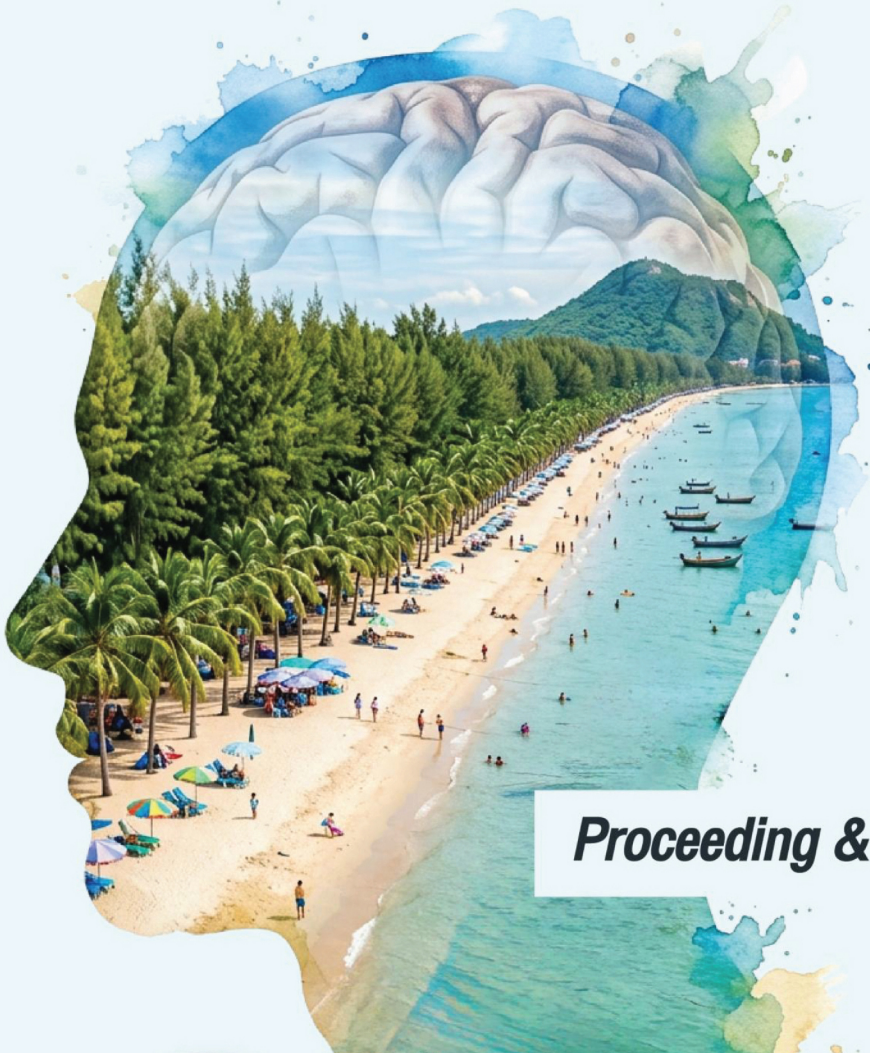




Proceeding & Abstracts

29th ANNUAL THAI

NEUROSCIENCE SOCIETY CONFERENCE
NEURODEVELOPMENT, AGING,
AND NEURODEGENERATIVE DISORDERS:
INNOVATION AND IMPLICATIONS

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THE 29TH ANNUAL THAI NEUROSCIENCE SOCIETY CONFERENCE

NEURODEVELOPMENT, AGING, AND
NEURODEGENERATIVE DISORDERS:
INNOVATION AND IMPLICATIONS

5-7 August, 2026 Amari Bang Saen, Chonburi, Thailand

PLENARY LECTURES



Prof. Pavich Tongroach
Past President of Thai Neuroscience Society



Prof. Anan Srikiatkachorn
Faculty of Medicine, King Mongkut's
Institute of Technology Ladkrabang, Thailand



Prof. Hitoshi Okamoto
RIKEN Brain Science Institute, Japan



Prof. Woong Sun
College of Medicine,
Korea University, South Korea



Prof. Alen Shih-Kuo Chen
College of Life Science,
National Taiwan University, Taiwan

PROF MASAO ITO'S LECTURE



Prof. Ingrid Liu
Institute of Medical Sciences,
Tzu Chi University, Taiwan



Prof. Steve Leu
Institute for Translation Research
in Biomedicine, Kaohsiung Chang Gung
Memorial Hospital, Taiwan



Asst. Prof. Tewarit Sarachana
Faculty of Allied Health Science,
Chulalongkorn University, Thailand



Assoc. Prof. Siriporn Chammiansawat
Faculty of Allied Health Sciences,
Burapha University, Thailand



Assoc. Prof. Amornpan Ajijmaporn
College of Sports Science and Technology,
Mahidol University, Thailand



Asst. Prof. Peeraporn Varintra
Institute of Medical Sciences,
Tzu Chi University, Hualien, Taiwan

SCIENTIFIC SESSION I : NEUROLOGICAL MODELS

SCIENTIFIC SESSION II : ATMPs & DRUG DISCOVERY

PROFESSOR DR ROONGTAM
LADPLI'S LECTURE



Prof. Siriporn Chattipakorn
Faculty of Medicine and Faculty of Dentistry,
Chiang Mai University, Thailand



Prof. Jintanaporn Wattanathorn
Faculty of Medicine,
Khon Kaen University, Thailand



Asst. Prof. Jacques Pak Kan Ip
School of Biomedical Sciences,
The Chinese University of Hong Kong, Hongkong



Asst. Prof. Jiraporn Panmanee
Institute of Molecular Biosciences,
Mahidol University, Thailand



Asst. Prof. Sukonthar Ngampramuan
Institute of Molecular Biosciences,
Mahidol University, Thailand



Dr. Suangsuda Supasai
Faculty of Tropical Medicine,
Mahidol University, Thailand

PROFESSOR DR PRASOP
RATANAKORN'S LECTURE



Prof. Jee Hyun KIM
Institute for Mental and Physical Health and
Clinical Translation, School of Medicine,
Deakin University, Australia

SCIENTIFIC SESSION III :
IBRO-TNS TRAVEL AWARD



Awardee 1: Irma Wati Ngadimon, University Malaysia, Malaysia

Awardee 2: Varsha Pai V, Manipal Academy of Higher Education, India

Awardee 3: Muhammad Tayyab Imtiaz, Monash University Malaysia, Malaysia

Awardee 5: Muhana Fawwazy Ilyas, Universitas Gadjah Mada, Indonesia

Awardee 4: Mingqing GU, The University of Hong Kong, China

Awardee 6: Jasmeet Singh Bindra, Indian Institute of Technology, India

SCIENTIFIC SESSION IV:
TNS COMMITTEE-SELECTED SPEAKERS



Prof. Somporn Kantharadussadee Triamchaisri
Faculty of Health Science, UCL Vietnam



Assoc. Prof. Chalerd Pichitpornchai
Faculty of Medicine Siriraj Hospital, Mahidol University, Thailand

SCIENTIFIC SESSION V: NEUROTECHNOLOGY



Asst. Prof. Sirawaj Itthipuripat
King Mongkut University of Technology Thonburi, Thailand



Dr. Sumeth Klomchitchareon
Institute of Molecular Biosciences, Mahidol University, Thailand

Important Dates



Abstract submission Deadline 30 June 2026
Early Bird Registration Deadline 30 June 2026
Regular Registration Deadline 4 August 2026

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Proceedings & Abstracts

29th Annual Thai Neuroscience Society Conference

TNS29

*Neurodevelopment, Aging, and Neurodegeneration:
Innovation and Implications*

AUGUST 5-7, 2026

Chonburi, Thailand

Venue	Amari Bangsaen Hotel, Chonburi, Thailand
Organized by	Thai Neuroscience Society (TNS) and Institute of Molecular Biosciences, Mahidol University
Hosted by	Institute of Molecular Biosciences, Mahidol University
Supported by	International Brain Research Organization Asia Pacific Regional Committee (IBRO-APRC) Mahidol University Thai Neuroscience Society

Editor
Scientific and Reviewing Committee TNS29

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29th Annual Conference of Thai Neuroscience Society (TNS29)

Program

Wed 5 AUGUST 2026 (DAY 1)				
	Time		Main Room (Pacific 1-2)	Workshop
DAY 1	08:30	-	Registration (Pacific Foyer) at Amari Bang Saen, Chonburi	
	09:30	10:00	Refreshment (Pacific Foyer)	
	10:00	12:00	TNS Neuro Quiz 2026 Final Competition Assoc. Prof. Chailerd Pichitpornchai, M.D., Ph.D. Mahidol University, Thailand Assoc. Prof. Akkradate Siriphorn, Ph.D. Chulalongkorn University, Thailand Asst. Prof. Prateep Amonruttanapun, Ph.D. Thammasat University, Thailand	
	12:00	13:00	Lunch (Pacific 3-4/Amaya)	
	Opening Ceremony			
	13:00	13:10	Opening remarks Prof. Supin Chompoopong, Ph.D. President of Thai Neuroscience Society Assoc. Prof. M.L. Saovaros Svasti, Ph.D. Director of Institute of Molecular Biosciences, Mahidol University	
	13:10	13:20	TNS NEURO QUIZ 2026 Award Ceremony	
	13:20	13:30	Group Photography	
	Plenary Lectures			Workshop
	13:30	14:05	PL-1: Reimagining Thai Education: From Systemic Challenges to Sustainable Transformation through the Neuroscience of Learning Prof. Pavich Tongroach, Ph.D. Past President of Thai Neuroscience Society	14:00-17:00 at Andaman 1-2 SKT workshop: The Elderly Memory and Health Healing (Thai language) Prof. Somporn Kantharadussadee Triamchaisri, Ph.D.
	14:05	14:40	PL-2: Medication Overused Headache – Possible Pathogenesis Prof. Anan Srikiatkhachorn, M.D. Faculty of Medicine, King Mongkut's Institute of Technology Ladkrabang	
	14:40	14:55	Refreshment (Pacific Foyer)	
	14:55	15:30	PL-3: Advancing Neural Organoid Technology for Better Understanding of CNS Development and Disease Modeling Prof. Woong Sun, Ph.D. College of Medicine, Korea University, South Korea	14:00-17:00 at Andaman 3 Innovative Learning Workshop Assoc. Prof. Chailerd Pichitpornchai, M.D., Ph.D., Asst. Prof. Pirom Chenprakhon, Ph.D., Supan Yodyingyong, Ph.D.
	15:30	16:05	PL-4: From Blue Light Photoreceptor ipRGCs to Behaviors: Computational Circuits for the Circadian Clock and Light Avoidance Prof. Alen Shih-Kuo Chen, Ph.D. College of Life Sciences, National Taiwan University, Taiwan	
	16:05	16:40	PL-5: The parallel pathways from the habenula to the interpeduncular nucleus control social conflict Prof. Hitoshi Okamoto, M.D., Ph.D. RIKEN Brain Science Institute, Japan	
	16:40	17:00	Poster setup	
			End of Day 1	



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29th Annual Conference of Thai Neuroscience Society (TNS29)

Thu 6 AUGUST 2026 (DAY 2)		
	Time	Main Room (Pacific 1-2)
DAY 2	Prof Masao Ito's Lecture	
	08:45	09:15
	HL1: Reversing Central Nervous System Dysfunction through Herbal Medicine and Systems Neuroscience Research Prof. Ingrid Liu, Ph.D. Institute of Medical Sciences, Tzu Chi University, Taiwan	
	Scientific Session I: Neurological Models	
	09:15	09:45
	SS1: The Emerging Role of Pnn in Neurodegenerative Diseases: From RNA Homeostasis to Neuronal Dysfunction Prof. Steve Leu, Ph.D. Institute for Translation Research in Biomedicine, Kaohsiung Chang Gung Memorial Hospital, Taiwan	
	09:45	10:15
	SS2: From the Blood to the Brain: Development of Human Brain Organoid-on-Chip from Peripheral Blood Mononuclear Cells of Thai Children Asst. Prof. Tewarit Sarachana, Ph.D. Faculty of Allied Health Sciences, Chulalongkorn University, Thailand	
	10:15	10:45
	Refreshment + Poster Presentation I (Pacific Foyer)	
	10:45	11:15
	SS3: Targeting Neuroinflammation and Synaptic Dysfunction with Bioactive Fraction from <i>Celastrus paricularus</i> Assoc. Prof. Siriporn Chamniansawat, Ph.D. Faculty of Allied Health Sciences, Burapha University, Thailand	
	11:15	11:45
	SS4: How Does Sport Shape the Brain? Neural Signatures of Visual Processing in Elite Gymnasts, Soccer Players, and Esport Athletes Assoc. Prof. Amornpan Ajjimaporn, Ph.D. College of Sports Science and Technology, Mahidol University, Thailand	
	11:45	12:15
	SS5: Natural Product-Based Intervention for Sleep Loss-Induced Cognitive Decline: Mechanistic Insights Into TCU411 (Adjunct) Asst. Prof. Peeraporn Varinthra, Ph.D. Institute of Medical Sciences, Tzu Chi University, Hualien, Taiwan	
	12:15	13:00
	Lunch (Pacific 3-4)/Amaya	
	13:00	13:30
	Poster Presentation II (Pacific Foyer)	
	Professor Dr Roongtam Ladpli's Lecture	
	13:30	14:00
	HL2: Broken Heart, Clouded Mind: Unmasking Cognitive Impairment in the Heart-Brain Axis Prof. Siriporn Chattipakorn, D.D.S., Ph.D. Faculty of Medicine, Chiang Mai University, and Faculty of Dentistry, Chiang Mai University, Thailand	
	Scientific Session II: ATPs & Drug Discovery	
	14:00	14:30
	SS6: Anthocyanins-Enriched Products, A Potential Candidate for Preventing and Mitigating Age-Related Neuropsychological Disorders Prof. Jintanaporn Wattanathorn, Ph.D. Faculty of Medicine, Khon Kaen University, Thailand	
	14:30	15:00
	SS7: Circular RNAs drive synaptic plasticity to promote learning and memory Asst. Prof. Jacque Pak Kan Ip, Ph.D. Chinese University of Hong Kong	
	15:00	15:30
	Refreshment + Poster Presentation III (Pacific Foyer)	
	15:30	16:00
	SS8: Structure-Based Drug Design: Exploring Small Molecules for Neurodegenerative Diseases Asst. Prof. Jiraporn Panmanee, Ph.D. Institute of Molecular Biosciences, Mahidol University, Thailand	



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Thu 6 AUGUST 2026 (DAY 2)

	Time		Main Room (Pacific 1-2)
	16:00	16:30	SS9: Bioengineered Exosomes for Neurorepair After Ischemic Stroke: Bridging Drug Discovery and Regenerative Medicine Asst. Prof. Sukonthar Ngampramuan, Ph.D. Institute of Molecular Biosciences, Mahidol University, Thailand
	16:30	17:00	SS10: Rebuild the Alzheimer-like brain: HMGB1 Box A gene therapy promotes neurogenesis and cognitive recovery Suangveda Supasai, Ph.D. Faculty of Tropical Medicine, Mahidol University, Thailand
			End of Day 2

Fri 7 AUGUST 2026 (DAY 3)

	Time		Main Room (Pacific 1-2)
DAY 3			Professor Dr Prasop Ratanakorn's Lecture
	08:45	09:15	HL3: Transcriptomics to Drive Drug Discovery in Psychiatry Prof. Jee Hyun KIM, Ph.D. Institute for Mental and Physical Health and Clinical Translation, School of Medicine, Deakin University, Australia
			Honorary Member Recognition
	09:15	09:20	Prof. Disya Ratanakorn, M.D. Chair of Professor Dr Prasop Ratanakorn Foundation
			Travel Grants Awarding
	09:20	09:30	Professor Dr Prasop Ratanakorn Travel Awards Mj. Gen. Chesda Udommongkol, M.D. Treasurer of Professor Dr Prasop Ratanakorn Foundation
			IBRO-TNS Travel Awards Prof. Supin Chompoopong, Ph.D. President of Thai Neuroscience Society
			Scientific Session III: IBRO-TNS Travel Award
	09:30	09:45	SS11: Neuroinflammation as a Converging Mechanism Linking Traumatic Brain Injury, Epileptogenesis, and Cognitive Decline Irma Wati Ngadimon, Ph.D. University Malaya, Malaysia
	09:45	10:00	SS12: Three-Dimensional Neural Tissue Models Elucidate Cellular and Molecular Mechanisms of Brain Aging Varsha Pai V Manipal Academy of Higher Education, India
	10:00	10:15	SS13: The Involvement of Oxidative Stress in Habenula Neurons in Social Stress Susceptibility in Adult Male Nile Tilapia Muhammad Tayyab Imtiaz Monash University Malaysia, Malaysia
	10:15	10:30	SS14: Artificial Intelligence for Nerve Conduction Studies: A Scoping review of Clinical Classification, Physiologic Modeling, and reporting Support Muhana Fawwazy Ilyas Universitas Gadjah Mada, Indonesia
	10:30	10:45	Refreshment (Pacific Foyer)



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29th Annual Conference of Thai Neuroscience Society (TNS29)

Fri 7 AUGUST 2026 (DAY 3)		
Time		Main Room (Pacific 1-2)
10:45	11:00	SS15: A Multi-Modal Framework for Point-of-Care Neurophysiological Monitoring and Foundation Model-Based Screening for Age-related Neurological Decline Jasmeet Singh Bindra Indian Institute of Technology, India
11:00	11:15	SS16: Deferiprone and Ferulic Acids Ameliorate Alcohol-Induced Cognitive Impairment by Modulating Iron Metabolism and Inhibiting Ferroptosis Minqing GU University of Hong Kong, China
11:15	11:45	IBRO Communication Prof. Sukumal Chongthammakun, Ph.D.
11:45	12:45	Lunch (Pacific 3-4/Amaya)
Scientific Session IV: TNS committee-selected speakers		
12:45	13:15	SS17: SKT Meditation Healing Exercise to Improve Health, Quality of Life and Healing Across the Life Span Prof. Somporn Kantharadussadee Triamchaisri, Ph.D. Faculty of Health Science, UCL, Vietnam
13:15	13:45	SS18: The Future Brain: How Artificial Intelligence is Reshaping Neuroscience in the Next Decade Assoc. Prof. Chailerd Pichitpornchai, M.D., Ph.D. Faculty of Medicine Siriraj Hospital, Mahidol University, Thailand
Scientific Session V: Neurotechnology		
13:45	14:15	SS19: A Model-Based EEG Framework for Investigating Attention Deficits in Mild Cognitive Impairment Asst. Prof. Sirawaj Itthipuripat, Ph.D. Learning Institute, King Mongkut University of Technology Thonburi, Thailand
14:15	14:45	SS20: Toward Translational NeuroAI & Neuroengineering for Human Restoration Sumeth Klomchitcharoen, Ph.D. Institute of Molecular Biosciences, Mahidol University, Thailand
14:45	15:00	Refreshment (Pacific Foyer)
TNS communication and CLOSING REMARKS		
15:00	15:45	Announcement of Award winners - Prof. Dr. Prasop Ratanakorn Foundation Award for Outstanding Research Achievement - Professor Roongtarn Ladpli Award - Associate Professor Naiphinich Kotchabhakdi Award - Professor Ingrid Y Liu Award - Professor Pavich Tongroach Award - Mr. Tay Khoon Yang Award (Hi-Tech Imaging Co. Ltd.) - Selected Abstract Award TNS Communication - TNS30 announcement Closing Address by Prof. Banthit Chetsawang, Ph.D.
		End of TNS29

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Welcome Remarks from President of Thai Neuroscience Society

Professor Supin Chompoonpong, Ph.D.

*President of Thai Neuroscience Society
Faculty of Medicine Siriraj Hospital,
Mahidol University, Thailand*

Distinguished Speakers, Attendees, and Esteemed Guests of the TNS29 International Conference

On behalf of the Thai Neuroscience Society (TNS) and the Conference Committee, it's my great pleasure to welcome you to the 29th Thai Neuroscience Society International Conference (TNS29) that is co-hosted by the Institute of Molecular Bioscience, Mahidol University. This collaborative event is also supported by IBRO Society Meeting Support 2026 for encouraging Asia Pacific Regional Country (APRC)'s activities.

TNS29 is held at the Amari Bang Saen, Chonburi, Thailand and scheduled for August 5-7, 2026. The theme of this year's meeting, "Neurodevelopment, Aging, and Neurodegenerative Disorders: Innovation and Implications," reflects our shared commitment to fostering progress in neuroscience across the APRC region.

Under this theme, **TNS29** designed to bring together neuroscience professionals by providing a vibrant platform for collaboration and idea exchange, the conference aims to inspire innovation in both basic and applied research. Over the course of three days, attendees will engage in a dynamic program featuring plenary lectures and honorary lectures by leading experts, scientific sessions including the early career researchers (ECR) covering a wide range of topics, poster presentations, award competitions & opportunities and special activity events.

In addition, **TNS29** would like to share the basis of neuroscience knowledge to the interested groups with our TNS special activities: 1) TNS Neuro Quiz 2026, which is the third year of competition (high school level) and 2) Innovative learning workshop that engages young minds in neuroscience, 3) SKT workshop that encourages interested participants to learn, practice health healing for promoting the elderly memory.

Beyond the scientific exchange, attendees will have the chance to experience the local flavors of Thai cuisine with unforgettable experience. Thank you for joining us, we look forward to your participation in making TNS29 a resounding success.



Welcome Message from Director of Institute of Molecular Biosciences, Mahidol University

**Associate Professor M.L. Saovaros
Svasti, Ph.D.**

*Director of Institute of Molecular Biosciences
Mahidol University, Thailand*

Welcome to the 29th Annual Thai Neuroscience Society Conference (TNS29).

On behalf of the Institute of Molecular Biosciences, Mahidol University, it is my great honor and pleasure to welcome all participants to the **29th Thai Neuroscience Society International Conference (TNS29)**.

The Institute of Molecular Biosciences is proud to co-host this conference with the Thai Neuroscience Society. Under the theme, **“Neurodevelopment, Aging, and Neurodegeneration: Innovation and Implications,”** TNS29 brings together distinguished scientists, clinicians, educators, and students from Thailand and around the world to share knowledge, exchange ideas, and strengthen collaborations that advance neuroscience research.

Scientific excellence is built through collaboration. The diverse participation of national and international experts at TNS29 reflects our shared commitment to addressing complex challenges in neuroscience through interdisciplinary research, innovation, and the open exchange of ideas. By fostering enduring partnerships across institutions and countries, we can build sustainable scientific networks that accelerate discovery, facilitate translation into improved human health, and cultivate the next generation of neuroscientists.

I hope this conference will inspire productive discussions, new collaborations, and lasting partnerships that extend well beyond this meeting and contribute to meaningful scientific and societal impact.

I would also like to express my sincere appreciation to the **Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University**, together with the organizing committee, speakers, authors, reviewers, sponsors, and all participants for their dedication and contributions in making TNS29 a success.

I wish you an inspiring, productive, and enjoyable conference.



Welcome Message from Co-Chair of Organizing Committee & Chair of Scientific committee

Professor Banthit Chetsawang, Ph.D.

*Co-Chair of Organizing Committee & Chair of Scientific
committee TNS29
Institute of Molecular Biosciences
Mahidol University, Thailand*

Welcome to the 29th Annual Thai Neuroscience Society Conference (TNS29).

On the occasion of the 29th Thai Neuroscience Society Annual Conference (TNS29), jointly organized by the **Thai Neuroscience Society (TNS)** and the **Institute of Molecular Biosciences, Mahidol University (MB-MU)**, from August 5-7, 2026 at Amari Bang Saen, Chonburi, Thailand.

As the representative of **MB-MU**, I would like to extend a warm welcome to all distinguished speakers and participants. This conference provides an excellent opportunity for scholars, students, and neuroscience research enthusiasts to attend keynote lectures by renowned international researchers from across the globe. Attendees will also have the chance to present their research findings through oral and poster presentations spanning various subfields of neuroscience, as well as exchange academic and research ideas.

Furthermore, this event serves as a valuable platform for **networking and building academic and research collaborations in neuroscience**. Thailand is the land of smiles. I sincerely hope that all participants will enjoy a memorable experience, marked by the warmth and hospitality of the Thai people. I also hope you will explore Thailand's rich culture and diverse soft power, including Thai cuisine and traditional attire.

We look forward to welcoming you all again on future occasions.

TNS29 Conference Information and Organizing Committee

TNS29 Conference Information

In 2026, the Thai Neuroscience Society, in collaboration with the Research Center for Neuroscience and the Institute of Molecular Biosciences, Mahidol University, will host the **29th Annual Thai Neuroscience Society Conference 2026 (TNS29)** under the theme “**Neurodevelopment, Aging, and Neurodegeneration: Innovation and Implication.**”

The **29th Annual Thai Neuroscience Society Conference 2026**, to be held on **August 5–7, 2026, at the Amari Bangsaen Hotel, Chonburi Province**, will serve as a premier platform for presenting the latest advances in neuroscience research and innovation. The conference will provide opportunities for in-depth scientific discussions, inspire new ideas through keynote and special lectures delivered by distinguished experts from Thailand and abroad, and foster collaborations and professional networks among researchers, clinicians, educators, and students in the neuroscience community.

We warmly invite neuroscientists, healthcare professionals, researchers, faculty members, postdoctoral fellows, graduate and undergraduate students, and all individuals interested in brain science to join **TNS29**. Your participation will contribute to the exchange of knowledge, the development of new collaborations, and the advancement of neuroscience research and innovation for the benefit of society. We look forward to welcoming you to Chonburi for this exciting scientific gathering.

Overview

Field: Neuroscience and Brain Research

Date: August 5–7, 2026

Venue: Amari Bangsaen Hotel, Chonburi Province

Theme: Neurodevelopment, Aging, and Neurodegeneration: Innovation and Implication

Official Language: English

Hosted by: Thai Neuroscience Society in collaboration with Institute of Molecular Biosciences, Mahidol University and the Research Center for Neuroscience, Mahidol University.

Committee Advisors

1. Professor Dr. Pavich Tongroach
2. Professor Emeritus Dr. Piyarat Govitrapong
3. Professor Emeritus Dr. Sukumal Chongthammakun
4. Associate Professor Dr. Kanokwan Tilokskulchai
5. Associate Professor Dr. M.L. Saovaros Svasti

TNS29 Organizing Committee

Professor Dr. Banthit Chetsawang	Chair of the organizing committee
Professor Dr. Supin Chompoonpong	Co-Chair of the organizing committee
Assistant Professor Dr. Narisorn Kitiyanant	Vice-Chair of the organizing committee
Professor Dr. Sutisa Thanoi	Committee Member
Professor Dr. Somporn Kantaradusdi Triamchaisri	Committee Member
Associate Professor Dr. Kulthida Chaithirayanon	Committee Member
Associate Professor Dr. Chairat Turbpaiboon	Committee Member
Associate Professor Dr. Chailerd Pichitpornchai	Committee Member
Associate Professor Dr. Narawut Pakaprot	Committee Member
Associate Professor Dr. Vorasith Siripornpanich	Committee Member
Associate Professor Dr. Akkradate Siriphorn	Committee Member
Assistant Professor Dr. Jiraporn Panmanee	Committee Member
Assistant Professor Dr. Prateep Amonruttanapun	Committee Member
Assistant Professor Dr. Manussabhorn Phatsara	Committee Member
Assistant Professor Dr. Adisorn Ratanayotha	Committee Member
Assistant Professor Dr. Amornpun Sereemasapun	Committee Member
Dr. Utcharaporn Kamsrijai	Committee Member
Miss Natthanit Srimasern	Committee Member
Miss Chonnicha Subkod	Committee Member
Associate Professor Dr. Sujira Mukda	Committee Member & Secretary
Mrs. Somsong Phengsukdaeng	Committee Member & Assistant Secretary
Miss Kaewklao Banjong	Committee Member & Assistant Secretary

Committee from Thai Neuroscience Society (TNS)

TNS Advisory committee

Pavich Thongroj
Kanokwan Tiloksakulchai
Piyarat Govitrapong
Sukumal Chongthammakun

TNS committee (2025-2027)

Supin Chompoonpong	President
Chailerd Pichitpornchai	Vice-President & Special Activity Committee
Sutisa Thanoi	Vice-President
Akkradate Siriporn	Scientific committee
Chairat Turbpaiboon	Deputy Scientific committee
Narawut Pakaprot	Committee & Hospitality
Adisorn Ratanayotha	Registrar
Sujira Mukda	Secretary General
Utcharaporn Kamsrijai	Deputy Secretary General
Banthit Chetsawang	Central Committee
Somporn Kantharadussadee Triamchaisri	Central Committee



Mahidol University
Institute of
Molecular Biosciences



Proceedings and Abstracts

August 5-7, 2026

29th Annual Thai Neuroscience Society Conference

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Proceedings and Abstracts

August 5-7, 2026

29th Annual Thai Neuroscience Society Conference

SPECIAL PROGRAMS

TNS Neuro Quiz 2026

Overview:

The **Thai Neuroscience Society Neuro Quiz 2026 (TNQ 2026)** aims to honor high-achieving students by providing them with the opportunity to participate in an exciting national and international neuroscience competition. The next generation of leaders in neuroscience will be encouraged through dynamic and interactive learning.

Participants will engage in a series of intellectually stimulating quiz rounds designed to promote active recall, critical thinking, and the application of knowledge. Scenario-based questions will challenge students to connect theoretical concepts with real-world contexts, enhancing both understanding and problem-solving skills.

Two progressive rounds will be organized:

- 1. Elimination Round Competition (Online):** The top 10 finalist teams will be listed.
- 2. International Final Competition (Onsite):** One winning team will be selected.

Each round increases in difficulty, offering a rewarding and challenging experience throughout the competition.

The top 10 finalist teams will be awarded sponsored one-night accommodation to compete in the International Finals, held onsite during the TNS29 Conference at **Amari Bangsaen, Chonburi, Thailand, on August 5, 2026**. The accommodation will be provided for one night on August 4, 2026.

This is a unique opportunity to showcase your knowledge, gain valuable experience, and connect with peers who share a passion for neuroscience.

Join us and be part of the future of neuroscience.

TNS Neuro Quiz 2026 – Program & Registration Details

Timeline:

Description	Date
First announcement details	16-19 April, 2026
Registration period	20 April - 20 May, 2026
Announcement of eligible teams	25 May, 2026
Elimination Round (Online) – Live Kahoot & MCQs Bell-Ringer Exam	6 June, 2026 (1.00 pm - 3.00 pm, GMT+7, Thailand Time)
Announcement of the Top 10 teams advancing to the Finals	13 June, 2026
International Final Round (Onsite) - TNS29 conference at Amari Bangsaen, Chonburi, Thailand	5 August, 2026 (10.00 am - 12.00 pm, GMT+7, Thailand Time)

Eligibility:

1. Open to all public, private, and parochial high schools in Thailand and international schools.
2. Participants must be students currently enrolled in Grades 10-12.
3. Each team must include one officially appointed coach or teacher, assigned by the school's principal or director.
4. Each team must consist of two primary student members.
5. Each school is allowed to register an unlimited number of teams.

Judging and Integrity:

All responses will be evaluated by a panel of experts in neuroscience to ensure fairness and academic rigor. Any form of dishonesty or plagiarism will result in immediate disqualification.

Registration:

1. Each team must consist of a coach (teacher) and two student members from the same school. If the coach is not a faculty member of the school, official documentation from the school is required to verify the team's eligibility for participation.
2. **Link for registration:** <https://forms.gle/u7soHrHTkEioctu59> or Scan the QR code below.
3. The participation fee is 500 Thai Baht (THB) per team, payable to Siam Commercial Bank PCL (SCB): **Thai Neuroscience Society, Account No. 016-2-95537-9**, Bank address: 2 Phran Nok Road, Siriraj Sub District, Bangkok Noi District, Bangkok 10700, Thailand.



Scan to register

Code of Conduct:

Participants must demonstrate excellent sportsmanship, showing respect to judges, organizers, and fellow competitors. Any use of inappropriate language or disruptive behavior will result in a warning or disqualification.

General Structure of the Quiz Format:

The competition will be conducted in two rounds:

1. **Elimination Round** – Conducted online through a Zoom meeting: **Live Kahoot platform** and **MCQs bell-ringer exam** to identify the top 10 finalist teams based on their highest combined scores.
2. **International Finals** – Conducted onsite at TNS29 conference at Amari Bangsaen, Chonburi, Thailand, on August 5, 2026, to select the winning team.

Elimination Round (online):

Rules

1. All registered participants are required to join the Zoom meeting using their unique team ID, followed by their full registered name (i.e., TNQ-01-CU-Full Name). The unique team ID will be communicated to the registered email address.
2. Participants must join within the scheduled time. Late arrivals (over 5 minutes) will be disqualified.
3. Zoom meeting recording will monitor participant behaviors for any suspicious activities.
4. Participants are required to remain in a well-lit environment, ensuring that no other individuals are visible within the camera frame. **Both webcam and microphone** must remain active for the duration of the competition.
5. No external assistance is allowed. No other person may participate or help in any form.
6. In the event of connection issues (e.g., disconnection, lag, frozen screen, or disruptions caused by phone or LINE calls), no extra time, pause, or compensation will be provided under any circumstances.

Regulations

1. Participants will be evaluated through **Live Kahoot** and **the MCQs bell-ringer exam** (conducted online via Google Form) in the Elimination Round to identify the top 10 finalist teams.
2. Each team member may complete the **individual examination independently** or **collaboratively with their teammates**; however, all responses must be submitted individually. The team's final score will be determined by combining all submitted answers.
3. All participants are required to use two devices: **(1) a PC, Mac, or iPad** to join the Zoom meeting (with camera and microphone enabled) and view the MCQ bell-ringer questions, and **(2) a second device, such as a mobile phone or iPad**, to submit answers in real time via Kahoot and Google Forms.
4. Participants using mobile phones are strongly advised to use a Wi-Fi connection (by turning on airplane mode and turning off the voice calls of the LINE application and other communication applications) instead of mobile data to prevent disruptions caused by incoming phone or LINE calls.
5. Please verify that your internet speed is sufficient.
6. The video conference will be held via Zoom meeting; ensure that the Zoom application is installed on your device beforehand.
7. The TNQ 2026 organizers will provide the quiz links (Live Kahoot and MCQ answer form) during the Zoom session on the test date.

8. The example of the recommended textbook for the TNS Neuro Quiz will be provided as a guideline, but its content may not directly correspond to the examination questions.
9. The team's ranking will be determined by the combined scores of all team members. The highest total team score will determine the top 10 finalist teams.
10. The official results of the Elimination Round will be communicated via email and published on the official website. Participants are expected to regularly check their email for updates and announcements.

International Final Round (onsite):

Rules

1. The team's composition must only include an accompanying teacher (coach) and two students of each team from the same school as registered.
2. Participants must join within the scheduled time. Late arrivals (over 5 minutes) will be disqualified.
3. In the event of connection issues (e.g., disconnection, lag, frozen screen, or disruptions caused by phone or LINE calls), no extra time, pause, or compensation will be provided under any circumstances.
4. No external assistance is allowed. No other person may participate or help in any form except for students from the same team.

Regulations

1. All participants are required to use **only one tablet per team** to submit answers via the MCQ Google Form and the live Kahoot platform.
2. Participants will be assessed through two types of questions onsite (MCQs and short-answer questions), administered via Google Forms and Kahoot during the TNS29 Conference at Amari Bangsaen, Chonburi, Thailand, on August 5, 2026.
3. Participants are allowed to use only the paper and pencil provided on their team's table to take notes. No additional materials or personal stationery are permitted.
4. The use of calculators, mobile phones, or any communication devices is strictly prohibited during the competition.
5. Any handwritten notes used during the competition must be submitted as instructed. Participants are not allowed to retain them after the competition concludes.
6. Team rankings will be determined based on the combined scores from Google Forms and Kahoot, with accuracy as the scoring metric.
7. No points will be deducted for incorrect answers.
8. The highest total score will determine the winners.
9. The official results of the International Final Round will be announced at the TNS Neuro Quiz 2026 Award Ceremony in the TNS29 conference on August 5, 2026, at Amari Bangsaen, Chonburi, Thailand.

Awards, Prizes, Gifts, and Certificates:

1. All certificates will be provided as digital e-certificates.

2. All participants will receive a Certificate of Participation.
3. In the final round, both files and printed copies will be provided for participants.
4. The top 10 teams advancing to the International Final Round who confirm their participation in the final round by the announced deadline will receive Certificates of Finalist and vouchers covering one-day event registration for the TNS29 conference (August 5, 2026). Further details regarding accommodation will be announced before final round confirmation.

TNS Neuro Quiz 2026 Committee of Competition

Assoc Prof Chailerd Pichitpornchai, MD, PhD	Faculty of Medicine Siriraj Hospital, Mahidol University, Thailand
Assoc Prof Akkradate Siriphorn, PhD	Faculty of Allied Health Sciences, Chulalongkorn University, Thailand
Assist Prof Prateep Amonruttanapun, PhD	Chulabhorn International College of Medicine, Thammasat University, Thailand
Sukrit Promtang, PT, MSc	Faculty of Allied Health Sciences, Pathumthani University, Thailand
Korlab Kobkoy, PT, BSc	The Siam Commercial Bank Public Company Limited, Thailand

PRESENTS

TNS NEURO QUIZ 2026

WHERE MINDS COMPETE & CHAMPIONS ARE MADE!

TEAM 1

TEAM 2

FROM
126
TEAMS
(83 SCHOOLS)

— QUALIFYING TO —
TOP 10 TEAMS

FINDING THE WINNER!

2ND PLACE

CERTIFICATE
+
CASH PRIZE 5,000 THB

WINNER

CASH PRIZE
25,000 THB
+ TNS TROPHY

3RD PLACE

CERTIFICATE
+
CASH PRIZE 5,000 THB

CHALLENGE YOUR BRAIN
Test your knowledge and think smart!

TEAMWORK MAKES SUCCESS
2 students per team
Stronger together!

COMPETE FOR THE BEST
Top 10 teams
move to the final round!

AMAZING PRIZES
Cash prizes & TNS Trophy!

83 SCHOOLS PARTICIPATED

- ANUKOOLNAREE SCHOOL
- Aranyaprathet School
- Assumption College Thonburi School
- Banglamung School
- Benjamacharutit School
- Bodindecha (Sing Singhassen) School
- Buriwitwittayalai School
- Burirampittayakhom School
- Chiangmai Tzu Chi School
- Chitralada School
- Chomthong School
- Chongfah Sinseung Wanich Bamrung School
- Chonkanayanukul School
- Churmpaeasaksa School
- Darussalam School
- Dattani School
- Demonstration School of Khon Kaen University, Secondary School (Education)
- Hatyai Wittayalai Sombonkulkanya School
- Hatyaiwittayalai School
- International Community School
- Islamic Science Demonstrations School, PSU
- Joseph Upatham School
- Kamnoetvidya Science Academy
- Kamphaeng School
- Kanchanapisit Wittayalai Nakhon Pathom School
- Kasetsart University Laboratory School, Kamphaeng Saen Campus, Educational Research and Development School
- Laem Sattigattana School
- Lukhaninwarinratchanarap School
- Mahidol University International Demonstration School
- Montfort College School
- Nakprasit School
- Naresuan University Demonstration School
- Nawamintrachinuthit Horwang Northaburi School
- Nawamintrachinuthit Suankulab Wittayalai Pathumthani School
- Panparat High School
- Pattumwan Demonstration School
- Petpittayakom School
- PHUKHIEO SCHOOL
- PINU WITTAYANUSORN SCHOOL
- Potsampittayakom School
- Prince of Songkla University Demonstration School (Secondary)
- Princess Chulabhorn Science High School Buriram
- Princess Chulabhorn Science High School Chonburi
- Princess Chulabhorn Science High School Loei
- Princess Chulabhorn Science High School Nakhon Si Thammarat
- Princess Chulabhorn Science High School Pathumthani
- Princess Chulabhorn Science High School Phetchaburi
- Princess Chulabhorn Science High School Satun
- Princess Sirindhorn's College
- Princess Ubolratana Rajakanya's College
- Phattthalung School
- Prommanusorn Phetchaburi School
- PSU Wittayanusorn School
- Roietwittayalai School
- Royal English Programme Banchang School
- Sa-nguan Ying School
- Saint Joseph Bangna School
- Saint Joseph Mueng-Nake School
- Samutsakhon Wittayalai School
- Satriwitthaya School
- Selaphum Pittayakom School
- Singapore International School of Bangkok (SISB Pracha Uthit)
- Singanur School
- Sirindhorn School
- Sirinatharadol University Prasarnmit Demonstration School (Secondary)
- Streemattarakom School
- Suankulab Wittayalai Rangsit School
- Suankulab Wittayalai School
- Suansirivittayakom School
- Sukasani School
- Sukarni Wittaya School
- Surawittayakom School
- The Newton Sixth Form School
- Triamudomsuksanomklao School
- Triamudomsuksanpattanakorn Nonthaburi School
- Triyit International School
- Udomakrun School
- Udonpittayakom School
- Wat Kaphang Surin School
- Wat Naumool School
- Wat Phraai Mahadhat Secondary Demonstration School, Phranakhon Rajabhat University
- Yothinburana School

ONE QUIZ. ONE CHAMPION. COUNTLESS FUTURES.





★ Congratulations to the ★

TOP 10

FINALIST TEAMS

— of TNS Neuro Quiz 2026 —

These 10 teams have demonstrated exceptional knowledge,
teamwork, and passion for neuroscience.

 12-TRI Trinity International School Coach: Rachen Singhakumar Students: Kwachuen Kongchim, Hyunsuk Roh	 46-NT3 The Newton Sixth Form School Coach: Yanisa Wassanatip Students: Mena Chitnukroh, Marisa Ise
 16-DSK Demonstration School of Khon Kaen University, Secondary School (Education) Coach: Panadda Wongwichitranon Students: Kongpipop Chan, Natchanon Sakhong	 60-KVI3 Kamnoetvidya Science Academy Coach: Tanawan Leeboonngam Students: Nada Ratchakitpipat, Paricha Liewluck
 19-PNS1 Princess Chulabhorn Science High School Nakhon Si Thammarat Coach: Nanticha Taweerat Students: Radanattakun Changsan, Chayada Preechatiwong	 74-SIB3 Singapore International School of Bangkok (SISB Pracha Uthit) Coach: Frans Brewis Students: Panawit (O'Neal) Maneeratanadamkirng, Tunchanok (Mint) Nontiwantok
 34-PM1 Wat Phrasri Mahadhat Secondary Demonstration School, Phranakhon Rajabhat University Coach: Suphasikant Lobbumrung Students: Nutt Phatkulthanut, Thanawint Ratchpuddee	 85-SW1 Satriwithaya School Coach: - Students: Wirulnut Iemsinworakul, Panpatchanan Yimluean
 44-YB Yothinburana School Coach: Yutthasak Sawangsamutchai Students: Pimboon Permpituk, Warunrak Junhasobhaga	 109-YS2 Hatyai Wittayalai Somboonkulkanya School Coach: - Students: Polpipat Phumkuea, Pornraiw Satholsumritphol



Well done to all our finalists!

We look forward to seeing you shine at the TNS Neuro Quiz 2026.





Announcement: Top 10 Finalist Teams – TNS Neuro Quiz 2026

Following the TNS Neuro Quiz 2026 Elimination Round held on June 6, 2026, with a total of 126 participating teams from 83 schools, we are pleased to officially announce the **Top 10 Finalist Teams**.

The TNS Neuro Quiz 2026 Organizing Committee is delighted to announce the Top 10 Finalist Teams that have qualified for the **International Final Round (Onsite)**, which will be held at the **Amari Bangsaen Hotel, Chonburi, Thailand, on August 5, 2026**. The qualified teams are listed by Team ID as follows:

Team ID	Participant
12-TRI	Trinity International School <i>Coach: Rachen Singhakumar</i> <i>Student: Kwachuen Kongchim, Hyunsuk Roh</i>
16-DSK	Demonstration School of Khon Kaen University, Secondary School (Education) <i>Coach: Panadda Wongwichitranon</i> <i>Student: Kongpiphop Chan, Natchanon Sakhong</i>
19-PNS1	Princess Chulabhorn Science High School Nakhon Si Thammarat <i>Coach: Nanticha Taweerat</i> <i>Student: Radanattakun Changsan, Chayada Preechatiwong</i>
34-PM1	Wat Phrasri Mahadhat Secondary Demonstration School, Phranakhon Rajabhat University <i>Coach: Suphasikant Lobbumrung</i> <i>Student: Nutt Phatkulthanut, Thanawint Ratchpuddee</i>
44-YB	Yothinburana School <i>Coach: Yutthasak Sawangsamutchai</i> <i>Student: Pimboon Permpituk, Warunrak Junhasobhaga</i>
46-NT3	The Newton Sixth Form School <i>Coach: Yanisa Wassanatip</i> <i>Student: Mena Chitnukroh, Marisa Ise</i>

TNS Neuro Quiz 2026

A part of the 29th Thai Neuroscience Society (TNS29) Annual Conference at Amari Bangsaen Hotel, Chonburi, Thailand



60-KV13	Kamnoetvidya Science Academy <i>Coach:</i> Tanawan Leeboonngam <i>Student:</i> Nada Ratchakitpipat, Paricha Liewluck
74-SIB3	Singapore International School of Bangkok (SISB Pracha Uthit) <i>Coach:</i> Frans Brewis <i>Student:</i> Panawit (O'Neal) Maneeratanadamkirng, Tunchanok (Mint) Nontiwantok
85-SW1	Satriwithaya School <i>Coach:</i> - <i>Student:</i> Wirulnut lemsinworakul, Panpatchanan Yimluean
109-YS2	Hatyai Wittayalai Somboonkulkanya School <i>Coach:</i> - <i>Student:</i> Polpipat Phumkuea, Pomraiwn Satholsumritphol

We sincerely congratulate all advancing teams on their outstanding performance and commend all participants for their enthusiasm, dedication, and passion for neuroscience.

The Thai Neuroscience Society (TNS) will provide partial support for each **Top 10 Finalist Team (one coach and two students)** to participate in the International Final Round Competition (Onsite) during the [TNS29 Conference](#) at the Amari Bangsaen Hotel, Chonburi, Thailand, on August 5, 2026.

Support for Top 10 Finalist Teams

1. Accommodation Reimbursement: Reimbursement will be provided upon submission of a **valid hotel payment receipt** issued in the name of the school, the coach, or a student. The maximum reimbursement amount is **2,000 THB per team**. Teams may arrange their own accommodation at Amari Bangsaen Hotel or any nearby hotel for the night of August 4, 2026. Reimbursement will be provided either in cash or QR code payment at the conference registration desk.

2. Registration Fee Waiver: The one-day registration fee for all Top 10 Finalist Teams will be fully waived on August 5, 2026. No additional registration is required.



International Final Round Tentative Program

August 5, 2026 (Wednesday, GMT+7)

08:30 – 10:00 Registration at the Ballroom, Amari Bangsaen Hotel, Chonburi, Thailand

10:00 – 12:00 International Final Round Competition (supported by the TNS Neuro Quiz 2026 and UFicon teams)

12:00 – 13:30 Lunch Break

13:30 – 13:40 Opening Remarks

13:40 – 13:50 TNS Neuro Quiz 2026 Award Ceremony

14:00 – 17:00 IL-MU Workshop for TNS Neuro Quiz Participants (supported by the Institute for Innovative Learning, Mahidol University)

Important Notice

Participation in the International Final Round (Onsite) requires the attendance of **both the coach and the two student participants** from each finalist team. Teams that fail to meet this requirement may be disqualified from the competition. **Please confirm your team's participation in the onsite final round with the Organizing Committee by June 15, 2026.** If any of the Top 10 Finalist Teams are unable to participate, the vacant position will be offered to the next highest-ranked team.



Assoc.Prof.Dr. Akkradate Siriphorn
Committee of the TNS Neuro Quiz 2026



Assoc.Prof.Dr. Chailerd Pichitpornchai
Chair of the TNS Neuro Quiz 2026



Asst.Prof.Dr. Prateep Amonruttanapun
Committee of the TNS Neuro Quiz 2026

SKT Workshop: The Elderly Memory and Health Healing

SKT Workshop is an intensive 3-hours learning sessions focused on applying specific SKT techniques to improve elderly memory and health problems.

Focus: Intensive learning and practical skill development Duration: Approximately 3 hours

Goal: SKT for elderly health enhancement and health problem healing in NCDs
Techniques: Training in SKT 1, 3, 7, and 8

Organization: The founder of SKT Meditation Healing Exercises 1-8, Prof. Dr. Somporn Kantharadussadee Triamchaisri and Thai Neuroscience Society (TNS): Prof. Dr. Supin Chompoopong and Assoc. Prof. Dr. Chailerd Pichitpornchai

Main Considerations:

SKT: The SKT (Short Cognitive Performance Test) is described as a tool used to assess cognitive impairment, specifically memory and attention, particularly in patients with dementia. It is also used to evaluate the effectiveness of therapies in improving these cognitive functions. However, the description mentions training in specific "SKT techniques" (1, 3, 7, 8) for memory and health improvement, suggesting the workshops may apply methods related to this test or perhaps a system of exercises called SKT Meditation Healing Exercises. Further information about the specific content of "SKT Meditation Healing Exercises 1-8" would be beneficial to understand the full scope of the workshops.

Scientific Basis: The workshops stimulate the exchange of scientific knowledge, suggesting an evidence-based approach to the training provided.

Focus: The explicit aim is to enhance elderly memory and health, indicating specialized techniques and practices that fit to a specific purpose to the group with Noncommunicable diseases; NCDs.

In conclusion, the SKT Workshops appear to be designed to provide focused, practical training in specific SKT-related techniques with the goal of improving the cognitive health of older adults, potentially through exercises and strategies aimed at enhancing memory and addressing health-related issues with self-healing, up skills and re skills.

Activities:

- S1. Introduction and Health
- S2. Mechanism and Safety of SKT Meditation Healing Exercises
- S3. Practicing SKT 1: Fit and firm brain cells/memory, body/mind, cardiovascular system and brain potential
- S4. Self-Management for Noncommunicable diseases (NCDs) S5. Discussion & Evaluation

Advisory of workshops

Professor Dr. Supin Chompoopong, TNS President 2025-2027

Emeritus Prof Dr. Sukumal Chongthammakun, TNS President 2023-2025



Mahidol University
Institute of
Molecular Biosciences



Proceedings and Abstracts

29th Annual Thai Neuroscience Society Conference

August 5-7, 2026

Speakers & Trainers of workshops

Professor Dr. Somporn Kantharadussadee Triamchaisri (Dr. SKT)

Assoc. Prof. Dr. Chailerd Pichitpornchai, MD

Dr. Ruchee Pornchai

Dr. Chanati Juntarachotichatchawal

Ajarn Thanaphon Sutsinsunthon

Ajarn Nuanchawee Netsangthip

Registration

All participants can register by completing the online registration form. Registration will close on July 29, 2026, 16.00 GMT+7. Participating in the workshop fee is 1,000 THB. Any more questions please send an email to meditationskt@gmail.com.

Who can attend?

Participants are health provider, researchers, students, patients, health volunteers from different backgrounds.

Languages

Thai language in August 5, 2026 (01:30 pm-16:30 pm)

SKT Workshop:

สมาธิ SKT เพื่อสร้างเสริมคุณภาพชีวิตผู้สูงอายุ
และเยียวยาโรคไม่ติดต่อเรื้อรังทุกกลุ่มวัยTNS29: The 29th Annual Thai Neuroscience Society Conference

การประชุมวิชาการนานาชาติของสมาคมประสาทวิทยาประเทศไทย ครั้งที่ 29

โรงแรม อมารี บางแสน จังหวัดชลบุรี วันที่ 5 สิงหาคม พ.ศ. 2569 เวลา 13.30 - 16:30 น.

S1. บทนำ และ การตระหนักรู้ด้านสุขภาพ (30 นาที)

S2. กลไกการทำงาน และ ความปลอดภัยในการฝึก SKT (30 นาที)

S3. นำฝึกปฏิบัติสมาธิบำบัด SKT (ภาคปฏิบัติหลัก) (60 นาที)

S4. การประยุกต์ใช้เพื่อจัดการโรคไม่ติดต่อเรื้อรังด้วยตนเอง (30 นาที)

S5. การประเมินผล และ สรุปปิดการอบรม (30 นาที)

SPEAKERS & TRAINERS

ศาสตราจารย์ ดร.สมพร กันทรดุษฎี เตรียมชัยศรี (Dr.SKT)

รองศาสตราจารย์ ดร. นพ.ชัยเลิศ พิชิตพรชัย

ดร.รุจิ พรชัย

ดร.ชนติ จันทโรทัย

อาจารย์ ธนากร สดสีสุนทร

อาจารย์ นวลฉวี เนตรแสงทิพย์

ที่ปรึกษากิจกรรม

ศาสตราจารย์ ดร.พันโทหญิงสุทิน ชมภูวงศ์
TNS President 2025-2027ศาสตราจารย์เกียรติคุณ ดร.สุมาล จงธรรมคุณ
TNS Former President

คณะกรรมการ

ผู้ช่วยศาสตราจารย์ ดร. นพ.อมรพันธุ์ เสริมพันธุ์

อาจารย์ ดร.ประทีป อมรัตน์พันธ์

อาจารย์ ดร.อรรถกร คำศรีใจ

การลงทะเบียน

ผู้เข้าร่วมทุกท่านต้องลงทะเบียนโดยการกรอกแบบฟอร์มลงทะเบียนออนไลน์.

การลงทะเบียนจะปิดรับ วันที่ 29 ก.ค. 2569 เวลา 16.00 น. GMT+7.

ค่าลงทะเบียน workshop ท่านละ 1,000 บาท.

สอบถามเพิ่มเติมได้ทางอีเมลล์ meditationskt@gmail.com



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TRAVEL GRANT AWARDEES

IBRO-APRC Travel Grant Awardees for TNS29

Travel Grant Awardees for TNS29



สมาคมประสาทวิทยาศาสตร์ไทย (Thai Neuroscience Society)

สำนักงานสมาคม: คณะวิทยาศาสตร์ มหาวิทยาลัยมหิดล, 272 ถนนพระราม 6 เขตราชเทวี กรุงเทพมหานคร 10400

TNS Office: Faculty of Science, Mahidol University, 272 Rama VI Road, Ratchathewi, Bangkok 10400

Website: www.thaineuroscience.org

IBRO-APRC Travel Grant Awardees for TNS29

The Scientific Committee of the **29th International Conference of Thai Neuroscience Society (TNS29)** is pleased to announce the recipients of the **IBRO-APRC Travel Grant**. These early-career researchers have been selected to present their work at our upcoming conference, “Neurodevelopment, Aging, and Neurodegenerative Disorders: Innovation and Implications,” held from August 5–7, 2026, at the Amari Bangsaen Hotel, Chon Buri, Thailand.

Each awardee will receive a travel grant (up to **USD 600**) and a **waived registration fee**.

List of Awardees

No.	Full Name	Institution	Abstract Title
1	Irma Wati Ngadimon	Universiti Malaya, Malaysia	Neuroinflammation as a Converging Mechanism Linking Traumatic Brain Injury, Epileptogenesis, and Cognitive Decline
2	Varsha Pai V	Manipal Academy of Higher Education, India	Three-Dimensional Neural Tissue Models Elucidate Cellular and Molecular Mechanisms of Brain Aging
3	Muhammad Tayyab Imtiaz	Monash University Malaysia	The Involvement of Oxidative Stress in Habenula Neurons in Social Stress Susceptibility in Adult Male Nile Tilapia
4	Minqing GU	The University of Hong Kong	Deferiprone and Ferulic Acids Ameliorate Alcohol-Induced Cognitive Impairment by Modulating Iron Metabolism and Inhibiting Ferroptosis
5	Muhana Fawwazy Ilyas	Universitas Gadjah Mada, Indonesia	Artificial Intelligence for Nerve Conduction Studies: A Scoping Review of Clinical Classification, Physiologic Modeling, and Reporting Support
6	Jasmeet Singh Bindra	Indian Institute of Technology Mandi, India	CereTron: A Multi-Modal Framework for Point-of-Care Neurophysiological Monitoring and Foundation Model-Based Screening for Age-Related Neurological Decline

Announced by:

TNS Scientific Committee

March 31, 2026



สมาคมประสาทวิทยาศาสตร์ไทย (Thai Neuroscience Society)

สำนักงานสมาคม: คณะวิทยาศาสตร์ มหาวิทยาลัยมหิดล, 272 ถนนพระราม 6 เขตราชเทวี กรุงเทพมหานคร 10400

TNS Office: Faculty of Science, Mahidol University, 272 Rama VI Road, Ratchathewi, Bangkok 10400

Website: www.thaianeuroscience.org

Official Announcement: Travel Grant Awardees

The 29th International Conference of the Thai Neuroscience Society (TNS29)

The Thai Neuroscience Society (TNS), in collaboration with the **Professor Dr. Prasop Ratanakorn Foundation**, is pleased to announce the official list of travel grant recipients for the upcoming **TNS29 Conference**, “*Neurodevelopment, Aging, and Neurodegenerative Disorders: Innovation and Implications*,” held at the Amari Bangsaen Hotel, Chon Buri, Thailand, from August 5–7, 2026.

This joint grant aims to support bright academic minds by providing **6,000 THB per applicant** (allocated as 4,000 THB for travel expenses and 2,000 THB for the TNS29 registration fee).

List of Awardees

Based on the eligibility criteria, the scientific committee has finalized the selection of recipients. Please review the official list of awardees below:

No.	First Name	Last Name	Academic Status / Career Stage	University / Research Institution
1	Wongsakorn	Siripan	Doctoral Candidate	Mahidol University
2	Pornkanok	Nimnoi	Doctoral Candidate	Mahidol University
3	Supatcha	Khosem	Graduate Student	Burapha University
4	Fahsai	Thaion	Graduate Student	Mahidol University
5	Sompong	Sansenya	Post-Doctoral / Lecturer	Praboromarajchanok Institute
6	Kotchapit	Maksri	Graduate Student	Mahidol University
7	Danuyada	Wattanaumadechakul	Post-Doctoral / Lecturer	Praboromarajchanok Institute
8	Ratanart	Permpikul	Graduate Student	Mahidol University

**สมาคมประสาทวิทยาศาสตร์ไทย (Thai Neuroscience Society)**

สำนักงานสมาคม: คณะวิทยาศาสตร์ มหาวิทยาลัยมหิดล, 272 ถนนพระราม 6 เขตราชเทวี กรุงเทพมหานคร 10400

TNS Office: Faculty of Science, Mahidol University, 272 Rama VI Road, Ratchathewi, Bangkok 10400

Website: www.thaineuroscience.org**Important Information for Awardees****Note to Recipients:**

- 1. TNS Membership Mandatory:** All grant recipients must be active members of the Thai Neuroscience Society. If you are not yet a member, please proceed with your **TNS membership registration** immediately.
- 2. Confirmation Deadline:** Awardees must reply to the official notification email to **confirm your acceptance (Confirmation) by June 15, 2026.**

If you have any immediate questions, please contact the organizing committee.

- **Contact Email:** thaineuroscience.sc@gmail.com

Congratulations to all the awardees! We look forward to seeing your research and presentations in Chon Buri this August.

Announced by:

The TNS Scientific Committee

In coordination with the Professor Dr. Prasop Ratanakorn Foundation

June 2026



Mahidol University
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AWARD & HONORARY LECTURE

Honorary Membership

Prof Masao Ito's Lecture

Professor Dr Prasop Ratanakorn's Lecture

Professor Dr Roongtam Ladpli's Lecture



2026 TNS Honorary Membership

Prof. Disya Ratanakorn, M.D.

*Professor of Neurology
President of the Professor Prasop Ratanakorn
Foundation*

It is with great pleasure that the Thai Neuroscience Society (TNS) announces the appointment of **Professor Dr. Disya Ratanakorn**, Professor of Neurology, as an **Honorary Member** of our esteemed society.

Professor Dr. Disya Ratanakorn is a distinguished neurologist and an expert in cerebrovascular diseases and neurosonology, including carotid duplex ultrasonography and transcranial Doppler studies. She is currently affiliated with the Department of Medicine, Faculty of Medicine, Ramathibodi Hospital, Mahidol University.

Her remarkable career spans academic, clinical, and leadership excellence. She formerly served as President of the Thai Stroke Society, Executive Committee Member of the World Stroke Organization, and Chairperson of the Neurological Research Foundation under the Royal Patronage. Currently, she serves as **Vice Chairperson of the Neurological Research Foundation** and **President of the Professor Prasop Ratanakorn Foundation**.

Trained in neurosonology at the University of Bonn, Germany, and as a Fellow in Stroke and Neurosonology at Wake Forest University School of Medicine, USA, she has been an International Lecturer in Neurosonology at Wake Forest since 2000. Her scholarly and clinical contributions have significantly advanced diagnosis, management, and education in stroke medicine.

Professor Dr. Disya Ratanakorn has also been instrumental in developing national training curricula in stroke neurology and neurosonology, aligning them with international standards. Her commitment to public education on stroke prevention and to supporting young neuroscientists—notably through initiatives under the Professor Prasop Ratanakorn Foundation and the Professor Prasop Ratanakorn Endowment Fund. Under her leadership, the Professor Prasop Ratanakorn Foundation has continuously supported Thai students to attend IBRO Schools and has provided sustained financial support for Thai student participation in the Thai Neuroscience Society Annual Conference for four consecutive years, from TNS26 (2022) to TNS29 (2026). Through these initiatives, the Foundation has played a vital role in nurturing and inspiring the next generation of Thai neuroscientists.

Therefore, it is with great honor that we recognize Professor Dr. Disya Ratanakorn as an Honorary Member of the Thai Neuroscience Society, in appreciation of her lifelong dedication and outstanding contributions to neuroscience in Thailand.

Congratulations, Professor Dr. Disya Ratanakorn, on this well-deserved distinction.

Given on the 5th day of August 2026.



HL1: Prof Masao Ito's Lecture

Prof. Ingrid Liu, Ph.D.

*Institute of Medical Sciences, Tzu Chi University, Taiwan***Reversing Central Nervous System Dysfunction through Herbal Medicine and Systems Neuroscience Research**¹Institute of Medical Sciences, Tzu Chi University, Hualien, 970473, Taiwan²Center for Brain, Compassion, and Altruism Research and Education, Tzu Chi University, Hualien, 970473, Taiwan³Center for Herbal Medicine and Natural Product Research, Tzu Chi University, Hualien, 970473, Taiwan*Corresponding author. Email: ycliu@gms.tcu.edu.tw**Abstract:**

Central nervous system (CNS) disorders, including neurodevelopmental and neurodegenerative diseases, are characterized by complex disruptions in neuronal signaling, synaptic plasticity, and neuroimmune homeostasis. Current therapeutic approaches are largely symptomatic and do not effectively modify the underlying disease mechanisms. Integrating systems neuroscience with herbal medicine represents a promising strategy for elucidating disease mechanisms and developing multi-target therapeutic interventions that restore neural function. The objective of this study was to investigate the molecular and cellular mechanisms underlying CNS dysfunction and to evaluate the therapeutic efficacy of both pharmacological and herbal interventions in reversing behavioral and neuropathological deficits in animal models of autism spectrum disorder (ASD) and Huntington's disease (HD). Behavioral, molecular, and histological analyses were conducted using Cav3.2 knockout mice as a genetic model of ASD and 3-nitropropionic acid (3-NP)-treated mice as a model of HD. Behavioral assessments included tests of social interaction, repetitive behaviors, fear memory, motor coordination (rotarod), locomotor activity (open-field), and neurological function. Brain tissues were examined by immunofluorescence, Western blotting, Nissl staining, TUNEL assay, and reactive oxygen species detection. Clonazepam was administered to enhance GABAergic neurotransmission in the ASD model, whereas the natural phenolic compound coniferaldehyde (CFA) was evaluated for its neuroprotective effects in the HD model. Loss of Cav3.2 induced ASD-like phenotypes, including impaired social novelty recognition, increased repetitive grooming, and memory deficits, which were associated with reduced GABAergic signaling in the frontal cortex. Low-dose clonazepam significantly ameliorated these behavioral abnormalities. In the HD model, CFA improved motor function, preserved neuronal architecture, attenuated oxidative stress and apoptosis, restored PKM2-mediated metabolic homeostasis, and suppressed neuroinflammation through inhibition of the JAK2/STAT3 signaling pathway. Our findings demonstrate that integrating systems

neuroscience with herbal medicine facilitates the identification of disease-specific mechanisms and the development of mechanism-based therapeutic strategies. Modulation of GABAergic signaling and multi-target herbal interventions represent promising therapeutic approaches for restoring CNS function and improving functional outcomes across a broad spectrum of neurological disorders.

Keywords: Herbal medicine, Huntington's disease, Cav3.2, autism spectrum disorder

Dr. Ingrid Y. Liu is a Full Professor at the Institute of Medical Sciences, Faculty of Medicine, Tzu Chi University (TCU). Her research focuses on elucidating the molecular mechanisms underlying brain health and disease, with particular emphasis on how sleep, neuroprotection, and natural products influence neuronal function and resilience against neurodegenerative disorders. She has received numerous honors, including the Research Excellence Award in 2021, Honorary Fellow of Thailand Neuroscience Society in 2019 and the TCU Journal Article Award multiple years since 2011. Dr. Liu also serves as an editorial board member at *Tzu Chi Medical Journal* and a reviewer for several academic journals, such as *Frontiers in Molecular Neuroscience*, *PLOS ONE*, *Brain Research*, *Frontiers in Cellular Neuroscience*, *Neuroscience Letters*, *Marine Drugs*, and *BMC Medical Genetics*.



HL2: Professor Dr Roongtam Ladpli's Lecture

Prof. Siriporn Chattipakorn, D.D.S., Ph.D.

*Professor of Neuroscience and Oral Biology
Neurophysiology Unit,
Cardiac Electrophysiology Research and Training Center
(CERT),
Faculty of Medicine, Chiang Mai University, Chiang Mai,
and
Department of Oral Biology and Diagnostic Sciences,
Faculty of Dentistry, Chiang Mai University, Chiang Mai,
Thailand*

Broken Heart, Clouded Mind: Unmasking Cognitive Impairment in the Heart-Brain Axis

Abstract:

Myocardial infarction (MI) and subsequent heart failure (HF) are associated with cognitive impairment, but the underlying mechanisms remain unclear. Our study investigated the pathways linking MI-induced cardiac dysfunction to cognitive decline and assessed the efficacy of several therapeutic interventions. MI-induced oxidative stress disrupted the blood-brain barrier, promoting neuroinflammation and brain mitochondrial dysfunction characterized by excessive reactive oxygen species production, reduced ATP generation, impaired mitochondrial fusion and biogenesis, and suppressed oxidative phosphorylation. These changes led to hippocampal apoptosis, synaptic loss, and deficits in learning, memory, and synaptic plasticity. Cognitive impairment emerged during the cardiac remodeling phase and occurred in both HFpEF and HFrEF, independent of the severity of cardiac dysfunction. Metabolic analyses revealed a compensatory shift toward glycolytic and ketone metabolism in response to impaired mitochondrial function. Mitochondrial-targeted therapies (Mdivi-1 and M1) effectively restored mitochondrial function and improved cognitive performance, while the pan-caspase inhibitor Z-vad enhanced neurogenesis and synaptic integrity, showing robust cognitive benefits. In contrast, enalapril and dapagliflozin improved cardiac outcomes and reduced systemic stress but provided limited protection against brain mitochondrial dysfunction and cognitive decline. Overall, our findings identify mitochondrial dysfunction and apoptosis-mediated synaptic loss as key drivers of post-MI cognitive impairment. Targeted mitochondrial and anti-apoptotic therapies may offer greater neuroprotection than conventional HF treatments, highlighting the therapeutic importance of the heart-brain axis after MI.

Keywords: Brain; Cognition; Heart Failure; Myocardial Infarction; Mitochondria

Professor Dr. Siriporn Chattipakorn has received many national and international scientific awards including the “**Outstanding Scientist 2025**” award, “**Thailand Best Researcher Award in Biomedical Sciences from the National Research Council of Thailand 2017**”, “**TRF-CHE SCOPUS Researcher Award 2012**”, the Thailand Research Fund, “**TRF-OHEC Clarivate Analytics Research Excellence Award 2017**”, Thailand Research Fund, “**TRF Best Research Project of the Year 2014**” from the Thailand Research Fund, “**Outstanding Visiting Scholar Award 2001**” from the University of Alabama at Birmingham, “**American Heart Association Fellowship Award, Epilepsy Fellowship Award 2001**”, from American Epilepsy Society, the “**Gold Elephant Award for Best Researcher in Medical Science 2014**” from the Chiang Mai University. In 2022, she received “**The Dushdi Mala Medal**” in Medical Science” from the King of Thailand. She has been appointed as the **TRF Senior Research Scholar** in the year 2017 and 2020 from the National Research Council of Thailand. In 2023, she has also been appointed as the **Distinguished Research Professor** from the National Research Council of Thailand. She has continuously received several research fundings from the Thailand Research Fund and the National Research Council of Thailand. She is currently the author of **565 peer-reviewed papers** and abstracts, and **13 international/1 national textbooks** in neuroscience and medicine. She has served as the editorial board and reviewer board for many international neuroscience and endocrinology journals. Professor Dr. Siriporn Chattipakorn's research interest is in the field of neurophysiology and the effects of the metabolic syndrome on the brain. She has been studying the electrophysiological changes in the brain and cognitive function of subjects with metabolic syndrome, diabetes, thalassemia and chemobrain using a wide range of study models ranging from brain mitochondria and animal models to the bedside level for these pathophysiological studies.



HL3: Professor Dr Prasop Ratanakorn's Lecture

Prof. Jee Hyun KIM, Ph.D.

*Institute for Mental and Physical Health and Clinical
Translation, School of Medicine, Deakin University, Australia*

Transcriptomics to Drive Drug Discovery in Psychiatry

Abstract:

Psychiatric disorders' heterogeneous etiology and poorly understood biology pose significant challenges to typical drug development paradigms that rely on targeting single proteins. Novel approaches are needed to discover new treatment options. Drug repurposing is an efficient and effective option, and its strategy includes finding the gene expression signature of the approved medications in combination to discover the common transcriptomic factors that may explain the efficacy of diverse medications. Then an existing drug that produces similar transcriptomic changes can be identified. As a proof of concept, human NT2-N neuronal cell cultures were treated with a cocktail of first-line bipolar depression medications (lithium, valproate, lamotrigine, and quetiapine) and RNAseq was used to discover the gene expression signature that best describes the overall medication effects. 960 compounds from the Prestwick library were then screened with qPCR. A comprehensive literature review was also conducted focussing on bipolar depression biology. Literature review identified that a novel therapy to treat bipolar depression must address the inter-related effects of reduced mitochondrial function with increased oxidative stress and inflammation. With this information and the similarity score to the combined bipolar medications, trimetazidine was identified as the most promising candidate. It was confirmed in a chronic social isolation rat model that trimetazidine (30 mg/kg) injected once-daily for 2 weeks had an antidepressant-like effect. Trimetazidine promotes glucose oxidation by selectively inhibiting 3-ketoacyl-CoA thiolase, a key enzyme in fatty acid β -oxidation. Trimetazidine also increases pyruvate dehydrogenase activity to decrease lactate accumulation, its effects going beyond addressing mitochondrial dysfunction to reducing inflammation and oxidative stress. Trimetazidine has been used for decades to treat angina pectoris for its cytoprotective and metabolic effects. Trimetazidine is now being assessed as a treatment for bipolar depression in an international multi-site clinical trial (ANZCTR registration: ACTRN12622000474752). Trimetazidine is highly novel in psychiatry, has an excellent safety profile, and crosses the blood brain barrier. Therefore, positive outcomes of the trial can fast-track repurposing of trimetazidine to treat bipolar depression. Transcriptomics to target the biology of psychiatric illnesses is providing a new hope for people with live experience.

Professor Jee Hyun Kim holds a PhD in Psychology from the University of New South Wales, Australia, and currently serves as a Professor at Deakin University within the Faculty of Health, the School of Medicine, and the Institute for Mental and Physical Health and Clinical Translation. Based at the Geelong Warrn Ponds Campus, her diverse fields of research encompass biological psychology, neurosciences, pharmacology and



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pharmaceutical sciences, as well as cognitive and computational psychology. Additionally, Professor Kim is actively available for the supervision of Masters by Research and PhD students.



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PLENARY LECTURES



PL-1:

Prof. Pavich Tongroach, Ph.D.*Past President, Thai Neuroscience Society*

Reimagining Thai Education: From Systemic Challenges to Sustainable Transformation through the Neuroscience of Learning

Abstract:

Education has become the most strategic investment of every nation in the twenty-first century. In an era characterized by artificial intelligence (AI), exponential technological advancement, demographic transition, global competition, and unprecedented societal uncertainty, the prosperity of nations increasingly depends not on natural resources or industrial capacity but on the quality of their human capital. Consequently, educational transformation has become a national imperative worldwide.

Thailand has invested heavily in education for several decades. School enrolment has reached nearly universal levels, educational opportunities have expanded considerably, and public expenditure on education remains among the highest sectors of the national budget. Nevertheless, learning outcomes have remained below expectations. International assessments such as the Programme for International Student Assessment (PISA), together with indicators of innovation and national competitiveness, consistently suggest that improvements in educational access have not translated proportionately into improvements in educational quality.

Over the past four decades, Thailand has undertaken numerous educational reforms. These reforms have included curriculum revisions, decentralization initiatives, quality assurance systems, teacher development programmes, digital technology implementation, assessment reforms, and repeated restructuring of educational administration. While many of these initiatives were well intentioned and produced localized improvements, none has fundamentally transformed the educational system.

This paper argues that previous reforms have shared one common limitation: **they have largely been designed from administrative, organizational, or political perspectives rather than from the perspective of how the human brain actually learns.**

The central thesis of this paper is therefore straightforward: **Thailand has never truly designed its educational system on neuroscientific principles.**

This statement does not imply that educational policymakers have ignored science. Rather, it recognizes that the science of learning has advanced dramatically during the past three decades, while educational systems – including that of Thailand –

continue to reflect assumptions originating from nineteenth- and twentieth-century industrial models of schooling.

Modern neuroscience has fundamentally transformed our understanding of learning. Learning is now recognized as an active biological process involving neuroplasticity, attention, executive function, motivation, emotional regulation, memory consolidation, social interaction, and continuous adaptation. The brain is not a passive recipient of information but an active constructor of knowledge. Consequently, educational environments should be designed to optimize these biological processes rather than inadvertently hinder them.

This paper therefore proposes a paradigm shift – from viewing educational reform primarily as institutional or administrative restructuring toward understanding it as the design of an integrated learning ecosystem grounded in neuroscience. Such a framework has important implications for curriculum development, assessment, teacher education, school organization, educational governance, and national policy.

Ultimately, sustainable educational transformation begins not merely with new legislation, revised curricula, or advanced technologies, but with a deeper scientific understanding of the learner. The science of learning is, fundamentally, the science of the brain.

Keywords: Neuroscience of learning; Thai education; educational reform; neuroplasticity; STEM education; executive function; educational policy; sustainable transformation.

1. INTRODUCTION

Education in the Age of Intelligence

Education has always been central to civilization. Throughout history, societies have transmitted knowledge, values, skills, and culture from one generation to the next through various forms of education. However, never before has education become as strategically important as it is today.

Human civilization is entering an era fundamentally different from any previous period. Artificial intelligence is transforming the nature of work. Automation is replacing routine occupations. Scientific discoveries occur at unprecedented speed. Climate change, geopolitical uncertainty, demographic ageing, pandemics, and economic volatility increasingly characterize the global environment. These developments collectively require individuals not merely to possess knowledge, but to continuously acquire, integrate, and apply new knowledge throughout life.

The competitive advantage of nations is consequently undergoing profound transformation. During the industrial era, economic prosperity depended primarily upon natural resources, manufacturing capacity, inexpensive labour, and physical infrastructure. Today, however, sustainable national competitiveness increasingly depends upon creativity, innovation, adaptability, critical thinking, scientific literacy, collaborative problem-solving, and lifelong learning. Human intelligence has become the principal strategic resource.

Education is therefore no longer merely a public service. It has become the operating system of national development.

This transformation is reflected internationally. Countries consistently achieving high educational outcomes – including Finland, Singapore, Estonia, Canada, and several East Asian economies – have increasingly recognized that education must prepare learners

not only for existing occupations but also for future challenges that cannot yet be fully anticipated.

The Organisation for Economic Co-operation and Development (OECD), UNESCO, the World Economic Forum, and numerous international organizations have consequently shifted educational discourse away from content acquisition toward competencies, adaptability, resilience, creativity, collaboration, and lifelong learning.

Underlying all these competencies lies one fundamental biological reality:

Every form of learning occurs within the human brain.

This observation appears almost self-evident, yet educational systems have seldom been designed from this perspective. Much educational reform continues to focus primarily upon organizational structures, curricula, regulations, administrative arrangements, examinations, funding mechanisms, and technological infrastructure. While each of these components is undeniably important, they represent only the external architecture of education.

The true engine of learning remains the learner's brain.

Understanding how the brain learns should therefore constitute the scientific foundation upon which educational systems are designed.

As both a neuroscientist and an educator, I have observed these two disciplines evolving largely in parallel. Neuroscience has dramatically expanded our understanding of cognition, memory, emotion, attention, executive function, neuroplasticity, and learning mechanisms. Simultaneously, educational reform has proceeded through curriculum revisions, governance restructuring, technological implementation, quality assurance, and institutional change.

Remarkably, these two fields have interacted far less than one might expect. This paper argues that sustainable educational transformation requires bringing them together. Educational policy should increasingly become informed by neuroscience, just as modern medicine has become informed by molecular biology and evidence-based clinical science.

The objective is not to transform teachers into neuroscientists, nor schools into neuroscience laboratories. Rather, it is to ensure that educational environments align with the fundamental biological principles through which every human brain learns, remembers, thinks, creates, and adapts.

In other words, educational reform should become **brain-compatible**.

The following sections examine why Thailand's educational reforms have produced only limited success despite decades of effort, and propose a neuroscience-informed framework for sustainable transformation

2. THE THAI EDUCATIONAL PARADOX

Thailand's educational development over the past half-century presents a striking paradox. On virtually every conventional indicator of educational expansion, the country has made remarkable progress. Yet when educational success is measured by learning quality, innovation, and the capacity of graduates to thrive in an increasingly complex world, the outcomes have fallen considerably short of national aspirations.

This paradox deserves careful examination because understanding the nature of the problem is the prerequisite for designing effective solutions.

Expansion without Transformation

Since the promulgation of the National Education Act in 1999, successive governments have consistently regarded education as a national priority. Considerable public

resources have been devoted to expanding educational opportunity, improving school infrastructure, increasing teacher numbers, introducing digital technologies, decentralizing educational administration, and reforming curricula.

Today, Thailand enjoys almost universal access to basic education. Educational participation rates compare favourably with many countries at similar stages of economic development. Universities have expanded rapidly, while vocational education, lifelong learning programmes, and digital learning initiatives continue to grow.

From a quantitative perspective, these achievements are undeniable. However, educational expansion alone does not necessarily produce educational excellence. The distinction between **schooling** and **learning** has become increasingly apparent. Children may spend more years in classrooms without necessarily acquiring deeper understanding, stronger reasoning abilities, greater creativity, or improved capacity for lifelong learning. Educational access, although indispensable, represents only the beginning of educational quality.

Persistent Learning Outcomes

International assessments provide one perspective on this challenge. Over the past two decades, Thailand has participated regularly in the Programme for International Student Assessment (PISA), administered by the OECD. Although these assessments do not capture every dimension of education, they provide internationally comparable evidence regarding students' ability to apply knowledge and reasoning to unfamiliar situations.

The findings have been sobering. Thai students consistently perform below the OECD average in reading literacy, mathematical literacy, and scientific literacy. More importantly, relatively little improvement has occurred despite numerous policy initiatives, curriculum revisions, and educational reforms. The concern extends beyond international rankings. PISA does not merely assess factual knowledge; it evaluates students' capacity to analyse, interpret, reason, solve problems, and transfer learning to novel contexts. These competencies increasingly determine success within knowledge-based economies.

Consequently, the issue is not national prestige but national capability. A country whose educational system produces graduates unable to adapt rapidly to technological and societal change inevitably encounters limitations in innovation, productivity, and long-term competitiveness.

The Illusion of Reform

One reason this paradox has persisted is that Thailand has never lacked educational reform.

Indeed, educational reform has been almost continuous.

Every decade has introduced new initiatives:

- curriculum revisions,
- teacher development programmes,
- school quality assurance,
- educational decentralization,
- ICT integration,
- digital classrooms,
- competency-based education,
- STEM education,
- coding education,
- English language initiatives,
- active learning,
- outcome-based education,

- educational innovation funds,
 - learning recovery programmes,
- and numerous others.

Each initiative has addressed an important aspect of education. Many have demonstrated local success. Yet collectively they have failed to produce the systemic transformation that policymakers, educators, parents, and society have long expected. This observation should not be interpreted as criticism of any particular reform. Rather, it suggests that educational problems cannot be solved simply by accumulating additional reforms. A system may undergo continuous change while remaining fundamentally unchanged.

Symptoms Rather than Causes

Much educational debate has traditionally focused on visible symptoms.

- Teachers are overworked.
- Students memorize excessively.
- Curricula are overcrowded.
- Schools face administrative burdens.
- Assessment encourages examination-oriented learning.
- Educational inequality persists.
- Digital technologies remain unevenly distributed.

Each observation is correct. Yet these represent manifestations rather than root causes. Attempting to solve educational problems one symptom at a time resembles treating fever without identifying the underlying disease. True transformation requires understanding the architecture of the system itself.

Education as a Complex Adaptive System

Modern educational systems should not be viewed as collections of independent components.

- Curriculum influences assessment.
- Assessment influences classroom practice.
- Classroom practice influences teacher behaviour.
- Teacher preparation influences curriculum implementation.
- Educational leadership influences school culture.
- School culture influences student motivation.
- Student motivation influences learning outcomes.
- Learning outcomes subsequently influence policy decisions.

These interactions create continuous feedback loops. Education therefore functions as a **complex adaptive system**, in which individual components cannot be optimized independently. Changes introduced into one part of the system inevitably affect every other component. This systems perspective explains why isolated reforms often generate disappointing results. Improving curricula alone cannot substantially improve learning if assessment continues to reward memorization. Introducing digital technology produces limited impact if pedagogy remains unchanged. Teacher development programmes achieve only modest success when teachers continue to operate within rigid administrative structures. Similarly, revising legislation alone cannot transform learning unless classrooms themselves evolve. Educational reform therefore requires coherence across the entire ecosystem.

An Overlooked Question

Despite decades of reform, one remarkably simple question has rarely occupied the centre of educational policy discussions: **How does the human brain actually learn?** This question appears almost deceptively obvious. After all, education exists for one purpose – to facilitate learning.

Learning occurs exclusively within the human brain. One might therefore reasonably expect educational systems to have been designed primarily around the biological principles governing learning itself. Surprisingly, this has seldom been the case. Historically, educational systems evolved largely in response to social, economic, political, religious, and industrial needs rather than neuroscientific knowledge. Class schedules reflected factory organization. Age grouping reflected administrative convenience. Curriculum organization reflected disciplinary traditions. Assessment reflected efficiency of large-scale examination systems. Teacher-centred instruction reflected historical constraints on information access. Most of these structures originated long before neuroscience began revealing how attention, memory, motivation, executive function, emotion, and neuroplasticity interact during learning. Consequently, many educational practices that appear entirely normal today were never consciously designed to align with the biology of learning. They simply evolved historically.

Some remain highly effective. Others may inadvertently impede learning.

The Missing Scientific Foundation

This observation leads to what may be the central proposition of this paper.

Thailand has invested enormous effort in reforming educational institutions. It has invested comparatively little effort in redesigning education according to the scientific principles of learning itself.

In other words,

Thai education has never truly been designed on neuroscientific principles.

This statement should not be interpreted as criticism of educators or policymakers.

Indeed, much of contemporary neuroscience did not yet exist when many current educational structures were established.

Rather, it reflects an extraordinary opportunity.

For the first time in history, educational reform can be informed not only by philosophy, sociology, economics, psychology, and public policy, but also by the rapidly expanding science of the human brain.

The challenge before us is therefore no longer whether neuroscience should contribute to education.

The challenge is how educational systems can be systematically redesigned so that every learner benefits from what neuroscience has revealed about how human beings learn most effectively.

The next section examines this scientific foundation and asks a fundamental question:

If learning occurs in the brain, what should every educational reformer understand about how the brain learns?

3. BRAIN-FIRST EDUCATION: A NEUROSCIENCE-INFORMED FRAMEWORK

Rather than reviewing neuroscience comprehensively, I will present only the educationally relevant concepts.

The section will focus on six fundamental principles.

1. Neuroplasticity

Learning physically changes neural networks. Every meaningful educational experience leaves biological traces within the brain.

2. Executive Functions

Successful learners are characterized not merely by intelligence, but by planning, working memory, inhibitory control, cognitive flexibility, and self-regulation.

3. Emotion and Motivation

The brain learns best when curiosity, intrinsic motivation, and emotional safety coexist. Fear and chronic stress impair hippocampal function and long-term memory formation.

4. Active Construction of Knowledge

Learning is not information transmission. It is active construction through questioning, experimentation, retrieval, reflection, and application.

5. Social Learning

The human brain evolved socially. Discussion, collaboration, communication, and peer interaction are essential components of effective learning.

6. Lifelong Brain Plasticity

Learning does not end with graduation. Throughout life, the brain retains remarkable capacity for adaptation and continuous development.

These six principles collectively suggest that education should no longer be organized around teaching alone but around optimizing learning.

Brain-First Education is proposed as a guiding philosophy for twenty-first century educational transformation. It recognizes that education is fundamentally the science and practice of facilitating learning, and that learning is ultimately a biological process occurring within the human brain. Consequently, every component of the educational ecosystem – including curriculum, pedagogy, assessment, teacher preparation, educational leadership, and governance – should be designed to align with the principles by which the brain learns most effectively.

4. IMPLICATION FOR REMAINING THAI EDUCATION

If learning is fundamentally a biological process occurring within the brain, educational reform should begin by designing environments that maximize learning rather than merely delivering instruction.

Several implications naturally follow.

First, curriculum should emphasize conceptual understanding rather than excessive factual coverage. In an era where information is universally accessible through AI and digital technologies, education should prioritize thinking over remembering. Second, assessment should evolve from measuring short-term recall toward evaluating reasoning, creativity, collaboration, communication, and problem solving. Third, teachers should increasingly become learning designers and mentors rather than transmitters of information. Their role is to create environments that stimulate curiosity, encourage inquiry, and facilitate deep learning. Fourth, interdisciplinary learning – including STEM education – should become a central pedagogical approach because real-world problems rarely respect disciplinary boundaries. Finally, educational governance should promote continuity, evidence-based policymaking, professional autonomy, and long-term institutional learning instead of fragmented reforms driven by short political cycles. Collectively, these changes represent not isolated innovations but elements of a coherent learning ecosystem.

5. CONCLUSION

Thailand has invested enormous effort in educational reform during the past several decades. Yet sustainable transformation remains elusive because reforms have largely focused on educational structures rather than on the science of learning itself.

This paper has argued that the next generation of educational reform should begin with a different question – not "How should we reorganize schools?" but rather "How does the human brain learn?"

Neuroscience does not provide ready-made educational policies, nor should educational decisions be determined by neuroscience alone. Education remains a multidisciplinary endeavour encompassing psychology, sociology, philosophy, economics, culture, and public policy.

Nevertheless, neuroscience offers something indispensable: a scientific understanding of the learner.

When combined with educational research and thoughtful public policy, this understanding enables us to design educational environments that are more compatible with the biological principles through which people learn.

Ultimately, reimagining Thai education is not simply about introducing new curricula, new technologies, or new legislation.

It is about redesigning an educational ecosystem in which every learner has the opportunity to develop his or her fullest potential.

The future of Thai education should therefore be guided by a simple but profound principle:

Education should be designed not merely around what society wishes to teach, but around how the human brain learns.

Only then can educational reform become both scientifically grounded and sustainably transformative.

Author's Perspective

The ideas presented in this paper are not intended as a comprehensive review of educational neuroscience, nor as a formal proposal for educational policy. Rather, they represent a personal reflection arising from a unique professional journey that has traversed three interconnected domains.

My academic career began in neuroscience. As a young investigator, my research focused on the fundamental mechanisms of brain function and synaptic plasticity – the biological processes through which experience modifies neural circuits and learning becomes possible. Like many neuroscientists of my generation, I was fascinated by one of nature's most remarkable questions: **how does the brain learn?**

My professional path subsequently took an unexpected direction. I entered higher education administration, serving as Dean of a Faculty of Pharmacy, Founding President of a newly established university, and later as Secretary-General responsible for Thailand's higher education system. These responsibilities shifted my attention from individual neurons to educational institutions, from laboratory research to educational leadership, and eventually to national policy.

During those years, I participated in numerous educational reforms involving university development, quality assurance, curriculum improvement, governance, and national policy. These experiences provided invaluable insights into the complexity of educational systems and the challenges of implementing large-scale change.

More recently, my work has focused on educational reform at the national level. This has prompted me to revisit an important question that first inspired me as a neuroscientist many decades ago:

If education exists for learning, and learning occurs in the brain, why have educational systems so rarely been designed according to the scientific principles of how the brain learns?

This paper represents my attempt to reconnect these two worlds.

It is not an argument that neuroscience alone can solve educational problems. Education is far too complex to be explained by biology alone. It is equally shaped by psychology, sociology, philosophy, economics, culture, and public policy. Nevertheless, neuroscience provides an indispensable scientific foundation by helping us understand the learner – the very person whom education seeks to develop.

As educational systems around the world confront the profound challenges of artificial intelligence, demographic change, and accelerating technological transformation, I believe the time has come to place the science of learning at the centre of educational thinking.

Reimagining Thai education therefore requires more than new curricula, better technologies, or revised legislation. It requires a shift in perspective – from designing educational systems around administrative convenience to designing them around human learning.

In that sense, this paper is not only about Thailand.

It is about rediscovering a simple but fundamental truth:

The science of learning is, ultimately, the science of the brain.

The Brain-First Framework for Educational Transformation

This is not another teaching method, nor another curriculum model. It is a design philosophy:

Every educational decision – from curriculum and assessment to teacher education and governance – should ultimately be judged by one question: *Does it support how the human brain learns?*

Dr. Pavich Tongorach is a distinguished neuropharmacologist and prominent educational leader whose academic foundation includes a B.Sc. in Pharmacy from Chulalongkorn University, a Ph.D. in Neuropharmacology from UCL, and postdoctoral training in neurophysiology under Dr. Masao Ito at the University of Tokyo. Throughout his extensive career in public and institutional administration, he has held highly influential roles in Thailand, including Dean of the Faculty of Pharmacy at Chulalongkorn University, Founding President of Mahasarakham University, Secretary General of the Commission on Higher Education, and Vice Minister for Education. An internationally recognized figure in scientific leadership, Dr. Tongorach has also served as President of the Thai Neuroscience Society, President of the Federation of Pan Asian Oceanic Neuroscience Societies, and Secretary General of IBRO's Pan Asian Oceanic Neuroscience Commission.



PL-2

Prof. Anan Srikiatkhachorn, M.D.

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Medication Overused Headache – Possible Pathogenesis**Abstract:**

Medication overuse headache (MOH), the development or worsening of chronic headache resulting from frequent and excessive intake of medications used for acute treatment of headache, is a common secondary headache disorder and is associated with significant personal and societal burdens. Despite no clear explanation regarding the pathophysiologic process leading to the headache progression, certain features such as increased sensory sensitivity, cutaneous allodynia, impaired habituation, identify the neuronal hyperexcitability as the plausible mechanism. Several lines of evidence indicate that chronic exposure to acute care migraine treatment may suppress endogenous serotonin (5-HT)-dependent antinociceptive systems. This alteration consequently facilitates the trigeminal nociceptive process via multiple mechanisms including enhancing nitric oxide production, phosphorylation of glutamate receptor and up-regulation of the calcitonin gene-related peptide (CGRP) system.

Keywords: iPSCs, neurodegeneration, neuron, lysosome, live-cell imaging

Prof. Anan Srikiatkhachorn, M.D., is a Professor of Neuroscience at King Mongkut's Institute of Technology Ladkrabang (KMITL), where he previously served as Founding Dean of the Faculty of Medicine. He received his M.D. from Mahidol University and completed research training at the University of New South Wales, Australia. A renowned authority in headache and pain medicine, his research focuses on migraine neurobiology, central sensitization, and medication-overuse headaches. With extensive publications on Google Scholar and Scopus, Prof. Anan remains a key figure in advancing Thai neuroscience research and medical education.



PL-3

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**Advancing Neural Organoid Technology for Better
Understanding of CNS Development and Disease
Modeling****Abstract:**

The development of the human central nervous system (CNS) is a highly orchestrated process, where disruptions can lead to severe structural abnormalities such as microcephaly and neural tube defects (NTDs). While human neural organoids offer unprecedented opportunities to study these conditions *in vitro*, their practical application has been severely limited by high batch-to-batch variability and the challenge of producing diverse CNS regions at scale. This study aims to overcome these limitations by developing a robust, scalable production pipeline capable of simultaneously generating diverse neural organoids with high reproducibility and precise regional identity. Building upon human spinal cord organoid (hSCO) technology – which successfully recapitulates neural tube-like morphogenesis and synchronized neural activity – we expanded this methodology into an integrated platform. This system allows for precise control over organoid size and regional identity along the anteroposterior and dorsoventral axes. We then utilized this platform as a high-content screening tool for toxicological and pathological evaluation. The established pipeline ensured high reproducibility across experimental batches. Through high-content screening, we successfully identified specific compounds that disrupt neural tube closure and analyzed the resulting structural malformations, effectively bridging the gap between basic embryology and human clinical pathophysiology. Our robust production pipeline provides a powerful foundation for investigating a wide spectrum of congenital CNS disorders. This versatile platform holds significant potential to accelerate toxicological screening and the development of personalized therapeutic strategies.

Keywords: Human neural organoids, Neural tube defects, Scalable production, High-content screening

Prof. Woong Sun is a Professor in the Department of Anatomy at Korea University College of Medicine and a leading expert in developmental neurobiology and organoid technology. He earned his Ph.D. from Seoul National University, followed by research fellowships at Osaka University and Wake Forest University, and a visiting professorship at UCSD. Dr. Sun is internationally recognized for his pioneering work on nervous system self-organization, mitochondrial dynamics, and advanced imaging and high-throughput organoid analysis platforms, boasting an exceptional H-index of 51. An influential figure in



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the global scientific community, he currently serves as the President of the Asian-Pacific Society for Neurochemistry (APSN) and is the President-elect of the Korean Society for Brain and Neural Sciences (KSBNS).



PL-4

Prof. Alen Shih-Kuo Chen, Ph.D.

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**From Blue Light Photoreceptor ipRGCS to Behaviors:
Computational Circuits and Light Avoidance****Abstract:**

Intrinsically photosensitive retinal ganglion cells (ipRGCs) project to subcortical structures to govern non-image-forming functions. However, how the deep hypothalamus transforms these retinal signals into neural representations that drive phase shifts or behavior has remained unclear due to significant optical and physical barriers. We sought to determine the in vivo circuit mechanisms and network principles within the master circadian clock—the suprachiasmatic nucleus (SCN)—and the supraoptic nucleus (SON) that compute luminance to govern photoentrainment and adaptive behavioral responses. We bypassed deep-brain imaging barriers by combining in vivo two-photon calcium imaging with GRIN endoscopes in live mice. We further manipulated phase-specific networks using TRAP and DREADD chemogenetic systems. In the SCN, we discovered a dynamic, bi-stable network model rather than a rigid labeled-line system. While most SCN neurons exhibit highly stochastic light responses in vivo, small subsets are consistently modulated: one activated at ZT16 (early night) and another inhibited at ZT22 (late night). Activating ZT16-trapped neurons induced phase delays at any circadian time, effectively breaking the photoentrainment dead zone. Paralleling this population strategy, individual SON oxytocin neurons also display trial-to-trial stochasticity but form dynamic ensembles. Driven by parallel ipRGC input and peri-SON GABAergic feed-forward inhibition, these ensembles transition from a high to a low excitatory-to-inhibitory ratio to encode luminance intensity. Our findings establish a new paradigm wherein hypothalamic circuits utilize distributed, flexible population codes rather than static relays. This provides a comprehensive computational framework for how ipRGC pathways integrate environmental luminance to gate discrete clock adaptations and immediate behavioral survival choices.

Keywords: circadian clock, avoidance, circuit, melanopsin, ipRGC



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Prof. Shih-Kuo (Alen) Chen is a Professor in the Department of Life Science at National Taiwan University (NTU). He earned his Ph.D. from the University of Houston and completed postdoctoral training at Johns Hopkins University. Dr. Chen's research focuses on intrinsically photosensitive retinal ganglion cells (ipRGCs), circadian rhythms, neural circuits, and non-image-forming visual functions. An award-winning scientist, he serves as a Council Member of the Taiwan Neuroscience Society and holds key administrative roles at NTU, including Deputy Director of the Center for Biotechnology.



PL-5

Prof. Hitoshi Okamoto, M.D., Ph.D.

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The Two Parallel Pathways from the Habenula to the Interpeduncular Nucleus Control Social Conflict**Abstract:**

The neural pathways from the habenula to the interpeduncular nucleus (IPN) are conserved throughout evolution. We have shown both in zebrafish and mouse that two parallel pathways in this circuit antagonize each other to make animals resilient against or prone to surrender in social conflict. We have recently expanded our closed-loop virtual reality system to allow zebrafish to swim in the two-dimensional virtual space. By setting up two fish independently in this system and showing the avatar of the opponent fish on each other's screen, we have succeeded in letting two avatars fight in the virtual space. We found that fish with the impaired winner-specific Hb-IPN pathway tend to fail in targeting the opponent correctly. Anterograde and retrograde poly-transsynaptic viral tracing from the IPN revealed reciprocal connections of the IPN with multiple brain regions for the interoception and the emotional regulatory system, suggesting the dual roles of this circuit both in precision of behavioral control and stress resilience.

Keywords: habenula, interpeduncular nucleus, social conflict, virtual reality, resilience

Prof. Hitoshi Okamoto is a world-renowned neuroscientist and RIKEN Honorary Scientist / Visiting Senior Scientist at the RIKEN Center for Brain Science (CBS) in Japan. He serves as the Project Leader of the National Bioresource Project (Zebrafish) and is the former Chair of the IBRO Asia-Pacific Regional Committee. Prof. Okamoto is internationally acclaimed for his pioneering research on neural circuit dynamics. Utilizing advanced zebrafish models, his work has successfully uncovered key brain mechanisms underlying decision-making, fear, and anxiety. Recipient of the prestigious Tokizawa Award (2014), his insights continue to drive global breakthroughs in behavioral neuroscience.



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SCIENTIFIC SESSION I: NEUROLOGICAL MODELS

SS1

**Prof. Steve Leu, Ph.D.**

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The Emerging Role of Pnn in Neurodegenerative Diseases: From RNA Homeostasis to Neuronal Dysfunction

Abstract:

Dysregulation of RNA-binding proteins (RBPs) is increasingly recognized as a key mechanism underlying neurodegenerative diseases. Pnn (Pinin) is a multifunctional RNA splicing regulator. However, its role in maintaining neuronal RNA-binding protein (RBP) homeostasis during aging remains largely unknown. Aim: To investigate whether neuronal Pnn deficiency contributes to age-related neurodegeneration and to determine its impact on RNA-binding protein homeostasis, synaptic function, and behavioral outcomes. Inducible neuron-specific Pnn knockout mice (CaMKII-CreERT2; Pnnflox/flox⁺) were generated. Brain structural changes were assessed by longitudinal magnetic resonance imaging (MRI). Behavioral phenotypes were evaluated using anxiety, social interaction, spatial learning, and nesting tests. Hippocampal synaptic plasticity was examined by long-term potentiation (LTP) recordings. Molecular alterations were characterized using Visium spatial transcriptomics, quantitative RT-PCR, Western blotting, and immunofluorescence analyses of disease-associated RNA-binding proteins. Neuronal Pnn depletion did not cause overt neuronal loss but induced progressive ventricular enlargement and hippocampal atrophy during aging. Pnn-deficient mice exhibited age-dependent anxiety-like behavior, impaired social novelty recognition, deficits in spatial learning strategies, and severe nesting impairment. While hippocampal LTP remained largely preserved at 6 months, significant impairment was observed at 12 months, indicating progressive synaptic dysfunction. Spatial transcriptomic analysis revealed dysregulation of genes involved in synaptic function and neuronal maintenance. Furthermore, neuronal Pnn deficiency disrupted RNA-binding protein homeostasis, characterized by altered expression, subcellular distribution, and pathological changes of multiple disease-associated RNA-binding proteins. Neuronal Pnn deficiency leads to progressive structural, molecular, synaptic, and behavioral abnormalities associated with age-related neurodegeneration. Our findings identify Pnn as an important regulator of neuronal RNA-binding protein homeostasis and suggest that disruption of RNA-binding protein homeostasis may contribute to disease progression.

Keywords: Pnn, Pinin, Neurodegeneration, RNA-binding proteins, Behavior deficits

Prof. Steve Leu is a Professor and Deputy Director at the Institute for Translational Research in Biomedicine, Kaohsiung Chang Gung Memorial Hospital, Taiwan. His research centers on RNA-binding proteins, RNA homeostasis, and the molecular mechanisms



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underlying neurodegenerative diseases and cardiomyopathy – with a strong focus on Pnn-mediated pathways and cellular homeostasis. Dr. Leu has authored over 100 peer-reviewed publications and serves as an executive committee member and editorial board member for prestigious international medical and scientific journals.

SS2

**Asst. Prof. Tewarit Sarachana, Ph.D.**

*Faculty of Allied Health Sciences, Chulalongkorn
University, Thailand*

From the Blood to the Brain: Development of Human Brain Organoid-On-Chip from Peripheral Blood Mononuclear Cells of Thai Children

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Abstract:

Medical technologists play an important role in advancing laboratory-based platforms that bridge clinical specimens, molecular diagnostics, and disease modeling. Human brain organoids derived from induced pluripotent stem cells (iPSCs) provide a promising model for studying neurodevelopment; however, conventional culture systems do not fully reproduce the dynamic microenvironment of human tissues. This study aimed to develop a human brain organoid-on-a-chip model using iPSCs generated from peripheral blood mononuclear cells (PBMCs) of Thai children, highlighting the translational value of medical laboratory science in precision health research. Aim(s): To develop and validate a human brain organoid-on-a-chip platform using PBMC-derived iPSCs from Thai children for neurodevelopmental modeling. Microfluidic chips were designed and simulated using COMSOL Multiphysics to evaluate fluid velocity and shear stress suitable for organoid culture. Chip molds were fabricated using high-resolution 3D printing, followed by polydimethylsiloxane casting and plasma bonding. PBMCs were reprogrammed into iPSCs using Yamanaka factors. Pluripotency was validated by RT-qPCR, immunofluorescence staining, and trilineage differentiation assays. Validated iPSCs were aggregated into embryoid bodies, induced toward neuroepithelial lineage, and cultured using both microfluidic chip-based and conventional organoid culture systems. Cerebral organoids were fixed, cryo-sectioned, and assessed for neuronal marker expression. The designed microfluidic chips demonstrated velocity and shear stress profiles compatible with brain organoid culture. PBMC-derived iPSCs expressed key

pluripotency markers, including *OCT4*, *NANOG*, and *SOX2*, and successfully differentiated into all three germ layers. Cerebral organoids generated from both culture systems expressed neuronal and neural progenitor markers, including *FOXP1*, *MAP2*, *PAX6*, *TUBB3*, and *TBR1*, and showed organized neuronal structures. This study successfully established a PBMC-derived human brain organoid-on-a-chip model from Thai children. The platform represents an original and clinically relevant laboratory model for studying neurodevelopmental disorders and demonstrates the expanding contribution of medical technologists to advanced biomedical innovation, molecular validation, and translational research.

Keywords: induced pluripotent stem cells, brain organoid, organoid-on-a-chip, medical laboratory science, neurodevelopment

Asst. Prof. Tewarit Sarachana is Assistant to the President of Chulalongkorn University and a faculty member in the Department of Clinical Chemistry, Faculty of Allied Health Sciences. He earned his Ph.D. in Molecular Medicine (Neuroscience) from George Washington University, USA. Dr. Tewarit leads the Chulalongkorn Autism Research and Innovation Center of Excellence (ChulaACE). His research integrates precision medicine, multi-omics, bioinformatics, and human brain organoids to investigate brain development, degeneration, and biomarker discovery in autism spectrum disorder and related neurological conditions.



SS3

**Assoc. Prof. Siriporn Chamniansawat,
Ph.D.**

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Thailand*

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**Targeting Neuroinflammation and Synaptic Dysfunction
with Bioactive Fractions from *Celastrus Paniculatus*****Abstract:**

Neurodegenerative diseases are characterized by progressive neuronal loss, chronic neuroinflammation, synaptic dysfunction, and cognitive decline. Emerging evidence suggests that effective therapeutic strategies should target both neuronal and glial pathways rather than neurons alone. *Celastrus paniculatus* (CP), a traditional medicinal plant, possesses antioxidant, anti-inflammatory, and neuroprotective properties; however, its multi-level mechanisms of action remain incompletely understood. Aim(s): To investigate the neuroprotective mechanisms of bioactive fractions isolated from *Celastrus paniculatus* across complementary cellular and animal models by evaluating their effects on neuronal survival, neuroinflammation, synaptic function, and memory. CP seed extracts were fractionated using silica gel chromatography, and the bioactive fractions (F6 and F7) were selected for mechanistic studies. Neuroprotective effects were evaluated in three experimental models: (i) high-dose DMSO-induced SH-SY5Y neuronal injury to assess neuronal viability, mitochondrial function, neurite outgrowth, and BDNF expression; (ii) scopolamine-induced memory impairment in Sprague–Dawley rats to evaluate spatial learning, memory, and hippocampal synaptic plasticity; and (iii) astrocyte-conditioned medium (ACM)-induced BV2 microglial activation to examine CD40/iNOS signaling, phagocytic activity, microglial morphology, and pro-inflammatory cytokine production. F6 and F7 significantly enhanced neuronal survival, improved mitochondrial function, promoted neurite outgrowth, and increased BDNF expression in SH-SY5Y cells. In vivo, treatment improved spatial learning and memory while preserving hippocampal synaptic plasticity. In activated BV2 microglia, both fractions suppressed CD40 and iNOS expression, reduced phagocytic activity, attenuated microglial activation, and decreased TNF- α and IL-6 production. Bioactive fractions F6 and F7 from *Celastrus paniculatus* provide neuroprotection through coordinated modulation of neuronal survival, synaptic plasticity, and microglia-mediated neuroinflammation. These findings support CP-derived bioactive fractions as promising multi-target therapeutic candidates for neurodegenerative diseases.

Keywords: *Celastrus paniculatus*, Neuroprotection, Microglia, Neuroinflammation

Assoc. Prof. Siriporn Chamniansawat is an Associate Professor in Neuroscience at the Faculty of Allied Health Sciences, Burapha University, Thailand. She earned her Ph.D. in



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Anatomy and Structural Biology from Mahidol University. Over the past 15 years, her research program has focused on hippocampal synaptic plasticity, neuroinflammation, astrocyte–microglia interactions, and brain-derived estrogen signaling in age-related cognitive decline. She also investigates bioactive natural products like *Celastrus paniculatus* for neuroprotection.

SS4

Assoc. Prof. Amornpan Ajjimaporn, Ph.D.

College of Sports Science and Technology, Mahidol
University, Thailand



How Does Sport Shape the Brain? Neural Signatures of Visual Processing in Elite Gymnasts, Soccer Players, and Esport Athletes

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Abstract:

Elite athletes are exposed to sport-specific visual environments that continuously challenge attention, perception, decision-making, and visuomotor coordination. Growing evidence suggests that long-term sport participation induces functional adaptations within the central nervous system; however, neural differences among athletes from sports with distinct visual demands remain poorly understood. Aim(s): To summarize a series of studies investigating sport-specific neural signatures of visual processing in elite female gymnasts, soccer players, and esports athletes using multimodal neurophysiological and visual assessments. Forty-two elite female athletes (14 gymnasts, 14 soccer players, and 14 esports athletes) completed a comprehensive assessment including resting-state electroencephalography (EEG), visual P300 event-related potentials (ERPs), eye-tracking, and visual ability tests. Resting cortical activity during eyes-closed and eyes-open conditions, neural responses to visual oddball stimuli, gaze behavior, visual reaction time, working memory, peripheral vision, and accommodation performance were compared across athlete groups. Distinct sport-specific neural profiles were identified. Gymnasts demonstrated greater neural efficiency, characterized by smaller eyes-closed to eyes-open alpha transitions and longer fixation durations, suggesting refined internal attentional control. Soccer players exhibited

enhanced frontal delta and theta activity during visual engagement together with superior response accuracy, indicating efficient visual attention and decision-making. Esports athletes showed faster near-far accommodation responses but prolonged frontal P300 latency, reflecting adaptations associated with prolonged screen-based visual processing. Collectively, resting EEG, visual ERPs, eye-tracking, and behavioral measures consistently demonstrated that different sporting environments are associated with unique neural adaptations supporting sport-specific visual performance. Long-term participation in different sports is associated with distinct neural signatures of visual processing. Integrating EEG, ERPs, eye-tracking, and visual performance assessments provides valuable insight into sport-specific brain adaptations and may facilitate the development of neuroscience-informed training strategies to optimize athletic performance.

Keywords: EEG, Visual Processing, Event-Related Potentials, Eye Tracking, Elite Athletes

Assoc. Prof. Amornpan Ajjimaporn is an Associate Professor at the College of Sports Science and Technology and a researcher at the Research Center for Neuroscience, Mahidol University, Thailand. She holds a Ph.D. in Neuroscience and specializes in exercise physiology, motor control, and neurorehabilitation. Her research integrates electroencephalography (EEG), non-invasive brain stimulation (rTMS), and movement analysis to investigate cognitive function, brain activity, and physical performance in athletes and individuals with neurodegenerative disorders such as Parkinson's disease.

SS5



**(Adjunct) Asst. Prof. Peeraporn
Varintra, Ph.D.**

*Institute of Medical Sciences, Tzu Chi University,
Hualien, Taiwan*

**Natural Product-Based Intervention for Sleep Loss-
Induced Cognitive Decline: Mechanistic Insights into
TCU411**

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Abstract:

Rapid eye movement sleep deprivation (REM-SD) is strongly associated with cognitive impairment and an increased risk of neurodegenerative disorders. Synaptic dysfunction, dysregulated GABAergic signaling, and actin cytoskeletal remodeling are key mechanisms underlying sleep loss-induced memory deficits. Despite growing evidence linking sleep disruption to cognitive decline, effective mechanism-based therapeutic interventions remain limited. TCU411, an indigenous herbal extract, has emerged as a promising natural product with neuroprotective potential. Aim(s): To investigate the therapeutic efficacy of TCU411 against REM-SD-induced cognitive impairment and to elucidate the molecular mechanisms underlying its neuroprotective effects, with particular emphasis on synaptic plasticity and actin cytoskeletal remodeling. REM-SD mouse models underwent behavioral assessments, including the T-maze and Morris water maze, to evaluate working memory, spatial memory, and memory flexibility. Hippocampal tissues were analyzed using proteomics, western blotting, and immunofluorescence to examine alterations in GABAergic signaling, synaptic proteins, protein kinase C (PKC) signaling, and actin remodeling. Active fractions of TCU411 were further evaluated to identify the components responsible for its neuroprotective effects. REM-SD impaired hippocampus-dependent learning and memory, accompanied by increased GABA_B receptor expression, reduced PSD-95 levels, dysregulated PKC signaling, and disrupted F-actin organization. Administration of TCU411 significantly improved cognitive performance across behavioral paradigms. Mechanistically, TCU411 extract normalized GABA_B receptor and PSD95 expression. The fraction of TCU411-F1 and

F2 restored PKC α /PKC γ signaling and promoted actin stabilization by activating the PAK1/LIMK1/cofilin pathway. TCU411 alleviates REM-SD-induced cognitive deficits by restoring synaptic plasticity and actin cytoskeletal dynamics through modulation of GABAergic and PKC signaling. These findings support TCU411 as a promising natural product for preventing sleep loss-induced cognitive decline by targeting actin remodeling.

Keywords: Indigenous herbal medicine, Sleep deprivation, TCU411, Actin cytoskeleton, Synaptic plasticity

Peeraporn Varinthra, Ph.D., is a Postdoctoral Fellow and Adjunct Assistant Professor in the Institute of Medical Sciences at Tzu Chi University, Hualien, Taiwan. She earned her Bachelor of Science in Physical Therapy (2011) and Master of Science in Anatomy (2014) from Mahidol University, Thailand, followed by a Ph.D. in Medical Sciences from Tzu Chi University (2021). Her research interests focus on herbal medicine and natural products, neurodegenerative diseases (such as Alzheimer's and Parkinson's disease), neuropsychiatric disorders, the impact of sleep on learning and memory, and synaptic functions. She has received numerous honors, including the 2026 NSTC France-Taiwan ORCHID Program-Researcher Exchange Project at CNRS in Montpellier, France, and international travel grants from IBRO. Additionally, she serves as a peer reviewer for various prominent scientific journals in neurobiology and ethnopharmacology.



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SCIENTIFIC SESSION II: ATMPS & DRUG DISCOVERY



SS6

Prof. Jintanaporn Wattanathorn, Ph.D.*Faculty of Medicine, Khon Kaen University, Thailand*

Anthocyanins – Enriched Products : A potential candidate for Preventing and Mitigating Age- Related Neuropsychological Disorders

At present, neuropsychological disorders are regarded as the leading cause of overall illness and disability worldwide, affecting over 3.4 billion people (more than 43% of the global population). These conditions are tied to the significant rising of the total health loss, and disability burdens. It has been demonstrated that most of the current prescription drugs that target single pathways often carry heavy risks while the natural bioactive compounds offer a safer, holistic, and sustainable approach to long-term brain health. Moreover, less prophylaxis drugs approach is available. Owing to the accumulative lines of evidence regarding the neuroprotective effect against neuropsychological disorders of the functional foods, the preventing and mitigating age-related neuropsychological disorders of anthocyanins-enriched product have gained much attention. Anthocyanins can improve neuropsychological disorders by many pathways. It can pass the blood-brain barrier directly, and improves oxidative stress status, inflammation, and neurotransmitters disturbance. In addition, it also exerts its indirect effect via the modulation of gut-brain-axis. Moreover, it also exerts the nutrigenomic effect by modulating genes that regulate different biological processes, including neurogenesis, inflammation, metabolism, cell to cell adhesion, cytoskeleton organization, and Alzheimer's and Parkinson's disease pathology. Taken together, integrated analysis revealed a multi-target mode of action of anthocyanin-rich products in the brain underlying the neuromodulation mechanisms.

Professor Dr. Jintanaporn Wattanathorn is a Professor in the Department of Physiology at the Faculty of Medicine, Khon Kaen University. She earned her Bachelor of Science (with Honors) in 1983, Master of Science in Physiology in 1986, and Ph.D. in Neuroscience in 1994, all from Mahidol University, where she graduated with a perfect GPA of 4.00. Her areas of expertise include functional food development, alternative medicine, personalized nutrition, and neuroscience. Throughout her distinguished career, she has served in prominent leadership roles, including Acting Director of the Human Performance and Health Promotion Research Institute and Head of the Department of Physiology at Khon Kaen University. A highly accomplished researcher and inventor, she has received numerous accolades, such as the Sarasin Distinguished Researcher Award, Mahidol University Alumni Honor Awards, and international gold medals for her innovations in functional foods and health products.



SS7

Asst. Prof. Jacque Pak Kan Ip, Ph.D.*Chinese University of Hong Kong*

Circular RNAs Drive Synaptic Plasticity to Promote Learning and Memory

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Abstract:

Neuronal circuits are known to be plastic and are subject to experience-driven changes. To gain a deeper understanding of the mechanisms underlying plasticity, we utilize monocular deprivation (MD) as an experimental model. MD involves briefly closing one eye, causing an imbalance in input to neurons in the primary visual cortex (V1). This manipulation serves as a valuable tool for studying ocular dominance plasticity, a specific form of visual cortical plasticity. During the critical period of visual development, MD leads to a weakening of functional responses in V1 neurons to the deprived (closed) eye, followed by a strengthening of responses to the non-deprived (open) eye. We aim to identify uncharacterized regulatory mechanisms of plasticity. Circular RNAs (circRNAs) are regulatory noncoding RNAs abundantly found in brain tissue. Their synaptically-enriched, activity-inducible, and developmentally-regulated properties suggest a role in experience-dependent synaptic plasticity. However, functional investigation of circRNAs in neurons is still limited. Relatively little is known about their role in experience-dependent plasticity. Previous studies have suggested that MD during the critical period induces dendritic spine remodeling, which contributes to the functional plasticity of V1 neuron responses. However, the precise mechanisms underlying dendritic spine remodeling and the functional characteristics of the eliminated spines in response to MD remain to be elucidated. Here, we identified unique activity-dependent circRNAs upon plasticity induction. We found that circRNAs were robustly and differentially regulated. We demonstrated the changes of circRNAs in cortical and hippocampal synaptic plasticity. Our study provides evidence of an experience-dependent circRNA that is a crucial regulator of synaptic development and plasticity as well as learning and memory.

Keywords: synaptic plasticity, circular RNA, visual cortex, hippocampus, learning and memory

Asst. Prof. Jacque Pak Kan IP is an outstanding neuroscientist currently serving as an Assistant Professor in the School of Biomedical Sciences at The Chinese University of Hong Kong (CUHK), where he is also a Core Faculty member of the Gerald Choa Neuroscience Institute. Following elite postdoctoral training at the Massachusetts Institute of Technology (MIT), his research deeply investigates the mechanisms of synaptic development, experience-dependent plasticity, and neurological disorders. His pioneering work has been featured in premier journals including *Science*, *Nature Neuroscience* and *Science Advances*. Globally recognized, Prof. Ip serves on the Council of the Asia-Pacific Society for Neurochemistry (APSN) and the IBRO Early Career Committee.



SS8

Asst. Prof. Jiraporn Panmanee, Ph.D.*Institute of Molecular Biosciences, Mahidol University,
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Structure-Based Drug Design: Exploring Small Molecules for Neurodegenerative Diseases

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Abstract:

Neurodegenerative diseases are characterized by progressive neuronal loss and currently lack effective disease-modifying therapies. Structure-based drug design (SBDD) has emerged as a promising strategy for accelerating the discovery of novel therapeutic compounds by integrating computational and experimental approaches. Aim(s): To establish an integrated SBDD workflow for the identification and validation of small-molecule modulators targeting proteins implicated in neurodegenerative diseases. Candidate compounds were identified through molecular docking-based virtual screening and further evaluated by molecular dynamics simulations to assess protein–ligand stability. Pharmacokinetic and drug-likeness properties were predicted using in silico ADMET analysis. Selected compounds were experimentally validated by differential scanning fluorimetry (DSF) and isothermal titration calorimetry (ITC). Selected small molecules were undergo experimental validation using complementary biochemical, molecular, and cell-based assays to confirm their biological activity and mechanisms of action. The integrated workflow is expected to identify promising lead compounds with favorable binding affinity, stability, and pharmacokinetic properties. Experimental validation will confirm target engagement and evaluate their neuroprotective potential and molecular mechanisms. Combining computational drug discovery with biochemical and cellular validation provides an efficient platform for identifying and characterizing novel therapeutic candidates for neurodegenerative diseases, facilitating the translation of promising compounds toward preclinical development.

Keywords: Structure-based drug design; Neurodegenerative diseases; Molecular docking; Drug discovery; Molecular dynamics

Asst. Prof. Jiraporn Panmanee is an Assistant Professor at the Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University. She earned her Ph.D. in Biological Sciences from the University of Liverpool. Her research integrates structural biology, computational drug design, protein biochemistry, and neurobiology to



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investigate neurodegenerative diseases, pesticide neurotoxicity, and target therapeutics like IDO-1. Recognized as one of Mahidol University's Top 100 Researchers in 2025, Dr. Panmanee has led multiple competitive research grants focusing on melatonin, mitochondrial dysfunction, and neuroprotection



SS9

**Asst. Prof. Sukonthar Ngampramuan,
Ph.D.***Institute of Molecular Biosciences, Mahidol University,
Thailand***Bioengineered Exosomes for Neurorepair after Ischemic
Stroke: Bridging Drug Discovery and Regenerative
Medicine**

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University, Nakhon Pathom 73170, Thailand*

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Abstract:

Ischemic stroke remains a leading cause of mortality and long-term disability worldwide. Despite advances in acute stroke management, effective therapies that promote neural repair and functional recovery remain limited. Bioengineered extracellular vesicles (EVs), particularly stem cell-derived exosomes, have emerged as promising Advanced Therapy Medicinal Products (ATMPs) due to their ability to deliver therapeutic cargo across the blood-brain barrier and modulate neuroregenerative processes. Aim (s): To investigate the therapeutic potential of engineered SHED-derived exosomes in experimental ischemic stroke and to evaluate their effects on functional recovery, neuroprotection, and neural repair mechanisms. A rat model of chronic cerebral ischemia was established using bilateral common carotid artery occlusion (BCCAO), with a reduction in cerebral blood flow confirmed by laser Doppler flowmetry. Animals received intravenous administration of either native SHED-derived exosomes or RVG-engineered SHED-derived exosomes. Neurological deficits, motor coordination, balance, and locomotor performance were evaluated using the modified neurological severity score (mNSS), beam balance, beam walking, and grid walking tests. Histopathological and molecular analyses were performed in the motor cortex and hippocampus to assess neuronal survival, astroglial activation (GFAP), and markers associated with neuronal regeneration. Exosome-treated animals demonstrated significant improvement in neurological and motor outcomes compared with untreated ischemic controls. Histological examination revealed reduced neuronal degeneration and preservation of neuronal architecture in vulnerable brain regions. Furthermore, exosome treatment attenuated reactive astrogliosis, as evidenced by reduced GFAP expression, and was associated with enhanced indicators of neural repair and regeneration. RVG-engineered exosomes produced more pronounced therapeutic effects than non-engineered exosomes. Bioengineered exosomes represent a promising ATMP platform for ischemic stroke by promoting neuroprotection, reducing neuroinflammation, and enhancing

endogenous repair mechanisms. These findings support further translational development of engineered EV-based therapeutics for neurological disorders.

Keywords: Exosomes; ATMPs; Ischemic Stroke; Neuroregeneration; Neuroprotection

Asst. Prof. Sukonthar Ngampramuan is an Assistant Professor at the Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Thailand. She holds a Ph.D. in Neurosciences from Mahidol University, with research training at Flinders University and Newcastle University in Australia. Her primary research interests focus on stress-related psychological disorders, animal models of psychiatric and brain diseases, and wearable biosensors for stress detection. Dr. Ngampramuan also serves as the Chair of the Institute Animal Care and Use Committee (MU-IACUC) and a Member of the Mahidol University Faculty Senate.



SS10

Suangsuda Supasai, Ph.D.

Faculty of Tropical Medicine, Mahidol University, Thailand

Rebuild the Alzheimer-like brain: HMGB1 Box A gene therapy promotes neurogenesis and cognitive recovery

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Abstract:

Alzheimer's disease (AD) is the leading cause of dementia, characterized by progressive cognitive decline associated with amyloid- β accumulation, tau pathology, neuroinflammation, synaptic dysfunction, and impaired neuronal plasticity. HMGB1 Box A has shown anti-inflammatory and anti-senescence effects, suggesting therapeutic potential in age-related neurodegenerative disorders. Aim(s): To determine whether HMGB1 Box A gene therapy ameliorates cognitive and neuropathological abnormalities and restores hippocampal neurogenesis-associated changes in a rat model of AD-like pathology. Male Wistar rats were chronically treated with AlCl_3 and D-galactose to induce AD-like features. Following disease development, calcium phosphate nanoparticle-encapsulated HMGB1 Box A plasmids were administered weekly at two doses for eight weeks. Behavioral, neuropathological, inflammatory, senescence-related, neurogenic, neurotrophic, neuronal activity, and MEK/ERK signaling outcomes were evaluated. Box A treatment improved locomotor and cognitive performance, reduced hippocampal amyloid- β accumulation and tau pathology, preserved neuronal and synaptic integrity, and attenuated microglial activation, astrogliosis, pro-

inflammatory responses, and cellular senescence. Box A also increased neurogenesis-associated markers across multiple developmental stages, including early neural progenitors, proliferating progenitors, intermediate progenitors, and mature neurons. These changes were accompanied by increased BDNF and CNTF expression, restoration of MEK/ERK signaling, modulation of excitatory/inhibitory neuronal markers, and increased neuronal activity, together with improved recognition memory. HMGB1 Box A gene therapy produced coordinated behavioral, molecular, and cellular recovery in AD-like rats. Its cognitive benefits may be mediated, at least in part, through the restoration of a hippocampal environment that supports neurogenesis, neuronal maturation, trophic signaling, and functional activity. These findings support HMGB1 Box A as a promising disease-modifying candidate for further preclinical development in AD.

Keywords: Alzheimer's disease; HMGB1 Box A; gene therapy; hippocampal neurogenesis; cognitive recovery

Dr. Suangsuda Supasai is a faculty member at the Faculty of Tropical Medicine, Mahidol University, with postdoctoral training from UC Davis. She specializes in neurobiology, neurotoxicology, and nutritional biology. Her research focuses on the molecular mechanisms of neurodegenerative disorders, particularly groundbreaking work on HMGB1 Box A gene therapy for Alzheimer's and Parkinson's diseases. Recognized internationally with awards such as the *Celebrating Women in Toxicology Award* and *Linus Pauling Institute's Young Investigator Award*, Dr. Supasai actively leads cutting-edge research in therapeutic strategies for brain health.



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Scientific Session III: IBRO-TNS Travel Award

SS11

Irma Wati Ngadimon. Ph.D.*University Malaya, Malaysia***Neuroinflammation as a Converging Mechanism Linking Traumatic Brain Injury, Epileptogenesis, and Cognitive Decline***Irma Wati Ngadimon¹, Mohamad Farooq Shaikh², Ching Soong Khoo³, Devi Mohan⁴**¹Department of Pharmaceutical Life Sciences, Faculty of Pharmacy, Universiti Malaya, Kuala Lumpur, Malaysia**²Neurology Unit, Department of Medicine, Hospital Canselor Tuanku Muhriz, Kuala Lumpur, Malaysia**³School of Dentistry and Medical Sciences, Charles Sturt University, Orange, New South Wales, Australia**⁴School of Public Health, The University of Queensland, Brisbane, Queensland, Australia***Abstract:**

Traumatic brain injury (TBI) is a leading cause of acquired epilepsy and is increasingly recognised as a contributor to long-term cognitive impairment and accelerated brain aging. While seizures represent a key clinical outcome, growing evidence indicates that persistent neuroinflammation plays a central role in driving epileptogenesis and progressive cognitive dysfunction following brain injury. This presentation examines neuroinflammatory signalling as a shared mechanistic pathway linking acute injury to chronic neurological sequelae. Focusing on inflammatory mediators such as high mobility group box-1 (HMGB1), interleukin-1 β , and tumour necrosis factor, I discuss how sustained activation of innate immune pathways – particularly the HMGB1-TLR4 axis – contributes to neuronal hyperexcitability, synaptic dysfunction, and impaired cognitive outcomes. Drawing on findings from clinical epilepsy cohorts and biomarker-based analyses, this talk highlights associations between inflammatory profiles, seizure development, and longitudinal cognitive performance. Rather than viewing epilepsy solely as a seizure disorder, this work frames post-traumatic epilepsy as a chronic brain condition with features overlapping neurodegeneration and aging-related decline. Understanding these shared mechanisms may enable earlier risk stratification and inform the development of targeted, disease-modifying interventions. By integrating clinical data with translational neuroscience concepts, this presentation aims to provide insight into how neuroinflammation shapes long-term brain outcomes after injury and its implications for aging populations.

SS12

Varsha Pai V

Manipal Academy of Higher Education, India

Three-Dimensional Neural Tissue Models Elucidate Cellular and Molecular Mechanisms of Brain Aging

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Abstract:

Brain aging is a complex process characterized by progressive cognitive decline, neurodegeneration, and impaired neural plasticity. Traditional two-dimensional (2D) cultures and animal models have limitations in replicating the intricate cellular microenvironment of the human brain. To address this issue, we developed a novel three-dimensional (3D) neural tissue model using a tissue engineering approach involving Wharton's jelly-derived mesenchymal stem cells (WJ-MSCs) and electrospun polycaprolactone (PCL)/gelatin scaffolds that mimic the brain tissue extracellular matrix. The electrospun PCL/gelatin scaffolds exhibited high porosity, mechanical stability, and cytocompatibility, supporting neuronal growth and maturation. Next, WJ-MSCs were isolated, characterized, and induced to differentiate into neuronal cells over 20 days on the PCL/gelatin scaffolds. Further, neuronal differentiation was confirmed by the expression of key neural markers, including nestin, synapsin-1, neural nuclei (NeuN), postsynaptic density protein 95 (PSD95), and neurofilamentlight chain (NF-L). Brain aging in the engineered neural tissue was induced by D-galactose (300 mM) treatment, which induced oxidative stress and cellular senescence. The 3D aged neural tissue demonstrated elevation in oxidative stress, senescence, and apoptotic markers, along with decreased autophagic activity. Furthermore, the system was validated using neuroprotective agents, which showed significant reversal of aging markers. Thus, the engineered 3D brain aging model offers a physiologically relevant, scalable, and reproducible platform for studying neuronal aging and high-throughput screening of anti-aging therapeutics.

SS14

Muhana Fawwazy Ilyas

Universitas Gadjah Mada, Indonesia

**Artificial Intelligence for Nerve Conduction Studies: A
Scoping review of Clinical Classification, Physiologic
Modeling, and reporting Support**

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Abstract:

Nerve conduction studies (NCS) generate rich tabular and waveform data, often interpreted alongside clinical history to produce electrodiagnostic conclusions. Recent work has applied computational methods to improve detection, grading, and reporting, but the studies vary widely in data type, task design, and clinical integration. Aim(s): To map how computational methods have been applied to NCS across diagnosis /severity grading, physiologic signal inference, and report interpretation /generation. To summarize common data modalities, performance trends, and gaps in real-world workflow adoption. A scoping approach was used to categorize the provided studies by setting (retrospective, prospective, simulation), data modalities (tabular NCS variables, waveforms, narrative reports, and multimodal combinations), task focus (diagnosis, severity grading, physiologic inference, report structuring, and report drafting), and evaluation type (benchmark performance vs clinician-facing impact). Findings were synthesized narratively to highlight patterns and remaining evidence gaps. Ten studies were included. The studies fall into three main themes: models using tabular NCS features for diagnosis and severity grading, waveform-based approaches for classification or physiologic inference, and language-model approaches for structuring or drafting electrodiagnostic reports from tables and clinical context. Condition-focused models, especially for carpal tunnel syndrome and neuropathy severity, often report strong performance when evaluated within well-defined datasets. Waveform-driven work shows promise at larger scale and for more complex differentiation tasks but is less often linked to end-to-end clinical interpretation workflows. Report-focused systems can improve interpretation quality in benchmarking settings, yet prospective testing suggests benefits may be limited by usability, efficiency, and fit within routine clinician workflows. Computational methods for NCS are advancing from narrow, high-performing classification tasks toward broader interpretation and reporting support. The biggest barrier to impact appears to be reliable integration into real clinical workflows with measurable improvements in efficiency or report quality.

Keywords: nerve conduction studies; electrodiagnosis; machine learning; waveform analysis; report generation

SS15

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Ceretrone: A Wearable Multi-Modal Framework for Point-of-Care Neurophysiological Monitoring and Foundation ModelBased Screening for Age-Related Neurological Decline

Jasmeet Singh Bindra¹, Shubhajit Roy Chowdhury²

Abstract:

Age-related neurological decline, encompassing mild cognitive impairment, early-stage dementia, and neurodegenerative conditions, represents a growing global health burden, yet accessible, continuous, and objective monitoring tools remain scarce, particularly in lowresource settings. We present Ceretrone, a low-cost wearable multi-modal neuroimaging headband coupled with a lightweight EEG foundation model for population-scale screening of neurological decline. The system is designed around three principles: clinical deployability, parameter efficiency, and generalizability across neurological conditions. The EEG foundation model is pre-trained on large-scale, multi-condition neurophysiological data using selfsupervised learning, enabling robust feature representations transferable to downstream tasks, including cognitive aging biomarker extraction, early dementia detection, and neurodegenerative disorder classification. Preliminary architectural design demonstrates that high-fidelity neural representations can be achieved within a compact parameter budget suitable for edge deployment. Ceretrone's non-invasive form factor and AI-driven backend position it as a scalable neurological screening tool for aging populations in both clinical and community settings. This work contributes a novel hardware-software framework at the intersection of wearable neuroimaging and foundation model neuroscience, directly addressing the global imperative for accessible neurodegenerative disease detection.

Keywords: Wearable EEG, Foundation Model, Neurodegenerative Disorders, Neurological Decline, Neuroscience AI

SS16

Minqing GU

University of Hong Kong, China

Deferiprone and Ferulic Acids Ameliorate Alcohol-Induced Cognitive Impairment by Modulating Iron Metabolism and Inhibiting Ferroptosis

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¹School of Chinese Medicine, LKS Faculty of Medicine, The University of Hong Kong, Hong Kong SAR, China

Abstract:

While alcohol-induced cognitive impairment and iron overload are recognized, the precise mechanisms linking alcohol to altered brain iron metabolism and subsequent cognitive decline remain unclear. This study investigated alcohol's impact on iron metabolism genes in cognitive brain regions and evaluated whether deferiprone (DFP) and ferulic acid (FA) ameliorate alcohol-induced memory impairment by modulating iron metabolism and ferroptosis. Mice underwent two experimental protocols. In Experiment 1, mice received oral alcohol (2.5 or 5 g/kg) daily for 28 days; ferrous iron levels were measured in the hippocampus, prefrontal cortex, striatum, midbrain, cerebellum and serum, and striatal expression of iron-metabolism genes (TFR1, DMT1, IRP2, TF, FTH1, FTL1, FPN1, HAMP) and MDA was assessed. In Experiment 2, another cohort received 5 g/kg alcohol with concurrent DFP (20 mg/kg) or FA (40 mg/kg). Cognitive function was evaluated using the Y-maze and Barnes maze. In the striatum, expression of TFR1, DMT1 and IRP2, ferroptosis markers (MDA, ACSL4, GPX4), synaptic proteins (SYP, PSD95), neuronal morphology and density, mitochondrial ultrastructure and dendritic spines were analyzed. Alcohol produced dose-dependent cognitive deficits and significantly increased ferrous iron levels in multiple brain regions and serum, with hippocampal and striatal iron elevation negatively correlating with cognitive scores. Striatal TFR1, DMT1, IRP2 and MDA were upregulated. DFP or FA treatment reversed alcohol-induced cognitive deficits and markedly reduced alcohol-elevated TFR1 and IRP2 expression, whereas DMT1 expression remained unchanged. Both compounds normalized ferroptosis markers (lower MDA and ACSL4, higher GPX4), prevented neuronal loss, restored SYP and PSD95 expression, ameliorated mitochondrial damage, increased dendritic complexity, total dendritic length and spine density in the striatum. DFP and FA attenuate alcohol-induced cognitive impairment in mice by normalizing brain iron metabolism, suppressing ferroptosis and oxidative stress, protecting dendritic and synaptic structure in the striatum.

Keywords: Alcohol, Cognitive impairment, Iron metabolism, Ferroptosis, Ferulic acid



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INTERNATIONAL BRAIN RESEARCH ORGANIZATION (IBRO) COMMUNICATION



IBRO Communication

Prof. Sukumal Chongthammakun, Ph.D.

*Emeritus Professor of Anatomy, Mahidol University,
Thailand*

*Member, IBRO Asia-Pacific Regional Committee
(2024-2025)*

President, Thai Neuroscience Society (2023-2025)

Unlocking Global Opportunities in Neuroscience: IBRO Programs for Research, Training, Publishing, and International Collaboration

Abstract:

The International Brain Research Organization (IBRO) is the world's leading federation of neuroscience organizations, dedicated to advancing neuroscience through equitable access to education, research, funding, and international collaboration. Through a diverse portfolio of programs, IBRO supports neuroscientists at all career stages while fostering scientific excellence and capacity building across its six global regions, including the Asia-Pacific.

An overview of IBRO's mission and strategic initiatives will be provided, highlighting opportunities that promote career development, research excellence, and international collaboration. Major funding schemes for individuals and organizations – including IBRO-supported Schools, Exchange Fellowships, Neuroscience Training Grants, Collaborative Research Grants, Rising Stars Awards, Parenthood Grants, Equitable Meeting Awards, and Brain Awareness Week Grants – will be introduced, together with practical considerations for identifying appropriate programs and developing competitive applications.

IBRO's expanding impact in the Asia-Pacific region will be highlighted through its support for neuroscience training, scientific meetings, collaborative research, and regional capacity building. The session will also introduce IBRO's official journals, *Neuroscience* and *IBRO Neuroscience Reports*, which provide high-quality publication platforms across all areas of neuroscience while generating essential revenue to sustain IBRO's global educational and scientific initiatives.

The upcoming **IBRO World Congress 2027** in Cape Town, South Africa, will be presented as a major opportunity for scientific exchange, networking, and international collaboration, with information on abstract submission, travel support, and participation opportunities. By increasing awareness of IBRO's global network and diverse portfolio of programs, the session aims to encourage neuroscientists throughout the Asia-Pacific region to engage more actively in international training, collaborative research, scientific publishing, and leadership activities, ultimately strengthening neuroscience across the region and contributing to the global neuroscience community.

Dr. Sukumal Chongthammakun, Ph.D., is an Emeritus Professor of Anatomy and Neuroscience at Mahidol University, Thailand. She earned her Ph.D. from Mahidol University

and completed postdoctoral training as an NIH Fellow at the University of Wisconsin. Her research focuses on neurodegeneration, neurotoxicology, neuroprotection, and neuroinflammation, with publications spanning four decades. Since 2000, she has played a leading role in advancing neuroscience in Thailand through scientific conferences, IBRO Associate Schools, STEM education, and international collaborations. She held successive leadership positions in the Thai Neuroscience Society, serving as General Secretary (1999–2010), Vice President (2010–2023), and President (2023–2025). She served as a member of the IBRO Asia-Pacific Regional Committee (2024–2025), contributing to the development of regional neuroscience initiatives, grant programs, and capacity-building activities. Prof. Chongthammakun has actively promoted IBRO programs, expanded access to travel grants and training opportunities, and fostered regional and international collaborations to advance neuroscience research, education, and scientific capacity across the Asia-Pacific region.



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Scientific Session IV: TNS committee- selected speakers



SS17

Prof. Somporn Kantharadussadee Triamchaisri, Ph.D.

*Faculty of Health Sciences, University of Cuu Long
Vietnam (UCL Vietnam)*

SKT Meditation Healing Exercise to Improve Health, Quality of Life and Healing Across the Life Span

Abstract:

The increasing prevalence of chronic diseases, mental health challenges, stress-related disorders, and population aging has created an urgent need for accessible, low-cost, and evidence-informed approaches to promote health and well-being throughout life. SKT Meditation Healing Exercise, developed in Thailand by Somporn Kantharadussadee Triamchaisri, integrates mindfulness, breathing regulation, body movement, relaxation, positive imagery, and self-healing practices. The approach has been applied across diverse populations, including children, adolescents, adults, older persons, patients with chronic illnesses, caregivers, healthcare professionals, and community members. This presentation aims to describe the conceptual framework, physiological mechanisms, and health outcomes of SKT Meditation Healing Exercise in improving health, quality of life, and healing across the lifespan. The SKT Meditation Healing Exercise model combines meditation, breathing exercises, gentle movement, posture adjustment, mindfulness practice, and positive suggestion. The program is grounded in holistic health principles addressing physical, psychological, social, and spiritual dimensions of well-being. Evidence from clinical studies, community interventions, and health promotion programs conducted in Thailand and other settings is reviewed to illustrate its effectiveness across different age groups. Findings from multiple studies indicate that SKT Meditation Healing Exercise contributes to reductions in stress, anxiety, depression, fatigue, pain perception, and physiological indicators associated with chronic stress. Improvements have been observed in autonomic nervous system balance, emotional regulation, sleep quality, cardiovascular health, immune function, memory performance, self-efficacy, and overall quality of life. Among older adults, regular practice has been associated with enhanced cognitive function, improved mental well-being, and greater resilience. In patients with chronic diseases, SKT Meditation Healing Exercise has demonstrated benefits in symptom management and health-related quality of life. Participants frequently report increased inner peace, self-awareness, positive thinking, and a stronger sense of meaning and connectedness.

Neuroscience Perspective

Emerging evidence suggests that meditation-based interventions may influence neuroplasticity, autonomic regulation, stress hormone pathways, inflammatory responses, and brain networks associated with attention, emotional processing, and self-regulation.

SKT Meditation Healing Exercise may support healing by promoting parasympathetic activation, reducing physiological stress responses, and enhancing mind–body integration. SKT Meditation Healing Exercise represents a practical, culturally adaptable, and sustainable approach for promoting health, healing, and quality of life throughout the lifespan. Its integration into healthcare, nursing practice, community health promotion, and healthy aging programs may contribute to holistic well-being and support global efforts toward preventive and person-centered care.

Keywords: SKT Meditation Healing Exercise, healing, quality of life, healthy aging, mindfulness, autonomic balance, neuroplasticity, holistic health, self-healing, lifespan.
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Professor Dr. Somporn Kantharadussadee Triamchaisri is an acclaimed Thai neuroscientist, public health nurse, and the developer of the clinically proven **SKT Meditation Therapy**. She holds a Ph.D. in Neuroscience and a Master's in Biostatistics from Mahidol University, supplemented by postdoctoral research at the University of California, San Francisco (UCSF). Over a four-decade career, including her tenure as an Associate Professor at Mahidol University, she has specialized in bridging neurophysiological principles with holistic alternative therapies to manage chronic conditions like hypertension, pre-diabetes, and complications in palliative care. Currently, she operates internationally as a Full Professor within the Faculty of Health Sciences at the **University of Cuu Long** in Vietnam, where she continues to publish breakthrough public health research, lead community health interventions, and expand cross-border mindfulness-healing programs.



SS18

**Assoc. Prof. Chailerd Pichitpornchai,
M.D., Ph.D.***Faculty of Medicine Siriraj Hospital, Mahidol University,
Thailand*

The Future Brain: How Artificial Intelligence is Reshaping Neuroscience in the Next Decade

Abstract:

Artificial Intelligence (AI) is rapidly transforming neuroscience by enabling researchers and clinicians to analyze complex datasets and uncover patterns that were previously difficult to detect. Advances in machine learning, deep learning, generative AI, and multimodal data analytics are accelerating discoveries across basic, translational, and clinical neuroscience. As neuroscience data continue to expand – from neuroimaging and electrophysiological recordings to behavioral, cognitive, and digital health data – AI has become an essential tool for generating actionable knowledge and supporting evidence-based decision making.

This lecture will explore how AI is reshaping neuroscience research and clinical practice and how these developments may influence the field over the next decade. Key topics include AI applications in neuroimaging, neurophysiology, cognitive neuroscience, brain-behavior research, and neurological disorders. Particular emphasis will be placed on digital biomarkers derived from wearable sensors, mobile devices, and continuous monitoring technologies that enable real-time assessment of brain health and neurological function in everyday environments.

The lecture will also highlight the growing role of AI in forecasting neurological outcomes, predicting disease progression, identifying individuals at risk of cognitive decline, and supporting personalized interventions. By integrating longitudinal data from wearable devices, physiological sensors, clinical records, and behavioral measures, AI systems are increasingly moving neuroscience beyond retrospective analysis toward predictive and preventive brain health. Emerging developments in foundation models, large multimodal models, and brain-computer interfaces will also be discussed as examples of technologies that may further transform neuroscience research and healthcare delivery.

Important challenges related to data quality, interpretability, algorithmic bias, privacy, ethics, and governance will also be addressed. By examining current innovations and future opportunities, this lecture aims to provide a forward-looking perspective on how AI may transform neuroscience from a predominantly descriptive science into a predictive, preventive, and precision-oriented discipline, ultimately contributing to improved brain health and quality of life across the lifespan.

Associate Professor Dr. Chailerd Pichitpornchai, MD, PhD, is an esteemed neurophysiologist, medical informatician, and educator serving in the Department of Physiology at the Faculty of Medicine Siriraj Hospital, Mahidol University. An alumnus of Mahidol University and Monash University with an M.D. with Honors and a Ph.D. in



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Medicine, his distinguished career spans significant academic leadership roles, including former President of Sukhothai Thammathirat Open University and Director of Mahidol University's Institute for Innovative Learning. Holding dual Principal and Senior Fellow recognitions from Advance HE alongside specialized expertise across cognitive neuroscience, medical ICT, higher education quality assurance, and public administration, Dr. Pichitpornchai bridges advanced medical science, instructional technology, and strategic institutional governance.



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Scientific Session V: Neurotechnology



SS19

Asst. Prof. Sirawaj Itthipuripat, Ph.D.*Learning Institute, King Mongkut University of Technology
Thonburi, Thailand*

A Model-Based EEG Framework for Investigating Attention Deficits in Mild Cognitive Impairment

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Technology Thonburi, Bangkok, Thailand*

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Abstract:

Selective Attention is a fundamental cognitive process that enables us to prioritize relevant information in a complex environment. Changes in attentional function occur across the lifespan and are often associated with aging and neurological disorders. Understanding how attention is represented in the brain is therefore essential for advancing both basic neuroscience and clinical applications.

In this talk, I will present a model-based framework that combines scalp electroencephalography (EEG) with machine learning to quantify the neural dynamics of selective attention with high temporal resolution. By applying an inverted encoding model (IEM) to alpha-band EEG activity, we can reconstruct moment-to-moment neural representations of attentional focus and characterize their strength, precision, and timing. I will discuss how this approach provides new insights into the neural mechanisms underlying attention in healthy individuals across adulthood, as well as its potential to detect subtle alterations associated with cognitive decline in aging society, particularly in patients with Mild Cognitive Impairment. These findings demonstrate the value of computational modeling for extracting meaningful neural signatures from non-invasive brain recordings and highlight the potential of EEG-based biomarkers for studying cognitive function in both research and clinical settings.

Finally, I will outline future directions for applying this framework to investigate attention across diverse populations and neurological conditions, with the goal of improving our understanding of brain function and supporting the development of objective, accessible tools for cognitive assessment.

Asst. Prof. Sirawaj Itthipuripat, Ph.D., is the Director of the Neuroscience Center for Research and Innovation at King Mongkut's University of Technology Thonburi (KMUTT), Thailand. He holds a Ph.D. in Neurosciences from UC San Diego and was recognized by FORBES Asia 30 Under 30. His research combines EEG, neuroimaging, brain stimulation, and computational methods to investigate cognitive mechanisms—including selective attention, learning, and memory—across the human lifespan. Dr. Sirawaj actively leads major initiatives in neurotechnology, computational neuroscience, and preventive cognitive health for aging populations.



SS20

Sumeth Klomchitcharoen. Ph.D.*Institute of Molecular Biosciences, Mahidol University,
Thailand***Toward Translational NeuroAI & Neuroengineering for
Human Restoration***¹Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol
University, Nakhon Pathom 73170, Thailand***Corresponding author. Email: sumeth.klo@mahidol.ac.th***Abstract:**

Human restoration is one of the most tremendous hindrances to a neuroscience breakthrough. For decades, the aspiration to restore lost brain function has arisen from human perseverance and tenacity, driving its feasibility. Neurotechnology lies beyond the understanding of the human brain, as it refers to how the rudimentary foundations of neuroscience translate into the fruitful reality of practical, applicable apparatus. The interdisciplinary field of neuroscience, artificial intelligence, and neuroengineering has accelerated the advancement of intelligent neurotechnologies capable of decoding neural activity, identifying disease-specific biomarkers, and restoring lost neurological function through technology-driven approaches. This remarkable paradigm has expanded the role of neurotechnology beyond fundamental scientific discovery toward clinically translatable solutions that improve disability, diagnosis, rehabilitation, and quality of life. Representative advances encompass brain-computer interface technologies for spinal cord injury patients, fundamental investigations of neural mechanisms in autism spectrum disorder (ASD), AI-assisted EEG biomarkers for the early detection of ASD and major depressive disorder (MDD), and emerging intelligent neural systems that broaden the future landscape of adaptive human restoration. The paramount goal of creating integrated neurotechnology ecosystems capable of ongoing adaptation and customised intervention is further demonstrated by new developments in intelligent neural systems, neuromorphic technology, organoid-inspired computing, brain-humanoid interface, and adaptive bioelectronic technologies. Ultimately, these ideas will set in motion the powerful potential of translational NeuroAI and neuroengineering to bridge fundamental neuroscience with real-world deployable technologies, paving the way for enhanced well-being, independence, diagnosis, rehabilitation, and human restoration.

Keywords: Brain-computer Interface, NeuroAI, Neuroengineering, Neurotechnology

Dr. Sumeth Klomchitcharoen is a Lecturer at the Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Thailand. He earned his Ph.D. in Biomedical Engineering from Mahidol University and completed his postdoctoral fellowship at Mahidol's Brain-Computer Interface (BCI) Lab. His multidisciplinary research integrates microelectrode-array organoid-computer interfaces, non-invasive BCIs, neuromorphic computing, and space biology – including neurophysiological studies on spaceflight gravity and radiation. An award-winning innovator, Dr. Sumeth led Thailand's BCI team at CYBATHLON 2024 and won the National James Dyson Award.



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POSTER PRESENTATION

P001

HYDROXYCHAVICOL ATTENUATES METHAMPHETAMINE-INDUCED ALZHEIMER'S DISEASE- LIKE NEURODEGENERATION VIA MODULATION OF NF- κB/NRF2 SIGNALING

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Abstract:

Background: Methamphetamine (METH) abuse is increasingly associated with neurotoxicity, cognitive decline and Alzheimer's disease (AD)-like neuropathology. Chronic METH exposure disrupts oxidative balance and promotes neuroinflammation through dysregulation of the nuclear factor kappa B (NF-κB) and nuclear factor erythroid 2-related factor 2 (Nrf2) signalling pathways. Hydroxychavicol, a bioactive phenolic compound derived from Piper betle, possesses potent antioxidant and anti-inflammatory properties; however, its neuroprotective role against METH-induced AD-like pathology remains unclear.

Aim(s): This study aimed to investigate the neuroprotective effects of hydroxychavicol on behavioural, biochemical and molecular alterations in a METH-induced neurodegeneration rat model.

Methods: A total of 40 male Sprague-Dawley rats were randomly divided into five groups: control, METH-administration and METH combined with low (7.5mg/kg), medium (15mg/kg) and high-dose (30mg/kg) hydroxychavicol groups (n = 8 per group). METH dependence was induced via subcutaneous administration (2 mg/kg, twice daily) for 14 days. Behavioural assessments included withdrawal severity, anxiety-like behaviour using the Elevated Plus Maze, depressive-like behaviour using the Forced Swim Test and cognitive performance using Novel Object Recognition and Morris Water Maze tests. Brain tissues were subsequently analysed for oxidative stress markers, pro-inflammatory cytokines, AD-related biomarkers and signalling mediators involving NF-κB and Nrf2 pathways.

Results: 30mg/kg of hydroxychavicol treatment significantly improved cognitive performance and reduced anxiety-, depressive-like and withdrawal-related behaviours compared to the METH-administered group. Biochemical analyses demonstrated significant reductions in lipid peroxidation and pro-inflammatory cytokine levels, alongside enhanced endogenous antioxidant activity. Molecular findings revealed suppression of NF-κB activation and upregulation of Nrf2 signalling, indicating restoration of redox homeostasis and attenuation of neuroinflammatory responses. Furthermore, AD-associated biomarkers were significantly reduced following hydroxychavicol treatment.

Conclusion: Hydroxychavicol exerts significant neuroprotective effects against METH-induced AD-like neurodegeneration by modulation oxidative stress and neuroinflammatory pathways, primarily through NF- κ B inhibition and Nrf2 activation. These findings support the therapeutic potential of hydroxychavicol as a natural neuroprotective candidate for substance abuse-associated neurodegenerative disorders.

Keywords: Hydroxychavicol; Methamphetamine; Alzheimer's disease; Neuroinflammation; NF- κ B/Nrf2 signalling

P002

EXPLORING THE LINK BETWEEN NIGHTMARES, SLEEP PARAMETERS, AND PREFRONTAL CORTEX DYSFUNCTION IN MILD COGNITIVE IMPAIRMENT (MCI): PRELIMINARY FINDINGS FROM A PILOT STUDY

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Abstract:

Background: Sleep disturbances, including nightmares, have been proposed as potential early indicators of cognitive decline and may be associated with prefrontal cortex (PFC) dysfunction. Our previous pilot study demonstrated the feasibility of sleep-related assessments and an Emotional Go/No-Go paradigm combined with functional near-infrared spectroscopy (fNIRS). Building upon these methodological findings, the emotional Go/No-Go paradigm was refined to improve signal stability and reduce participant fatigue.

Aim(s): To evaluate the feasibility of a refined emotional Go/No-Go paradigm combined with fNIRS and to explore associations between sleep parameters and PFC activation as a preliminary step toward MCI research.

Methods: Eight healthy adults (5 females, 3 males; age 27.9 ± 5.1 years) completed the Pittsburgh Sleep Quality Index (PSQI), Perceived Stress Scale (PSS-10), Montreal Cognitive Assessment (MoCA), and a refined emotional Go/No-Go task during fNIRS recording. Task feasibility was evaluated using behavioral performance and fNIRS signal quality. Oxyhemoglobin (HbO) responses were extracted from the bilateral inferior frontal gyrus (IFG). Associations between questionnaire measures and IFG activation were assessed using Spearman correlations.

Results: Participants showed excellent task performance, with Go accuracy of $99.4 \pm 0.6\%$, No-Go accuracy of $99.3 \pm 1.3\%$, and reaction time of 491 ± 66 ms. fNIRS captured task-related hemodynamic responses in the bilateral IFG (mean HbO = 0.033 ± 0.165 μ M). No significant associations were observed. However, a tendency toward lower IFG activation was observed with higher PSQI scores ($\rho = -0.599$, $p = 0.124$). A weaker pattern was observed for perceived stress ($\rho = -0.455$, $p = 0.258$), whereas no apparent pattern was observed for MoCA scores ($\rho = -0.050$, $p = 0.936$).

Conclusion: The refined emotional Go/No-Go paradigm demonstrated high feasibility, excellent behavioral compliance, and successful acquisition of task-related fNIRS data. Preliminary findings suggest a potential link between poorer sleep quality and reduced IFG activation during emotional inhibitory control. These findings support application of the refined protocol in future MCI studies.

Keywords: Nightmares, Sleep Quality, fNIRS, Prefrontal Cortex, Mild cognitive impairment

Background

Mild cognitive impairment (MCI) is considered an intermediate stage between normal aging and dementia, characterized by measurable cognitive decline despite relatively preserved daily functioning. Individuals with MCI are at substantially increased risk of progressing to dementia, making early identification of cognitive vulnerability an important research priority (Roberts & Knopman, 2013). As global populations continue to age, the prevalence of MCI and dementia is expected to increase considerably, creating significant personal, social, and economic burdens. Consequently, there is growing interest in identifying accessible behavioral and physiological markers that may facilitate earlier detection of cognitive decline before irreversible neurodegenerative changes become established.

Among the potential markers of cognitive vulnerability, nightmares have recently attracted increasing attention. Nightmares are vivid and distressing dreams that typically occur during rapid eye movement (REM) sleep and are often accompanied by abrupt awakenings and intense negative emotions (American Academy of Sleep Medicine, 2014). Although traditionally viewed as a sleep-related symptom, accumulating evidence suggests that frequent nightmares may be associated with cognitive dysfunction and neurodegenerative processes. Several longitudinal studies have reported that individuals experiencing frequent nightmares are at greater risk of future cognitive decline and dementia, suggesting that nightmare experiences may precede the onset of clinically apparent neurodegenerative disorders (Otaiku, 2022). These findings raise the possibility that nightmares could serve as a simple, non-invasive behavioral marker for identifying individuals at risk of cognitive impairment.

One proposed mechanism underlying the association between nightmares and cognitive decline involves dysfunction of the prefrontal cortex (PFC). The PFC plays a critical role in executive function, emotional regulation, and top-down control of limbic structures involved in threat processing and emotional memory (Ochsner & Gross, 2005; Passingham et al., 2012). Emerging evidence suggests that frequent nightmares may be associated with alterations in frontal brain activity and impaired emotional regulation processes, potentially reflecting reduced prefrontal control over affective responses (Marquis et al., 2019; Nadorff et al., 2019). Similar abnormalities in prefrontal and executive-control networks have also been reported in individuals with MCI, where reduced neural efficiency and disrupted executive functioning are associated with cognitive decline (Liu et al., 2021; Xu et al., 2020). These findings suggest that nightmares and cognitive impairment may share common neurobiological pathways involving prefrontal dysfunction.

Because the prefrontal cortex is critically involved in executive control, alterations in PFC function may be reflected in impaired inhibitory processing. Inhibitory control refers to the ability to suppress inappropriate or unwanted responses in accordance with current goals and is considered a core component of executive function. Among the cortical regions involved in this process, the inferior frontal gyrus (IFG), particularly within the right hemisphere, has been consistently identified as a key neural substrate of response inhibition (Aron et al., 2014). Consequently, assessment of inhibitory control may provide a sensitive approach for detecting subtle changes in prefrontal function. The Emotional Go/No-Go paradigm is widely used to evaluate inhibitory control under emotionally salient conditions by requiring participants to respond selectively to target stimuli while withholding responses to non-target stimuli. Neural evidence from Go/No-Go paradigms consistently implicates the inferior frontal gyrus and related prefrontal regions in inhibitory processing (Simmonds et al., 2008). Compared with traditional

Go/No-Go tasks, the emotional variant engages both executive and affective processing systems, providing a more ecologically relevant measure of cognitive control.

Functional near-infrared spectroscopy (fNIRS) provides a non-invasive method for investigating prefrontal function by measuring task-related changes in oxygenated and deoxygenated hemoglobin concentrations. Due to its portability, safety, and tolerance to movement, fNIRS has become increasingly valuable for studying executive function in both healthy and clinical populations (Ferrari & Quaresima, 2012; Pinti et al., 2020). Despite growing evidence linking nightmares, sleep disturbances, and prefrontal dysfunction, these factors have largely been investigated independently. Furthermore, no study has comprehensively examined the relationships among nightmare, sleep-related measures, and prefrontal activation using an fNIRS-compatible emotional inhibitory control paradigm. As part of an ongoing research project investigating the relationships among nightmares, sleep parameters, prefrontal cortex dysfunction, and mild cognitive impairment, we previously developed an Emotional Go/No-Go paradigm combined with fNIRS recording. Although the initial protocol demonstrated feasibility, its relatively long duration may have increased participant burden and contributed to variability in hemodynamic responses. Therefore, the paradigm was refined into a shorter block-design version to improve practicality, reduce fatigue, and optimize signal acquisition. The present pilot study aimed to evaluate the feasibility of the refined Emotional Go/No-Go paradigm combined with fNIRS and to explore preliminary associations among sleep quality, perceived stress, cognitive function, and task-related prefrontal cortical activation in healthy adults prior to its application in future MCI studies.

Materials and Methods

Participants

Eight healthy adults (5 females and 3 males; mean age = 27.9 ± 5.1 years; mean BMI = 22.5 ± 5.8 kg/m²) participated in this pilot study. Participants were recruited through convenience sampling and were required to be right-handed, have normal or corrected-to-normal vision, and report no history of neurological or psychiatric disorders. The study protocol was approved by the Mahidol University Central Institutional Review Board (MU-CIRB; Protocol Ref. No. MU-CIRB 2026/359.0406). Written informed consent was obtained from all participants prior to data collection, and all procedures were conducted in accordance with the Declaration of Helsinki.

Questionnaires

Prior to the experimental session, participants completed a battery of questionnaires assessing sleep quality, perceived stress, and cognitive function. Subjective sleep quality was assessed using the Thai-translated version of the Pittsburgh Sleep Quality Index (PSQI), which evaluates sleep quality and sleep-related disturbances over the previous month (Sitasuwan et al., 2014). Perceived stress was measured using the Thai-translated version of the 10-item Perceived Stress Scale (PSS-10), which assesses the degree to which individuals perceive their lives as stressful (Wongpakaran & Wongpakaran, 2010). Global cognitive function was evaluated using the Thai-translated version of the Montreal Cognitive Assessment (MoCA), a cognitive screening instrument assessing multiple domains including attention, memory, language, visuospatial ability, and executive function (Hemrungronj et al., 2021). Following questionnaire completion, participants underwent fNIRS recording while performing the Emotional Go/No-Go task.

Emotional Go/No-Go Task

Following completion of the questionnaires, participants underwent fNIRS recording while performing a refined Emotional Go/No-Go task

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designed to assess emotional inhibitory control. The task was programmed and presented using PsychoPy (version 2025.2.4beta). Prior to task performance, participants completed 3-min eyes-open (EO) and 3-min eyes-closed (EC) resting-state recordings. The experimental paradigm employed a block-design structure consisting of 12 active blocks and 12 rest blocks. During each active block, participants completed 10 facial-expression trials comprising 7 Happy-Go and 3 Angry No-Go trials. Trial order was randomized separately within each block while maintaining the 7:3 ratio. For each trial, a facial image was randomly selected with replacement from the corresponding emotion category, meaning that the same image could be presented more than once. Because the trial sequence and images were randomized for each experimental run, they could differ across blocks and participants. Participants were instructed to respond as quickly and accurately as possible to Happy-Go stimuli by pressing the space bar while withholding responses to Angry No-Go stimuli. Emotional stimuli consisted of photographs depicting happy and angry facial expressions obtained specifically for use in the study. Each trial consisted of a fixation cross presented for 500 ms, followed by a facial stimulus presented for 800 ms and an inter-trial interval of 700 ms, resulting in an active block duration of 20 s. Active blocks were alternated with 20-s rest periods during which a fixation cross was displayed at the center of the screen. The entire task consisted of 12 active-rest cycles and lasted approximately 8 min, excluding task instructions. Behavioral performance was evaluated using Go accuracy, No-Go accuracy, and reaction time (RT), which were automatically recorded by PsychoPy during task performance.

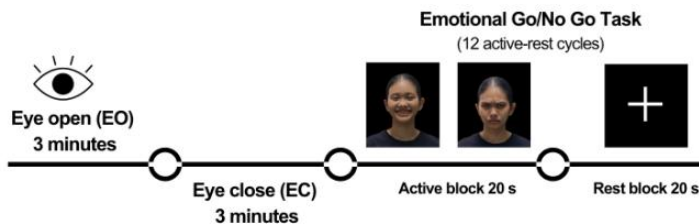


Figure 1. Experimental paradigm of the Emotional Go/No-Go protocol, consisting of 3-min eyes-open (EO), 3-min eyes-closed (EC), and 12 active-rest cycles.

fNIRS Data Acquisition and Processing

Prefrontal cortical activity was recorded using a NIRSport2 functional near-infrared spectroscopy (fNIRS) system (NIRx Medical Technologies LLC, Glen Head, NY, USA). Data were acquired at wavelengths of 760 and 850 nm using a prefrontal cortex montage covering regions associated with executive function and inhibitory control. Optodes were positioned according to the international 10–20 system. The fNIRS recording was initiated immediately before the 3-min eyes-open resting-state condition and continued without interruption throughout the eyes-open, eyes-closed, and Emotional Go/No-Go task conditions. Task onset was manually marked in the fNIRS recording, and the timing of the subsequent active and rest blocks was aligned according to the predefined PsychoPy task sequence. Raw light intensity signals were processed using Homer3 software. Signals were converted to optical density, corrected for motion artifacts, and transformed into concentration changes in oxygenated hemoglobin (HbO) and deoxygenated hemoglobin (HbR) using the modified Beer–Lambert law. Temporal filtering was applied to reduce physiological and instrumental noise. Task-related hemodynamic responses were estimated using block averaging based on the Emotional Go/No-Go paradigm. Because HbO is commonly used as an indicator of task-evoked cortical activation, subsequent analyses focused

on HbO responses. Mean HbO values were calculated within the 5–15 s post-stimulus window. The primary fNIRS outcome was bilateral IFG activation, calculated by averaging HbO responses from predefined left and right IFG channels.

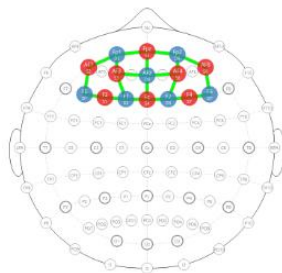


Figure 2. Prefrontal fNIRS optode configuration based on the international 10–20 system.

Statistical Analysis

Descriptive statistics were calculated for demographic characteristics, questionnaire scores, behavioral performance, and fNIRS outcomes. Data are presented as mean $\pm$ standard deviation (SD). Associations between sleep quality (PSQI), perceived stress (PSS-10), cognitive performance (MoCA), and task-related bilateral IFG activation were examined using Spearman's rank correlation coefficient. Statistical analyses were performed using R software (version 4.4.1), and statistical significance was set at $p < 0.05$.

Results (Figure and/or Table)

Behavioral Performance and Questionnaire Measures

Participants performed the Emotional Go/No-Go task with high accuracy and compliance. Mean Go accuracy was $99.4 \pm 0.6\%$, while mean No-Go accuracy was $99.3 \pm 1.3\%$. Mean reaction time was 489 ± 66 ms. Despite the healthy status of the sample, PSQI scores showed variability (mean = 6.50 ± 4.31), suggesting differences in subjective sleep quality across participants. Sex-based subgroup analysis showed no significant differences in questionnaire measures, behavioral performance, or bilateral IFG HbO responses between male and female participants (all $p > 0.05$). Descriptive statistics for questionnaire measures, behavioral performance, and fNIRS outcomes are summarized in Table 1.

Table 1. Descriptive statistics for questionnaire, behavioral, and fNIRS measures (N = 8).

Measure	Mean $\pm$ SD
PSQI Total Score	6.50 ± 4.31
PSS-10 Total Score	15.63 ± 6.00
MoCA Total Score	27.50 ± 1.41
Go Accuracy (%)	99.4 ± 0.6
No-Go Accuracy (%)	99.3 ± 1.3
Reaction Time (ms)	489 ± 66
Bilateral IFG HbO Response (μM)	0.033 ± 0.165

Task-Related Hemodynamic Responses

Task-related fNIRS recordings revealed measurable hemodynamic responses during the Emotional Go/No-Go task. The representative hemodynamic response

function (HRF) demonstrated an increase in oxyhemoglobin (HbO) following task onset, peaking approximately 6–8 s after stimulus presentation. Consistent with the HRF findings, the HbO topographic map at 10 s demonstrated task-related changes in prefrontal activation during task performance (Figure 3).

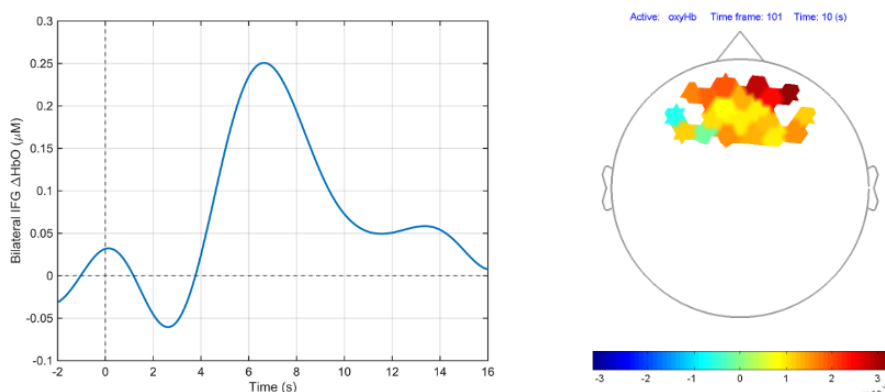


Figure 3. Representative fNIRS findings during the Emotional Go/No-Go task. (Left) Bilateral IFG HbO hemodynamic response function (HRF). (Right) HbO topographic map at 10 s after task onset showing PFC activation.

Associations Between Sleep Measures and IFG Activation

Correlation analyses did not reveal statistically significant associations between questionnaire measures and bilateral IFG activation. Nevertheless, the relationship between PSQI scores and IFG HbO responses showed a negative trend ($\rho = -0.599$, $p = 0.124$) (Figure 4). Associations with perceived stress (PSS-10) and cognitive performance (MoCA) were weaker ($\rho = -0.455$, $p = 0.258$ and $\rho = -0.050$, $p = 0.936$, respectively).

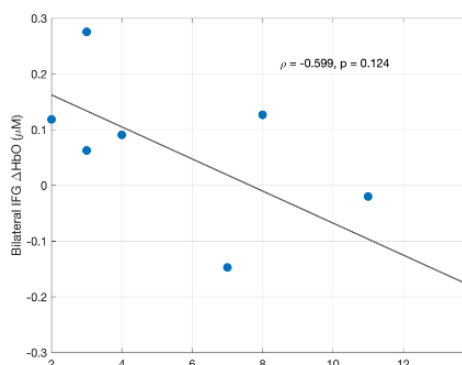


Figure 4. Scatter plot showing the relationship between PSQI total score and bilateral IFG HbO responses. A negative trend was observed, although the association did not reach statistical significance (Spearman's $\rho = -0.599$, $p = 0.124$).

Discussion and Conclusion

This pilot study demonstrated the feasibility of a refined Emotional Go/No-Go paradigm combined with fNIRS for assessing prefrontal activation during emotional

inhibitory control. Participants showed excellent behavioral performance, with near-ceiling Go and No-Go accuracies and stable reaction times, indicating high task compliance and successful completion of the protocol. In addition, task-related hemodynamic responses were successfully detected during the Emotional Go/No-Go task. The representative HRF demonstrated the expected increase in HbO following task onset, whereas topographic mapping revealed task-related changes across prefrontal regions. Together, these findings suggest that the refined paradigm successfully elicited measurable cortical responses and is suitable for investigating emotional inhibitory control using fNIRS.

Although no statistically significant associations were observed between questionnaire measures and bilateral IFG activation, a negative trend was observed between PSQI scores and IFG HbO responses. Participants with poorer subjective sleep quality tended to exhibit lower task-related IFG activation, consistent with previous reports linking sleep disturbances to altered prefrontal functioning. Sleep disturbances such as insomnia have been associated with prefrontal hypoactivation, while sleep deprivation has been linked to impairments in cognition, executive function, and emotional processing (Altena et al., 2008; Killgore, 2010). While the present findings support the theoretical framework underlying the present study and suggest that sleep may influence prefrontal activity during emotional inhibitory control, the absence of statistically significant associations may partly be explained by the limited sample size and the relatively homogeneous characteristics of the healthy young participants. In addition, near-ceiling behavioral performance and limited variability in cognitive measures may have reduced the ability to detect significant relationships between questionnaire measures and task-related brain activation.

The potential relationship between sleep quality and IFG activation may therefore be particularly relevant in the context of emotional inhibitory control, as sleep-related alterations in prefrontal function may affect both executive control and emotional regulation. Previous studies using Go/No-Go paradigms have demonstrated the involvement of prefrontal regions, including the IFG, during response inhibition and executive processing (Aron et al., 2014; Simmonds et al., 2008). Emotional Go/No-Go paradigms additionally combine affective stimulus processing with inhibitory control and engage neural systems involved in emotion regulation and cognitive control (Hare et al., 2005). Moreover, response-inhibition paradigms more broadly involve right prefrontal and inferior frontal regions associated with action withholding and inhibitory processing (Sebastian et al., 2013). Because emotional inhibitory control requires the integration of both executive and affective processes, the present paradigm may be particularly sensitive to sleep-related alterations in prefrontal function.

Such alterations in prefrontal function may be particularly relevant in populations at risk for cognitive decline, such as individuals with mild cognitive impairment (MCI), in whom both prefrontal dysfunction and sleep disturbances have frequently been reported. Previous fNIRS work in MCI has demonstrated altered prefrontal hemodynamic responses during executive function tasks, including N-back, Stroop, and verbal fluency paradigms, suggesting that PFC activation patterns may serve as potential biomarkers for early MCI detection (Yang et al., 2019). These findings support the use of fNIRS as a non-invasive tool for detecting subtle functional changes in prefrontal networks that may not be fully captured by behavioral performance alone.

Sleep disturbance may represent one potential contributor to these prefrontal alterations, as sleep problems are common among individuals with MCI and may accompany progressive cognitive decline. Sleep disturbances in MCI commonly include poor sleep quality, increased sleep fragmentation, prolonged sleep latency, reduced sleep

efficiency, and alterations in sleep architecture (Cassidy-Eagle & Siebern, 2017). Disturbed sleep has also been linked to cognitive decline and dementia risk, supporting the relevance of sleep assessment in populations at risk for neurodegenerative disease (Wennberg et al., 2017). In addition, disturbing dreams and nightmares have recently been proposed as potential early indicators of cognitive decline. A prospective cohort study reported that higher distressing dream frequency was associated with increased risk of cognitive decline and incident dementia in middle-aged and older adults (Otaiku, 2022). Nightmare symptoms may therefore reflect dysfunction of neural systems involved in emotional regulation, memory processing, and executive control.

Taken together, these findings provide a rationale for examining the relationship between sleep quality, nightmare symptoms, emotional inhibitory control, and prefrontal activation in individuals at risk for cognitive decline. Several limitations should be acknowledged. First, the small sample size limits statistical power and the generalizability of the findings. Second, the present study was conducted in healthy young adults rather than individuals with MCI, limiting direct clinical interpretation. Third, only prefrontal activation was examined, whereas additional cortical and subcortical regions may contribute to emotional inhibitory control and sleep-related cognitive processes.

Overall, the present findings support the feasibility of using a refined Emotional Go/No-Go paradigm combined with fNIRS to investigate emotional inhibitory control and prefrontal function. The protocol provided stable behavioral performance, measurable hemodynamic responses, and reduced task burden compared with the previous version. Although this pilot study was conducted in healthy young adults, the observed relationship between sleep quality and IFG activation, together with previous evidence of prefrontal dysfunction and sleep disturbances in MCI, supports the application of this refined paradigm in future studies investigating whether sleep disturbances and nightmare symptoms are associated with altered IFG activation during emotional inhibitory control tasks in individuals with mild cognitive impairment. Such investigations may improve our understanding of the relationship between sleep disturbances, prefrontal dysfunction, and cognitive decline and may contribute to the identification of early markers of neurodegenerative disease.

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The authors would like to thank Prof. Nuanchan Chutabhakdikul for assistance with the fNIRS equipment and Miss Fahsai Kaewpikul for photographing the emotional facial-expression stimuli used in the Emotional Go/No-Go task. The authors also gratefully acknowledge Assoc. Prof. Solapat Hemrungronj for the Thai version of the Montreal Cognitive Assessment (MoCA), Assoc. Prof. Tulaya Sitasuwan for the Thai version of the Pittsburgh Sleep Quality Index (PSQI), and Prof. Tinakon Wongpakaran and Prof. Nahathai Wongpakaran for the Thai version of the Perceived Stress Scale (PSS-10). Finally, the authors thank all participants for their time and cooperation.

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P003

THE DEVELOPMENT OF THE ASIAN FACIAL EMOTIONAL GO/NO-GO TASK TO STUDY THE RELATIONSHIP BETWEEN INHIBITORY CONTROL AND RUMINATION

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Abstract:

Background: Rumination has been described as a passive dwelling on negative emotions in individuals experiencing depressive symptoms and was considered a significant cognitive risk factor for depression. It has been described as a passive dwelling on negative emotions in individuals experiencing depressive symptoms. Although its relationship with inhibitory control remains controversial, rumination has typically been assessed through self-report measures, which alone do not provide sufficient behavioral evidence.

Aim: The present study aimed to develop and validate a culturally adapted facial emotional Go/No-Go (GNG) task to investigate the relationship between inhibitory control and rumination in an Asian context.

Methods: The GNG task is a classic task for examining inhibitory control and impulsive behavior by presenting stimuli in random order. A culturally adapted GNG task was developed, comprising four stimulus types: button-press responses to female faces (f-Go), button-press responses to male faces (m-Go), response inhibition to female faces (f-No-Go), and response inhibition to male faces (m-No-Go). The task consisted of two phases: participants responded to smiling faces in the first phase and angry faces in the second, with 192 Go stimuli and 48 No-Go stimuli per phase. A 5-minute break was provided between phases, and both response time and accuracy were recorded.

Results: Behavioral accuracy analysis showed that Go-trial accuracy did not differ between smile-cued and angry-cued trials, $t(4) = 1.19$, $p = .300$, whereas Inhibit accuracy was significantly higher for angry-cued than smile-cued No-Go trials, $t(4) = 5.10$, $p = .007$, $d_z = 2.28$. At Pz, Response-related P3 amplitude (individually scored) was significantly larger for smile-cued than angry-cued Go trials, $t(4) = 2.92$, $p = .043$, $d_z = 1.30$. In contrast, Inhibit-related P3 latency at FCz was shorter for angry-cued than smile-cued No-Go trials, $t(4) = -2.86$, $p = .046$, $d_z = -1.28$, converging with the behavioral advantage for angry-cued inhibition.

Conclusion: The culturally adapted Asian Facial Emotional Go/No-Go task appears to be a feasible behavioral paradigm for assessing inhibitory control in a Thai population, with ERP responses largely consistent across facial expression cues, supporting its use as a foundation for future research examining the relationship between inhibitory control and rumination in Asian populations.

Keywords: Inhibition, Go/No-Go Task, Rumination, Electroencephalogram (EEG)

Background

Rumination has been described as a passive dwelling on negative emotions, particularly prevalent in depressed individuals (Nolen-Hoeksema, 1991). Previous studies strongly suggest that rumination is associated with impairments in cognitive flexibility and difficulty in regulating negative thoughts. Furthermore, psychopathology has been found to develop in young adolescents with a history of rumination (Corrieri et al., 2014). Rumination is considered a significant risk factor for depression. However, the relationship between rumination and cognitive control remains controversial (Aker et al., 2014; Park et al., 2024; Zetsche et al., 2018). Rumination has typically been assessed through self-report measures, and sufficient behavioral evidence of its relationship with cognitive control remains limited, underscoring the need for objective, task-based paradigms capable of capturing inhibitory control processes in the context of emotional information.

The Emotional Go/No-Go (GNG) Task is a classic task for examining inhibitory control and impulsive behavior by presenting facial emotional stimuli in random order. This task is effective in evaluating the impact of emotional stimuli on conscious behavioral responses. Beringer et al. (2022) reported that healthy participants tend to make more errors when trying to inhibit the expression of joy or smiling compared to fear or disgust. In other words, positive emotions are harder to inhibit than negative emotions during the task. In a subsequent experiment, participants engaged in face-to-face interactions. The results showed that direct eye contact increased the rate of inhibition when an interlocutor made eye contact and smiled. The social pull is so strong that participants struggled intensely to maintain a neutral face during No-Go trials, while negative expressions did not significantly increase error rates. The researchers theorize that this may be related to the social function of emotion. Positive emotions are a key to building social bonds and trigger the autonomic mimicry response, making it hard to suppress a smile (Hess & Fischer, 2013). However, in a rumination study, high levels of rumination were associated with higher error rates and diminished perceptual sensitivity. These individuals lack the flexibility to inhibit all emotions due to difficulty adapting (Whitmer & Banich, 2007). Despite the growing body of literature on emotional inhibitory control, the majority of existing GNG tasks have relied predominantly on Western facial stimuli, which may not adequately represent the emotional expressions of Asian populations. Cultural differences in emotional display rules and facial expressivity have been shown to influence both behavioral performance and neural responses during emotion categorization tasks (Matsumoto et al., 2008), suggesting that findings based on Western stimuli may not generalize to non-Western samples. Since rumination and its cognitive correlates are increasingly studied across diverse cultural contexts, the validity of task-based assessments depends on the cultural appropriateness of the stimuli employed. Therefore, the present study aimed to develop and validate a culturally adapted Asian facial emotional GNG task, and to examine whether trait rumination moderates inhibitory control performance in response to positive and negative emotional facial stimuli within this culturally representative paradigm.

Materials and Methods

The GNG task is a classic task for examining inhibitory control and impulsive behavior by presenting different stimuli randomly (Donders, 1969). At least two stimuli are presented on the screen, requiring a response (pressing the button: Go) and inhibiting responding (No-Go). This study will focus on social and emotional responding inhibition, and the task will be modified from Sun et al. (2020) using STIM2. Prior studies have examined the association between rumination and ERP indices of inhibitory control, though results have not been consistent across the literature. One study employing a

Go/NoGo vigilance paradigm to assess N2 and P3 components demonstrated that the reflective dimension of rumination played a moderating role in inhibitory functioning.

Asian facial emotion images were captured against a black background. The images were adjusted and cleaned of artifacts using Adobe Photoshop 2024 (Figure 1). The GNG task is a widely used measure of inhibitory control and impulsive behavior. The task was created using the program STIM2, in which participants were presented with four types of stimuli: a response condition with female faces (f-Go), a response condition with male faces (m-Go), a response-inhibition condition with female faces (f-No-Go), and a response-inhibition condition with male faces (m-No-Go). The task consisted of two phases, each comprising 240 trials. In the first phase, participants were presented with 192 Go stimuli (Smile-Go; 91 males, 91 females) and 48 No-Go stimuli (Angry-No-Go; 24 males, 24 females) and were instructed to respond to smiling faces. In the second phase, 192 Go stimuli (Angry-Go) and 48 No-Go stimuli (Smile-No-Go) were presented, with participants responding to angry faces. A 5-minute break was provided between phases. Each trial began with a fixation cross presented for 400 ms, followed by a randomly selected facial image displayed for 1,000 ms, and ended with a blank screen (Figure 2).

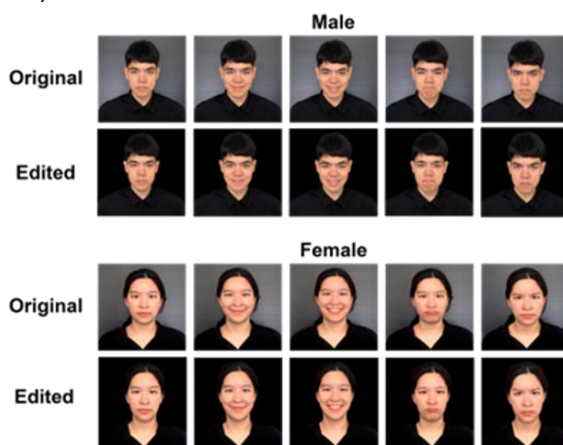


Figure 1 Edited Asian facial emotion image from original to artifact removal

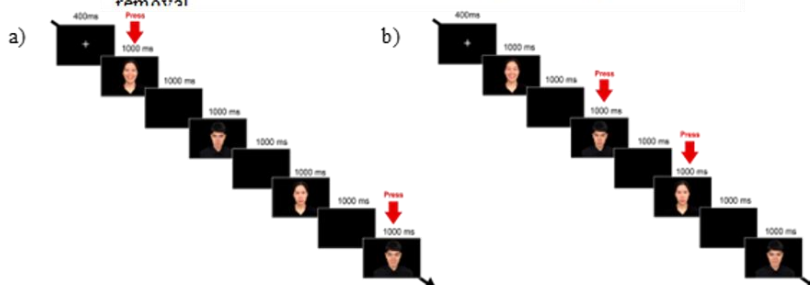


Figure 2 Emotional inhibition Go/No-Go Task; a) Phase1 GNG Task requiring responses to smile facial expression (Smile-Go) and b) Phase2 GNG Task requiring responses to an angry facial expression (Angry-Go)

Results

Behaviorally, Overall Go-trial (Response) accuracy was high and did not differ significantly between smile-cued (Phase 1: $M = 96.56\%$, $SD = 4.01$) and angry-cued (Phase 2: $M = 91.88\%$, $SD = 12.78$) trials, $t(4) = 1.19$, $p = .300$, $d_z = 0.53$, indicating that participants could reliably execute the required button-press response regardless of which facial expression cued it. In contrast, No-Go (Inhibit) trial accuracy differed significantly by cuing expression: angry-cued No-Go trials were inhibited more accurately ($M = 82.08\%$, $SD = 22.08$) than smile-cued No-Go trials ($M = 76.67\%$, $SD = 23.82$), $t(4) = 5.10$, $p = .007$, $d_z = 2.28$. Per-subject accuracy is shown in Table 1; one participant (FS) showed markedly lower inhibition accuracy on both No-Go conditions relative to the rest of the sample, contributing much of the observed variability given the small sample ($n = 5$).

Table 1. Per-subject and group accuracy (%) by trial type and phase.

Subject	Smile-Go (Phase 1)	Angry-No-Go (Phase 1)	Smile-No-Go (Phase 2)	Angry-Go (Phase 2)
1	98.44	95.83	93.75	98.44
2	98.44	43.75	35.42	98.96
3	89.58	85.42	79.17	69.27
4	96.88	97.92	91.67	94.27
5	99.48	87.50	83.33	98.44
Mean $\pm$ SD	96.56 $\pm$ 4.01	82.08 $\pm$ 22.08	76.67 $\pm$ 23.82	91.88 $\pm$ 12.78

This behavioral pattern converges with the ERP findings. Inhibit-related P3 latency at FCz was significantly shorter for angry-cued than smile-cued No-Go trials, $t(4) = -2.86$, $p = .046$, $d_z = -1.28$, suggesting that an angry facial expression facilitates faster response inhibition at the neurophysiological level, consistent with the behavioral inhibition advantage described above. At Pz, Response-related (Go-trial) P3 amplitude, quantified from individually scored peaks, was significantly larger for smile-cued than angry-cued Go trials ($M = 10.15 \mu V$, $SD = 3.57$ vs. $M = 4.47 \mu V$, $SD = 3.80$), $t(4) = 2.92$, $p = .043$, $d_z = 1.30$, even though Go-trial response accuracy itself did not differ between the two cuing expressions. Peak latency at Pz did not differ significantly between conditions (smile-cued: $M = 460.0$ ms, $SD = 123.6$; angry-cued: $M = 405.2$ ms, $SD = 34.8$), $t(4) = 0.83$, $p = .455$, $d_z = 0.37$; however, the markedly larger latency SD for smile-cued trials, which driven chiefly by one participant whose peak occurred at 680 ms, which indicates greater inter-individual latency jitter in this condition. This amplitude difference was less pronounced when measured from the grand-averaged waveform (Figure 3) than from individually scored peaks. The smile-cued grand-average P3 at Pz peaked at approximately $5.2 \mu V$ (~ 350 ms; Figure 3a), whereas the angry-cued grand-average P3 peaked at approximately $4.1 \mu V$ (~ 370 – 420 ms; Figure 3b), which a gap far smaller than the $5.68 \mu V$ difference obtained from individually scored peaks. The angry-cued grand-average peak ($4.1 \mu V$) closely matched its individually scored mean ($4.47 \mu V$), consistent with the tighter latency clustering in that condition ($SD = 34.8$ ms). By contrast, the smile-cued grand-average peak ($5.2 \mu V$) was roughly half its individually scored mean ($10.15 \mu V$), consistent with greater inter-individual latency jitter ($SD = 123.6$ ms) attenuating the peak under grand-averaging, since misaligned single-subject peaks partially cancel when waveforms are averaged together. Together, these results indicate that the emotional valence of the cue modulated both the amplitude and the inter-individual timing consistency of neural engagement during responding, without affecting overt Go-trial response accuracy. All other electrode, component, and measure comparisons were non-significant, and the significant ERP effects did not survive correction for multiple comparisons.

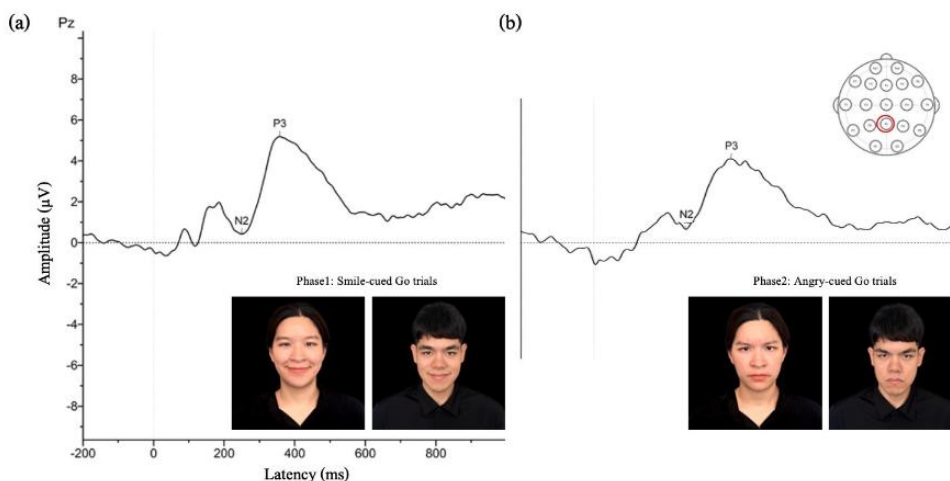


Figure 3. Grand-average ERP waveforms at Pz for Go trials. (a) Smile-cued Go trials (Phase 1), P3 peak $\approx 5.2 \mu\text{V}$. (b) Angry-cued Go trials (Phase 2), P3 peak $\approx 4.1 \mu\text{V}$.

Discussion

The necessity of developing a culturally adapted Asian facial set is underscored by established cross-cultural variations in display rules and facial expressivity, which substantially shape both behavioral performance and neural dynamics during emotion categorization and cognitive control tasks (Matsumoto et al., 2008). Cross-cultural studies confirm the presence of "other-race effects" and display-rule variations during facial emotion processing, demonstrating that standardized Western stimuli applied to non-Western cohorts can introduce confounding perceptual biases (Jiang et al., 2023). It was this gap that motivated the present study: to develop and validate a culturally adapted Asian Facial Emotional Go/No-Go (GNG) task, offering an objective behavioral and electrophysiological framework for evaluating emotional inhibitory control within an Asian demographic. Following the classic Go/No-Go logic of examining response inhibition and impulsive control through randomized stimulus conditions (Donders, 1969), the present paradigm replaced conventional Western facial sets with culturally congruent, artifact-free Asian facial stimuli within a Thai population, directly addressing these established cross-cultural variations and substantially enhancing ecological validity for neuroscientific research across Asian cohorts.

Both the behavioral and event-related potential (ERP) indices derived from this task demonstrate that emotional cue valence significantly modulates response inhibition performance and neural resource allocation. At the behavioral level, response execution accuracy remained high and comparable across both Go conditions, with participants achieving 96.56% accuracy for smile-cued trials and 91.88% accuracy for angry-cued trials, indicating that motor responses could be reliably executed regardless of the emotional expression cueing the trial. Response inhibition accuracy, by contrast, differed significantly by emotional valence: participants showed superior response suppression when withholding responses to angry facial cues (82.08%) compared to smiling facial cues (76.67%). This behavioral advantage during angry-cued inhibition converged directly with the electrophysiological findings, in which inhibit-related P3 latency at FCz was significantly shorter for angry-cued than smile-cued No-Go trials. Because frontocentral

No-Go P3 latency reflects the speed and efficiency of motor suppression processes, this shorter latency indicates accelerated neural processing when halting a motor command in response to threat-related social cues. Response-related P3 amplitude at Pz followed a complementary pattern, emerging significantly larger for smile-cued than angry-cued Go trials, which is indicating that positive emotional valence recruits heightened neural engagement during target response execution even though overt motor accuracy remained unchanged.

Together, these results align with existing literature showing that positive emotional expressions exert a powerful "social pull" that increases the difficulty of response inhibition: positive emotions foster autonomic mimicry and social bonding, making it inherently harder to suppress actions in response to smiling expressions (Hess & Fischer, 2013), and healthy individuals typically commit more errors inhibiting joyful expressions than negative or threat-related ones (Beringer et al., 2022). Angry facial expressions, conversely, function as immediate threat cues that trigger rapid cognitive vigilance and urgent behavioral stopping, which is consistent with both the faster No-Go P3 latency and the higher inhibition accuracy observed here for angry-cued trials. Integrating this kind of objective, task-based paradigm with high-temporal-resolution ERP recordings offers evidence that extends meaningfully beyond traditional self-report measures, which is a distinction that matters most for rumination. Rumination is recognized as a major cognitive risk factor for the onset and maintenance of depressive disorders (Nolen-Hoeksema, 1991; World Health, 2025) and is closely linked to deficits in executive function, reduced cognitive flexibility, impaired disengagement from negative content, and heightened vulnerability to stress (Aker et al., 2014; Joormann & Gotlib, 2010; Koster et al., 2011; Ruscio et al., 2015). Recent neuroimaging and ERP investigations further demonstrate that rumination is accompanied by over-recruitment of cognitive control networks and altered N2/P3 dynamics during goal-directed tasks (Field; Connell et al., 2020; Gan et al., 2019; Owens et al., 2021; Park et al., 2024). Because the precise relationship between trait rumination and objective executive control remains actively debated (Zetsche et al., 2018), relying solely on self-report questionnaires is insufficient for isolating the specific cognitive mechanisms involved. By pairing culturally adapted social cues with N2 and P3 ERP components, the present paradigm therefore provides a sensitive behavioral-neurophysiological platform for evaluating how individual differences in rumination alter emotional disengagement and motor inhibition (Shimony et al., 2021; Whitmer & Banich, 2007).

Limitations

Several limitations of the present investigation warrant consideration. First, the validation dataset was restricted to a preliminary sample size ($n=5$), which constrained overall statistical power. Consequently, while key primary contrasts regarding behavior and P3 latency reached statistical significance, secondary ERP comparisons did not survive strict corrections for multiple testing. Second, while the present study established the task's feasibility in healthy Thai adults, future studies must recruit larger, clinically diverse samples, including individuals with high trait rumination or clinical depression, and examine whether rumination moderates false alarm rates or modulates neural components during emotional inhibition.

Conclusion

In conclusion, the culturally adapted Asian Facial Emotional Go/No-Go task represents a feasible, ecologically valid paradigm for evaluating emotional inhibitory control. The observed behavioral facilitation and reduced P3 latency during angry-cued

trials demonstrate the distinct cognitive demands imposed by threat- versus reward-oriented social cues. Ultimately, this validated paradigm establishes a robust foundation for future empirical research investigating executive dysfunction, emotional dysregulation, and rumination across Asian populations.

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P004

REWIRING MEMORY NETWORKS: NON-INVASIVE NEUROMODULATION IN THE HUMAN BRAIN

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Abstract:

Background: Working memory, the capacity to hold and manipulate information over short timescales, is a core cognitive function impaired in many neurological and psychiatric conditions. Transcranial magnetic stimulation (TMS) shows promise but findings remain inconsistent. A key limitation is the use of uniform stimulation protocols that overlook the individual variability in the functional brain networks underlying working memory functions.

Aim(s): In a multi-session, randomized crossover design, we tested whether individualized, network-guided TMS, optimizing stimulation target and frequency per participant, produces reliable working-memory gains.

Methods: Using concurrent TMS and functional magnetic resonance imaging (fMRI), we first map each individual's personalized functional networks to identify subject-specific stimulation targets. We then deploy a real-time neural decoder, built on long short-term memory recurrent neural networks, to track stimulation-evoked brain dynamics and empirically determine the optimal stimulation frequency for each participant from a tested set. We then deliver repetitive TMS at participant-specific optimal and suboptimal frequencies to these individualized targets in 19 healthy adults while they engage in a working memory task.

Results: Personalized optimal-frequency stimulation produced significantly faster working memory responses at longer retention delays i.e., 4 sec delay ($F(2, 36) = 5.60$, $p = 0.008$, and the 12 sec delay ($F(2, 36) = 3.33$, $p = 0.047$), without compromising accuracy, whereas suboptimal-frequency stimulation at the same individualized target did not. Effects were specific to working memory and depended on target–frequency interactions rather than any single uniform frequency. Optimal frequencies varied widely across individuals, and real-time decoder outputs predicted subsequent behavioral improvements.

Conclusion: Together, these findings demonstrate that respecting the biological individuality of each brain can transform inconsistent stimulation outcomes into reliable cognitive enhancement. This work offers a generalizable, biomarker-driven framework for precision neuromodulation, with implications for both basic understanding of memory networks and the development of individualized cognitive and clinical interventions.

Keywords: working memory; transcranial magnetic stimulation; personalized functional networks; concurrent TMS-fMRI; closed-loop neuromodulation

P005

BEHAVIORAL AND NEURAL ACTIVITY OF THE EMOTIONAL MEMORY TASK: A PILOT STUDY FOR COGNITIVE SLEEP RESEARCH

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Abstract:

Background: Sleep is recognized as a vital biological process for human cognitive performance and emotional functioning. Sleep plays an important role in memory consolidation and long-term memory formation. Emotional memory is a fundamental cognitive process that supports encoding, storage, and retrieval of emotionally salient experiences. Within the domain of cognitive retrieval, emotional contents are well known to be remembered better than neutral contents. An emotional event elicits a response that includes subjective feelings, behaviors indicating approach or avoidance and various physiological responses. However, underlying neural mechanisms that regulate these emotional memory processes, especially the temporal dynamics of distinct brain waves during recognition, remain unclear.

Aim(s): This pilot study aimed to validate the feasibility of an emotional memory task and characterize behavioral performance and neural activity during emotional memory retrieval in healthy young adults using electroencephalography (EEG), prior to its application in future sleep research.

Methods: Five healthy young adult volunteers aged 20-25 years encoded emotional memory pictures from the Open Affective Standardized Image Set (OASIS), a validated database of valence- and arousal-rated negative and neutral images, on the first day. On the following day, participants completed a retrieval session in which they recognized old and new pictures. During the task, EEG and behavioral data were recorded and analyzed using event-related potentials (ERPs).

Results: Preliminary analyses revealed identifiable FN400 and LPC components over regions of interest, including Fz, Cz, and Pz. Correlation analysis showed that, during the old neutral condition, longer reaction times were associated with lower FN400 amplitude over the C3 region ($p < 0.05$). No significant associations were observed between behavioral performance and LPC measures.

Conclusion: Preliminary findings indicate that the emotional memory paradigm successfully captures FN400 ERP markers associated with familiarity-related neural processes during emotional memory recognition. Future studies with larger sample sizes and refined experimental designs are needed to further validate these findings.

Keywords: Sleep; Long-term memory; emotional memory; EEG; ERP

P006

DEVELOPMENT OF AN INTEGRATED tDCS AND SELF-MANAGEMENT TRAINING PROTOCOL FOR ADULTS WITH AUTISM SPECTRUM DISORDER IN A VOCATIONAL CONTEXT

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Abstract:

Background: Adults with Autism Spectrum Disorder (ASD) continue to experience poor vocational outcomes despite preserved intellectual ability. Executive Function (EF) dysfunction is a major contributor to these difficulties. Although self-management interventions are evidence-based, they remain resource-intensive and require sustained support, limiting accessibility for many adults with ASD. To date, no protocol has integrated transcranial direct current stimulation (tDCS) with Applied Behavior Analysis (ABA)-based self-management training in a vocational context.

Aim(s): To develop an integrated intervention protocol targeting vocational self-management behaviors in adults with high-functioning ASD (HF-ASD) by combining ABA-based self-management training with concurrent transcranial direct current stimulation (tDCS).

Methods: The protocol was developed through literature synthesis integrating evidence from cognitive neuroscience to identify the stimulation target, from ABA-based self-management literature to develop the behavioral intervention, and neuromodulation research to determine stimulation parameters.

Results: An integrated intervention protocol combining concurrent anodal tDCS targeting the left dorsolateral prefrontal cortex (left dlPFC) with structured ABA-based self-management training was developed for subsequent pilot evaluation. The implementation and evaluation in adults with HF-ASD were beyond the scope of this study. The protocol comprised five individualized sessions and included standardized intervention procedures, behavioral training materials, and monitoring tools targeting vocationally relevant executive behaviors.

Conclusion: This study developed an integrated intervention protocol combining ABA-based self-management training with concurrent tDCS for adults with HF-ASD in a vocational context. The protocol provides a structured framework for future pilot studies evaluating its feasibility and preliminary efficacy.

Keywords: Autism Spectrum Disorder, Executive Function, Self-Management, Transcranial Direct Current Stimulation, Vocational Rehabilitation

Background

Autism Spectrum Disorder (ASD) is a lifelong neurodevelopmental condition characterized by persistent difficulties in social communication and restricted, repetitive patterns of behavior. Despite average or above-average intellectual ability, many autistic adults continue to experience poor vocational outcomes (Syvan & Pearlman-Avni, 2019). More than half of adults with ASD remain unemployed or underemployed, and vocational participation remains substantially lower than in the general population and in people with other disabilities (Bury et al., 2024). Evidence suggests that these difficulties are frequently related not to deficits in technical competence, but to impairments in adaptive workplace behaviors, including task initiation, time management, organization, cognitive flexibility, and workplace self-regulation (Pezzimenti et al., 2024). These behavioral difficulties limit vocational independence even among adults without intellectual disability (Gal et al., 2015).

Executive dysfunction has been consistently identified as one of the principal cognitive mechanisms underlying these vocational challenges (Pugliese et al., 2016). Neuroimaging and cognitive studies demonstrate atypical recruitment of executive-control networks involving the dorsolateral prefrontal cortex (dlPFC), which may contribute to impairments in executive function domains, including working memory, inhibitory control, planning, and cognitive flexibility (Chan et al., 2022). Together, these deficits may reduce the capacity to regulate goal-directed behavior in complex environments such as workplace settings (Holc et al., 2025).

Behavioral interventions, particularly self-management approaches derived from Applied Behavior Analysis (ABA), have demonstrated effectiveness in improving vocationally relevant behaviors by promoting self-monitoring, self-evaluation, and self-regulation (Beaver et al., 2017). Self-management strategies have also been incorporated into comprehensive vocational programs, such as Project SEARCH plus ASD Supports, which have demonstrated improvements in vocational independence and skill generalization (Wehman et al., 2017). However, implementation typically requires intensive individualized support, limiting scalability and accessibility for many adults with ASD (McConkey, 2022; Wehman et al., 2013).

Transcranial direct current stimulation (tDCS) has emerged as a potential neuromodulation technique capable of facilitating activity-dependent plasticity within executive-control networks during behavioral learning (Yatan et al., 2023). Although behavioral interventions and tDCS have each demonstrated potential for improving executive functioning, no intervention protocol has integrated concurrent tDCS with ABA-based self-management training within a vocational rehabilitation context for adults with ASD.

Materials and Methods

The intervention protocol was developed through a current evidence synthesis process integrating three complementary domains: cognitive neuroscience to identify the stimulation target, ABA-based self-management interventions literature to develop the behavioral components, and transcranial direct current stimulation (tDCS) literature to determine stimulation parameters.

Based on evidence identifying the dorsolateral prefrontal cortex (dlPFC) as a central hub for executive control rather than a disorder-specific lesion, the dlPFC was selected as the stimulation target (Todd F Heatherton & Dylan D Wagner, 2011; Miller & Cohen, 2001).

Within the ABA literature, self-management was selected as the core intervention because it directly targets independent regulation of behavior and has demonstrated

effectiveness in improving vocationally relevant behaviors in adults with ASD (Scheibel et al., 2024).

Core intervention components were selected based on the tripartite framework of self-management, comprising self-monitoring, self-evaluation, and self-reinforcement (Erhard et al., 2022). Behavioral Skills Training (BST), incorporating instruction, modeling, rehearsal, and feedback, together with task analysis, guided practice, and homework assignments, was incorporated to facilitate skill acquisition and generalization to workplace settings (Brighenti et al., 2023).

The tDCS parameters were determined through evidence synthesis from executive-function neuromodulation, ASD intervention studies, and current safety recommendations (Bikson et al., 2018). A conventional anodal F3–cathodal Fp2 montage was adopted because of its widespread application in executive-function research and its compatibility with repeated behavioral intervention (Salehinejad et al., 2022). Stimulation was delivered at 2 mA for 20 minutes using 5 × 7 cm saline-soaked sponge electrodes, maintaining current density within established safety recommendations while providing practical and reproducible implementation (Antal et al., 2026).

Finally, five concurrent stimulation sessions were selected to balance evidence supporting cumulative neuroplasticity, participant burden, and feasibility within a vocational rehabilitation setting (Arturi et al., 2025).

The preliminary protocol was iteratively refined through consultation with specialists in behavioral intervention and neuromodulation to improve feasibility and applicability within a vocational rehabilitation setting. The finalized protocol comprised five individualized intervention sessions integrating concurrent tDCS with structured ABA-based self-management training.

Results

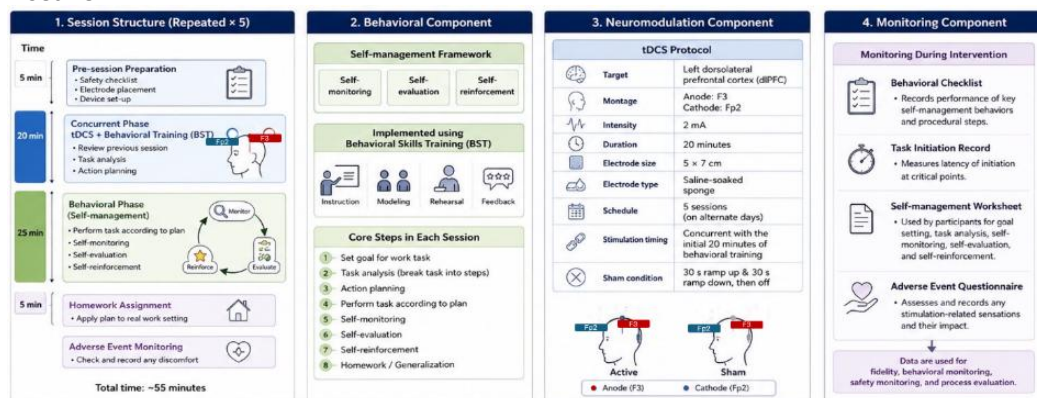


Figure1. Overview of the Developed Intervention Protocol

An integrated intervention protocol combining concurrent transcranial direct current stimulation (tDCS) with ABA-based self-management training was successfully developed for a subsequent pilot study for adults with high-functioning ASD in vocational rehabilitation. Implementation and evaluation of the protocol were beyond the scope of the present study. The protocol integrates neuromodulation and behavioral intervention into a structured learning framework targeting vocational self-management behaviors.

It comprises five individualized intervention sessions delivered on alternate days, with tDCS administered concurrently during the initial phase of behavioral training.

(1) Self-management Worksheet

Goal (What do I want to achieve?)	Task Analysis (What steps should I do?)	Action Plan (When/Where/How?)
Self-monitoring (Did I do it?)	Self-evaluation (How did I do?)	Self-reinforcement (What will I reward myself?)

Purpose: Guides participants through the self-management cycle.

(2) Task Analysis Form

Step	Behavior (What to do)	Completed (✓)
1		
2		
3		
4		
5		
...		

Purpose: Breaks work tasks into observable, sequential steps.

(3) Behavioral Checklist

Behavior Components	Correct (✓)	Incorrect (X)	NA
1. Write goal			
2. Start task analysis			
3. Include all components			
4. Start first step			
5. Finish first step			
...			

Purpose: Records key behaviors and procedural fidelity.

(4) Task Initiation Record

Event	Latency (sec)
1. Instruction A → start TA (A)	
2. Finish TA (A) → start first step (A)	
3. Instruction B → start TA (A & B)	
4. Finish TA (A & B) → start first step	

Purpose: Measures initiation latency at critical transition points.

(5) Adverse Event Questionnaire

Itchiness	None	Mild	Moderate	Considerable	Strong
	☐	☐	☐	☐	☐
Pain	None	Mild	Moderate	Considerable	Strong
	☐	☐	☐	☐	☐
Burning	None	Mild	Moderate	Considerable	Strong
	☐	☐	☐	☐	☐
...					

Purpose: Monitors stimulation-related sensations and safety.

Figure 2. Developed Intervention Material

Discussion and Conclusion

The present study developed an integrated intervention protocol combining concurrent transcranial direct current stimulation (tDCS) with ABA-based self-management training for adults with high-functioning ASD within a vocational rehabilitation context. The protocol integrates existing evidence from cognitive neuroscience, behavioral intervention, and neuromodulation into a structured intervention specifically designed to address vocationally relevant executive behaviors.

The design of the protocol was guided by the observation that executive dysfunction is one of the most consistently reported factors contributing to vocational difficulties in adults with ASD (Pugliese et al., 2016; Sullivan et al., 2024). Based on evidence identifying the left dorsolateral prefrontal cortex (left-DLPFC), as a central hub for executive control, it was selected as the stimulation target (Fried et al., 2014; T. F. Heatherton & D. D. Wagner, 2011; Miller & Cohen, 2001; Panikratova et al., 2020).

While behavioral interventions based on Applied Behavior Analysis (ABA) have demonstrated effectiveness in improving adaptive and vocational behaviors, these approaches have traditionally relied on behavioral mechanisms alone. Conversely, previous tDCS studies have primarily focused on improving laboratory-based cognitive performance using computerized cognitive training (Han et al., 2022; Ratsapbhayakul et al., 2024). The present protocol attempts to bridge these two approaches by pairing concurrent neuromodulation with an evidence-based behavioral intervention targeting real-world vocational self-management.

A further strength of the protocol is its emphasis on ecological validity. Instead of training isolated cognitive processes, the behavioral component focuses on vocationally relevant executive behaviors, including planning, task initiation, self-monitoring, self-evaluation, and self-reinforcement (Erhard et al., 2022; Hume et al., 2009). These skills are directly applicable to workplace functioning and reflect the executive demands encountered during vocational rehabilitation. Delivering tDCS concurrently with behavioral training was intended to maximize activity-dependent plasticity during engagement of executive-control networks, providing a theoretically grounded rationale for integrating neuromodulation with behavioral learning (Fritsch et al., 2010; Kronberg et al., 2020).

In addition to the intervention procedures, the present study developed a standardized intervention package consisting of structured session manuals, self-management worksheets, task-analysis forms, behavioral monitoring tools, and safety-monitoring procedures. These materials may facilitate treatment fidelity, improve reproducibility across settings, and support future implementation in vocational rehabilitation programs.

Several limitations should be acknowledged. The present study focused exclusively on protocol development and therefore does not provide evidence regarding clinical efficacy, feasibility, or participant acceptability. Furthermore, stimulation parameters were selected based on currently available evidence rather than individualized neurophysiological measures. Future pilot studies are required to evaluate the feasibility of the protocol, estimate preliminary treatment effects, and inform subsequent randomized controlled trials.

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P007

QUANTITATIVE ASSESSMENT OF REGIONAL NEURONAL DAMAGE FOLLOWING SHED-DERIVED EXOSOME AND RVG-MODIFIED EXOSOME TREATMENT IN A RAT BCCAO MODEL

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Abstract:

Background: Bilateral common carotid artery occlusion (BCCAO) induces chronic cerebral hypoperfusion and region-selective neuronal injury. The motor cortex, striatum, and hippocampal CA3 are particularly vulnerable to hypoperfusion and are commonly examined for region-specific neuropathological changes. Exosomes derived from stem cells of human exfoliated deciduous teeth (SHED-EXO) exhibit neuroprotective potential. Rabies virus glycoprotein (RVG) modification is hypothesized to enhance brain targeting through interaction with neuronal nicotinic acetylcholine receptors, increasing neuronal uptake and delivery of exosome cargo.

Aim(s): To quantitatively compare neuronal damage in the motor cortex, striatum, and hippocampal CA3 following treatment with SHED-EXO or RVG-modified SHED-derived exosomes (RVG-EXO) in rats subjected to BCCAO.

Methods: Male Sprague-Dawley rats were assigned to Sham + PBS, BCCAO + PBS, BCCAO + SHED-EXO, or BCCAO + RVG-EXO groups (n = 3/group). Exosomes (100 µg/rat) were administered intravenously 3 h after BCCAO. On day 14, brain sections were stained with hematoxylin and eosin (H&E) and cresyl violet (Nissl). Neuronal damage was quantified as the percentage of neurons with shrunken cell bodies, pyknotic nuclei, and disrupted neuronal architecture. Data were analyzed using one-way ANOVA with Tukey's multiple-comparisons test (GraphPad Prism 10.2.3; mean ± SD; P < 0.05).

Results: In H&E-stained hippocampal CA3, neuronal damage was 7.88 ± 5.33% in Sham + PBS, 76.05 ± 22.66% in BCCAO + PBS, 23.34 ± 11.96% after SHED-EXO, and 7.06 ± 2.83% after RVG-EXO. Both treatments significantly reduced neuronal damage compared with BCCAO + PBS (P < 0.01 and P < 0.001, respectively). Although RVG-EXO showed a

more favorable numerical outcome than SHED-EXO, the difference was not statistically significant. Nissl staining showed similar findings.

Conclusion: Both SHED-EXO and RVG-EXO demonstrated neuroprotective effects against BCCAO-induced neuronal injury. RVG-EXO showed the lowest mean neuronal damage in the hippocampal CA3, supporting further investigation in larger mechanistic studies.

Keywords: Bilateral common carotid artery occlusion (BCCAO), SHED-derived exosomes, rabies virus glycoprotein (RVG), neuronal damage, histopathological analysis

P008

LCN2/LCN2R AXIS TRIGGERS NEURONAL DEATH IN HEPATIC ENCEPHALOPATHY THROUGH GRB2 RECRUITMENT AND MITOCHONDRIAL ENERGY DEFICITS

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Abstract:

Background: Hepatic encephalopathy (HE) is a severe neuropsychiatric complication of liver failure characterized by a spectrum of cognitive and motor impairments. Lipocalin-2 (Lcn2) is a multifunctional secretory protein and inflammatory mediator predominantly released by glial cells in response to brain insults, whereas its receptor, Lcn2R, is expressed in cells. Nevertheless, the precise role of this Lcn2/Lcn2R axis in HE pathogenesis remains poorly understood.

Aim(s): To investigate the cellular mechanisms by which the Lcn2/Lcn2R axis triggers neuronal death in HE.

Methods: Acute liver failure was induced in C57BL/6 mice using azoxymethane (AOM; 100 µg/g, i.p.). Primary neuronal cultures from postnatal day 1 mice were used for mechanistic studies. RNA sequencing, immunofluorescence, proximity ligation assay, co-immunoprecipitation, mitochondrial respiration analysis, pharmacological inhibition, and siRNA knockdown were performed.

Results: RNA sequencing and immunofluorescence identified Lcn2 as the most significantly upregulated gene in the hippocampus, with its receptor, Lcn2R, expressed in neurons. Mechanistically, Lcn2 binding to Lcn2R recruits Grb2 to activate the downstream MEK/ERK signaling pathway. This pathway upregulates pyruvate dehydrogenase kinases (PDKs), leading to the phosphorylation and inactivation of pyruvate dehydrogenase (PDH). Consequently, mitochondrial respiration experiments revealed that Lcn2 significantly impairs mitochondrial energy metabolism by reducing ATP production and coupling efficiency. *In vitro* silencing of Lcn2R or inhibition of MEK (U0126) and PDK (dichloroacetate) reversed these metabolic deficits and prevented neuronal death. Furthermore, *in vivo* intracerebroventricular administration of an Lcn2-neutralizing antibody successfully restored cerebral ATP levels and reduced neuronal apoptosis in HE mice.

Conclusion: The Lcn2/Lcn2R axis contributes to HE pathogenesis by activating Grb2-mediated MEK/ERK/PDK signaling, which impairs mitochondrial ATP synthesis and ultimately induces neuronal death, highlighting this pathway as a potential therapeutic target for HE.

Keywords: Hepatic encephalopathy, Acute liver failure, Mitochondrial respiration, Apoptotic cell death

P009

AWAKE CRANIOTOMY WITH FUNCTIONAL BRAIN MAPPING PRESERVES MOTOR AND LANGUAGE FUNCTION IN ELOQUENT BRAIN TUMOR SURGERY: A CASE SERIES FROM THAILAND

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Abstract:

Background: Preservation of neurological function is a major objective in the surgical management of tumors involving eloquent brain regions. Awake craniotomy with intraoperative functional mapping enables individualized identification of motor and language cortex, facilitating maximal safe resection while minimizing postoperative neurological deficits. However, clinical experience from Thailand remains limited.

Aim(s): To evaluate the feasibility, safety, and functional outcomes of awake craniotomy for resection of intracranial tumors located within eloquent brain regions.

Methods: We retrospectively reviewed consecutive patients who underwent awake craniotomy with direct electrical stimulation for motor and/or language mapping at Chulabhorn Hospital, Bangkok, Thailand, between 2021 and 2025. All procedures employed a standardized asleep-awake-asleep anesthetic protocol. Extent of resection was determined from postoperative imaging by radiologist interpretation and surgeon review and categorized as gross total or near-total resection. Neurological outcomes were assessed preoperatively, immediately postoperatively, and at 90- and 180-day follow-up. Seizure outcomes were classified according to the International League Against Epilepsy (ILAE). Given the small sample size, data were analyzed descriptively.

Results: Eight patients (mean age 50.6 ± 12.3 years, range 34–72) underwent awake craniotomy. Histopathology included four brain metastases from lung cancer, three WHO grade II gliomas, and one WHO grade IV glioma. Cortical mapping was successfully completed in all patients without conversion to general anesthesia. Gross total resection was achieved in five patients (62.5%) and near-total resection in three (37.5%). Motor function was preserved or improved in seven patients (87.5%), with one persistent motor deficit attributed to intraoperative venous compromise rather than mapping failure. Language function was preserved or improved in all patients, including those with preoperative aphasia. Seizure control improved in all patients with preoperative epilepsy. One intraoperative seizure (12.5%) was successfully managed without

conversion to general anesthesia. No perioperative mortality, surgical site infection, cerebrospinal fluid leakage, or reoperation occurred.

Conclusion: Awake craniotomy with functional brain mapping is a safe and feasible approach for tumors involving eloquent brain regions, preserving motor and language function while achieving satisfactory extent of resection. These findings support broader implementation of functional brain mapping in neuro-oncological surgery in Thailand.

Keywords: Awake craniotomy; Brain mapping; Neuro-oncology; Eloquent cortex; Functional preservation

P011

DEEP BRAIN MODULATION AND SEIZURE CONTROL VIA TEMPORAL SUMMATION OF INTERSECTIONAL SHORT PULSES

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Abstract:

Background: Conventional transcranial electrical stimulation frequently struggles with inadequate depth penetration and poor spatial precision, restricting its clinical usefulness. To resolve these challenges, intersectional short pulse (ISP) stimulation utilizes fast, alternating electrical pulses that nerve cell membranes integrate over time.

Aim(s): This research sought to determine the underlying biophysical principles of temporal summation and assess its effectiveness for modulating deep brain regions and halting seizures *in vivo*.

Methods: We integrated finite-element modeling (FEM using ROAST) and in situ field mapping in two human cadavers using subgaleal and stereoelectroencephalography (sEEG) electrodes to characterize induced electric field dynamics. Cellular temporal summation mechanisms were evaluated using in vivo whole-cell patch-clamp recordings in the somatosensory cortex of female Wistar rats combined with NEURON software simulations. Finally, to test therapeutic efficacy, closed-loop LFP-guided ISP stimulation

was applied during hippocampal-kindled temporal lobe epilepsy (TLE) in eleven male Long-Evans rats across >500 seizure events.

Results: Patch-clamp recordings and cellular simulations revealed that neuronal membranes accumulate sequential pulses via scalar temporal integration rather than extracellular vectorial field overlap. While FEM and cadaveric sEEG recordings showed comparable instantaneous field strengths between conventional and short-pulse techniques, ISP achieved significantly more consistent neuronal excitability across varying tissue depths. In the TLE model, closed-loop LFP-triggered ISP stimulation dramatically outperformed conventional methods, reducing hippocampal seizure duration by 45% compared to sham treatments and 35% compared to standard stimulation, while sharply decreasing motor seizure severity.

Conclusion: This temporally structured approach offers a superior, targeted non-invasive technique for reaching deep neural structures. By demonstrating membrane-level mechanisms and successfully suppressing epileptic activity, these results strongly support clinical translation for neurological disorders.

Keywords: Intersectional Short Pulse (ISP) stimulation; Temporal summation; Transcranial electrical stimulation (TES); Neuromodulation; Seizure Suppression

P012

CLOSED LOOP, SUBGALEAL INTERSECTIONAL SHORT-PULSE STIMULATION FOR THE TREATMENT OF THERAPY RESISTANT EPILEPSY IN ADULTS

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Abstract:

Background: Approximately one-third of patients with epilepsy are resistant to antiseizure medications (ASMs) and are not candidates for surgical intervention. Existing neuromodulation therapies, including deep brain stimulation (DBS), vagus nerve stimulation (VNS), and responsive neurostimulation (RNS), are limited by invasiveness, limited targeting precision, delayed therapeutic effects, and lower adaptability. Therefore, a minimally invasive, personalized, closed-loop neurostimulation therapy capable of rapidly terminating seizures remains an unmet clinical need.

Aim(s): To evaluate the safety, feasibility, and acute therapeutic efficacy of Intersectional Short-Pulse (ISP) stimulation, a novel minimally invasive closed-loop neurostimulation approach for seizure control in adult patients with drug-resistant epilepsy and focal seizure profile.

Methods: ISP stimulation was delivered through subgaleally implanted electrode strips using brief, high-intensity pulses arranged spatially and temporally to maximize stimulation within seizure onset zones while minimizing off-target effects. In this first clinical study, seizures were detected using a machine-learning algorithm and closed-loop ISP stimulation was delivered manually or automatically based on patient-specific tolerance thresholds. Ethical approvals were obtained from the National Institute of Pharmacy and

Nutrition, Hungary (OGYÉI/9674/2021) and the Medical Research Council, Hungary (IV/1920-1/2021/EKU). ClinicalTrials.gov identifier: NCT07041619.

Results: Twelve patients received implanted electrodes and closed-loop ISP stimulation during 116 patient-days of video-EEG monitoring. Mean perception and tolerance thresholds were 8.1 mA and 28.2 mA, respectively. Ictal ISP stimulation reduced seizure duration by 52% ($p < 0.001$; $N = 272$ baseline vs. 224 stimulated seizures from 11 patients), altered ictal spectral characteristics, and inhibited secondary generalization. In a within-subject comparison of five patients with multiple daily seizures, cumulative electroclinical seizure incidence decreased by 85% within 48 hours. Among 102 adverse events (75 grade 1, 25 grade 2, and 2 unrelated grade 3), no serious adverse events were attributable to implantation or ISP stimulation, and all resolved spontaneously or following treatment.

Conclusion: Closed-loop ISP stimulation via subgaleal electrodes is a safe, feasible, and minimally invasive therapy that acutely reduces seizure burden. These findings support further studies to assess long-term efficacy and optimize stimulation parameters.

Keywords: (epilepsy, neuromodulation, transcranial electrical stimulation, ISP)

P013

TASK-RELATED EEG CONNECTIVITY AND OSCILLATORY ALTERATIONS DURING ASSOCIATIVE LEARNING IN ADULT MIGRAINE

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Abstract:

Background: Migraine is increasingly recognized as a disorder involving altered sensory and cognitive processing beyond recurrent headache attacks. Task-related EEG may provide temporally precise biomarkers of these alterations during cognitive paradigms.

Aim(s): This study aimed to investigate event-related oscillatory activity and functional connectivity during associative learning in migraine.

Methods: Sixty-four-channel EEG was recorded from twenty-six migraine patients without aura and matched healthy controls during an associative learning task, at least three days after the patients' last headache. Stimulus-onset-locked and button-press-locked epochs were extracted to separate stimulus evaluation, decision-related and reward-related processes. Analyses included scalp power, time–frequency decomposition and region-of-interest-based connectivity. Bilateral frontal, temporal, parietal, and occipital ROIs were examined. Functional coupling was assessed using weighted phase lag index (wPLI), while directed phase lag index (dPLI) was used to characterize interregional phase-leading relationships. Group differences in power were assessed using cluster-based permutation tests; connectivity differences were tested using permutation procedures with FDR correction.

Results: Migraine-related group differences were consistently observed in the beta frequency range. Stimulus-locked analyses revealed altered post-stimulus scalp power and time–frequency responses, particularly in posterior regions during the test phase. Button-press-locked analyses showed broader beta-dominant alterations involving frontal, parietal, temporal and occipital regions across pre- and post-response time windows. Patients showed higher baseline connectivity in several ROI pairs but weaker task-related increases than controls (FDR-corrected $p < 0.05$). These connectivity differences involved frontal, parietal and temporal ROI pairs. dPLI indicated similar anterior-to-posterior phase-leading organization in both groups, with frontal regions predominantly phase-leading parietal and temporal regions.

Conclusion: Stimulus- and button-press-locked analyses provided complementary information on migraine-related cognitive processing and may offer a neural context for the lower behavioral performance previously reported in migraine using the same task. Reduced task-related connectivity enhancement may reflect less adaptive network recruitment and may contribute to potential task-related EEG biomarkers of migraine.

Keywords: migraine; EEG; associative learning; time-frequency analysis; connectivity

P014

ATTENTION MODULATION ACROSS CONTRAST LEVEL THROUGH AN INTEGRATED EEG AND FNIRS APPROACH

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Abstract:

Background: Previous studies have reported inconsistent findings regarding attentional modulation across electrophysiological and hemodynamic measurement techniques. For instance, obtaining contrast response function from different tools yields different models of modulation. Previous studies have primarily shown an increased response to all stimulus equally in fMRI measurements, while EEG have shown contrast-dependent modulation.

Aim(s): This study aims to investigate the effect of attention on neural activity with different stimulus contrast using simultaneous measurement.

Methods: Thirty participants were asked to identify contrast change in the target stimulus while EEG and fNIRS data were being recorded. SSVEP and fNIRS response were analyzed using contrast response function models.

Results: EEG results reveal increase in response across all contrast levels in contralateral occipital area. FNIRS results do not yield any main effect.

Conclusion: EEG analyses revealed significant modulation of neural responses across contrast conditions, particularly over occipital regions. In contrast, fNIRS analyses did not reveal significant hemodynamic differences between conditions. These findings suggest that electrophysiological and hemodynamic measures may differ in their sensitivity to attentional modulation under rapid visual stimulation paradigms.

Keywords: Attention, EEG, Contrast, fNIRS

Background

Visual attention is a selective process that prioritizes stimuli in the environment (Carrasco, 2011). Features of visual stimuli can enhance attention and increase the neuronal activities (Fang & Liu, 2019). Many studies have used various types of designs and brain imaging techniques to explore mechanisms of attention (Hillyard & Anllo-Vento, 1998; Itthipuripat et al., 2023; Sawetsuttipan et al., 2023; Slotnick, 2018).

Previous studies have suggested that attentional modulation may depend on stimulus contrast. According to normalization-based models of attention, attention can alter neural responses through mechanisms such as contrast gain or response gain (Reynolds & Heeger, 2009). Consequently, the magnitude and pattern of attentional effects may vary across different stimulus contrast levels. Understanding how attention

interacts with visual contrast remains an important topic in vision science and cognitive neuroscience.

There are many tools used to study brain activity, each having their capabilities and limitations. Electroencephalography (EEG) is one method, measuring the electrical signals of the neurons near the scalp (L'Abate & Kaiser. 2012). Another example is functional magnetic resonance imaging (fMRI), which monitors blood volume and oxygen level changes relating to brain function (Logothetis et al., 2001). Similarly, functional near-infrared spectroscopy (fNIRS) also monitors blood volume and oxygen level changes relating to brain functions (Izzetoglu et al., 2005), although at a lower spatial resolution. In principle, these methods should reflect the same underlying mechanisms of the brain functions, complementing each other.

However, evidence suggests that different neuroimaging techniques do not always yield complementary findings. For example, Itthipuripat et al. (2019) demonstrated that fMRI and EEG measurements yielded qualitatively different patterns of attentional modulation. EEG measurements demonstrated contrast-dependent attentional modulation, whereas fMRI responses showed an additive increase across contrast levels. However, the measurements were acquired in separate experimental sessions because simultaneous EEG-fMRI acquisition was not employed in that study.

These findings raise an important question regarding whether the discrepancy arises from differences in the underlying neural mechanisms being measured or from differences in the measurement techniques themselves. Because EEG and fMRI are typically acquired in separate experimental sessions, variations in task performance, participant state, and experimental conditions may contribute to the observed differences. Simultaneous multimodal recordings may therefore provide a more direct comparison between electrophysiological and hemodynamic responses.

Given the similarities between fMRI and fNIRS, simultaneous EEG-fNIRS recordings provide a practical approach for examining attentional modulation using electrophysiological and hemodynamic measurements under identical experimental conditions. Although fNIRS has lower spatial resolution than fMRI, simultaneous acquisition allows both modalities to be measured during the same trials, reducing potential variability arising from separate experimental sessions.

Simultaneous EEG-fNIRS measurements provide an opportunity to directly compare electrophysiological and hemodynamic responses during visual attention. Such measurements may help clarify whether previously reported discrepancies arise from differences in neural signals, measurement sensitivity, or experimental procedures.

As such, this study aims to integrate EEG and fNIRS measurements to investigate attentional modulation across different visual contrast levels and to compare electrophysiological and hemodynamic responses obtained under identical experimental conditions.

Materials and Methods

Participants

30 neurologically healthy participants aged 20-25 with normal or corrected-to-normal vision were recruited from the Mahidol University group chat.

Materials

Simultaneous EEG and functional near-infrared spectroscopy (fNIRS) recordings were acquired during the visual attention task. EEG data were recorded using a Neuroscan acquisition system (NeuroScan Version 4.3, Neurosoft Inc.) with a standard EEG cap, scalp electrodes, amplifier, and recording software.

Hemodynamic responses were recorded using a continuous-wave fNIRS system (NIRSport2, NIRx Medical Technologies, USA) consisting of 15 light sources and 15 detectors. Data were sampled at 5.43 Hz. Probe placements can be found in figure 1.

Visual stimuli were presented on a computer monitor using a dedicated stimulus presentation computer. EEG and fNIRS recordings were synchronized using shared event triggers to ensure temporal alignment between neural and hemodynamic measurements.

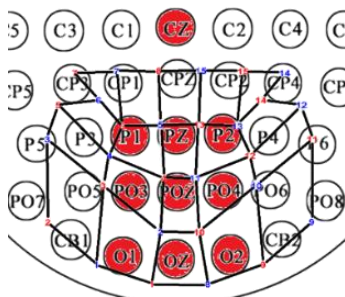


Figure 1. Probes Placement. Red areas represent EEG probe. Black lines represent fNIRS channels

Task

The visual attention task was designed to investigate attention modulation across contrast levels. Participants were asked to identify changes in the contrast of stimulus. The task consists of cue phase and stimulus phase. During the cue phase, a fixation cue appeared in the middle of the screen for 500 ms. This cue can be one of three things: a cross, a right-pointing arrow, or a left-pointing arrow. The cue instructed participants to direct their attention to the center of the screen, the right side of the screen, or the left side of the screen respectively. Then, stimulus appeared to the left or right side of the screen for 2000 ms. The stimulus is a flickering checkerboard at 15Hz. The stimulus may have one of 6 levels of contrast, at Michelson contrast level of 100, 70, 35, 17.5, 8.75, or 4.375. In 25% of trials, the stimulus had its contrast reduced while flickering. Participants were instructed to press a button if they detect a change in contrast. The cue phase and stimulus phase make 1 trial. There is a 1500 ms - 2500 ms of intertrial interval between each trial. There are 96 trials per block of trials. Participants each complete 4 blocks of trials, split in 2 2-blocks sessions with a 10 minutes break in between.

Analysis

EEG data were preprocessed and analyzed using BrainAnalyzer software. Data were segmented into 3000 ms epoch from -500 ms to 2500 ms from stimulus presentation. Data were preprocessed using BrainAnalyzer software. EEG data were re-referenced to the 2 mastoid electrodes. Then, 0.1 Hz high-pass and 45 Hz low-pass filter were applied. ICA ocular correction were then performed. Artifact rejection was performed using $\pm 60\mu V$ for Horizontal eye movement. Each trial were then segmented into each conditions. Baseline correction was performed at -200-0ms. The remaining data were averaged for each participant and exported for statistical inference in MATLAB.

fNIRS data were preprocessed using Satori software. Preprocessing includes channel rejection, motion correction using spike removal and temporal derivative distribution repair. Physiological component removal was performed using PCA. Temporal filtering used Butterworth filter of 0.01Hz and 0.4Hz high-pass and low-pass filter respectively. Events were synchronized from EEG trigger data. Event related averages

were obtained and exported for analysis in MATLAB software. GLM were performed on -2s to 5s of stimulus onset. Beta values were obtained for each channel for each participant for statistical analysis.

Data were rearranged to present contralateral brain regions relative to stimulus location on the left side of the topological brain. Similarly, the right side of the topological brain corresponds to ipsilateral stimulus location.

Results

EEG analyses revealed a significant main effect of contrast over contralateral occipital regions. Neural responses increased as stimulus contrast increased, indicating contrast-dependent modulation of visual processing. No significant main effect of attention was observed. Figure 2 illustrates representative SSVEP responses recorded from occipital channels.

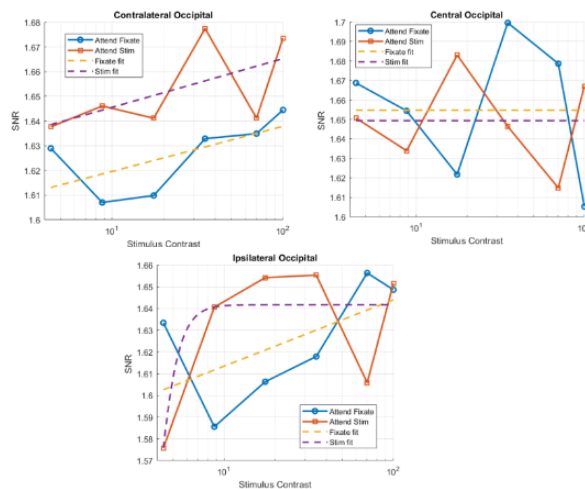


Figure 2. SSVEP analysis on occipital area.

For fNIRS, GLM analyses did not reveal significant main effects of attention or contrast across occipital channels. Similarly, no significant differences were observed between attend-fixate and attend-stimulus conditions in the fitted contrast-response parameters. Overall, hemodynamic responses showed substantial inter-individual variability and did not demonstrate a consistent modulation pattern across contrast levels. Figure 3 illustrate some fNIRS results from occipital area.

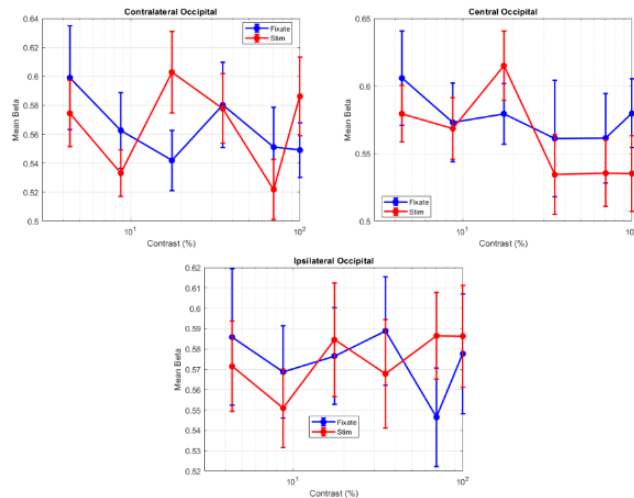


Figure 3. Beta values on occipital area

Discussion and Conclusion

The present study demonstrated significant EEG responses during the visual attention task, indicating that neural activity was sensitive to stimulus contrast. However, corresponding fNIRS responses did not reach statistical significance. This discrepancy may reflect differences in temporal sensitivity between electrophysiological and hemodynamic measurements, particularly under rapid event-related stimulation paradigms.

Although GLM analysis was employed to account for overlapping hemodynamic responses, the relatively short inter-stimulus interval may have reduced sensitivity to subtle contrast-dependent changes in HbO signals. Future work should investigate whether longer inter-trial intervals, optimized channel selection, or region-of-interest analyses improve the detection of hemodynamic modulation.

Limitation

Several limitations should be acknowledged. First, the study employed a rapid event-related design with short and jittered inter-stimulus intervals, which may have reduced the detectability of hemodynamic responses. Second, statistical analyses were performed on a channel-by-channel basis, potentially reducing sensitivity due to multiple-comparison effects. Finally, EEG-fNIRS coupling analyses were not performed in the present study and remain an important direction for future investigation.

Acknowledgements

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P015

FROM FAMILIAR FACES TO FOREIGN THREATS: FUSIFORM FACE AREA DEVELOPMENT, THE ONSET OF STRANGER ANXIETY, AND THE NEURODEVELOPMENTAL ROOTS OF SOCIAL BIAS

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Abstract:

Background: Stranger anxiety, wariness toward unfamiliar faces, emerges reliably in the second half of the first year and intensifies through toddlerhood. While well characterized behaviorally, its neurobiological substrate remains poorly understood. The Fusiform Face Area (FFA), central to face perception and familiarity processing, matures across this same period, making it a compelling candidate mechanism linking face-processing development to early social wariness.

Aim(s): This review examines the relationship between FFA neurodevelopment and the onset of stranger anxiety in toddlers, and evaluates a broader hypothesis: that FFA-mediated familiarity processing may contribute to the earliest psychological partitioning of social experience into "in-group" and "out-group" categories, with possible relevance to later social bias.

Methods: Following PRISMA guidelines, we searched PubMed/MEDLINE, PsycINFO, Web of Science, and Scopus, with Google Scholar and citation tracking. Boolean clusters combined: (1) "fusiform face area" OR "FFA" OR "face processing" OR "facial recognition" OR "fusiform gyrus"; (2) "stranger anxiety" OR "stranger wariness" OR "fear of strangers" OR "social wariness"; and (3) "infant" OR "toddler" OR "developmental" OR "neurodevelopment" OR "maturation". We included peer-reviewed empirical, review, and theoretical work, then critically assessed proposed causal links and methodological gaps.

Results: The literature is consistent with a framework in which a maturing face-recognition system consolidates a database of familiar individuals, such that unfamiliar faces may engage threat-response circuitry, manifesting as stranger anxiety. However, direct longitudinal evidence linking FFA development to social-categorization outcomes is scarce, and the inferential leap from normative stranger anxiety to adult xenophobia remains largely speculative.

Conclusion: Decoding early face-familiarity processing may illuminate developmental origins of in-group bias and inform cognitive-structural interventions delivered during sensitive windows. We outline a research agenda integrating developmental neuroimaging, behavioral assessment, and intervention science to test these claims rigorously.



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Keywords: Fusiform Face Area, stranger anxiety, face processing, developmental neuroscience

P016

THE EFFECT OF ALL-TRANS-RETINOIC ACID ON CELL DEATH IN ALZHEIMER'S DISEASE-LIKE MODEL

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Abstract:

Background: Alzheimer's disease (AD), the most prevalent neurodegenerative disorder, is characterized by cognitive decline, extracellular accumulation of β -amyloid ($A\beta$) plaques, and intracellular aggregation of neurofibrillary tangles (NFTs) composed of hyperphosphorylated tau. All-trans-retinoic acid (atRA), a known ligand for nuclear retinoic acid receptors (RAR/RXR), demonstrates significant efficacy in inhibiting cell death and enhancing survival. However, whether atRA modulates cell survival pathways in early-onset Alzheimer's disease (EOAD) remains poorly understood.

Aim(s): This project investigates the effect of atRA treatment on EOAD models, focusing on AD-related markers and cell survival pathways.

Methods: To assess the effects of atRA in EOAD, APPswe/Ind-overexpressing SH-SY5Y cells were treated with or without atRA for 72 h. Western blot analysis was performed to assess the expression levels of apoptosis and AD-associated markers.

Results: We found that APPswe/Ind-overexpressing SH-SY5Y cells led to an increase in the accumulation of pathological proteins involved in AD development. The ratio of pTau/total Tau, p53, and Bax significantly increased to $207.3 \pm 12.48\%$ ($p < 0.01$), $220.3 \pm 39.19\%$ ($p < 0.05$), and $213 \pm 35.41\%$ ($p < 0.05$), respectively, as compared to the control (non-overexpressing cells). While the level of Bcl-2, the anti-apoptotic marker, was reduced to $35.41 \pm 10.88\%$ ($p < 0.01$) when compared to the control. Treatment with atRA effectively reduced the levels of pTau/total Tau, p53 and Bax to $74.34 \pm 2.78\%$ ($p < 0.001$), $58.52 \pm 8.45\%$ ($p < 0.01$), and $78.74 \pm 6.6\%$ ($p < 0.05$), respectively, compared to untreated APPswe/Ind-overexpressing cells, while atRA increased the expression of Bcl-2 to $169 \pm 12.14\%$ ($p < 0.05$).

Conclusion: atRA can reduce AD pathology and apoptotic markers in EOAD. Therefore, atRA may be a good treatment option for EOAD in the future.

Keywords: Alzheimer's disease, All-trans-retinoic acid, Cell death, Neurofibrillary tangles, β -amyloid

P017

ANTIOXIDANT ACTIVITY OF AA001, A POTENTIAL ANTI-ALZHEIMER'S DRUG, BY THE DPPH ASSAY

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Abstract:

Background: Oxidative stress plays a central role in the pathogenesis of Alzheimer's disease (AD), contributing to neuronal damage, mitochondrial dysfunction, and amyloid- β aggregation. Compounds with free radical scavenging properties are therefore of interest as potential preventive or disease-modifying agents. AA001 is a novel compound in the aromatic dithiocarbamate group proposed as a candidate anti-Alzheimer's drug, but its antioxidant capacity has not been characterized.

Aim(s): This study aimed to evaluate the *in vitro* antioxidant activity of AA001 using the DPPH assay, with vitamin C (ascorbic acid) as a positive control.

Methods: The free radical scavenging activity of AA001 was determined by the 2,2-diphenyl-1-picrylhydrazyl (DPPH) assay. AA001 was tested across a concentration range of [0-100 μ g/mL], and the decrease in DPPH absorbance was measured at 517 nm after 30 min of incubation in the dark. Ascorbic acid served as the positive control. Percentage radical inhibition was calculated, and the half-maximal inhibitory concentration (IC₅₀) was derived from the concentration-response curve. Assays were performed in triplicate.

Results: AA001 showed concentration-dependent DPPH radical scavenging activity, with an IC₅₀ of 115.6202 ± 4.1329 μ g/mL, with significant difference from vitamin C (IC₅₀ = 976.2894 ± 73.5715 μ g/mL). These results indicate that AA001 possesses strong antioxidant capacity *in vitro*.

Conclusion: AA001 demonstrates potent antioxidant activity comparable to vitamin C, supporting its potential as an anti-Alzheimer's drug candidate capable of counteracting oxidative stress. Further studies are warranted to clarify its antioxidant mechanisms and to confirm neuroprotective efficacy in cellular and *in vivo* AD models.

Keywords: Alzheimer's disease, Antioxidant, Aromatic dithiocarbamate

P018

SHED-DERIVED EXOSOMES ALLEVIATE COGNITIVE IMPAIRMENT AND ASTROCYTE ACTIVATION FOLLOWING GLOBAL ISCHEMIC STROKE INDUCED BY CHRONIC CEREBRAL HYPOPERFUSION IN RATS

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Abstract:

Background: Global ischemic stroke, characterized by a critical reduction of cerebral blood flow, induces selective neuronal injury in the hippocampus, a brain region vital for learning and memory. Current therapeutic strategies remain limited. Stem cells from human exfoliated deciduous teeth (SHED)- derived exosomes have emerged as promising cell-free therapeutics due to their neuroprotective and regenerative properties.

Aim(s): To evaluate whether SHED-derived exosomes improve cognitive deficits and reduce astrocyte activation following chronic cerebral hypoperfusion.

Methods: Male Sprague-Dawley rats underwent modified bilateral common carotid artery occlusion (BCCAO) and received either vehicle or SHED-derived exosomes (100 µg, i.v.). Neurological function was evaluated using mNSS, while spatial learning and memory were assessed by the Morris water maze. Hippocampal astrocyte activation was examined by GFAP Western blot analysis. All animal procedures were approved by the Mahidol University Institute Animal Care and Use Committee (COA.NO.IMB-ACUC 2024/010).

Results: BCCAO induced significant neurological deficits, spatial learning and memory impairment, and increased hippocampal GFAP expression. SHED-derived exosome treatment significantly improved cognitive performance in the Morris water maze, as evidenced by reduced escape latency, increased time spent in the target quadrant, and increased platform crossings. Furthermore, SHED-derived exosomes markedly attenuated GFAP expression in the hippocampus.

Conclusion: SHED-derived exosomes improve spatial learning and memory deficits and attenuate hippocampal astrocyte activation following chronic cerebral hypoperfusion. These findings suggest that SHED-derived exosomes may represent a promising cell-free therapeutic approach for cognitive impairment associated with global ischemic stroke.

Keywords: Global ischemic stroke, Chronic cerebral hypoperfusion, SHEDs-derived exosomes, Spatial learning and memory, Astrocyte activation

P019

NANOZYME-ENHANCED PAPER-BASED SENSOR FOR SALIVARY α -AMYLASE DETECTION: A NON-INVASIVE PLATFORM FOR STRESS ASSESSMENT

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Abstract:

Background: Stress and anxiety disorders are increasingly prevalent and commonly assessed using subjective questionnaires or self-reporting, which may limit diagnostic accuracy and real-time monitoring. Salivary α -amylase is a non-invasive physiological biomarker associated with sympathetic nervous system activation and has potential for objective stress assessment.

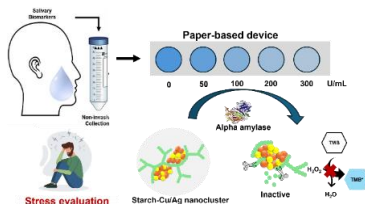
Aim(s): This study aimed to develop a portable paper-based colorimetric biosensor for rapid detection of salivary α -amylase for stress monitoring.

Methods: A starch-stabilized Cu/Ag nanocluster was synthesized and used as a nanozyme catalyst for the TMB-H₂O₂ colorimetric reaction. The nanocluster was characterized using UV-Vis spectroscopy and transmission electron microscopy. The nanozyme was then integrated into a paper-based analytical device. Analytical performance was evaluated through assay optimization, calibration, selectivity testing, interference studies, and recovery analysis using artificial saliva.

Results: The device enabled visual and quantitative detection of α -amylase without specialized laboratory equipment. It showed a linear detection range of 5–500 U/mL, with a limit of detection of 1.3 U/mL. Negligible matrix interference and acceptable recovery values confirmed its reliability.

Conclusion: This nanozyme-driven paper-based biosensor offers a low-cost, portable, and non-invasive platform for point-of-care stress assessment.

Keywords: Salivary α -amylase; Stress screening; Paper-based analytical device; Colorimetric sensor; Nanoclusters



P020

UPREGULATION OF SOX2 AND NCAM EXPRESSION BY ANDROGRAPHOLIDE IN A ZIKV- INFECTED CHICK EMBRYO MODEL

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Abstract:

Background: Andrographolide (Andro), its potential to be used as a broad-spectrum anti-flavivirus infection drug, has optimum binding affinity with non-structural protein including NS3 helicase, NS3 proteases, and NS5 of Japanese encephalitis virus. Absorption, distribution, metabolism, excretion, and toxicity analysis has revealed good pharmacokinetic and safety profiles for Andro. Zika virus (ZIKV) is a mosquito-borne flavivirus that causes severe neurological disorders, such as microcephaly in fetuses. The ZIKV NS2B-NS3 protease, which is involved in viral replication, has been considered as a promising target for Zika virus drug development. Andro has also been reported the inhibitory effects on viral spread by the interactions at this target protein, ZIKV NS2B-NS3 protease.

Aim(s): To determine whether Andro regulates embryonic neurogenesis in a ZIKV-infected chick embryo model.

Methods: Embryos were divided into three groups: Control, ZIKV-infected, and ZIKV-infected group treated with andrographolide, with 10 embryos per group. Fertilized chicken eggs were inoculated via the yolk sac (YS) at embryonic day 2 (E2) with 10^4 FFU/mL of ZIKV, followed by treatment with 5 μ M Andro at E3 via YS. The eggs were incubated until E6 or E14. Brain tissues were collected for histopathological evaluation with H&E staining and immunofluorescence staining. Images were quantified using ImageJ. Data were analyzed using one-way ANOVA, with $p < 0.05$ considered statistically significant.

Results: ZIKV-infected embryos exhibited abnormal growth and reduced brain size (68.38 ± 9.83 mm²). Neuronal cell death and decreased expression of neuronal proteins including NCAM and Sox2 were observed in the brain tissues. Sox2 is a key regulator of neural stem cell maintenance and self-renewal, whereas NCAM is highly expressed during axonal growth and synaptogenesis in mature neurons. Treatment with 5 μ M Andro improved brain size (76.01 ± 3.04 mm²) and cortical thickness (837.1195 ± 29.66 μ m), supporting normal embryonic development from E6 to E14. At E6, Andro treatment increased Sox2 immunoreactivity in the optic tectum (26.20 ± 2.59 %Area)

compared with untreated ZIKV-infected group. At E14, Andro-treated group showed an apparent increase in NCAM immunoreactivity in the brain tissue (60.82 ± 3.72 %Area) compared to ZIKV-infected group.

Conclusion: Andrographolide treatment attenuated ZIKV-associated alterations in embryonic and brain morphology and partially restored Sox2 and NCAM immunoreactivity in infected chick embryos. These findings suggest a potential protective effect of andrographolide against ZIKV-associated neurodevelopmental alterations; however, its antiviral and neurogenic mechanisms require further investigation.

Keywords: Neurogenesis, Andrographolide, NCAM, Sox2, ZIKV infection

P021

OPTIMIZATION OF ZIKA INFECTION AND EVALUATION OF ANDROGRAPHIS PANICULATA EXTRACT IN EMBRYONATED CHICKEN EMBRYOS

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Abstract:

Background: Zika virus (ZIKV) can replicate in embryonated chicken eggs, similar to human infection during the first trimester of pregnancy although the cell types and tissues supporting ZIKV replication differ. Previous studies demonstrated that ZIKV replicates in embryonic brain tissue following yolk sac inoculation at E2.5 and E5 during early neurogenesis, resulting in delayed hatching and neurological signs after hatching. Differential susceptibility to timing and dose of ZIKV remains to be understood during human pregnancy, and these questions could be refined and narrowed with a chicken ZIKV embryo model. This *in vivo* model is relatively fast, inexpensive, ease of manipulation, and suitability for direct manipulation.

Aim(s): To (i) determine appropriate dose ranges and assess baseline outcomes, including embryo survival and compound-associated toxicity, (ii) evaluate the feasibility of co-administration of ZIKV, *Andrographis paniculata* extract (APE), and andrographolide (Andro).

Methods: Embryonated chicken eggs (n = 5/group) were used. ZIKV (10¹–10⁵ FFU/mL; 100 µL) was inoculated into the yolk sac at embryonic day 2 (E2). APE (2, 6, and 8 mg/mL) and the positive comparator, Andro (5, 10, and 20 µM) were administered via the same route at embryonic day 3 (E3), one day after ZIKV inoculation. Embryo survival and gross morphology, and liver histology were assessed.

Results: Embryo survival differed among viral doses, with survival rates of 100% at ZIKV, 10¹–10² FFU/mL, 66.6% at 10³ FFU/mL, 75% at 10⁴ FFU/mL, and 25% at 10⁵ FFU/mL. A dose of 10⁴ FFU/mL was selected as the working concentration for subsequent analyses. APE at 2 and 6 mg/mL no signs of toxicity were observed whereas 8 mg/mL showed adverse effects. Andro at 20 µM reduced hepatic cell density and dilation of venous canals and blood sinusoids, while lower doses (5, 10 µM) showed no apparent abnormalities.

Conclusion: These findings demonstrate that embryonated chicken eggs can serve as a feasible platform for dose optimization of ZIKV infection and compound testing. A viral dose of 10⁴ FFU/mL provided a suitable balance between embryo survival

and reproducible developmental and brain pathological changes, making it appropriate for evaluating treatment efficacy. Low-dose APE (2 mg/mL) and andrographolide (5 μ M) were identified as non-toxic conditions suitable for subsequent efficacy studies.

Keywords: Zika virus (ZIKV), Chicken embryo model, Andrographis paniculata, Andrographolide

P022

PRELIMINARY BEHAVIORAL AND ELECTROENCEPHALOGRAPHIC EVALUATION OF ASARININ IN NORMAL MICE

Myat Ko

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Abstract:

Background: Asarinin is a naturally occurring lignan isolated from medicinal plants of the Asarum genus and has been reported to possess various neuroprotective properties. Recently, virtual screening conducted by our research group identified Asarinin as a potential dual orexin receptor antagonist (DORA), suggesting a possible role in regulating sleep-wake processes. However, the in vivo effects of Asarinin on behavioral and electroencephalographic (EEG) activity remain unknown.

Aim(s): To evaluate the sedative-like effects of Asarinin using behavioral and EEG assessments in normal mice and to compare its effects with those of the dual orexin receptor antagonist, Lemborexant, a medication used to treat insomnia.

Methods: Adult male mice (6 weeks old) were assigned to vehicle (normal saline solution), Asarinin (10 mg/kg), and Lemborexant (10 mg/kg) (n = 2 per group). Locomotor activity was evaluated using the open field test for 30 min following a single intraperitoneal injection. EEG activity was recorded before treatment (baseline) for 24 hours and for 24 hours after drug administration. Preliminary analyses included locomotor activity parameters and EEG activity profiles.

Results: Both Asarinin- and Lemborexant-treated mice exhibited reduced locomotor activity compared with vehicle-treated controls. Total distance traveled, mean velocity, and time spent in highly active states showed a decreasing trend following drug administration, whereas inactive duration increased in the treatment groups. Lemborexant produced the greatest reduction in locomotor activity, while Asarinin demonstrated an intermediate effect. Although these trends were consistent across behavioral parameters, no statistically significant differences were observed among groups, likely due to the limited sample size of this pilot study. Preliminary EEG recordings further demonstrated reduced activity profiles in both treatment groups compared with vehicle controls. Further analyses of EEG spectral power and sleep architecture are ongoing.

Conclusion: Preliminary findings suggest that Asarinin may produce behavioral and EEG effects comparable to Lemborexant, underscoring its potential for further development as a novel compound or drug for the treatment of insomnia. Further studies with larger sample sizes and detailed EEG analyses are warranted.

Keywords: asarinin, EEG, dual orexin receptor antagonist, sleep regulation, locomotor activity

P023

ROLE OF CN8 MYELINATION IN ASD PATHOLOGY

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Abstract

Background: Sensory processing abnormalities affect up to 90% of individuals with Autism Spectrum Disorder (ASD). While CNS myelination abnormalities in ASD are documented, whether peripheral myelination of the vestibulocochlear nerve (CN VIII) contributes to auditory/vestibular symptoms has not been yet posited.

Aim(s): To determine whether CN VIII conduction integrity differs between ASD and typically developing (TD) individuals across development, and whether this correlates with sensory symptom severity. We hypothesize ASD participants will show CN VIII conduction abnormalities relative to TD controls that scale with symptom severity, reflecting dysmyelination (hypo- vs. hypermyelination).

Myelination Assessment: Direct in vivo imaging of CN VIII myelin is not resolvable with standard clinical MRI. Conduction velocity will serve as its physiological correlate: ABR interpeak latencies/amplitudes will index cochlear nerve conduction; cervical/ocular VEMP will index vestibular nerve conduction. Abnormalities will be interpreted as dysmyelination broadly, not presumed hypomyelination, since hypermyelination can also impair conduction.

Methods: A planned two-arm cross-sectional study. *Arm 1 (in silico):* analysis of open-access ASD diffusion/structural MRI datasets for g-ratio/myelin-water-fraction proxies; GROMACS simulation of myelin lipid structure; and biophysical modeling of a myelinated CN VIII fiber in NEURON, built as a node-of-Ranvier/internode cable model with Hodgkin-Huxley kinetics and myelin-dependent internodal capacitance, swept across physiological and pathological g-ratios to generate conduction-velocity predictions under distinct hypo- vs. hypermyelination scenarios. *Arm 2 (clinical):* 60 participants (ages 8–12, 18–22), ASD (DSM-5-confirmed) vs. TD, undergoing ABR/VEMP testing. Group differences via t-tests/ANOVA/MANCOVA (age-adjusted); regression for symptom-severity association; Bonferroni correction.

Anticipated Contribution: Preliminary simulations show thicker myelin alone increases conduction velocity, contradicting a simple "hypermyelination slows conduction" account and implicating additional structural disruption. Future modeling framework will generate a more robust conduction-velocity predictions to interpret forthcoming ABR/VEMP data and resolve the direction of CN VIII dysmyelination in ASD.

P024

SIMULTANEOUS ISOLATION OF THREE PRIMARY EMBRYONIC RAT BRAIN CELL TYPES FROM A SINGLE LITTER: A REFINED PROTOCOL SUPPORTING REDUCTION AND CHARACTERIZATION OF NEURONAL, ASTROCYTIC, AND MICROGLIAL PHENOTYPES

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Abstract:

Background: Primary embryonic rat brain cells are widely used models in neuroscience research; however, conventional isolation protocols typically require separate animal cohorts to obtain distinct cell populations, raising concerns about inter-batch biological variability and excess animal use.

Aim(s): This study aimed to develop and validate a simultaneous isolation protocol yielding three primary cell types, hippocampal neurons, cortical astrocytes, and cortical microglia from a single embryonic rat litter (E16–E18), in alignment with the 3R principles of Reduction and Refinement.

Methods: Embryonic rats (E16–E18) from a single litter were used. Hippocampi were dissected for neuronal isolation, while cortical tissue was processed for astrocyte and microglial isolation in parallel. Cells were cultured under standard conditions and characterized by immunofluorescence microscopy. Neurons were identified using PSD-95 (postsynaptic density marker); astrocytes using GFAP; and microglia using Iba-1 as a pan-microglial marker. Microglial phenotype was assessed by stimulating cultures with lipopolysaccharide (LPS) to induce M1 polarization (iNOS, CD86) or with IL-4 for M2 polarization (Arg-1). Morphological characterization was performed on all three cell types.

Results: All three cell populations were successfully isolated from a single embryonic rat litter. Hippocampal neurons displayed characteristic morphology with confirmed synaptic marker expression (PSD-95). Cortical astrocytes exhibited typical stellate morphology with robust GFAP immunoreactivity. Microglia were confirmed by Iba-1 staining and demonstrated plasticity in phenotypic polarization, expressing iNOS and CD86 under M1-inducing conditions and Arg-1 under M2-inducing conditions. Cell viability and morphological integrity were consistent across all populations.

Conclusion: This refined simultaneous isolation protocol provides a practical and reproducible approach for obtaining neurons, astrocytes, and microglia from a single embryonic rat litter, substantially reducing inter-batch variability and animal use. This platform supports future neuroinflammation and neuroprotection studies requiring multi-cell-type experimental designs.

Keywords: primary cell isolation, embryonic rat brain, neuron, microglia, astrocyte, 3R principles

P025

THE MEDIATING ROLE OF KYNURENINE IN THE ASSOCIATION BETWEEN PHYSICAL FITNESS AND COGNITIVE FUNCTION: IMPLICATIONS FOR COGNITIVE RESILIENCE IN AGING

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Abstract:

Background: Kynurenine pathway (KYN), an inflammation-related metabolic pathway, has been implicated in neurodegeneration and cognitive impairment. Cognitive decline in aging populations is a major public health challenge with no effective pharmacological or supplement-based treatment. Physical fitness is currently recommended to improve early cognitive impairment, but whether kynurenine metabolites mediate the association between physical fitness and cognitive function remains unclear.

Aim: To examine whether sweat KYN mediates the association between physical fitness and cognitive function among pre-elderly and older adults.

Methods: This cross-sectional study included 60 healthy Thai people aged 45–86 years. Participants with neurodegenerative diseases or anti-inflammatory use within 24 hours were excluded. KYN were derived from sweat collected during physical fitness tests and analyzed the metabolites by HPLC, with sodium measured by MP-AES for normalization. Cognitive functions were assessed by Mini-Cog test. Path analysis was conducted to examine the mediating role of KYN in these associations, controlling for age.

Results: Individuals with better physical fitness in strength domain demonstrate lower sweat level of KYN ($\beta = -0.262$, $p = .036$), which are in turn relatively strong association with better cognitive function ($\beta = -0.431$, $p < .001$). The indirect effect of physical fitness on cognition through KYN was significant ($\beta = 0.113$, $p = .031$, 95% CI [0.010, 0.215]), accounting for 60.1% of the total association. The association between physical fitness and cognitive function was fully mediated by KYN, with no evidence of a direct effect ($\beta = 0.076$, $p = .522$).



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Conclusion: Sweat KYN significantly mediated the association between physical fitness and cognitive function in pre-elderly and older adults, supporting its potential as a non-invasive biomarker of cognitive resilience. Enhancing physical fitness may help modulate KYN activity and promote cognitive reserve in aging populations.

Keywords: Kynurenine, Physical fitness, Cognitive function, Older Adults

P026

ANTHOCYANIN-RICH THAI COLORED RICE EXTRACTS IN A MOUSE MODEL OF VASCULAR COGNITIVE IMPAIRMENT

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Abstract:

Background: Vascular cognitive impairment caused by cerebral hypoperfusion results in cell excitotoxicity, release inflammatory cytokines releasing, and microglial activation. While white rice (WR) is a dietary staple, anthocyanin-rich variants like Black Glutinous (BR) and Riceberry (RR) rice possess many antioxidant properties.

Aim(s): This study investigated whether BR and RR could attenuate neuroinflammation and cognitive deficits in a modified bilateral common carotid artery occlusion (mBCCAO) mouse model and whether they were improved by the more total phenolic and pro-anthocyanidin contents in colored rice extracts.

Methods: Forty male mice were subjected to mBCCAO or sham surgery and treated with WR, RR, or BR extracts (12.5 mg/kg). Cognitive performance was evaluated via Morris water maze and trace fear conditioning tests, and neuroinflammation, myelin integrity, and microglial markers were assessed using ELISA, Western blot, and immunohistochemistry.

Results: Colored rice significantly improved cognitive performance in the Morris water maze and trace fear conditioning tests compared with the occlusion group ($p < 0.05$). Colored rice also significantly increased hippocampal p-ERK1/2 expression, a marker associated with long-term memory formation, relative to the occlusion group. In addition, colored rice attenuated white matter damage, as indicated by increased MBP expression (RR: 1.11 ± 0.10 ; BR: 0.60 ± 0.02 , vs. 0.47 ± 0.03 in the occlusion group; approximately 2.4-fold and 1.3-fold increases, respectively; $p < 0.05$), and reduced IBA1 immunoreactivity in the hippocampus, suggesting reduced microglial reactivity. Colored rice also attenuated the mBCCAO-induced increase in the pro-inflammatory cytokine TNF- α (occlusion: 4.11 ± 0.1 pg/mg vs. sham: 0.56 ± 0.03 pg/mg; $p < 0.001$), reducing levels to 0.583 ± 0.01 pg/mg with RR and 0.16 ± 0.1 pg/mg with BR. These findings suggest that the colored rice ameliorates cognitive decline and white matter damage, potentially through modulation of microglia activation and TNF- α , which may be attributable to its higher total phenolic and pro-anthocyanidin contents compared with white rice.

Conclusion: Anthocyanin-rich Thai colored rice (Riceberry and Black Glutinous rice) attenuated neuroinflammation, white matter damage, and cognitive deficits in a mouse model of vascular cognitive impairment, likely through modulation of microglial activation and TNF- α associated with their higher phenolic and pro-anthocyanidin content. These findings support colored rice as a dietary candidate warranting further investigation for mitigating vascular cognitive impairment.

Keywords: Colored Rice, Microglia activation, Vascular Cognitive Impairment

P027

FROM FUNCTIONAL FOODS TO NANOMEDICINE: HARNESSING PLANT-DERIVED NANOVESICLES TO COMBAT AGE-RELATED NEURODEGENERATION

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Abstract:

Background: Age-related neurodegenerative diseases are driven by oxidative stress and chronic neuroinflammation, while current therapies remain limited by poor blood–brain barrier (BBB) penetration and concerns regarding immunogenicity. Plant-derived nanovesicles (PDNVs) have emerged as promising natural nanotherapeutics owing to their low immunogenicity, excellent biocompatibility, potential to cross BBB, intrinsic bioactive cargo, cost-effective production, and scalability from renewable plant sources. Among medicinal and edible plants, *Bacopa monnieri* (Brahmi) possesses well-established cognitive-enhancing properties, whereas *Wolffia globosa* (Khai-Phum) is a sustainable functional food rich in bioactive compounds, making both attractive sources of neuroprotective PDNVs. However, their nanovesicles have not yet been isolated and evaluated for neuroprotective activity

Aim: This study investigated the neuroprotective potential of PDNVs isolated from Brahmi and Khai-Phum.

Methods: PDNVs were isolated by sequential centrifugation and ultrafiltration, followed by characterization using nanoparticle tracking analysis, zeta potential measurement, and cryogenic electron microscopy. Cellular uptake was examined by immunocytochemistry. Untargeted proteomic and metabolomic analyses were performed to identify bioactive cargo, while intrinsic antioxidant activity was evaluated using DPPH and FRAP assays. Neuroprotective effects were assessed in lipopolysaccharide (LPS)-, hydrogen peroxide (H₂O₂)-, and methylglyoxal (MGX)-induced injury models using human

HMC3 microglia, mouse SIM-A9 microglia, and mouse catecholaminergic CAD neurons. Cell viability (MTT), intracellular ROS (DCFH-DA), and nitric oxide production (Griess assay) were evaluated.

Results: Both Brahmi- and Khai-Phum-derived nanovesicles exhibited nanoscale size, a characteristic lipid bilayer structure, and negative surface charge. Fluorescently labeled PDNVs were efficiently internalized by recipient brain cells, supporting their biocompatibility. OMIC-based cargo profiling identified abundant antioxidant-associated metabolites and proteins, consistent with strong intrinsic antioxidant activity. Brahmi-derived nanovesicles significantly protected HMC3 microglia against H₂O₂-induced cell death and suppressed LPS-induced nitric oxide production in SIM-A9 cells, whereas Khai-Phum-derived nanovesicles attenuated MGX-induced neurotoxicity and cellular ROS accumulation in CAD neurons. Further mechanistic and in vivo studies will be important to support their development as plant-based nanotherapeutics for neurodegenerative diseases.

Keywords: Plant-derived nanovesicles, *Bacopa monnieri*, *Wolffia globosa*, Interkingdom interaction, Nanotherapeutics

P028

BIOACTIVE COMPOUNDS PROFILE RELATED TO ACETYLCHOLINESTERASE INHIBITORY ACTIVITIES AND THE EFFECT OF AIR-COLD PLASMA JET ON THE BIOACTIVE COMPOUNDS PROFILE OF EDIBLE CHINESE PINK FLOWER (DIANTHUS CHINENSIS L.)

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Abstract:

Background: Chinese pink flower is a medicinal edible flower. Cold plasma (CP) pretreatment enhances the extraction of plant bioactive compounds.

Aim(s): This study investigated the bioactive compounds profile and biological activities across flower colours. The red and pink flowers contain higher phenolic and flavonoid contents and exhibit greater antioxidant potency than the white flowers.

Methods: Importantly, (1) Acetic acid, 3-(6,6-dimethyl-2-methylenecyclohex-3-enylidene)-1-methylbutyl ester and (2) 3-O-Methyl-d-glucose are found in the flowers, and these compounds have the potential acetylcholinesterase (AChE) correlates with the content.

Results: The docking study reveals that (1) binds to the active sites of AChE with binding affinities and inhibition constant (K_i) of -8.4 kcal/mol and (0.7 μ M), respectively. Furthermore, air-cold plasma jet (CPJ) pretreatment enhances the phenolic and flavonoid contents of the flowers. These results indicate that the flowers are rich in bioactive compounds and exhibit inhibitory activities against AChE.

Conclusion: The air-CPJ pretreatment method is appropriate for extracting bioactive compounds from plant samples.

Keywords: Edible flower, Bioactive compound, Non-communicable disease, Air-cold plasma jet

P029

GAMBOGIC ACID DERIVED FROM GARCINIA HANBURYI IMPROVES GUT BARRIER INTEGRITY AND BRAIN FUNCTION IN TYPE 2 DIABETIC RATS

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Abstract:

Background: Type 2 diabetes mellitus (T2DM) is a chronic metabolic disease characterized by insulin resistance and systemic inflammation. This condition is associated with complications affecting multiple organs, particularly the brain and gastrointestinal tract. T2DM often involves decreased BDNF levels, increased systemic inflammation and compromised intestinal barrier integrity. Gambogic acid (GA) has shown potential anti-inflammatory and protective effects in various models, but its impact on the gut-brain axis in T2DM remains uninvestigated.

Aim(s): This study aimed to investigate the therapeutic effects of GA on gut and brain integrity in a streptozotocin-induced T2DM rat model.

Methods: Eight-week-old male rats were fed a high-fat diet and subsequently received a single intraperitoneal injection of streptozotocin (STZ; 35 mg/kg body weight) to establish a T2DM rat model. DM rats were treated once daily with gambogic acid (GA; 3 mg/kg, i.p.). We assessed behavioral changes using the open field test, levels of biochemical markers, duodenal histology by hematoxylin and eosin staining, and protein levels of tight junctions by Western blot analysis.

Results: GA improved behavioral outcomes, increased serum BDNF levels, and significantly suppressed systemic inflammation by reducing serum IL-6 levels. GA treatment significantly restored duodenal architecture, increasing villus height, width, and goblet cell populations while decreasing crypt depth compared to the diabetic group. Furthermore, molecular analysis discovered that GA significantly upregulated the level of tight junction proteins Zonula Occludens 1 (ZO-1) and Occludin.

Conclusion: These findings suggest that GA may preserve intestinal barrier integrity and attenuate T2DM-associated neuroinflammation, thereby supporting its potential protective role against diabetes-related complications.

Keywords: Gambogic acid, Type 2 diabetes, Gut-brain axis, Intestinal barrier, Neuroinflammation

Background

Diabetes mellitus (DM) is a serious and chronic condition characterized by the development of hyperglycemia from either a defect in insulin secretion or decreased

insulin sensitivity (Association, 2013). The onset of diabetes can be influenced by genetic inheritance and environmental factors including, high-fat diet and low physical activity, and stress (Khudhair Alhadrawi et al., 2025; Nolan et al., 2011; Ostenson, 2001). Diabetes mellitus (DM) comprises two major types: type 1 diabetes mellitus (T1DM) and type 2 diabetes mellitus (T2DM), with T2DM accounting for 90% of all cases (Ruze et al., 2023). T2DM is caused by the combination of both primary factors, including the tissues respond inappropriately to the presence of insulin, and pancreatic β -cells dysfunction (Galicia-Garcia et al., 2020). Insulin resistance is a pathological condition which reduced insulin responsiveness of tissues lead to disruption of glucose and metabolic regulation and increase the risk of metabolic syndrome and T2DM (Seibenhener & Wooten, 2015). The complications of T2DM are associated with several vital organs especially brain, nervous system, and digestive system (Reed J, 2021). The neurological complications of T2DM are shown in several studies. T2DM is linked directly to physiological responses within the nervous system, including oxidative stress, neuroinflammation, death of neuronal cells and synaptic alteration (Ranjan et al., 2026). Furthermore, T2DM significantly increases the risk of neurological disorders through chronic inflammation and insulin resistance, both of which contribute to dysfunction of neurons, neuropathy, cognitive decline, and depression (Amalliah et al., 2026). Hyperglycemia-induced systemic inflammation can impair brain function and contribute to altered emotional regulation, anxiety, and depressive symptoms through neuronal damage (Xu & Chen, 2025). Furthermore, insulin resistance disrupts energy metabolism, thereby adversely affecting the brain, a highly energy-demanding organ. (Zhao et al., 2023).

Brain cells use glucose as the main source of energy to support neuronal activity and synaptic plasticity (Mergenthaler et al., 2013). Brain-derived neurotrophic factor (BDNF) is a neurotrophin generally expressed throughout the nervous system, particularly in the brain. BDNF serves as a key biomarker of neuronal survival and neuroplasticity and is also involved in the maintenance of pancreatic β -cell function by activating tropomyosin receptor kinase B (TrkB)-mediated signaling, which enhances glucose-stimulated insulin secretion, promotes β -cell survival, and supports glucose homeostasis (Fulgenzi et al., 2015). Reduced BDNF levels are associated with increased anxiety-like behaviors and impaired stress-coping ability (Rosenhauer et al., 2019). Several studies have reported significantly lower serum BDNF levels in individuals with diabetes mellitus, diabetes-associated depression, and T2DM-related cognitive impairment compared to normal controls (He et al., 2024; Moosaie et al., 2023). The systemic inflammation from DM, usually indicated by the level of inflammatory cytokines that circulate in the system. Previous studies showed that in T2DM patients, there is an increase in pro- and anti-inflammatory cytokine such as tumor necrosis factor alpha (TNF α) and interleukin group including IL-4, IL-6, IL-10, IL-17, and IL-18 (Cao et al., 2024; Velikova et al., 2021). Especially, IL-6 can be used as a biomarker for T2DM (Velikova et al., 2021).

The small intestine serves as a primary organ for nutrient and mineral absorption. The duodenum is the first part of the small intestine that is the initial site for post-gastric digestion, it has unique roles in sensing nutrient intake and coordinating metabolic signaling pathways (Lerkdumnerkit et al., 2022). In the gut, hyperglycemia and IR events lead to the alteration of gut health, including changes in mucus, increased gut permeability and decreased tight junction integrity between intestinal epithelial cells, which allows toxic substances to invade the system (Daryabor et al., 2020). The gut-brain axis is a bidirectional complex that linking between the gastrointestinal tract and the central

nervous system. (Kearns, 2024). T2DM involved with dysregulation of gut composition and chronic low-grade inflammation, which decreases intestinal integrity and further causes leaky gut. This disruption facilitates the translocation of endotoxins, such as lipopolysaccharide (LPS), into the circulation, thereby exacerbating systemic inflammation and contributing to neurological dysfunction. (Boyadzhyan et al., 2026; Rai et al., 2023).

Conventional treatments for T2DM, patients are primarily given oral antidiabetic drugs. However, these treatments are often associated with adverse effects and are still unable to fully control glucose homeostasis (Xu et al., 2018). Thus, alternative treatments for diabetic patients are still needed to minimize the limitations of current treatments. Gambogic Acid (GA) is a natural compound derived from the *Garcinia hanburyi* tree (Gilani et al., 2020). GA has been reported to exert multiple biological activities against cancer progression, such as anti-metastatic, anti-inflammatory, autophagic, pro-programmed cell death, anti-angiogenic, and cell cycle arrest effects (Ishaq et al., 2014; Kashyap et al., 2016; Seo et al., 2019). Recently, Thongrungsri and colleagues have reported that GA evidently improved the structure of renal and function in diabetic rats and exhibited renal protective effects by reducing oxidative stress and inflammation in diabetic nephropathy (Thongrungsri et al., 2025). However, the effects of GA on gut-brain integrity in diabetic models have not been investigated. Therefore, this study aimed to investigate the effects of GA on the gut-brain integrity in T2DM rats.

Materials and Methods

Animal and Treatment

Animal studies were conducted at the Animal Center of Thammasat University, Thailand, under an approved animal care protocol (No. 021/2021). Twenty-five male Wistar rats (8 weeks of age; 200–220 g) were used in this study. To induce diabetes, rats were injected with a single intraperitoneal injection of streptozotocin (35 mg/kg body weight, and DM-induced rats were maintained on a high-fat diet throughout the study period (Lapmanee et al., 2024). Diabetic status was confirmed one week after STZ administration by measuring fasting blood glucose levels, with values exceeding 180 mg/dL considered indicative of diabetes (Thongrungsri et al., 2025). Gambogic acid was administered intraperitoneally at a dose of 3 mg/kg body weight/day for four weeks. Rats were randomly assigned to one of three groups: control, diabetic, or diabetic treated with 3 mg/kg GA. After treatment, rats were euthanized, and the brain and duodenum were harvested, immediately frozen, and stored at -80°C for further analyses. All downstream experiments were performed at the Faculty of Science, Mahidol University, Thailand (Protocol No. MUSC68-031-801).

Open field test (OFT)

The open field test was performed in a box (76×57×35 cm), with a grid on the floor that was separated into inner and outer zones. An individual rat was placed into the corner of the box and given time to explore the box for 5 min. Behavioral parameters, including time spent in the inner zone and total line crossings, were automatically quantified using ANY-maze behavioral tracking software (Stoelting Co., Wood Dale, IL, USA). Automated video analysis minimized observer bias and manual counting errors. All videos were analyzed using identical tracking parameters by an investigator blinded to the treatment groups to reduce sampling bias. The total line crossings were used as an indicator for locomotor activity (total movement), and the time spent in the inner zone

serves as an indicator of anxiety-like behavior, where less time spent in the center typically suggests higher anxiety (Seibenhener & Wooten, 2015) .

Evaluation of biochemical markers in serum

Enzyme-linked immunosorbent assay (ELISA) kits (Abcam, Cambridge, UK) were used to analyze the levels of brain-derived neurotrophic factor (BDNF) and interleukin-6.

Histological analysis

The duodenum part was embedded in paraffin and stained with hematoxylin and eosin (H&E) to observe the destruction of the duodenum. Villus height and width, crypt depth, goblet cell and villus-to-crypt (VC) ratio were determined by the following formula:

$$\text{VC Ratio} = \frac{\text{Villus Height}}{\text{Crypt Depth}}$$

Western blot analysis for protein analysis

After 4 weeks of treatment, duodenum part was removed for protein expression. 1X RIPA buffer (Abcam) with phenylmethylsulfonyl fluoride (PMSF) were added into grinded duodenum tissues. The BCA Protein Assay kit (Thermo Scientific, USA) was used to determine the protein concentration. 20 ug protein of each sample was added into each well of 10% Gel SDS-PAGE, running gel was transferred onto nitrocellulose membranes (Merck Millipore, Ireland) with 100 mV for 1.30 hr. The membranes were incubated with 1:1,000 Rat monoclonal anti-ZO-1 antibody (catalog no. sc-33725), 1:1,000 Mouse monoclonal anti-Occludin antibody (catalog no. sc-271842) (Santa Cruz Biotechnology, USA), or 1:5,000 rabbit monoclonal anti-β-actin antibody (catalog no; 4970S) (Cell Signaling, Danvers, USA) at 4°C overnight. The membranes were washed and incubated with 5% non-fat dry milk with 1:5,000 secondary antibodies linked to rat and mouse horseradish peroxidase (HRP) for 1 hr at 25°C. An enhanced chemiluminescence (ECL) substrate was added onto each membrane. The protein bands were placed on the black tray and detected using an Azure 600 Gel Imaging System. The intensity of the protein band was determined by the ImageJ program. The protein level relative to β-actin in the control was normalized to 1.

Statistical Analysis.

All statistical analysis were performed with GraphPad Prism (Version 9.1.1), using one-way ANOVA. Data are presented as mean ± SEM.

Results

GA exerts neuroprotective effects in a T2DM rat model

After 4 weeks of treatment with GA in T2DM rats, changes were observed in both the behavioral assessments (Figure 1). Behavioral assessment demonstrated by OFT, the result showed higher anxiety-like behaviors across both tests in DM. Compared with controls, DM rats had a significantly lower total crossing line ($p < 0.001$) (Figure 1A) and time spent in the inner zone of the box ($p < 0.001$) (Figure 1B). Notably, four weeks of GA treatment (3 mg/kg BW) significantly improved locomotor activity and exploratory behavior compared with the DM group ($p < 0.001$) (Figure 1A-B). These results demonstrate that GA significantly mitigates anxiety-like behaviors and enhances neurotrophic support.

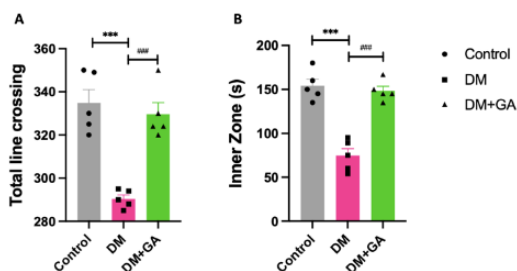


Fig. 1. The effect of Gambogic acid (GA) on behavioral changes in T2DM rats. Graphs showing quantitative data for behavioral assessments: (A) Total line crossing and (B) Time spent (s) in inner zone activity in the open field test. Data are presented as mean $\pm$ SEM. Statistical significance is indicated by symbols where *** $p < 0.001$ compared to control, and ### $p < 0.001$ or ## $p < 0.01$ compared to the DM group.

GA exerts anti-inflammatory effects in a T2DM rat model

After 4 weeks of treatment with GA in T2DM rats, changes were observed in biochemical analysis (Figure 2). The BDNF level in serum was significantly reduced in the DM group (Figure 2A). Moreover, the IL-6 level was significantly increased in the DM group compared to the control (Figure 2B). After four weeks of GA treatment (3 mg/kg BW), the BDNF levels were significantly increased in the GA group compared to the DM group ($p < 0.01$) (Figure 2A). In contrast, GA exhibited potent anti-inflammatory effects, as evidenced by significantly reduced serum IL-6 levels ($p < 0.001$) (Figure 2B). These results indicated that GA exerts potent anti-inflammatory effects in the T2DM rat model.

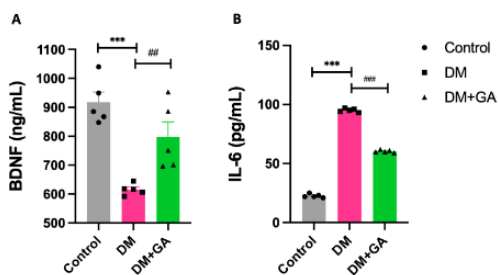


Fig. 2. The effect of Gambogic acid (GA) on biochemical markers in T2DM rats. Graphs representing serum levels of (A) Brain-derived neurotrophic factor (BDNF) and (B) Interleukin-6 (IL-6) in control, diabetic (DM), and DM+GA (3 mg/kg BW) treated groups. Data are presented as mean $\pm$ SEM. Statistical significance is indicated by symbols where *** $p < 0.001$ compared to control, and ### $p < 0.001$ or ## $p < 0.01$ compared to the DM group.

GA demonstrates a restorative effect on the histological pathology of the duodenum in the T2DM rat model

Histopathological changes were observed in the duodenum of the diabetic rat model after 4 weeks of treatment with GA (Figure 3A). Compared with control, DM rats exhibited significant structural damage, characterized by a marked reduction in villus height and number of goblet cells, an increased crypt depth, and a significantly lower villus-to-crypt (VC) ratio ($p < 0.001$) (Figure 3B–F). Markedly, 4 weeks of treatment with GA (3 mg/kg BW) significantly improved the histological architecture of the duodenum compared to the DM group, as evidenced by increased villus height, restored

goblet cell counts, normalized crypt depth, and a significantly improved VC ratio ($p < 0.001$) (Figure 3B–F). These results demonstrate that GA exerts a potent restorative effect on duodenal mucosal integrity in this T2DM rat model.

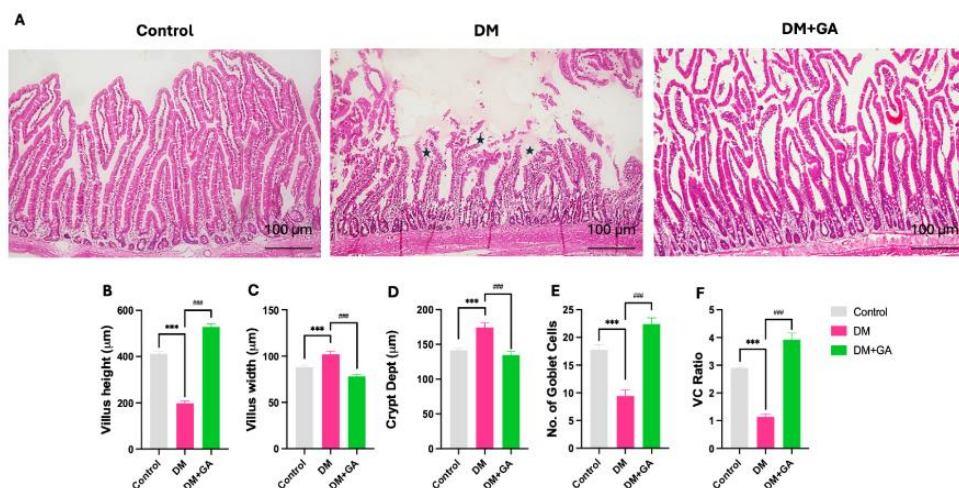


Fig. 3. The effect of Gambogic acid (GA) on duodenum histological pathology in T2DM rat model. (A) Representative of hematoxylin and eosin (H&E) staining. ★ represents destruction of villi (B-F) Graphs showing quantitative data of villus height, villus width, crypt depth, goblet cells, and VC ratio in normal rats (control), diabetic rats (DM), and DM with GA treated at a dose of 3 mg/kg BW (DM+GA). Data are presented as mean $\pm$ SEM. Statistical significance is indicated by symbols where *** $p < 0.001$ compared to control, and ### $p < 0.001$

GA positively influences the level of tight junction proteins in the duodenal tissue of in T2DM rat model.

The level of tight junction proteins in the duodenal tissue was evaluated to assess intestinal barrier integrity (Figure 4). Compared with controls, the DM group showed a marked downregulation in the level of ZO-1 and Occludin proteins (Figure 4A–C), which indicates the loss of gut integrity. Interestingly, 4 weeks of treatment with GA at a dose of 3 mg/kg BW significantly upregulated the level of these tight junction proteins compared to the DM group, with a statistically significant increase observed for ZO-1 ($p < 0.05$) (Figure 4B–C). These results indicate that GA treatment effectively enhances the level of key tight junction proteins, suggesting a protective role in maintaining intestinal barrier function in this T2DM rat model.

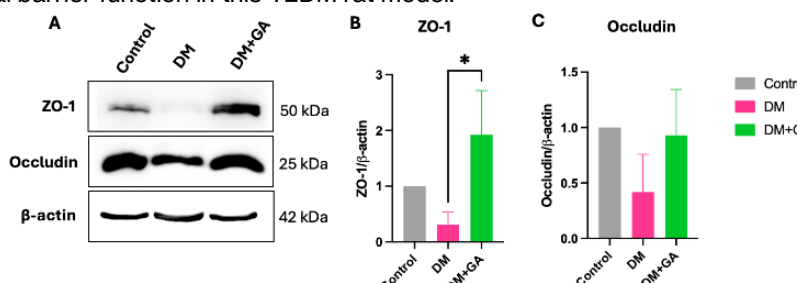


Fig. 4. The effect of Gambogic acid (GA) on tight junction protein levels in the duodenum of T2DM rat model. (A) Representative Western blot images showing the protein levels of ZO-1 and Occludin, with β -actin used as a loading control. (B–C) Quantitative densitometric analysis of (B) ZO-1 and (C) Occludin protein levels normalized to β -actin in control, diabetic (DM), and DM+GA (3 mg/kg BW) treated groups. Data are presented as mean $\pm$ SEM; * $p < 0.05$ indicates significant difference.

Discussion and Conclusion

Previous studies demonstrated that GA treatment improved hyperglycemia and glucose homeostasis while attenuating diabetes-associated oxidative stress and inflammation (Cui et al., 2018; Thongrungs et al., 2025). These findings support the glucose-lowering and protective effects of GA in T2DM-related complications. This study demonstrates the potential of GA to ameliorate the gut barrier integrity and brain function of STZ-induced T2DM rat model. The results exhibited improvements in neurobehavioral performance (i.e., anxiety-like behavior and locomotor activity), systemic inflammation, histological structure, and intestinal barrier integrity in the STZ-induced rat model. Diabetic rats exhibited severe duodenal pathology, which was characterized by structural destruction and a compromised intestinal barrier. The study showed that there is an increase in villus height in the DM rat model (Lerkdumnerkit et al., 2022). This differs from our histological analysis and may be due to the different doses of STZ used in our experiment. However, the other histological analysis including villus width, crypt depth, number of goblet cells and VC ratio, are similar to previous studies (Lerkdumnerkit et al., 2022). Our findings reveal that GA treatment facilitates the recovery of intestinal architecture, specifically by increasing villus height and width, decreasing crypt depth, and restoring goblet cell populations. ZO-1 is a peripheral transmembrane protein that plays a crucial role in the formation of tight junctions and gut permeability, to support gut health (Runkle & Mu, 2013).

When inflammation occurs, it directly downregulates ZO-1 and claudin-1 (Yu et al., 2023). In gut, occludin acts as an integral membrane that facilitates gut permeability and barrier (Dubreuil, 2017). Therefore, the restoration of these proteins is critical, as tight junctions maintain epithelial barrier function, preventing the leaky gut phenotype often associated with systemic inflammation in metabolic diseases. The effects of GA on gut structural recovery are further supported by the molecular evidence of increased levels of key tight junction proteins, especially ZO-1.

GA demonstrated significant neuroprotective and anti-inflammatory potential. Diabetic rats showed impaired behavioral performance, including reduced exploration in the open field test, which correlated with lower levels of BDNF and elevated serum IL-6. A previous study has demonstrated that BDNF alleviates depressive-like behaviors and is associated with reduced IL-6 expression (Li et al., 2022). BDNF is a neurotrophic protein that contributes to neuronal maintenance, maturation, and synaptic plasticity. Through its regulatory effects on neural development and synaptic function, BDNF supports cognitive processes, including learning and memory, while facilitating neuronal adaptation and recovery following injury (Azoulay & Horowitz, 2022). Beyond the central nervous system, emerging evidence suggests that BDNF also participates in peripheral metabolic regulation by modulating pancreatic β -cell function, insulin secretion, and glucose homeostasis, thereby linking neurotrophic signaling with metabolic health (Fulgenzi et al., 2020). GA significantly increased serum BDNF levels, suggesting enhanced neuroplasticity and neuronal support, which may contribute to the observed improvements in locomotor and exploratory behaviors in the open-field test. Interleukin-6 (IL-6) is a pleiotropic cytokine that plays a central role in regulating immune and

inflammatory processes and is commonly elevated in chronic metabolic diseases. The study suggested that selective inhibition of IL-6 might be beneficial for inflammatory diseases and metabolic conditions of bone (Fonseca et al., 2009). The observed reduction in serum IL-6 levels indicates that GA effectively attenuates the systemic inflammatory state characteristic of T2DM. A limitation of the present study is that direct neuronal analyses, including neuronal survival, morphology, and synaptic connectivity, were not comprehensively evaluated. Although GA improved neurobehavioral outcomes and reduced neuroinflammatory responses, additional investigations using neuronal-specific markers and molecular analyses are required to confirm its direct neuroprotective mechanisms.

The data collectively suggest that GA exerts a multi-faceted therapeutic effect. By strengthening the intestinal barrier, GA may limit the translocation of pro-inflammatory bacterial products into systemic circulation, thereby lowering systemic IL-6. These findings highlight GA as a promising therapeutic candidate for addressing the gut-brain axis disruptions prevalent in Type 2 Diabetes Mellitus.

Acknowledgements

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P030

THE REGULATION OF TRYPTOPHAN METABOLISM: A POTENTIAL TARGET TO PREVENT NEUROINFLAMMATION-RELATED NEURODEGENERATION IN METHAMPHETAMINE- INDUCED NEUROTOXICITY

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Abstract:

Background: Methamphetamine (METH) is a psychostimulant drug whose effects on the toxicity of mammalian brains induce neurodegeneration. Prolonged use of methamphetamine causes dopamine accumulation in the brain, leading to dopamine auto-oxidation, which generates reactive oxygen species (ROS), resulting in mitochondrial damage and oxidative stress. Several studies substantially demonstrate that METH contributes to brain inflammation by persistently activating microglia, leading to neuroinflammation and increased production of pro-inflammatory cytokines. Moreover, several lines of evidence suggest a role for tryptophan metabolism in neuroinflammation-induced neuronal degeneration.

Aim(s): This study aimed to elucidate the inhibitory effect of a new candidate compound for neuroprotection, Rupatadine, on tryptophan metabolism enzyme-IDO-1 (Indoleamine 2,3-dioxygenase 1) activation and neuroinflammation in METH-induced degeneration in neuroblastoma SH-SY5Y cell cultures.

Methods: The SH-SY5Y cells were treated with 1.0 mM METH for 24 hours. Some cells were treated with 1 μ M PF06840003 (a highly selective, brain-penetrant IDO-1 inhibitor) or Rupatadine for 6 hours following METH exposure. Cell viability, IDO-1 expression levels, IDO-1 activity, intracellular ROS levels, inflammatory cytokine levels, and cell degenerative markers were determined in SH-SY5Y cells.

Results: Exposure to METH can reduce cell viability but increase IDO-1 expression levels, IDO-1 activity, intracellular ROS levels, inflammatory cytokine (IL-6) levels, and cell degenerative markers. Post-treatment with PF06840003 or Rupatadine can diminish METH-induced IDO-1 activation and the induction of ROS and IL-6 levels, as well as cell degenerative markers.

Conclusion: These findings suggest a potential role of Rupatadine in attenuating neuroinflammation-related neurodegenerative processes and the involvement of tryptophan metabolism in METH-induced neurotoxicity.

Keywords: neurodegeneration, neuroinflammation, tryptophan metabolism, IDO-1, methamphetamine

P031

STABLE OVEREXPRESSION OF AMYLOID PRECURSOR PROTEIN (APP) IN SH-SY5Y CELLS AS AN *IN VITRO* PLATFORM FOR INVESTIGATING APP PROCESSING MECHANISMS

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Abstract:

Background: Alzheimer's disease (AD) is pathologically characterized by the accumulation of amyloid-beta (A β) plaques, which are derived from the sequential proteolytic cleavage of the amyloid precursor protein (APP). Investigating the precise molecular dynamics of amyloidogenic and non-amyloidogenic APP processing requires a robust, reproducible human *in vitro* neuronal model. While human neuroblastoma SH-SY5Y cells are widely used in neurobiology, their endogenous APP expression levels are often insufficient for robust mechanistic and kinetic studies of downstream processing fragments.

Aim(s): The objective of this study was to establish and validate a stable APP-overexpressing SH-SY5Y cell line to serve as an optimized *in vitro* platform for investigating APP processing pathways and secretase activities.

Methods: A recombinant lentiviral vector harboring the human APP cDNA sequence was constructed. Lentiviral particles were generated in HEK293T packaging cells and subsequently used to transduce target human SH-SY5Y cells. Successfully transduced cells were selected using antibiotic resistance markers to establish a stable, homogenous cell pool overexpressing the APP gene.

Results: To confirm the efficiency of the genetic modification, total protein was harvested from the stable cell lines and analyzed. Western blot analysis using an APP-specific monoclonal antibody demonstrated a robust and highly significant increase in APP protein expression in the lentivirus-transduced SH-SY5Y cells compared to the non-transduced parental control cells.

Conclusion: We have successfully established a stable, high-expression APP SH-SY5Y cellular model using a lentiviral delivery system. This validated system provides a reliable, scalable *in vitro* tool to dissect the upstream regulatory mechanisms of APP cleavage and to screen potential therapeutic agents targeting amyloid pathology in Alzheimer's disease.

Keywords: Alzheimer's disease, Amyloid precursor protein (APP), SH-SY5Y neuroblastoma cells, Lentiviral transduction, *In vitro* disease modeling

P032

THE ROLE OF HYPER-FOCUS CHARACTERISTICS AS A TRANSDIAGNOSTIC ATTENTIONAL TRAITS ON COGNITIVE FLEXIBILITY IN AN ADULT POPULATION

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Abstract:

Background and Aim(s): This study aimed to examine the transdiagnostic role of hyper-focus experiences on cognitive flexibility patterns according to transdiagnostic traits in Autism Spectrum Disorder (ASD trait) and Attention-Deficit/Hyperactivity Disorder (ADHD trait) under both non-emotional (Cold EF) and emotional (Hot EF) conditions.

Methods: The methodology employed a quantitative research design with a sample of 61 Thai adults aged 18–60 years who completed online data collection tasks. The research instruments consisted of self-report scales, including the RAADS-14, ASRS v1.1, and AHQ-D, alongside a behavioral task using the 10-stage Intra-Dimensional/Extra-Dimensional (IDED) set-shifting paradigms. Notably, the researcher incorporated a 10th stage providing false negative feedback to assess the affective stress cost. Data were analyzed using multiple regression, simple mediation analysis, and simple moderation analysis via Jamovi and PROCESS.

Results: The results revealed that ADHD traits and ASD traits significantly and positively co-predicted the level of hyper-focus experience, confirming its nature as a transdiagnostic trait. However, behavioral testing provided no statistically significant evidence that hyper-focus moderated the relationship between neurodevelopmental traits and set-shifting errors under the Cold EF condition, or the ability to manage behavior when facing situations that conflict with expectations under the Hot EF condition. Additionally, no statistically significant mediating effect on cognitive effort efficiency was observed. These non-significant behavioral findings may be explained by the Cognitive Flexibility Paradox, wherein impairments might be masked by compensatory mechanisms in highly educated individuals within structured environments, alongside limitations regarding statistical power and the online research format involving uncontrollable device latency and external distractions.

Conclusion: This study suggests utilizing laboratory-based behavioral tests combined with physiological measures and recommends developing clinical interventions that integrate emotion regulation skills when encountering negative outcomes.

Keywords: Hyper-Focus, Transdiagnostic Framework, Cognitive Flexibility, ASD Trait, ADHD Trait

P033

FENDOSAL MODULATES RHEB/MTOR SIGNALING AND INFLAMMATORY RESPONSES UNDER ISCHEMIA-REPERFUSION-LIKE CONDITIONS

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Abstract:

Background: Ischemic stroke induces astrocyte activation, inflammatory dysregulation, and cell death-associated signaling, contributing to secondary brain injury. The Ras homolog enriched in brain/mechanistic target of rapamycin complex 1 (Rheb/mTORC1) pathway is a key regulator of cellular metabolism, autophagy, and stress responses, but its role in astrocytes under ischemia-reperfusion-like conditions remains unclear. Fendosal, a non-steroidal anti-inflammatory compound, may provide cytoprotective effects through modulation of inflammatory and survival pathways.

Aim(s): This study aimed to investigate whether fendosal modulates Rheb/mTOR-associated signaling, inflammatory cytokine secretion, and apoptosis-associated protein expression in C8-D1A astrocytes subjected to oxygen-glucose deprivation/reperfusion (OGD/R).

Methods: C8-D1A astrocytes were exposed to 3 hours of oxygen-glucose deprivation (OGD) followed by 24 hours of reperfusion. Cell viability was assessed after treatment with fendosal or the Rheb inhibitor NR1. Rheb, phospho-mTOR, Bax, and Bcl-2 protein expression were evaluated by Western blotting. Cytokine secretion in conditioned media was measured by ELISA. NR1 was used as a pharmacological comparator to investigate the involvement of the Rheb pathway. All experiments were performed at least three independent biological replicates. Data were analyzed using one-way ANOVA followed by Tukey's *post hoc* test.

Results: Fendosal and NR1 did not reduce astrocyte viability across the tested concentrations. OGD/R induced changes in Rheb/mTOR signaling, including Rheb expression during OGD and altered phospho-mTOR levels during reperfusion. OGD increased IL-1 β , TNF- α , IL-10, and IL-18 secretion, whereas reperfusion partially normalized several cytokine responses. Fendosal and NR1 modulated the OGD/R-induced cytokine profile, including suppression of IL-10 secretion during reperfusion, suggesting context-dependent regulation of inflammatory balance. OGD/R also increased the Bax/Bcl-2 ratio following ischemic stress, which was attenuated by fendosal and NR1 treatment.

Conclusion: Fendosal may protect astrocyte under ischemia-reperfusion-like condition by modulating the Rheb/mTOR signaling pathway, inflammatory cytokine release, and apoptosis-associated signaling. These findings support further investigation of fendosal as a potential astrocyte-targeted modulator of post-ischemic neuroinflammation.

Keywords: Fendosal, Rheb, mTOR, Astrocytes, Ischemic stroke

P034

INTRAMUSCULAR AUTOLOGOUS MESENCHYMAL STEM CELL THERAPY FOR CHRONIC PROGRESSIVE MYELOPATHY IN A DOG: A CASE REPORT

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Abstract:

Background: Chronic progressive myelopathy often causes severe neurological deficits with limited treatment options. Mesenchymal stem cells (MSCs) are an emerging regenerative option for spinal cord injury. Following allogeneic MSCs transplantation, a 5-year-old male French Bulldog with grade 5/5 posterior paresis improved to grade 3/5 and regained normal spinal reflexes. However, localized thigh muscle weakness persisted.

Aim(s): This study aims to evaluate how the localized effects of autologous MSCs can improve neuromuscular recovery and restore motor function.

Methods: The dog received a total autologous MSCs dose of 3×10^7 cells allocated as 1×10^7 cell intravenously, 1×10^7 cells injected into the epaxial muscle (5×10^6 per side), and 1×10^7 cells injected into the thigh muscles (5×10^6 per side). Outcome assessments included quadriceps femoris muscle thickness measurements, shear wave elastography (SWE), and paresis grading.

Results: Three weeks post-injection, a decrease in quadriceps femoris muscle size was observed, with the left side decreasing from 1.55 cm to 1.43 cm and the right side decreasing from 1.35 cm to 1.31 cm. Conversely, SWE of the vastus lateralis muscle was demonstrated an increase from 38.22 kPa to 90.46 kPa on the left side and from 11.63 kPa to 47.71 kPa on the right side. Although the paresis grade remained at grade 3, the dog demonstrated an improved ability to stand independently, reflecting enhanced motor function.

Conclusion: These findings suggest that MSC therapy can promote neuromuscular adaptation and functional motor recovery in dog with chronic progressive myelopathy.

Keywords: autologous MSCs, Shear wave elastography, chronic progressive myelopathy

P035

FENDOSAL ATTENUATES INFLAMMATORY RESPONSES AND APOPTOSIS IN NEURONAL CELLS UNDER ISCHEMIA-REPERFUSION CONDITIONS

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Abstract:

Background: Ischemia-reperfusion injury significantly contributes to neuronal loss following ischemic stroke, primarily due to excessive inflammatory responses and the activation of apoptotic pathways. Fendosal, a derivative of a nonsteroidal anti-inflammatory drug (NSAID), has demonstrated anti-inflammatory activity in chronic inflammation models; however, its potential neuroprotective effects under ischemia-reperfusion conditions remain uncertain.

Aim(s): This study aimed to investigate whether Fendosal modulates inflammatory cytokine production and apoptosis-related signaling in HT22 neuronal cells subjected to oxygen-glucose deprivation (OGD) and subsequent re-oxygenation (OGD/R).

Methods: HT22 hippocampal neuronal cells were exposed to 6 hours of oxygen-glucose deprivation followed by 18 hours reoxygenation. During the reoxygenation phase, cells were treated with or without Fendosal at 1 nM or 5 nM. Levels of interleukin-1 β (IL-1 β), interleukin-6 (IL-6), interleukin-10 (IL-10), and tumor necrosis factor- α (TNF- α) in the conditioned media were measured using ELISA. Apoptosis-related signaling was assessed by Western blot analysis of Bax and Bcl-2 protein expression.

Results: OGD/R induced a significant inflammatory and pro-apoptotic response in the HT22 cells, as evidenced by increased secretion of IL-1 β and TNF- α , along with enhanced expression of Bax. Treatment with Fendosal at 5 nM significantly reduced the OGD/R-induced IL-1 β and TNF- α levels, indicating attenuation of pro-inflammatory cytokine production. Additionally, 5 nM Fendosal also significantly increased IL-6 secretion while decreasing IL-10 levels, suggesting a selective regulatory effect on the inflammatory response during reoxygenation. Concurrently, Fendosal significantly decreased the levels of the pro-apoptotic protein Bax and increased the levels of the anti-apoptotic protein Bcl-2, thereby reducing the Bax/Bcl-2 ratio and shifting apoptosis-related signaling toward a cell survival-associated profile.

Conclusion: These findings demonstrate that Fendosal attenuates the inflammatory responses and apoptotic signaling following OGD/R in HT22 neuronal cells, suggesting Fendosal may have therapeutic potential for mitigating neuronal injury linked to ischemia-reperfusion conditions.



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Keywords: Oxygen-glucose deprivation, Neuroinflammation, Apoptosis, Fendosal, Neuron

P036

MULTIMODAL ASSESSMENT OF SLEEP QUALITY, COGNITIVE FUNCTION, AND AUTONOMIC REGULATION IN PRIMARY SCHOOL CHILDREN WITH AND WITHOUT ADHD: A PILOT STUDY

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Abstract:

Background: Sleep disturbances are common in children with attention-deficit/hyperactivity disorder (ADHD) and may adversely affect cognitive function and behavioral regulation. The autonomic nervous system (ANS) is believed to mediate the relationship between sleep quality and cognitive performance; however, evidence integrating these domains in primary school children remains limited.

Aim(s): To investigate the relationships among sleep quality, cognitive function, and ANS activity in primary school children with and without ADHD.

Methods: Four children aged 7–9 years (ADHD, $n = 2$; controls, $n = 2$) participated in this pilot study at the Institute of Molecular Biosciences, Mahidol University. Parents completed the Children's Sleep Habits Questionnaire (CSHQ), OSA-18, and SNAP-IV. Cognitive function was assessed using the Wisconsin Card Sorting Test (WCST) and Attention Network Test (ANT). During cognitive testing, ANS responses were continuously recorded during a 5-min baseline, cognitive tasks (15–20 min), and a 5-min recovery period using biopotential signals, including electrodermal activity (EDA) and electrocardiography. Heart rate (HR), R-R interval (RRI), and sympathetic-vagal indices were subsequently derived.

Results: In this pilot sample, ADHD tended to exhibit poorer sleep quality (CSHQ: 50.0 vs. 45.0), higher SNAP-IV scores (43.5 vs. 8.5), increased parasomnia, and greater sleep duration difficulties. Descriptive observations from the WCST indicated fewer perseverative errors and greater cognitive flexibility, while ANT showed faster processing speed and higher accuracy but reduced orienting efficiency. Simultaneous ANS recordings during both cognitive tasks demonstrated higher EDA, HR, sympathetic activity, and sympathetic-vagal ratio, accompanied by lower vagal activity in ADHD, indicating a trend toward sympathetic predominance during cognitive performance.

Conclusion: Preliminary observations suggest potential relationships among sleep quality, cognitive performance, and ANS activity, with a trend toward sympathetic predominance during cognitive tasks in ADHD. Larger controlled studies are needed to validate these multimodal measures and further clarify the interactions among sleep, cognition, and ANS regulation.



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Keywords: Attention-Deficit/Hyperactivity Disorder (ADHD), Sleep Quality, Cognitive Function, Autonomic Nervous System, Heart Rate Variability

P037

INDIRUBIN AS A POTENTIAL MODULATOR OF KAINIC ACID-INDUCED ASTROCYTE REACTIVITY AND INFLAMMATORY RESPONSES

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Abstract:

Background: Kainic acid is widely used as an excitotoxic stimulus in experimental models of neurological disorders, particularly epilepsy and excitotoxic brain injury. Although excitotoxicity is commonly associated with neuronal damage, astrocytes are also critically involved in this process through the regulation of glutamate homeostasis, inflammatory responses, and redox balance. Under excitotoxic stress-related conditions, astrocytes may become reactive and release pro-inflammatory cytokines, including interleukin-1 β (IL-1 β), tumor necrosis factor- α (TNF- α), and interleukin-6 (IL-6), which can further amplify neuroinflammation and neuronal vulnerability. Indirubin, a bioactive compound derived from *Indigo naturalis*, has been reported to exert anti-inflammatory, antioxidant, and neuroprotective properties. However, its potential role in modulating kainic acid-induced astrocyte reactivity remains unclear.

Aim(s): This study aimed to investigate the therapeutic potential of indirubin in attenuating kainic acid-induced reactive astrocyte model associated with excitotoxic stress, with emphasis on its ability to modulate astrocyte reactivity and inflammatory cytokine production.

Methods: Mouse astrocyte C8-D1A cells were cultured and exposed to kainic acid at concentration ranging from 100 nM to 500 μ M to determine suitable conditions for inducing astrocytes responses. Non-toxic concentration of indirubin, ranging from 500 nM to 100 μ M, were determined using the MTT assay. Cells were then pretreated with selected concentrations of indirubin before exposure to kainic acid. The levels of inflammatory cytokines, including IL-1 β , TNF- α , and IL-6, were evaluated using enzyme-linked immunosorbent assay.

Results: MTT findings identified concentration ranges of kainic acid and indirubin suitable for subsequent experiments. These results provide the basis for further investigation of whether indirubin can attenuate kainic acid-induced astrocyte reactivity and inflammatory cytokine production.

Conclusion: This study provides an experimental condition for investigating indirubin as a potential modulator of kainic acid-induced astrocyte reactivity. The findings may contribute to a better understanding of indirubin-mediated neuroprotection in excitotoxicity-related neurological disorders.



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Keywords: Reactive Astrocyte, Excitotoxicity, Indirubin, Kainic acid, Inflammatory cytokine

P038

AHR INHIBITION PROTECTS BV2 MICROGLIA AGAINST PM2.5-LIKE PARTICULATE MATTER-INDUCED CYTOTOXICITY UNDER ISCHEMIA-REPERFUSION-LIKE CONDITIONS

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Abstract:

Background: Ischemic stroke is a leading cause of mortality and long-term disability worldwide. Following ischemic injury, microglia are rapidly activated and contribute to secondary brain injury by releasing pro-inflammatory mediators. The aryl hydrocarbon receptor (AhR), a ligand-activated transcription factor responsive to environmental pollutants, has emerged as an important regulator of inflammatory responses in the central nervous system (CNS). However, its role in microglial responses to ischemia-reperfusion-like conditions, particularly under fine particulate matter exposure, remains unclear.

Aim(s): This study aimed to investigate the involvement of AhR signaling in BV2 microglial responses under oxygen-glucose deprivation/reperfusion (OGD/R) conditions and to evaluate the effects of PM2.5-like particulate matter on microglial viability.

Methods: BV2 microglial cells were subjected to oxygen-glucose deprivation (OGD) followed by reperfusion. To identify an appropriate reperfusion period for inflammatory activation, IL-1 β release in conditioned media was measured after 3, 18, and 24 hours of reperfusion using ELISA. The cytotoxic effects of the AhR antagonist CH-223191 and PM2.5-like particulate matter were evaluated using the MTT assay. A non-cytotoxic concentration of CH-223191 was selected for co-treatment experiments.

Results: IL-1 β release was significantly increased after 24 hours of reperfusion, indicating enhanced inflammatory activation of BV2 microglia. Based on the MTT assay, 0.05 μ M CH-223191 was selected for subsequent experiments. PM2.5-like particulate matter significantly reduced microglial viability at 50 μ g/mL. Co-treatment with CH-223191 significantly attenuated the reduction in cell viability induced by PM2.5-like particulate matter.

Conclusion: These findings suggest that PM2.5-like particulate matter induces cytotoxicity in BV2 microglial, while AhR inhibition may partially protect against PM2.5-associated cell death. OGD followed by 24 hours of reperfusion appears suitable for studying inflammatory activation in this microglial model. This study provides preliminary evidence linking AhR signaling, particulate matter exposure, and microglial responses relevant to post-stroke neuroinflammation.

Keywords: Ischemic stroke, microglia, aryl hydrocarbon receptor, PM2.5, oxygen-glucose deprivation

P040

THE ANTAGONISTIC ROLE OF ASARININ AGAINST OREXIN RECEPTOR ACTIVATION IN ORX2R-OVEREXPRESSING CELLS: AS A POTENTIAL DUAL OREXIN RECEPTOR ANTAGONIST FOR THE TREATMENT OF INSOMNIA

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Abstract:

Background: Insomnia is a prevalent sleep disorder affecting 10%-40% of the adult population, with an increasing trend. Chronic insomnia is characterized by difficulties in initiating or maintaining sleep or waking up too early, occurring at least three times per week and persisting for at least three months, with significant impairment in daily functioning. Current pharmacotherapies include dual orexin receptor (ORXR) antagonists (DORAs), which block both ORX1R and ORX2R, thereby increasing total sleep time primarily by promoting REM sleep. Our research team, through virtual drug screening, identified asarinin, a lignan extracted from *Asarum sieboldii* Miq., as a promising candidate DORA that inhibits orexin receptor activation.

Aim(s): To investigate the antagonistic role of asarinin as a DORA on orexin-2 receptor (ORX2R) downstream signaling cascades in ORX2R-overexpressing neuroblastoma cells, suggesting its potential therapeutic application for insomnia.

Methods: ORX2R-overexpressing neuroblastoma SH-SY5Y cells were established as an *in vitro* model to examine the potential mechanism of action of asarinin inhibiting ORX2R-mediated cell signaling and a PKC-mediated calcium influx. Phosphorylation of ERK1/2 was evaluated by Western blot analysis, and the cytosolic calcium levels were determined using the green fluorescent calcium indicator Fluo-4, following receptor activation with the selective ORXR agonist HY-19320 and treatment with either asarinin or the clinically used DORA, lemborexant.

Results: Asarinin at 50 μ M partially reduces HY-19320-induced ERK1/2 phosphorylation. However, lemborexant at 20 nM and 40 nM significantly decreased HY-19320-induced increase in ERK1/2 phosphorylation. Additionally, asarinin at 50 μ M and lemborexant at 40 nM significantly decreased HY-19320-induced increase in cytosolic Ca^{2+} levels.

Conclusion: The findings suggest that asarinin acts as a potential ORXR antagonist. Our results demonstrated that asarinin effectively suppressed both HY-19320-induced increases in intracellular Ca^{2+} levels and ERK1/2 phosphorylation. This dual inhibition of key Gq downstream effectors provides functional evidence that asarinin acts as an ORX2R antagonist. Moreover, since ORX2R activation involves multiple GPCR pathways sustaining neuronal excitation, further studies should explore additional downstream pathways to evaluate the potential of asarinin as a novel insomnia therapeutic.

Keywords: asarinin, dual orexin receptor antagonist, insomnia, orexin, orexin-2 receptor

P042

OXIDATIVE STRESS-INDUCED ASTROCYTE- CONDITIONED MEDIUM IMPAIRS NEURONAL VIABILITY AND NEURITE INTEGRITY IN DIFFERENTIATED SH-SY5Y CELLS

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Abstract:

Background: Neuroinflammation mediated by dysfunctional astrocytes contributes to neuronal injury during neurodegenerative processes. However, the direct effects of conditioned medium from oxidatively stressed astrocytes on neuronal viability and neurite integrity remain unclear.

Objective: To investigate the effects of astrocyte-conditioned medium derived from H₂O₂-treated CTX-TNA2 cells (ACM-H) on neuronal viability and neurite integrity in SH-SY5Y cells.

Methods: CTX-TNA2 astrocytes were exposed to 200 μ M H₂O₂ for 1 h, followed by 3 days of recovery before collecting astrocyte-conditioned medium (ACM-H). SH-SY5Y cells were treated with 25%, 50%, 75%, or 100% ACM-H for 24 h. Cell viability was determined using the MTT assay. Cellular morphology was examined by bright-field microscopy, and neurite integrity was evaluated by β III-tubulin immunofluorescence staining.

Results: ACM-H significantly reduced SH-SY5Y cell viability in a concentration-dependent manner ($P < 0.05$). Bright-field microscopy revealed progressive neurite shortening and disruption of neuronal networks with increasing ACM-H concentrations. β III-tubulin immunofluorescence further demonstrated impaired neurite integrity and loss of neuronal processes in ACM-H-treated cells, suggesting that soluble neurotoxic factors released from oxidatively stressed astrocytes contribute to neuronal injury.

Conclusion: Conditioned medium from oxidatively stressed astrocytes impaired neuronal viability and neurite integrity in SH-SY5Y cells. This in vitro model may provide a useful platform for investigating astrocyte–neuron interactions and identifying therapeutic strategies targeting astrocyte-mediated neuronal injury.

Keywords: Astrocyte, SH-SY5Y, ACM-H, Neurite morphology

P043

FUNCTIONAL AND EFFECTIVE NEURONAL CONNECTIVITY ALTERATION AMONG THAI ADULT METHAMPHETAMINE USERS

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Abstract

Background: Methamphetamine (MA) is a prevalent drug of abuse in Thailand. Chronic use alters the excitability of Medium Spiny neurons, amplifying the Direct Pathway of the Basal Ganglia circuit and leading to their excitotoxicity. Cognitive circuits could receive the consequences. In this study, long-range connectivity was hypothesized to change due to the drug in overall coherence and Granger causality, with the sub-connection involving the Superior Longitudinal Fasciculus II (SLFII).

Aim(s): To establish patterns of altered functional connectivity among MA users that could be used as a biomarker for guiding treatment and rehabilitation.

Methods: Spectral EEG from 10 controls, 8 MA users without psychosis and 17 MA users with psychosis were taken. Conditions included Eye closed, Eye open, Go-No go task and One-Back task. The MNE and MNE-connectivity libraries in Python 3.13 were used to extract coherence and Granger causality in both the alpha and beta bands. The output was entered into Excel, sorted by conditions and participants' IDs, and analyzed using SPSS (30th edition). One-way ANOVA and Tukey analysis were performed.

Results: An overall reduction in alpha and beta coherence was observed among MA abusers during cognitive tasks. The sub-connection, depending on SLFII, exhibited lower beta coherence and Granger causality, in contrast to the overall enhanced beta Granger causality in the MA user groups' Frontoparietal circuit.

Conclusion: Long-range cognitive circuits, visualized via functional and connectivity analyses, are altered by MA use, with enhanced beta-oscillation effective connectivity and weakened sub-component linkage. The beta band profile observed here could align with the 'abnormal beta oscillation' reported in recent literature, which occurs after drug use and contributes to altered neuronal connectivity.

Keywords: Methamphetamine abuser, Coherence, Granger causality, Abnormal beta oscillation, neuronal connectivity, Superior Longitudinal Fasciculus II



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TNS29 AWARDS

TNS29 Awards Announcement

The TNS29 Awards recognize outstanding research contributions presented at the conference. This year, a total of 14 awards were presented to recognize excellence in research, creativity, and scientific communication.

1. **Selected Abstract Award as Oral presentation** (2 Awards) (Professor Ingrid Y Liu award 2,000 THB)
2. **Best Poster Presentation Awards**
 - 2.1 **Prof. Dr. Prasop Ratanakorn Foundation Award for Outstanding Research Achievement** (Receives a Certificate and a cash prize of 4,000 THB)
 - 2.2 **Professor Roongtarn Ladpli Award** (Receives a Certificate and a cash prize of 2,000 THB)
 - 2.3 **Associate Professor Naiphinich Kotchabhakdi Award** (Receives a Certificate and a cash prize of 2,000 THB)
 - 2.4 **Professor Ingrid Y Liu Award** (2 Awards) (Receives a Certificate and a cash prize of 2,000 THB)
 - 2.5 **Professor Pavich Tongroach Award** (2 Awards) (Receives a Certificate and a cash prize of 2,000 THB)
 - 2.6 **Mr. Tay Khoon Yang Award** (Hi-Tech Imaging Co. Ltd.) (Receives a Certificate and a cash prize of 2,000 THB)



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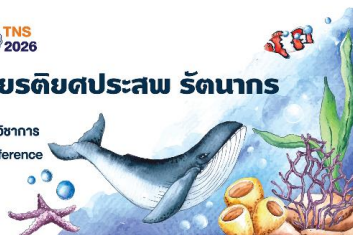


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- ชุดน้ำยาวิเคราะห์และตรวจวัดโปรตีน



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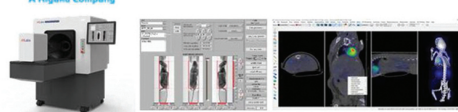


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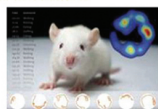


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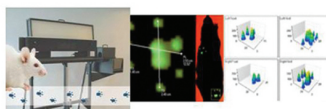
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ทะเบียนเลขที่ จ.๓๖๕๔/๒๕๖๘

ส.ศ.๕

ใบสำคัญแสดงการจดทะเบียนการแก้ไขเพิ่มเติมข้อบังคับ

ใบสำคัญฉบับนี้ออกให้เพื่อแสดงว่า สมาคมประสาทวิทยาศาสตร์ไทย สำนักงานใหญ่ตั้งอยู่ ณ ภาควิชากายวิภาคศาสตร์ คณะวิทยาศาสตร์ มหาวิทยาลัยมหิดล เลขที่ ๒๗๒ ถนนพระรามที่ ๖ เขตราชเทวี กรุงเทพมหานคร มีการจดทะเบียนการแก้ไขเพิ่มเติมข้อบังคับของสมาคม และนายทะเบียนสมาคม กรุงเทพมหานครได้รับจดทะเบียนการแก้ไขเพิ่มเติมข้อบังคับของสมาคม ตามมาตรา ๘๔ แห่งประมวลกฎหมายแพ่งและพาณิชย์แล้ว ดังต่อไปนี้

แก้ไขเพิ่มเติมข้อบังคับ ข้อ ๑๔ ความว่า

ข้อ ๑๔ การดำรงตำแหน่งของนายกสมาคมและกรรมการของสมาคม

๑๔.๑ นายกสมาคมสามารถอยู่ในตำแหน่ง ได้วาระละ ๒ ปี และไม่เกิน ๒ วาระติดต่อกัน

๑๔.๒ กรรมการของสมาคม จะหมดวาระตามนายกสมาคม

เมื่อคณะกรรมการอยู่ในตำแหน่งครบกำหนดตามวาระแล้ว แต่คณะกรรมการชุดใหม่ ยังไม่ได้รับอนุญาตให้จดทะเบียนจากทางราชการ ก็ให้คณะกรรมการที่ครบกำหนดตามวาระรักษาการไปพลางก่อน จนกว่าคณะกรรมการชุดใหม่จะได้รับอนุญาตให้จดทะเบียนจากทางราชการ และเมื่อคณะกรรมการชุดใหม่ได้รับอนุญาตให้จดทะเบียนจากทางราชการเป็นที่เรียบร้อยแล้ว ก็ให้ทำการส่งและรับมอบงานกันระหว่าง คณะกรรมการชุดเก่าและคณะกรรมการชุดใหม่ ให้เป็นที่เสร็จสิ้นภายใน ๓๐ วัน นับตั้งแต่วันที่คณะกรรมการชุดใหม่ได้รับอนุญาตให้จดทะเบียนจากทางราชการ

ให้ไว้ ณ วันที่ ๐๑ ตุลาคม พ.ศ. ๒๕๖๘



(นางสาวสิริณัฐ นุตระรังค์)

นายอำเภอเวียงเก่า รักษาการในตำแหน่ง

ผู้อำนวยการส่วนทะเบียนมูลนิธิ และสมาคม ปฏิบัติราชการแทน

อธิบดีกรมการปกครอง

นายทะเบียนสมาคมกรุงเทพมหานคร



ทะเบียนเลขที่ จ.๓๖๕๔/๒๕๖๘

ส.ค.๖

**ใบสำคัญแสดงการจดทะเบียนการแต่งตั้งกรรมการของสมาคมขึ้นใหม่ทั้งหมด
หรือการเปลี่ยนแปลงกรรมการของสมาคม**

ใบสำคัญฉบับนี้ออกให้เพื่อแสดงว่า สมาคมประสาทวิทยาศาสตร์ไทย สำนักงานใหญ่ตั้งอยู่ ณ ภาควิชากายวิภาคศาสตร์ คณะวิทยาศาสตร์ มหาวิทยาลัยมหิดล เลขที่ ๒๗๒ ถนนพระรามที่ ๖ เขตราชเทวี กรุงเทพมหานคร มีการแต่งตั้งกรรมการของสมาคมขึ้นใหม่ทั้งหมด และนายทะเบียนสมาคมกรุงเทพมหานคร ได้รับจดทะเบียนการแต่งตั้งกรรมการของสมาคมขึ้นใหม่ทั้งหมด ตามมาตรา ๘๕ แห่งประมวลกฎหมายแพ่งและพาณิชย์แล้ว ดังต่อไปนี้

๑. นางสาวพิน	ชมภูพงษ์	นายกสมาคม
๒. นายชัยเลิศ	พิชิตพรชัย	อุปนายก คนที่ ๑ และกรรมการกิจการพิเศษ
๓. นางสาวทิสา	ถ่าน้อย	อุปนายก คนที่ ๒
๔. นายบัณฑิต	เจตน์สว่าง	กรรมการ
๕. นางสาวพร กัณฑ์ขุฑ	เตรียมชัยศรี	กรรมการ
๖. นายอัศวเดช	ศิริพร	กรรมการและวิชาการ
๗. นายชัยรัตน์	เดิปปิบูลย์	กรรมการและผู้ช่วยวิชาการ
๘. นายรวาภูมิ	ภาคาพรต	กรรมการและปฎิคม
๙. นายอดิสร	รัตนโยธ	กรรมการและนายทะเบียน
๑๐. นางมนัสพร	พัสระ	กรรมการและประชาสัมพันธ์
๑๑. นายอมรพันธุ์	เสรีมาศพันธุ์	กรรมการและผู้ช่วยกิจการพิเศษ
๑๒. นายประทีป	อมรรัตน์พันธุ์	กรรมการและผู้ช่วยกิจการพิเศษ
๑๓. นางสาวกุลธิดา	ชัยธีระยานนท์	กรรมการและเหรัญญิก
๑๔. นางสาวสุจิรา	มุกดา	กรรมการและเลขธิการ
๑๕. นางสาวอัจฉราภรณ์	คำศรีใจ	กรรมการและผู้ช่วยเลขธิการ

ให้ไว้ ณ วันที่ ๐๑ ตุลาคม พ.ศ. ๒๕๖๘

(นางสาวสิริยุรค์ นุตตะรังค์)

นายอำเภอเวียงเก่า รักษาการในตำแหน่ง

ผู้อำนวยการส่วนทะเบียนมูลนิธิ และสมาคม ปฏิบัติราชการแทน

อธิบดีกรมการปกครอง

นายทะเบียนสมาคมกรุงเทพมหานคร

Announcement of the TNS28 Academic Achievement Awards

TNS are delighted to announce the winners of the **TNS28 Awards**. This year, a total of **15 awards** were presented to recognize excellence in research, creativity, and scientific communication.

The awardees are as follows:

1. Selected Abstract Award as Oral presentation (4 Awards)

1.1 Awardee: Napasakorn Verasaksuriya

P-045: REPURPOSING UBIQUITIN C-TERMINAL HYDROLASE L1 (UCHL1) INHIBITION INTO A SENESCENCE-DRIVING STRATEGY.

Napasakorn Verasaksuriya, Nattawadee Panyain, Kowit Hengphasatporn, Yasuteru Shigeta, Krisada Rungsang, Monruedee Sukprasansap, Tewin Tencomnao, Sunhapas Soodvilai, Benjamin Ongnok

Receives a Certificate and a cash prize of 4,000 THB

(Professor Ingrid Y Liu award 2,000 THB and CMUTEAM 2,000 THB)

1.2 Awardee: Vikas Kumar Tiwari

P-015: A NEW BRAIN SIGNAL RECORDED FROM TMS IN PRIMATES: PREDICTING MOTOR PARALYSIS OUTCOMES

Tiwari VK, Honda Y, Ogawa K, Nakamura S, Tsutsui KI

Receives a Certificate and a cash prize of 3,000 THB

(Professor Ingrid Y Liu award 2,000 THB and CMUTEAM 1,000 THB)

1.3 Awardee: Anda Cimpean

P-002: 4-METHYLBELLIFERONE REVERSES AGE-RELATED CHANGES IN NEUROPLASTICITY AND NEUROINFLAMMATION

Anda Cimpean, Jana Dubisova, Pavla Jendelova, James W. Fawcett, Jessica C.F. Kwok

Receives a Certificate and a cash prize of 2,000 THB

(Professor Ingrid Y Liu award 2,000 THB)

1.4 Awardee: Raj Katariya

P-031: THERAPEUTIC TARGETING OF CENTRAL AGMATINE SIGNALING FOR INTERVENTION OF HUNTINGTON'S DISEASE

Raj Katariya, Brijesh Taksande

Receives a Certificate and a cash prize of 2,000 THB

(Professor Ingrid Y Liu award 2,000 THB)

2. Flash Talk Award as Oral presentation (3 Awards)

Each receives a Certificate and a cash prize of 1,000 THB from TNS

2.1 Awardee: Tara Sudhadevi

P-060: INHIBITION OF SPHINGOSINE KINASE 1 BY PF543 IMPROVES AIRWAY HYPERREACTIVITY AND NEUROCOGNITIVE OUTCOMES IN A MURINE MODEL OF BRONCHOPULMONARY DYSPLASIA

Tara Sudhadevi, Prathima Basa, Ian Adams and Anantha Harijith

2.2 **Awardee:** Chattraporn Nantawanichakorn

P-058: THERAPEUTIC POTENTIAL OF EXOSOMES DERIVED FROM STEM CELLS FROM HUMAN EXFOLIATED DECIDUOUS TEETH (SHEDs) IN IMPROVING NEUROLOGICAL AND MOTOR RECOVERY AFTER ISCHEMIC STROKE IN RATS

Chattraporn Nantawanichakorn, Hathaitip Sritanaudomchai, Kovit Pattanapanyasat, Dungdol Narasirilk, Jinnipa Matchwong, Sukonthar Ngampramuan

2.3 **Awardee:** Yaowapa Trangan

P-066: NON-INVASIVE ASSESSMENT OF AGE-RELATED SWEAT 3-HYDROXYANTHRANILIC ACID LEVELS: IMPLICATIONS FOR EARLY DETECTION OF COGNITIVE DECLINE

Yaowapa Trangan, Kanlaya Prapainop Katewongsa, Preeyanuch Manohon, Rungrat Palakai, Sirinapa Mysook, Banthit Chetsawang, Piyawat Katewongsa

3. Best Poster Presentation Awards

3.1 Prof. Dr. Prasop Ratanakorn Foundation Award for Outstanding Research Achievement

Receives a Certificate and a cash prize of 4,000 THB

Awardee: Tai Chaiamarit

P-055: REGULATORY MECHANISMS OF MEMBRANE TRAFFICKING TRANSCRIPT LOCALIZATION AT THE DROSOPHILA NEUROMUSCULAR SYNAPSE

Tai Chaiamarit, Danail Stoychev, Francesca Robertson, Jeffrey Y Lee, Ilan Davis

3.2 Professor Roongtarn Ladpli Award

Receives a Certificate and a cash prize of 2,000 THB

Awardee: Jiasui Yu

P-063: MUSCLE-DERIVED APELIN MEDIATES EXERCISE-INDUCED ANTIDEPRESSANT EFFECTS AND HIPPOCAMPAL NEUROPLASTICITY

Jiasui Yu, Suk-yu Yau

3.3 Associate Professor Naiphinich Kotchabhakdi Award

Receives a Certificate and a cash prize of 2,000 THB

Awardee: Pacharaporn Suklai

P-068: A HUMAN STEM CELL-DERIVED BIOENGINEERED PLATFORM WITH DEFINED CIRCUIT ARCHITECTURE FOR SYNAPTIC CONNECTIVITY ANALYSIS

Pacharaporn Suklai, Andrea Serio

3.4 Professor Ingrid Y Liu Award

Receives a Certificate and a cash prize of 2,000 THB

Awardee: Nuttanan Pholngam

P-025: HIPPOCAMPAL NEURODEGENERATION AND COGNITIVE IMPAIRMENT IN AGING β -THALASSEMIA MICE

Nuttanan Pholngam, Parinda Jamrus, Kittikun Viwatpinyo, Benjaporn Kiatpakdee, Jim Vadolas, Pornthip Chaichompoo, Sukonthar Ngampramuan, Saovaros Svasti

3.5 Mr. Tay Khoon Yang Award (Hi-Tech Imaging Co. Ltd.)

Receives a Certificate and a cash prize of 2,000 THB

Awardee: Mubashir Raza

P-043: TCU412 AMELIORATES AMYLOID BETA-INDUCED TOXICITY IN HT22 CELLS AND MEMORY IMPAIRMENTS IN 3XTG-AD MICE

Mubashir Raza, Ingrid Y. Liu

3.6 Professor Pavich Tongroach Award (2 Awards, 4000 THB)

3.6.1 **Awardees:** Sukrit Promtang

P-014: METASCAPE-GUIDED TRANSCRIPTOMIC ANALYSIS OF 2-BTHF TREATMENT FROM HOLOTHURIA SCABRA IN A 6-OHDA-EXPOSED C. ELEGANS MODEL OF PARKINSON'S DISEASE

Sukrit Promtang, Pawanrat Chalorak, Darunee Rodma, Rungsarit Sunan, Apinya Sayinta, and Krai Meemon

Receives a Certificate and a cash prize of 2,000 THB

3.6.2 **Awardees:** Shin-Meng Deng

P-041: NEURONAL PNN DEFICIENCY LEADS TO BRAIN STRUCTURAL ALTERATIONS AND BEHAVIORAL ABNORMALITIES IN MICE

Shin-Meng Deng, Sujira Mukda, Steve Leu

Receives a Certificate and a cash prize of 2,000 THB

Congratulations to all award recipients for their outstanding contributions and dedication to advancing neuroscience research and innovation.

President, Thai Neuroscience Society

October 29-31, 2025



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The 29th Annual Thai Neuroscience Society Conference 2026 (TNS29)

หัวข้อ Neurodevelopment, Aging, and Neurodegeneration: Innovation and Implication

ด้วยสมาคมประสาทวิทยาศาสตร์ไทย (Thai Neuroscience Society; TNS) ร่วมกับสถาบันชีววิทยาศาสตร์โมเลกุล มหาวิทยาลัยมหิดล ได้กำหนดจัดการประชุมวิชาการนานาชาติด้านประสาทวิทยาศาสตร์ครั้งที่ 29 (The 29th Annual Thai Neuroscience Society Conference 2026 (TNS29) ภายใต้หัวข้อ “Neurodevelopment, Aging, and Neurodegeneration: Innovation and Implication” โดยการประชุมวิชาการดังกล่าวจัดร่วมกับ TNS Neuro Quiz 2026 และ SKT Workshops ระหว่างวันที่ 5 – 7 สิงหาคม พ.ศ. 2569 ณ โรงแรมอมารีนางาแสน จังหวัดชลบุรี โดยมีวัตถุประสงค์เพื่อติดตามวิทยาการความก้าวหน้าในวิชาการด้านประสาทวิทยาศาสตร์จากวิทยากรผู้ทรงคุณวุฒิ มีการแสดงผลงานทางวิชาการจากคณาจารย์ นักวิทยาศาสตร์ นักวิจัย นักศึกษา นักเรียนมัธยมปลาย สมาชิกสมาคมฯ และบุคคลทั่วไปที่สนใจ เพื่อเป็นการส่งเสริมและกระตุ้นให้ผู้เข้าร่วมประชุมฯ เข้าถึงความรู้ ความก้าวหน้าทางด้านประสาทวิทยาศาสตร์ที่ทันสมัย สามารถนำไปประยุกต์ใช้ทั้งในการดำเนินชีวิต และพัฒนาการจัดการเรียนการสอนในหลักสูตรวิทยาศาสตร์สุขภาพที่เกี่ยวข้อง รวมทั้งได้เผยแพร่ผลงานวิจัย แลกเปลี่ยนความรู้และวิทยาการต่าง ๆ ในสาขาวิชาประสาทวิทยาศาสตร์ และสาขาอื่น ๆ ที่เกี่ยวข้อง เสริมโอกาสสร้างความร่วมมือของนักวิชาการ นักวิจัยระหว่างสถาบันทั้งในประเทศ และระหว่างประเทศ

เพื่อให้การดำเนินงานเป็นไปด้วยความเรียบร้อย คล่องตัว และมีประสิทธิภาพ อาศัยอำนาจตามความในมาตรา 37 แห่งพระราชบัญญัติมหาวิทยาลัยมหิดล พ.ศ. 2550 ผู้อำนวยการสถาบันชีววิทยาศาสตร์โมเลกุล โดยความเห็นชอบของคณะกรรมการประจำสถาบันชีววิทยาศาสตร์โมเลกุล ในการประชุมครั้งที่ 14 (1/2569) เมื่อวันที่ 29 มกราคม 2569 จึงแต่งตั้งคณะกรรมการจัดการประชุมวิชาการนานาชาติด้านประสาทวิทยาศาสตร์ครั้งที่ 29 (The 29th Annual Thai Neuroscience Society Conference 2026 (TNS29) ภายใต้หัวข้อ “Neurodevelopment, Aging, and Neurodegeneration: Innovation and Implication” ดังมีรายนามต่อไปนี้

ที่ปรึกษาคณะกรรมการ

1. ศาสตราจารย์ ดร. ภาวิธ ทองโรจน์
2. ศาสตราจารย์เกียรติคุณ ดร. ปิยะรัตน์ ไกรวิตรพงศ์
3. ศาสตราจารย์เกียรติคุณ ดร. สุขุมาล จงธรรมคุณ
4. รองศาสตราจารย์ ดร. เกสกรหญิงกนกวรรณ ตีลกสกุลชัย
5. รองศาสตราจารย์ ดร. ม.ล.เสาวรส สวัสดิ์วัฒน์

คณะกรรมการดำเนินการประชุมวิชาการ

1. ศาสตราจารย์ ดร.บัณฑิต เจตน์สว่าง	ประธานกรรมการ
2. ศาสตราจารย์เกียรติคุณ ดร. พันโทหญิงสุพิน ชมภูพงษ์	ประธานกรรมการร่วม
3. ผู้ช่วยศาสตราจารย์ ดร.นริศร กิตยานันท์	รองประธานกรรมการ
4. ศาสตราจารย์ ดร.สุทิสลา ถาน้อย	กรรมการ
5. ศาสตราจารย์ ดร.สมพร กันทรดุษฎี เตรียมชัยศรี	กรรมการ
6. รองศาสตราจารย์ ดร.กุลธิดา ชัยธีระยานนท์	กรรมการ
7. รองศาสตราจารย์ ดร. นพ.ชัยรัตน์ เต็บไพบูลย์	กรรมการ
8. รองศาสตราจารย์ ดร. นพ.ชัยเลิศ พิธิพรชัย	กรรมการ
9. รองศาสตราจารย์ ดร. นพ.นราวุฒิ ภาคาพรต	กรรมการ
10. รองศาสตราจารย์ ดร. นพ.วรสิทธิ์ ศิริพรพาณิชย์	กรรมการ
11. รองศาสตราจารย์ ดร. ภก.อัศเดช ศิริพร	กรรมการ
12. ผู้ช่วยศาสตราจารย์ ดร.ประทีป อมรัตน์พันธ์	กรรมการ
13. ผู้ช่วยศาสตราจารย์ ดร.มนัสพร พัสระ	กรรมการ
14. ผู้ช่วยศาสตราจารย์ ดร. นพ.อดิสร รัตนโยธา	กรรมการ
15. ผู้ช่วยศาสตราจารย์ ดร. นพ.อมรพันธุ์ เสริมาศพันธุ์	กรรมการ
16. อาจารย์ ดร.อัษฎราภรณ์ คำศรีใจ	กรรมการ
17. รองศาสตราจารย์ ดร.สุจิตรา มุกดา	กรรมการและเลขานุการ
18. ผู้ช่วยศาสตราจารย์ ดร.จิราพร ปานมณี	กรรมการและเลขานุการ
19. นางสมทรง พึ่งสุขแดง	กรรมการและผู้ช่วยเลขานุการ
20. นางสาวแก้วเกล้า บรรจง	กรรมการและผู้ช่วยเลขานุการ

โดยให้คณะกรรมการดังกล่าว มีหน้าที่

- วางแผน กำหนดเนื้อหาทางวิชาการของการประชุม เตรียมงาน และประสานงานกับฝ่ายต่าง ๆ
- ดำเนินการ จัดประชุมและกิจกรรมที่เกี่ยวข้อง และสรุปติดตามการดำเนินงาน

คณะกรรมการฝ่ายวิชาการ

1. ศาสตราจารย์ ดร.บัณฑิต เจตน์สว่าง	ประธานอนุกรรมการ
2. รองศาสตราจารย์ ดร.อัศเดช ศิริพร	ประธานอนุกรรมการร่วม
3. รองศาสตราจารย์ ดร. นพ.ชัยรัตน์ เต็บไพบูลย์	รองประธานอนุกรรมการ
4. ศาสตราจารย์ ดร.สุทิสลา ถาน้อย	อนุกรรมการ
5. รองศาสตราจารย์ ดร.นวลจันทร์ จุฑาภักดีกุล	อนุกรรมการ
6. รองศาสตราจารย์ ดร. นพ.วรสิทธิ์ ศิริพรพาณิชย์	อนุกรรมการ
7. รองศาสตราจารย์ ดร.สุจิตรา มุกดา	อนุกรรมการ
8. ผู้ช่วยศาสตราจารย์ ดร.จิราพร ปานมณี	อนุกรรมการ

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| 9. ผู้ช่วยศาสตราจารย์ ดร.นริศร กิตยานันท์ | อนุกรรมการ |
| 10. ผู้ช่วยศาสตราจารย์ ดร.สุคนธา งามประมวญ | อนุกรรมการ |
| 11. อาจารย์ ดร.สุเมธ กล่อมจิตเจริญ | อนุกรรมการ |
| 12. อาจารย์ ดร.เอกพจน์ คงกล้า | อนุกรรมการ |
| 13. อาจารย์ ดร.สิริประภา บุปผาหอม | อนุกรรมการและเลขานุการ |

โดยให้คณะอนุกรรมการดังกล่าวมีหน้าที่

1. ติดต่อวิทยากรด้วยการออกจดหมายเชิญ จัดเตรียมประวัติวิทยากร บทความ หรือบทคัดย่อของวิทยากร
2. จัดลำดับการบรรยาย กำหนดประธาน รองประธานในแต่ละช่วงการประชุม
3. รับผลงานทางวิชาการจากผู้เข้าร่วมประชุมเพื่อพิจารณาคัดเลือกผลงานสำหรับเข้าร่วมประชุม พร้อมกำหนดลำดับหัวข้อการนำเสนอผลงานทางวิชาการ
4. ดำเนินการเรื่องรางวัลการนำเสนอผลงาน กำหนดกรรมการ และพิจารณาตัดสินผลสำหรับผลงานทางวิชาการของผู้เข้าร่วมประชุม
5. จัดทำต้นฉบับหนังสือบทความหรือบทคัดย่อของการบรรยายของวิทยากร ผลงานทางวิชาการที่เข้าร่วมนำเสนอในการประชุมและเอกสารผู้สนับสนุนการประชุมในรูปแบบอิเล็กทรอนิกส์ และ/หรือรูปเล่ม

คณะอนุกรรมการฝ่ายกิจกรรมพิเศษ: TNS Neuro Quiz, SKT workshop และ IL workshop

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| 1. รองศาสตราจารย์ ดร. นพ.ชัยเลิศ พิชิตรพชัย | ประธานอนุกรรมการ |
| 2. ศาสตราจารย์ ดร.สมพร กันทรดุษฎี เตรียมชัยศรี | รองประธานอนุกรรมการ |
| 3. ศาสตราจารย์เกียรติคุณ ดร.พันโทหญิงสุพิน ชมภูพงษ์ | อนุกรรมการ |
| 4. ผู้ช่วยศาสตราจารย์ ดร. นพ.อมรพันธุ์ เสรีมาศพันธุ์ | อนุกรรมการ |
| 5. อาจารย์ ดร.ประทีป อมรรัตนพันธ์ | อนุกรรมการ |
| 6. ดร.รุจี พรชัย | อนุกรรมการ |
| 7. ดร.ชนติ จันทระโชติชัชวาล | อนุกรรมการ |
| 8. อาจารย์ ดร.อัจฉราภรณ์ คำศรีใจ | อนุกรรมการและเลขานุการ |

โดยให้คณะอนุกรรมการดังกล่าวมีหน้าที่

1. จัดทำสื่อประชาสัมพันธ์กิจกรรม TNS Neuro Quiz, SKT workshop และ Innovative Learning Activity workshop
2. จัดทำการตอบคำถามรอบคัดเลือกแบบออนไลน์ และประสานงานด้านการเตรียมความพร้อมสื่อคอมพิวเตอร์เวที และห้องประชุมเพื่อการตอบคำถามรอบชิงชนะเลิศแบบ On-site
3. ดำเนินการจัดกิจกรรม SKT Workshop
4. ดำเนินการจัดกิจกรรม Innovative Learning Activity workshop
5. จัดหาผู้สนับสนุนหลัก เพื่อการมอบผลิตภัณฑ์ ของที่ระลึก โล่ รางวัล สำหรับ TNS Neuro Quiz
6. จัดเตรียมใบประกาศนียบัตรการเข้าร่วมกิจกรรม

คณะกรรมการฝ่ายพิธีการ

- | | |
|---|------------------------|
| 1. ผู้ช่วยศาสตราจารย์ ดร.สุคนธา งามประมวญ | ประธานอนุกรรมการ |
| 2. ศาสตราจารย์ ดร.พนัสนิธิพิณ ชมภูพงษ์ | อนุกรรมการ |
| 3. รองศาสตราจารย์ ดร. นพ.ชัยรัตน์ เต็บไพบูลย์ | อนุกรรมการ |
| 4. รองศาสตราจารย์ ดร. นพ.นราวุฒิ ภาคาพรต | อนุกรรมการ |
| 5. นางสาวทรง พึ่งสุขแดง | อนุกรรมการและเลขานุการ |

โดยให้คณะกรรมการดังกล่าวมีหน้าที่

1. จัดเตรียมพิธีการ และพิธีกรตลอดการประชุม
2. ประสานงานด้านการนำเสนอในการบรรยายและโปสเตอร์ การเตรียมความพร้อมสื่อคอมพิวเตอร์
3. ดำเนินการประชุม จัดการเรื่องเวลาการนำเสนอ ป้ายชื่อวิทยากร และการมอบของที่ระลึก เกียรติบัตร รางวัล และโล่ประกาศเกียรติคุณ

คณะกรรมการฝ่ายประชาสัมพันธ์และลงทะเบียน

- | | |
|--|------------------------|
| 1. ผู้ช่วยศาสตราจารย์ ดร.จิราพร ปานมณี | ประธานอนุกรรมการ |
| 2. อาจารย์ ดร.สิริประภา บุปผาหอม | อนุกรรมการ |
| 3. นายสุจิต วัชรไทย์ | อนุกรรมการ |
| 4. นายชนกฤต นิมนวล | อนุกรรมการ |
| 5. นางสาวปิณิจา รัชตเมธาวิ | อนุกรรมการ |
| 6. นางสาวณัฏฐณิชา ศรีมาเสริม | อนุกรรมการและเลขานุการ |

โดยให้คณะกรรมการดังกล่าวมีหน้าที่

1. ประชาสัมพันธ์การประชุมวิชาการให้หน่วยงานทั้งภายใน และภายนอกทราบผ่านทางรูปแบบต่าง ๆ จัดการและควบคุมเกี่ยวกับข้อมูลที่ได้รับจากการลงทะเบียน และจัดการด้านจดหมายอิเล็กทรอนิกส์
2. ดำเนินการด้านการลงทะเบียนการประชุมผ่านทางเว็บไซต์และสื่อออนไลน์อื่น
3. ประสานงานรายชื่อผู้เข้าร่วมกิจกรรมต่าง ๆ จัดทำสรุปจำนวนรายชื่อผู้เข้าร่วมกิจกรรม วิทยากร และจัดทำเอกสารลงทะเบียน
4. ดำเนินการรับลงทะเบียนและแจกเอกสารให้ผู้เข้าร่วมกิจกรรม
5. จัดเตรียมวัสดุ เอกสารลงทะเบียน ป้ายชื่อ และใบประกาศนียบัตรการเข้าร่วมการประชุม
6. จัดทำรูปแบบประเมินโครงการ และสรุปการประเมินโครงการ

คณะกรรมการฝ่ายสถานที่ และ招待ศุภกรณ์

- | | |
|---|------------------|
| 1. ผู้ช่วยศาสตราจารย์ ดร.นริศร กิตยานันท์ | ประธานอนุกรรมการ |
| 2. อาจารย์ ดร.สุเมธ กล่อมจิตเจริญ | อนุกรรมการ |
| 3. นางสาวกรกนก พรหมเทพ | อนุกรรมการ |
| 4. นายสุจิต วัชรไทย์ | อนุกรรมการ |

5. นางสาวแก้วเกล้า บรรจง

อนุกรรมการ

6. นางสาวปิ่นจุฑา รัชตเมธาวิณ

อนุกรรมการและเลขานุการ

โดยให้คณะอนุกรรมการดังกล่าวมีหน้าที่

1. จัดเตรียม/ประสานงานด้านสถานที่และโสตทัศนูปกรณ์ เช่น เครื่องเสียง เครื่องคอมพิวเตอร์ และอุปกรณ์อื่น
2. จัดเตรียมป้ายประชาสัมพันธ์สถานที่
3. จัดหาอุปกรณ์ บอร์ด และควบคุมการจัดตั้งสำหรับการนำเสนอผลงานในรูปแบบโปสเตอร์และบูธนิทรรศการ

คณะอนุกรรมการฝ่ายจัดการรายได้และการเงิน

1. รองศาสตราจารย์ ดร.กุลธิดา ชัยธีระยานนท์

ประธานอนุกรรมการ

2. ผู้ช่วยศาสตราจารย์ ดร.นริศร กิตยารักษ์

รองประธานอนุกรรมการ

3. ศาสตราจารย์เกียรติคุณ ดร.พินโทหญิงสุพิน ชมภูพงษ์

อนุกรรมการ

4. ศาสตราจารย์เกียรติคุณ ดร.สุพมาล จงธรรมคุณ

อนุกรรมการ

5. รองศาสตราจารย์ ดร.สุจิตรา มุกดา

อนุกรรมการ

6. นางสาวอมรรัตน์ อ่อนจันทร์

อนุกรรมการและเลขานุการ

โดยให้คณะอนุกรรมการดังกล่าวมีหน้าที่

1. ดูแลงบประมาณ และการเบิกจ่ายให้กับคณะอนุกรรมการฝ่ายต่าง ๆ ทำบัญชีการเงินรายรับ – รายจ่ายของการประชุม รวมถึงการเก็บรวบรวม และตรวจสอบเอกสารประกอบการเบิกจ่าย
2. ทำเอกสารเพื่อหารายได้ และผู้สนับสนุนการจัดประชุม เพื่อจัดแสดงนิทรรศการในงาน

ทั้งนี้ ตั้งแต่บัดนี้เป็นต้นไป

สั่ง ณ วันที่ 11 กุมภาพันธ์ พ.ศ. 2569

18115 สรอสจธ

(รองศาสตราจารย์ ดร. หม่อมหลวงเสาวรส สวัสดิ์วัฒน)

ผู้อำนวยการสถาบันชีววิทยาศาสตร์โมเลกุล



คำสั่งสถาบันชีววิทยาศาสตร์โมเลกุล มหาวิทยาลัยมหิดล

ที่ 51/2569

เรื่อง แก้ไขเพิ่มเติมคำสั่งแต่งตั้งคณะกรรมการจัดการประชุมวิชาการนานาชาติด้านประสาทวิทยาศาสตร์

The 29th Annual Thai Neuroscience Society Conference 2026 (TNS29)

หัวข้อ Neurodevelopment, Aging, and Neurodegeneration: Innovation and Implication

อนุสนธิคำสั่งสถาบันชีววิทยาศาสตร์โมเลกุล มหาวิทยาลัยมหิดล ที่ 18/2569 ลงวันที่ 11 กุมภาพันธ์ พ.ศ. 2569 ได้แต่งตั้งคณะกรรมการจัดการประชุมวิชาการนานาชาติด้านประสาทวิทยาศาสตร์ The 29th Annual Thai Neuroscience Society Conference 2026 (TNS29) นั้น เนื่องจากมีความจำเป็นต้องปรับปรุงเพิ่มเติมรายชื่อคณะกรรมการ เพื่อให้การดำเนินงานเป็นไปด้วยความเรียบร้อยและมีประสิทธิภาพมากขึ้น

อาศัยอำนาจตามความในมาตรา 37 แห่งพระราชบัญญัติมหาวิทยาลัยมหิดล พ.ศ. 2550 ผู้อำนวยการสถาบันชีววิทยาศาสตร์โมเลกุล จึงให้แก้ไขเพิ่มเติมคำสั่งดังกล่าว ดังนี้

คณะกรรมการดำเนินการประชุมวิชาการ

เดิม

- | | |
|---|----------------------------|
| 17. รองศาสตราจารย์ ดร.สุจิตรา มุกดา | กรรมการและเลขานุการ |
| 18. ผู้ช่วยศาสตราจารย์ ดร.จิราพร ปานมณี | กรรมการและเลขานุการ |
| 19. นางสมทรง พึ่งสุขแดง | กรรมการและผู้ช่วยเลขานุการ |
| 20. นางสาวแก้วเกล้า บรรจง | กรรมการและผู้ช่วยเลขานุการ |

แก้ไขเป็น

- | | |
|---|----------------------------|
| 17. ผู้ช่วยศาสตราจารย์ ดร.จิราพร ปานมณี | กรรมการ |
| 18. นางสาวชลนิชา สืบโกฏ | กรรมการ |
| 19. รองศาสตราจารย์ ดร.สุจิตรา มุกดา | กรรมการและเลขานุการ |
| 20. นางณัฏฐณิชา ศรีมาเสริม | กรรมการ |
| 21. นางสมทรง พึ่งสุขแดง | กรรมการและผู้ช่วยเลขานุการ |
| 22. นางสาวแก้วเกล้า บรรจง | กรรมการและผู้ช่วยเลขานุการ |

คณะกรรมการฝ่ายประชาสัมพันธ์และลงทะเบียน

เดิม

6. นางสาวณัฏฐณิชา ศรีมาเสริม

อนุกรรมการและเลขานุการ

แก้ไขเป็น

6. นางสาวชลนิชา สืบโกฏ

อนุกรรมการ

7. นางสาวณัฏฐณิชา ศรีมาเสริม

อนุกรรมการและเลขานุการ

8. นางสาวลลิกษา เอี่ยมอินทร์

อนุกรรมการและผู้ช่วยเลขานุการ

คณะกรรมการฝ่ายจัดหารายได้และการเงิน

เดิม

6. นางสาวอมรรรัตน์ อ่อนจันทร์

อนุกรรมการและเลขานุการ

แก้ไขเป็น

6. นางสาวทิชากร นาคสีหมอก

อนุกรรมการ

7. นางสาวอมรรรัตน์ อ่อนจันทร์

อนุกรรมการและเลขานุการ

โดยให้คงข้อความอื่น ๆ ในคำสั่งสถาบันชีววิทยาศาสตร์โมเลกุล มหาวิทยาลัยมหิดล ที่ 18/2569 ลงวันที่ 11 กุมภาพันธ์ พ.ศ. 2569 ไว้ตามเดิมทุกประการ

ทั้งนี้ ตั้งแต่บัดนี้เป็นต้นไป

สั่ง ณ วันที่ 7 เมษายน พ.ศ. 2569

(รองศาสตราจารย์ ดร. หม่อมหลวงเสาวรส สวัสดิ์วัฒน)

ผู้อำนวยการสถาบันชีววิทยาศาสตร์โมเลกุล



Amari

BANGSAEN

Caspian Room

MAITREE

Hotel Lobby

AMA'YA
FOOD GALLERY

Pacific
Grand Ballroom

Atlantic
Room

EV Charger
Station

Car Park

Caribbean Room

Andaman Room

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11

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BAR & LOBBY

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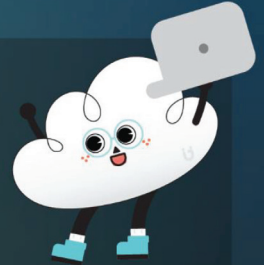
Marlin Fish
Fountain

Naha

Bangsaen Beach Road

สาขา U•Store by UFicon

U•Store at Siriraj Piyamaharajkarun Hospital
U•Store at Srinakharinwirot University
U•Store at Srinakharinwirot University Ongkharak Campus
U•Store at Suratthani Rajabhat University
U•Store at Prince of Songkla University
U•Store at Ubon Ratchathani Rajabhat University
U•Store at Walailak University
U•Store at Walailuk University Hospital



สาขา HUAWEI Store by UFicon

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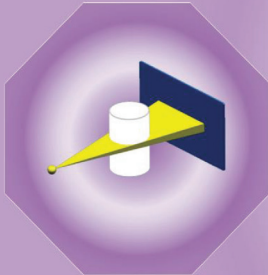
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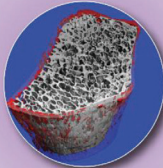
HI-TECH IMAGING Co., Ltd.

Solutions Beyond Expectation

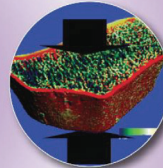
SCANCO MEDICAL Multi-dimensional X-ray Computed Tomography



Dental Implant



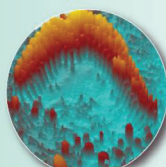
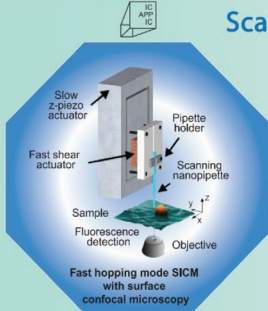
Human radius
cortical porosity



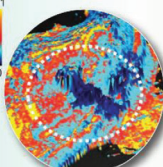
Human radius
FE analysis



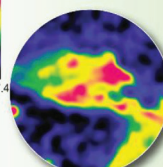
Scanning Ion Conductance Microscopy (SICM)



3D Stereocilia



3D Nanoscale Mechanics

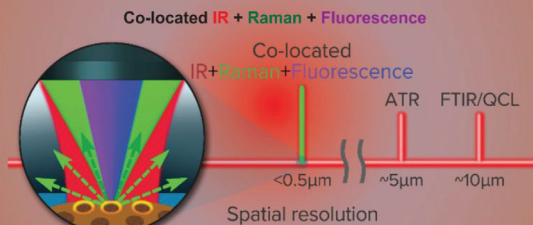
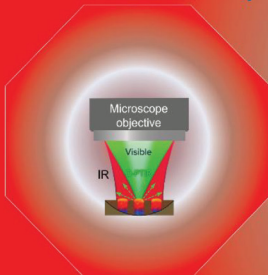


3D pH mapping

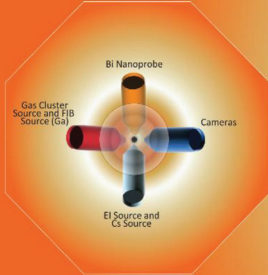


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Spectrum

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Image

Depth Profiling



Depth Profiling

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