SNCI-2024

XXXVIII Annual Meeting of Society for Neurochemistry (INDIA)

International conference

on

INNOVATIONS AND FUTURE PERSPECTIVES IN NEUROCHEMISTRY

September 26 - 28, 2024

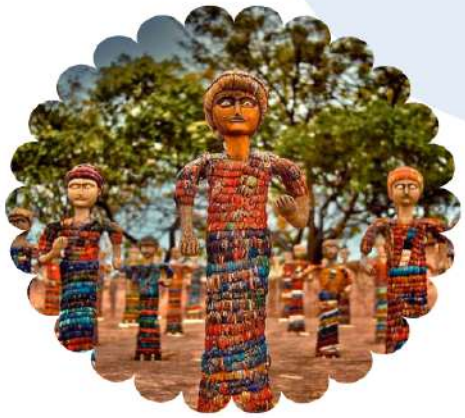
Abstract Book

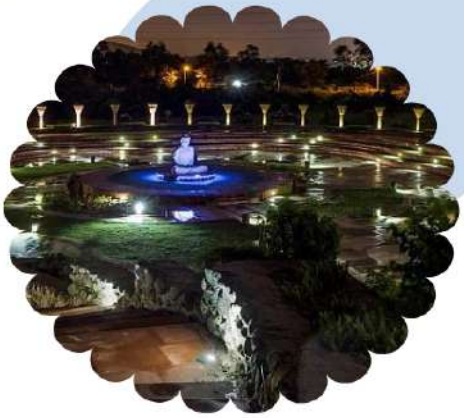
Pre-Conference Workshop on

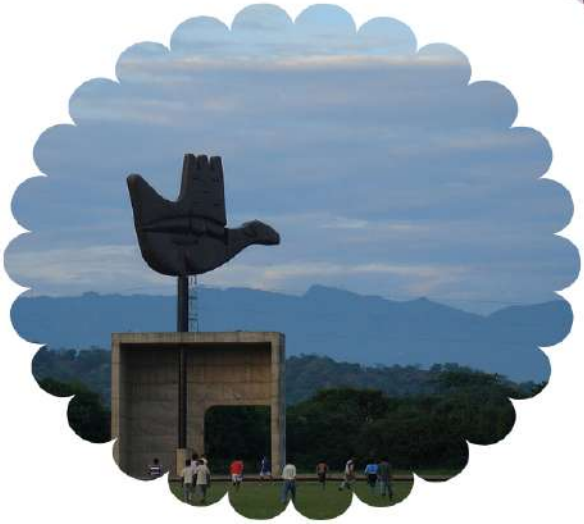
ADVANCED TECHNIQUES IN MOLECULAR NEUROBIOLOGY

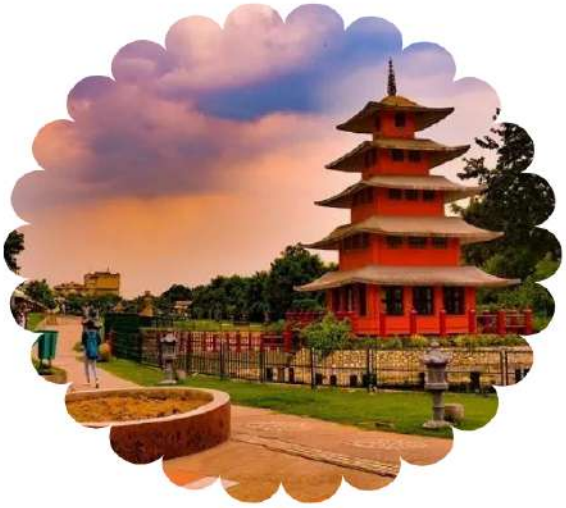
September 23 - 25, 2024

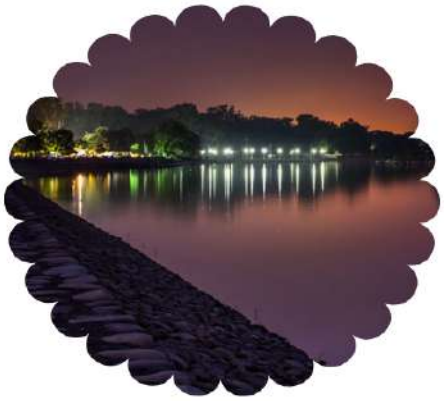
Glimpses of Chandigarh

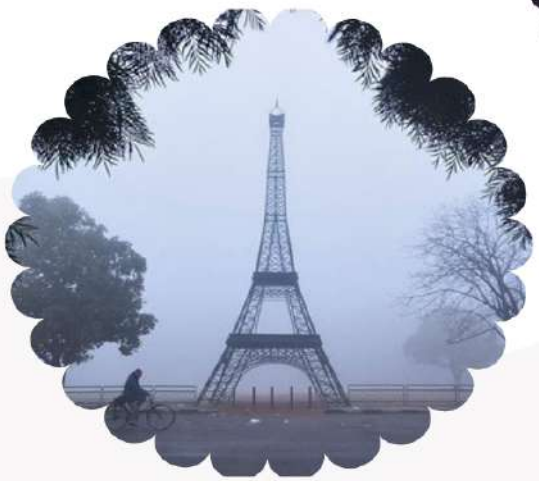












Professor Renu Vig
Vice - Chancellor

PANJAB UNIVERSITY
CHANDIGARH, *India* 160 014

MESSAGE

It is indeed a great pleasure to announce that the Panjab University is organizing the **38th Annual Meeting of Society for Neurochemistry (India)** and International Conference on the theme '*Innovations and Future Perspectives in Neurochemistry*', **SNCI 2024** from September 26-28, 2024. It is proud moment that this event is being organised in Chandigarh for the first time and Panjab University happens to to be the host of the event. I am also delighted that a three-day workshop on the theme '*Advanced Techniques in Molecular Neurobiology*' was also conducted from September 23-25, 2024.

Panjab University takes immense pride in promoting initiatives that contribute to the global advancement of knowledge and SNCI 2024 is a perfect example towards this commitment. The conference, a flagship academic event, focuses on highlighting the prominence of neurochemistry and cutting-edge innovations taking place in the field of neuroscience. We know the world faces an enormous health challenge from increasing number of cases of neurological disorders. Hence, the theme *"Innovations and Future Perspectives in Neurochemistry",* underscores the importance of progressing scientific research and advances in the field of neuroscience. The conference promises to be a dynamic platform for discussions, covering wide range of topics such as mental health & aging, gut-brain axis, glial biology, neurotherapeutics, neurobiology of pain, addiction biology, neurometabolic disorders potential of 'Omics, articificial intelligence and role of alternative medicine in preventing and treating neurodegenerative disorders.

I am glad to note that the conference will feature presentations from both delegates and budding neuroscientists showcasing their work in the domain. I have learnt that this the conference has particiaption from basic scientists, clinicians and engineers which will be a perfect platform to foster meaningful exchanges for innovations in the field.

As the conference unfolds, my warmest wishes to all attendees for a productive and enlightening experience. I fervently believe that this conference will boost the quality of the research and collaborations and ultimately advance our understanding of neuroscience thereby reducing the suffering/burden from neurological conditions.

My heartfelt congratulations to the organizers, participants, and sponsors for their efforts to shape SNCI 2024 into an exceptional event.

(Renu Vig)

FROM THE DESK OF ORGANIZING SECRETARY, SNCI 2024

Prof. Rajat Sandhir

Dear Participants and Esteemed Colleagues,

It is with immense excitement and delight that I extend a warm welcome to you for the 38th Annual Meeting of Society for Neurochemistry (India) and International Conference on 'Innovations and Future Perspectives in Neurochemistry (SNCI 2024), organized by the Department of Biochemistry of Panjab University. This historic event is scheduled to take place from September 26 -28, 2024, with an exciting pre-conference workshop on "Advanced Techniques in Molecular Neurobiology" from September 23- 25, 2024.

The conference will delve into a spectrum of topics covering mental health, aging, gene-environment interactions, glia-associated disorders, neurobiology of pain, gut-brain axis, cognitive & behavioral neuroscience, addiction, neurochemistry in clinical practice, biomarkers in neurodegenerative conditions, neurochemical networks and systems biology, innovations in neuroscience, optogenetics 2D & 3D neuronal cultures and artificial intelligence. The conversations will explore the most recent advancements in neurochemistry aimed at neurodegenerative disorders, molecular targets for therapies and evolution of techniques for better understanding Our delegates represent a rich tapestry of knowledge including renowned neuroscientists, clinicians and practitioners of various medical disciplines and engineers.

The program encompasses a diverse array of sessions, including plenary sessions, invited lectures, young scientist award sessions for oral and poster presentations I am confident that these sessions will foster insightful discussions and catalyze innovative research collaborations.

I extend our heartfelt gratitude to the pivotal support from Panjab University and esteemed government funding agencies like CSIR, SERB (DST), IBRO, UT-DST and DBT. Special thanks are due to our corporate sponsors that include Gentech, SciFinder (CAS) and others for generous contributions that have played a crucial role in making this conference a reality. My heartfelt appreciation goes out to our collaborating institutions such as PGIMER, NABI, PEC, CSIR-CSIO, CSIR-IMTECH for their instrumental role in every aspect of the conference. I would also like to express our sincere thanks to the organizing committee, and the volunteers from the Department of Biochemistry, and everyone involved in contributing to make this event truly memorable.

Finally, I would also like to express our gratitude to the Society for Neurochemistry (India) for entrusting us with the responsibility of hosting this esteemed conference. Your belief in our capabilities is deeply appreciated, and I am honored to have had the opportunity to collaborate with you.

To all the participants, thank you for your active involvement in this meeting and for encouraging your colleagues and students to be a part of this conference. I am confident that your participation will be an enriching and rewarding experience.

Finally, I hope that your stay in the modest accommodations provided in different guest houses of Panjab University will be comfortable. Any inconvenience caused is deeply regretted.

At the last let us all keep the the motto of the SNCI in our mind that "*Brains at work to know how brain works*"

Best regards,

Prof. Rajat Sandhir
Organizing Secretary, Department of Biochemistry, Panjab University, Chandigarh-160014

Date: 25-09-2024

Secretary General Message

***

The Society for Neurochemistry, India, (SNCI) started way back in 1979. The SNCI is a registered body in Hyderabad, Telangana with the University of Hyderabad as its Headquarters with a clear mandate to train young minds for neurochemistry/neuroscience. The society inducts life members into its fold.

During annual meetings, young students are encouraged with various awards, the best poster and best oral presenter. The society also encourages mid-career scientists with the Shri Akundi Narayana Murty award for the scientists/ faculty members below 45 years. Further, the society started an oration award for the clinicians in the name of its founder President Dr. B. Ramamurthi an eminent neurosurgeon and founder Secretary, Prof. K. Subba Rao Oration award for the basic neuroscientists. These awards will promote young students and faculty to take up and continue neuroscience as their career. The society also instituted Lifetime achievement award to honour the senior members who have contributed immensely to the Neuroscience/ Neurochemistry during their career.

The society with the help of life members of the society, organises annual meetings at different places in the country. The society is very keen to organise workshops along with the annual meetings to provide hands-on training to the young neuroscience researchers. Further, the society encourages members to organize local chapter meetings and workshops in their respective institutes on a focused theme.

I am happy that 38th annual meeting is being organized by Prof. Rajat Sandhir, Department of Biochemistry, Panjab University, Chandigarh from 26th to 28th September 2024. This annual meeting precedes the hands-on workshop for the selected participants on the advanced techniques of neurochemistry/ neuroscience.

Sincerely,

(P. Prakash Babu)

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- **About the Conference:**

We are pleased to announce that the "International Conference on Innovations and Future Perspectives in Neurochemistry" under the aegis of the XXXVIII Annual Meeting of Society for NEUROCHEMISTRY (INDIA)" will be held at Panjab University, Chandigarh from September 26 - 28, 2024.

SNCI-2024 is expected to foster knowledge, research expertise and provide a podium for interaction and exchange of ideas between young participants and scientists of national and international repute. The theme of the current conference is "**Pioneering Neurochemical Frontiers: Bridging Molecular Insights and Therapeutic Innovations**". The conference will be a platform to discuss the various aspects of neurochemistry, focusing on recent molecular discoveries and their potential to inform and revolutionize therapeutic approaches.

Numerous scientists from across the world including India with contributions in the field are expected to participate, which would provide opportunities for networking and sharing of ideas. In addition, the conference emphasizes the cutting-edge advancements and forward-looking aspects of neurochemistry, encouraging discussions on the latest research, technologies, and theories that will shape the future of the field. It invites a wide range of topics and participants, fostering collaboration and innovation. We cordially invite you, your colleagues and students to participate in this academic extravaganza.

- **About Chandigarh:**

Chandigarh-The City Beautiful is the first planned city of Modern India. The master plan of the city was developed by Swiss-French architect Charles-Édouard Jeanneret, also known as Le Corbusier. The city is located in the foothills of Shivaliks, which forms a part of the Himalayan ecosystem. Chandigarh derives its name from deity 'Chandi', the goddess of power and has a temple named 'Chandi Mandir' while 'garh' means 'fort'. Chandigarh is the capital of Punjab and Haryana that borders the city. The emblem of Chandigarh "Open hand" signifies openness of thoughts and ideas. The city is known for its modernist architecture and urban design, featuring wide boulevards, spacious parks, and well-organized sectors. The city has a vibrant culture with a blend of traditional Punjabi with modern influences. Chandigarh is home to prestigious institutions like the Postgraduate Institute of Medical Education and Research (PGIMER) and Punjab University.

- **Tourist attractions:**

Some of the best places to visit in Chandigarh include Zakir Hussain Rose Garden, Sukhna Lake, Rock Garden, Chandigarh Bird Park, Terraced Garden, Pinjore Garden, Leisure Valley, City Centre, Government Museum & Art Gallery, Le Corbusier Center, International Doll Museum, Butterfly Park, Fun City and Open Hand Monument. Other nearby attractions include: the Golden Temple at Amritsar (228 kms), Virasat-E-Khalsa at Anandpur Sahib (84 kms), Morni Hills (45 kms), Naina Devi Temple (106 kms) and the hill station of Shimla (113 kms) at a few hours journey from Chandigarh.

More: https://chandigarhtourism.gov.in/

Pre-Conference Workshop Program

DAY 1 September 23rd, 2024

Venue: CIL Auditorium, Panjab University	
8:30 am - 9:30 am	Registration
9:30 am - 10:30 am	Inauguration Ceremony
10:30 am - 11:00 am	Ice-Breaking Session and High Tea
11:00 am - 11:40 am	***Lecture I:*** "***In vitro* and *in vivo* models of Parkinson's Disease"** *Prof. Deepak Sharma* CSIR-IMTECH, Chandigarh, India
11:40 am - 12:20 pm	***Lecture II:*** "**Animal Models for Neurodegeneration Research**" *Prof. Rajat Sandhir* Department of Biochemistry, Panjab University, Chandigarh, India
12:20 pm - 1:00 pm	***Lecture III:*** "**Establishment and Maintenance of Primary Culture as a Preclinical Model for Neurosciences**" *Dr. Ashutosh Rai* Department of Biochemistry, Panjab University, Chandigarh, India
1:00 pm - 2:00 pm	Lunch Break
2:00 pm - 5:00 pm	***Demonstration Session I:*** **Primary Culture, Behaviour, Molecular Techniques** Dept. of Biochemistry, Panjab University

DAY 2 September 24th, 2024

Venue: CIL Auditorium, Panjab University	
9:00 am - 10:00 am	***Lecture I:*** "**Development of Optogenetic Tools for Understanding Neural Signalling and Associated Diseases**" (Online) *Prof. Suneel Kateriya* School of Biotechnology, Jawaharlal Nehru University (JNU), New Delhi, India
10:00 am - 11:00 am	***Lecture II:*** "**Deep Learning Methods for Understanding Olfaction**" *Dr. Ritesh Kumar* CSIO, Chandigarh, India
11:00 am - 11:30 am	Tea Break
11:30 am - 1:00 pm	***Lecture III:*** "**Genotoxicity Assays and Demonstration**" *Dr. Mani Chopra* Department of Zoology, Panjab University, Chandigarh, India
1:00 pm - 2:00 pm	Lunch Break
2:00 pm - 5:00 pm	***Demonstration Session II:*** **Omics & In Vivo Imaging** *Dr. Vikas Rishi, NABI, Mohali* *Dr. Mohit Kumar, NABI, Mohali*

DAY 3
September 25th, 2024

Venue: CIL Auditorium, Panjab University	
9:00 am - 10:00 am	***Lecture I:*** **"Electrophysiology Techniques in Neurobiology"** *Dr. Pradeep Punnakkal* Department of Biophysics, PGIMER, Chandigarh, India
10:00 am - 11:00 am	***Lecture II:*** **"2D and 3D Culture for Neuroscience Research"** *Dr. Pankaj Seth* National Brain Research Centre (NBRC), Manesar, Haryana, India
11:00 am - 11:30 am	Tea Break
11:30 am - 12:15 pm	***Lecture III:*** **"Methodology for conducting Systematic Reviews and Meta-Analysis"** *Dr. Amit Pal* Department of Biochemistry, All India Institute of Medical Sciences, Kalyani, West Bengal, India
12:15 pm - 12:45 pm	Valedictory Session
12:45 pm - 1:30 pm	Lunch Break
1:30 pm - 5:00 pm	***Demonstration Session III:*** **Electrophysiology** *PGIMER, Chandigarh*

Scientific Program

SNCI-2024

XXXVIII Annual Meeting of Society For Neurochemistry (India) and International Conference on

"Innovations and Future Perspectives in Neurochemistry"

26th-28th September, 2024

Venue: Golden Jubilee Hall, Panjab University

DAY 1 September 26th, 2024

8:30 am - 9:30 am	**Registration**
9:00 am - 9:45 am	**Inauguration Ceremony**
9:45 am - 10:15 am	**Dr. B. Ramamurthy Oration Award** **Chairperson**: **Prof. Amitabha Chattopadhyay**, CSIR-CCMB, Hyderabad, India "***Psyche, soma, spirituality & modern medicine***" **Dr. Lokendra Singh**, CIIMS, Nagpur, India
10:15 am - 10:45 am	**Prof. K Subba Rao Oration Award for Senior Scientist** **Chairperson**: **Prof. M.K. Thakur**, BHU, Varanasi, India "***Metabolomics: Real-world applications in managing Neurometabolic Disorders***" **Prof. Rita Christopher**, NIMHANS, Bengaluru, India
10:45 am - 11:15 am	**Plenary Talk** **Chairperson**: **Dr. Rajpal Singh Kashyap,** CIIMS, Nagpur, India "***Cholesterol Sensitivity of a Neurotransmitter G Protein-Coupled Receptor: Signalling and Endocytosis***" **Prof. Amitabha Chattopadhyay**, CSIR Bhatnagar Fellow, CSIR-CCMB, Hyderabad, India
11:15 am - 11:45 am	**High Tea & Group Photo**
11:45 am - 12:15 pm	**Keynote Lecture** **Chairperson**: **Prof. P. Prakash Babu**, UoH, Hyderabad, India "***Elucidating Cellular & Molecular Mechanisms Underlying Murine ß-Coronavirus-Induced Demyelination & Neuroinflammation: Parallels with SARS-CoV2***" **Prof. Jayasri Das Sarma**, IISER Kolkata, India

DAY 1 September 26th, 2024

Symposium IV: Targeting Glioblastomas **Chairpersons: Dr. Ravi Shankar Akundi**, SAU, New Delhi, India **& Prof. D.K. Dhawan**, Panjab University, Chandigarh	
4:40 pm - 5:00 pm	IL 10: "***Antibrain Cancer Potential of Emblica officinalis***" **Dr. Anuradha Sharma**, LPU, Phagwara, India
5:00 pm - 5:20 pm	IL 11: "***Nanotechnology-Based Therapy for Glioblastoma***" **Dr. Fahima Dilnawaz,** Centurion University, Bhubaneswar, India
5:20 pm - 5:40 pm	IL 12: "***Purinergic Receptor Antagonists as Anti-Tumor Agents***" **Dr. Ravi Shankar Akundi**, SAU, New Delhi, India
5:40 pm - 6:00 pm	IL 13: "***Neurochemical Signaling in Tumor Microenvironment of Glioblastoma***" **Dr. Nandakumar Dalavaikodihalli Nanjaiah,** NIMHANS, Bengaluru, India
6:00 pm - 6:20 pm	IL 14: "***Acid & Alkaline Phosphatase Activity as Biomarkers in Brain Tumor Grading***" **Dr. M Prabha**, RIT, Bengaluru, India
6:20 pm - 6:40 pm	IL 15: "***Application and importance of Systematic review and meta-analysis in Experimental & Clinical Research, and Evidence based Medicine***" **Dr. Amit Pal, AIIMS**, Kalyani, India
6:40 pm - 7:00 pm	**General Body Meeting**
7:00 pm - 8:00 pm	**Cultural Program**
8:00 pm onwards	**Dinner**

DAY 2
September 27th, 2024

Venue: Golden Jubilee Hall, Panjab University

8:30 am - 9:30 am	**Registration**
9:00 am - 9:30 am	**Lifetime Achievement Award of SNCI** Chairperson: **Prof. BN Srikumar**, NIMHANS, Bengaluru, India
	Shri Akundi Narayana Murty Memorial Medal for Early Career Scientists **Chairpersons: Dr. M. Varalakshmi**, UoH, Hyderabad, India **&** **Prof VR Sinha**, Panjab University, Chandigarh
9:30 am – 9:45 am	"***A mouse model of Parkinson's disease to evaluate sex differences in the progression***" **Dr. Poonam Thakur**, IISER, Thiruvananthapuram, India
9:45 am – 10:00 am	"***Walking and Talking Proteins: Role in Trafficking of Nociceptors and Chronic Pain***" **Dr. Vinod Tiwari**, IIT-BHU, Varanasi, India
10:00 am – 10:15 am	"***Determining 'Brain Age' as a Measure of Neuroanatomic and Cognitive Health: Unravelling the Vascular Insults of White Matter Hyperintensity on Brain Health***" **Dr. Vivek Tiwari**, IISER, Berhampur, India
10:15 am – 10:30 am	"***Tau-mediated endocytic trafficking of microglial CX3CR1 upon internalization***" **Dr. Subhashchandrabose Cinnathambi**, NIMHANS, Bengaluru, India
10:30 am- 10:50 am	"***Identification of a new molecular player controlling Huntington's Disease pathology***" **Dr. Shuvadeep Maity,** BITS, Pilani – Hyderabad Campus, India
	Symposium V: Mental Health and Aging **Chairpersons: Dr. A.J. Vanisree**, Madras University, Chennai, India **&** **Dr. Aastha Thakkar**, PGIMER, Chandigarh, India
10:50 am- 11:10 am	IL 1: "***CXCL10 perpetuates brain aging: implications neurodegenerative disorder***" **Dr. PN Yadav**, CSIR-CDRI, Lucknow
11:10 am – 11:30 am	IL 2: "***Effects of Finasteride on Learning, Memory and Synaptic Plasticity: Implications for Ageing and Dementia***" **Dr. Bettadapura N Srikumar**, NIMHANS, Bengaluru, India

DAY 2 September 27th, 2024

11:30 am – 11:50 am	IL 3: "***Amino acid-Peptide-Metal based Theranostic Nanoparticles as Potential Neuroprotective Agents for Alzheimer 's disease***" **Dr. Jiban Jyoti Panda**, INST, Mohali, India
11:50 am – 12:10 pm	IL 4: "***Neurovascular coupling in treatment resistant depression: Role of nitric oxide***" **Dr. Kalpana Kumari Barhwal**, AIIMS-Bhubaneshwar, India
Symposium VI: Cognitive and Behavioural Neuroscience **Chairpersons: Dr. Urmi Chatterjee**, University of Calcutta, Kolkata, India **and Dr. Shalmoli Bhattacharya**, PGIMER, Chandigarh, India	
12:10 pm – 12:30 pm	IL 5: "***Neurophysiological correlates of elevated curiosity-like behavior in adolescence rats predisposed to early life stress***" **Prof. T.R. Laxmi**, NIMHANS, Bengaluru, India
12:30 pm – 12:50 pm	IL 6: "***Mitochondrial SIRT3 activation by HKL reverses neurodegenerative changes in the hippocampus of moderate grade hepatic encephalopathy***" **Dr. Anamika Jaiswal**, Ramjas College, DU, Delhi, India
12:50-1:20	IL 7: "***Role of biomarkers in Neurological Disorders***" **Dr. Karthik Vinay Mahesh**, PGIMER, Chandigarh, India
1:20 pm-1:40 pm	IL 8: "***LiH:The New Addition to Metabolic Syndrome***" **Dr. Aastha Thakkar**, PGIMER, Chandigarh, India
1:40 pm-2:00 pm	**Lunch Break**
Symposium VII: Neurobiology of Addiction **Chairpersons: Dr. Preeti Arun**, GMCH-32, Chandigarh, India **& Dr. Abhishek Ghosh**, PGIMER, Chandigarh, India	
2:00 pm – 2:20 pm	IL 8: "***Validation of the Immersive Virtual Reality and Mobile games for Cognitive Assessment: Results from the 82 Young Participants***" **Dr. Veeky Baths**, BITS Pilani, Goa, India
2:20 pm – 2:40 pm	IL 9: "***Sugar Addiction: Is It for REAL?***" **Dr. Mohit Kumar**, NABI, Mohali, India
2:40 pm – 3:00 pm	IL 10: "***Your Gut, Your Mood!!!***" **Dr. Urmi Chatterjee**, University of Calcutta, Kolkata, India

DAY 2 September 27th, 2024

3:00 pm – 4:15 pm	**Dr. G. M. Taori memorial Award for Best Oral Presentation** **Chairpersons**: **Dr. Vikas Rishi**, NABI, Mohali, India **&** **Prof. T.R. Laxmi**, NIMHANS, Bengaluru, India
4:15 pm – 4:40 pm	**Suven Life Sciences Pharma Award for Best Poster Presentation** & **Networking Tea** **Chairpersons**: **Dr. Nitin Singhal**, NABI, Mohali, India & **Dr. Rachna Mehta**, Amity University, Noida, India
Symposium VIII: Neuroinflammation **Chairpersons: Prof. Dinesh Bhatia**, NEHU, Shillong, India **&** **Dr. Jyotdeep Kaur**, PGIMER, Chandigarh, India	
4:40 pm – 5:00 pm	IL 11: "***17β-estradiol and testosterone may regulate epilepsy-associated neuroinflammation in anterior temporal lobe and hippocampus of male rats***" **Dr. Amrita Bakshi**, Ramjas College, DU, Delhi, ndia
5:00 pm – 5:20 pm	IL 12: "***Dysregulated Glutamate Trafficking Driven by Neuroinflammation in Major Depressive Disorder***" **Dr. Neelima Dubey**, DRILS, Hyderabad, India
5:20 pm – 5:40 pm	IL 13: "***Antiviral and neuroprotective potential of Ocimum basilicum against Japanese encephalitis virus in experimental models***" **Dr. Debapriya Garabadu**, CUoP, Bathinda, India
5:40 pm – 6:00 pm	IL 14: "***Olfml3 and Tmem119 could be a new player in microglia functions***" **Dr. Shashank Kumar Maurya**, DU, Delhi, India
6:00 pm – 6:30 pm	IL 15: "***Extracellular vesicles and microRNA crosstalk: Modulators of neuroinflammation in HIV-1 and opioid-associated neurocognitive disorders***" **Prof. Shilpa Buch**, University of Nebraska, Omaha, USA
8:00 pm onwards	**Dinner**

DAY 3 September 28th, 2024

Venue: Golden Jubilee Hall, Panjab University

8:30 am- 9:00 am	IL 1: "***Decoding Parkinson's: The Quest for Answers***" **Dr. Wael Mohamed**, IIUM, Malaysia
8:30 am- 9:00 am	**Registration**
	Symposium IX: Clinical Neuroscience **Chairpersons: Dr. Debapriya Garabadu**, CUoP, Bathinda, India **& Dr. Indu Verma**, PGIMER, Chandigarh, India
9:00 am – 9:20 am	IL 2: "***A Shotgun Metagenomics Sequencing Approach for Studying the Etiology of Undiagnosed Meningoencephalitis Infection in a Tertiary Care Hospital Setting***" **Dr. Rajpal Singh Kashyap**, CIIMS, Nagpur, India
9:20 am – 9:40 am	IL 3: "***Customized Nanoparticle-Based Strategies For The Management of Neurological Disorders***" **Dr. Rehan Khan**, INST, Mohali, India
9:40 am – 10:00 am	IL 4: "***Brain Tissue Segmentation from MRI Scans using Artificial Intelligence***" **Dr. Poonam Saini**, PEC, Chandigarh, India
	Symposium X: Plasticity **Chairpersons: Dr. Shashank Kumar Maurya**, DU, Delhi, India **& Dr. Saravana Babu Chidambaram**, JSS College Mysuru, India
10:00 am – 10:20 am	IL 5: "***Association Of Dietary Patterns With Cognition And Mental Health: From A Vegetarianism Perspective"*** **Dr. M. Varalakshmi**, UoH, Hyderabad, India
10:20 am – 10:40 am	IL 6: "***New Horizons of Neuronal Optogenetics***" **Dr. Suneel Kateriya**, JNU, New Delhi, India
10:40 am – 11:00 am	IL 7: "***Synaptic Plasticity in the Epileptic Brain***" **Dr. Pradeep Punnakal**, PGIMER, Chandigarh, India

DAY 3
September 28th, 2024

Symposium XI: Depression and Stress Disorders
Chairpersons: **Dr. Nandakumar DN**, NIMHANS, Bengaluru, India **&** **Dr. Suneel Kateriya**, JNU, New Delhi, India

11:00 am-11:20 am	IL 8: "***Unravelling the Molecular Nexus of Antenatal Depression and Gestational Diabetes Mellitus***" **Dr. Gokulakrishnan Kuppan**, NIMHANS, Bengaluru, India
11:20 am-11:40 am	IL 9: "***Designing of a modified spontaneous alternation behaviour test to study the working memory paradigms***" **Dr. Akash Gautam**, UoH, Hyderabad, India
11:40 am – 12:00 pm	IL 10: "***Human Microglial Cell Line, Hmc3 Is An Ideal Cellular Model To Investigate Sporadic Amyotrophic Lateral Sclerosis Associated Pathomechanisms***" **Dr. Vijayalakshmi K**, NIMHANS, Bengaluru, India
12:00 pm – 12:20 pm	IL 11: "***Neuroprogression and accelerated aging in psychiatric disorders***" **Prof. Pawan Kumar Maurya**, CUoH, Mahendergarh, India
12:20 pm – 12:40 pm	IL 12: ""***Myasthenia Gravis: Complications and management***" **Dr. Shripad Patil**, NIMHANS, Bengaluru, India
12:40 pm – 1:00 pm	IL 13: "***Impact of Social Isolation Stress on Depression and Anxiety-like Behavior in Zebrafish***" **Dr. Sunil Kumar**, Amity University, Noida, India
1:00 pm-2:00 pm	**Lunch Break**

Symposium XII : Innovations in Neuroscience
Chairpersons: **Dr. Anant B. Patel**, CSIR-CCMB, Hyderabad, India **&** **Dr. Vijayalakshmi K**, NIMHANS, Bengaluru, India

2:00 pm – 2:20 pm	IL 14: "***Blue Brain***" **Dr. Shyamal Mandal**, NEHU, Shillong, India
2:20 pm – 2:40 pm	IL 15: "***Amlodipine Prevents Neuronal Cell Death in Zebrafish***" **Prof. Manorama Patri**, CUHP, Dharamsala, India
2:40 pm – 3:00 pm	IL 16: "***Neurobiology of Opioid Use Disorder***" **Dr. Abhishek Ghosh**, PGIMER, Chandigarh, India

DAY 3
September 28th, 2024

3:00 pm – 3:20 pm	IL 17: "***Alzheimer's disease: reactive astrocytes play a key role in disease pathogenesis***" **Dr. Subhash C. Biswas**, CSIR-IICB, Kolkata, India
3:20 pm – 3:40 pm	IL 18: "***Prenatal VPA instigated mitochondrial damage resulting in autism-like phenotype and its mitigation by bioflavonoid***" **Dr. Mani Chopra**, PU, Chandigarh, India
3:40 pm – 4:00 pm	IL 19: "***Identification of autism disorder using EEG signals***" **Dr. Padmavati**, PEC, Chandigarh, India
4:00 pm – 5:00 pm	**Valedictory Session, Prize Distribution and Vote of Thanks**

ABSTRACTS

PLENARY TALK

Cholesterol Sensitivity of a Neurotransmitter G Protein-Coupled Receptor: Signaling and Endocytosis

Prof. Amitabha Chattopadhyay

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G protein-coupled receptors (GPCRs) are the largest class of molecules involved in signal transduction across membranes, and represent major drug targets in all clinical areas. The serotonin$_{1A}$ receptor is an important neurotransmitter receptor of the GPCR superfamily and is implicated in the generation and modulation of various cognitive, behavioural and developmental functions. In our earlier work, we demonstrated that membrane cholesterol is necessary for ligand binding, G-protein coupling and signalling of serotonin$_{1A}$ receptors. In the overall context of recent high-resolution structures of GPCRs showing bound cholesterol molecules, we previously reported the presence of cholesterol recognition/interaction amino acid consensus (CRAC) motifs in the serotonin$_{1A}$ receptor. In our recent work, we explored the molecular basis of cholesterol sensitivity exhibited by the serotonin$_{1A}$ receptor by generating site-specific mutants of key residues in CRAC motifs in transmembrane helices (TM) 2 and 5 of the receptor supported by all-atom MD simulations. Notably, we showed that a lysine residue (K101) in one of the CRAC motifs is crucial for sensing altered membrane cholesterol levels (Kumar *et al.* (2021) *Science Advances* 7: eabh2922 (recommended in Faculty Opinions (F1000Prime)). These results constitute one of the first reports comprehensively demonstrating that cholesterol sensitivity could be knocked out by a single point mutation at a cholesterol binding site. Our observations are further supported from all-atom molecular dynamics simulations which reveal a tightly bound cholesterol molecule between TM1 and TM2 by establishing polar contacts with K101 that leads to stabilization of extracellular loop 1 (ECL1). Interestingly, the position of this cholesterol molecule is almost identical to a co-crystallized cholesterol molecule in the recently reported high-resolution cryo-EM structure of the serotonin$_{1A}$ receptor, thereby strongly validating the molecular mechanism for cholesterol sensitivity of the serotonin$_{1A}$ receptor proposed by us.

KEYNOTE LECTURE

Elucidating the Cellular and Molecular Mechanisms Underlying Murine β-Coronavirus -Induced Demyelination and Neuroinflammation: Parallels between Murine β-Coronavirus -induced CNS pathologies and SARS-CoV2

Prof. Jayasri Das Sarma, Ph.D., FNASc, FASc

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Neurotropic mouse hepatitis virus (MHV-A59/RSA59) infection in mice leads to acute neuroinflammation due to direct damage to neural cells, which progresses to demyelination, with or without axonal loss. These are key pathological features of multiple sclerosis (MS), a human neurological disease. Our recent studies using the RSA59-induced neuroinflammation model of MS have highlighted a protective role for CNS-infiltrating CD4+ T cells, in contrast to their pathogenic role in autoimmune models. We further explored the molecular interactions between CD40 ligands expressed on CD4+ T cells and CD40 on microglia/macrophages using CD40L-/- mice. Our studies highlight alterations in CD40L expression in the CNS following RSA59 infection. CD40L-/- mice were found to be more susceptible to RSA59 infection, exhibiting reduced microglia/macrophage activation and significantly impaired recruitment of effector CD4+ T cells to the CNS at day ten post-infection. Moreover, CD40L-/- mice showed severe demyelination caused by phagocytic microglia/macrophages, axonal damage, and ongoing poliomyelitis during the chronic phase of infection. These results suggest that CD40-CD40L signaling plays a protective role in mitigating RSA59-induced demyelination, offering a potential target for developing prophylactic strategies against virus-induced demyelination and axonal loss, in contrast to approaches focusing on immunosuppression in autoimmune-driven inflammatory demyelination. Furthermore, since MHV-A59 is a β coronavirus belonging to the same family as SARS-CoV-2, studies in this mouse model have provided insights into the pathogenesis of human CoV's.

INVITED LECTURES

DAY 1

IL-1

Targeting Transient Receptor Potential Channels for the Treatment of Neuropathic Pain Using Pharmacological Approach

Prof. Shyam S. Sharma

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Transient Receptor Potential (TRP) Channels family has been postulated for their involvement in oxidative stress and Ca2+- mediated neuronal death. These channels are a superfamily of nonselective cation channels, which was first cloned from Drosophila melanogaster in the late 1970s and till date, a total of 28 members have been discovered in mammals. There are 28 members of mammalian TRP channels, which are divided into six subfamilies: 1) TRPC (canonical), 2) TRPV (vanilloid), 3) TRPM (melastatin), 4) TRPA (ankyrin), 5) TRPP (polycystic kidney disease) and 6) TRML (mucolipin). TRP channels are activated by various stimuli (polymodal sensors), including receptor stimulation, heat, osmotic pressure, mechanical stress, environmental irritants, menthol, noxious cold stimuli and cellular redox status. These channels are reported to be involved in many disease conditions diabetic complications (neuropathy and cognitive impairment). The most common and severe complication of diabetes is diabetic neuropathy (DN), which affects approximately 50 % of all diabetics. Diabetic peripheral neuropathy (DPN) damages sensory and motor nerves leading to burning pain, allodynia, hyperalgesia, paraesthesia, muscle weakness and night cramps. DPN is not only associated with neuropathic pain. There are several limitations of existing symptomatic treatment. Therefore, there are always need of neuropharmacological agents targeting new pathways for the treatment of diabetic neuropathy. However, their involvement has not been investigated in diabetic neuropathy. Therefore, in our studies we have investigated the role of redox-sensing TRPC5 channels in diabetic peripheral neuropathy using pharmacological interventions. This lecture will emphasize on the therapeutic potential of pharmacological interventions targeting TRP channels (Acknowledgement: Financial support from SERB (CRG/2020/003019)).

IL-2

Prospective study on the effectiveness of Neuromodulation techniques in the treatment of Vestibular Migraine employing tone burst and click stimulation

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Vestibular Migraine (VM) is one of the most common vestibular disorders with unknown etiology and pathophysiology. It is often misdiagnosed with other vestibular and neurological disorders because of familial symptomatology resulting as a challenging disorder with the availability of limited diagnostic methods and limited management options for its treatment. Vestibular Evoked Myogenic Potential (VEMP) is a biphasic response elicited by saccule and utricle using two kinds of stimulus mostly click and tone bursts (TB). The present study includes a total of 60 subjects between the age group of 23-46 years which were assigned into three groups: Group A consisting of Vestibular Migraine, Group B consisting of other variants of Migraine and Group C consisting of Controls. The subjects were tested with OVEMP and CVEMP according to the established protocols and with two different kind of stimulus namely, click and TB (500hz) at 95 db nhl and 105 db nhl respectively. The results showed that when click and TB stimulus were compared in Vestibular Migraine (Group A) at 105 db Nhl in CVEMP and oVEMP in both the ears, only P1 and N1 amplitudes showed significant differences($p>0.05$) at 95 db nhl in right ear, whereas left ear showed no significant differences ($p<0.05$) between tone burst and clicks for both P1 and N1 amplitudes at 95 and 105 db nHL. Hence, it may be concluded that OVEMP is more reliable than CVEMP in diagnosing VM, although cVEMP can also provide a good test-retest reliability for VM. Though, TB is more reliable stimulus in getting better amplitude than clicks; but for diagnosing VM both click and TBs needs to be considered in both CVEMP and OVEMP with different intensities.

Keywords: Vestibular Migraine, Repetitive Transcranial Magnetic Stimulation (r-TMS), t-DCS (Transcranial Direct Current Stimulation), Neuromodulation, Migraine Episode

IL-3

Restoring mitochondrial health: a Promising therapeutic strategy for diabetic peripheral neuropathy

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Diabetic peripheral neuropathy (DPN) is a prevalent and debilitating complication of diabetes, affecting up to 50-60% of diabetic patients. It is characterized by progressive damage to peripheral nerves and manifests sensory, motor, and autonomic dysfunction, leading to pain, numbness, and impaired motor control, which significantly diminishes patients' quality of life. Despite its widespread occurrence, current therapeutic options for DPN are primarily limited to symptomatic pain relief, leaving the underlying pathophysiological mechanisms largely unaddressed. Mitochondrial dysfunction has emerged as a key factor in DPN pathogenesis. Hyperglycemia-driven oxidative stress induces excessive production of reactive oxygen species (ROS), which damages mitochondrial DNA, proteins, and lipids, leading to impaired mitochondrial function. This disruption in mitochondrial activity results in reduced ATP synthesis, causing an energy imbalance in neurons. The resultant accumulation of dysfunctional mitochondria exacerbates neuronal degeneration, contributing to the progression of DPN. Therapeutic interventions targeting mitochondrial health may be beneficial in addressing the root causes of DPN. Antioxidants such as sulforaphane and resveratrol are being investigated for their ability to mitigate ROS-induced damage and support mitochondrial function. Additionally, activation of critical regulators of mitochondrial health, including PGC1α and SIRT1 has demonstrated potential in restoring mitochondrial dynamics, improving neuronal energy homeostasis, and enhancing cellular repair processes. Our research further explored neuroprotective pathways implicated in DPN, focusing on oxidative stress responses (Nrf2, NF-κB, NLRP3) and mitochondrial regulation (AMPK, PGC1α, SIRT1) in both cellular and animal models. Our results suggest that targeting mitochondrial dysfunction, mitophagy, and restoring mitochondrial health can significantly reduce functional, behavioral, and biochemical deficits in DPN.

IL-4

Understanding α-Synuclein Oligomer Structure by Raman Spectroscopy

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α-Synuclein is a presynaptic neuronal protein and is linked genetically and neuropathologically to Parkinson's disease (PD). One of the major hypotheses is that alpha synuclein oligomer are the main toxic agent that is directly linked to the pathology of PD and it shows more dopaminergic neuronal cell death. We have made and purified two different types of oligomers e.g., lyophilized oligomer (LO) and heat induced oligomer (IO). Lyophilized oligomers showed more toxicity compared to induced oligomer in SH-SY5Y neuronal cell line. The structural changes were monitored by circular dichroism (CD) and Raman spectroscopic analysis. We also studied and hat compared to wild type aS, the S129A and S129W mutants displayed increased structural stability and a high tendency to adopt α-helical secondary structures. Cytotoxicity tests conducted on SH-SY5Y neuronal cell lines demonstrated that the aggregates formed by the mutant proteins were potentially less harmful than those from the wild type protein. We also are attempting to study the morphological features of the oligomers by AFM and TEM analysis.

IL-5

Effect of rapid eye movement sleep (REMS) deprivation on expressions of tyrosine hydroxylase and monoamine oxidase in different areas of rat brain: implications with REMS in health and diseases

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Rapid eye movement sleep (REMS) loss affects almost all physiological processes, and itself is affected in most altered conditions. Interactions among the pedunculo-pontine (PPT) cholinergic REM-ON neurons, and locus coeruleus (LC) noradrenaline (NA)-ergic REM-OFF neurons form the basic scaffold for REMS regulation. REMS helps maintain NA at an optimum level in a healthy individual, while it is increased upon REMS loss; and the increased NA is associated with REMS- loss associated disorders. The effective level of neurotransmitter (including NA) depends on its synthesis, release, and degradation. As symptoms of REMS-loss vary, we proposed that underlying cause for associated changes in the level of NA could have a bearing with variations in REMS-loss associated symptoms. Therefore, we proposed that an understanding of the transcriptional and translational regulation of factors modifying the synthesis and degradation of NA might help us explain REMS-loss associated pathophysiological symptoms. In this study, rats were REMS deprived for 96h using classical flowerpot method. Brain regions related to REMS regulation viz. LC, PPT and area unrelated to REMS viz. hippocampus, were dissected out for evaluation of protein and gene expressions and associated histone modifications of tyrosine hydroxylase (TH), and monoamine oxidase-A (MAO-A). Upon REMSD, TH gene expression increased significantly and showed increased H3K14-acetylation and decreased H3K9-dimethylation, while opposite modifications were seen for MAO-A gene in LC and PPT. All the altered gene and protein expressions returned or tended to return to normal levels after recovery and prazosin treatment. The findings support our contention, which may be exploited for amelioration of REMS-loss associated disorders.

Keywords: Gene expression; Histone modifications; Noradrenaline; Protein expression; REMS loss.

IL-6

Association of Pax6, Vitronectin, Trim2, Trim9, and the ratio of α-Syn and ß-Syn with non-motor and motor functions in brain of MPTP-treated mouse

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Pax6, a multifunctional proteins and transcriptional regulator, proves critical for maintaining functional anatomy of the brain, spinal cord, eyes, pituitary, nose, and pancreatic alpha-cells. Patients, who suffer from tremors, impaired movement, and cognition, show reduced levels of Pax6 (Paired Box6) and the presence of Lewy bodies. The knockdown of Pax6 affects S100β, Gfap, Bdnf, Tgfβ, Ngn2, and p53. However, overexpression of Pax6 in MPTP (1-methyl- 4-phenyl-1,2,3,6-tetrahydropyridine)-treated cell line increased survival and decreased oxidative stress. It is presumed that Pax6 regulates α-synuclein, a part of Lewy bodies, and the Pax6-Tgfβ axis is critical. The Pax6 may serve as a connecting link between motor and non-motor symptoms of PD. This report presents a comprehensive evaluation of metabolomics and proteomics of the brain of PD mouse model treated with MPTP and immunoprecipitation assay followed with bioinformatics analysis. Adult (6-8 weeks) mice of AKR strain were used for experiments as per approved guidelines (IAEC No. BHU/DoZ/IAEC/2018-19/002 dated 14.08.2019). The adult mice were injected MPTP (2mg/kg) intra-peritoneal for 21days. The control mice were also given normal saline for consecutive 21 days. Thereafter, mice were sacrificed, and brains were isolated and processed for metabolomic as well as proteomic studies, ChIP and ChIP-sequencing, RNA- sequencing and functional analysis. Enriched genes and pathways were evaluated through Eukaryotic Promoter Database, DAVID, g: Profiler, GENEMANIA and STRING database, KEGG pathway. The Pax6 interacts with genes and proteins like Snca, Sncb, App, PaxIP, TnfIP, S100β, Tgfβ, p53, Gfap, Trim2, 9, Nptx1, Nptxr, Tau, Slc25a12, Ctnnd1, Cntn1, Stx12, Gabarap11, and Gabarap12. Pax6 is involved in neuroprotection and neurodegeneration by regulating protein aggregation, mitochondrial dysfunctions, neuroinflammation, neurotransmitter metabolism, and oxidative damage. Pax6 regulates cascades of genes and proteins involved in motor and non-motor functions. Levels of Pax6 and the ratio of α-Synuclein and ß-Synuclein would be useful for accessing the pathological status and differential diagnosis of Parkinson's disease. Enolase and neurofilaments may represent the markers for neural stress.

IL-7

Ashwagandha Bioactives to Bioactivities: Biochemistry, Biotechnology and Bioinformatics aided molecular insights

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Ashwagandha (*Withania somnifera*) is most commonly used and highly trusted for a variety of health benefits in Indian traditional system of home medicine, Ayurveda. We have been investigating bioactive components and molecular mechanisms of their action by cell culture based molecular assays. Alcohol, water and DMSO based extracts of Ashwagandha leaves were prepared. A variety of human cancer and normal cell types were treated with these extracts, and subjected to a wide range of biochemical, bioinformatics, molecular and imaging assays. Alcoholic extract of leaves (i-Extract) showed the presence of Withaferin-A (Wi-A) and Withanone (Wi-N), and caused selective killing of cancer cells. Molecular mechanism of the later as resolved by bioinformatics and experimental assays endorsed activation of tumor suppressor protein p53, accumulation of oxidative stress and mitochondrial dysfunction, inactivation of telomere maintaining mechanisms, inactivation of NF-kB signaling. Intriguingly, cells treated with low concentrations of these compounds showed better viability under stressed conditions. Besides, methoxy-Withaferin A (mWi-A) lacked anticancer activity, but exhibited remarkable antistress effect. In an independent project, we performed a three-way blinded screening of natural compounds for antistress activity, 4/70 compounds were found to provide protection against oxidative and metal stress. Molecular assays of the stressed cells treated with low nontoxic concentrations of these four (Wi-A, Wi-N, methoxyWi-A, and triethylene glycol, TEG) compounds revealed protection against apoptosis, ROS accumulation, mitochondrial dysfunction, DNA damage and protein aggregation. Wi-N and TEG offered protection against intrinsic replicative stress accumulation in normal human fibroblasts and attenuated stemness of cancer cells as validated by decrease in their clustering and increase in differentiation ability. The findings largely support antistress, antiaging and anticancer potentials of Ashwagandha bioactives. In context of chronic stress and aging being closely connected with neurodegeneration and cancer, our studies suggest the multiple benefits of chemotypically defined dose-dependent applications of Ashwagandha leave extracts.

IL-8

Stroke Induces Subcellular Organelle and Distal Organ Dysfunction

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Stroke is a significant global health issue, ranking as the second leading cause of death and disability. In India, it is the fourth leading cause of mortality. The complexity of stroke pathology, which activates multiple cell death pathways, necessitates thorough investigation. Currently, thrombolytics are the only direct treatment available, highlighting the need for alternative therapeutic strategies. This study explores post-translational modifications (PTMs) of proteins, such as ubiquitination, SUMOylation, and phosphorylation, which influence neuronal death in stroke. Clinical samples were collected from patients who underwent decompressive craniotomy or craniectomy following large Middle Cerebral Artery (MCA) infarcts. Additionally, rat models of MCA occlusion were used to analyze protein ubiquitination. The findings include altered necrotic morphology, nuclear expression of SUMO-2/3, accumulation of ubiquitinated proteins, and phosphorylation of eukaryotic initiation factor 2 alpha (peIF2α), a marker of endoplasmic reticulum (ER) stress. Stroke not only activates multiple cell death pathways but also impacts other organs, such as the kidneys. The study highlights the systemic complications of stroke, emphasizing the importance of understanding these non-neurological consequences. A notable discovery was that apocynin, an antioxidant, reduced stroke-induced brain damage by regulating oxidative and ER stress. In rats treated with apocynin after MCA occlusion, there was reduced ER stress in the kidneys and improved glomerular morphology, although ubiquitination persisted. The findings suggest that stroke triggers ER stress signaling in the kidney, contributing to acute and chronic kidney dysfunction. The study underscores the need to explore brain-kidney interactions in stroke pathology and could inform the development of novel therapeutic approaches for stroke-induced systemic complications. Understanding PTMs in stroke could provide new insights into stroke pathophysiology and potential treatments for both brain and kidney damage.

Keywords: Stroke; Ubiquitin; SUMO; peIF2α; Neuroprotection; Organ stress; Kidney dysfunction; ER stress; Nephroseq.

IL-9

Changing circadian dynamics with Aging and Neurodegeneration: Therapeutic Interventions

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Aging is associated with significant changes in the basic parameters of the circadian timing system (CTS), leading to circadian dysfunction. The interplay between the core circadian machinery and a network of interconnected transcriptional and translational feedback loops enables the SCN to maintain daily 24-hour rhythms including the sleep-wake cycle. To understand clock dynamics, we have extensively investigated various behavioural, histological, biochemical, and molecular parameters in aging and Neurodegeneration. Furthermore, we have studied the therapeutic effects of melatonin and herbal nutraceuticals towards developing novel treatments for circadian dysfunction, promoting good health, and increasing longevity.

Male Wistar rats of various age groups and animal models for Parkinson and Alzheimer Disease were used. Tissue samples were collected at various time points Zeitgeber time (ZT) such as 0/24, 6,12 and 18. Various techniques behavioural, histological, biochemical, and molecular such as enzyme assays, RP-HPLC, qRT-PCR, 2-D protein profiling etc. along with statistical tools such as R-program Pearson correlation ,one way ANOVA were used for data analysis.We observed alterations in daily locomotor rhythms, clock gene expression, serotonin metabolism, protein profiles, antioxidant enzyme activity, immune gene expression, and molecular markers of inflammation, learning, and memory. Our findings suggested differential alterations in the various parameters in aging, PD and AD. Therapeutic interventions used helped restore the functional integrity of the CTS differentially. This work has implications for developing novel treatments for circadian dysfunction, promoting good health, and increasing longevity.

IL-10

Anti-brain Cancer Potential of *Emblica officinalis*: A Promising candidate for complementary approach of cancer therapeutics

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Brain cancer remains one of the most challenging malignancies to treat due to its complex nature and limited therapeutic approaches. In recent years, there has been growing interest in exploring natural compounds with anticancer properties as alternative or complementary therapies. Emblica officinalis (Indian gooseberry), known for its rich phytochemical content, has shown potential as a potent anticancer agent in various malignancies, however, there is no evidence of anti-brain cancer potential of this plant. So, the current study investigates the anti-brain cancer potential of E. officinalis fruit extracts, focusing on its effects on glioblastoma multiforme (GBM), the most aggressive form of brain cancer. Our preliminary in vitro studies revealed that different E. officinalis extracts (Aqueous, 50% aqueous ethanolic and methanolic) inhibited the proliferation of human glioblastoma cells (U87-MG) in a dose-dependent manner with methanolic extract showing most effective results. The tested extracts also showed anti-migratory potential in wound scratch assay with lowest percentage gap closure showed by methanolic extract. Phytochemical analysis and GC-MS examination further suggested the anti-oxidant rich nature of these extracts. We are further optimising the nano-delivery of these extracts to enhance the efficacy and preparing for detailed molecular studies. These preliminary findings thus suggest that this highly popular plant may also serve as potential target for developing novel therapeutics for brain cancers. However, further investigation into the molecular mechanisms underlying the anti-brain cancer effects of. E. officinalis and its potential application in clinical settings are required.

Keywords: *Emblica officinalis*; Glioblastoma; Brain cancer; Anti-oxidant; Complementary therapy

IL-11

Nanotechnology-based therapy for glioblastoma

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Glioblastoma multiforme (GBM), the most aggressive form of brain tumor, has limited clinical prognosis. Further, the aggressive and heterogeneous nature of GBMs has made the standard treatment options more challenging. The presence of blood brain barrier (BBB) impedes the available treatment and makes adequate treatments such as radiotherapy and chemotherapy unattainable. Recent advancement of nanotechnology, has enabled the efficacious treatment of glioblastoma through different types of nanocarriers. Introduction of nanomedicine has proven the potentiality for persistent brain disorders. Future therapy will be profoundly dependent on the development of nanocarriers to address the specific need of the glioblastoma patient.

IL-12

Purinergic receptor antagonists as potential anti-tumor agents

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The pro-inflammatory enzyme cyclooxygenase 2 (COX-2) has been known to impart metastatic property to cancer cells. However, blocking of COX-2 with nonsteroidal anti-inflammatory drugs or COX-2- specific inhibitors has failed in clinical trials due to adverse effects associated with their prolonged use. We hypothesize that chemotherapy increased levels of extracellular ATP (eATP) in the tumor microenvironment, which in turn enhances COX-2 expression several-fold by acting on purinergic (P2) receptors. In this study, we show that blocking of P2 receptors significantly reduced tumor growth in a mouse model of lymphoma. Tumors were induced in mice through subcutaneous injection of syngeneic EL4 lymphoma cells. Various P2 receptor antagonists were injected within the tumors after they were palpable. The broad-spectrum P2 receptor antagonist, suramin, P2X7 receptor-specific antagonist, oATP, P2Y6 receptor-specific antagonist, MRS 2578, and P2Y12 receptor-specific antagonist, AR-C 69931, all showed significant arrest in tumor growth. Both suramin and AR-C 69931-treated tumors showed strong reduction in COX-2 expression and modulation of various metastatic markers. Disaggregated cells from AR-C 69931-treated tumors, when injected intravenously in naïve mice, did not exhibit metastasis in various tissues which was observed in mice injected with cells from saline-treated tumors. Our results show that blocking of P2 receptors through the use of specific P2 receptor antagonists, dubbed as P2 receptor-based anti-inflammatory drugs (PBAIDs), therefore, is a therapeutic alternative to inhibit COX-2 expression, and thereby, arrest tumor progression and metastasis.

IL-13

Neurochemical signaling in tumor microenvironment of glioblastoma and targets

Dr. Nandakumar Dalavaikodihalli Nanjaiah
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Glioblastoma belongs to WHO grade 4 as per CNS tumor classification. It is the most common primary brain tumor, with poor prognosis and frequently relapse despite neurosurgical resection and radio-chemotherapy. It is highly invasive having extensive migratory and infiltrative growth. Also, it develops resistance to the current repertoire of chemotherapeutic agents limiting therapeutic success. The tumour microenvironment of glioblastoma is highly heterogeneous and multiple factors play role in its growth and progression. Many oncogenic pathways are simultaneously active in one tumor. Hence, new therapeutic approaches need to be elucidated for better management of glioblastoma.

IL-14

Acid and Alkaline Phosphatases Specific activities and Expression as Efficient Biomarkers in Brain Tumor Grading and Correlation with primary culture and lithium effect on GBM cell lines

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Acid Phosphatase (ACP) and Alkaline Phosphatases (ALP) are hydrolases remove phosphate groups from protein and nucleic acid respectively for the regulation of cell function from ACP as lysosomal defence function and ALP membrane-bound as a barrier of the cell.

The ACP and ALP-specific activities of Meningiomas (n = 75) and gliomas (n = 81) were compared among brain tumors, normal brain, and derived primary cell culture. Total Protein and Phosphatases assays estimated by Spectrophotometer and Native PAGE Gel Electrophoresis. Brain tumor and primary explant lysosome studies were performed with an electron microscope.

Average ACP specific activity exhibited 9.32617 ± 4.1144 for meningiomas (n = 55) and 5.91 ± 5.8305 for gliomas (n = 60) respectively as compared to normal brain 7.104 ± 1.33 (n = 120) nm/min/mg of protein. Average ALP exhibited 37.1862 ± 39.91 (n = 36) for meningiomas and 5.91 ± 5.83 (n = 60) for gliomas respectively as compared to normal brain (n = 117) 2.463 ± 1.01 nm/min/mg of protein. ACP and ALP exhibited higher activities for meningiomas but not for gliomas as compared to normal brain, in contrast, both expressed more activities in the majority of glioma cell lines and lower in meningioma cell lines. Interestingly gliomas exhibited similar average specific activities for ACP and ALP. While GBM IV exhibits lower ALP activities and higher ACP activity correlate too many storage lysosomes from Electron microscopic observation as compared to meningiomas.

Higher ALP activities can be surrogate markers from meningiomas G-I, G-II to G-III respectively. However, meningiomas G-III are similar to gliomas excluding Anaplastic Oligodendroglioma G- III similar to Meningiomas G-I.

The GBM cell lines total protein decreased with the lithium chloride in two GBM cell lines including LN229 and U251 co culture. ALP activities with Lithium may answer for understanding the cancerous cell properties and is a positive modulator for activating ACP in glioblastoma cell lines showed anti-cancerous activity.

Hence Lithium can be conjugated with anti-cancerous drugs for their efficiency and targeting on cancer cells. Therefore, an ALP level in meningiomas indicates complementary diagnosis as antibody-ALP conjugates with anticancer drugs for efficiency in targeting brain tumor reduction.

Keywords: Specific activity; Acid Phosphatase; Alkaline Phosphatase; Anaplastic Oligodendroglioma G-III; Meningiomas G-I; GBM Cell lines; Lithium

IL-15

Application and importance of Systematic review and meta- analysis in Experimental & Clinical Research and Evidence based Medicine

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Despite years of extensive research on copper (Cu) in the neurodegeneration/Alzheimer's disease/Cancer field, the definite causation/contributing effect is not completely established although the role of Cu in the physiological/pathological mechanisms in various diseases, particularly neurodegeneration and cancer, has increased tremendously. With the discovery and establishment of Cuproptosis (Copper dependent cell death) pathway, Cu has again caught the focus of the researchers with more and more research focusing on primarily on exploiting the translational role of cuproptosis in neurodegeneration/cancer. However, the field of Cu research is marred with various laboratory limitations and lack of high quality/reproducible Systematic Review & Meta-Analysis (SRMA) in the era of evidence-based medicine.

Issues related to Cu mediated research in neurodegeneration/cancer: Laboratory limitations

- Lack of multi-centric high-powered research studies
- Gold standard vs non-Gold standard equipment for Cu quantification
- Lack of uniform tissue sample preparation methodology for Cu quantification
- Lack of studies providing Cu levels in different stages/grades of the neurodegeneration/cancer
- Lack of specific biomarker for Cu status
- Multi-elemental & multi-nutrients interaction in the biological setting

INVITED LECTURES

Day 2

IL-1

CXCL10 perpetuates brain aging: implications neurodegenerative disorder

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Chemokines are 8 to 14kda small secreted proteins that recruit and activate immune and non-immune cells both in vivo and in vitro. Chemokine receptors belong to the GPCR family, and multiple lines of emerging evidence suggest that several chemokines are elevated in the brain of neurodegenerative disorders. We observed increased CXCL10 expression in an age-dependent manner in mice brains. This leads us to hypothesize that CXCL10, being a component of SASPs, may aggravate/perpetuate the brain aging process and, finally, neurodegenerative diseases. To test this hypothesis, we treated the primary cortical neuron (DIV-7-8) and found increased expression of proteins that regulate cellular senescence. Additionally, we found that CXCL10 attenuates the autophagy in brain tissues as well as in primary cortical neurons. Finally, we demonstrated that increased CXCR3 (cognate receptor of CXCL10) signaling negatively alters glutamatergic neurotransmission in vivo as well as in vitro. Overall, our observation supports the hypothesis that CXCL10, an agonist of CXCR3, facilitates brain aging and could be targeted for the management of ageing-associated CNS disorders.

IL-2

Effects of Finasteride on Learning, Memory and Synaptic Plasticity: Implications for Ageing and Dementia

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One of the key enzymes responsible for the neurosteroid production is 5alpha- Reductase (5alpha-R), which catalyzes a rate-limiting step. 5alpha-R inhibitors such as finasteride are used in older men to treat benign prostatic hyperplasia. Clinical reports indicate that finasteride treatment results in depression and cognitive dysfunction in some subjects. However, the molecular mechanisms have not been completely understood. Accordingly, we examined the effects of short-term 5alpha-R inhibition using finasteride on learning and memory in different spatial and non-spatial paradigms such as radial arm maze, Morris water maze, novel object recognition and location tasks. We subjected male Wistar rats to repeated finasteride administration (10, 30 or 100 mg/Kg, s.c.) over a period of 6 days followed by cognitive evaluation. Short-term finasteride administration at 100 mg/Kg. s.c. resulted in impairment in novel object location but not recognition tasks. In the radial arm maze task, it impaired retention of the task. In the Morris water maze task, acquisition and retention was assessed. Administration of finasteride before but not after the acquisition trial, impaired learning. When finasteride was administered after acquisition, memory was impaired. To examine the mechanisms, we evaluated synaptic plasticity in the hippocampus and plasma corticosterone levels. Basal synaptic transmission was impaired by finasteride, while paired-pulse facilitation was not affected. Interestingly, ex-vivo field potential recordings in the Schaffer Collateral-CA1 synapses also showed that hippocampal LTP is impaired. Further, corticosterone levels were increased by finasteride administration. These results indicate interesting effects of 5-alpha reductase inhibition on cognition in male rats. These not only have implications for the adverse effects of finasteride in the treatment of BPH, but also for the role of neurosteroids in cognition in ageing-associated dementia. Further research in this area has potential for development of novel neurosteroid-based therapeutics to treat age-associated dementia and cognitive disorders.

Keywords: finasteride, treatment-resistant depression, anxiety, finasteride, neurosteroid, cognition

IL-3

Amino acid-Peptide-Metal based Theranostic Nanoparticles as Potential Neuroprotective Agents for Alzheimer ’s disease

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Alzheimer's disease (AD) is a progressive neurodegenerative and age-related disorder characterized by cognitive decline, particularly in memory, executive function, and language. It is the furthermost common form of dementia among older adults. The key neuropathological feature of AD is the accumulation of neurotoxic amyloid-β (Aβ) polypeptides, which form abnormal extracellular senile plaques that damage the hippocampus and prefrontal cortex. Current treatment options are limited, focusing primarily on symptom management. While cholinesterase inhibitors and NMDA receptor antagonists provide some cognitive stabilization, their effects on cognitive derailment are minimal.[3] Developing highly efficient scavengers that target β-amyloid protein (Aβ) plaques has been identified as a promising approach for preventing and treating AD. In response to this challenge, we synthesized various peptide/amino acid and catecholamine-metal nanocomposites as neuroprotective and anti-amyloid agents. These nanocomposites not only prevent amyloid-β fibrillization and promote fibril disaggregation but also alleviate the damage caused by amyloid-β plaques. This includes normalizing the oxidative microenvironment and rescuing neuronal death and synaptic loss, leading to significant improvements in N2A and SH-SY5Y neuronal cells. Some of these nanocomposites are even self-fluorescent and specifically target beta-amyloid, making them a promising tool for diagnosis. To further enhance neuroprotection, some nanocomposites were loaded with brain-derived neurotrophic factor (BDNF), a neuronal growth factor, resulting in a marked increase in the protection of neuronal cells. Thus, these powerful nanocomposites, skillfully fabricated using brain targeting moieties, amyloid inhibitors and antioxidants as precursors, present a new approach for designing multifunctional scavengers that target amyloid plaques and exhibit neuroprotective effects. Additionally, their potential use in diagnostics makes them valuable theranostic tools for Alzheimer’s and other age-related mental disorders.

Keywords: Alzheimer’s disease; Catecholamines; Neuroprotection; Anti-inflammation, Brain targeting.

IL-4

Neurovascular coupling in treatment resistant depression: Role of nitric oxide

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Despite considerable focus on mental health in recent years, treatment resistant depression (TRD) continues to considerably influence global burden on health. TRD is defined as a current episode of depressive disorder where in there is no remittance after at least two adequate trials of antidepressant compounds with different mechanism of action. With a high prevalence of nearly 30-40% of the MDD patients, TRD has emerged as a major challenge for clinicians worldwide. Our previous study suggests strong association of TRD with serum tetrahydrobiopterin and intima media thickness of carotid artery in middle aged population. The findings suggest lower reward responsiveness (BAS-RR) and higher BIS scores in TRD patients along with differentially higher intima-media thickness of left internal carotid artery. Higher depression severity at all stages of the study was correlated with lower tetrahydrobiopterin and BAS-RR scores. We, therefore, suggest that vascular depression resulting due to increased intima-media thickness of left carotid artery and lower tetrahydrobiopterin could be contributing factors for treatment resistance in middle- aged MDD patients. Tetrahydrobiopterin is known to stimulate nitric oxide (NO) production which in turn is a vasodialator that improves cerebral perfusion. Hence, improving NO concentration through dietary supplementations could probably improve efficacy of antidepressant drugs in TRD.

Keywords: Treatment resistant depression; Neurovascular coupling; Tetrahydrobiopterin; Nitric oxide

IL-5

Neurophysiological correlates of elevated curiosity-like behavior in adolescence rats predisposed to early life stress

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Early life stress (ELS), particularly during the stress hypo-responsive period (SHRP), significantly impacts brain development. This period is crucial for synaptic pruning, circuit formation and maturation. Adverse events during this period such as physical, emotional or sexual abuse can impose long lasting effects on brain function, cognitive and emotional development causing an increased risk for psychopathologies later in life. We previously showed that ELS induced an increased anxiety-like behaviour, an increase in fear generalization, increased fear memory retention and reduced fear extinction in adolescence and young age groups, but not in older age groups. They also showed an increased spatial learning, attention and repetitive behaviour. An increased neuronal activity in the PVN of hypothalamus and reduced activities in medial prefrontal cortical (mPFC) regions during fear memory recall, with a persistent increased activities in PVN and reduced mPFC activities in young adulthood. Adolescence age is characterized with natural tendency for sensation-seeking behavior when they are mostly with peers such as elevated curiosity-like behavior, and risk-taking behavior. Also, rewiring, reorganizing and reestablishing the permanent connectivity between the reward and limbic circuit, as a result the personality traits are established. It is unknown an exposure to early life stress whether it impacts the natural adolescent behavior. Our previous studies have shown that at adolescence age, ELS caused an increased curiosity and risky decision-taking behavior associated with an increased plasma corticosterone and Dopamine. However, there are no studies yet to reveal the neurophysiological mechanisms of early life stress on curiosity-like at adolescence in rats predisposed to ELS. In the present lecture, I am going to demonstrate the characteristic features of neural oscillations in the reward circuit – PL, NAc and CA1 per se – and how early life stress affects the oscillatory patterns during the curiosity-like behaviour in adolescence age. Thus, we showed that curiosity is one of the hallmark behavioral phenotypes of adolescence, and early adverse experiences are known to affect such behavior in enhancing the risk for substance abuse disorders later in adolescence age.

IL-6

Mitochondrial SIRT3 activation by HKL reverses neurodegenerative changes in the hippocampus of moderate grade hepatic encephalopathy

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Excessive release of glutamate, an excitatory neurotransmitter, results in overactivation of NMDAR leading to neuronal derangements and development of several neurodegenerative disorders such as AD, PD, ALS etc. In this respect, SIRT3, mitochondria localized member of SIRTUIN family protein deacetylase, is emerging as cogent target for preventing mitochondrial dysfunction led neuronal damage in various animal model of excitotoxicity. SIRT3 functions as a master regulator in maintaining cytoplasmic-mitochondrial metabolite trafficking and thus protecting mitochondrial structure and function and therefore, it is speculated to serve as a strategic therapeutic target for preventing neuronal derangements during excitotoxicity. Hepatic encephalopathy is a neuropsychiatric disorder caused due to persistent hyperammonemia led elevated extracellular glutamate in brain and thereby causing NMDAR overactivation. The present studies were undertaken in the hippocampus of the neurobehavior characterized MoHE rats, developed by administration of 100 mg/kg bw of thioacetamide i.p. for 10 days, and of the MoHE rats post treated with HKL (10 mg/Kg b.w.) for 7 days. The results suggested that honokiol dependent SIRT3 activation, could enhance the expression of SIRT3 at protein and mRNA level which was consistent with recovery in its MoHE associated deranged upstream modulators like FoxO3a and PGC1α. This was also found to be associated with restoration of neuroprotective NR2A dominated NMDAR combination from a neurodegenerative NR2B dominated neurodegenerative combination seen in the hippocampus of the MoHE rats. SIRT3 activation could also restore the mitochondrial calcium homoeostasis while normalizing the enhanced mPTP opening and apoptosis by regulating the Ca2+ influx and could also modulate oxidative stress by deacetylating its immediate target Mn-SOD. The findings suggest SIRT3 as a potential therapeutic target for managing mitochondrial dysfunction led neurodegeneration.

IL-7

Role of biomarkers in Neurological Disorders

Dr.Karthik V Mahesh
PGIMER, Chandigarh, India

Biomarkers play a crucial role in neurology by serving as objective indicators of disease processes, aiding in diagnosis, prognosis, and treatment response. This lecture will delve into the types of biomarkers, including genetic, proteomic, and imaging markers, and their applications in conditions such as Alzheimer's disease, Parkinson's disease, and multiple sclerosis, cancer. We will explore how biomarkers enhance understanding of disease mechanisms and contribute to personalized medicine, ultimately improving patient outcomes. Additionally, the lecture will discuss challenges in biomarker validation and integration into clinical practice, highlighting ongoing research efforts aimed at identifying new and more effective neurological biomarkers.

IL-8

LiH: The New Addition to Metabolic Syndrome

Dr. Aastha Thakkar

PGIMER, Chandigarh, India

IL-9

Validation of the Immersive Virtual Reality and Mobile games for Cognitive Assessment: Results from the 82 Young Participants

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Dementia is a global public health problem. Given the absence of reliable cure for dementia, it is critical that our scientific efforts are invested in its early detection. Such an approach allows opportunities for timely intervention and cognitive rehabilitation that promises slower cognitive deterioration and intact quality of life. Traditional neuropsychological tests are unsuited for early indication of cognitive deterioration because they lack ecological validity and do not inform the real-world functional cognitive abilities of people. As a result of this limitation, these tests cannot tap into the asymptomatic deteriorating cognitive abilities. Given this backdrop we need to discover and invent alternative tools that can detect cognitive deterioration early. In this context, goal-oriented games based on end-to-end software become extremely relevant. Prior research has shown that such games have the capacity to measure the cognitive abilities of the players, however the vision with which they are developed is limited to cross-sectional cognitive assessment without any goal of using them for a disease like dementia. Moreover, the approach towards validating these games lacks creativity because it is based primarily on correlational analyses. Through our work, we demonstrate not only the limitless possibility of goal-oriented games in immersive and non-immersive VR for early detection of cognitive red flags, but also show the multi- dimensional validation approach that helps us to vet the reliability and potential of these games from multiple dimensions. In this talk I will demonstrate the quantitative and qualitative evidence on the potential of VR and mobile based goal-oriented games for cognitive assessment using data collected in a pilot study we conducted on 82 young participants (aged 18-28 years). The selection of this cohort was based on extensive evidence on the neurobiology of aging which establishes that cognitive decline begins in the 2nd decade of life. This study forms an important piece of evidence on the role of goal-oriented software based games for cognitive assessment and informs our scientific efforts to develop technology-based solutions for dementia care.

IL-10

Sugar Addiction: Is It for REAL?

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Abstract

Sugar bingeing induces maladaptive neuroadaptations to decrease dietary control. Neuroadaptations induced by sugar bingeing lead to severe cravings and withdrawal effects when sugar is no longer available in the diet. However, the concept of sugar addiction is debatable and has gained much attention recently, whether or not clinical features of addiction such as withdrawal effects are applied to sugar feeding. This pre-clinical study investigated the sex differences in sucrose bingeing and withdrawal-induced negative mood effects and underlying neuroimmune response in the prefrontal cortex and nucleus accumbens of C57BL/6 male and female mice. We used a two-bottle sucrose choice paradigm to develop sucrose dependence in mice. Female mice consumed more sucrose than male mice when given free access to water and 10% sucrose for eleven weeks. A significant increase in the mRNA expression of neuroinflammatory markers (*Il1β*, *Tnfα*) was found in the prefrontal cortex of males exposed to sucrose withdrawal. Sucrose bingeing and subsequent withdrawal showed elevated protein levels of pro-inflammatory cytokines/chemokines/growth factors in the prefrontal cortex (IL-1β, IL-6, TNFα, IFN-γ, IL-10, CCL5, VEGF) and nucleus accumbens (IL-1β, IL-6, IL-10, VEGF) of male mice as compared to their water controls. These effects were concurrent with reduced mRNA expression of neuronal activation markers (*cFos*) in the prefrontal cortex of sucrose withdrawal males. One week of sucrose withdrawal showed anxiety-like behavior in male mice, not females. In conclusion, this study demonstrates that repeated access to sucrose induces depression and anxiety-like behavior when the sugar is no longer available in the diet and these effects are male-specific. Elevated neuroinflammation in reward neurocircuitry may underlie these sex-specific effects.

Keywords: Anxiety; Depression; Sex difference; Sucrose withdrawal; Sugar addiction

IL-11

Your Gut, Your Mood!!!

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Balancing various emotions are a daily necessity, culminating in either stressful or appeasing effects. As a consequence, maintaining sound mental health is increasingly recognized as a crucial necessity for the overall well-being, particularly in aging populations. Numerous studies have highlighted that exposure to and subsequent infestations of pollutants, high-fat/high-sugar diets, alcohol, sedentary lifestyles, and lack of exercise have immense disastrous effects on mental health. Recently, a key cause of disrupted brain function has been demonstrated to be an outcome of altered gut microbiota, leading to dysbiosis. Of different pollutants to which organisms may be inadvertently exposed, severe toxic effects of arsenic on human physiology have been of immense concern worldwide. Arsenic contamination causes irrevocable structural and functional disruption of tissues, leading to major diseases in chronically exposed individuals. However, it is yet to be resolved whether the effects are a result of direct deposition and persistence of arsenic in tissues, or via activation of indirect signaling components. Emerging evidences suggest that gut inhabitants play an active role in orchestrating various aspects of brain physiology, as the gut-brain axis maintains cognitive health, emotions, learning and memory skills. Agents triggering dysbiosis, such as arsenic-induced alterations in the gut microbial population, may consequentially evoke neurotoxicity, eventually leading to anxiety and depression. In order to delineate the mechanism of action, mice were exposed to different concentrations of arsenic. Behavioural studies revealed significant onset of anxiety and depression-like behaviour in a dose-dependent manner. Altered levels of neurotransmitters, microglial activation and appearance of pyknotic nuclei in the hippocampus were also observed. Finally, to confirm whether the neurotoxic effects are specifically a consequence of modulation of gut microbiota by arsenic and not direct deposition of arsenic in the mice brain, fecal microbiota transplantations (FMT) were performed. Perturbed gut microbial population and severe structural, functional and behavioral alterations were observed in the FMT groups. The deleterious effects were comparable, and sometimes more pronounced in FMT mice, compared to arsenic-treated mice. 16S rRNA gene sequencing indicated major alterations in the gut bacterial population. Moreover, suppression of TLR4 using vivo-morpholino indicated restoration of the altered parameters towards normalcy in FMT mice, confirming direct involvement of the gut bacteria in inducing neurotoxicity in mice via an arsenic-gut-brain axis. Interestingly, psychobiotics, a subset of probiotics, specifically modulate the functions of the central nervous system, in concordance with the immune, humoral, neural and metabolic networks, to enhance antidepressant and anxiolytic effects. Thus, a comprehensive understanding of the psychobiotics, and defining the causative factors influencing mental health which may be ameliorated by psychobiotics, will aid in developing effective psychotherapeutic treatment strategies in the near future.

IL-12

17β-estradiol and testosterone may regulate epilepsy-associated neuroinflammation in anterior temporal lobe and hippocampus of male rats

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Epilepsy is a prevalent neurological disorder that affects more than 50 million people and is characterized by unprompted repeated seizures. In humans, the most common form of chronic epilepsy is temporal lobe epilepsy (TLE) wherein seizures originate in anterior temporal lobe (ATL), hippocampus, amygdala, and entorhinal cortex. The disrupted endocrine milieu is reported in TLE with decreased testosterone (T) levels in TLE men, and increased allopregnanolone in male children. Disrupted reproductive hormones influence seizures through genomic and non-genomic actions of mechanisms. Although altered levels of hormones are studied under epileptic conditions, expression of their receptors is meagrely explored in the significant regions including hippocampus, and ATL. Hence, the present work deals with understanding the changes in levels of two primary sex steroid hormones, testosterone (T) and 17β-estradiol (E2), and their receptors Ar, Erα, Erβ in hippocampus and ATL of pilocarpine-induced chronic model of TLE male rats. Total steroid extraction from blood, hippocampus, and ATL, followed by estimation of T and E2 was done using specific ELISA kits. Further, gene expression of receptors Ar, Erα, Erβ along with key neuro-inflammatory (Tnf-α) and -trophic factors (Bdnf, Tgf-β) in hippocampus and ATL was estimated using qRT-PCR. Further, a correlation was established between (a) hormones (T/ E2) and neuro- inflammatory (Tnf-α) and -trophic factors (Bdnf, Tgf-β), (b) hormone receptors (Ar, Erα, Erβ) and neuro-inflammatory (Tnf-α) and -trophic factors (Bdnf, Tgf-β), and (c) hormones (T/ E2) and their receptors (Ar, Erα, Erβ), in both hippocampus and ATL using Pearson's correlation test ($p < 0.05$). In hippocampus, E2 was increased while T was decreased whereas in ATL, both E2 and T were seen to be increased. Elevated levels of E2 but decreased levels of T were found in serum of TLE rats. Expression of Erα, Erβ, Ar, Bdnf, Tgf-β, and Tnf-α were upregulated in hippocampus and ATL of TLE rats. In the ATL, E2 exhibited a negative association with Erα but a positive association with Erβ. Further, a positive correlation was observed between steroidal receptors and neuroinflammatory markers, thereby suggesting a regulatory role of sex steroid hormones in TLE pathogenesis via modulating neuroinflammation.

IL-13

Dysregulated Glutamate Trafficking Driven by Neuroinflammation in Major Depressive Disorder

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Major Depressive Disorder (MDD) is a genetically- complex neuropsychiatric disorder and is one of the leading causes of global disease burden. There are several target genes and factors implicated in the pathophysiology of MDD, however, the complete set of signaling pathways contributing to their etiology remains largely unidentified and poorly characterized. In the present study from our lab, we employed an unbiased approach to identify differentially expressed genes and investigate their associated signaling pathways that may contribute to the pathophysiology of MDD in women. We performed next-generation sequencing on whole blood samples from women with MDD and their well-matched healthy counterparts, generating transcript profiles of differentially expressed genes from Peripheral Blood Mononuclear Cells (PBMCs). We used robust bioinformatics tools to carefully identify the significant differentially expressed genes (DEG) with a high fold change and performed network analysis to get insights into their corresponding signaling pathways and potential biological mechanisms associated with MDD. Our result suggests that the Neuroactive ligand-receptor interaction pathways with glutamate ion channel activity and inflammatory mechanisms might play an important role in the pathophysiology of MDD in women. Further, we propose that the neuroactive ligand-receptor interaction pathway may be crucial in the disruption of neuronal calcium ion homeostasis. Calcium signaling, both within neurons and in the extracellular environment of the brain, is essential for neuronal plasticity, which underlies learning, memory, and neuron survival. We believe that the results of this study may provide novel insights into the molecular mechanisms of MDD and other depressive disorders, paving the way for the discovery of potential therapeutic interventions.

IL-14

Antiviral and neuroprotective potential of *Ocimum basilicum* against Japanese encephalitis virus in experimental models

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Japanese encephalitis virus (JEV), a member of the Flaviviridae family, threatens over three billion people worldwide despite existing vaccines. This study aimed to find effective treatments by exploring 265 phytocompounds from *Ocimum basilicum* (OB) for their antiviral and neuroprotective properties. The research involved in-silico, in vitro, and in vivo evaluations. In the in-silico study, we identified CA, rutin, and salvianolic acids A as the topmost hits phytocompounds of *Ocimum basilicum* (OB) via molecular docking in four targets of JEV (Envelope protein, NS5 RdRp, NS3 helicase, and NS3 protease) using Schrodinger software. In the in vitro study, CA and rutin exhibited variable antiviral potency with IC50 values ranging from 11.03 μM to 24.04 μM and 16.45 μM to 26.84 μM in different treatment approaches respectively. They demonstrated strong antiviral effect via inhibition of the virus entry into the host cells. In addition, treatment of JEV-infected SH-SY5Y cells with these compounds significantly reduced the intracellular viral load, the proportion of apoptotic cells, and the ROS level in a dose-dependent manner. In the in-vivo experiments, rutin demonstrated a dose-dependent increase in survival. Rutin (50 mg/kg) significantly improved survival rates and reduced the severity of encephalitis symptoms in JEV-challenged mice. An in-depth analysis of brain and serum samples from treated mice revealed a substantial reduction in infectious viral particles, viral RNA, and viral NS3 protein levels, particularly at 25 and 50 mg/kg. Rutin also enhanced antiviral gene expression, including IFNα, IFNβ, and IFIT1, suggesting a robust antiviral response. Additionally, rutin significantly mitigated JEV-induced neuroinflammation by decreasing microglial activation, inflammasome formation, and proinflammatory cytokine levels. In conclusion, OB could be a potential therapeutic candidate in Japanese encephalitis.

Keywords: Antiviral, Chicoric acid, Japanese encephalitis virus, Neuroprotection, *Ocimum basilicum*, Rutin.

IL-15

Olfml3 and Tmem119 could be a new player in microglia functions

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Neuroinflammation is a hallmark of many neurological disorders characterized by microglia activation and infiltration of blood-borne macrophages. Microglia plays an important role in the regulation of neuroinflammation-driven neurological disorders. Recently, Olfml3 and Tmem119 have been identified as microglia markers whose expression has been shown to alter during neurodegenerative diseases. However, information about the Olfml3 and Tmem119 protein and its possible involvement in neuroinflammation is still lacking. Therefore, the neuroinflammatory mice model was developed by intraperitoneal injection of lipopolysaccharide (750 μg/kg) for 7 days. The expression pattern of Olfml3 and Tmem119 in the brain at transcript and protein levels was done by qPCR and western blotting, respectively. In silico analysis was done to identify Olfml3 and Tmem119-specific interacting proteins and signaling pathways. It was followed by modeling Olfml3 and Tmem119 protein, molecular docking, and MD simulation to check their possible interacting partner in microglia. The expression of Olfml3 and Tmem119 at both transcript and protein levels increased in LPS-induced mice compared to the control. They were found to interact with proteins essential for microglia proliferation, activation, and migration, extracellular matrix organization, immunoregulatory interactions between a lymphoid and a non-lymphoid cell, and integrin cell surface interaction. Olfml3 was observed to co-localize with Iba1, and the number of Olfml3 and Iba1 dual-positive cells increased in the brain of the neuroinflammatory mice model. Thus, Olfml3 and Tmem119 could be involved in microglia functions.

IL-16

Extracellular vesicles and microRNA crosstalk: Modulators of neuroinflammation in HIV-1 and opioid-associated neurocognitive disorders

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HIV-associated neurocognitive disorders (HAND) persist as a significant challenge in HIV-1-infected individuals, especially in the context of opioid abuse, such as morphine use, even in those receiving combination antiretroviral therapy. Recent studies have highlighted the critical role of extracellular vesicles (EVs) as paracrine signaling mediators that modulate HAND pathogenesis via the transfer of cargoes, including microRNAs (miRNAs) between glial cells, neurons, and other CNS components. Our work has demonstrated that HIV-1 transactivators of transcription (Tat) protein and morphine enhance neuroinflammation and neurodegeneration by potentiating this miRNA-mediated cellular crosstalk.

HIV-1 Tat protein triggers the release of miR-9-enriched EVs from astrocytes, which are taken up by microglia. This leads to the downregulation of phosphatase and tensin homolog (PTEN) through direct binding to its 3′-UTR, resulting in microglial migration. Additionally, HIV-1 Tat-induced miR-7-enriched EVs are internalized by neurons, downregulating Neuroligin 2 and contributing to synaptic alterations, which exacerbate neuronal dysfunction.

In parallel, morphine exposure induces the release of miR-138-enriched EVs from astrocytes, which activate microglia by binding the GUUGUGU motif of miR-138 to endosomal toll-like receptor 7. This triggers the activation of the NFκB signaling pathway, further promoting neuroinflammation. Intranasal administration of a miR-138 inhibitor in morphine-treated mice models attenuated this microglial activation, underscoring the therapeutic potential of miRNA-based interventions. Morphine-stimulated astrocytes also release miR-23a-enriched EVs that are internalized by pericytes, downregulating PTEN expression and increasing pericyte migration. This disrupts the integrity of the blood-brain barrier by causing pericyte loss, facilitating the influx of peripheral monocytes into the CNS, and contributing to ongoing inflammation. Our findings also highlight the therapeutic application of engineered EVs. Delivery of miR-124-loaded EVs (EV-Cy5-miR-124) in cocaine-treated mice reduced microglial activation and diminished the expression of pro-inflammatory markers like TLR4 and STAT3.

These findings collectively emphasize the crucial role of EV-mediated miRNA signaling in the pathological interplay between glial cells, neurons, and pericytes in HAND pathogenesis. Understanding this glial-neuronal and glial-pericyte communication offers potential avenues for developing therapeutic interventions aimed at mitigating the neuroinflammatory consequences of HIV-1 infection and opioid abuse in HAND.

INVITED LECTURES

Day 3

IL-1

Decoding Parkinson's: The Quest for Answers

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Parkinson's disease (PD) is a complex neurodegenerative disorder that continues to challenge researchers and clinicians alike. Despite substantial progress in understanding its clinical manifestations—such as motor symptoms like tremors, rigidity, and bradykinesia—the precise mechanisms driving PD remain largely unclear. Numerous unanswered questions persist regarding the underlying pathophysiology of PD, including the causes of selective dopaminergic neuronal loss, the roles of genetic and environmental factors in disease onset, and the involvement of non-motor symptoms such as cognitive decline and sleep disturbances. Current therapies for PD are symptomatic and fail to address the root cause of the disease, with no available treatment able to halt or slow its progression. This highlights an urgent need for novel approaches in both understanding and treating PD.

One promising avenue for addressing these unsolved questions lies in the use of **zebrafish (Danio rerio)** as a model organism. Zebrafish share many genetic and physiological similarities with humans, making them an ideal model for studying the complex genetic and molecular mechanisms underlying PD. Their transparent embryos and rapid development allow real-time visualization of neurodegeneration and high-throughput screening of potential therapeutic compounds. Additionally, zebrafish models enable the study of both genetic mutations linked to PD and the effects of environmental toxins that may contribute to the disease, offering a holistic approach to understanding PD's multifaceted nature. By leveraging the zebrafish model, researchers may be able to unlock critical insights into PD's pathogenesis and accelerate the discovery of disease-modifying treatments, ultimately bringing us closer to finding answers for this debilitating disorder.

II-2

A Shotgun Metagenomics Sequencing Approach for Studying the Etiology of Undiagnosed Meningoencephalitis Infection in a Tertiary Care Hospital Setting

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Meningoencephalitis (ME) is a severe and often life-threatening neurological emergency that involves inflammation of the brain and meninges. Despite advances in clinical diagnostics, ME continues to be a significant cause of mortality and morbidity, especially in tertiary neuro care ICU settings in India. Conventional diagnostic methods, such as culturing, serology, and molecular assays, frequently fail to identify the causative pathogens, leading to inadequate treatment and potentially severe outcomes. Given the high rate of undiagnosed cases in Indian settings, there is an urgent need for alternative diagnostic approaches to better understand the etiology of ME. Shotgun metagenomics sequencing (mNGS) is a novel, hypothesis- free approach that enables the detection of a wide range of pathogens, including viruses, bacteria, fungi, and parasites, from a single cerebrospinal fluid (CSF) sample. This technology offers a promising new direction in the diagnosis of ME, particularly for cases where conventional methods fail to identify the causative agent. Although mNGS has shown potential in studies from other countries, there is limited data on its utility in the Indian context. This study, conducted in the neurological intensive care unit (NICU) of Dr. G. M. Taori Central India Institute of Medical Sciences (CIIMS), Nagpur, aims to address this gap by using mNGS to investigate the etiology of undiagnosed ME cases. The study involved 90 patients and processed for metagenomic sequencing. Pathogen identification was performed using the CZ-ID bioinformatics platform, which analyzes unaligned human sequences to detect microbial DNA and RNA. Results revealed that mNGS identified pathogens in 29 out of 50 suspected meningitis cases, with a detection rate of 64.7% in known etiology cases, showing 100% concordance with routine investigations. For unknown etiology cases, mNGS identified potential pathogens in 18 out of 28 idiopathic cases (64.28%). Interestingly, Mycobacterium tuberculosis emerged as the predominant pathogen in many cases of both known and unknown etiology. The findings from this study highlight the significant translational potential of mNGS in clinical practice. By improving diagnostic accuracy and reducing reliance on empirical treatments, mNGS can help mitigate the risk of antibiotic resistance, enhance patient outcomes, and inform public health strategies. Furthermore, the development of customized bacterial panels for molecular diagnostics could lead to more sensitive and specific testing, ultimately revolutionizing the management of ME in India. This study represents a pioneering effort in applying mNGS to the Indian population, offering critical insights into the etiology of undiagnosed ME cases and laying the groundwork for future diagnostic innovations.

IL-3

Customized Nanoparticle-Based Strategies For The Management of Neurological Disorders

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Hematological disorders constitute about 0%-8% of all causes of acute stroke. It is frequently associated with severe cellular deterioration and death. There is widespread agreement that the NLRP3 inflammasome pathway plays a crucial role in the development of cerebral ischemia-reperfusion injury.

In this study, MCC-950 loaded transferrin-conjugated pH-responsive polymeric nanomicelles (NMs) were synthesized and characterized using infrared (IR) spectroscopy and 1H NMR. We characterized the size, shape, and surface potential using dynamic light scattering (DLS), zeta sizer, scanning electron microscopy (SEM), transmission electron microscopy (TEM) and atomic force microscopy (AFM).

Nanomicelles specifically binds to the transferrin receptor 1 (TFR1) expressed on the cells of blood-brain barrier (BBB) and thus help to cross the BBB. NMs also exhibited the enhanced release of MCC-950 drug, which is NLRP3 inflammasome inhibitor, in slightly acidic pH, which represents pH of the ischemic region of the brain. Furthermore, therapeutic potential of NMs was evaluated against in vitro, in ovo, and in vivo models which represent cerebral ischemia. NMs were injected into the common carotid artery (CCA) of middle cerebral artery occlusion (MCAO) rat model to achieve maximum accretion of nanomicelles into the brain as blood flows towards the brain in the CCA. The current study reveals that the treatment of NMs significantly reduced the levels of NLRP3, ASC, cleaved caspase1, active IL-1β, and active IL-18 which were found to be increased in oxygen glucose deprivation (OGD)-induced SH-SY5Y cells, right vitelline artery (RVA) of chick embryo, and Middle cerebral artery occlusion (MCAO) rat model. The supplementation of NMs significantly increased the overall survival of MCAO rats. Overall, nanomicelles exerted the therapeutic effects against cerebral ischemic injury, which might be due to the suppression of the activation of the NLRP3 inflammasome.

IL-4

Brain Tissue Segmentation from MRI Scans using Artificial Intelligence

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The brain is one of the most unexplored parts of the human body, and its complex and delicate structure has scientists worldwide looking for answers to its intricacies. Nowadays, with the increase in life expectancy and the extravagant use of technology, it is evident that neurological diseases are on the rise. Therefore, it becomes essential that such diseases can be diagnosed at an early stage of their occurrence. Technologies like Artificial Intelligence have emerged as a boon for such early diagnostic systems. The proposed work performs data acquisition and preprocessing to extract a clear image of the brain from MRI scans for the classification of Alzheimer's disease.

IL-5

Association Of Dietary Patterns With Cognition And Mental Health: From A Vegetarianism Perspective

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Plant-based diets, rich in antioxidants, vitamins, minerals, and phytochemicals, have been associated with improved cognitive function and neuro protective effects. Additionally, vegetarian diets have been linked to reduced inflammation and oxidative stress, both of which are factors contributing to cognitive decline. The study aimed to investigate the impact of vegetarianism on cognition and neuropsychological status in healthy adults. A cross-sectional study was conducted among adults (N=304) aged 40 years and above in R.R district, Telangana. Cognitive function was assessed using the Mini Mental State Examination (MMSE), while psychological measures were evaluated through the Depression, Anxiety, and Stress Scale (DASS-21 questionnaire). Significantly the mood was better among vegetarians (n=155) compared to non-vegetarians (n=149), as indicated the DASS subscales for depression (10.0±0.06 vs 17.0±0.07, p=<.001), anxiety (4.0±0.05 vs 6.0±0.07, p=0.005), and stress (8.0±0.02 vs 10.0±0.05, p=0.007). Cognitive function scores also higher in vegetarian group compared to non-vegetarians (26.0±0.04 vs 24.0±0.03, p=<.001). Cognitive measures were inversely correlated with depression, anxiety and stress for vegetarians (rho=-0.371, p=0.000 ; rho=-0.027, p=0.734 ; rho=-0.105, p=0.914) respectively. A positive correlation between depression and anxiety (rho=0.147 ,p= 0.068) as well as depression and stress (rho=0.003 , p= 0.966) was observed. Adherence to a vegetarian diet shows to have positive effect on cognitive health in the aging population. However, further research is necessary to understand the underlying mechanisms and long- term implications of vegetarianism on cognition and brain health. The present study findings contribute to the existing literature on dietary choices and cognitive health, recommending the need for public health policy interventions and understanding the role of diet in cognitive aging holds significant potential for promoting brain health and overall quality of life.

Keywords: Cognition, Mental health, Vegetarianism, Nutrition, Plant-based diet

IL-6

New Horizons of Neuronal Optogenetics

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Optogenetics is an emerging field which focuses on controlling variety of cellular functions by utilizing light-activated proteins. Optogenetics has tremendous potential in neuroscience as it showed superior spatiotemporal resolution over conventionally used surgical, electrical, and pharmacological methods for studying neurobiology. A plethora of investigations utilised optogenetic tools to study neuronal physiology, brain circuit functions as well as for developing new rehabilitative and therapeutic strategies for associated pathological conditions/neurodegenerative diseases. Neuroscientist have used Channelrhodopsins (ChRs), a blue light responsive light-gated ion channel, to control intracellular ion flux and/or membrane potential of animal cells, simply by illumination. The ChR2 optogenetics was successfully improved working memory and short-term memory in an Aβ-injected mouse model of Alzheimer's disease (AD). The application of ChRs to remotely control the neural activity has encouraged researchers to identify new light-activated proteins with different spectral and ion selectivity. Recently, our group have characterized novel modular channelrhodopsin (light-activated ion channel proteins), cyclase and Serine/Threonine specific protein kinase, respectively, having immense optogenetic potential of these proteins in the field of neuroscience. The molecular, functional and their applications in opto-modulation of neuronal signalling and optogenetic therapy for the associated disease will be presented in detail .

Keywords: Optogenetics, neuroscience, light-activated proteins, neurodegenerative diseases

IL-7

Synaptic Plasticity in the Epileptic Brain

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Epilepsy is a common neurological disorder, affecting about 1% of India's population. The change in the ratio of excitatory to inhibitory neurotransmission is regarded as the main cause of epilepsy. Patients with epilepsy suffer many co-morbidities including memory impairment and cognitive problems. Hippocampus is the region of the brain, which is mainly associated with learning and memory and cognitive functions. Long term potentiation (LTP) and long-term depression (LTD) are the two main models to study memory at the cellular level. In the brain LTP/LTD is mostly NMDA receptor dependent. Here, we studied the role of NMDA receptor function in the epileptiform activity. Moreover how different NMDA receptor subtypes contribute to epileptiform activity in the hippocampus. We found that NMDA receptor subtypes contribute differently during development. Next we studied the effect of epileptiform activity in LTD by studying the CA1-Schaffer collateral pathway in the hippocampal slices. LTD was induced in the CA1-Schaffer collateral pathway by low-frequency stimulation (1Hz, 900 pulses), which induced 20% LTD in control hippocampal slices. But when we induced LTD after epileptiform activity the LTD protocol induced 20% LTP instead of LTD. The change in the direction of synaptic plasticity was NMDA receptor subtype dependent. We also investigated the excitatory and inhibitory ratio in control and epileptiform induced slices, and found that there was no change in the excitatory current amplitude and inter-event interval, but epileptiform activity reduced the GABA A current amplitude and inter-event interval significantly when compared with control slices. The change in GABA A currents and loss of LTD in epileptic slices may explain the learning and memory impairments in patients with epilepsy.

IL-8

Unravelling the Molecular Nexus of Antenatal Depression and Gestational Diabetes Mellitus

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Antenatal depression (AND) and gestational diabetes mellitus (GDM) are increasingly recognised as co-morbid conditions with profound implications for both maternal and fetal health. While traditionally studied in isolation, emerging evidence highlights a molecular nexus linking these conditions, rooted in shared biological pathways, yet the molecular mechanisms underlying their co-occurrence remain inadequately understood. Key pathways, including inflammation, oxidative stress, and insulin resistance, are central to AND and GDM. Elevated levels of pro-inflammatory cytokines, have been identified in both conditions, suggesting a convergent inflammatory response. Furthermore, dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, a hallmark of antenatal depression, may impair glucose metabolism, contributing to insulin resistance and subsequent GDM. We will present our recent findings on epigenetic predispositions, mitochondrial dysfunction and telomeres in regulating glucose metabolism, and neuroendocrine function. Additionally, the role of maternal psychosocial stress and epigenetic modifications in modulating this molecular cross-talk will be discussed paving the way for novel biomarkers and integrated therapeutic strategies. Ultimately, our goal is to improve the overall health and well- being of pregnant women and their offspring by addressing the complex interplay between mental health and metabolic disorders during pregnancy.

Keywords: Antenatal depression, gestational diabetes mellitus, molecular mechanisms, epigenetics

IL-9

Designing of a modified spontaneous alternation behaviour test to study the working memory paradigms

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The Spontaneous Alternation Behaviour (SAB) test is a widely used behavioural assay in rodents to assess their ability to explore a novel environment and their tendency to alternate between different choices. SAB test is often used to evaluate cognitive functions such as working memory, attention, and decision-making through a Y-maze. During the test trials, the rodent is placed at the centre of the Y-maze and is allowed to explore it freely, and the sequence of arm choices is recorded. The alternation ratio is calculated as the number of alternations (i.e., entering a different arm on consecutive trials) divided by the total number of possible alternations. A higher alternation ratio indicates a stronger tendency to alternate between choices. However, the traditional SAB test shows low alternation rates (~65%) and unreliable evaluation of cognitive functions. Therefore, we designed a modified SAB test named delayed-SAB for this purpose. The methodology of the delayed SAB test involves using a Y-maze apparatus with specific dimensions and gates to control rodent movement. We modified the testing procedure by introducing delays after each choice. We verified this test by assessing spatial working memory in three mice groups: No-Delay, Delay, and Scopolamine-Delay (positive control). After calculating the alternation percentage, we observed a significant increase in the alternations by introducing inter-choice delays. Moreover, Inter Session Interval-1 of 30 min had a significant effect on session-3 alternations. Both parameters were measured using frame-by-frame analysis with a standard video player. We used the Kruskal-Wallis test and Dunn's multiple comparisons (post hoc test) for statistical analysis.

IL-10

Human Microglial Cell Line, Hmc3 is an Ideal Cellular Model to investigate Sporadic Amyotrophic Lateral Sclerosis Associated Patho-mechanisms

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Contribution of non-neuronal cells, specifically the microglia, have been implicated in several neurodegenerative disorders including, Amyotrophic lateral sclerosis (ALS), an adult-onset progressive neurodegenerative disorder. ALS pathomechanisms still remains obscure and hence no curative therapy is currently available. Therefore, it is critical to delineate the explicit role of non-neuronal cells including microglia, in view of its immense involvement to neuroinflammatory responses observed in ALS. Studies from our laboratory which focus primarily on sporadic ALS representing 90% of the total cases have shown detrimental effects of CSF from ALS patiebosents (ALS-CSF) not only on motor neurons but also induced marked gliosis in rodent models. Proteomic analysis of ALS-CSF revealed up-regulation of Chitotriosidase (CHIT-1) by more than 20 folds. Further, CHIT- 1 was found to be expressed exclusively by microglia amongst the various neural cells, specifically upon exposure to ALS-CSF. These CHIT-1 expressing microglia were found to be activated and were skewed to a pro-inflammatory form. The precise mechanism of action of CHIT-1 is not known. Although primary microglial cells are beneficial in understanding these aspects, they have limitations for gene silencing and proteomic studies where, large homogenous cell population is required. For such experiments, microglia in the form of cell line would be more ideal. In this study, we investigated whether microglial cell line of human origin, namely, Human Microglial Cell 3 (HMC3), a well-characterized cell line can be an ideal model to study the molecular mechanisms associated with ALS-CSF and recombinant CHIT-1(rCHIT-1). HMC3 cells were exposed to ALS-CSF (10%v/v) and rCHIT-1 (18pg/µl) at different time points (6h, 12h, 24h and 48h) and were evaluated for viability, activation by phenotypic changes, ROS levels and inflammatory markers. Phenotypic changes were studied by live cell imaging and phase contrast microscopy while the ROS levels were measured spectrophotometrically by DCFDA assay. Our findings show prominent activation of HMC3 cells post exposure to both ALS-CSF and rCHIT-1. Morphologically, most of the cells transformed from the ramified resting form to the phagocytic amoeboid form post exposure. Enhanced ROS levels with increased proliferation confirmed the activation. Concurrently, the cells showed significant increase in the expression of pro-inflammatory markers including TNF-α. Interestingly, the ALS-CSF and rCHIT-1 exposed groups showed similar findings hinting that microglial activation is driven by CHIT-1 in ALS-CSF. This study therefore, provides an insight on the reliable use of HMC3 as a cellular model for deeper investigations on the contributions of CHIT-1 to the pathophysiology of sporadic ALS.

Keywords: Sporadic ALS, ALS-CSF, recombinant CHIT-1, HMC-3, ROS, inflammatory markers

IL-11

Neuro-progression and accelerated aging in psychiatric disorders

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Although neuroprogression became a consolidated concept in Biological Psychiatry, mechanisms involved in neuroprogression remain largely unknown and only few explanatory models exist. Aging in humans refers to a persistent decline in the age-specific fitness components of an organism due to internal physiological degeneration and it is necessarily understood as a multidimensional process of physical, neuropsychological, and social changes. The present study aimed to compare the leukocyte telomere length (TL) in patients with psychiatric disorders. Participants with Ultra high risk for psychosis, first episode psychosis, Schizophrenia and Healthy Control were enrolled in this study. Telomere lengths were determined using a multiplex qPCR assay. After adjustment for age, sex, ethnicity, and education, patients in UHR, compared with HC groups, had shorter telomere length. After adjustment for age, sex, ethnicity, years of education, and smoking, patient with Schizophrenia had longer telomere length compared with HC groups ($p < 0.01$). Shorter leukocyte telomere length in Ultra high risk for psychosis could represent early signs of accelerated aging in this population.

IL-12

Myasthenia Gravis: Complications Diagnosis And Management

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Myasthenia gravis is a T cell mediated auto-immune neuro-muscular disorder. Autoantibodies generated against acetyl choline receptors impair the muscle function through the formation of complement mediated membrane attack complex, rapid degradation of post-synaptic AchR and allosteric blocking of AchR. This impairment in signal transduction causes muscle weakness and fatigability which are classical hallmark of MG.

To begin with the smallest muscles like eyelid muscle are affected which leads to dropping of eyelids. Blurred vision, double vision, swallowing difficulties and generalised weakness of limbs and finally the respiratory muscle involvement leading to ventilatory support.

Detection of anti-AchR antibodies in the sera of suspected cases of MG will help in better management of the patients and saving them from going into more severe complications. AchR (acetyl choline receptor) is indigenously prepared from human muscle extract. Using these receptors in specific concentration an ELISA was developed to detect anti-acetyl choline receptor antibodies. The ELISA has been significantly helpful in severe cases and in the initial stages of MG.

The study found more males effected with MG than females. The results have been quite helpful in managing patients with MG so that they are saved from entering in the more severe forms like respiratory distress.

IL-13

Impact of Social Isolation Stress on Depression and Anxiety-like Behavior in Zebrafish

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Social isolation has been known for long to increase the risk of psychological impairments, illness and even death of people. Social isolation has been found to be strongly associated with post-traumatic stress disorder (PTSD), depression and suicidal ideation. While the effects of isolation have been documented in mammals and various fish species, comprehensive studies on socially isolated zebrafish remain scarce. In this study, zebrafish were used to examine the effects of social isolation on depression and anxiety-like behavior. Zebrafish were socially isolated from for 8-12 weeks followed by the well-established behavioural tests for anxiety and depression. The findings revealed that social isolation induce depression-like effects and significantly altered the exploratory behavior of zebrafish. Anxiety-like behavior was observed in zebrafish following social isolation. Additionally, it was found that the isolated zebrafish responded significantly more to social stimuli compared to the control group, indicating an increased preference or need for conspecifics after prolonged social isolation. Our findings demonstrate that zebrafish subjected to social isolation display significant behavioural changes, potentially impacting the quality of life which can leads to major depressive disorders and suicidal ideation.

IL-14

Blue Brain

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The human Brain, the most valuable creation of God. The man is called intelligent because of the brain. Today, we are devolved due to our unique capacity of thought, a trait distinct from most other animals, yet this knowledge is lost after the death of the human body. That knowledge might have been used for the development of society. The biological neural network consists of a network of neurons that are connected together by axons and dendrites and it received signals from other neurons and transmit though axon to others cell body. The human brain consists of a large number of neurons, approximately 86 billion. The Blue Brain Project is a Swiss endeavor in brain research with the objective of constructing a digital replica of the mouse brain. The project was established in May 2005 by the Brain Mind Institute at École Polytechnique Fédérale de Lausanne (EPFL) in Switzerland. The objective of this organization is to utilize biologically accurate digital reconstructions and simulations of the mammalian brain to determine the underlying principles governing its structure and function. Scientists at the Blue Brain Project used algebraic topology to create an algorithm, Topological Neuronal Synthesis, that generates a large number of unique cells using only a few examples, synthesizing millions of unique neuronal morphologies. This allows them to replicate both healthy and diseased states of the brain.

IL-15

Amlodipine prevents carrageenan-induced oxidative stress by attenuating neuronal cell death in adult zebrafish

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Carrageenan (Carr) obtained from seaweed (Rhodophyceae), a sulfated polysaccharides extracted from a species of red algae (Chondrus crispus) is a reversible gel mostly used in eye-drop preparation due to its hydrocolloid nature. But, Carr-induced inflammation causing oxidative stress in animal has not yet been evaluated. It was reported that amlodipine (AML) amlodipine can inhibit inflammatory cytokines by lowering intracellular calcium concentration increasing antioxidant defenses. In this study we hypothesised to understand the Carr-induced oxidative stress causing free radial mediated neurodegeneration and behavioral alteration in zebrafish and impact of AML as ameliorative agent. We aimed to evaluate the behavioral alteration in zebrafish using novel tank diving test (NTDT) and light dark preference test (LDPT) as invaluable tools for analyzing visual functions. To know the role of antioxidant defense system, oxidative stress biomarkers like lipid peroxidation, and CAT activity were analysed. RGCs of the retina of the eye and neurons of the optical tectum (TeO) in the brain were investigated by histopathological studies. Carr-treated groups swim in random patterns and showed increased number of transitions and spending more time in alter zones, whereas controls prefer perceived motion. AML significantly attenuates the increased lipid peroxidation level and decreased CAT activity by Carr administration. The histopathological analysis showed an increased in percentage of pyknotic cell counts after Carr treatment, whereas AML supplementation significantly reduced the pyknotic RGCs cell count in retina and TeO of the brain. Findings showed Carr-induced oxidative stress can be ameliorated by AML that act as antioxidant and neuroprotective agent by reducing pyckonsis process in developmental RGCs due to its possible repair mechanism in adult zebrafish. This study may be useful to know the underlying mechanisms of weakening and eliminating central neurons for synaptic connections in brain as well as in visual system giving scope for the treatment of neurodegenerative disorders.

Keywords: Carrageenan; Amlodipine; Glaucoma; Retinal ganglionic cells; Zebrafish

IL-16

Neurobiology of Opioid Use Disorder

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Opioid Use Disorder (OUD) is associated with significant neurobiological changes, particularly in brain regions responsible for reward, memory, and executive functions. Chronic opioid exposure disrupts the mesolimbic pathway, leading to dysregulated dopamine signaling and compulsive drug-seeking behavior. I will present our work on neurocognitive functions and neuroimaging studies, including Diffusion Tensor Imaging (DTI) and Surface-Based Morphometry (SBM), which reveal significant impairments in several cognitive domains and indicate white matter (WM) microstructural abnormalities, particularly in the frontostriatal circuits (FSC), and gray matter reductions in frontotemporal and limbic regions.

Research findings demonstrate that buprenorphine maintenance treatment (BMT) induces neuroadaptive changes, including altered cortical thickness, particularly in occipital and temporal regions, with potential benefits for sensory processing and memory. However, certain structural features like sulcal depth and gyrification remain stable over a six-month treatment period, indicating the selective impact of BMT on neuroplasticity.

DTI studies further reveal decreased fractional anisotropy (FA) in the inferior frontal gyrus (IFG) and orbitofrontal cortex (OFC) in opioid-dependent individuals, linked to impaired executive function and increased impulsivity. Importantly, some WM changes in the left IFG appear to predate addiction, suggesting a pre-existing vulnerability that is exacerbated by chronic drug use. These changes partially reverse following abstinence, as evidenced by higher FA in abstinent individuals, particularly in the right IFG.

These findings underscore the complexity of OUD's neurobiology, highlighting the role of structural and functional brain alterations in addiction development, vulnerability, and recovery. The results suggest that pharmacological treatments, such as BMT may optimize brain recovery and improve cognitive outcomes in individuals with OUD.

IL-17

Alzheimer's disease: reactive astrocytes play a key role in disease pathogenesis

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Astrocytes, the most abundant of the glial cells, undergo morphological, biochemical, molecular, and functional changes during the pathogenesis of neurodegenerative diseases including Alzheimer's disease (AD) termed as reactive astrogliosis. Reactive astrocytes are rich reservoirs of cytokines which may exert both beneficial and detrimental impacts on neuronal health. Anti-inflammatory molecules were the major entities during early hours of Amyloid-β (Aβ) exposure. However, pro-inflammatory molecules were up-regulated upon prolonged Aβ exposure. Our study identified cytokines secreted by reactive astrocytes in response to Aβ at early hours such as intercellular adhesion molecule-1 (ICAM-1), Pentraxin-3 (Ptx3) and tissue inhibitor of matrix metalloproteinase-1 (TIMP-1) as major neuroprotective candidates that ameliorate cognitive deficits in AD mouse models. We have also found ER mediated unfolded protein response (UPR) in astrocytes. ER membrane bound stress sensor proteins like PERK and IRE1α are activated as well as altered in its protein level in a time dependent manner in response to Aβ. Interestingly, we found strikingly diminished levels of TIMP-1 in the brain of six-month-old 5xFAD mice (a transgenic model of AD) versus wild-type mice. Intracerebroventricular injection of TIMP-1 in 5xFAD mice ameliorated their cognitive functions. TIMP-1 not only ensured neuronal viability in the AD model, but also conferred synapse–specific effects. Synaptosomal analysis revealed that TIMP-1 elevates dendritic spine size and protein levels. Electrophysiology study revealed that it promotes post-synaptic long-term potentiation in hippocampus, independent of pre-synaptic activity. ICAM-1 protected neurons and improved cognitive behaviours in 5xFAD mice by NF-kB signalling. Therefore, we identify cytokines of reactive astrocyte origin, with strong protective mechanisms-of-action on neurons which cognitive behavioural benefits and propose them as promising therapeutic candidates in AD.

IL-18

Prenatal VPA instigated mitochondrial damage resulting in autism-like phenotype and its mitigation by bioflavonoid

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Autism is a type of neurodevelopmental disorder and its pathophysiology is linked with abnormal behavioural habits, mitochondrial damage, blood-brain barrier disruption, and various other mechanisms. One of the main pathways that can contribute to the pathophysiology of autism is altered canonical Wnt signalling. The purpose of this study was to examine how canonical Wnt signalling contributes to the onset of autism-related symptoms and how the bioflavonoid "fisetin" targets it therapeutically in a prenatal VPA model of autism. To induce autism in the first generation of offspring, dams were exposed to valproic acid sodium salt (VPA) at the moment of neural tube closure i.e., gestational day 12.5 (GD 12.5). Fisetin was administered in two treatment regimens i.e., gestational (GD 13 till delivery) and post-weaning fisetin (PND 23-32). Our findings supported the notion that exposure to developmental VPA raised reactive oxygen species, disrupted mitochondrial function, and resulted in oxidative stress in the brain. In the prenatal VPA model of autism, this ROS-induced mitochondrial damage dysregulated canonical Wnt signalling, further disrupted the blood-brain barrier (BBB), apoptosis, and caused neuronal damage. The combination of these processes led to the behavioural abnormalities and developmental delays that characterise autism. Treatments with gestational and post-weaning fisetin effectively reduced the increased ROS, mitochondria dysfunction, and overactivation of Wnt signalling. Regulation of the mitochondrial directed-Wnt signalling axis improved the neurodevelopmental impairments and BBB permeability, alleviating symptoms of autism-like behaviour. Taken together, our findings revealed that fisetin evoked modulation of the mitochondria-*Wnt* signaling cascade successfully relieved the associated symptoms of autism in the prenatal VPA model of autism and could be used as a potential bioceutical against autism.

Valproic acid, fisetin, oxidative stress, mitochondria, canonical *Wnt* Signaling, blood brain barrier, neuroprotection

IL-19

Identification of autism disorder using EEG signals

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SRI AKUNDI NARAYANA MURTY MEMORIAL MEDAL for Early Career Scientists

ECS-1

A mouse model of Parkinson's disease to evaluate sex differences in the progression

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Parkinson's disease (PD) is a neurodegenerative disorder caused by the loss of dopaminergic (DA) neurons of the substantia nigra (SN). The pathological hallmark of PD is aggregation of α- synuclein protein in Lewy bodies and their progressive spreading in the brain. 60% of neuronal death occurs before the motor symptoms appear. Males have a higher incidence of PD than females. Hence, developing a mouse model replicating these progressive pathological and behavioural changes in both sexes is essential. To generate a PD model, we injected AAV-SNCA and α-synuclein pre-formed fibrils into medio- lateral SN. The behavioural outcomes were evaluated using wirehang, cylinder, and openfield tests. We studied the extent of neurodegeneration, α-synuclein aggregation and neuroinflammation at 4 (early), 12 (intermediate) and 24W (late) post-surgery to study stages of disease pathology. We observed mild motor deficits in mice consistent with other synuclein-based models. Males showed a stronger tendency of motor deficits while females displayed more anxious behaviors. There was a progressive reduction in DA neurons in SN and striatal fibre density, although no sex difference in neurodegeneration was observed. Further, we observed aggregated α-synuclein across time-points at SN that spread trans-synaptically in striatum and cortex. However, the accumulation of aggregates was absent at 24W in the striatum and cortex indicating their resistance to aggregation pathology. We also observed a significant increase in activated microglial and astrocytes across time-point. This activation is strongest at early time-points and attenuates over time. The model replicated PD pathology with progressive neurodegeneration and striatal fibre loss, accumulation of p-syn. This correlated with increased neuroinflammation in SN. We also observed a differential presentation of behavioural outcomes in male and female mice thus faithfully recapitulating the key features of PD.

ECS-2

Walking and Talking Proteins: Role in Trafficking of Nociceptors and Chronic Pain

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Kinesins (KIF's) are the motor proteins which are recently reported to be involved in the trafficking of nociceptors leading to chronic pain. Aurora kinases are known to be involved in the regulation of KIF proteins which are associated with the activation of N-methyl-D-aspartate (NMDA) receptors. Here, we investigated the effect of tozasertib, a pan-Aurora kinase inhibitor, on nerve injury-induced evoked and chronic ongoing pain in rats and the involvement of KIF17-NR2B crosstalk in the same. Rats with chronic constriction injury showed a significantly decreased pain threshold in a battery of pain behavioural assays. We found that tozasertib (10, 20, and 40mg/kg (i.p.) treatment showed a significant and dose-dependent inhibition of both evoked and chronic ongoing pain in rats with nerve injury. Tozasertib (40 mg/kg i.p.) and
gabapentin (30 mg/kg i.p.) treatment significantly inhibits spontaneous ongoing pain in nerve injured rats but did not produce any place preference behaviour in healthy naïve rats pointing towards their non-addictive analgesic potential. Moreover, tozasertib (10, 20, and 40mg/kg i.p.) and gabapentin (30 mg/kg i.p.) treatment did not altered the normal pain threshold in healthy naïve rats and didn't produce CNS-associated side effects as well. Western blotting and rt-PCR studies suggested enhanced expressions of NR2B and KIF-17 along with increased NFkβ, TNF-α, IL-1β, and IL-6 levels in dorsal root ganglion (DRG) and spinal cord of nerve injured rats which was significantly attenuated on treatment with different does of Tozasertib. Findings from the current study suggests that inhibition of pan-Aurora kinase decreased KIF-17 mediated NR2B activation which further leads to significant inhibition of evoked and chronic ongoing pain in nerve-injured rats.

Keywords: Aurora Kinase; Kinesin; Spontaneous ongoing pain; NR2B; NMDA; Neuroinflammation

ECS-3

Determining 'Brain Age' as a Measure of Neuroanatomic and Cognitive Health: Unravelling the Vascular Insults of White Matter Hyperintensity on Brain Health

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White matter hyperintensity (WMH), a brain lesion resulting from cerebral small vessel diseases with chronological aging, is depictive of fiber loss and fiber pruning. WMH load beyond a threshold is likely to pose vascular insults to neuroanatomic structures thereby leading to altered cognitive and brain health compared to the subjects without WMH.

Our investigations in aging cohort of NACC and ADNI showed that WMH increases exponentially with age, wherein, the periventricular WMH (PVWMH) progresses ~3 times faster than Deep white matter hyperintenisty (DWMH) in cognitively normal individuals. A combined approach implicating WMH quantification alongside cognitive measurements suggests that Total WMH burden with a load >3.8 mL is strongly associated with deficits in 'executive functioning', as revealed by poor performance on the Trail Making Test (TMT)-B, forward and backward Digit Span Tests (DST), semantic memory and episodic memory test. PVWMH threshold >1.8 ml and DWMH threshold >2.5 ml appears to impinge cognitive deteriorations associated with executive functions, semantic and episodic memory. Using WMH load and neuroanatomical segmentation, we developed a quantitative platform to estimate 'Brain Age (BA)': an index representative of Brain health at a given chronological age. From a cohort of cognitively normal subjects (N = 528) were used to develop the BA estimation model. The Brain Age model was used to predict BA and Brain Age Gap (BAG) using the following equation.

$$BAG = \text{Chronological Age} - \text{Estimated Brain Age}$$

BA was not significantly different from CA in the CN subjects with low WMH. The CN subjects who had high WMH (5-10 ml) load in the brain had significantly higher Brain Age Gap at the early (2.5 ± 2.9 years, $p < 0.001$) and intermediate age groups (2.2 ± 3.3 years, $p < 0.001$) compared to the subjects with low WMH (<1.5 ml). In this study we establish a unique brain age model that incorporates WMH load, for the first time, along with neuroanatomic quantities as a potential clinical measure of Cognitive status and Brain Health.

ECS-4

Tau-mediated endocytic trafficking of microglial CX3CR1 upon internalization

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Misfolded Tau protein clearance from extracellular space is highly impaired in the Alzheimer's disease brain. Microglia, the brain resident macrophage cells, are majorly involved in the active clearance of Tau oligomers and aggregated fibrils during the initial stages of disease progression. In recent research, GPCRs have gained more attention in the field of Alzheimer's disease therapy. Extracellular Tau protein is involved in direct binding to several GPCRs that promote receptor activation, downstream signalling, Tau internalization, and receptor endocytosis. Upon Tau binding, receptors undergo vesicular trafficking by several cellular mechanisms, leading to extracellular Tau internalization and degradation. CX3CR1 is a microglia chemokine receptor that maintains microglia at rest upon fractalkine binding in neurons. Extracellular Tau also binds the CX3CR1 receptor for microglial activation and Tau phagocytosis. In this study, we are interested in understanding CX3CR1 interaction with Tau oligomers and aggregates promoting clathrin-mediated receptor endocytosis. Further, we studied the association of the Tau-CX3CR1 complex with early, late, recycling endosomal and lysosomal markers to visualize the role of CX3CR1 in extracellular Tau clearance. Here, we have used confocal fluorescence microscopy to visualize the immunostained samples at different time intervals. Our study clearly demonstrated the association of the microglial chemokine CX3CR1 receptor in sensing and promoting extracellular Tau internalization, accumulation, and degradation, which would shed some light in the field of misfolded Tau protein clearance by microglial GPCRs in Alzheimer's disease.

ECS-5

Identification of a new molecular player controlling Huntington's Disease pathology

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In Huntington's disease (HD), the accumulation of polyglutamine expansion (PolyQ) is known to cause mitochondrial dysfunction along with proteotoxic stress. Clinical patient and experimental model studies also linked HD with a severe decline in mitochondrial functionality and oxidative stress, which ultimately leads to cell death. Despite extensive efforts, our knowledge regarding HD pathology is still incomplete. To gain greater insights into mitochondrial dysfunction, we performed a transcriptome- wide meta-analysis followed by extensive in vivo and in vitro experiments. Our combined in silico and experimental approach identified organellar fission as one of the important aspects linked to HD pathology. We observed that maintenance of peroxisomal and mitochondrial division via Peroxin isoforms controls the survivability of cells in HD conditions. In HD condition, the altered expression of genes coding for mitochondrial and peroxisomal fission/fusion proteins caused mitochondrial and peroxisomal dysfunction. Moreover, a unique correlation was observed between the expression of OXPHOS and Peroxin isoform expression. The administration of chemical chaperones that improve peroxisomal proliferation enhanced cell survivability and improved mitochondrial dynamics. Taken together, our study established the role of peroxisomes and their relation to mitochondria as one of the crucial links in the development of HD pathology. Furthermore, chemical chaperone-based rescue opens up possibilities for potential therapeutic approaches for HD treatment.

Dr. G. M. Taori memorial Award for Best Oral Presentation

OP1: Crosstalk between Alzheimer's Disease and Metabolic Disorders: An 1H-NMR Metabolomics Study

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Alzheimer's disease (AD) is a major neurological affliction which adversely impacts the elderly population globally. Recent studies have indicated linkages between AD and metabolic disorders like Diabetes mellitus (DM) and Obesity (OB). The present study is an attempt to evaluate metabolic alterations in the serum and brain through NMR spectroscopy. AD was induced in rats by stereotactic intra-cerebral-ventricular (ICV) injection of oligomerized Aβ-42 peptide into the brain. DM and OB were induced by intra-peritoneal injection of Streptozotocin (STZ) and feeding on High Fat Diet (HFD) respectively. The metabolic alterations obtained were analysed by ^{1}H-NMR spectra subjected to multivariate analysis by Principal Component Analysis (PCA) and Partial Least-squares Discrimination Analysis (PLS-DA). In serum, the levels glycolytic and osmolyte related metabolites were increased in AD, DM and OB rats. On the other hand, the metabolite profile from brain indicated altered levels of metabolites were related to amino acid cycle, glucose metabolism and neurotransmitter function. The alterations in neurotransmitters and cerebral energy metabolism were accompanied by deficits in cognition assessed by Morris Water Maze (MWM) in AD, DM and OB rats. The perturbed metabolic profiles were accompanied by presence of pathogenic amyloid deposits. Therefore, the study highlights shared metabolic signatures in AD, DM and OB, which may be involved in AD pathology.

Key Words: Alzheimer's Disease; Biomarkers; Brain; Metabolomics; Diabetes; Obesity

OP2: Expression analysis of the Brain Renin-Angiotensin System in diabetes-induced amnesic rat model

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Diabetes is a chronic metabolic disorder characterized by sustained hyperglycemia and has been implicated in various neurological complications, including cognitive impairment and the development of neurodegenerative disorders. The Renin Angiotensin System (RAS) is a complex hormonal cascade responsible mainly for regulating blood pressure and maintaining water and electrolyte balance. Numerous studies have provided evidence for the critical role of RAS components like Plasma Renin and the main pressor component of the RAS, Angiotensin II (Ang II), in regulating blood pressure. The actions of Ang II are mediated mainly via the AT1 and AT2 receptors, which are responsible for the manifestation of the RAS functions. However, it has been observed that the RAS is dysregulated under diabetic conditions. A separate component of the RAS within the brain has been reported and further implicated in conferring neurodegeneration and sometimes neuroprotection under diabetic conditions. Assuming that the role of brain RAS is also altered in diabetes, we speculate that this could lead to cognitive impairment, particularly amnesia, as a manifestation of neurodegeneration. Therefore, the current study was carried out to determine the involvement of brain RAS in diabetes-induced amnesic conditions.

To fulfil this aim, we used male Sprague Dawley (SD) rats and divided them into four groups: Vehicle, diabetic, amnesic, and diabetic+amnesic. Diabetes was induced by a single IP injection of Streptozotocin (STZ) at a dose of 55 mg/kg body weight. Amnesia was induced by an IP injection of Scopolamine at a dose 1 mg/kg body weight dissolved in normal saline for 15 days. Forty-eight hours after STZ administration, blood glucose levels were determined in the animals. Animals with blood glucose levels above 300 mg/dl were considered diabetic and were included in the diabetic group. The animals were maintained for two weeks, and physiological data like body weights, water consumption and food consumption were measured daily. Amnesia was assessed in all groups through behavioral tests, such as the Morris Water Maze and Novel object recognition test. Biochemical assays: acetylcholinesterase and choline acetylcholine transferase rates and expression analysis of synaptic plasticity genes Arc and

BDNF in the cortex and hippocampus. Finally, the expression level of brain-RAS components AT1 and AT2 was analyzed through RT-PCR in these two brain regions.

Physiological data showed that diabetic groups had significant weight loss but had increased food and water consumption when compared to amnesic and vehicle groups. As revealed from the behavioral, biochemical and molecular assays, we observed significant memory loss in the diabetic groups compared to the control group. For the brain-RAS components, the mRNA expression analysis showed that AT_1 mRNA levels were significantly elevated ($p < 0.001$). However, AT_2 mRNA levels were decreased significantly ($p < 0.001$) in the diabetic and amnesic groups compared to the control group in both regions of the brain. The AT_1R activation in the brain is linked to memory impairment, neuroinflammation and overall negative effects, while the AT_2R activation has been demonstrated to provide neuroprotection and enhance learning and memory. In conclusion, our findings reveal that increased AT_1 expression in the diabetic group might contribute to memory impairment, while the simultaneous decrease in AT_2 expression does not confer any noticeable neuroprotection.

OP3: Differential regulation of alternative splicing regulators during Metabolic Syndrome-induced progression of Type 2 diabetes in the brain: A comparative approach

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Metabolic syndrome (MetS) is one of the most widespread medical conditions worldwide, encompassing conditions such as obesity, dyslipidemia, hyperglycemia and hypertension. Hyperinsulinemia or insulin resistance (IR) is an important mechanism for disease progression. The hypothalamus is the key brain part involved in controlling food intake and energy metabolism. Current research works are focused on delineating the signaling pathway, the role of different proteins, associated factors and their crosstalk in MetS-induced IR and diabetic conditions in the brain. Alternative splicing (AS) is an important post-transcriptional mechanism that dictates gene expression at the level of availability of functional protein, effectively regulating several cellular processes including glucose metabolism. AS results from the combinatorial action of multiple RNA-binding proteins (RBPs) and small nuclear ribonucleoproteins (snRNP) that recognize sequence-specific motifs in the pre-mRNA, promoting or repressing the splicing of precursor mRNA. Activated serine/threonine-rich protein (SR proteins) also balance the whole mechanism. We hypothesize differential regulation of hnRNP, snRNP and SR proteins at different time points of diabetes progression will affect the functioning of the hypothalamus. In-vivo, the MetS mice model was prepared by feeding a high-fat diet for 8 weeks and 14 weeks. An increase in body weight and a change in their glucose tolerance level were observed. RT-PCR and qPCR analysis of several universal as well as brain-specific snRNPs and RBPs showed significant variation in their expression level at the early and late onset of pre-diabetic mice models. We also observed varied splicing patterns of target genes connected to MetS at different weeks. Taken together, we categorized diabetes progression-associated splicing regulators as early and late regulators.

OP4: 'Fast but NOT Furious': Gliomas with IDH Mutation Reprogram the Metabolic and Genetic Landscape but are 'Good Tumors'

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Gliomas are a class of Brain Tumors with an incidence of 27% of all brain tumors, wherein 60% harbor a gain of function mutation in IDH gene: A TCA cycle enzyme. Tumors harboring IDH mutations have good response to treatment, better prognosis, and higher overall survival (5 years to 15 years). Indeed, WHO revised its glioma classification and introduced a mandatory molecular classification of gliomas (IDH mutant / wildtype) in clinical setup. This study aimed to investigate if IDH mutant gliomas exhibit: (1) unique cellular and proliferative characteristics compared to IDH wild-type gliomas, (2) distinct metabolic and genetic landscapes, and (3) remodeling of the metabolic branch point at α-ketoglutarate, favoring 2-hydroxyglutarate synthesis over conventional glutamate synthesis. Metabolic and genetic analyses were conducted on LN229 glioma cell lines. Stable LN229 cell lines expressing IDH1 wild type and IDH1-R132H mutation were established. IDH1-R132H cells exhibited increased proliferation, reduced senescence, and altered morphology with a significantly increased nuclear-cytoplasmic ratio. Metabolically, IDH1 mutant cells showed reduced acetate and increased glutamine intracellular pool. ^{1}H-NMR based Consumption and Release (CORE) studies on LN229 cell lines revealed differential consumption and release of lactate, acetate, and glycerol across the LN229 naive, IDH1-WT and IDH1-mutant cell lines. Key carbon sources, including acetate, branched-chain amino acids, and glucose, as well as their associated metabolic pathways, were downregulated, along with reduced intracellular levels of these metabolites. In contrast, IDH1-mutant cells showed relatively higher intracellular pool of glutamine and glutamine hydrolysis enzyme expression. Additionally, IDH1 mutant tumors exhibit increased EM transition genes, increased expression of SLCs: a solute transporter, which might drive efficient uptake of chemotherapy drugs in IDH mutant gliomas. Binding energy studies further indicated that αKG favored binding to IDH1-mutant enzyme rather than GDH.

OP5: Neural Correlates of Training-specific Music Cognition: a Neuroimaging Study to understand Cognitive and Therapeutic Interventions in Parkinson's disease

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Neurological mechanisms involved in music perception has application in clinical interventions in motor disorders such as Parkinson's disease (PD). Construction of Music Perception through its features need more investigation to understand how neuroplasticity developed through music help in improving motor movements in PD patients. We studied construction of music perception through its features using PROMS-S introduced by Zentner (2012) as function of duration of training. Differences in integrity of white matter tracts comparing musicians and non-musicians using Diffusion Tensor Imaging (DTI) in fMRI was also investigated. Our objective was to identify training-specific and training-independent features and their associated neural correlates to provide insights on Music Therapy used in PD. Total 80 participants (36 musicians, 44 non-musicians) performed PROMS-S offline including eight subtests of perceptual features of music. The participants were required to compare reference and target stimuli and report whether they are similar/dissimilar and their scores were recorded. We used TBSS (Tract Based Spatial Statistics) in *fsl* to analyze data obtained from DTI and generate contrast for white matter tracts comparing musicians and non-musicians. We performed Pearson's correlation analysis to understand association between Fractional Anisotropy of white matter tracts and performance on auditory features obtained through PROMS-S. Because of multicollinearity among features, we performed principal component analysis (PCA) and chose to work with PC1 as it accounts for maximum variation in data. We found tempo to be the highest contributing feature for non-musicians in constructing music perception, which points towards tempo having a universally low discrimination threshold, thereby making it a significantly important clinical component for intervention studies. The white matter integrities in the genu of corpus callosum are equivalent for musicians and non-musicians for tempo, which backs up the universality of tempo as a feature and its clinical significance. Factor analysis showed that for non-musicians, Tempo, Tuning and Rhythm are unique features, whereas for musicians, unique features are Melody, Rhythm, Pitch and Tuning. This indicates that for musicians, rhythmic understanding develops from concept of melody, whereas for non-musicians, it develops from concept of tempo.

Pearson's correlation analysis revealed that for musicians, association between PC1 and melody is stronger, whereas for non-musicians, it was between PC1 and embedded rhythm. We also found that the genu of corpus callosum shows higher white matter integrity for musicians compared to non-musicians for melody, rhythm and pitch, while splenium show higher white matter integrity for embedded rhythm and tempo for non-musicians. This is indicative of a double dissociation between genu and splenium of corpus callosum between musicians and non-musicians. The overall study focuses on the neural correlates of training-specific and training-independent features of music perception that may have further clinical and neurological implications in PD.

OP6: Ursolic acid attenuates cuprizone-induced alteration in cortical ceramide synthase-2, sphingomyelin synthase-1, and serine palmitoyl transferase-1 expression in rodent model of multiple sclerosis

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Ceramide plays an important role in the myelination process; thus, the ceramide biosynthetic pathway could be considered a potential target in managing multiple sclerosis (MS). Fingolimod, an approved drug for MS, is considered a standard modulator of the ceramide biosynthetic pathway in the experimental model of MS. Ursolic acid (UA) exhibited neuroprotective activity in Cuprizone (CPZ)-induced animal model of MS. However, the protective activity of UA is yet to be established in the experimental model of MS. Therefore, the objective of the study was to evaluate the effect of UA (50, 100, and 200 mg/kg; p.o.) on the level of expression of ceramide synthase 2 (CS2), sphingomyelin synthase 1 (SMS1) and serine palmitoyl transferase 1 (SPTLC1) in CPZ-induced animal model of MS. CPZ (6 mg/kg; p.o.) was administered for 30 days once daily to male Swiss Albino mice to induce MS. CPZ significantly caused a decrease in the number of squares crossed in Open field, decrease in the muscular strength in Kondziela's inverted screen, decrease in the ratio between open to closed arm in elevated plus maze, and decrease in the number of head dip in hole board tests in the animals. Further, CPZ increased the levels of reactive oxygen species, GFAP, and Iba-1 and decreased the levels of MBP, NeuN, and Olig2 in mice cortex. Interestingly, CPZ increased the levels of CS2 and SPTLC1, and decreased the level of SMS1 in mice cortex. UA (100 and 200 mg/kg) significantly attenuated CPZ-induced behavioural, biochemical and molecular parameters in the animals. Thus, UA could be a potential therapeutic alternative in the management of MS. Further, the biomarkers of ceramide biosynthetic pathways such as CS2, SMS1, and SPTLC1 could be potential therapeutic targets in the management of MS.

Keywords: Multiple sclerosis; Ursolic acid; Ceramide synthase-2; Sphingomyelin synthase-1; Serine palmitoyl transferase-1; Ceramide; Fingolimod

OP7: Female Chronic social defeat stress: A novel model for investigating female depression

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Female depression is a complex and widespread disorder affecting millions of women worldwide, with its underlying mechanisms remaining poorly understood, largely due to a gender bias in preclinical research that has predominantly focused on male models. To bridge this gap, we introduce a novel female Chronic Social Defeat Stress (fCSDS) rodent model specifically designed to investigate depression in females. Our research aimed to characterize a female-specific model of depression that accurately reflects the distinct biological, behavioral, and molecular aspects of depression in female mice. We employed a meticulously developed protocol that involved inducing aggression in parous CD1 females through extended cohabitation with castrated male partners. Adult female C57BL/6J mice were then repeatedly exposed to these aggressive CD1 females over a 10-day period, simulating chronic social defeat stress without male influence. Additionally, label-free Quantitative MS-MS analysis was performed on the nucleus accumbens, a brain region crucial for reward and motivation. Behavioral assessments, including the sucrose preference test, forced swim test, elevated plus maze, and social interaction test, revealed significant stress-induced depressive-like behaviors in the experimental group. Molecular analyses showed dysregulation of estrogen receptors (ESR1 and ESR2), histone modifications (H3K9me3 and H3K27ac), and synaptic markers (SYP and PSD-95) across brain regions associated with depression. Further analysis identified significant reductions in EAAT1 and increased glutamate levels in the caudate putamen, indicating glutamate excitotoxicity, alongside elevated serum cortisol levels, suggesting an enhanced stress response. Notably, mass spectrometric analysis of the nucleus accumbens revealed disruptions in synaptogenesis signaling pathways and pathways associated with mitochondrial cytopathy. These findings demonstrate that the fCSDS model effectively replicates key aspects of female depression. This model addresses a critical gap in depression research and offers a foundation for developing targeted therapeutic strategies for women.

OP8: Exploring the Role of Electron Transport Chain Genes in Depression Pathogenesis Using a Reserpine-Induced Animal Model

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Depression is a pervasive mental disorder with profound impacts on individuals, families, and society, contributing significantly to disability and healthcare costs. The complex nature of depression involves genetic, environmental, and psychological factors, often comorbid with other health conditions. Effective treatment and prevention require a deep understanding of its mechanisms. So, the current study was planned to validate a pharmacological model of depression using reserpine in male Wistar rats and to further investigate the changes in the expression of genes associated with electron transport chain in mitochondria.

Reserpine drug was administered intraperitoneally to induce depression like symptoms, and neuro-behavioral tests were done to validate the model of depression in male Wistar rats. Behavioral tests such as actophotometer, elevated plus maze, marble burying test, and forced swim test and open field test were used to confirm the induction of depression. Depressed rats exhibited the symptoms such as reduced locomotion, decreased exploration, increased anxiety-like behaviors, and anhedonia. The study involved the examination of change in expression of ETC genes of mitochondria of nuclear origin in the hippocampus and Pre-frontal cortex region of the rat brain tissues using real-time PCR. Out of 11 genes selected based on previous literature search and using bioinformatic tools, 2 genes of ETC i.e., Ndufs7 (complex I) and Uqcrc2 (complex III) showed significant upregulation in the reserpine induced depressed rats. Dysregulation of genes in the complex I and complex III might be leading to an imbalance in the electron flow in the ETC eventually leading to the generation of Reactive Oxygen Species causing mitochondrial dysfunction subsequently resulting in the pathogenesis of depression.

The above findings suggest mitochondrial dysregulation in the animal model of depression as a potential cause for its pathogenesis. Further research is needed to explore the role of mitochondrial changes in neuroplasticity, neurogenesis, and neurotransmitter metabolism to enhance understanding and treatment of depression.

OP9: Green Synthesis of Ag NPs and Their Applications For Electrochemical Detection of miRNA-128

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Alzheimer's Disease (AD) is the third most predominantly occurring disease. It is an untreatable neurodegenerative disease characterized by progressive deterioration of brain cells and change in behavior, personality, orientation on time and space, functional capacity which affects the daily life of patient. Currently, AD is diagnosed by detecting deformities in patient's brain using sophisticated techniques including Magnetic Resonance Imaging, Positron Emission Tomography and Near Infrared. We report an electrochemical biosensor for detection of AD by using the miRNA-128 as a efficient biomarker. In this context, silver nanoparticles (Ag NPs) were synthesized using $AgNO_3$ and *Bryophyllum pinnatum* leaf extract as reducing agent. The Ag NPs were further capped with tri-sodium citrate (Ag/Cit) were deposited on Fluorine tin oxide (FTO) and followed by treatment with streptavidin. The biotinylated cDNA was conjugated with streptavidin through avidin-biotin interactions The final sensing platform c-DNA/SV/Ag/Cit/FTO was utilized to detect the miRNA-128.The characterization of modified electrode was done by Field emission scanning electron microscopy, and electrochemical characterizations including cyclic voltammetry, electrochemical impendence spectroscopy. The sensor was optimized with Concentration of c-DNA and reponse time. The linear range and limit of detection were 0.1 fM to 100 nM and 0.060 fM, respectively. It provides high selectivity from complementary DNA from non-complementary DNA for miRNAs and presented a long shelf life of 35 days. In this regard, this biosensor can be an ideal alternative owing to its high sensitivity, easy-to-use procedure, cost effectiveness, and compactness. Detailed results will be presented during the conference.

OP10: Vitamin D Ameliorates Mitochondrial Dysfunction and Spatial Memory Deficits in a 3-NP Induced Huntington's Disease Mouse Model

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Huntington's Disease (HD) is a neurodegenerative disorder characterized by motor deficits, cognitive decline, and mitochondrial dysfunction, often accompanied by proteostatic stress, particularly endoplasmic reticulum (ER) stress. Emerging evidence suggests that Vitamin D (VD) deficiency is a significant risk factor for neurodegenerative diseases, including HD. Previously we reported VD supplementation can rescue the motor deficits in a 3-nitropropionic acid (3-NP) induced HD mouse model. In this study, we extended our investigation to explore the impact of VD on spatial memory loss associated with HD. Remarkably, VD supplementation restored spatial memory function in HD mice. To understand the molecular basis behind the cognitive improvement we systematically dissect signalling events which proposed to be linked with neurodegenerative diseases causing ER and mitochondrial dysfunction. Both *in vivo* and *in vitro* model studies indicated VD mediated improvement of ER and mitochondrial homeostasis. We observed a significant imbalance in mitochondrial fission and fusion proteins, elevated reactive oxygen species (ROS) production, and disruptions in the oxidative phosphorylation (OXPHOS) system. VD supplementation restored mitochondrial homeostasis maintaining its dynamics. To ensure it is a direct effect of Vitamin D, we overexpressed of CYP27A1, a mitochondrial cytochrome P450 enzyme crucial for the conversion of inactive to active VD, in the *in vitro* HD model. Indeed overexpression of VD metabolic enzyme restored organellar homeostasis with significant improve in survivability. Taken together our findings underscore the therapeutic potential of VD in mitigating both cognitive deficits and ER-mitochondrial dysfunction in HD, highlighting the role of organellar health in the pathogenesis of neurodegenerative diseases and the protective effects of VD.

OP11: Anti-seizure medication eslicarbazepine acetate modulates excitatory and inhibitory currents and impairs synaptic plasticity at clinically relevant concentrations

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Anti-Seizure medications (ASM) are the first line of treatment for epilepsy. However, ASMs, including ESL, are frequently associated with cognitive and memory deficits. ESL acts via blocking the voltage gated sodium channel but the reason behind memory and cognitive impairment is not known. This study investigates the effects of ESL on hippocampal synaptic transmission and plasticity, key processes in learning and memory. Hippocampal slices from Wistar rats (P14-P28) were used to perform patch clamp and field recordings on CA1 pyramidal neurons. ESL (100 μM), at clinically relevant concentrations, reduced spontaneous AMPA and GABA-A receptor-mediated currents, while increasing the inter-event interval of GABA-A receptor currents. Additionally, ESL enhanced the amplitude of evoked field excitatory postsynaptic potentials (fEPSPs) via antagonism of adenosine A1 receptors, which typically inhibit synaptic activity. Importantly, ESL impaired long-term potentiation (LTP), a critical mechanism for synaptic plasticity, learning, and memory. These findings suggest that ESL not only targets sodium channels but also affects AMPA, GABA-A, and adenosine A1 receptor-mediated transmission, which may contribute to the cognitive impairments observed in epilepsy patients treated with ASMs.

Suven Life Sciences Pharma Award for Best Poster Presentation

PP1: Bypassing the need of Gadolinium injected Post Contrast Tumor imaging: A novel Radiomic approach for improved clinical management of Gliomas

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Gadolinium (Gd), is a paramagnetic material, widely used as a contrast-agent in MRI studies for identifying lesions and tumors in brain and other body parts. Recent measurements in brain autopsy of subjects who have had Gadolinium-enhanced MRI at some stage in lifetime revealed substantial deposits of gadolinium in the brain tissues. Gadolinium deposition from Gd-enhanced MRI may lead to cellular toxicity, hypersensitive reactions, deposition in bone and yet to be deciphered adverse effects. We aim to bypass the need of Gd-injection for generating the tumor tissue contrast and structural details by using a novel combinatorial approach of Structural-MRI (3D-T1w, 3D-T2w, 3D-T2-FLAIR) in glioma patients and healthy brain together with Deep-Learning neural network analysis. This study introduces a two-pronged approach for the development and implementation of Gd-free MRI for identifying the necrotic, non-enhancing and enhancing components of the Glioma-mass without injection of Gadolinium. Using TCIA cohort of LGG (N=65) and HGG (N=101), we are establishing a deep-learning approach which utilizes T1w, T2-FLAIR, and T2w capable of generating an output similar to that obtained from post-Gd-T1w MRI. Another approach is mathematical-approach of manipulating the addition and subtraction combination of T1w, T2w, and T2-FLAIR to generate a T1-Gd like image. We have successfully generated tumor subcomponents masks depicting enhancing, non-enhancing and edema components in the Glioma-mass. An optimal combination of mathematical operations between MRI modalities have shown more than 80% of similarity with post-contrast T1-w image. Quantitative analysis of images generated after applying equations with different combinations of mathematical operation shows Structural similarity index (SSIM) of 85% ± 5%, Peak to noise ratio of 82% ± 5% and mean squared error of 0.02 ± 0.03. Success of the project will also open avenues for aging based MRI investigations evaluating blood-brain-barrier permeability without application of contrast-enhanced imaging.

PP2: Fisetin as a Caloric Restriction Mimetic: Investigating its Neuroprotective Effects via Autophagy Induction in Amyloid Beta-Induced Neurotoxicity

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Caloric restriction mimetics (CRMs) are the compounds that mimic health-promoting effects of caloric restriction at the molecular, cellular, and physiological levels. Fisetin a plant flavonoid, is a well-established CRM that can induce autophagy but its role in neuroprotection against the aggregation of amyloid beta plaques via inducing autophagy is not clearly known. In this study, attempts have been made to examine neuroprotective effect of fisetin against amyloid beta-induced neurotoxicity using in silico and transcriptional approaches. The binding affinity of fisetin with two autophagy regulatory proteins (AMPK & mTORC) was performed using the molecular docking tool. We also checked the mRNA expression of autophagy regulatory genes (AMPK & mTORC), autophagy related genes (ATG101, ATG13), autophagy marker genes (ULK1, p62), mitophagy genes (PINK1), mitochondrial dynamics genes (Parkin), synaptic marker genes (PSD95, SYP) and neuropathy marker genes (AChE) in differentiated SHSY5Y cells under the exposure of amyloid-beta 42 and fisetin via qRT-PCR. Our findings suggested that the fisetin has high binding affinity with AMPK & mTORC. Moreover, transcriptional data demonstrated that fisetin has a significant impact such autophagy regulatory gene. While the gene expression of ATG101, ATG13, p62 and ULK1 has been significantly induced under the cotreatment of amyloid beta and fisetin. Similarly, fisetin also shows a significant induction of synaptic markers, mitophagy marker and mitochondrial dynamic gene. On the other hand, fisetin shows a significant reduction in neuropathy marker. So, we can conclude that fisetin shows neuroprotective effects via inducing the autophagy. Therefore, the present study indicates that fisetin treatment can have protective effects in an in vitro model of Alzheimer's disease. The results suggested fisetin as a potential CRM that may be considered as a therapeutic candidate for the management of age-related neurodegenerative diseases.

Key words: Caloric restriction mimetics; Fisetin; Neurodegenerative diseases; Autophagy; Gene expression

PP3: Early Biomarkers of Perinatal Asphyxia-Induced Hypoxic-Ischemic Encephalopathy: A Novel Combined Strategy of AI and Metabolomics

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Hypoxic-Ischemic Encephalopathy (HIE) is a critical neonatal condition caused by oxygen deprivation during birth, often leading to long-term neurological disabilities. Globally, HIE affects approximately 2% of live births, with higher prevalence in developing countries. In India, the state of Odisha reports one of the highest neonatal mortality rates, with 32 deaths per 1,000 live births, 29.09% of which result from moderate to severe perinatal asphyxia. Early detection and timely intervention are essential in minimizing the risk of irreversible neurological damage. However, there is NO definitive quantitative early clinical marker signature that can establish whether the neonate has undergone HIE. Whether HIE has set in becomes apparent only at the first-milestone i.e. after 6-months. This study presents a novel 'Neuroimaging and Blood Based Metabolomics and Cytokine Profiling' integrated with AI models for highly precise determination of HIE within an hour of Birth. We hypothesize that biomarkers associated with brain energy metabolism, antioxidants, and neuroinflammatory cytokines will be deregulated in response to the severity and duration of hypoxia. Blood samples from neonates are collected at 1, 6, and 48 hours post-birth across multiple hospitals in different parts of Odisha, followed by plasma isolation for Metabolic profiling at 700 MHz NMR spectrometer, while cytokine levels are quantified via ELISA. In parallel, Infant Brain MRI are segmented using Infant segmentation pipelines for neuroanatomic quantification. Plasma NMR analysis revealed significantly elevated concentrations of lactate (3.8-fold), creatine phosphate (1.7-fold), and choline (1.2-fold) in neonates with birth asphyxia over time compared to non-HIE subjects. Concurrently, neuroimaging segmentation indicated that HIE subjects exhibit ventricular enlargement (10%) and cortical thinning (7.7%). These neuroimaging metrics, along with plasma metabolite concentrations, are being integrated into AI models to discriminate between HIE and non-HIE subjects.

PP4: Potential Therapeutic Implications of a Ketogenic Diet for Sex-Specific Anxiety Disorders: A Focus on Microglial and Mitochondrial Modulation

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Preclinical studies have demonstrated the beneficial effects of the ketogenic diet (KD) on cognitive function and anxiety reduction. However, the underlying molecular mechanisms, particularly concerning sex-specific differences, remain unclear. This study investigated the impact of KD on anxiety-like behavior and microglial function in the hippocampus and prefrontal cortex (PFC) of male and female C57BL/6J mice. Results revealed that KD significantly reduced anxiety-like behaviors and enhanced motor function in male mice, but not in females. This sex-specific effect was accompanied by alterations in microglial morphology and gene expression in the male PFC, as well as changes in mitochondrial bioenergetics and dynamics. Specifically, β-hydroxybutyrate (BHB) increased mitochondrial respiration and reduced reactive oxygen species production in HMC3 cells. These findings suggest that the anxiolytic and motor-enhancing effects of KD may be mediated by sex-specific modulation of microglial function and mitochondrial dynamics, potentially providing a novel therapeutic avenue for anxiety disorders.

PP5: Metagenomics next-generation sequencing to characterize potential etiologies of infectious meningitis and encephalitis in Central India

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The burden of meningitis in low- and middle-income countries remains substantial, yet the infectious causes are largely unknown, hindering the implementation of evidence-based treatment and prevention decisions. To elucidate the etiologies of meningitis in Central India, we conducted a validation and application study utilizing unbiased metagenomic next-generation sequencing (mNGS). This DNA mNGS study was performed on cerebrospinal fluid (CSF) specimens collected from patients admitted to G.M. Taori CIIMS hospital, a tertiary care neurological hospital, with known meningitis cases (n=5), unknown meningitis cases (n=40), and no infection (n=2), as well as three environmental samples, between February 2023 and October 2023. We employed the IDseq bioinformatics pipeline and machine learning to identify potentially pathogenic microbes, which were then confirmed through orthogonal methods and followed up through phone and home visits. In samples with known etiology and without infections, there was 100% concordance between mNGS and conventional testing. In idiopathic cases, mNGS identified a potential bacterial or fungal etiology in 60%. We detected 16 instances of Mycobacterium tuberculosis (MTB), whose contigs exhibited 99% identity to each other and to a TB strain previously recognized to cause brain TB in India. Identification of other rare pathogens through mNGS as causes of meningitis highlights the need for development of customized bacterial panels for molecular diagnostic assays and the improvement of patient outcomes. This study highlights the potential of mNGS as a diagnostic tool for meningitis, enabling targeted and effective treatment strategies.

PP6: Virtual Screening and Antiviral Evaluation of Diallyl Disulfide against Japanese Encephalitis Virus

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Japanese encephalitis (JE) is the most common viral encephalitis caused by the Japanese encephalitis virus (JEV), affecting primarily children. Currently, there is no effective antiviral drug approved for the treatment of JEV infection. Thus, we conducted a virtual screening in four drug targets of JEV such as envelope protein (PDB ID: 3P54), NS3 helicase (PDB ID: 4R8T) NS3 protease (PDB ID: 2Z83), and NS5 RdRp (PDB ID: 4HDG) and evaluated antiviral activity of diallyl disulfide (DADS) in JEV challenged SH-SY5Y cells using MTT, plaque, and immunohistochemistry assays. Moreover, apoptosis and ROS assays were performed to check the cytoprotective effect of the compound in the JEV-infected cells. The drug showed variable antiviral potency in different treatment approaches with a significant cell viability of 75.4% and 77.85% in prior, and 78.3% and 83.62% in simultaneous treatments at 100 µM and 200 µM concentrations, respectively. A significant cell viability was observed in post-infection treatments even at lower concentrations with average cell viability of 75.87%, 79.15%, and 85.05% at 50, 100, and 200 µM concentrations, respectively. The antiviral activity of the drug was further validated in plaque titer reduction assay, in which DADS reduced plaque titer by 25.0%, 43.2%, and 59.8% in prior, simultaneous, and post-infection treatment approaches with median inhibitory concentration (IC_{50}) of 545.5, 227.4, and 165.8 µM, respectively. Similar antiviral effect was observed in immunocytochemistry assay in post-infection treatment mode. On top of this antiviral activity, DADS also demonstrated significant cytoprotective effect in JEV-challenged cells via its antioxidant activity. In conclusion, the results gave insight into the antiviral and cytoprotective potential of DADS in the management JEV infection.

Key words: Antiviral, Diallyl disulfide, Japanese Encephalitis virus, Organosulfur compounds, Virtual screening

PP7: Sex-specific effects of sucrose withdrawal on anxiety-like behavior and neuroimmune response

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Maladaptive neuroadaptations brought on by sugar bingeing exacerbate withdrawal symptoms and reduce dietary control. This study examined sex differences in sucrose bingeing, the negative mood effects of sucrose withdrawal, and the underlying neuroimmune response in the nucleus accumbens (NAc) and prefrontal cortex (PFC) of male and female C57BL/6J mice. Two-bottle sucrose choice paradigm was used to develop sucrose dependence in mice. Female mice consumed more sucrose than male mice when given free access to water and 10% sucrose for four weeks. A significant increase in the mRNA expression of neuroinflammatory markers (Il1β, Tnfα) was found in the PFC of males exposed to sucrose withdrawal. Sucrose bingeing and subsequent sucrose withdrawal showed elevated protein levels of pro-inflammatory cytokines/chemokines/growth factors in the PFC (IL1β, IL-6, TNFα, IFN-γ, IL-10, CCL5, VEGF) and NAc (IL-1β, IL-6, IL-10, VEGF) of male mice as compared to their water controls. These effects were concurrent with reduced mRNA expression of neuronal activation marker (cFos) in the PFC of sucrose withdrawal males. One week of sucrose withdrawal after prolonged sucrose consumption showed anxiety-like behavior in male mice, not in females. In conclusion, this study demonstrates that repeated access to sucrose induces anxiety-like behavior when the sugar is no longer available in the diet and these effects are male-specific. Elevated neuroinflammation in reward neurocircuitry may underlie these sex-specific effects.

PP8: Interplay between sex steroid hormones, receptors, and neuroinflammation in brain regions of temporal lobe epileptic rats

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Temporal lobe epilepsy (TLE) is one of the most common forms of drug-resistant focal epilepsy and is characterized by seizures that arise from the temporal lobe. There is a complex, bidirectional interaction between sex-steroid hormones and epilepsy. In addition, the occurrence of recurrent seizures in TLE is influenced by nearby temporal lobe structures besides the hippocampus. However, region-specific differences in the expression level of sex steroid hormones & their receptors are still elusive. Thus, the present study is designed to investigate the region-specific levels of sex-steroid hormones, their receptors, and neuroinflammatory markers in the TLE rat model. Adult male rats were administered with pilocarpine to generate the chronic model of TLE. Brain regions, namely, the anterior temporal lobe (ATL), hippocampus, and blood were extracted. Steroid extraction from blood and tissues followed by estimation of testosterone (T) and 17β-estradiol (E_2) were performed using specific ELISA kits. The qPCR of estrogen receptors (*Erα*, *Erβ*), androgen receptors (*Ar*), neurotrophic /growth factors (*Bdnf*, *Tgf-β*), astrogliosis marker (*Gfap*), microglial markers (*Iba1*, *Olfml3*) and pro-inflammatory marker (*Tnf-α*) was performed followed by Pearson's correlation test ($p < 0.05$). Expression of *Erα*, *Erβ*, *Ar*, *Bdnf*, *Tgf-β*, *Iba1*, *Olfml3, Gfap, Tnf-α* were upregulated in the hippocampus and ATL of the TLE rat. In hippocampus E_2 expression was increased and T was found to be reduced whereas in ATL both E_2 & T were found to be increased. Elevated levels of E_2 but decreased levels of T were found in the serum of TLE rats. *Erα*, *Erβ* showed positive correlation with E_2 in hippocampus and in ATL *Erα* & E_2 were negatively, *Erβ* & E_2 were positively correlated. Further, a positive correlation was observed between steroidal receptors and neuroinflammatory markers. There is a strong association between levels of hormones, their receptors, and neuroinflammatory/trophic factors in TLE pathogenesis.

PP9: The Neurotoxic Effects of Atrazine on Rat Brain Mitochondria: The Role of Betaine Intervention

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Atrazine, a broad-spectrum herbicide, is known for its potential risks to human health and the environment due to its persistence and mobility, which has led to harmful effects including the induction of oxidative stress and neuroinflammation. Betaine (BET), a compound well-regarded for its antioxidant properties in agriculture and human health, boosts non-enzymatic antioxidant defences through the methionine-homocysteine cycle and creates a protective membrane around cells. This study examines the impact of atrazine on oxidative stress markers and the expression of autophagy and mitophagy genes in the brains of Wistar rats, while also investigating the neuroprotective properties of betaine. Wistar rats were used as experimental models and were orally administered 0.9% saline (control), 100mg/kg atrazine, 400 mg/kg betaine, and a combination of both for 28 days. Biochemical analyses and molecular studies, including real-time PCR, were performed to assess oxidative stress levels and gene expression related to autophagy and mitophagy pathways. Biochemical analyses showed a notable reduction in catalase and superoxide dismutase activity in brain tissues treated with atrazine. Gene expression analysis indicated that atrazine interfered with pathways of autophagy and mitophagy, resulting in the accumulation of damaged organelles and proteins, which in turn contributed to neuronal damage. Co-administration of betaine with atrazine slightly improved antioxidant enzyme activities, and ameliorated atrazine-induced oxidative stress and disruptions in autophagy and mitophagy, as evidenced by gene expression changes. Atrazine induces neurotoxic effects in the brain by triggering oxidative stress and disrupting cellular processes. Betaine administration mitigated the harmful effects of atrazine, indicating that betaine could be a promising antioxidant for countering atrazine-induced oxidative stress in Wistar rats.

Keywords: Atrazine, Betaine, Oxidative Stress, Autophagy, Mitophagy, Wistar Rats, Neuroprotection, Antioxidant Defence system

PP10: Metagenomic Next-Generation Sequencing for Diagnosing Infectious Encephalitis and Meningitis: Addressing Technical Challenges and Enhancing Accuracy

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Infectious encephalitis and meningitis are severe conditions with high global morbidity and mortality rates, presenting significant diagnostic challenges due to difficulties in pathogen identification. Conventional microbiological methods, including smears, cultures, and polymerase chain reactions, often fall short because of the low volume of cerebrospinal fluid (CSF) and the protective blood-brain barrier. These limitations are exacerbated by the clinical and CSF similarities among viral, bacterial, tuberculous, and fungal infections, which can lead to inappropriate treatments and adverse outcomes. Metagenomic next-generation sequencing (mngs) has emerged as a promising solution, offering simultaneous and unbiased detection of all microorganisms in CSF samples. This study investigates the efficacy of mNGS for diagnosing infectious encephalitis and meningitis in Central India, where a significant proportion of cases remain undiagnosed and are treated empirically, resulting in substantial neurological morbidity. A total of 200 CSF samples, including 180 from cases of unknown etiology, 15 from known etiology, and 5 non-infectious controls, were collected and subjected to mNGS library preparation using the Illumina I seq platform. While mNGS offers comprehensive pathogen detection, several technical challenges were encountered, such as the low volume of CSF, contamination with host DNA that can obscure pathogen signals, and the need for advanced bioinformatics tools to accurately interpret diverse microbial genomes. Despite these challenges, the findings highlight mNGS's potential to improve diagnostic accuracy compared to traditional microbiological methods and emphasize the need for its broader adoption to address the diagnostic gap in neuroinfections, particularly in regions with high rates of undiagnosed cases.

Keyword: Meningoencephalitis, mNGS, India

PP11: GRAPE SEED POWDER SUPPLEMENTATION AND NEUROCOGNITIVE HEALTH AMONG ADULTS: A Randomized Controlled Trial

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Cognitive decline is a pressing public health issue demanding innovative preventive approaches. Preclinical studies have shown grape polyphenolic compounds (GPs) can mitigate cognitive impairment in Alzheimer's disease models. However, translating these findings to human research remains a challenge. Given grape polyphenols potential antioxidant properties and ability to protect neurons, a randomized, double-blind, placebo-controlled trial was conducted to evaluate the effects of grape seed powder (GSP) on cognition and neuropsychological status among adults with mild cognitive impairment (MCI). Adults between the age group of 40-90 years were enrolled and after preliminary survey, 120 participants received either GSP or placebo daily for 12 weeks (46g/day). Cognitive function was assessed using the Mini-Mental State Examination (MMSE), while neuropsychological status was measured with the Depression, Anxiety, and Stress Scale (DASS-21) at baseline, midpoint and after the 12 weeks. GSP supplementation significantly improved MMSE scores (3.50 ± 0.36, $p<0.001$) compared to placebo (0.50 ± 0.14, $p=0.005$) with respect to baseline scores. GSP also reduced depression levels (-1.68 ± 0.23, $p=0.001$) and anxiety (-0.61 ± 0.60, $p=0.041$) scores, while the placebo group experienced slight increases for depression and anxiety. Both groups reported minimal stress changes, but the increase was significant in the placebo group (0.48 ± 0.24, $p=0.010$). GSP was safe and well-tolerated. The findings of the study suggest that GSP may represent promising non-pharmacological interventional strategy to enhancing cognitive health and for mitigating depressive and anxiety symptoms in adults with MCI. However, further research employing biomarkers and brain scans is necessary to elucidate the underlying mechanisms, optimize intervention parameters, and explore the long-term effects of these interventions.

Keywords: Cognitive decline, Grapes, Mental health, Mild cognitive impairment, polyphenols.

PP12: Unraveling the Therapeutic Potential of Pramipexole in Disrupting Protein Aggregation Pathways: A Promising Approach for Alzheimer's Disease

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Amyloid aggregates are highly ordered, cross-β-sheet-rich protein/peptide structures associated with both human diseases and native functions. These aggregates can form under various conditions, resulting in regular arrays of β-sheet filaments or fibers of indefinite length, often coiled together in higher-order structures. The all-α helix multi-domain protein bovine serum albumin (BSA) is known to aggregate at elevated temperatures. Pramipexole (PPX) has been identified as a potential candidate for inhibiting and disaggregating preformed fibrils of BSA. The current investigation focuses on analyzing the recent trend in treating neurodegenerative diseases, which often face delays in medication due to their asymptomatic nature. The medication approach aims to alleviate symptoms rather than prevent the disease entirely. To address this, the study explores an "amyloid-aggregation" study as a potential solution for disease prevention. Various biophysical methods, including Thioflavin T (ThT) binding assays, 90° light scattering (RLS), and Congo red assay (CR), and in-vitro studies including Cell cytotoxicity assay and Cell proliferation assay using N2a Cell line have been conducted at different time points to gain insights into this area of research. It was observed that the equimolar ratio of BSA and PPX promotes the aggregation of native BSA whereas at higher concentrations ([BSA]/[PPX], 1:2 and 1:4) it inhibits the same. Congo red assay was used to confirm the presence of amyloid fibrils. The shift in the spectra of the BSA or BSA-PPX complex from the spectra of only CR confirms the presence of amyloid fibrils. The study concludes that PPX inhibits the formation of thermally-induced BSA aggregates by stabilizing the native conformation of BSA in vitro. The study suggests that further research on animal models is necessary to understand the efficiency of this process in intending to prevent the disease rather than just alleviating symptoms. The study highlights the importance of

understanding the molecular mechanisms of amyloid formation and the potential of small molecules as therapeutic interventions for neurodegenerative diseases.

Keywords: Aggregation, Amyloid fibrils, Bovine Serum Albumin, Pramipexole, Thioflavin T assay, Congo Red assay, Cell proliferation assay, Cell cytotoxicity assay

PP13: Dyslipidemia In Patients With Idiopathic Intracranial Hypertension

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Idiopathic intracranial hypertension (IIH) is characterized by increased intracranial pressure (ICP) occurring predominantly in women with obesity without an identifiable etiology. It causes blindness (in up to 24% of individuals) and long-term disabling headaches. There is positive correlation between central adiposity and IIH, despite not all IIH patients being obese, raises questions about IIH as a systemic metabolic disease. The patients presenting at PGIMER (Chandigarh) and fulfilling the revised diagnostic criteria (modified Friedman criteria, 2013) for IIH were included in this prospective pilot study carried out. They were assessed for neurological, visual and radiological outcomes. The demographic and clinical findings including the lipid profile {Cholesterol, Low Density Lipoproteins (LDL), Very Low-Density Lipoproteins (VLDL), Triacylglycerols (TAGs)} were explored. Thirty-six female IIH patients with mean age 35.22±10.6 were assessed. The mean BMI (kg/m2) of disease onset was 29.2±4.8. The median CSF opening pressure reported was 280mm (150, 400). The median Cholesterol, Triglyceride, LDL, HDL and VLDL reported were 169mg/dL (119, 354), 134mg/dL (58,309), 96mg/dL (37, 257), 46.7mg/dL (29.7, 116) and 27mg/dL (11.6, 51) respectively. There was significant correlation between lipid profile and the age. The patients above 34 years (N=16) of age had significantly higher levels of Cholesterol (p= 0.03) and LDL(p=0.012). Although no correlation was found between BMI and the lipid profile. It can be concluded that the higher age group in IIH patients are more prone to obesity although there was weak positive correlation between BMI and age (pearson correlation coefficient=0.05) in the cohort. The patients with higher CSF opening pressure reported low levels of HDL {Pearson correlation coefficient= -0.02 (negative correlation)}. The IIH patients are generally obese but not all the obese individuals develop IIH. However, we noted the differential increase in lipidogram in these patients suggestive of dysregulated lipid metabolic pathways.

PP14: Neurobehavioral and Gastrointestinal Effects of DSS-CUMS Integrated Model in Balb/c Mice: A Study on Gut-Brain Axis

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Irritable bowel syndrome (IBS) is a disabling functional gastro-intestinal disorder (FGID) characterised by altered bowel habits, bloating, visceral hypersensitivity. Despite the high clinical prevalence, there are limited models to mimic the disease. We investigated the effects of a combined model of dextran sulfate sodium (DSS) and chronic unpredictable mild stress (CUMS) on neurobehavioral and gastrointestinal (GI) functions in male Balb/c mice (8 weeks old). Mice were divided into four groups: Control, DSS, CUMS, and DSS-CUMS, with 8 animals in each group. Macroscopic parameters such as daily weight, food and water intake, colon length and weight, and visceral hypersensitivity (via abdominal withdrawal reflex) were measured. Disease Activity Index (DAI) was calculated based on weight loss, stool consistency, and bleeding. GI permeability was assessed using phenol red extravasation to estimate small bowel transit. Histological analysis of gut tissue was performed using H&E staining to estimate inflammatory cell infiltration. Neurobehavioral assessments were conducted using the open field test, sucrose preference test, elevated plus maze (EPM), marble burying test (MBT), and novel object recognition. Our results revealed significant neurobehavioral alterations, including changes in cognition, spatial memory, and anxiety. Additionally, colon morphology and oxidative stress markers indicated exacerbation of gut pathology in the DSS-CUMS group. However, motor activity, assessed via the pole test and actophotometer, showed no significant impairment. These findings highlight the relevance of the DSS-CUMS model in studying gut-brain interactions in irritable bowel syndrome (IBS).

PP15: Integrated Multi-Omics and Meta-Analysis Reveal Molecular Signatures and Therapeutic Targets Associated with Mitochondrial Dynamics and Peroxin in Alzheimer's and Huntington's Disease

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Recent studies have highlighted the significance of vitamin supplementation in maintaining mitochondrial function and energy production. Our multi-omics analysis revealed decreased levels of vitamins B2, B5, and B6 cofactors in Alzheimer's disease (AD) patients, leading to mitochondrial dysfunction and disease progression. We also observed deregulation of the OXPHOS system, including genes like NDUFS1, SDHB, and UQCRC1, which are essential for mitochondrial energy production. To extend our insights over neuronal diseases which has common phenotypes like mitochondrial dysfunction, we performed a comparative analysis AD and Huntington's disease (HD). Both diseases showed decreased expression of major OXPHOS genes, consistent with previous studies. Additionally, we found altered organellar fission, including mitochondria and peroxisomes. PEX11β, a key protein in peroxisomal division and biogenesis, was downregulated in AD and HD. Previous studies have shown that PEX11β expression is co-regulated with mitochondrial ATP synthase and other electron transport chain components. We confirmed the decreased PEX11β expression in *an in vitro* HD cell line and found that restoring its expression improved cell survivability. Our analysis establishes the involvement of peroxisomes in AD and HD pathology. Our study highlights the crucial role of vitamins B2, B5, and B6 cofactors in maintaining mitochondrial function and energy production in AD. We also demonstrate the link between peroxisomal dysfunction and AD/HD pathology, emphasizing the importance of proper peroxisome and mitochondria function in AD and HD. Our findings open up potential therapeutic targets for the treatment of these neurodegenerative diseases.

PP16: Screening of Phytocompounds for Developing Nanoformulation for Glioblastoma

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Glioblastoma (GBM) is the most frequent and deadly malignant adult tumor of the central nervous system (CNS). The five-year survival rates for high-grade gliomas are under 5%, even with improvements in treatment modalities. It is difficult to treat due to its quick progression, intra-tumoral heterogeneity characteristic, and difficulty in drug delivery across the blood-brain barrier (BBB). To address this issue, dietary flavonoids that target many molecular targets are the best choice for high-grade adult-type diffuse glioma therapy. In this context, nanoformulations are emerging as a potentially effective approach to improve the effectiveness of therapeutic agents. We are currently screening the range of phytocompounds from which the lead compound will be taken for the development of nanoformulations. Further research will be carried out on cytotoxic activity in the GBM cell line, by *in vitro* and *in vivo* investigations.

PP17: Pharmacological Investigations on Myricetin Encapsulated Chitosan Nanoparticles in Animal Model of Type I Diabetes Mellitus

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Diabetic Peripheral Neuropathy (DPN) is a prevalent and debilitating complication of diabetes, characterized by pain, tingling, and numbness, particularly in the lower limbs. Currently, available treatments majorly attenuate symptoms however it does not treat the underlying autonomic nerve deficit. Myricetin (MYR), a naturally occurring flavonoid with potent antioxidant properties, has shown promise in the treatment of diabetes by enhancing insulin action and reducing oxidative stress. However, the limited bioavailability of MYR has limited its therapeutic potential. Myricetin-loaded chitosan nanoparticles (MYR-CHT-NPs) are designed to increase MYR's oral bioavailability and efficacy. The aim of the study is to investigate both the acute toxicity profile and the therapeutic effects of MYR-CHT-NPs in the STZ-induced animal model of diabetic peripheral neuropathy. MYR-CHT-NPs effectively maintain glucose, albumin, AST, ALT, AP, bilirubin, cholesterol, creatinine, and electrolyte levels within the normal range, indicating no significant adverse effects. It also reduces blood glucose levels, maintains body weight within the normal range, and exhibits normal feed consumption and water intake. Further, we performed pain behavioral assays in diabetic rats which suggests that MYR-CHT-NPs treatment significantly attenuates thermal hyperalgesia, mechanical hyperalgesia, and cold hyperalgesia in diabetic rats. The study further demonstrated that MYR-CHT-NPs not only reduced elevated biomarkers such as urea and HbA1c but also effectively maintained normal levels of liver enzymes (AST, ALT) and electrolytes (Na, K), which are critical indicators of metabolic health. Thus, it can be concluded that MYR-CHT-NPs have the potential to overcome peripheral neuropathy associated with diabetes mellitus, thereby improving the lifestyle of patients.

PP18: Protective Role of Stigmasterol On Arsenic-Induced Neurotoxicity: An Integrated *In Silico* And *In Vivo* Study

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Arsenic, a toxic metalloid, is widely present in the environment, contaminating air, water, soil, and other resources, with groundwater being a major source of arsenic-induced toxicity in mammals. This study begins with an *in-silico* investigation, where stigmasterol was docked with key proteins involved in oxidative stress. The *in-silico* studies revealed that stigmasterol can interfere with the KEAP1-Nrf2 binding, as well as with BAX-Bcl2 interactions, showing strong binding affinities and antioxidant and antiapoptotic properties. In contrast, stigmasterol exhibited strong binding interactions with IKKβ and IKKα, proving its anti-inflammatory properties *in-silico*. Building on the *in silico* study, *in vivo,* experiments were conducted on male albino Wistar rats, divided into four groups: control, stigmasterol-treated (50 mg/kg), arsenic-treated (25 ppm), and a combined arsenic + stigmasterol-treated group. Both arsenic and stigmasterol were administered intragastrically for 28 days. Arsenic exposure reduced total antioxidant levels in brain tissues and increased ROS, indicating oxidative stress and neuronal damage. Histopathological analysis of the rat cerebellum using Hematoxylin and Eosin (H&E), Congo red, and Nissl staining provided additional evidence of stigmasterol's protective effects. This study highlights stigmasterol's potential as a therapeutic agent against arsenic-induced neurotoxicity in the rat brain. Biochemical assays for Superoxide Dismutase (SOD), catalase, and Acetylcholinesterase (AChE) activity further supported the findings, showing that stigmasterol mitigated oxidative stress and enzymatic disruptions caused by arsenic exposure.

Keywords: Arsenic, Neurotoxicity, Stigmasterol, Molecular Docking, In-Vivo study

PP19: HMGCR inhibitor restores mitochondrial dynamics by regulating signalling cascades in a rodent Alzheimer's Disease model

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Atorvastatin an HMGCR inhibitor may play a role in enhancing spatial, and long-term memory and combating anxious behaviour deficits induced by $A\beta_{1-42}$. Behavioural deficits studies, immunoblotting for the antioxidant/apoptotic protein expression, flow cytometry (FACS) for mitochondrial ROS, membrane potential (▲ψm), and histopathological alterations were performed against $A\beta_{1-42}$ toxicity. $A\beta_{1-42}$ was infused directly into the brain through *i.c.v* for the establishment of the AD model. Atorvastatin (ATOR) was administered orally used to treat AD in adult male Wistar rats aged between 200-250 g. We confirmed that ATOR administration significantly attenuates the $A\beta_{1-42}$-induced cognitive decline targeted mitochondrial-mediated age-dependent disease progression. Nrf2 stabilizes to interact SOD2 antioxidant enzyme, allowing transcriptional activity by the steep increase in ▲ψm and a reduction in ROS by activating mitochondrial superoxide scavenger and Nrf2-dependent pathway. The findings confirmed that ATOR has the potential efficacy to modulate the interference in cognitive decline induced by $A\beta_{1-42}$.

Keywords: Alzheimer's disease, $A\beta_{1-42}$, Atorvastatin, Neurodegeneration, Neuroprotection

PP20: Modulation of inflammation through COX-2 expression in LPS-treated J774A.1 macrophage using recombinant CD73

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Inflammation is a consequence of the innate immune response, the first line of the body's defense system, which recognizes the pathogen or any foreign particle and initiates the clearing and repair process. It is mediated by the production of cytokines, chemokines, reactive oxygen species, and secondary messengers such as cAMP and prostaglandins. Resolution of inflammation is necessary, but failing to do so can lead to chronic inflammatory conditions such as neurodegeneration, rheumatoid arthritis, and/or cancer. We have previously identified extracellular ATP (eATP) as a synergistic enhancer of cyclooxygenase 2 (COX-2)-mediated inflammation in macrophages and found that activation of purinergic P2 receptors enhanced the synthesis and stability of COX-2 mRNA, thereby sustaining inflammation. Therefore, the removal of eATP is an essential step for the resolution of inflammation. Ectonucleotidases such as CD39 and CD73 sequentially dephosphorylate ATP to ADP and AMP and further to adenosine, respectively. Several reports suggest that adenosine acts as an anti-inflammatory molecule by binding to P1 receptors. Therefore, we hypothesize that increased expression of ectonucleotidases, especially the rate-limiting enzyme CD73, might be a potential mechanism for the control of inflammation. Based on this hypothesis, in this project, we cloned, overexpressed, and purified CD73 in a bacterial system and tested its ability to reduce inflammation in lipopolysaccharide (LPS)-treated J744A1 macrophage cells. Modulation of inflammation by using recombinant ectonucleotidase would be an alternative to non-steroidal anti-inflammatory drugs (NSAIDs) to combat chronic inflammatory conditions, autoimmune disorders, and neurodegeneration.

PP21: Oxidative stress associated with rapid eye movement (REM) sleep loss and REM sleep deprived Parkinson's disease rat model: Role of Noradrenaline

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Parkinson's disease (PD) is associated with neurodegeneration and is accompanied with rapid eye movement (REM) sleep loss. Independent studies have shown that the latter (REMS—loss) elevates noradrenaline (NA) levels and promotes neurodegeneration in the brain. We hypothesized that elevated NA due to REM sleep loss is a key factor driving oxidative stress (OS) and neurodegeneration in PD as well. In this study we have examined the effects of REM sleep deprivation (REMSD) on OS markers in various brain regions of an induced PD rat model and evaluated the role of NA in modulating OS in PD. A PD model in rats was created by injecting 6-OHDA unilaterally into substantia nigra (SN). The injected rats showed significant motor and exploratory deficits in rotarod and open field tests, with immunohistochemistry confirming dopaminergic cell loss in SN. In other sets of experiments, 2- or 4-weeks post-injection, the rats were subjected to 96 hours REMSD by the standard platform method. 6-OHDA post-REMSD, the levels of reactive oxygen species (ROS) and superoxide dismutase (SOD) activity were estimated in locus coeruleus (LC), brain area rich in NA neurons and responsible for regulation of REMS and in SN. We observed a significant increase in ROS and a decrease in SOD in both regions of REMSD (LC) and in 6-OHDA injected PD rat models. REMSD n PD rats exhibited highest ROS and significantly reduced SOD activity, suggesting accelerated OS. REMSD in non-PD but 6-OHDA injected rats caused increased OS than in control non-REMSD PD rats, suggesting REMSD induced OS even without neuronal degeneration. Prazosin (α-1 adrenergic receptor antagonist) in REMSD rats reduced ROS levels and enhanced SOD activity, suggesting role of NA in REMSD-associated generation of OS markers. We propose that REMSD induced elevated NA exacerbates OS and accelerates progression of PD. The findings may be exploited for therapeutic benefits.

Keywords: Parkinson's disease; REM sleep deprivation; Oxidative stress; Neurodegeneration; Noradrenaline; Prazosin

PP22: Purinergic receptor-mediated amplification of COX-2 and PGE2 in inflammation

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Inflammation is a defense mechanism in biological systems, including responses against harmful stimuli, where the innate immune system offers immediate protection and the adaptive system provides targeted, lasting defense. Prostaglandin E_2 (PGE_2) is a key inflammatory mediator produced by cyclooxygenases (COX-1 and COX-2), which convert arachidonic acid into prostaglandins. COX-1 maintains normal physiological functions, whereas COX-2 is induced during inflammation, leading to increased PGE_2 production. Nonsteroidal anti-inflammatory drugs (NSAIDs) target COX activity to alleviate inflammation, but their effectiveness in chronic conditions is often limited, and they can cause adverse effects such as gastrointestinal ulcers, renal impairment, and cardiovascular risks. We propose a two-hit hypothesis for inflammation progression. The first hit involves macrophage activation by stimuli like bacterial lipopolysaccharide (LPS), tissue damage, or amyloid-beta deposits, initiating inflammation and cell damage. This process releases damage-associated molecular patterns (DAMPs), including extracellular ATP (eATP) and other nucleotides. The second hit occurs when these nucleotides activate purinergic (P2) receptors on immune cells, further amplifying inflammation through increased COX-2 expression. Under inflammatory conditions, eATP levels can rise from nanomolar to millimolar concentrations. Our research investigates how eATP modulates LPS-induced COX-2 expression and explores the potential of regulating eATP levels with ectonucleotidases such as recombinant CD73 (rCD73) and apyrase. We aim to understand the molecular mechanisms by which eATP elevates COX-2 levels, focusing on the signaling pathways involved. We also examined eATP and eUDP's roles in LPS- induced inflammation, conducting a purinergic receptor screening to identify receptors involved in enhancing COX-2 expression and PGE_2 production. Using RAW 264.7 macrophages, peritoneal macrophages, N9 microglial cells, and rat primary microglia, we demonstrated that LPS induces COX- 2 and PGE_2 synthesis, which is further amplified by eATP co-stimulation. Our screening revealed high expression of $P2Y_6$, $P2X_7$, and $P2X_4$ receptors, with $P2Y_6$ significantly enhancing LPS-induced COX-2 expression across immune cell types. Mechanistic studies indicated that eATP and eUDP activate the MEKK signaling pathway, leading to COX-2 transcriptional upregulation and stabilization of COX-2 mRNA through the p38 MAPK pathway. CDK-9 regulates transcription elongation, promoting inflammatory mediator expression, while transcription factors like IκB impact the NF-κB pathway, crucial for inflammation. Targeting P2X and P2Y receptors offers a promising strategy for developing Purinergic Receptor-Based Anti-Inflammatory Drugs (PBAIDs), potentially treating chronic inflammation by mitigating inflammatory responses while preserving COX enzyme functions.

PP23: Polyethylene-nanoplastic induced oxidative stress mediated neurotoxicity through disruption of gut permeability in Wistar rats

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Polyethylene micro and nanoplastics (MNPs) are increasingly recognized as emerging environmental contaminants with potential detrimental health effects. Multiple studies have documented MNPs mediated neurotoxicity, but underlying mechanisms remain enigmatic. Thus, the present study aimed to investigates the mechanism based toxicological profile of PE-NPs on behavioural alterations, and neurotoxicity in Wistar rats. For the present work environmentally relevant doses of PE-NP were orally administered to adult Wistar rats for a period of 35 days. A significant increase in oxidative stress marker were observed in both gut and brain including elevated levels of reactive oxygen species (ROS), malondialdehyde (MDA) and reduced antioxidant enzymes. Additionally, intestinal permeability was also impaired, as demonstrated by histological alterations in the ileum of the gastrointestinal (GI) tract. Disrupted gut permeability could have led to entry of PE-NP into systemic circulation and inflammation. Furthermore, systemic inflammation and PE-NP induced corporal injury could be responsible for observed Blood-Brain Barrier (BBB) disruption, altered AChE level and neuro-behavioural impairments. ROS mediated leaky gut and disrupted BBB formed the basis for MNPs induced impaired cognitive functions and depression-anxiety-like behaviour. Therefore, these findings highlight the potential hazards of polyethylene nanoplastic exposure to neurological health.

Keywords: Microplastics, Nanoplastics, Gastrointestinal toxicity, Neurotoxicity, Neurobehavioral deficits.

PP24: Establishing a model of Streptozotocin induced sporadic Alzheimer's Disease in Zebrafish

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Alzheimer's Disease is a global burden with no viable treatment options available till date. Rodents and primates have been extensively used for understanding AD pathogenesis. Recently, zebrafish have become a popular choice of model organism for neuroscience research. Their high similarity to humans, conservation of main genes causing diseases, relatively simpler nervous system, shorted duration of model induction and cost effectiveness have promoted chemically induced and transgenic models of zebrafish models for studying AD. In this study, a robust and reliable model of chemically induced sAD has been established and validated. ICV-STZ injection was given to adult zebrafish in different doses and their survival rate was observed. The selected doses where subsequently used to evaluate behavioural, molecular and histological symptoms of sAD. The STZ injected fish displayed anxiety and stress with cognitive impairment. They exhibited increased expression of genes and proteins involved in AD pathology. Histological examination revealed marked signs of neurodegeneration with amyloid plaques and taupathy. Electron microscopy revealed the presence of fibrils with twists. The ICV-STZ injection of 5 mg/kg body weight for 7 days proved to be the most effective dose for inducing sAD pathology. Based on literature review, this is the first study of its kind to induce ICV-STZ sAD model in zebrafish.

PP25: White Matter Hyperintensity Load: A Key Determinant of Cognitive Status and 'Brain Age'

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White matter hyperintensity (WMH), a brain lesion resulting from cerebral small vessel diseases with chronological aging, is depictive of fiber loss and fiber pruning. WMH load beyond a threshold is likely to pose vascular insults to neuroanatomic structures thereby leading to altered cognitive and brain health compared to the subjects without WMH. While studying the T2-FLAIR MRI and Cognitive data of the National Alzheimer's Coordinating Center (NACC) and Alzheimer's Disease Neuroimaging Initiative (ADNI), it was intriguing to note that even within the set of subjects classified as Cognitively normal (CN), a significant subset of subjects had WMH load in the brain while another subset of the subjects had no/very low-WMH. It motivated us to investigate the impact of WMH on Brain Age in subjects with WMH load compared to the subjects without WMH load. We conducted a comprehensive MRI segmentation of neuroanatomic structures along with WMH quantification in groups of CN, cognitively impaired (CI), and cognitive impairment owing to Alzheimer's Disease (CI-AD) from the NACC and ADNI cohort using T1-weighted and T2-FLAIR MRI. Furthermore, the quantified volume/thickness of 178 neuroanatomic structures, periventricular WMH, and deep WMH volume were utilized to train a boosting algorithm to predict Brain Age. Brain Age Gap (BAG) was calculated as the difference between CA and estimated Brain Age. Using the neuroanatomic and WMH volume and the supervised machine learning models, we established that a minimum set of three brain quantities, Total brain (GM+WM), CSF, and WMH volume, provides the Optimal quantitative features discriminative of cognitive status as CN, CI, and CI-AD. We also observed that the CN subjects with elevated WMH load (5 - 10 ml) had increased Brain Age (+0.6 to +4 years) than the chronological age. Increased 'Brain Age' in the subjects with elevated WMH is suggestive of WMH-induced vascular insult leading to accelerated and early structural loss than expected for a given chronological age. Henceforth, this study establishes that quantification of WMH together with an optimal number of neuroanatomic features is important to delve into the biological underpinning of aging and aging-associated cognitive disorders.

PP26: Curcumin-Loaded Gelatin Nanoparticles: A Promising Therapeutic Approach for Glioblastoma Treatment.

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The most common and deadly malignant tumour of the adult central nervous system is called glioblastoma (GBM). For high-grade gliomas, the five-year survival rate is still less than 5%, even with advances in treatment. Treatment for GBM is extremely challenging due to its aggressive progression, intratumoral heterogeneity, and the difficulty of getting medications across the blood-brain barrier (BBB). Curcumin, a naturally occurring polyphenol with multi-targeted effects, has showed promise as a treatment alternative for GBM. Its quick breakdown and low absorption, however, restrict its clinical use. Curcumin-loaded gelatin nanoparticles have become a unique strategy to address these issues, improving curcumin's stability, bioavailability, and targeted delivery to GBM cells. Curcumin-loaded gelatin nanoparticles are presently being developed and optimised. In vitro and in vivo studies will be carried out to assess the cytotoxic potential of these particles against GBM cell lines.

PP27: Kynurenine Pathway Dysregulation and its Link to Subclinical Inflammation in Drug-Naïve and treated Schizophrenia Patients: Insights from an Indian Cohort

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Schizophrenia (SCZ) is a complex neuropsychiatric disorder influenced by both genetic predisposition and environmental factors. While classical neurotransmitters has been extensively studied in SCZ, the role of neuromodulators, particularly kynurenine pathway (KP) metabolites resulting from tryptophan catabolism, remains less explored. As dysregulation of the KP metabolites contributes to neuroinflammation, neurodegeneration, and cognitive deficits, we studied the systemic levels of KP metabolites and their association with inflammation, aiming to identify potential markers of brain dysfunction and systemic inflammation in SCZ patients. We recruited 230 individuals, patients with Drug Naïve SCZ (DN) (n =66), risperidone-treated SCZ (RT) (n = 87), and healthy controls (n=75) (HCs), from a large tertiary neuropsychiatry center. Serum levels of Tryptophan (TRY), kynurenine (KYN), Kynurenic acid (KYNA) and 3-Hydroxy kynurenine (3-HK) were measured by LC-MS-MS, TNF-α, IFN-γ, IL-10 were quantified using ELISA. We found lower systemic levels of TRY and higher KYN, KYNA in patients with DN and RT compared to healthy controls ($p<0.001$). However, 3-HK was elevated only in DN compared to HCs ($p<0.05$). TNF-α, IFN-γ and IL-10 were significantly elevated in SCZ group (both DN & RT) compared to HCs ($p<0.001$). KP metabolites were significantly correlated with inflammatory cytokines ($p<0.01$). Individuals with SCZ had higher independent odds of TRY (OR: 0.37, 95% CI: 0.25–0.55), KYN (OR: 1.97, 95% CI: 1.39–2.78), KYNA (OR: 3.43, 95% CI: 2.32–5.08), and KYN-TRP ratio (OR: 6.27, 95% CI: 3.35–11.72) after adjusting for the potential confounders namely Age, Body mass index (BMI), Family history of SCZ, Fasting blood sugar (FBS), Cholesterol (CHO), Triglycerides (TRIG) and Neutrophil-to-Lymphocyte Ratio (NLR). Our study demonstrates an association of KP metabolites in Asian Indian SCZ patients and correlates with systemic inflammation. The observed disparities in KP metabolites between individuals with SCZ and HC suggest a potential impact on brain kynurenine levels, which are critically involved in modulating symptom severity and cognitive function in SCZ. The interplay between the KP metabolites & systemic inflammation appears to be an important factor in the pathophysiology of schizophrenia and a promising area for further research and potential interventions.

PP28: Biomarkers of Oxidative Stress in Idiopathic Parkinson's Disease

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This study attempted to comprehend the role of oxidative stress in Idiopathic parkinson's disease (IPD). IPD is a type of Parkinson's disease, which is the second most neurodegenerative conditions affecting the central nervous system (CNS). In IPD root cause of occurrence of disease is completely unknown, it is characterized by cognitive dysfunction and motor impairments. The primary cause of this disease are degeneration of dopaminergic (DA) neurons and accumulation of α-synuclein in the substantia nigra pars compacta. There is currently no proven cure for the illness. Finding novel therapeutics that help IPD patients stop their disease from progressing is the focus of many studies. Since IPD is age-related and metabolic disorder oxidative stress plays a major role in progression of disease. The present study aims to evaluate the levels of two oxidative stress biomarkers, superoxide dismutase (SOD) and Malondialdehyde (MDA) in blood of IPD patients. The venous blood of the around 80 subjects was collected using venipuncture method for the study with their informed consent. The study SOD in around IPD patients and healthy control were performed using pyrogallol auto-oxidation assay. MDA activity was calculated using TBARS assay in human blood samples. Based from the data evaluated the levels of SOD and MDA were significantly high in IPD patients as compared to healthy control. The etiology of IPD is significantly influenced by neuroinflammation, mitochondrial dysfunction and DA metabolism. Its still unclear which substances could lead to oxidative stress in IPD. In coming years, neuroprotective treatments will need to address a variety of pathogenic pathways, including neuroinflammation, oxidative stress, mitochondrial dysfunction. The level of oxidative stress biomarkers is statistically higher as compared to age-matched healthy control. Based on the present study it signifies the role of oxidative stress in the initiation of cascade of metabolic pathways that leads to neuronal demise in IPD patients.

PP29: Trazodone mitigates behavioral disturbances in scopolamine-induced cognitive deficits by modulating BDNF-CREB signaling in the hippocampus of rats.

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Trazodone, an antidepressant may play a role in enhancing long-term memory combining anxious behavior deficits induced by scopolamine. We propose the potential novel mechanistic insights that link up between oxidative stress and memory biomarkers including BNDF and CREB pathways to modulate the pathogenesis of AD-like symptoms. Behavioural deficits studies, biochemical determination of lipid peroxidation and acetylcholinesterase activities, and immunohistochemistry of BDNF and CREB were performed against scopolamine-induced AD-like symptoms. Moreover, histopathological alterations were also performed against an AD-like model. $A\beta_{42}$ proteins immunofluorescence was performed due to its known mechanism under AD. Scopolamine-induced intraperitoneal into the rats for the establishment of an AD-like model. The findings confirmed that TRAZ could be useful in mitigating the negative effects of scopolamine-induced cognitive impairment and lowering oxidative stress by enhancing memory indicators.

Keywords: Trazodone, Scopolamine, Neurodegeneration, Neuroprotection

PP30: Investigating the Therapeutic Role of Atomoxetine in Ministration of Rheumatoid Arthritis Pain

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Rheumatoid Arthritis (RA) is a chronic autoimmune disorder characterized by significant pain due to synovial inflammation and joint damage. Recent studies shows the potential of atomoxetine in chronic pain conditions by inhibiting the neurotransmitters release and modulating pain pathways. The central sensitization of pain contributing to hyperalgesia and allodynia through neurotransmitters like glutamate and substance P. This study investigates the atomoxetine, a selective norepinephrine reuptake inhibitor and NMDA receptor blocker as a potential treatment for RA-associated pain. In a Complete Freund's Adjuvant (CFA)-induced rat model of RA atomoxetine significantly reduced thermal, mechanical, cold hyperalgesia and allodynia indicating its efficacy in managing evoked pain. Atomoxetine significantly alleviates ongoing pain in CFA-induced rats and do not show any drug seeking behaviour in naive rats. Additionally, atomoxetine restored oxidative and nitrosative stress markers including glutathione (GSH), malondialdehyde (MDA) and nitrite levels in the sciatic nerve highlighting its potential neuroprotective effects. Importantly, atomoxetine did not induce any CNS toxicities as evidenced by the open field and Rotarod tests confirming its safety at therapeutic doses. These findings suggest that atomoxetine could be a novel therapeutic option for addressing the unmet medical need of chronic pain in RA and offering hope for improved quality of life in patients suffering from this debilitating condition.

PP31: Nanostrategies for overcoming multidrug resistance

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Every year, cancer accounts for a vast portion of deaths worldwide. Chemotherapy involves the use of specific chemodrugs to inhibit the proliferation of cancer cells, but the frequent emergence of multidrug-resistant cancer cells poses a tremendous threat. Multidrug resistance (MDR) can lead to metastatic progression and disease relapse. Therapy has limited success in treating MDR due to the varying pharmacokinetic properties of drugs and tumor heterogeneity. These remain significant challenges for many cancer patients. Currently, different nanoformulations are developed to enhance therapeutic efficiency. Hollow mesoporous nanoparticles will be developed and characterized for glioma therapy both in vitro and in vivo.

PP32: The Domino Effect: Mapping the Havoc Triggered by Carbon Black Nanoparticles in Mice Brain through Oral Intake

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Carbon black nanoparticles (CBNPs) are witnessing a significant surge in demand and are increasingly being exposed to humans due to their widespread use in many sectors and applications from the scientific to the industrial levels, making it crucial to carry out thorough examinations of their potential effects on human health. CBNPs fed orally to mice for 30 days using varying quantities, specifically 5mg/kg, 10mg/kg, and 20mg/kg of the mice's body weight provoked detrimental effects in the mice. The adverse impact of CBNPs was assessed by many factors like physiological responses of the brain and genetic makeup. Prolonged exposure to CBNPs resulted in physiological alterations of the brain evidenced by GABA and Dopamine being affected drastically. Total proteins, Catalase, Alkaline Phosphatase, and Total ATPases were also seen to alter. The cytoarchitecture of the brain showed significant changes, including cell inflammation, Purkinje cell degeneration, tissue necrosis as well as CBNPs accumulation in the cerebellum (C), cerebral cortex (CO), medulla oblongata (MO), olfactory lobes (OL), and hippocampus (H) tissue. Genotoxicity was evident via the DNA fragmentation revealed by the Comet assay. Chromosomal aberrations also showed deletion and duplication in the experimental groups. Consequently, it may be inferred that exposure to CBNPs may result in a disruption of physiological processes, which could ultimately lead to the development of severe and potentially fatal diseases.

PP33: Rutin Inhibited Viral Entry and Replication and Exerted neuroprotection against JEV

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Japanese encephalitis virus (JEV), belonging to the virus family of Flaviviridae, is a threat to more than three billion people globally despite the availability of vaccines. This study considered the urgent need for an effective drug against JEV by evaluating the antiviral and neuroprotective activities of chicoric acid (CA) and rutin in the *in vitro* and *in vivo* models of JE. In the *in vitro* study, although CA and rutin exhibited variable antiviral potency with IC_{50} values ranging from 11.03 μM to 24.04 μM for CA and 16.45 μM to 26.84 μM for rutin in different treatment approaches, they demonstrated strong antiviral effect via inhibition of the virus entry into the host cells. In addition, treatment of JEV-infected SH-SY5Y cells with these compounds significantly reduced the intracellular viral load, the proportion of apoptotic cells, and the ROS level in a dose-dependent manner. In the *in-vivo* experiments, rutin demonstrated a dose-dependent increase in survival, with the highest dose (50 mg/kg) significantly improving survival rates and reducing the severity of encephalitis symptoms. An in-depth analysis of brain and serum samples from treated mice revealed a substantial reduction in infectious viral particles, viral RNA, and viral NS3 protein levels, particularly at 25 and 50 mg/kg. Rutin also enhanced antiviral gene expression, including IFNα, IFNβ, and IFIT1, suggesting a robust antiviral response. Additionally, rutin significantly mitigated JEV-induced neuroinflammation by decreasing microglial activation, inflammasome formation, and proinflammatory cytokine levels. Collectively, these findings gave insight into the antiviral and neuroprotective potential of rutin in the development of drugs for JE.

Key Words: Antiviral, chicoric acid, Japanese encephalitis virus, neuroprotective, phytocompounds, rutin

PP34: Investigating Therapeutic Potential Of Bergenin For The Treatment Of Chronic Constriction Injury-Induced Neuropathic Pain

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Neuropathic pain, a widespread and debilitating condition, arises from nerve damage and is frequently associated with comorbidities such as diabetes, stroke, and cancer. Current therapeutic options are limited by significant adverse effects, highlighting an urgent need for novel treatments. This study investigates the therapeutic potential of bergenin, a bioactive compound, on chronic constriction injury-induced neuropathic pain and unravels the cellular and molecular mechanism of neuropathic pain. Behavioral assays were conducted to assess pain responses before and after CCI induction at various time points following bergenin and gabapentin administration. To evaluate potential central nervous system (CNS) toxicity, locomotor and motor activities were monitored, given that these are common adverse effects associated with standard neuropathic pain medications. Oxidonitrosative stress parameters were measured to assess the biochemical impact of bergenin treatment on nerve-injured rats. Furthermore, we examined the expression of key molecular markers such as tumor necrosis factor-alpha (TNF-α), ionized calcium-binding adapter molecule 1 (IBA1), intercellular adhesion molecule 1 (ICAM1), transient receptor potential vanilloid 1 (TRPV1), and calcitonin gene-related peptide (CGRP) in the spinal cord of affected rats. Our findings revealed that bergenin administration significantly ameliorated the CCI-induced alterations in pain perception and reduced the expression of pro-inflammatory and oxidative stress markers. The data suggest that bergenin exerts its analgesic effects by modulating the neuroinflammatory cascade and inhibiting TRPV1 nociceptors in the spinal cord. These results indicate that bergenin may serve as a promising therapeutic agent for the management of neuropathic pain, offering an effective alternative to conventional treatments with fewer adverse effects. This study provides valuable insights into the potential use of bergenin for the development of improved therapeutics targeting neuropathic pain.

PP35: MITOCHONDRIAL DYSFUNCTION IN EXPERIMENTAL CEREBRAL MALARIA

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Cerebral malaria is a neurodegenerative disease which is caused by infection with the protozoan *Plasmodium falciparum* in humans. Even after clearance of the parasite, 25% children develop cognitive impairment. Iron chelation therapy along with antimalarials has been shown to enhance recovery in comatose patients. Iron-sulfur clusters serve as an important cofactor for various proteins including the Iron Regulatory protein 1 (IRP1). IRP1 can modulate the translation of proteins involved in iron transport by binding to Iron Responsive Elements (IREs) in the 5' or 3' untranslated regions. Inhibition of mitochondrial complex I of the electron transport chain has been shown to inhibit the synthesis of Iron-sulfur clusters which can therefore alter iron transport. In this study, we want to check mitochondrial metabolism in experimental model of cerebral malaria (ECM) and its effect on IRP activity and ultimately on the expression of iron transport proteins. Additionally, we want to check if activation of SIRT3, a histone deacetylase known to regulate mitochondrial metabolism can restore Complex I activity and prevent neurocognitive sequelae in infected animals. So far, we have shown that there is increased ROS production in the brain tissue of the infected animals which can disrupt the activity of electron transport chain. In line with this, we also found reduction in the activity of Complex I and loss of ferroportin, the only protein known to export iron out of the cell hence proving that there is iron accumulation.

PP36: Vocalization Patterns as Early Indicators of Autism Spectrum Disorder: Insights from a Chick Model Study

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Autism Spectrum Disorder (ASD) is a complex neurodevelopmental condition primarily characterized by impaired social communication, as well as repetitive and restrictive behaviors. The heterogeneous nature of ASD complicates early diagnosis as current detection methods are largely behavioral and lack evidence-based tools. Infant cries are an innate form of communication observed from birth and can provide insights into an infant's neurological and physical state. This study hypothesizes that vocalization differences that can be observed in animal models like chicks (vocal at hatching) could serve as a proxy for early communication patterns linked to ASD. For this study, Chicks were divided into a Control group and an Experimental group (Valproic acid-induced ASD group). Vocalizations were recorded in a soundproof room using a brooding cage to reduce external noise and stress-related sounds. Each recording session lasted about five minutes, providing sufficient data for analysis. Acoustic parameters such as frequency, amplitude, and pitch were analyzed using RAVEN PRO 1.6 software, which generated detailed spectrograms. The spectrogram analysis highlighted atypical patterns in the Valproic acid-treated group and further analysis of the vocalizations revealed distinct acoustic variations between the control, vehicle control, and experimental groups. Chicks treated with Valproic acid exhibited significant alterations in pitch, frequency range, and increased variability compared to both the control and vehicle control groups. These changes suggest disruptions in normal vocalization patterns, possibly due to underlying neurodevelopmental alterations associated with ASD. These acoustic anomalies mirror the atypical vocal patterns observed in human infants with ASD, and hence, these findings suggest that the acoustic markers identified could serve as early indicators of ASD at birth, offering a non-invasive method for early diagnosis leading to timely medical intervention during early post-natal life. Further research is needed to refine the model and explore the translational potential of these findings.

PP37: Sex-specific alteration in longevity and metabolic profile in Alzheimer's disease flies: Combinational impact of Curcumin and Resveratrol

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Alzheimer's disease (AD) is one of the most prevalent neurological conditions, but effective treatments to prevent or reverse AD are still elusive. Curcumin and Resveratrol, members of two different families of phytochemicals, are reported to alleviate the symptoms of AD. However, their combined and sex-specific impact on AD pathophysiology is not known. Thus, AD flies were reared on 1) Standard Banana- Jaggery media, 2) Media containing Curcumin (C), 3) Resveratrol (R) and 4) Combination of Curcumin and Resveratrol (C+R) and maintained at 25°C under 12L:12D condition. Survival assay, Whole body protein content, Lipid content, Carbohydrate content, and Trehalose content were measured for all groups of AD flies on different developmental ages, i.e. Days 0,7,10,14 and 21 for both males and females. The lifespan of male and female AD flies fed with C+R-containing media was significantly improved compared to other groups. The C+R group showed a significant increase in whole-body protein content of males at day 0, 14, and 21, whereas at only day 10 in females. Further, lipid content significantly decreased at Day 0 in male flies but was significantly higher as the diseases progressed at the age of 14 and 21 days. In females, it was significantly high at day 10 in the C+R group. The trehalose content was significantly low at day 0 in males and was significantly high at day 14 in females in the C+R group. Further, total carbohydrate content was observed as high at day 14 in males and at day 14 and 21 in females in the C+R group. Hence, Curcumin and Resveratrol, in combination, improve metabolite levels and increase the longevity of AD flies in a sex-specific manner.

PP38: Antiepileptic effect of naringenin loaded nanoparticles in the experimental model of post-traumatic epilepsy

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Epilepsy, a severe neurological disorder is characterized by the prevalence of sudden and recurrent seizures. The neurological disorder is known to affect millions of human populations around the world. Naringenin is a flavonoid compound known for its anti-inflammatory, antidiabetic, antioxidant, and other pharmacological properties. However, low bioavailability makes it challenging to establish as an effective therapeutic agent. Hence, we intended to evaluate the therapeutic potential of naringenin-loaded nanoparticles in iron-induced experimental epilepsy. The polymer-based naringenin loaded nanoparticles were prepared by nanoprecipitation method and characterized. Epilepsy was induced in the rats by injecting $FeCl_3$ (5 µl of 100 mM solution) intracorticaly and 15 days post-surgery, free naringenin and naringenin loaded nanoparticles were orally administered for 15 consecutive days. Electrophysiological, behavioral, and molecular analyses were used to assess the disease-modifying potential of naringenin-loaded nanoparticles. The results showed that naringenin-loaded nanoparticle attenuate epileptic seizures and cognitive impairment in epileptic rats. Further, the study demonstrated that the naringenin loaded nanoparticles modulated the glial activation and reduced TNF-α expression in the brain of epileptic rats. Overall, the study suggest that naringenin-loaded nanoparticle possesses potential anti-seizure and anti-inflammatory effect in the iron-induced experimental epilepsy.

Keywords: Naringenin, nanoparticles, iron-induced epilepsy, inflammation, seizures

PP39: Involvement of PDGFRα and Integrin activated signalling pathways in Glioblastoma Anoikis Resistance

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Anoikis resistance, the evasion of programmed cell death when cells detach from the extracellular matrix (ECM), is a critical feature of glioblastoma (GBM) malignancy, contributing to tumor survival, spread, and resistance to therapy. We focused on the role of growth factor receptors, particularly PDGFRα, and integrin expression patterns in mediating this resistance. Through *in silico* analyses, we observed significant correlations between PDGFRα and integrins, highlighting their role in activating survival pathways in GBM cells. We also identified specific miRNAs that enhance anoikis resistance by targeting PDGFRα and integrins. To experimentally investigate these mechanisms, GBM cells were cultured under non-adherent conditions using poly-HEMA-treated plates to induce anoikis resistance. We performed various assays, including cell survival, migration, sphere formation, Western blotting, and qPCR. In patient-derived GBM tissue samples, we observed elevated protein levels of PDGFRα and integrins, reinforcing our hypothesis about their involvement in anoikis resistance. To delineate the role of PDGFRα signaling in anoikis resistance, we further employed pharmacological inhibitors of key signalling molecules such AG1295 (PDGFRα blocker), HS173 (PI3K Inhibitor), U0126 (ERK Inhibitor), AG490 (JAK-STAT Inhibitor), etc., which led to a decrease in cell survival, proliferation, and migration. Additionally, Western blot analysis showed upregulation of Integrin β1 and Integrin β3 in both adherent and non-adherent cells, along with the presence of different glycosylated integrin forms, suggesting modifications that may enhance anoikis resistance. In conclusion, our findings indicate that PDGFRα and integrins are central to the mechanisms driving anoikis resistance in GBM, with additional contributions from growth factors, miRNAs, and kinases. Targeting these pathways could offer new therapeutic strategies for combating GBM.

PP40: *In-silico* Identification and *In-vivo* Evaluation of Liver X Receptors Modulators in Sporadic Alzheimer's Disease

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Alzheimer's disease (AD) is the most common cause of dementia. Evidence indicates that progression of AD is linked to aberrant cholesterol metabolism and inflammation and can be modulated by liver X receptors (LXRs). Therefore, present study was designed to evaluate the potential of vitamin D (D3) and podocarpic acid (PA) as LXR modulators in sAD model. Structure-based virtual screening (SBVS) was employed to identify the LXR modulators from natural curated library. Male Sprague Dawley rats weighing 300-350g were used to evaluate the selected LXR modulation effects. Cognitive deficits were done using MWM, Y-maze, NOR and OFT tests. Gene and protein expressions of LXRs and its target genes were done using qPCR and immunoblotting. Histological analysis was done using H&E, Nissl staining and NeuN immunofluorescence. Golgi-Cox was performed to evaluate the axonal and synaptic plasticity. SBVS identified vitamin D3 (D3) and podocarpic acid (PA) as potential LXR modulators. Neuro-behavior tests showed significant improvement in memory and cognition following both the treatments. Gene expression of LXRs and its target genes (ABCA1) were significantly altered in STZ-treated animals and found differentially restored on D3 and PA treatment. D3 treatment significantly decreased the cell loss. Moreover, Golgi-cox and synaptic plasticity marker expression studies suggested D3 rescued synaptic plasticity loss. D3 evidenced efficacy in attenuating STZ-induced neuronal damage and cognitive deficits displaying neurotherapeutic effects. On treatment with D3 and PA, LXRs transcriptional activation seems to play a role in the above-mentioned beneficial effects. In conclusion, our results reveled that D3 and PA treatment attenuated ICV-STZ induced neurobehavioral and histological abnormalities. The neuroprotective effect of these treatments could be attributed to the activation of LXRs in modulating memory deficits and morphological alterations. Therefore, strategies like modulating the activity of LXRs in brain of AD individuals might offer prophylactic strategies to combat the disease.

PP41: To investigate the impact of High Fat Diet induced metabolic dysfunction on Cognitive performance in mice model

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Non-Alcoholic Fatty Liver Disease (NAFLD) is a rapidly growing multifactorial chronic disease characterized by excessive accumulation of hepatic triglycerides. Recent estimates indicate a global prevalence of NAFLD at 32.4%, projected to increase to 56% in the next decade, with the incidence doubling over the past ten years. The clinical burden of NAFLD extends beyond liver-related morbidity and mortality, with emerging evidence suggesting that it significantly affects cognitive performance and behavior. This study investigated the impact of high-fat diet-induced metabolic dysfunction on cognitive performance in a mouse model. We assessed various anthropometric parameters, including body weight, thoracic circumference, abdominal circumference, and body mass index (BMI), to evaluate the presence of NAFLD in the rodents. Neurobehavioral alterations were systematically examined using established tests: the Rotarod Test assessed motor coordination and balance, while the Open Field Test (OFT) measured general locomotor activity and anxiety levels. The Elevated Plus Maze (EPM) provided insights into anxiety behaviors, and the Morris Water Maze (MWM) evaluated spatial learning and memory capabilities. Additionally, the Novel Object Recognition (NOR) Test assessed visual memory
formation. All behavioral tracking was performed using ANY-maze video tracking software, ensuring accurate data collection throughout the experiments. The results highlight effect of diet high in fat on brain functions, thereby supporting the notion that metabolic dysfunction has a significant impact on overall brain health and cognitive function.

PP42: Modulation of PI3K Attenuates Cognitive Dysfunction in Rat Amyloid-beta Model of Alzheimer's Disease

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Phosphoinositide 3-kinase (PI3K) plays a critical role in phosphorylating PIP2 and PIP3, which subsequently activates Akt and various downstream signaling molecules essential for neuronal health, growth, plasticity, and memory formation. In this study, the neuroprotective effects of two phytoconstituents, Polydatin and Santonin, PI3K modulators, were evaluated in rat models of Alzheimer's disease (AD) induced by bilateral intracerebroventricular (icv) administration of amyloid beta. After a week of recovery, Polydatin (25, 50, and 100 mg/kg), Santonin (25, 50, and 100 mg/kg) & Donepezil (5 mg/kg) were administered by oral gavage in Aβ-injected rats for 21 days. Morris Water Maze (MWM) and Novel Object Recognition Test (NORT), were used for the assessment of learning and memory. Polydatin treatment showed a significant reduction in escape latency ($p<0.001$) from day 2 onwards, with notable improvements in probe trials (day 5) including increased platform crossings ($p<0.01$) and target quadrant time ($p<0.01$) at higher doses. In the Novel Object Recognition Test, Polydatin treatment significantly ($p<0.01$) improved the discrimination index of Aβ-injected rats, while Donepezil showed stronger effects ($p<0.001$). Similarly, Santonin demonstrated significant reductions in escape latency ($p<0.001$) by day 2, with enhancements in probe trial crossings ($p<0.05$) and time spent in the target quadrant ($p<0.001$). In NORT, the discrimination index of rats also improved significantly post Santonin treatment ($p<0.001$). Findings from the present study suggests that both Polydatin and Santonin have promising neuroprotective effects in rat model of Aβ-induced Alzheimer's Disease. Further studies including biochemical, molecular & cellular assays are underway to investigate the possible mode of action of Polydatin and Santonin.

Key words: Phosphoinositide 3-kinase (PI3K); Alzheimer's; Polydatin and Santonin; Amyloid beta; Cognition

PP43: Environment Enrichment Promotes Memory Consolidation *Via* Behavioral Tagging

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The behavioural tagging (BT) hypothesis provides crucial insights into the mechanism of long-term memory (LTM) consolidation. Novelty exposure in BT is a decisive step in activating the molecular machinery of memory formation. Several studies have validated BT using different neurobehavioral tasks; however, the novelty given in all studies is open field (OF) exploration. Environment enrichment (EE) is another key experimental paradigm to explore the fundamentals of brain functioning. Recently, several studies have highlighted the importance of EE in enhancing cognition, LTM, and synaptic plasticity. Hence, in the present study, we investigated the effects of different types of novelty on LTM consolidation and memory-related protein synthesis using the BT phenomenon. Radial Arm maze was used as the learning task for rodents (male Wistar rats), while OF and EE were two types of novel experiences provided to the rodents. Our results indicated that EE exposure efficiently leads to LTM consolidation through the BT phenomenon. In addition, EE exposure significantly enhances Post Synaptic Density Protein (PSD-95) synthesis in the hippocampus region of the rat brain. However, the OF exposure did not lead to significant PSD-95 expression. Further, our results did find alterations in PSD-95 expression after EE and OF exposure in the hippocampus in the at 15 mins after novelty. Hence, it is concluded that different types of novelty mediate the BT phenomenon up to the same extent at the behavioural level. However, the implications of different novelties may differ at molecular levels.

Methods: A study involving 15 adult male Wistar rats (180-220g) divided into three groups—Control, BT(OF) (Behavioural Tagging with Open Field), and BT(EE) (Behavioural Tagging with Environmental Enrichment)—aimed to assess memory consolidation. All groups underwent a Radial arm Maze (RAM) task for three days, with habituation, training, and testing. Control rats received no novelty exposure, while BT(OF) explored an open field, and BT(EE) experienced an enriched environment post-RAM training. After testing, hippocampal tissue was analysed for protein expression via Western blotting. Data analysis was done using ANOVA and Mann-Whitney U tests.

Results: It was observed that the exposure to after 15 mins of EE novelty led to long term memory formation and synthesis of memory related protein (PSD-95) after a single exposure.

Conclusion: Different forms of novelty do produce different behavioural outputs; however, differences are reflected in the neurobehavior machinery ,5 min EE exposure as a novelty is sufficient for synthesizing and expressing LTM- and synaptic density-specific protein PSD-95 in the hippocampus region. EE as a novelty exposure follows the specific trajectory in the hippocampus region for facilitating the LTM consolidation through the BT phenomenon.

Key words: long-term memory; novel object recognition; novelty exposure; plasticity-related proteins

PP44: *Bacopa monnieri* extract (CDRI-08) ameliorated recognition memory in mice exposed to hypoxia, correlated with downregulation of NMDA receptor levels in the hippocampus

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The brain, the body's largest oxygen consumer, depends on oxygen to support normal physiological activity. It is highly sensitive to oxygen deprivation and responds to oxidative stress and impairment of cognitive function. The body's oxygen-sensing system activates gene expression to protect brain tissues from hypoxia. Hypoxia is a significant stressor, stabilized hypoxia-inducible factor-1α (HIF-1α) regulating the expression of numerous genes leads to various biochemical, molecular, physiological, and genomic changes. Herbal medicines have been widely used for managing various toxicological effects and disorders, including hypoxia; however, the data on safety, efficacy, and molecular mechanism that increases vulnerability / lethality against hypoxia is still lacking and urgently needed to be investigated. In the current study, we have shown that hypoxia-induced recognition memory turn-down is associated with a significant upregulation of NMDA receptor subunits NR2A/2B and trafficking proteins like PSD 95 in the hippocampus of CoCl2-treated (40 mg/kg BW) mice. *Bacopa monnieri* extract (CDRI-08) (200 mg/kg BW) treatment reversed the expression of NMDA receptor subunits and their scaffolding/trafficking proteins, with varying effects depending on whether CDRI-08 was administered as a pre-treatment or co-treatment with CoCl2. Additionally, the expression of numerous genes, including EPO, GLUT-1, and VEGF, was significantly restored to levels similar to those in control mice compared to co-treatment. Together these, the current findings may provide mechanisms of hypoxia-induced deformity in the recognition memory, neurodegeneration, and neuroprotective action of CDRI-08 and provide a base for its therapeutic use in the recovery of hypoxia-led memory impairment.

PP45: *Toxoplasma gondii* infection induces neurodegeneration through altered host gene expression of α-synuclein network

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Neurodegeneration is a pathological condition that includes the process of losing structure and functions of neuronal cells resulting in functional defects in central nervous system. Among various factors in neurodegenerative instigations, pathogenic infections and misfolded protein aggregations are major risk factors for causing neurodegenerative disorders including Alzheimer's and Parkinson's disease. α-synuclein is a presynaptic neuronal protein involved in many functions majorly synaptic plasticity, neurotransmitter release, transcriptional regulation and immunological responses. α-synuclein can change its conformation under different types of stress and dysregulation of α-synuclein leads to neuroinflammation, neurodegeneration due to synuclein aggregations which transform in to development of prion like synucleinopathies. Dysfunctions in neurotransmission may cause neural associated disorders including Alzheimer's, Parkinson's, and Huntington's disease etc. *Toxoplasma gondii* is an apicomplexan protozoan parasite causes toxoplasmosis with serious neuropsychiatric symptoms and altered neurotransmission in infected hosts. In infectious diseases, it is assumed that α-synuclein plays an important role in pathogenesis of infection-mediated neurodegeneration. However, the role of α-synuclein in altering various host genes involved in neurodegeneration during *T. gondii* infection has not been elucidated. This study majorly focused on the expression analysis of α-synuclein related multi targeted genes including *GCH1, PRKN2, LRRK2, ANXA2, FUBP3, LGALS1, PRSS12, PPARG, MAOA, TLR2, SIRT1, STAT3, IFN-γ,* and *IL-1β* during *T. gondii* infection. We have observed altered expression in the genes related to α-synuclein. The mechanism, and expression/aggregation patterns of α-synuclein during *T. gondii* infection is under investigation. This study may unravel cues to understand the effect of *T. gondii* infection in altering various cellular mechanisms leading to neurodegeneration.

Keywords: *Toxoplasma gondii,* α-synuclein, Neurodegeneration, Alzheimer's disease, Parkinson's disease.

PP46: Female Chronic social defeat stress (fCSDS): A novel model for investigating female depression

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ABSTRACT

The study introduces a novel rodent model of female Chronic Social Defeat Stress (fCSDS) to better understand the biological, behavioral, and molecular mechanisms of depression specific to females, an area often neglected in preclinical research dominated by male-focused studies. In this model, parous CD1 female mice, induced into aggression through prolonged cohabitation with castrated males, were used to repeatedly expose adult female C57BL/6J mice to aggressive encounters over a period of 10 days, mimicking chronic social defeat without male involvement. Behavioral assessments revealed notable stress-induced depressive symptoms, as evidenced by altered performance in the sucrose preference test, forced swim test, elevated plus maze, and social interaction test. On the molecular level, significant changes were observed in estrogen receptors (ESR1 and ESR2), histone modifications (H3K9me3 and H3K27ac), and synaptic proteins (SYP and PSD-95) across brain regions implicated in depressive disorders. Additionally, the study identified disruptions in glutamate signaling, with elevated glutamate levels and reduced EAAT1 in the caudate putamen, suggesting excitotoxicity, alongside increased serum cortisol levels, indicative of heightened stress responses. Label-free quantitative mass spectrometry of the nucleus accumbens revealed abnormalities in synaptogenesis and mitochondrial pathways, further supporting the link between stress and neuroplasticity changes in this model. Overall, the fCSDS model successfully captures essential characteristics of female depression, offering a valuable tool for exploring sex-specific pathways and identifying potential therapeutic targets for treating depression in women.

PP47: Mitophagy as a Therapeutic Target in Neurodegenerative diseases

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Neurodegenerative diseases are a group of disorders characterized by the progressive degeneration and loss of neurons, the fundamental units of the nervous system. These conditions gradually impair cognitive functions, affect memory, and lead to severe physical disabilities. Common examples include Alzheimer's disease, Parkinson's disease, Huntington's disease, and amyotrophic lateral sclerosis (ALS). The etiology of these disorders is complex, involving both environmental and genetic factors. Hallmarks of neurodegenerative diseases include abnormal protein accumulation, neuroinflammation, neuronal loss, protein misfolding, and mitochondrial dysfunction. Recent research has highlighted mitophagy, a selective form of autophagy that eliminates dysfunctional or damaged mitochondria, as a promising therapeutic target for neurodegenerative diseases. Impaired mitophagy has been closely linked to the pathogenesis of various neurodegenerative disorders. Maintaining a delicate balance between mitochondrial biogenesis and mitophagy is crucial for preserving energy homeostasis and neuronal health. Disruption of this balance can lead to the accumulation of damaged mitochondria, increased oxidative stress, and ultimately, neuronal death. Several therapeutic strategies have been explored to enhance mitophagy for neuroprotection. These include lifestyle interventions such as regular exercise, stress management, and the administration of natural or synthetic pharmacological compounds. Emerging research focuses on developing targeted approaches to modulate mitophagy pathways, potentially offering new avenues for the treatment and prevention of neurodegenerative diseases.

Keywords
Neurodegenerative diseases, Neuronal degeneration, Alzheimer's disease, Parkinson's disease, Huntington's disease, Amyotrophic lateral sclerosis (ALS), Protein accumulation, Neuroinflammation, Protein misfolding, Mitochondrial dysfunction, Mitophagy, Parkin dependent, PINK1/Parkin-dependent mitophagy, PINK1/Parkin-independent mitophagy

PP48: Molecular Mechanisms For Targeted Autism Spectrum Disorder Treatment

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This poster presents an overview of the molecular mechanisms underlying Autism Spectrum Disorder (ASD) and potential targeted treatment strategies. ASDs are characterized by a heterogeneous array of symptoms, including deficits in social interaction and communication, often accompanied by other clinical challenges such as intellectual disability and epilepsy. Recent advancements in genetic research have identified various ASD-risk genes and their associated pathways, revealing common molecular disruptions in neurogenesis, synaptogenesis, and protein homeostasis. This review emphasizes the importance of understanding critical developmental windows for effective therapeutic interventions and suggests stratifying ASDs into distinct molecular subgroups to facilitate personalized treatment approaches. Notably, the exploration of chromatin remodeling, synaptic plasticity, and signaling pathways offers promising avenues for targeted therapies. The findings underscore the necessity for continued research into the interplay between genetic and environmental factors during crucial developmental periods, paving the way for innovative treatments that could significantly improve outcomes for individuals with ASD.

KEYWORDS: Autism Spectrum Disorder, molecular mechanisms, neurogenesis, synaptic plasticity, developmental windows, personalized treatment.

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