of 18 compounds were identified, and kaempferol was the major component, with 40.01 µg/g (30%). More strikingly, the top-scoring compounds (catechin, and kaempferol) exhibited good binding affinity, and interactedfavorably with amino acids residues around and within the active sites of AChE and BuChE and also obeyed drug-likeness rules, and did not show a tendency towards toxicity when placed side by side with rivastigmine which is immunogenic. Thus, A. melegueta seeds contain safe bioactive chemicals, which could be a veritable remedy for managing Alzheimer's and other neurodegenerative diseases.

Keywords: Aframomum melegueta, acetylcholinesterase, antioxidants, butyrylcholinesterase, drug-likeness, antioxidants.

S2-11

Antidepressant-like potential of stigmasterol against lipopolysaccharide-induced depression-like behaviors in mice

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Abstract

With a global prevalence of 3.8% and affecting 280 million people, depression remains a global burden calling for renewed efforts including novel antidepressants to address treatment gaps. Stigmasterol (STIG) is an unsaturated naturally abundant phytosterol that has exhibited anti-neuroinflammatory and antioxiant activity in preclinical studies. With the significant involvement of neuroinflammation and oxidative stress in the pathogenesis of treatment resistant depression, we induced depression-like behaviors using lipopolysaccharide (850 ug kg) and assessed the antidepressant-like effects of STIG at doses 1, 3, 10, 30 and 100 mg kg-1 using the force swim test (FST) and tail suspension test (TST) in mice. STIG significantly ameliorated depressive-like behavior as evident from the results obtained from the FST and TST, reducing the duration of LPS-induced immobility in a dose dependent manner. STIG pretreatment also reduced LPS-induced anhedonic behaviour measured in sucrose preference test and also increased social interaction time. Additionally, whole brain antioxidant levels assayed indicated an increase in oxidative stress observed after LPS injection was also attenuated by STIG with elevated concentrations of brain glutathione as well as reduced activity of catalase enzyme in the brain. Based on results obtained, we concluded that STIG exhibits antidepressant-like activity in the LPS-induced model for antidepressants by mitigating neuroinflammation in a dose-dependent manner.

Keywords Mood disorders, Neuroinflammation, Neuropsychiatry, Drug discovery, Antidepressant

S2-12

Neuropharmacological properties of ethanol extract of the leaves of *Olax subscorpioidea oliv.* in mice

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Abstract

Background: Sensory input to the central nervous system is the primary means by which, an individual responds to variation in their physical and biological environments. Interestingly, some psychoactive plants have provided valuable insight into the neurochemistry of many central nervous system disorders. Medicinal plants have been highly used regularly for thousands of years by people as medicine for the masses. Among the notable ones (herbs) used in psychiatric conditions in African folkloric medicine is O. *subscorpioidea. O. subscorpioidea*, which is locally called “ifon” belongs to the family *olaceacea*. It is a shrub distributed in Ghana and some parts of Africa. *Olax subscorpioidea* is used in the management of mental illness, fever, convulsion, and pain in traditional medicine.

However, there is scant information on the neuropharmacological activities that support its use. Aim: The aim of the study is to investigate the neuropharmacological properties of ethanol extract of *O. subscorpioidea* leaves (EEOSL) in mice. Method: The leaves were air-dried (150 g), pulverized and soaked in 50% ethanol (1.5 L) for 48 hours. The filtrate was concentrated and evaporated to dryness (8.7 g). Twenty-five Swiss male albino mice (20-22 g) were allotted into five treatment groups viz: control (distilled water), and Ethanol Extract of *Olax subscorpioidea* Leaves (EEOSL) (3.1, 6.3, 12.5, 25 mg/kg) given with five animals in each group. They were pretreated thirty minutes intraperitoneally (i.p.), before neurobehavioural effects of EEOSL on novelty-induced behaviours (rearing and grooming) and frequency of head dips were investigated using open-field and hole-board tests respectively. Another twenty male mice (22-25 g) were divided into four treatment groups: the control would be (distilled water) and EEOSL (12.5, 25, 50 mg/kg) as well. Thirty minutes after i.p. treatment, pentobarbitone-induced sleeping time was investigated. For the analgesic study, eighty male mice (22-25 g) were allocated into four treatment groups: control (distilled water), and EEOSL (12.5, 25, 50 mg/kg) with five animals in each group. Thirty minutes after i.p. treatment they were subjected to acetic-acid induced writhing, formalin, tail immersion and hot plate tests. Similarly, for the antidepressant study, another set of eighty male mice (22-25 g) were randomly allotted into four treatment groups: control (distilled water), and EEOSL (6.3, 12.5, 25 mg/kg) with five animals in each group. Thirty minutes after i.p. treatment, animals were subjected to despair, tail suspension, reserpine-induced depression, and yohimbine lethality tests.

S2-13

Spondias mombin polyphenols Intervention on Hypothalamic Neuropeptide Ff-1 Receptor Signalling in Dehydroepiandrosterone-Induced Polycystic Ovary Syndrome in Rat

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Abstract

Introduction: Polycystic ovary syndrome (PCOS) affects 6-20% of women, leading to infertility, menstrual irregularities, and metabolic issues. Studies show varying prevalence, with high rising rates in Nigerians. PCOS is linked to insulin resistance and hormonal imbalances, affecting brain function. We investigated the potential efficacy of spondias mombin (S.mombin) in modulating hypothalamic neuropeptide FF-1 receptor (NPFF1R) signalling to improve ovulation rates in Wistar rat model of dehydroepiandrosterone (DHEA)-induced PCOS. Method: The study explores in silico docking of flavonoid-enriched fractions from S.mombin against NPFFR1, involving protein modeling, ligand preparation, molecular docking, and binding free energy calculations to assess binding

stability. Next, a total of 36 female juvenile Wistar rats (30-50g) were divided into six groups (n=6). Control-(G1) received sesame oil, S.mombin low dose-(G2) orally received S. mombin (50 mg/kg), S.mombin high dose-(G3) orally received S.mombin (100 mg/kg), PCOS-(G4) received DHEA (6 mg/100 g) via daily subcutaneous injection, PCOS+S.mombin low dose-(G5) and PCOS+S.mombin high dose-(G6). All treatment was done for 21 days, followed by hormonal, histological, immunohistochemical and gene examination. Result: Phytoconstituents of S.mombin polyphenols demonstrate strong binding affinities with NPFF1R, highlighting bioactivity. DHEA-induced PCOS significantly disrupts the levels of luteinizing hormone, follicle-stimulating hormone, oestradiol, fasting serum insulin, fastingblood glucose and HOMA-IR. It also downregulated NPFF1R and upregulated GnRH/cAMP/PKA mRNAs expression, and revealed cystic follicles in the ovaries with degenerative-like features in the hypothalamus. Conclusion: Taken together, our findings clarify the role of DHEA-induced PCOS and S. mombin mechanism in counteracting these effects by preserving ovarian and neuronal cells integrity, and reducing anovulatory and reproductive issues associated with PCOS.

Keywords: Polycystic ovary syndrome; Spondias mombin; Anovulation; Neuropeptide FF-1 receptor; Infertility

S2-14

Assessment of galectin-3 expression in the retino-cortico-hippocampal tract of glaucomatous rats comorbid with neuroinflammation

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Abstract

Background: There is a noted connection between Alzheimer's disease and glaucoma, particularly in the elderly population, where individuals with glaucoma may exhibit clinical signs of dementia and vice versa. Objective: This study investigates Galectin-3 (Gal-3) expression in activated microglia in the eye and brain during neuroinflammation induced by lipopolysaccharide (LPS) in conjunction with hypertonic saline-induced glaucoma (HSIG). Methods: Twenty-four Long-Evans rats (280-300g) were divided into four groups(n=6). SHAM (distilled water and normal saline), LPS (500 µg/kg for 7 days), HS (single 50 µl dose of hypertonic saline), and HS+LPS (50 µl saline followed by 500 µg/kg LPS for 7 days). Experimental animals were anesthetized and sacrificed for histological, biochemical, immunohistochemistry, and immunofluorescence analyses of eye and brain tissues. Results: Gal-3

expression increased in the retina, correlating with activated microglia (CD16+ and Iba1+). CD16+ cells were linked to ganglion cell layer degeneration, triggering an inflammatory response. A significant increase in recruitment of activated microglia and co-localization of Gal-3 with degenerative cells was observed in the cortex. Notably, a cluster of Gal-3 was not found to co-localize with Iba1+ cells, and some Iba1+ cells did not express Gal-3. LPS-AD/HSIG disrupted tissue architecture, reduced catalase activity, and increased TNF-α and IL-10 levels in both the eye and brain. Conclusion: The interaction of Gal-3 and activated microglia phenotypes plays a deleterious role in mediating inflammatory assault in the retina and cortical areas. This highlights the need for further research on microglial phenotypes in glaucoma and neuroinflammation.
Keywords: Neuroinflammation, Glaucoma, Microglia, LPS, Hypertonic saline

S2-15

Corn oil and soybean oil effects as vehicles on behavioral and oxidative stress profiles in developmentally exposed offspring mice

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Abstract
Corn and soybean oils are among the most frequently used vehicles for water-insoluble compounds in toxicological studies. These two vegetable oils are nutrients and may induce some biological effects on animals that might interfere with the experimental results. However, their chronic effects on a developing brain have not been reported. This study aims to evaluate the neurobehavioral and brain biochemical effects of both oils on male and female Swiss albino mice. Pregnant female mice were exposed to 1µl/g/d of either tap water, con oil (CO), or soybean oil (SO) from early gestation (GD1) until weaning then offspring mice were exposed to the same treatment regimen until adulthood (PND70). Our results showed that developmental exposure to both oils induced body weight changes in offspring mice. In addition, we detected some behavioral abnormalities where both oil-treated groups showed a significant decrease in locomotor activity and greater levels of anxiety behavior. Moreover, our results suggest that continuous exposure to these oils may alter motor coordination, special memory and induce depression-like behavior in adult mice. These alterations were accompanied by increased malondialdehyde, superoxide dismutase, and glutathione peroxidase activities in specific brain regions. Together, these data suggest that exposure to CO and SO as vehicles in developmental studies may interfere with the behavioral response and brain redox homeostasis in offspring mice.

Poster Presentations

S2-16

Modulation of Brain-Kidney Crosstalk by Olanzapine in Aluminum Chloride-Induced Memory Impairment in Male Mice

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Abstract

Background and Objective: Chronic kidney disease (CKD) is associated with an increased risk of neuropsychiatric disorders, cognitive decline, dementia due to the release of cytokines and chemokines, production of reactive oxygen species, and the generation of trophic factors. The second-generation antipsychotic olanzapine (OLN) has been studied for its neuroprotective and antioxidant properties that may enhance cognitive function. However, limited research has explored the interaction between brain cytokines and kidney biomarkers, which could contribute to pathological conditions. This study aimed to assess the effects of OLN on cognitive function, oxidative stress, and the relationship between brain and kidney markers in mice with aluminum chloride (AlCl)-induced memory impairment. Methods: Twenty-five male mice (20-30 g) were randomly assigned to five groups (n = 5 each). Group 1 served as the control, receiving only distilled water (10 ml/kg). Group 2 received AlCl (100 mg/kg). Groups 3 and 4 were pre-treated with OLN (0.5 mg/kg and 1.0 mg/kg, respectively) one hour before AlCl administration. Group 5 was pre-treated with Donepezil (1 mg/kg). All treatments were administered orally for seven consecutive days. On the final day, neurobehavioral tests, including the Y maze and open field tests, were conducted to evaluate memory impairment. The mice were then euthanized; their brain and kidney tissues were analyzed for tumor necrosis factor-alpha (TNF-α), antioxidant markers, urea, and total protein levels, along with histopathological examination of the hippocampus and prefrontal cortex. Results: Both doses of OLN significantly improved cognitive function in the Y maze and open field tests, reduced oxidative stress by decreasing malondialdehyde and increasing SOD, catalase, and glutathione levels, and lowered brain TNF-α levels. Histopathological assessments indicated that OLN reduced neurodegeneration in the hippocampus and prefrontal cortex. There is also decreased kidney urea and increased protein concentration. Discussion: OLN significantly improves memory and antioxidant levels while reducing cytokine levels, although its effects on kidney function are limited. These results underscore the intricate relationship between OLN, cognitive function, and renal health.
Keywords: Antioxidant markers, Olanzapine, TNF-α, Urea, Y-maze

S2-17

Nutritional status of children with neurodevelopmental disorders aged 3 - 12 years followed up at the Neuro-Psychopathological Center and the University Clinics of Kinshasa

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Abstract

Background and Objective: Nutritional disorders are observed in patients with neurodevelopmental conditions. In this context, nutritional disorders are poorly investigated in our area. This study sought to determine the nutritional status of children with neurodevelopmental disorders. Methods: A retrospective study was conducted from June to October 2022 on medical records of 388 patients aged 3 to 12 years followed up in pediatric neurology at the Neuro-Psycho-Pathological Center (CNPP) and the University Clinics of Kinshasa (CUK) from January 2010 to June 2022. The nutritional assessment was carried out using the World Health Organization (WHO) anthropometric indices (Weight/Age, Height/A/A, Weight/Height, Body mass index). Results: We observed a male predominance with a sex-ratio of 1.47 and the mean age (± standard deviation) was 5.75 (±2.82) years. The most common neurodevelopmental disorder was intellectual disability (intellectual disability and global delay in psychomotor development) with 26.28% (102/388) of subjects. Normal nutritional status was found in 23.19% (90/388). Acute malnutrition accounted for 52.1% (202/388) of which 31.18% (121/388) were moderate, chronic malnutrition 22.40% (87/388) and overnutrition 2.31% (9/388). Among children diagnosed with autism spectrum disorder, the frequency of moderate acute malnutrition was 42.25% (30/71). Only 4 out of 289 malnourished children (1.4%) had benefited from nutritional rehabilitation. The dietary supplement (omega 3 and 6 and neurotropic vitamins) was noted in children with dietary supplementation and good evolution in the order of 70.9% (202/285). Conclusion: Nutritional disorders are associated with neurodevelopmental conditions. This should be taken into account in further studies and in the management of neurodevelopmental disorders.

Keywords: Malnutrition, Autism spectrum disorder, Intellectual disability, Nutritional rehabilitation, Omega 3 and 6.

S2-18

Peripheral neuropathy in sciatic and vagus nerve of mice undergoing sub-chronic exposure to vanadium

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Abstract

Vanadium, a renowned neurotoxic environmental pollutant, induces oxidative damage, neuroinflammation, and demyelination in biological systems. While in-depth studies have been done on its effect on the central nervous system (CNS), there is a paucity of data on the extent of damage it causes to the peripheral nervous system (PNS). This is however important as some populations for instance the inhabitants of the Niger Delta are practically exposed to vanadium through the burning of fossil fuels for a lifetime with a danger of subchronic and chronic peripheral neuropathies. This work is designed to ascertain the peripheral neuropathies resulting from sub-chronic vanadium administration and investigate the associated neurobehavioural changes. A total of 72 male BALB/c mice will be used for the experiment, which will be divided into three groups representing 1 month, 2 months and 3 months of exposure. The mice will be dosed with sodium metavanadate via intraperitoneal injection (i.p.) at 3 mg/kg body weight. From postnatal day 1 to 14 days, dams will be dosed daily while the pups will be dosed from day 15 to 30, 60 and 90 days, every 72 hours, respectively for each treatment group. Age-matched controls will receive a placebo throughout the experimental period, and daily body weight will be taken for the entire period of exposure. Afterwards, the mice will be subjected to behavioural tests, including open field, inverted square grid and hanging wire tests. The sciatic and vagus nerves will be harvested for histological analysis using (haematoxilin and eosin and cresyl violet staining) and immunohistochemistry (S100 and myelin basic protein). Building on previous findings regarding the CNS where demyelination of axons and activation of glial cells have been established, along with recent observations of misfolded protein aggregation in cases of chronic vanadium exposure, we anticipate similar effects in the PNS. We expect to observe severe peripheral axonopathies compared to those seen after sub-acute exposure, further supporting the proposed possibility of vanadium crossing the blood-nerve barrier and this will be reported at the conference.

S2-19

Molecular Docking Analysis of Butyrate-Neurotransmitter Interactions: Insights into Anxiety in Autism Spectrum Disorders

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Abstract

The exploration of butyrate's interaction with serotonin and dopaminergic pathways holds promise for advancing therapeutic approaches for neurodevelopmental disorders such as autism spectrum disorder (ASD). Understanding these interactions may open new avenues for addressing the complex behavioral challenges associated with ASD. This research aims to enhance our knowledge of butyrate-based interventions, potentially optimizing treatment strategies to manage the behavioral complexities characteristic of ASD. To achieve the objectives of this study, standardized protocols were used to conduct neurodevelopmental and behavioural assessments, evaluating cognitive and motor functions in an animal model exposed to valproic acid. Following these assessments, the brains of rats were fixed in paraformaldehyde, embedded in paraffin, sectioned, stained with hematoxylin and eosin, and examined microscopically. For molecular docking studies, 3D structures of target proteins and ligands were prepared, and simulations were conducted using the AutoDock tool to predict binding affinities and interaction sites. Results show that AVP-exposed rats exhibit neurological and behavioral changes in motor and sensory system development, neuronal changes, increased anxiety, and reduced social behaviors. Molecular docking further indicates a strong affinity of butyrate for dopaminergic and serotonergic receptors associated with anxiety-like behaviors. Prenatal exposure to valproic acid in rats induces behavioral and neurodevelopmental alterations, highlighting neuronal changes linked to anxiety. Molecular docking proves essential for exploring the specific interactions of butyrate with dopaminergic and serotonergic receptors, supporting the development of targeted therapies to alleviate anxiety symptoms.

Keywords: Neurotransmitters, molecular docking, butyrate, anxiety, autism spectrum disorders.

S2-20

Sex-Specific Neurobehavioral and Reproductive Effects of Developmental Exposure to Glyphosate in Offspring Mice

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Abstract

Herbicides and pesticides are widely used in agriculture to control weeds and pests, with glyphosate (GLY) being a prominent active ingredient in many non-selective herbicides. Despite increasing concerns about its neurotoxic and endocrine-disrupting potential, the effects of GLY on

developmental, behavioral, and reproductive health remain incompletely understood. Our study aims to evaluate the sex-specific neurobehavioral and reproductive effects of early-life exposure to glyphosate in Swiss albino mice. Pregnant mice were exposed to a low concentration of 50 mg/L GLY through drinking water from gestation up to the weaning of the pups, which were individually exposed to a similar dose regimen until adulthood. Our results show that glyphosate exposure delayed puberty onset in females, indicated by delayed vaginal opening and prolonged estrous cycles, signifying endocrine disruption. Behavioral outcomes were markedly sex-dependent. Females exhibited increased anxiety-like behaviors, impaired memory performance, and elevated depression-like tendencies, while males showed heightened anxiety, impaired memory, and significant deficits in social interactions. These findings reveal that chronic early-life exposure to glyphosate disrupts neurobehavioral and reproductive health, with distinct sex-specific vulnerabilities. Our results contribute to understanding the differential susceptibilities to glyphosate exposure and underscore the need for targeted regulatory actions and further research into its mechanisms of action.

S2-21

Investigation of *Pleurotus ostreatus* Aqueous Extract on Oxidative Stress Markers, hippocampus and Memory Functions of Wistar Rats: The Study of Scopolamine-induced Neurotoxicity

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Abstract
The present study investigated the neuroprotective effects of *Pleurotus ostreatus* aqueous extract (POAE) on scopolamine-induced changes in memory, oxidative stress markers and hippocampus of adult Wistar rats. Twenty-five male Wistar rats were randomly divided into five groups (n = 5). Group I received normal saline, orally from day 1 to 14 and then intraperitoneally from day 15 to 21. Group II received NS, orally from day 1 to 14 and 3 mg/kg of scopolamine intraperitoneally from day 15 to 21. Groups III, IV and V, respectively received 200 mg/kg POAE, 400 mg/kg POAE and 20 mg/kg memantine orally from day 1 to 21, and each of the groups received 3 mg/kg scopolamine intraperitoneally from day 15 to 21. Memory was assessed using the T-maze and novel object recognition tests. The rats were euthanized, and blood samples and brain homogenate were collected for oxidative stress analysis. The hippocampus was harvested, processed, and stained with Haematoxylin and Eosin. The results showed that the increase in MDA levels and decrease in catalase activities were not significant in group II when compared with groups I, III, IV and V. The pretreatment of POAE for 14 days enhanced recognition memory significantly by increasing the DI when compared with the control group. However, POAE seems to enhance working and recognition

memory in groups III, IV, and V, but it is non-significant when compared with group II. The pretreatment of POAE in groups III and V prevents the histological alterations in CA1 and CA3 regions of the hippocampus when compared with group II administered with scopolamine. The treatment of POAE at higher doses could significantly improve memory and prevent oxidative stress and histological alterations in a rat model. Therefore, further study should be carried out, administering higher doses for both POAE and scopolamine for a long period.

S2-22

Rescue role of catechin and oleanolic acid in a lead-induced Drosophila melanogaster model of Alzheimer's disease

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Abstract
Alzheimer's disease (AD) is a common neurodegenerative disorder marked by cognitive decline, memory loss, and progressive neuronal dysfunction. Environmental toxins like lead have been shown to exacerbate AD symptoms, contributing to oxidative stress and neuronal damage. Catechin and oleanolic acid, two naturally occurring compounds with known antioxidant properties, may counteract these effects. This study investigates the combined neuroprotective potential of catechin and oleanolic acid in a Drosophila melanogaster model of lead-induced AD, focusing on the neurobehavioural, biochemical outcome and microanatomical changes. The experiment utilized Drosophila melanogaster, which were divided into six groups (n=6). Flies were exposed to lead, catechin, oleanolic acid, and a combination of catechin and oleanolic acid for seven days. Neurobehavioral assays such as geotaxis and crawling activity were conducted. Biochemical analyses measured acetylcholinesterase (AChE) activity, total protein, glutathione-S-transferase (GST) activity, and oxidative stress markers. Microanatomy of the optic lobe and mushroom body of the Drosophila brain was evaluated using histology (H&E) and Amyloid Beta (A?) immunostaining techniques. Lead exposure resulted in significant locomotor impairments, increased oxidative stress, and neuroanatomical damage in the optic lobe and mushroom body as evidenced by histological staining and aggregation of A? plaques showed by the immunostaining analysis. The combination of catechin and oleanolic acid showed synergistic effects, further enhancing antioxidant activity and reducing lead-induced toxicity; improving geotaxis and crawling performance and significantly reducing oxidative stress. Histological examination revealed preservation of brain architecture, and immunostaining showed enhanced synaptic density and reduced neuronal loss in treated groups and restoring brain morphology closer to control levels. Co-administration of catechin and oleanolic acid significantly ameliorates lead-induced neurotoxicity in Drosophila melanogaster preserving both behavioral functions and brain histoarchitecture. Thereby suggesting their potential therapeutic role

in mitigating environmental toxin-induced neurodegenerative conditions such as Alzheimer's disease.

S2-23

Impaired Brain Redox Status in Rats Fed Selenium-Restricted Diet, and the Ameliorative Effect of Dietary Inclusion of African Eggplant (Solanum macrocarpon L.) Leaves

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Abstract

Impaired Brain Redox Status in Rats Fed Selenium-Restricted Diet, and the Ameliorative Effect of Dietary Inclusion of African Eggplant (Solanum macrocarpon L.) Leaves Opeyemi Babatunde Ogunsuyi1, Olawande Chinedu Olagoke2, Adedayo Oluwaseun Ademiluyi3, Ganiyu Oboh3 1Department of Medical Biochemistry, Federal University of Technology Akure (FUTA), Nigeria 2Division of Gastroenterology, Department of Medicine, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA 3Department of Biochemistry, FUTA, Nigeria Background and Objectives: Selenium (Se) deficiency is associated with neurological disorders due to its antioxidant properties. This study investigated the neuroprotective effects of African eggplant (Solanum macrocarpon L) on rats fed Se-restricted diets. Methods: African eggplant was cultivated on soil containing 8.36±2.02 mg/100g Se. Thereafter, the AE leaves were harvested, dried and pulverized for subsequent analyses. The Se content in the pulverized leaves was subsequently determined. For the feeding experiment, adult Albino rats were divided into four groups (n=6) consisting of Se control (rats fed basal diet with Se), Se restricted control (rats fed basal diet without Se), 4% AE (rats fedSe-restricted diet plus 4% AE leaf) and 8% (rats fed Se-restricted diet plus 8% AE leaf). The feeding experiment lasted for fourteen (14) days after which the rats were euthanized and brain tissue rapidly isolated and homogenized. This was followed by assaying the brain tissue homogenate for neural antioxidant and redox markers. Results: These showed that the AE leaves bioaccumulated Se to the level of 5.91±0.02 mg/100g. Rats fed Se-restricted diet elicited significantly reduced ($p<0.05$) levels of neural total thiol and non-protein thiol, as well as significantly ($p<0.05$) elevated level of lipid peroxidation, when compared to the control rats fed basal diet containing Se. In addition, activities of GPx, GST and catalase were significantly reduced ($p<0.05$) in rats fed Se-restricted diet. However, the impaired redox/antioxidant indices were significantly ameliorated ($p<0.05$) in rats fed Se-restricted diet plus AE (4% and 8%). Discussion: This study therefore revealed that AE leaves significantly bioaccumulated Se from the soil and was able to ameliorate impairments to neural antioxidant markers observed in rats fed Se-restricted diets.

Keywords: Nutritional disorder; Selenium; Brain; Functional food; Solanum

S2-24

Roles of oxidative stress and pro-inflammatory cytokines in copper sulfate-induced Depression-like disorders and abnormal neuronal morphology in mice

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Abstract
Epidemiological studies have implicated copper as one of the key environmental risk factors for the pathogenesis of major depression. However, the precise mechanism by which copper contributes to the genesis of depression, particularly the involvement of oxidative stress-driven neuroinflammation, is yet to be fully elucidated. Thus, this study was designed to evaluate the effects of copper sulfate ($CuSO_4$) on depression-like behaviors and the role of oxidative stress and pro-inflammatory cytokines in mice. Forty male Swiss mice were distributed into control and three test groups (n = 10), and were treated orally with distilled water (10 mL/kg) or $CuSO_4$ (25, 50 and 100 mg/kg) daily for 28 days. Afterwards, the tail suspension, forced swim, and sucrose splash tests were used for the detection of depression-like behaviors. The animals were then euthanized and the brains were processed for the estimation of biomarkers of oxidative stress and pro-inflammatory cytokines (tumor necrosis factor-alpha and interleukin-6). The histomorphological derangements and neuronal viability of the prefrontal cortex, hippocampus and striatum were also evaluated. Mice exposed to $CuSO_4$ displayed depression-like features when compared with controls. The brain concentrations of malondialdehyde, nitrite and pro-inflammatory cytokines were elevated in mice exposed to $CuSO_4$. Mice exposed to $CuSO_4$ also had reduced brain antioxidant status (glutathione, glutathione-s-transferase, total thiols, superoxide-dismutase and catalase), as well as altered histomorphological features, and decreased population of viable neuronal cells. These findings suggest that $CuSO_4$ induces oxidative stress and pro-inflammatory cytokines, and abnormal neuronal morphology to elicit depression-like effects in mice.
Keywords: Copper sulfate, Depression, Oxidative stress, Pro-inflammatory cytokines.

S2-25

Hair Lead and Cadmium Levels among Moroccan Children Diagnosed with Attention Deficit Hyperactivity Disorder

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Abstract

Attention deficit hyperactivity disorder (ADHD) is a neurodevelopmental disorder characterized by inattention, hyperactivity, and impulsivity, typically diagnosed in childhood and potentially persisting into adulthood. While genetic, environmental, and neurobiological factors contribute to ADHD, its precise causes remain unclear. Environmental factors, including prenatal exposure to neurotoxic metals, may increase the risk of developing the disorder. This study aimed to investigate the potential association between lead (Pb) and cadmium (Cd) exposure, alongside other environmental risk factors, and ADHD in children from the city of Marrakech. The study included 42 children, divided into two groups: 21 children diagnosed with ADHD according to DSM-5 criteria and Conners' scale, and 21 healthy controls with no history of psychiatric or medical conditions. Parents completed questionnaires on environmental risk factors, and hair samples were collected for analysis using Flame Atomic Absorption Spectrophotometry (FAAS). Our results revealed significant associations between ADHD and prenatal exposure to environmental pollutants, including proximity to industrial zones, heavy traffic, and maternal exposure to paint and toxic products ($p = 0.03$, $p = 0.04$). Additionally, children with ADHD had significantly higher median levels of lead (2.670 µg/g, $p = 0.02$) and cadmium (0.12 µg/g, $p = 0.03$) in their hair compared to controls. The risk of developing ADHD was 1.46 times higher for lead and 1.33 times higher for cadmium. Gender analysis showed that boys with ADHD had higher levels of these metals than girls, suggesting a potential gender influence on heavy metal accumulation and ADHD development. Our findings support previous research suggesting that heavy metals may contribute to the development of ADHD. Further research is necessary to explore the underlying mechanisms of these associations.
Keywords: ADHD, Children, Environmental Risk Factors, Lead, Cadmium

S2-26

Evaluation of the Neurocognitive Potential of Extracts of Phoenix dactylifera and Parquetina nigrescens

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Abstract

Objectives: To evaluate the ethnomedicinal uses of crude methanol extracts of the seeds of Phoenix dactylifera (SPD) and leaves of Parquetina nigrescens (LPN) for their possible neurocognitive and neuropharmacological potentials Methods: The extracts were evaluated for their anti-inflammatory, phytochemical screening, antioxidant, and anticholinesterase activities using standard in vitro assays; and neuropharmacological potentials in vivo. Data were expressed as mean±standard error of mean (SEM) and analysed using One-way Analysis of Variance followed by a post hoc test. Results: Both SPD and LPN extracts possessed glycosides, tannins I & II, saponins, alkaloids, phenols, flavonoids, and anthraquinones. The SPD extract showed higher phenolic (807.77 ± 19.10) and flavonoid (103.99 ± 1.15) contents. Comparingly, SPD exhibited higher antioxidant activity in almost all the in vitro antioxidant assays: TAC (166.71±8.80), FRAP (336.12±0.52), CUPRAC (146.70±1.53), ABTS (1.03±0.16). However, both SPD and LPN significantly inhibited acetylcholinesterase with IC50 = 1.76 ± 0.08 mg/mL and 0.41 ± 0.03 mg/mL respectively. They consistently showed dose-dependent anti-inflammatory activities ($p < 0.05$). The LD50 of SPD and LPN were 5000 mg/kg. Both SPD and LPN extracts significantly stimulated the frequencies of rearing ($F_{6,31} = 7.068$, $p < 0.0001$) and grooming activities ($F_{6,31} = 55.9$, $p < 0.0001$) in mice but devoid of significant effect on their short-term memory. However, LPN showed a significant increase ($F_{7,41} = 8.610$, $p < 0.0001$) in the time spent in the open arm of the elevated plus maze (EPM). Conclusion: The extracts of the seeds of Phoenix dactylifera (SPD) and leaves of Parquetina nigrescens (LPN) used ethnomedicinally in the management of dementia showed promising antioxidant, anti-cholinesterase, anti-inflammatory, and neuropharmacological potentials attributed to the presence of some polyphenols.

S2-27

Investigating Tramadol Addiction and its Role in Early onset Movement Disorders: Exploring the Therapeutic Potential of Spondias mombin

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Abstract

Background: Substance abuse is a growing global health burden, with tramadol being widely misused, particularly among young individuals. Concurrently, there has been an alarming rise in early-onset movement disorders, raising questions about a potential link between substance abuse and disruptions in the nigrostriatal pathway. This study aims to investigate this connection using a tramadol addiction model and evaluate the neuroprotective effects of Spondias mombin, a medicinal plant with reported therapeutic potential. Methods: Twenty-eight adult male Wistar rats were divided into four groups of seven: Group A (control): Received normal saline and distilled water; Group B: Received 8 mg/kg tramadol for 14 days; Group C: Received 200 mg/kg Spondias mombin extract; Group D: Received tramadol and Spondias mombin concurrently. Behavioural tests (open

field, beam walk, gait, and grip strength) assessed motor function. Histological and immunohistochemical analyses evaluated neuronal integrity and dopaminergic markers in the nigrostriatal pathway. Biochemical assays, ELISA, and gene expression studies (Cabl and α-synuclein) explored oxidative stress, neuroinflammation, and molecular pathways influenced by tramadol and Spondias mombin. While data analysis is ongoing, preliminary observations suggest a potential disruption in motor functions and neuronal integrity following tramadol exposure. The study is poised to address whether tramadol addiction contributes to the rising prevalence of early-onset movement disorders in young individuals and whether Spondias mombin can counteract these effects by preserving neuronal integrity and mitigating neuroinflammation. The outcome of this study has the potential to provide valuable insights into the neurotoxic effects of tramadol and the role of plant-based therapies in managing substance abuse-related neurodegenerative conditions.

Keywords: substance abuse, tramadol addiction, early-onset movement disorders, nigrostriatal pathway, Spondias mombin, neuroprotection.

S2-28

Phytochemical aspect, safety, and antihyperglycemic effect of the aqueous extract of Carum carvi L. from the Béni Mellal-Khénifra region

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Abstract

Carum carvi L. is a biennial plant from the Apiaceae family, and its seeds have been used since antiquity for the treatment of diabetes in traditional healing systems across vast geographical areas, including the Béni Mellal-Khénifra region. The objective of this study is to analyze the phytochemistry of the aqueous extract of Carum carvi L. seeds from this region, evaluate its antihyperglycemic activity, and assess its safety. The aqueous extract of the seeds was obtained using the decoction technique. The identification of secondary metabolites was performed using HPLC. The antihyperglycemic effect was evaluated using the oral glucose tolerance test in normal Albinos mice. The safety of the aqueous extract was assessed using the acute toxicity test and the hypoglycemic effect test on baseline blood glucose levels in normal mice. The extraction yield was 8%. Phytochemical analysis of the aqueous extract revealed 11 compounds, with the most abundant being syringic acid (40.02%), cinnamic acid (26.54%), and 4-hydroxybenzoic acid (10.22%). The aqueous extract showed a significant antihyperglycemic effect in normal Albinos mice. In addition to this pharmacological effect, the aqueous extract did not pose any risk of hypoglycemia on baseline blood glucose levels in Albinos mice and showed no signs of short-term toxicity. Based on these very encouraging and positive results, which confirm the efficacy of using this plant in traditional medicine, this plant could be used to treat diabetic patients.

Keywords: HPLC, toxicity, antihyperglycemic, Carum carvi L. from Béni Mellal-Khénifra.

S2-29

Inhibitory effect of the enzymatic activity of pancreatic α-amylase and intestinal β-glucosidase from Carum carvi L. sourced from the Béni Mellal-Khénifra region: in vitro and in vivo studies

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Abstract
Carum carvi L. is a biennial plant from the Apiaceae family, and its seeds have been used since antiquity for the treatment of diabetes in traditional healing systems across vast geographical areas, including the Béni Mellal-Khénifra region. The objective of this study is to analyze the phytochemistry of the aqueous extract of Carum carvi L. seeds from this region, evaluate its antihyperglycemic activity, and assess its safety. The aqueous extract from the seeds of this plant was obtained using the decoction technique. The quantification of polyphenol and flavonoid content was determined using the Folin-Ciocalteu method and the Aluminum Chloride (AlCl3) method. The antihyperglycemic effect was evaluated by examining the effect of the aqueous extract on the enzymatic activity of pancreatic α-amylase and intestinal β-glucosidase both in vitro and in vivo. The extraction yield is 8%. The results of the quantitative phytochemical analysis showed that the aqueous extract of this plant contains 0.57 ± 0.07 mg GAE/mg DM and 0.27 ± 0.08 mg QE/mg DM for polyphenols and flavonoids, respectively. Regarding the antihyperglycemic effect, the aqueous extract exhibited an inhibitory effect on the enzymatic activity of pancreatic α-amylase with an IC50 value of 0.139 ± 0.023 mg/mL and on intestinal β-glucosidase with an IC50 value of 56.66 ± 1.247 µg/mL in vitro. Furthermore, the aqueous extract showed a significant inhibitory effect in vivo on the enzymatic activity of both enzymes.
Keywords: Carum carvi L., diabetes, α-amylase, β-glucosidase, in vivo and in vitro.

S2-30

Automated scoring of autism-like behaviors in the valproic acid mouse model: enhancing accuracy and efficiency with machine learning tools (DeepLabCut and SimBA)

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Abstract

Preclinical research often relies on animal observation and subsequent behavioral analysis to study brain function. However, traditional methods are often perceived as time-consuming and prone to human error. Recent advancements in machine learning now allow for automated tracking of freely moving animals. DeepLabCut (DLC) is a markerless pose estimation method for tracking animal body parts, while SimBA (Simple Behavioral Analysis) uses supervised machine learning to classify behaviors based on these tracked positions. In this study, we applied DLC and SimBA to introduce a scoring model for autism spectrum disorder (ASD)-like behaviors in the valproic acid (VPA) mouse model, accounting for sex differences. Our results revealed significant and consistent core ASD-like symptoms in VPA-exposed mice. The automated tracking of specific body parts, such as the animal's nose, provided higher precision and accuracy in detecting sociability deficits in VPA-exposed female mice compared to traditional behavior tracking software (e.g., EthoVision©). Notably, VPA-exposed mice exhibited significantly more repetitive behaviors, typically scored manually. However, this method assessed these behaviors with improved accuracy, thereby reducing the time and potential bias associated with manual scoring. Our approach demonstrates the power of combining DLC and SimBA to enhance the precision of scoring ASD-like behaviors in mice while addressing the limitations of traditional scoring methods and outperforming commercial automatic tracking systems. This method holds great potential for advancing research, particularly in the precise scoring of ASD-like behaviors, across various fields of behavioral neuroscience.

Keywords: Autism spectrum disorder, Valproic Acid, Mouse model, DeepLabCut, SimBA, Machine Learning.

S2-31

Amnesic properties of extra-virgin avocado oil (Persea americana Mill., Lauraceae) on anAlcl3/D-galactose Co-administration-induced model of Alzheimer's disease in ovariectomized Wistar rats

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Abstract

Background: Alzheimer's disease (AD) is the main cause of dementia worldwide, accounting for 60-70% of cases. This pathology remains incurable today because of its multifactorial origin. Previous studies showed that extra-virgin Avocado oil could protect against Alzheimer’s disease. The aim of this study was to assess the antiamnesic properties of extra virgin avocado oil (Persea americana Mill., Lauraceae) on an AlCl3/D-galactose coadministration-induced model of Alzheimer's disease in

ovariectomized Wistar rats. Material and methods: To accomplish this, 42 were ovariectomized (OVX) and 12 underwent white surgery (SHAM). Fourteen days after surgery, the rats were randomly distributed in nine groups (n = 6) and concomitantly treated with extra-virgin avocado oil (0.25; 0.5 and 1 mL/kg, p.o.) and Alcl3/D-gal (300 mg/kg/150 mg/kg i.p.) for one week. Donepezil and estradiol valerate (1 mg/kg, p.o.) were used as reference drugs. Memory disorders were evaluated using the Object Recognition, Y-Maze, and Morris water maze tests. Amyloid-beta peptide 1-42 (Aβ1-42), tau phosphorylated protein (pτ), acetylcholine, interleukin 1β (IL-1β), and tumor neutrosis factor α (TNF-α) levels, were evaluated in the hippocampus homogenate, and a histological analysis was performed in the brain tissue. Results: It was found that extra-virgin avocado oil significantly decreased the concentrations of Aβ1-42, phosphorylated tau protein at doses of 0.5 and 1 ml/kg, TNF-α and IL-1β ($p < 0.001$) at the doses tested in the hippocampus compared with the OVX+Alcl3/ D-Gal negative control group. Conclusion: Taken together, these results suggest that avocado oil could be an alternative for the prevention and treatment of AD.
Keywords: Alzheimer's disease, aluminum chloride, D-galactose, ovariectomy, avocado oil (Persea americana Mill., Lauraceae).

S2-32

Thymus vulgaris aqueous extract as a potential Intervention for prenatal alcohol-induced cognitive and anxiety disorders in mouse offspring

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Abstract

Background: Prenatal alcohol exposure (PAE) is a significant concern that leads to Fetal Alcohol Spectrum Disorders (FASD), which are characterized by cognitive, behavioural, and physical impairments. Thymus vulgaris (T.v) is a traditional medicinal plant known for its potential to treat various brain disorders. Objectives: This study aimed to evaluate the neuroprotective effects of T.v on a mouse model of PAE. Methodology: A total of 21 mice (14 females and 7 males) were utilized to generate offspring. Females were randomized into a control group (n = 2) and a prenatal alcohol exposure group (n = 12), with subgroups receiving 25, 50, 100, and 200 mg/kg of T.v. Males served exclusively as breeders in a 1:2 male-to-female ratio. The PAE model was developed through a modified protocol consisting of four periods: sensitization, coupling, gestational, and postnatal.

Females self-administered varying concentrations of ethanol, and maternal weights, litter sizes, and pup weights were monitored. Behavioural tests were conducted before sacrificing the mice for analysis. Results: T.v at doses of 25 and 100 mg/kg significantly increased spontaneous alternation and the discrimination index in the Y-maze and Novel Object Recognition tests, respectively. In the EPM, T.v significantly enhanced the number of entries and time spent in the open arms. Additionally, T.v reduced levels of malondialdehyde, nitric oxide, Acetylcholinesterase, ALT and AST while increasing total protein, serotonin and catalase levels. Histopathological evaluations revealed that T.v mitigated neuronal death in the hippocampus. Conclusion: The findings suggest that T.v exhibits neuroprotective properties against PAE.

S2-33

Ameliorative effects of the “tenghõ’s” extract on cell proliferation, neuronal cell viability, and growth

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Abstract
The pathogenesis of neurodevelopmental disorders involves structural alteration in neurons' growth and morphology, which disrupt communication between specific brain regions. While a previous study explored the analgesic and anti-inflammatory properties of the “tenghõ’s” extract in an animal model, it’s effect on neuronal cells was not examined. Rooted in Cameroonian traditions, the “tenghõ’s” extract combines ginger (Zingiber officinale), garlic (Allium sativum), lemongrass (Cymbopogon citratus), parsley (Petroselinum crispum), basil (Ocimum basilicum), and hive products such as propolis. These components may protect neuronal cells by promoting survival, growth, and morphology stability in response to neurotoxic or adverse stimuli. This study investigated the effects of “tenghõ’s” extract on cell proliferation, viability and morphology on primary neurons from the midbrain, striatum and hippocampus of neonatal rat pups. MTT assay was used to assess both cell proliferation and survival on SH-SY5Y neuroblastoma cell line. Primary neurons, freshly isolated from neonatal rats? midbrain, striatum, and hippocampus were used for morphological studies. Cells were cultured in a 5% CO2 humidified atmosphere at 37°C and treated with or without 0.1, 1 and 10 µg/mL of “tenghõ’s” extract. Primary neurons exposed to the “tenghõ’s” extract showed significantly increased neurite outgrowth, particularly in the midbrain, striatum and

hippocampus compared to the control. The longest neurite length increased notably. Additionally, SH-SY5Y cells exhibited improved viability at the lowest concentration (0.1 µg/mL). These findings suggest that the “tenghõ’s” extract can enhance neuronal structure, potentially contributing to its neuroprotective effect.

Poster Presentations

Session 3

S3-01

Plastinated Specimen in Anatomy Education: Babcock University Students' Knowledge, Perception and Awareness -A Cross-Sectional Study

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Abstract
The study of Anatomy has been in existence for thousands of years, according to scientific literature yet Anatomical Education in Africa (Nigeria) has over the years been taught mainly with traditional methodologies. Plastination is a recent method of permanently preserving biological specimens and tissue in a life-like state whereby the fluids of the body (fat and water) are replaced with synthetic materials (silicon). This research was aimed at investigating the perception and awareness of plastinated specimens among Anatomy Students of Babcock University, Ilishan-Remo, Ogun-State, Nigeria. This study was done using a research instrument (questionnaire with informed consent) from 91 participants. The Participants were systematically selected and divided into 4 groups. Group A was a member from the 2017/2018 set, Group B was a member from the 2018/2019 set, Group C was a member from 2019/2020 set, and Group D are member of the 2020/2021 set. The research instrument has four sections which include the participants? biodata, participants? knowledge, awareness level, and perception of plastination were obtained. The results of this study were analyzed using IBM SPSS and there was no significant association found between gender and level of perception at (p=0.05). The need for plastinated samples to be used as teaching aids and the technique to be taught is apparently inevitable especially in Africa (Nigeria) to make it a normal routine.

S3-02

Effect of an Experimental Model of Neuroinflammation on Reflex Regulation of the Heart in Rats

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Abstract
Experimental and clinical data demonstrate a pronounced effect of neuroinflammation on brain reflexes involved in the regulation of the cardiovascular system. This study aimed to investigate the

influence of a bacterial model of neuroinflammation on the characteristics of cardiac reflex regulation in rats. The experiment was conducted on 20 male rats of the Wistar line. The acute inflammation model was created by a single injection of lipopolysaccharide (LPS) from Salmonella typhi (n=12; 5 mg/kg; i.p.). The control group rats (n=8) were injected with a physiological solution. Hematological and hemodynamic changes were analyzed 6 hours after the LPS was injected. Various blood circulation indicators were evaluated: heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean blood pressure (MBP), and total volume of blood in the tail vessels (TVBtv). The reflex heart regulation in both groups was assessed according to the degree of bradycardia on noradrenaline (0.03 mg/kg; i.p.). Results. It was found that LPS administration resulted in the appearance of characteristic signs of inflammation (leukocytosis associated with an increase in the number of neutrophils; increase in IL-6; increase in C-reactive protein; increase in endotoxemia index et al.). In 6 hours after LPS administration pronounced changes in hemodynamics were observed: decrease of SBP by 23.4 %, DBP by 21.7 %, MBP by 21.9 %. Changes in hemodynamics occurred against the background of an increase of HR by 31.5 % and a decrease of TVBtv by 26.3 %. LPS-induced model of inflammation has a modulating effect on the reflex mechanisms of heart regulation. Norepinephrine in the control group resulted in sustained bradycardia associated with the activation of baroreceptors of reflexogenic zones and strengthening of vagus nerve influence. For rats injected with LPS, norepinephrine resulted in weaker reflex bradycardia. The peripheral and central action of anti-inflammatory cytokines may have a pronounced influence on the functional activity of both blood vessels and the heart. The effect of anti-inflammatory cytokines at the brain level can reduce the effectiveness of reflex mechanisms that provide the adaptive potential of the cardiovascular system.
Keywords: neuroinflammation, cardiac reflexes, hypertension, bradycardia.

S3-03

Axonal degeneration in hemorrhagic stroke: a systematic review

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Abstract
Hemorrhagic stroke occurs due to a rupture of a blood vessel in the brain. This leads to initial mechanical damage and secondary injuries on nearby cells, including axonal degeneration (AxD). Since axons are critical for all brain functions, we systematically reviewed studies that focused on AxD in hemorrhagic stroke to understand how and to what extent AxD develops and unravels the

underlying mechanisms and potential therapeutic targets. After screening 817 publications published until September 18, 2024, we identified 69 records to be included. AxD was detected in patients as early as 24 hours and animal models as early as 6 hours by the release of neurofilaments into the CSF and blood, the accumulation of beta-amyloid precursor protein at the sites of axonal damage, an increased serum or cerebral level of tau protein, degenerative changes in the axonal density and shape achieved from light and electron microscopy, or a reduced fractional anisotropy in diffusion tensor imaging. AxD is correlated with hematoma volume and worsening of clinical outcomes. It occurs in various locations, especially in the hemorrhagic center and perihemorrhagic zone, and its extent increases over time. Targeting neuroinflammation, improving energy metabolism, inhibiting microtubule breakdown, and stimulating axonal growth and regeneration were assessed as therapeutic options. Further investigations are needed to understand better the mechanisms underlying AxD. This will form the basis for the development of novel interventions for hemorrhagic stroke and potentially other neurological diseases.

Keywords: Hemorrhagic stroke, axonal degeneration, neuroinflammation, biomarkers

S3-04

Attention deficit hyperactivity disorder (ADHD): Diagnosis, etiology, and comorbidity

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Abstract

ADHD (Attention Deficit Hyperactivity Disorder) is a chronic neurodevelopmental disorder characterized by a symptomatic triad of inattention, hyperactivity, and motor impulsivity. In this work, we summarize current knowledge on diagnostic tools, etiology, and comorbidities associated with ADHD in children, focusing on the challenges in managing this disorder. We analyzed data published in the ScienceDirect, Cochrane, and PsycInfo databases since 2018 to provide an updated synthesis. Furthermore, our selective study strategy was refined by using precise filters to limit results to relevant publications. The selected articles are in English or French and focus exclusively on the diagnosis, etiology, comorbidities associated with ADHD in children, and its management. Our study specifically includes peer-reviewed publications (reviews, clinical trials, meta-analyses, and observational studies) and studies based on reliable, detailed methodologies. The analysis of selected articles shows that ADHD is multifactorial, caused by a complex interplay of genetic and environmental factors acting early in brain development. Our results also demonstrate that this disorder is often accompanied by various neurodevelopmental comorbidities that can complicate diagnosis and management. Additionally, the diagnostic tools for ADHD do not capture all

manifestations of the disorder, necessitating further studies to improve their effectiveness. Moreover, our analytical study reveals the lack of involvement of public service professionals in the diagnostic process, raising questions about their role in evaluating this morbidity. Furthermore, stigma often presents a challenge to the diagnosis and management of ADHD due to judgments and rejection attitudes, as well as feelings of shame, devaluation, and even self-stigmatization in the affected child. This synthesis study highlights the need to improve access to quality information on ADHD to raise public awareness. In addition, a thorough evaluation is essential for the diagnosis and management of ADHD and its comorbidities. Keywords: ADHD - Child - Diagnosis - Etiology - Comorbidity

S3-05

Sleep Sexual Dimorphism in Mitopark Mouse Model of Parkinson's disease

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Abstract

Sleep is regulated by a complex interplay of biological processes that govern the timing, duration, and quality of sleep. Four main factors modulate sleep; the circadian process (Process C), the homeostatic process (Process S), the masking effects of light and darkness, as well the motivational status that was recently shown to be a powerful modulator of sleep/wake behavior. Clinically, it is also known that depression and mood regulation are sexually dimorphic, which, in turn, affects sleep patterns. Sleep disturbances are common in Parkinson's disease (PD). While much research has focused on motor symptoms, non-motor problems such as fragmented sleep, excessive daytime sleepiness (EDS), insomnia, and REM sleep behavior disorder (RBD) are less understood, particularly how these problems vary between males and females. This study aimed to investigate the sexual dimorphism of sleep patterns in PD using the Mitopark mouse model. To this end, we analyzed a previously acquired EEG/EMG data set from Mitopark mice. This animal model replicates the chronic nature of the disease and also replicates the majority of sleep disturbances associated with PD. Our study found that while sex does not significantly affect the overall sleep-wake amount in these PD mouse models, it does influence sleep architecture. Specifically, female Mitopark mice exhibit more fragmented sleep than males, characterized by shorter and more frequent sleep episodes. This fragmentation might correlate with higher rates of insomnia observed in women with PD. These differences in sleep patterns are not due to altered homeostatic or circadian regulations or masking effects. Instead, our findings suggest the potential role of emotional and motivational processes in regulating sleep.

Importantly, we observed that theta mobilization during wakefulness, which reflects motivation valence, is altered following dopamine loss in female Mitopark mice, which may explain the sex-specific manifestations of sleep disturbances.

Keywords: Sleep, Sex, Parkinson, Motivation.

S3-06

Early life events but trait resilience differentially predict mental health outcomes in face of lifelong adversity: future perspectives and insights into the psychopathology of PTSD

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Abstract

Background and Objective. Mental health is essential to demonstrate resilience and strive for well-being, globally. We searched for determinants of post-traumatic disorders (PTSD) with or without anxiety, depression, or psychosis in Eastern Congo, a theatre of decade-long armed conflicts, precarity, and adversity. Methods. A case-control design enrolled 148 subjects with PTSD and tentatively age- and gender-matched 142 presumably healthy controls [mean (SD) years: 32.96 (12.8) years] PTSD was ascertained using the PTSD Checklist for DSM-5 (PCL-5). Measures of resilience trait (RT), anxiety, or depression were performed using the Connor and David Resilience scale, the Spielberger scale (anxiety trait), or the Beck scale, respectively. Current psychotic disorder (CPD), lifetime psychotic disorder (LTPD), and current major depressive disorder (CMDD) were ascertained at the Mini-International Neuropsychiatric Interview. Prediction was carried out using logistic regression at the 0.05 significance level (STATA, v.16). Results. PTSD was observed in 50.4% of men vs. 51.5% of women ($p>0.05$). The overall resilience trait appeared to be moderate (48.1%) vs. low or high in 30 % or 22% of subjects, respectively; with no significant association with PTSD. Distinct psychopathological features included anxiety state by Beck in 64.5%, LTPD in 56.8%, depression by Beck in 56.4%, CMDD in 37.3%, and CPD in 31% of subjects. Only LTPD [OR: 2.4, 95%CI: 1.9-3.4; $p= 0.01$] or CPD [OR: 1.8, 95%CI: 1.1-3.1; $p=0.02$] were significantly associated with PTSD after adjusting for age, gender, anxiety state, and resilience trait. All co-morbidities but LTPD were significantly associated with earlier life stressors including but not limited to poor health during the first 2 years

of life [OR: 1.47, 95%CI:1.1; p=1.98; p = 0.012] and history of physical violence [OR:1.8, 95%CI: 1.0-3.12; p=0.04] for CPD after adjustments. LTPD however was associated with familial history of mental health [OR:1.7, 95%CI: 1.0-2.8; p=0.04]. Conclusion. Early life stressors but resilient traits predict mental health outcomes in contexts of lifelong adversity. Symptoms of psychosis in contexts of PTSD may reflect different mechanisms of psychopathology. Psychopathology in Eastern Congo may help disentangle the impact of biological vulnerability, early life stressors, and lifelong adversity on mental health.

S3-07

Emotional intensity linked to current psychotic disorder and major depression associated with PTSD under conditions of lifelong adversity in Eastern Congo

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Abstract

Background and Objective. Mental health is of paramount importance in contexts of war as poor outcomes affect one's quality of life, performance in everyday activities, and post-traumatic resilience. The present study was aimed at unveiling dimensions of individual memory experiences at the core of post-traumatic stress disorder (PTSD) in Eastern Congo. Methods. A case-control design enrolled 148 subjects with PTSD and tentatively age- and gender-matched 142 presumably healthy controls [mean (SD) years: 32.96 (12.8) years]. PTSD was ascertained using the PTSD Checklist for DSM-5 (PCL-5). Measures of resilience trait (RT), anxiety, or depression were performed using the Connor and David Resilience (CD-RISC-10) or Beck scale as deemed appropriate. Current psychotic disorder (CPD), lifetime psychotic disorder (LTPD), and current major depressive disorder (CMDD) were ascertained at the Mini-International Neuropsychiatric Interview. Autobiographical memory was assessed by the Memory Experiences Questionnaire (MEQ), a ten-dimensional short version that explored self-experienced memories, assessing vividness, coherence, accessibility, emotional intensity, sensory details, and the perspective from which they recall traumatic events. Association studies were carried out using Spearman correlations or logistic regression at the 0.05 significance level (STATA, v.16). Results. PTSD was observed in 50.4% of men vs. 51.5% of women (p>0.05).

Distinct psychopathological features included anxiety state in 64.5%, LTPD in 56.8%, depression by Beck in 56.4%, CMDD in 37.3%, and CPD in 31% of subjects. Emotional intensity score was the unique MEQ dimension that significantly correlated to both PTSD (r=0.14; p=0.18) and resilience (r=-0.25, p=0.00) scores. After adjusting for age, gender, and level of resilience, emotional intensity was significantly associated with PTSD (Adjusted OR: 1.09, 95%CI: 1.02-1.16; p = 0.015), and more specifically, PTSD with current psychotic disorder (Adjusted OR: 1.09, 95%CI: 1.01-1.16; p = 0.025) or current major depressive disorder (Adjusted OR: 1.14, 95%CI: 1.06-1.22; p = 0.00). Conclusion. Emotional intensity is central to the psychopathology of mental health outcomes in war-torn Eastern Congo. Further studies will help understand how emotional experiences impact cognition, and more specifically, memory formation, retrieval, and subjective experience of remembering events that occur in situations of uncertainty and adversity imposed by war; and their influences on PTSD psychopathology.

S3-08

Aspirin as a modifier of epigenetic responses: DNA methylation changes in a social instability stress model of depression in female Wistar rats

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Abstract
Depression is the most common psychiatric disorder that poses a significant public health concern. Recent studies have implicated DNA methylation, an epigenetic modification, as a potential mechanism for increased susceptibility to depression. Aspirin [acetylsalicylic acid (ASA)] has been reported to have a possible antidepressant effect. This study aimed to determine the mechanisms of the antidepressant effect of aspirin in a social instability stress (SIS) model of depression. Eighty adult female Wistar rats (180?220 g) were acclimatised for twenty-one days. Ten were in the control group, and 70 were exposed to SIS to induce depression-like behaviours. Before and after induction, rats were subjected to behavioural tests to determine resilient and susceptible ones. Rats were divided into nine groups (n=7), and treated as follows: (i) control+distilled water (DW) (1 ml/kg) (ii) resilient+DW (1 ml/kg) (iii) susceptible+DW (1 ml/kg) (iv) susceptible+escitalopram (ESC) (10 mg/kg) (v) susceptible+RG108 (0.4 mg/kg) (vi) susceptible+ASA (10 mg/kg); (vii) susceptible+ASA (10 mg/kg)+ESC (10 mg/kg); (viii) susceptible+ASA (100 mg/kg); and (ix) susceptible+ASA (100 mg/kg)+ESC (10 mg/kg). ASA (oral), ESC (oral), and RG108 (intraperitoneal) were administered once daily for twenty-one days. Rats were euthanised, and brain samples were collected for gene expression and whole genome methylation studies. Data were analysed using one-way analysis of

variance, Tukey posthoc test with a significance level of $p< 0.05$ using GraphPad Prism 8.02. The result showed: (i) reversal of depression-like behaviours on behavioural tests; (ii) upregulation and downregulation of DNMT3L gene expression in the susceptible+ASA (100 mg/kg) and susceptible+ESC groups respectively, compared to the control; (iii) knockout of methylated depression susceptibility genes in the susceptible+ASA (100 mg/kg) and susceptible+RG108 (0.4 mg/kg) groups. However, all other groups showed differential methylation of several depression susceptibility genes. In conclusion, aspirin had DNA methylation inhibitory properties comparable to the standard DNA methyltransferase inhibitor - RG108, and exerted its antidepressant effect through this epigenetic mechanism. This study recommends that aspirin should be used as an epigenetic-targeted (adjunct) antidepressant.

Keywords: Acetylsalicylic acid (ASA); Aspirin; Depression; DNA methylation; Gene expression; Whole genome methylation sequencing

S3-09

Assessment of the stress in patients with multiple sclerosis in Morocco

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Abstract

Introduction: Multiple sclerosis (MS) is a disease of unknown origin. However, certain factors may contribute to its onset, notably stress, which the WHO defines as a state of mental worry or tension caused by difficult circumstances. The association between life stress and subsequent MS exacerbations has been demonstrated in several previous studies. Aims: This study aims to assess stress levels in MS patients in Morocco and to identify its determinants and impact on disease progression. Methods A descriptive study was conducted to explore this topic. To evaluate patients' stress levels, we used the Arabic adaptation of the Perceived Stress Scale (PSS). Results The study results reveal that a significant majority of respondents (76%) suffer from stress, with a mean score of 20.81%. Women are relatively more exposed to stress (21.23%) than men (19.73%), and individuals aged 20 to 30 years show the highest stress scores. Patients from the Eastern region had an average score of 19.23%, while those from the Tangier-Tetouan-Al Hoceima region scored 22.14%. Additionally, patients with the progressive form of multiple sclerosis exhibited slightly higher stress scores than those with the relapsing-remitting phenotype. Patients experiencing only one relapse per year had a stress score of 20.10%, whereas those with more than three relapses scored 22.85%. Furthermore, the study found a correlation between disease duration and stress levels: patients whose disease had lasted less than five years scored 21.10%, compared to 20.67% for those with a disease duration of more than five years. Discussion This study highlights that stress levels among surveyed MS patients in Morocco are relatively high. Moreover, stress scores vary according to

patients' socio-demographic and clinical characteristics

S3-10

Combined Effect of Melatonin and Metformin in Mitigating Anxiety and Depression-Related Behaviors in Diabetic Mice Under Immobility Stress: Role of Oxidative Stress

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Abstract

Previous epidemiological findings indicate an association between type 2 diabetes mellitus (T2DM) and behavioral alterations, with stress recognized as a factor contributing to psychiatric disorders. Melatonin (MEL), known for its potent antioxidant properties and various benefits against hyperglycemia and stress-related complications, is particularly intriguing. The primary objective of this study was to investigate whether combining Melatonin and Metformin (MET) could ameliorate biochemical and behavioral changes associated with persistent hyperglycemia, specifically by reducing lipid peroxidation (LPO), a key marker of oxidative stress (OS), in T2DM mice exposed to chronic immobilization stress (CIS). Mice were divided into four groups: normal-control (NC), diabetic treated with MET (D-Met), stressed diabetic treated with MET (D-Ims-Met), and stressed diabetic treated with a combination of MET and MEL(D-Ims-Met-Mel). T2DM was induced experimentally by exposing mice to fructose for 14 days followed by a single intraperitoneal injection of streptozotocin (STZ). Stress symptoms were induced using the chronic immobilization stress paradigm. Anxiety and depression-related behaviors were evaluated using three behavioral tests after treatment periods of 30 days (T1) and 60 days (T2), respectively. Blood samples collected post-sacrifice were analyzed to assess lipid parameters and corticosterone (CORT) levels. Additionally, organs (pancreas and brain) were dissected to measure LPO levels as indicators of OS. The data underwent a one-way analysis of variance, followed by Bonferroni's post hoc test for inter-group comparisons. The results revealed that combining MET with MEL alleviated OS in the brain and pancreas of diabetic mice subjected to CIS for two months by reducing LPO accumulation ($P<0.05$). This combination treatment effectively regulated CORT levels, improved lipid profiles, and reduced the atherogenic index in these mice ($P<0.05$). Moreover, the combined therapy significantly improved depression-related behaviors in this mouse model ($P<0.001$). These findings suggest that the combination of MEL and MET could be promising as a potential antioxidant supplement for patients with T2DM. By mitigating oxidative stress and metabolic disorders, this combination could improve disease management and the quality of life for patients.

S3-11

High-Frequency Stimulation of the Anterior Cingulate Cortex Mitigates Aggression in Socially Isolated Mice

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Abstract
Background: Aggressive behavior is a common symptom in patients with various neuropsychiatric disorders. While standard treatments effectively manage aggression in most patients, a small group remains resistant to pharmacological interventions and is classified as treatment-refractory. Neuromodulation therapies, such as High-Frequency Stimulation (HFS), have emerged as potential alternative treatments for this subset of patients. The Anterior Cingulate Cortex (ACC) is crucial in regulating aggression, with previous studies showing reduced activation of the ACC in socially aggressive mice. Objective: This study aimed to determine whether direct HFS of the ACC can reduce aggressive behavior in mice by enhancing its activation (i.e., long-term potentiation, LTP). Methods: Swiss mice were socially isolated for six weeks, a condition known to increase territorial and aggressive behaviors when introduced to intruder mice. Electrodes were implanted in the ACC of resident mice, which were then divided into two groups: stimulated and non-stimulated. The stimulated group received two trains of 150 pulses at 250 Hz, spaced 60 seconds apart, before encountering the intruder, while the non-stimulated group served as the control. Results: ACC HFS significantly increased the latency to the first attack and reduced the total number of attacks compared to the control group. Furthermore, the stimulation enhanced social behaviors, as evidenced by increased frequency and duration of investigatory actions. Conclusion: These findings suggest that HFS of the ACC may have therapeutic potential in mitigating aggression in socially isolated mice.

S3-12

Trans-cinnamaldehyde attenuates neuronal cytotoxicity and memory impairment in comorbid exposure to sleep deprivation and formalin inhalation in rat model

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Abstract
Background: Synapse impairment is associated with decline in learning ability and memory loss,

which can be activated by enhanced oxidative stress, inflammation and hypermyelination of neurons in the prefrontal cortex (PFC), hippocampus (Hipp), and other brain regions. Thus, we examined trans-cinnamaldehyde (TCA) neuroprotective mechanisms in cortico-hippocampal axis of rat deprived of sleep with concomitant long-time exposure to formalin inhalation. Methods: Thirty-six (36) healthy male Wistar rats (average of 170 ± 10g) were randomly assigned into six experimental groups (n=6). Positive control group (CON), TCA (40mg/kg bw) control group (TCA), sleep deprivation exposure group (SD), 20% formalin inhalation group (FEx), SD+FEx group (SD+FEx) and SD+FEx+TCA group (SD+FEx+TCA). 20% formalin exposure was done via inhalation, with sleep deprivation for 8hr a day, 5 days/week, from day 1 to 56 while TCA was given intraperitoneally, from day 28 to 56 and thereafter, behavioral, biochemical, immunohistochemistry and histomorphology was assessed Results: SD, FEx and SD+FEx exposure provokes loss of body weight across days, impair short- and long-term memory functions and heightened pro-oxidants molecules with marked decreased endogenous antioxidants status in cortico-hippocampal brain region of the animal. The degree of cortico-hippocampal proinflammatory mediators were further upregulated supported with loss of pyramidal neurons, severe white matter degeneration, axonal loss and hypermyelination. In addition, SD+FEx exposure exacerbates cortico-hippocampal Nrf2 immunopositive cells. Herein, all these assaults were mitigated by TCA treatment, due to itsability to modulate Nrf2 protein, oxido-inflammatory signaling molecule and restore cortico-hippocampal neurons. Conclusion: Taken together, our findings showed the positive neuroprotective effects of TCA against neurodegeneration associated with SD+FEx exposure, by reducing oxido-inflammatory molecules, increasing the endogenous antioxidant defense system modulating Nrf2 protein and abating the loss of cortico-hippocampal neurons and histoarchitecture.

S3-13

Neuropsychology and celiac disease: Analysis of neuro-cognitive impacts in children

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Abstract

Background and objective: Celiac disease, a common chronic enteropathy, is a long-term immune disorder linked to an abnormal immune response to ingested gluten in genetically predisposed patients. Celiac disease is systemic, characterized by a multifactorial pathogenesis, with a multifaceted etiology ranging from digestive and extra-digestive symptoms to endocrinological, rheumatological, dermatological, and neuropsychological symptoms. Indeed, according to the

literature, celiac disease is strongly associated with cognitive dysfunction, and psychiatric disorders, which manifest themselves from the juvenile period through to adulthood. This pathology is linked to several neuropsychiatric-type complications, such as cognitive deficits, including memory disorders, acalculia and confusion. Neurodevelopmental disorders are also present in this child population, namely autism spectrum disorders and attention deficit hyperactivity disorder (ADHD). This medical condition, which presents a complex profile, often affecting the infant population and integrating the immunological, genetic and epigenetic axes, can have an impact on the neuropsychological development of sufferers, particularly children. To address this issue, we set out to evaluate the neuropsychological profile of children with celiac disease. Materials and methods: This is a descriptive cross-sectional study, the target population includes children with celiac disease, aged 3 to 14 years, followed at the Children's Hospital, CHU IBN SINA in Rabat. The evaluation of their neuropsychological development will be carried out through the use of adapted (valid and reliable) neuropsychological tests to assess the following cognitive and intellectual dimensions, and also through interviews with the parents of the recruited children. Expected Results and Discussion: We anticipate that this study will provide valuable information on the neuropsychological profile of affected children. Given the known impacts of the disease on mental and cognitive health, we expect to observe significant variability in cognitive performance, and particularly that the analyses will reveal cognitive deficits. We will compare them to previous norms and research. Conclusion: This research will contribute to a better understanding, in our daily practice, of the specific neuropsychological needs of children with celiac disease. The identification of deficient cognitive functions could lead to more targeted remediation, in conjunction with the development of multidisciplinary and interprofessional therapeutic intervention plans for quality socio-educational integration.

S3-14

Exposure to Ibuprofen Differentially Alters the Morphology and Synaptic Integrity of the Prefrontal Cortex and Amygdala of Adult Male Wistar Rat

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Abstract
Background: Ibuprofen, a widely used NSAID with analgesic and anti-inflammatory properties, has shown conflicting effects on the nervous system; some studies suggest neuroprotective effects, while others indicate potential harm. Aim: This research aimed to investigate the specific effects of long-term ibuprofen exposure on the amygdala and prefrontal cortex. Methods: Twenty-four male Wistar rats were divided into three groups of eight animals each and treated orally as follows: Group 1

received distilled water for 28 days, Group 2 received 50 mg/kg of ibuprofen for 28 days, and Group 3 received 200 mg/kg of ibuprofen for 28 days. Body weight was recorded, and behavioral assays (open field, tail suspension, and tail flick tests) were conducted. We assessed oxidative redox parameters, acetylcholinesterase levels, and total protein profiles in the brain and amygdala using biochemical assay kits. Morphological examinations of the prefrontal cortex and amygdala were conducted using H&E and Cresyl Violet staining, along with assessments of astrocytic morphology, synaptic integrity, and neuronal markers using anti-GFAP, anti-Synaptophysin, and anti-NeuN antibodies. Molecular docking analyses were also performed to examine interactions between ibuprofen and specific target proteins (MAOA, oxytocin, and BCHE). Results: Molecular docking indicated that ibuprofen strongly interacted with MAOA, oxytocin, and BCHE in their inhibitory conformations. In the animal study, ibuprofen had minimal impact on the prefrontal cortex; however, effects on the amygdala were pronounced, resulting in behavioral deficits, oxidative stress, neuronal loss, reduced Nissl bodies, astrogliosis, and impaired synaptic plasticity. Conclusion: This study reveals that prolonged use of ibuprofen negatively affects amygdala-related functions, potentially through oxidative stress and oxytocin inhibition, underscoring the need for further research to understand the mechanisms and clinical implications of long-term ibuprofen use.

Keywords : Empathy, Ibuprofen, Amygdala, Prefrontal cortex, Neurons

S3-15

Gender-Based Analysis of Comorbidity between Post-Traumatic Stress Disorder and Social Anxiety Disorder in Rodents

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Abstract

Post-traumatic stress disorder (PTSD) and social anxiety disorder (SAD) often co-occur, yet the underlying mechanisms of this comorbidity remain unclear. This gap is particularly evident in animal models, where studies examining the interaction between PTSD and SAD are scarce. We investigated SAD-related symptoms in mice exposed to traumatic events, aiming to explore the comorbidity between PTSD and SAD. Mice were subjected to two electric shocks and assessed for PTSD symptoms (freezing, avoidance, fear sensitization) and SAD-related behaviors (social interaction, collective and individual exploration, and individual exploration preference). Both male and female mice were included, as sex differences may influence anxiety and fear learning. Our results indicated that PTSD symptoms were more pronounced in males, while both sexes exhibited significantly lower sociability compared to controls. These findings suggest a link between PTSD and SAD, emphasizing the need for

further research to refine treatment approaches for individuals with both disorders.

S3-16

Overview of neuropsychological aspects in the assessment and non-pharmacological interventions for Moroccan Migrants with Dementia

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Abstract

Background and objectives: Dementia diagnosis in Moroccan migrants may present unique challenges and barriers. While non-pharmacological interventions encompass a wide range of neurocognitive and behavioral treatments, it's crucial to address them culturally and linguistically speaking. This presentation explores the complexities of using conventional and Western-based neuropsychological assessments and care for dementia in this population. Methods: A review of cultural, linguistic and clinical barriers can significantly impact test performance. Standardized assessments often rely on language fluency, educational background, and familiarity with specific cultural references. Results: Consequently, Moroccan migrants with limited literacy may score poorly due to cultural differences, leading to misdiagnosis or overdiagnosis of dementia in their host countries. The findings emphasize the need for culturally sensitive and linguistically appropriate neuropsychological assessment. This could involve incorporating tasks and stimuli that are relevant to Moroccan cultural contexts. Additionally, utilizing Arabic-language or adapted Berber versions of assessments is crucial when delivering tailored clinical services for Moroccan migrants. Discussion: Furthermore, this review highlights the importance of considering pre-migration experiences, stress, and potential risk factors for cognitive decline specific to Moroccan migrants. Factors such as lower baseline education levels, social isolation due to migration, and limited access to healthcare in the new country could contribute to cognitive decline. Regarding potential non-pharmacological interventions for dementia management in Moroccan migrants, this might include culturally tailored neuro-cognitive stimulation therapies, social engagement activities that incorporate Moroccan traditions, and music therapy that utilizes familiar musical styles and repertoires. Family involvement and co-therapy are also emphasized as crucial aspects of intervention. By adopting a specific clinical assessment and care, will improve the accuracy of dementia diagnosis and quality of care for Moroccan migrants experiencing cognitive decline in their host countries with respect to their cultural and linguistic features.

Keywords: Migrants; Mental; Health; Migration; Moroccan

Poster Presentations

S3-17

Carrageenan induces hyperactive behaviour and an impairment of social interaction in mouse model

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Abstract
Carrageenan is a compound of interest in the field of the food industry, where it is widely used as a gelling agent or emulsifier (under the category of food additives). It is also utilized in scientific research, where it is injected into the paws of rodents to induce edema for evaluating the anti-inflammatory properties of plants of interest. In the present study, carrageenan (E407) was administered to weaned NMRI mice for a duration of 4 weeks at a dose of 2500 mg/kg/day. Subsequently, behavioral tests were conducted to evaluate the effect of carrageenan consumption on locomotor activity, exploratory activity, anxiety-like behavior, compulsive behavior, and sociability of the mice. The results demonstrate that carrageenan induces hyperactive behavior with an anxious tendency, accompanied by an increase in compulsive behavior, as well as impairment in social behavior among the mice. Thus, our study reveals that the widely consumed food additive (E407), particularly by the general population and children, leads to negative effects on health, specifically on behavior.
Keywords: Carrageenan, Behavioral tests, Central nervous system, Food additive, Mice.

S3-18

Biochemical Assessments following Cannabis sativa Administration in a Rat Model of Stress Triggered Hippocampal Changes

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Abstract
Stress continues to be a rising global health concern, which can trigger neurodegenerative diseases. Available treatment options focus more on alleviation of specific symptoms rather than delay progression of stress triggered neurodegeneration. Cannabis sativa l. (C. Sativa) at a moderate dose is widely regarded as a potent psychoactive, medicinal plant with antioxidative properties. This study assessed the neuroprotective effect of hydroethanolic extract of C. sativa (HCS) on stress-induced changes in the brain of Wistar rats. Twenty-five (25) Wistar rats were used in the present study and

were divided into 5 groups (n=5). Group I (H2O 1 ml/kg); Group II (2 hours of stress); Group III (Diazepam (5 mg/kg) + Stress; Group IV (250 mg/kg C. Sativa. + Stress); and Group V (750 mg/kg of C. sativa + Stress). Treatment was via oral route for 21 days. The neuroprotective effect of HCS was evaluated using biochemical analysis of oxidative stress bio makers (MDA, SOD, CAT and GPx) and endogenous enzyme (AchE) activity of the brain of Wistar rats. Biochemical results revealed alteration in the concentration levels of oxidative stress parameters of the hippocampus in stress-exposed groups. HCS significantly ($p<0.05$) ameliorated stress-induced alterations of biochemical parameters. Findings from this study suggest the efficacy of low-dose C. sativa. in ameliorating stress-induced biochemical alterations in the brain and could be a potential candidate for application in the management and treatment of stress-induced neurodegeneration.

S3-19

Neuroanatomical adaptations in the ocular and visual systems of African tree squirrels, crucial for their arboreal lifestyle.

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Abstract

Background and Objectives: African tree-dwelling squirrels are diurnal rodents known for their exceptional visual acuity; an adaptation crucial for survival in their arboreal habitats. This study aimed to investigate the neuroanatomical adaptations of these squirrels by conducting a detailed histological and immunohistochemical analysis of their ocular structures and key visual brain regions, providing insight into the mechanisms supporting their tree-dwelling lifestyle. Methods: We examined ocular and brain tissues from five squirrels captured at the University of Ibadan, Nigeria. Paraffin-embedded samples were subjected to routine histological processing, special staining, and immunohistochemical analyses. Results: Histological analysis revealed densely packed stromal fibers in the cornea and undulations in the innermost corneal epithelial layer. The basement membrane of the corneal epithelium was strongly PAS-positive, and pronounced pigmentation was noted in the choroid, iris, and ciliary epithelia. The retinal architecture was multilayered with densely packed ganglion cells. Immunohistochemistry showed strong glial fibrillary acidic protein expression in the retinal nerve fiber layer and optic nerve. In the brain, neuroglial cells resembling astrocytes were found in association with pinealocytes and capillaries in the pineal gland. The rostral colliculus exhibited an enlarged structure with densely packed neurons of varying sizes, organized into two laminae. Discussion: The immunohistochemical findings demonstrate robust neural support, particularly in the retinal and optic nerve regions. Additionally, the analysis of key visual brain structures reveals a complex neural organization, comparable to that observed in primates. These results highlight significant neuroanatomical adaptations in the ocular and visual brain regions of African tree squirrels, which are critical for their exceptional visual acuity and sensorimotor coordination, key factors for thriving in arboreal environments. This study offers the first in-depth

characterization of these adaptations, paving the way for future research into the neurobiological mechanisms that support arboreal lifestyles.

Keywords: Ocular adaptations, Neuroanatomy, Arboreal rodents, Histology, Visual processing.

S3-20

Ameliorative Effects of Caffeine on Chronic Stress-Induced Neurobehavioral, Neurochemical and Glial Alterations in Wistar Rats

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Abstract

Prolonged exposure to stress has detrimental effects on health and the consumption of caffeine, mostly contained in energy drinks, has become a widely adopted stress-coping strategy. Currently, there is limited information regarding the effects of caffeine intake on chronic stress exposure. Thus, this study investigated the effects of caffeine administration on chronic stress-induced behavioral deficits, neurochemical alterations, and glial disruptions in experimental rats. Thirty male Wistar rats were randomly assigned to five groups (n = 6): non-stress control, stress control, and caffeine groups of doses 12.5, 25, and 50 mg/kg. The stress control and caffeine groups were subjected to an unpredictable chronic mild stress (UCMS) protocol daily for 14 days. The rats were evaluated for phenotypic and neurobehavioral assessments. Thereafter, the rat brains were processed for biochemical and immunohistochemical assays. Caffeine administration was found to ameliorate behavioral dysfunctions in rats exposed to UCMS. The UCMS-induced changes in brain levels of monoamines, cholinesterases, and some oxidative stress biomarkers were reversed by caffeine. Caffeine administration also produced mild protective effects against UCMS-induced GFAP and Iba-1 expression changes in stress-specific brain regions. These results showed that low and moderate doses of caffeine reversed most of the stress-induced changes, suggesting its ameliorative potential against chronic stress-induced alterations.

Poster Presentations

S3-21

Effects of prenatal stress on puberty onset and reproductive function in mice

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3- Higher Institute of Nursing Professions and Health Techniques Er-Rachidia, Ouarzazate Annex

Abstract
Stress during pregnancy has a major effect on brain development can negatively affect reproductive health. This study aims to use physiological and histological techniques to investigate how prenatal restraint stress affects the onset of puberty, the regularity of the female's estrous cycle, and the alterations in the male testicular in mice. From embryonic day 7.5 (E7.5) to delivery, pregnant mice were exposed to restraint stress for 3 hours daily. The offspring's postnatal and adult development was observed for reproductive problems after weaning. In female offspring, prenatal stress was associated with precocious puberty, suggesting alterations in the timing of HPG axis activation. In addition, adult females exhibited irregular estrous cycles, characterized by prolonged cycle phases, potentially indicating long-term impairments in hormonal regulation and ovarian function. moreover, for male offspring, histological analysis of the testes shows structural anomalies, such as decreased epithelial and seminiferous tubule heights and increased luminal diameter.

S3-22

Unravelling the Complexity of Autism: Exploring the Synaptopathology in a Valproic Acid Sprague-Dawley Rat Model

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Abstract
Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by persistent deficits in reciprocal social communication and interaction, alongside excessive and persistent restricted, repetitive behaviors. Affecting approximately 0.6-2% of children worldwide, its prevalence continues to rise. Understanding the synaptopathology of ASD may provide novel pharmaceutical targets, yet current data on this subject remains inconsistent. The proteins, post-synaptic density-95 (PSD-95) and Vesicular glutamate transporter 1 (VGluT1), integral to excitatory-inhibitory (E/I) balance, play critical roles in synaptic function in both human and animal models. To assess the E/I balance, we quantified and compared the levels of these proteins in the left insular cortex of the Sprague-Dawley (SD) valproic acid (VPA) autism model and compared the protein levels

to behavioral test outcomes. Ethical approval for this research was obtained (2022/03/07/B). Pregnant dams were administered VPA or saline, and male and female pups were used for behavioral and biochemical analyses. Behavioral testing began on postnatal day 23 to assess social interaction (three-chamber test), non-verbal communication (cotton-tip olfactory test), anxiety and repetitive behaviors (light/dark test), cognition (novel object recognition), and motor skills (balance beam test). The PSD-95 and VGluT1 protein levels were quantified using ELISA. Results indicated that VPA-exposed pups exhibited altered physical morphometries, suggesting impaired development. Female VPA-exposed models demonstrated heightened locomotion, repetitive behaviors, and impaired sociability. Male VPA-exposed models exhibited deficits in habituation and dishabituation, coupled with increased anxiety-like behaviors that limited environmental exploration. ELISA analysis revealed elevated PSD-95 and VGluT1 levels in the insular cortex of VPA-exposed models compared to controls, implicating prenatal VPA exposure in increased excitatory synaptic protein concentrations and a consequent E/I imbalance. Our findings highlight the heterogeneity of ASD phenotypes, with male and female models exhibiting distinct behavioral and synaptic profiles. This aligns with the heterogeneity outlined in DSM-5 and underscores the need for broader analyses across cortical regions to fully understand the synaptopathology of autism. These insights provide a foundation for targeted investigations into synaptic dysfunction as a therapeutic avenue in ASD.

S3-23

Testosterone acts as non-photic cue on suprachiasmatic nucleus-driven locomotor activity in the male Jerboa (Jaculus Orientalis)

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Abstract
Jaculus orientalis is a wild rodent that inhabits the high continental shelves of the Middle Atlas Mountains in Morocco. It reproduces seasonally, and a shorter autumnal period of daylight is correlated with lower testosterone concentrations and inversely correlated with metabolic activity in the suprachiasmatic nucleus. We thus investigated whether testosterone acted as a non-photic cue in regulating locomotor activity governed by the suprachiasmatic nucleus using a circadian activity measurement system. Fifty sexually active male jerboas captured in the field during early spring were kept in captivity for two weeks and divided into three groups: sexually active controls, castrated jerboas, and castrated jerboas with testosterone implants or injections. Locomotor activity was recorded for two weeks under a constant long photoperiod, followed by three months in darkness. All rhythm parameters were analyzed using specialized software. Actograms showed that locomotor activity started at the transition from the light to the dark phase and continued during darkness. The

end of activity was observed before light was on for all animals. Under constant darkness, the rhythm was fundamentally circadian with an endogenous period of around 23.9 hours. Two phenotypes were observed during darkness, with most animals exhibiting a phase advance and some showing a phase delay. The period of the rhythm did not change among the three groups; however, the duration of nighttime activity and overall locomotor activity were lower in castrated jerboas. Testosterone implants restored nighttime activity levels to those of the control group. Castrated jerboas showed a higher onset error compared to the controls and the testosterone-treated groups. These findings highlight that in sexually active jerboas, locomotor activity rhythm is circadian and synchronized to a 24-hour cycle through the light/dark cycle. This rhythm is influenced by the sex hormone testosterone, which likely reduces locomotor activity by affecting suprachiasmatic nucleus activity. However, testosterone does not synchronize the rhythm activity.

S3-24

From event enjoyment to career aspirations: how science engagement shapes participant perspectives

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Abstract

Engaging young people in science is essential for fostering curiosity, enhancing science literacy, and inspiring potential careers in scientific fields. However, disparities in access to science education persist, with underrepresented communities such as girls and individuals from low socio-economic backgrounds often excluded from STEM opportunities. This study examines the impact of a one-day science outreach event we organized during Brain Awareness Week 2024 in a suburban town near Paris, targeting underserved communities. Designed to detach science from traditional academic settings, the event introduced participants to neuroscience concepts through interactive booths covering evolution, genetics, programming, behavior, and chemistry topics. Participants engaged in hands-on activities, interacted with scientists from diverse backgrounds and explored various scientific career paths. Pre- and post-event surveys assessed participant demographics, interest in science, perceptions of scientific careers, and feedback on the event's effectiveness. Results indicated that the interactive and inquiry-based format significantly enhanced interest in science and shifted perceptions of scientific careers, particularly among young attendees and female participants. The event also demonstrated that meaningful science outreach can be achieved in a cost-effective manner by leveraging local partnerships. This study underscores the potential of well-designed, community-focused science outreach events to inspire future generations, promote diversity in STEM fields, and enhance public understanding of science. The findings offer a replicable model for inclusive and impactful science engagement initiatives that prioritize accessibility, interaction, and representation. Future efforts should continue to emphasize inclusivity and sustainability to broaden the reach and

effectiveness of science outreach.

S3-25

Distinct ventral hippocampal inhibitory microcircuits regulating anxiety and fear behaviors

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2-International Institute for Integrative Sleep Medicine, University of Tsukuba, Tsukuba, Japan. In emotion research, anxiety and fear have always been interconnected, sharing overlapping

Abstract
brain structures and neural circuitry. Recent investigations, however, have unveiled parallel long-range projection pathways originating from the ventral hippocampus, shedding light on their distinct roles in anxiety and fear. Yet, the mechanisms governing the emergence of projection-specific activity patterns to mediate different negative emotions remain elusive. Here, we show a division of labor in local GABAergic inhibitory microcircuits of the ventral hippocampus, orchestrating the activity of subpopulations of pyramidal neurons to shape anxiety and fear behaviors in mice. These findings offer a comprehensive insight into how distinct inhibitory microcircuits are dynamically engaged to encode different emotional states.

S3-26

Evidence for the involvement of the kisspeptin receptor, Kiss1R, in memory retention

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Abstract
The present study was undertaken to evaluate the involvement of the kisspeptin receptor (Kiss1R) in the development and the expression of conditioned place preference for sexual cues. WT and Kiss1R-KO male mice were conditioned by exposure to sexually receptive females. Following four pairings to each compartment of the conditioned place preference apparatus, two preference tests were performed. WT and Kiss1R-KO were found able to show a preference for the compartment associated with the sexual encounter during the first preference test. However, 24 hours later, Kiss1R-KO males lost their initially observed preference suggesting a deficiency in memory retention. Interestingly, histological assessment revealed that the kisspeptin receptor is highly expressed in key brain regions involved in memory, including the cortex, the hippocampus and the amygdala, suggesting its

involvement in cognitive processing. Further studies are needed to study in depth the involvement of the Kiss1R in memory and cognition.
Keywords: Kisspeptin receptor, Kiss1R, conditioned place preference

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Hippocampal morphology changes and oxidative stress in autism spectrum disorder mouse model following prenatal valproic acid exposure

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Abstract
Autism spectrum disorder (ASD) is a lifelong neurodevelopmental condition characterized by impairments in social communication, along with repetitive and restrictive behaviors, with a higher incidence in males. Valproic acid (VPA), a commonly prescribed antiepileptic and mood-stabilizing drug, has been implicated in the etiology of ASD when used during pregnancy. Maternal exposure to VPA has been linked to congenital abnormalities, cognitive impairments, and an elevated risk of ASD in offspring. Moreover, ASD involves changes in brain connectivity, including atypical neurons and dendrites morphology. To explore this connection, we employed a prenatal VPA exposure (PE-VPA) mouse model of ASD, where pregnant dams received a single injection of VPA. Our study aimed to investigate potential morphological changes and alterations in antioxidant enzyme levels in the hippocampus of PE-VPA mice. Behavioral assessments of the offspring revealed core ASD-like traits, such as reduced sociability and increased repetitive behaviors. Sholl analysis indicated enhanced dendritic branching in the granule and CA1 pyramidal neurons of male PE-VPA mice. In contrast, female PE-VPA mice exhibited reduced dendritic arborization in dentate gyrus granule cells. Both sexes showed elevated dendritic spine density. Additionally, heightened oxidative stress status was observed in the hippocampi of PE-VPA mice, as indicated by significant changes in oxidative stress markers. Our work highlights sex differences in the effects of prenatal VPA exposure and suggests a possible role for hippocampal morphology and oxidative stress in the pathophysiology of ASD. As a result, we point to the need to develop gender-specific, targeted interventions for this disorder.
Keywords: Valproic acid, Autism spectrum disorder, Hippocampal morphology, Oxidative stress.

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Serum C-Reactive Protein and Prediction of Stroke in Hemodialysis Patients

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Poster Presentations

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Abstract

Uremic patients undergoing maintenance hemodialysis (HD) are a population with a high prevalence of cognitive impairment and risk of stroke. Inflammatory biomarkers play a crucial role in stroke prediction in these patients. This study aimed to analyze the variations of the main inflammatory markers with a significant association with stroke prediction in this population. The Scopus, ScienceDirect, and Web of Science databases were searched for articles published between 2020 and 2024. Included articles focused only on hemodialysis patients. Articles describing biomarkers that can predict cardiovascular events in hemodialysis patients were also included. Among the main inflammatory markers predicting this risk of stroke, C-reactive protein (CRP) stands out as a robust indicator of cardiovascular risk and systemic inflammation in the hemodialysis population. Recent studies have revealed that an elevated serum CRP level is positively correlated with an increased risk of stroke in this population, although its clinical use is still limited by the lack of consensus on specific thresholds. The underlying mechanisms mainly include arterial stiffness and an imbalance of thrombo-inflammatory processes, which are directly linked to the development of stroke. The assessment of (CRP) variations, as an inflammatory biomarker, in patients with (HD) could allow a more precise stratification of stroke risk. Further studies will be necessary to integrate this biomarker into routine clinical practices.

Keywords: Inflammatory biomarkers, hemodialysis, stroke.