INTERNATIONAL BRAIN IBRO RESEARCH ORGANIZATION

THE THAI NEUROSCIENCE SOCIETY

CMU CHIANG MAI UNIVERSITY

FACULTY OF MEDICINE CHIANG MAI UNIVERSITY

Neuroplasticity Across the Lifespan

28th Annual Thai Neuroscience Society Conference

29 – 31 OCT 2025

Kantary Hills Hotel

IBRO-APRC Supported Associate School

27 OCT - 2 NOV 2025, Faculty of Medicine, Chiang Mai University

supported by INTERNATIONAL BRAIN IBRO RESEARCH ORGANIZATION

Proceedings & Abstracts

tns28.info@gmail.com

https://thaineuroscience.org/conference/tns28/

The 28th Annual Thai Neuroscience Society Conference

แบรนด์
ซุปไก่สกัด
เปิดวัน
เปิด
แบรนด์
BRAND'S
SUNTORY
แบรนด์
ซุปไก่สกัด
ออริจินัล
มีวิตามิน บี12
คาร์โนซีน
35 มก. / ขวด**
ปริมาตรสุทธิ 70 มล.
วิตามินบี 12 มีส่วนช่วยในการทำงานตามปกติของระบบประสาทและสมอง
วิตามิน บี12
คาร์โนซีน
Scan Me
ข้อมูลข่าวสารผลิตภัณฑ์
ฆอ.439/2568
บริษัท ซันโทรี่ เบเวอเรจ แอนด์ ฟู้ด (ประเทศไทย) จำกัด 88 อาคารเดอะปาร์ค ชั้น 15
ถนนรัชดาภิเษก แขวง/เขตคลองเตย กรุงเทพฯ 10110 โทร 02-079-7979


INTERNATIONAL BRAIN IBRO RESEARCH ORGANIZATION
CMU CHIANG MAI UNIVERSITY
FACULTY OF MEDICINE CHIANG MAI UNIVERSITY
Neuroplasticity Across the Lifespan
28th Annual Thai Neuroscience Society Conference
29 - 31 OCT 2025
Kantary Hills Hotel, Chiang Mai, Thailand
IBRO-APRC Supported Associate School
27 OCT - 2 NOV 2025, Faculty of Medicine, Chiang Mai University, Thailand
TNS Neuro-Quiz 2025
International Final Round on October, 29, 2025,
10.00-12.00 at Kantary Hills Hotel, Chiang Mai, Thailand
TNS NEURO QUIZ
Assoc. Prof. Chailerd Pichitpornchai
Mahidol university, thailand
Assoc. Prof. Akkradate Siriphorn
Chulalongkorn University, Thailand
Asst. Prof. Prateep Amonruttanapun
Thammasat University, Thailand
SKT Workshop Supported by BRANDS
• Thai Language
29 OCT 2025, 2 PM - 5 PM
• English Language
31 OCT 2025, 9 AM - 12 PM
Kantary Hills Hotel, Chiang Mai, Thailand
Prof. Somporn Kantharadussadee Triamchaisri
PLENARY LECTURES
Distinguished Prof.
Siriporn Chattipakorn
Chiang Mai University, Thailand
Prof. Vorasuk Shotelersuk
Chulalongkorn University,
Thailand
Prof. Daesoo Kim
Korea Advanced Institute of
Science & Technology, Korea
PROFESSOR DR PRASOP RATANAKORN'S LECTURE
Distinguished University Chair
Prof. Long-Jun Wu
University of Texas, USA
PROFESSOR DR ROONGTAM LADPLI'S LECTURE
Prof. Ingrid Liu
Tzu Chi University,
Taiwan
I: Mechanisms of Neuroplasticity Across Development and Aging
Dr. Luca Lo Piccolo
Chiang Mai University,
Thailand
Dr. Arnaud Monteil
Institute of Functional
Genomics, France
Prof. Wael Mohamed
International Islamic
University, Malaysia
Assoc. Prof.Vorasith Siripornpanich
Mahidol University,
Thailand
Dr. Ranchana Yeewa
Chiang Mai University,
Thailand
II: Advanced Imaging and Omics Technologies for Studying Neuroplasticity
Prof. Sutisa Nudmamud-Thanoi
Naresuan University,
Thailand
Assoc. Prof. Ha Thi Thanh Huong
International University Vietnam
National University, Vietnam
Dr. Catleya Rojviriya
Synchrotron Light Research
Institute, Thailand
Prof. Banthit Chetsawang
Mahidol University,
Thailand
III. Gene Editing and Cell-Based Approaches to Modulate Neuroplasticity
Prof. Toshihide Yamashita
The University of Osaka,
Japan
Assoc. Prof. Hideki Yoshida
Kyoto Institute of Technology,
Japan
Dr. Nithi Asavapanumas
Mahidol University,
Thailand
IV: Pharmacological and Lifestyle Interventions to Enhance Neuroplasticity
Dr. Hiranya Pintana
Chiang Mai University,
Thailand
Dr. Salinee Jantrapirom
Chiang Mai University,
Thailand
Assoc. Prof. Narawut Pakaprot
Mahidol University,
Thailand
V: Neuroplasticity Across Systems: From Social Behavior to Cognitive Aging and Brain-Gut Interactions
Distinguished Prof. and Chief
Director Ishwar Parhar
University of Toyama, Japan
Dr. Lalitta Suriya-Arunroj
Chulalongkorn University,
Thailand
Asst. Prof. Amornpun Sereemaspun
Chulalongkorn University,
Thailand
IMPORTANT DATES
Abstract Submission : 1 - March 2025 - 30 September 2025
Early Bird Registration : 1 - March 2025 - 30 September 2025
Regular Registration : 1 - March 2025 - 31 October 2025
tns28.info@gmail.com
https://thaineuroscience.org/conference/tns28/
The 28th Annual Thai Neuroscience Society Conference
BRANDS
SCI-ED
HI-TECH IMAGING CO., LTD. Your Ultimate Strategic Partner
Bio Design
THERA TRADING

Proceedings & Abstracts

28th Annual Thai Neuroscience Society Conference

TNS28

"Neuroplasticity Across the Lifespan: Advancing Neuroplasticity Research through cutting-edge methodologies"

OCTOBER 29-31, 2025

Chiang Mai, Thailand

Venue	Kantary Hills Hotel, Chiang Mai, Thailand
Organized by	Thai Neuroscience Society (TNS) and Faculty of Medicine, Chiang Mai University
Hosted by	Chaing Mai University
Supported by	International Brain Research Organization Asia Pacific Regional Committee (IBRO-APRC) Chaing Mai University Thai Neuroscience Society

Editor
Scientific and Reviewing Committee TNS28

Cover design by Chansunee Panto
Flyer design by Chansunee Panto
TNS Neuro Quiz 2025 Logo design by Sukrit Promtang

The Proceedings and Abstracts of 28th Thai Neuroscience Society Annual Conference, October 29th-31st, 2025

150 Copies / October 2025
ISBN 978-616-629-699-0

IBRO-APRC Supported Winter School on Neuroplasticity Across the Lifespan & 28th Annual Conference of Thai Neuroscience Society (TNS28)

Program

Wed 29-10-2025 (DAY 1)				
	Time		**Main Room** (DOI SUTHEP 1-2)	**Workshop**
DAY 1	08:30	-	**Registration** at Kantary Hotel, Chiang Mai	
	09:30	10:00	Time break	
	10:00	12:00	**International Final Round: TNS Neuro Quiz 2025** **Assoc. Prof. Chailerd Pichitpornchai, MD, PhD** Mahidol University, Thailand **Assoc. Prof. Akkradate Siriphorn, PhD** Chulalongkorn University, Thailand **Asst. Prof. Prateep Amonruttanapun, PhD** Thammasat University, Thailand	
	12:00	13:30	Lunch	
	Opening Ceremony			
	13:30	13:40	**Opening remarks** **Prof. Dumnoensun Pruksakorn, MD, PhD** Head of CMUTEAM Faculty of Medicine, Chiang Mai University, Thailand	
	13:40	13:45	**Opening remarks** **Prof. Supin Chompoong, PhD** President of Thai Neuroscience Society Faculty of Medicine Siriraj Hospital, Mahidol University, Thailand	
	13:45	13:50	**TNS NEURO QUIZ 2025 Award ceremony**	
	13:50	13:55	**Announcement of Honorary Members**	
	13:55	14:10	**Group Photography**	
	Plenary Lectures			
	14:10	14:50	**Distinguished Prof. Siriporn Chattipakorn, DDS, PhD** Faculty of Medicine, Chiang Mai University, Thailand **Obesity, aging and cognitive impairment: mechanistic insight and non-pharmacological approaches**	14:00-17:00 (DOI NUA) **IL MU workshop** **Assoc. Prof. Chailerd Pichitpornchai, MD, PhD** (30 participants, free) 14:00-17:00 (DOI NANG at 2nd Fl) **SKT workshop (Thai language)** **Prof. Somporn Kantharadussadee Triamchaisri, PhD and Brand company** (50 registered participants)
	14:50	15:20	Time break	
	15:20	16:00	**Prof. Vorasuk Shotelersuk, MD** Faculty of Medicine, Chulalongkorn University, Thailand **Precision medicine: a new Era of healthcare**	
	16:00	16:40	**Prof. Daesoo Kim, PhD** KAIST institute for Biocentury KAIST institute for Health Science KAIST institute for Artificial Intelligence, Korea **Neuroscience takes AI to Tango**	
	17:00	17:30	**Poster put up** (PARTY ROOM)	
	17:30	-	**Welcome dinner*** ** ONLY for Invited speakers, IBRO lecturers, TNS, TNS28 organizing committee, IBRO students*	

IBRO-APRC Supported Winter School on Neuroplasticity Across the Lifespan & 28th Annual Conference of Thai Neuroscience Society (TNS28)

Thu 30-10-2025 (DAY 2)				
	Time		**Main Room** (DOI SUTHEP 1-2)	**Workshop**
DAY 2	**Professor Dr Prasop Ratanakorn's Lecture**			
	08:45	09:15	**Prof. Long-Jun Wu, PhD** University of Texas Health Science Center at Houston, USA **Road-shaped microglia regulate neuronal activity in TDP-43 neurodegeneration**	
	Plenary Session I: Mechanisms of Neuroplasticity Across Development and Aging			
	09:15	09:45	**Arnaud Monteil, PhD** University of Montpellier, France **Paving the path to therapies for genetic diseases linked to the NALCN channelosome**	
	09:45	10:15	**Prof. Wael Mohamed, MD, PhD** International Islamic University Malaysia, Malaysia **Knowing the unknown: Parkinson's hidden face**	
	10:15	10:45	Time break + **Poster evaluation** (PARTY ROOM)	
	10:45	11:15	**Assoc. Prof. Vorasith Siripornpanich, MD, PhD** Mahidol University, Thailand **Mind at rest, brain in motion: sleep and synaptic plasticity**	
	11:15	11:45	**Ranchana Yeewa, PhD** Chiang Mai University, Thailand **Epigenetic–Metabolic crossroads in neuroplasticity and brain aging**	
	11:45	13:30	Lunch	
	Professor Dr Roongtam Ladpli's Lecture			
	13:30	14:00	**Prof. Ingrid Liu, PhD** Tzu Chi University, Taiwan **From sleep to synapse: neuroplasticity impairment via Actin depolymerization in REM sleep-deprived mice**	
	Plenary Session II: Advanced Imaging and Omics Technologies for Studying Neuroplasticity			
	14:00	14:30	**Ha Thi Thanh Huong, PhD** International University, Vietnam National University, Vietnam **Comprehensive AI in Alzheimer's: from early diagnosis to progression insights across multiple modalities**	
	14:30	15:00	**Prof. Sutisa Nudmamud-Thanoi, PhD** Naresuan University, Thailand **Proteomic insights into cognitive performance: age-dependent sex differences in healthy Thai subjects**	
	15:00	15:30	Time break + **Poster evaluation** (PARTY ROOM)	
	15:30	16:00	**Catleya Rojviriya, PhD** Synchrotron Light Research Institute (SLRI), Thailand **Super-High resolution tomography imaging in neuroscience**	

Thu 30-10-2025 (DAY 2)				
	Time		**Main Room** (DOI SUTHEP 1-2)	**Workshop**
	16:00	16:30	**Prof. Banthit Chetsawang, PhD** Mahidol University, Thailand **The regulation of kynurenine pathway: a potential targeting to prevent neurodegeneration in methamphetamine-induced toxicity model**	
	16:30	16:45	**Selected Abstract #1** **Miss Napasakorn Verasaksuriya** Faculty of Science, Mahidol University **Investigating the Role of UCHL1 Inhibition in Autophagic Disruption and Cellular Senescence in Microglia.**	
	16:45	17:00	**Selected Abstract #2** **Assoc. Prof. Vikas Kumar Tiwari** Graduate School of Medicine, Tohoku University, Japan; SGMCHRC, MGUG, India **A New Brain Signal Recorded from TMS in Primates: Predicting Motor Paralysis Outcomes**	
	17:00	17:15	**Flash talk #1** **Dr. Tara Sudhadevi** University Road, South Kalamassery, Kalamassery **Inhibition of Sphingosine Kinase 1 by PF543 Improves Airway Hyperreactivity and Neuro-Cognitive Behavior in a Murine Bronchopulmonary Dysplasia Model**	
			Flash talk #2 **Miss Chattraporn Nantawanichakorn** Research Center for Neuroscience, Institute of Molecular Biosciences **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**	
			Flash talk #3 **Miss Yaowapa Trangan** MBNS, Mahidol University **Non-Invasive Assessment of Age-Related Sweat 3-Hydroxyanthranilic Acid: Implications for Early Detection of Cognitive Decline**	
			End of DAY 2	

IBRO-APRC Supported Winter School on Neuroplasticity Across the Lifespan & 28th Annual Conference of Thai Neuroscience Society (TNS28)

Fri 31-10-2025 (DAY 3)				
	Time		Main Room (DOI SUTHEP 1-2)	Workshop
DAY 3	**Plenary Session III: Gene Editing and Cell-Based Approaches to Modulate Neuroplasticity**			
	08:45	09:15	**Prof. Toshihide Yamashita, MD, PhD** Osaka University, Japan **Repulsive guidance molecule regulates glial and immune function under neurological diseases**	09:00-12:00 (DOI NUA) **SKT workshop (English language)** **Prof. Somporn Kantharadussadee Triamchaisri, PhD and Brand company** (30 registered participants)
	09:15	09:45	**Assoc. Prof. Yoshida Hideki, PhD** Kyoto Institute of Technology, Japan **Diversity of molecular mechanisms underlying mRNA localization**	
	09:45	10:15	**Nithi Asavapanumas, MD, PhD** Mahidol University, Thailand **Induced Pluripotent stem cells (iPSCs)-derived brain models in the study of neurodevelopment and neurodegeneration**	
	10:15	10:45	Time break + **Poster evaluation** (PARTY ROOM)	
	Plenary Session IV: Pharmacological and Lifestyle Interventions to Enhance Neuroplasticity			
	10:45	11:15	**Hiranya Pintana, PhD** Chiang Mai University, Thailand **The Impact of 5-Alpha-Reductase Inhibition on Anxiety- and Depression-Like Behaviors in Obese-Insulin Resistant or Early-Senescent Male Rats**	
	11:15	11:45	**Salinee Jantrapirom, PhD** Chiang Mai University, Thailand **UBQLN2-Associated Neurodegeneration: Molecular Mechanisms and Therapeutic Perspectives**	
	11:45	12:15	**Assoc. Prof. Narawut Pakaprot, MD, PhD** Mahidol University, Thailand **In Search for the Molecular Target of Centella Asiatica Extract**	
	12:15	13:30	Lunch	
	Plenary Session V: Neuroplasticity Across Systems: From Social Behavior to Cognitive Aging and Brain-Gut Interactions			
	13:30	14:00	**Distinguished Prof. Ishwar Parhar, PhD** University of Toyama, Japan **Neural Plasticity in Reproduction and Social Behaviors**	
	14:00	14:30	**Lalitta Suriya-Arunroj, Dr. rer. nat.** Chulalongkorn University, Thailand **Cognition, Clocks, and Computation: Cognitive Decline and Epigenetic Aging in Monkeys - Bridging Computational and Translational Neuroscience**	
	14:30	15:00	**Assist. Prof. Amornpun Sereemaspun, MD, PhD** Chulalongkorn University, Thailand	

IBRO-APRC Supported Winter School on Neuroplasticity Across the Lifespan & 28th Annual Conference of Thai Neuroscience Society (TNS28)

Fri 31-10-2025 (DAY 3)				
	Time		Main Room (DOI SUTHEP 1-2)	Workshop
			Emerging Trends of Microbiome-related products used for Gut-Brain Axis: Cutting-Edge Clinical Application	
	15:00	15:30	Time break + **Poster evaluation** (PARTY ROOM)	
	15:30	15:45	**Selected Abstract #3** **Miss Anda Cimpean** Institute of Experimental Medicine, Czech Academy of Sciences, Prague **4-Methylumbelliferone as a Modulator of Neuroinflammation and Neural Plasticity During Aging**	
	15:45	16:00	**Selected Abstract #4** **Mr. Raj Katariya** Division of Neuroscience, Smt. Kishoritai Bhoyar College of Pharmacy, Kamptee, Nagpur **Therapeutic Targeting of Central Agmatine Signaling for Intervention of Huntington's Disease**	
	16:00	16:25	**IBRO communication**	
	16:25	16:35	**Announcement Award winners** (Best poster, Awarding for the selected abstracts and flash talks)	
	16:35	17:00	**TNS communication and CLOSING REMARKS**	
			END of TNS28	

Table of Contents

Welcome Message from the Chairman of Center of Multidisciplinary Technology for Advanced Medicine (CMUTEAM) *1*

Welcome Remarks from the President of Thai Neuroscience Society *2*

Welcome Message from the Co-Chair of Organizing Committee & Chair of Scientific Committee *3*

Welcome Message from the TNS28 Organizing Committee *4*

TNS28 Conference Information and Organizing Committee *5*

List of Reviewers *8*

SPECIAL PROGRAMS *9*

TNS Neuro Quiz 2025 .... *10*

SKT Course & Workshop: The Elderly Memory and Health Healing *23*

IBRO-APRC Supported Associate School 2025 .... *26*

AWARD *35*

2025 TNS Honorary Membership *36*

Professor Dr. Siriporn Chattipakorn 36

Professor Dr Prasop Ratanakorn's Lecture *37*

Rod-shaped microglia regulate neuronal activity in TDP-43 neurodegeneration *37*

Prof. Long-Jun Wu, PhD 37

Professor Dr Roongtam Ladpli's Lecture *39*

From Sleep to Synapse: Neuroplasticity Impairment via Actin Depolymerization in REM Sleep-Deprived Mice *39*

Prof. Ingrid Y Liu, Ph.D. 39

SCIENTIFIC PROGRAM *40*

Aging, Obesity, and Cognitive Impairment: Mechanistic Insights and Non-Pharmacological Approaches *41*

Prof. Siriporn Chattipakorn, DDS, PhD 41

Advanced DNA Sequencing Technologies in Neuroscience and Human Health *43*

Prof. Vorasuk Shotelersuk, MD 43

Neuroscience Takes AI to tango *45*

Prof. Daesoo Kim, PhD 45

Paving the path to therapies for genetic diseases linked to the NALCN channelosome *46*

Arnaud Monteil, PhD 46

Knowing the Unknown: Parkinson's Hidden Face *48*

Assoc. Prof. Wael Mohamed, MD, PhD 48

Mind at Rest, Brain in Motion: Sleep and Synaptic Plasticity *50*

Assoc. Prof. Vorasith Siripornpanich, M.D., Ph.D. 50

Epigenetic–Metabolic Crossroads in Neuroplasticity and Brain Aging *51*

Ranchana Yeewa, PhD 51

Comprehensive AI in Alzheimer's: From Early Diagnosis to Progression Insights Across Multiple Modalities *52*

Ha Thi Thanh Huong, PhD 52

Proteomic Insights into Cognitive Performance: Age-Dependent Sex Differences in Healthy Thai Subjects *54*

Prof. Sutisa Nudmamud-Thanoi, PhD 54

Super High-Resolution Tomography Imaging in Neuroscience *56*

Catleya Rojviriya, PhD 56

The Regulation of Kynurenine Pathway: A Potential Targeting to Prevent Neurodegeneration in Methamphetamine-Induced Toxicity Model *57*

Prof. Banthit Chetsawang, PhD 57

Development of therapeutic strategies to repair neuronal network for the central nervous system diseases *59*

Prof. Toshihide Yamashita, MD, PhD 59

Diversity of molecular mechanisms underlying mRNA localization *61*

Assoc. Prof. Hideki Yoshida, PhD 61

Induced Pluripotent Stem Cells (iPSCs)-Derived Brain Models in the Study of Neurodevelopment and Neurodegeneration *62*

Nithi Asavapanumas, MD, PhD 62

The Impact of 5-Alpha-Reductase Inhibition on Anxiety- and Depression-Like Behaviors in Obese Insulin Resistant or Early Senescent Male Rats *64*

Hiranya Pintana, PhD 64

UBQLN2-Associated Neurodegeneration: Molecular Mechanisms and Therapeutic Perspectives *66*

Salinee Jantrapirom, PhD 66

In Search for the Molecular Target of Centella Asiatica Extract *67*

Assoc. Prof. Narawut Pakaprot, MD, PhD 67

Neural Plasticity in Reproduction and Social Behaviors *69*

Prof. Ishwar Parhar, PhD 69

Cognition, Clocks, and Computation: Cognitive Decline and Epigenetic Aging in Monkeys - Bridging Computational and Translational Neuroscience *71*

Lalitta Suriya-Arunroj, Dr. rer. nat. 71

Emerging Trends of Microbiome-related products used for Gut-Brain Axis: Cutting-Edge Clinical Application *72*

Assist. Prof. Amornpun Sereemaspun, MD, PhD 72

POSTER PRESENTATION *73*

P-001: *74*

LCN2/LCN2R SIGNALING IMPAIRS MITOCHONDRIAL BIOENERGETICS TO PROMOTE NEURONAL DEATH IN HEPATIC ENCEPHALOPATHY *74*

Ching-Yi Tsai[1*] 74

P-002: *75*

4-METHYLUMBELLIFERONE REVERSES AGE-RELATED CHANGES IN NEUROPLASTICITY AND NEUROINFLAMMATION *75*

Anda Cimpean[1,2], Jana Dubisova[1], Pavla Jendelova[1], James W. Fawcett[1,3], Jessica C.F. Kwok[1,4] 75

P-003: *76*

PROBUCOL AND RIFAMPICIN AMELIORATE 3-NITROPROPIONIC ACID-INDUCED HUNTINGTON'S DISEASE BY DOWNREGULATING HMGB1/TLR4/NF-KB SIGNALING PATHWAY *76*

Vishal Kumar[1], Puneet Kumar[1*] 76

P-004: *77*

IMPLICATION OF COLORED RICE TREATMENT ON MICROGLIAL RESPONSE AND CHOLINERGIC DYSFUNCTION UNDER MILD CEREBRAL HYPOPERFUSION IN MICE *77*

Danuyada Wattanaumadechakul[1,2], Chairat Turbpaiboon[1], Panapat Uawithya[3], Nanthanit Pholphana[4], Nuchanart Rangkadilok[4], Supin Chompoopong[1*] 77

P-005: *78*

TREATMENT WITH CAULERPA LENTILLIFERA ATTENUATED ALPHA-SYNUCLEIN ACCUMULATION IN MPP-INDUCED PARKINSON'S-LIKE MODEL *78*

Darunee Rodma[1,5], Chairat Turbpaiboon[2], Rungsarit Sunan[1,5], Ladawan Khowawisetsut[3] Krai Meemon[4], Prasert Sobhon[4], Supin Chompoopong[2], Morakot Sroyraya[4*] 78

P-006: *79*

HALYMENIA DURVILLEI EXTRACT MITIGATES NEUROINFLAMMATORY CYTOKINES IN VASCULAR COGNITIVE IMPAIRMENT MICE *79*

Rungsarit Sunan[1,2], Chairat Turbpaiboon[3], Darunee Rodma[1,2], Ladawan Khowawisetsut[4], Morakot Sroyraya[5], Krai Meemon[5], Prasert Sobhon[5], Supin Chompoopong[3*] 79

P-007: *80*

DEVELOPMENT OF A SELF-EMULSIFYING FORMULATION OF HUPERZIA CARINATA EXTRACT FOR IMPROVING HUPERZINE A SOLUBILITY AND EVALUATING EFFICACY IN AN MPTP-INDUCED PARKINSONIAN MURINE MODEL *80*

Namfa Sermkaew[1,2], Nuttapon Songnaka[1,2], Wuttipong Marsraksa[3], Amit Jaisi[1], Yosita Yongyuen[1], Pannatorn Buntha[1], Nadiah Syafiqah Nor Azman[4], Juntratip Jomrit[1], and Phetcharat Boonruamkaew[1,*] 80

P-008: *82*

ADOLESCENCE AS A WINDOW FOR RECALIBRATION: PUBERTAL STRESS MODULATES BEHAVIORAL AND MICROBIOTA CONSEQUENCES OF EARLY-LIFE STRESS *82*

Muhammad Jahangir[1], Dai Wenjing[1,2], Li Tao[1], Guo Wan-Jun*[1] 82

P-009:.. 84

DECIPHERING THE ROLE OF AURANOFIN-LOADED NANOPARTICLES AGAINST ANIMAL MODEL OF PARKINSON'S DISEASE: VIA GSK-3B/ NRF2/HO-1 SIGNALING PATHWAYS........................ 84

Divya Soni[1], Yogesh Garg[2], Shubham Upadhayay[1], Amit Bhatia[2] and Puneet Kumar[1]* 84

P-010:.. 86

LIRAGLUTIDE IMPROVES COGNITIVE DEFICITS IN TYPE 2 DIABETIC MICE............................. 86

Yoottana Janthakhin[1*], Theerawat Khunsanthia[3], Sirikran Juntapremjit[2], and Sutin Kingtong[3] 86

P-011:.. 87

ERβ-SELECTIVE MODULATION AND NEUROPLASTICITY ENHANCEMENT BY TINOSPORA CORDIFOLIA IN A PERIMENOPAUSAL CONTEXT 87

Anuradha Sharma[1,*], Anuj Babbar[1] 87

P-012:.. 88

PHARMACOLOGICAL EVALUATION OF NEUROMEDIN B IN SEPSIS-INDUCED COGNITIVE DYSFUNCTION IN MOUSE MODEL.. 88

Manisha Suri[1] and Anjana Bali[1*] 88

P-013:.. 90

Withdrawn 90

P-014:.. 91

METASCAPE-GUIDED TRANSCRIPTOMIC ANALYSIS OF 2-BTHF TREATMENT FROM HOLOTHURIA SCABRA IN A 6-OHDA-EXPOSED C. ELEGANS MODEL OF PARKINSON'S DISEASE 91

Sukrit Promtang[1,2*], Pawanrat Chalorak[3], Darunee Rodma[1,2], Rungsarit Sunan[1,2], Apinya Sayinta[1,4], and Krai Meemon[4*] 91

P-015:.. 93

A NEW BRAIN SIGNAL RECORDED FROM TMS IN PRIMATES: PREDICTING MOTOR PARALYSIS OUTCOMES 93

Tiwari VK[1,2], Honda Y[1], Ogawa K[1], Nakamura S[1], Tsutsui KI[1] 93

P-016:.. 94

EVENT-RELATED POTENTIAL BLIND SOURCE SEPARATION USING RECURRENT NEURAL NETWORK: EVALUATION ON SIMULATED DATA 94

Jamie A. O'Reilly[1] 94

P-017:.. 95

PROLONGED P1 LATENCY IN SIMULTANEOUS AUDITORY-SOMATOSENSORY INTEGRATION AND THE RELATIONSHIP WITH SENSORY BEHAVIORAL PERFORMANCE IN CHILDREN WITH AUTISM SPECTRUM DISORDER 95

Nutthida Phianchana[1], Timothy W. Budd[2], Vorasith Siripornpanich[1]* 95

P-018: ... *108*

EXTRACTS DERIVED FROM MULBERRY LEAVES, COMBINED WITH BUTTERFLY PEA FLOWERS SYNERGISTICALLY ENHANCE REM SLEEP AND COGNITIVE PERFORMANCE THROUGH A $GABA_A$ RECEPTOR-DEPENDENT MECHANISM IN RATS ... *108*

Jakkrit Nukitram[1, 2, 3, *], Jintanaporn Wattanathorn[3, 4] ... 108

P-019: ... *110*

IRON OVERLOAD IMPAIRS MOTOR COORDINATION AND TENDS TO AFFECT RECOGNITION MEMORY IN b-THALASSEMIA MICE ... *110*

Benjaporn Kiatpakdee[1], Pornthip Chaichompoo[2], Paranee Yatmark[3], Parin Suwannaprapha[3], Rujapope Sutiwisesak[4], Jim Valodas[5], Saovaros Svasti[1*] ... 110

P-020: ... *111*

IRON OVERLOAD INDUCES BLOOD-BRAIN BARRIER DAMAGE AND HIPPOCAMPAL NEURONAL LOSS CONTRIBUTING TO LEARNING AND MEMORY IMPAIRMENT IN β-THALASSEMIA MICE . *111*

Parinda Jamrus[1], Nuttanan Pholngam[1,2], Benjaporn Kiatpakdee[1], Chinarat Changsangfa[3], Jim Vadolas[4,5], Sukonthar Ngampramuan[6], Pornthip Chaichompoo[7], Saovaros Svasti[1,8,*] 111

P-021: ... *113*

EFFECTS OF COVID-19 INFECTION ON CARDIAC AUTONOMIC FUNCTION AND COGNITIVE FUNCTION ... *113*

Suman Pokhrel[1*], Dilip Thakur[2], Amrendra Jha[2], Ashata Dahal[3] ... 113

P-022: ... *114*

MECHANICAL ALLODYNIA AND DRG PLASTICITY ARE MODULATED BY ANDROGRAPHOLIDE AFTER SCIATIC NERVE INJURY IN RATS ... *114*

Adisorn Ratanayotha[1,2], Kanyaratana Bamrungsuk[3], Napatsorn Jongapirattanakul[1], Bhornluck Paepetch Suato[1], Chairat Turbpaiboon[1], Supin Chompoopong[1] ... 114

P-023: ... *115*

NEW INSIGHTS INTO RECESSIVE ATAXIA: CA8 AND MAG SPLICE DONOR MUTATIONS IN PAKISTANI FAMILIES ... *115*

Rabia Akram[1,2], Tehreem Iman[1], Shahid Mahmood Baig[3], Stephanie Efthymiou[2], Henry Houlden[2], Ghulam Hussain[1*] ... 115

P-024: ... *116*

LIVER AND NEURONAL DEVELOPMENTAL CHANGES IN ZIKV-INFECTED CHICK EMBRYOS .. *116*

Wongsakorn Siripan[1], Pornkanok Nimnoi[1], Chairat Turbpaiboon[2], Boonrat Tassaneetrithep[3], Supin Chompoopong[2*] ... 116

P-025: ... *117*

HIPPOCAMPAL NEURODEGENERATION AND COGNITIVE IMPAIRMENT IN AGING β-THALASSEMIA MICE ... *117*

Nuttanan Pholngam[1,2], Parinda Jamrus[2,3], Kittikun Viwatpinyo[4,5], Benjaporn Kiatpakdee[2], Jim Vadolas[6,7], Pornthip Chaichompoo[3], Sukonthar Ngampramuan[4]*, Saovaros Svasti[2,8]* 117

P-026: ... *119*

REPEATED LEARNING IMPROVE COGNITIVE FUNCTION IN β-THALASSEMIA MICE *119*

Parinda Jamrus[1], Nuttanan Pholngam[1,2], Benjaporn Kiatpakdee[1], Chinarat Changsangfa[3], Suparada Aiemongkot[1], Jim Vadolas[4,5], Sukonthar Ngampramuan[6], Pornthip Chaichompoo[7], Saovaros Svasti[1,8,*] 119

P-027: *121*

NEUROPSYCHIATRIC MANIFESTATIONS IN CADASIL: A 10-YEAR RETROSPECTIVE STUDY AT SIRIRAJ HOSPITAL *121*

Chatchawan Rattanabannakit[1]*, Suprakit Jiraratwattana[2] 121

P-028: *122*

FENDOSAL SELECTIVELY MODULATES CYTOKINE SECRETION IN ASTROCYTES UNDER ISCHEMIA-REPERFUSION CONDITIONS *122*

Sudichhya Tamrakar[1], Kornkanok Promthep[1], Sovaritthon Chansaengsee[1,2], Jiraporn Panmanee[1], Narisorn Kitiyanant[3], Surapon Piboonpocanun[3], Sujira Mukda[1]* 122

P-029: *130*

OXYBERBERINE ATTENUATES VASCULAR DEMENTIA VIA MODULATION OF RHO-KINASE SIGNALING IN A RAT MODEL OF CHRONIC CEREBRAL HYPOPERFUSION *130*

Manoj P. Dandekar[1,*], Harapriya Baral[1], Simranjit Kaur[1], Arbaz Sujat Shaikh[2], K. Venkata Rao[2] 130

P-030: *131*

SCANNING ION-CONDUCTANCE MICROSCOPY FOR STUDYING β-AMYLOID AGGREGATE FORMATION ON LIVING CELL SURFACES *131*

Vasilii S. Kolmogorov[1], Vangene Tay[2], Petr V. Gorelkin[1,3*] 131

P-031: *132*

THERAPEUTIC TARGETING OF CENTRAL AGMATINE SIGNALING FOR INTERVENTION OF HUNTINGTON'S DISEASE *132*

Raj Katariya[1], Brijesh Taksande[2*] 132

P-032: *134*

HUMAN STEM CELL FROM APICAL PAPILLA: THE POTENTIAL ALTERNATIVE CELL SOURCE FOR NEURONAL DIFFERENTIATION *134*

Anupong Thongklam Songsaad[1*], Tatcha Balit[2], Tarinee Chodchavanchai[3], Amarin Thongsuk[3], Nisarat Ruangsawasdi[4], Charoensri Thonabulsombat[3] 134

P-033: *135*

PROTECTIVE EFFECT OF MELATONIN AGAINST OXIDATIVE STRESS IN CEREBRAL ISCHEMIC RATS *135*

Naparat Promyoo[1], Jirakhamon Sengking[2], Mathurada Saephu[1], Chainarong Tocharus[2], Jiraporn Tocharus[1*] 135

P-034: *136*

PROTECTIVE EFFECT OF APOCYNIN ON COGNITIVE IMPAIRMENT IN CHRONIC CEREBRAL HYPOPERFUSION-INDUCED RATS *136*

Mathurada Saephu[1], Phakkawat Thangwong[2], Jirakhamon Sengking[3], Naparat Promyoo[1], Chainarong Tocharus[3], Jiraporn Tocharus[1]* 136

P-035: *143*

RED ONION OLIGOSACCHARIDE SUPPLEMENTATION COUNTERACTS HIGH-FAT DIET-INDUCED COGNITIVE AND METABOLIC ABNORMALITIES IN RATS *143*

Juefang Gan[1,2], Jittiporn Wongpun[3], Jirakhamon Sengking[1], Sivanan Sivasinprasasn[1], Apinun Kanpiengjai[4], Jiraporn Tocharus[5], Chainarong Tocharus[1]* 143

P-036: *145*

TCU412 EXTRACT EXERTED A PROTECTIVE EFFECT AGAINST CORTICOSTERONE-INDUCED CYTOTOXICITY IN HT22 CELLS, POTENTIALLY THROUGH MODULATION OF THE FERROPTOSIS PATHWAYS *145*

Nhan Do Thi Thuy[1], Peeraporn Varinthra[1], Ingrid Y. Liu[1*] 145

P-037: *146*

ESTABLISHMENT OF A DROSOPHILA MODEL OF CHARCOT-MARIE-TOOTH TYPE 4B3 .... *146*

Titaree Yamsri and Hideki Yoshida[1,*] 146

P-038: *147*

NEUROPROTECTIVE EFFECTS OF TCU 451 AGAINST AMYLOID BETA-INDUCED APOPTOSIS AND OXIDATIVE STRESS IN HT22 CELLS *147*

Ismat Noor Paromi [1], Peeraporn Varinthra [1], Ingrid Y. Liu [1]* 147

P-039: *148*

PRUNUS DOMESTICA L. EXTRACT ATTENUATES D-GALACTOSE–INDUCED IMPAIRMENTS IN HIPPOCAMPAL NEUROGENESIS *148*

Puncharatsm Pannin[1], Nataya Sritawan[1], Anusara Aranarochana[1], Apiwat Sirichoat[1], Jariya U Welbat[1*] 148

P-040: *149*

PROTEOMIC INSIGHTS INTO HIPPOCAMPAL NEUROINFLAMMATION IN CUMS-INDUCED ANXIETY: THERAPEUTIC EFFECTS OF COMBINED CANNABIDIOL AND BRAHMI *149*

Plaiyfah Janthueng[1,7], Wanfrutkon Waehama[1,7], Sawanya Charoenlappanit[2], Sittiruk Roytrakul[2], Prapapan Temkitthawon[3,4], Paweena Kaewman[5,7], Jureepon Roboon[5,7], Samur Thanoi[6], and Sutisa Nudmamud-Thanoi[2,7*] 149

P-041: *151*

NEURONAL PNN DEFICIENCY LEADS TO BRAIN STRUCTURAL ALTERATIONS AND BEHAVIORAL ABNORMALITIES IN MICE *151*

Shin-Meng Deng[1], Sujira Mukda[2], Steve Leu[3*] 151

P-042: *152*

EFFECT OF CAFFEINE ON AMYLOID PRECURSOR PROTEIN AND ALZHEIMER'S MARKERS IN AMYLOID BETA-TREATED SH-SY5Y NEUROBLASTOMA CELLS *152*

Chonnicha Subkod[1], Kornkanok Promthep[1], Theptharin Charuraksa[2], Jiraporn Panmanee[1] and Narisorn Kitiyanant[3]* 152

P-043: *153*

TCU412 AMELIORATES AMYLOID BETA-INDUCED TOXICITY IN HT22 CELLS AND MEMORY IMPAIRMENTS IN 3XTG-AD MICE *153*

Mubashir Raza[1], Ingrid Y. Liu[1]* 153

P-044: *154*

NEURONAL ACTIVATION IN CORTICOSTRIATAL CIRCUITS FOLLOWING REPEATED ANODAL-TRANSCRANIAL DIRECT CURRENT STIMULATION IN MICE *154*

Siripaporn Kesyou[1,2], Bahrie Ramadan[2], Stephanie Dumontoy[2], Sutisa Nudmamud-Thanoi[3,4*], Vincent Van Waes[2*] 154

P-045: *156*

IMP-1710: REPURPOSING UBIQUITIN C-TERMINAL HYDROLASE L1 (UCHL1) INHIBITION INTO A SENESCENCE-DRIVING STRATEGY *156*

Napasakorn Verasaksuriya[1], Nattawadee Panyain[2], Kowit Hengphasatporn[3], Yasuteru Shigeta[3], Krisada Rungsang[1], Monruedee Sukprasansap[4], Tewin Tencomnao[5], Sunhapas Soodvilai[6,7], Benjamin Ongnok[6,8,*] 156

P-046: *158*

COMPUTATIONAL DRUG REPURPOSING YIELDS EFFECTIVE GSK3β INHIBITORS IN HT-22 HIPPOCAMPAL CELLS *158*

Hanwen Mi[1], Utid Suriya[2], Monruedee Sukprasansap[3], Tewin Tencomnao[4], Sunhapas Soodvilai[1,5], Benjamin Ongnok[1,6,*] 158

P-047: *160*

CBD-ENRICHED HEMP EXTRACT AND CBD ISOLATE ATTENUATE STRESS-INDUCED ANXIETY VIA ENDOCANNABINOID SYSTEM MODULATION IN RATS *160*

Wanfrutkon Waehama[1,2], Jureepon Roboon[2,3], Prapapan Temkitthawon[4,5], Samur Thanoi[6], Sawanya Charoenlappanit[7], Sittiruk Roytrakul[7], Sutisa Nudmamud-Thanoi[2,3*] 160

P-048: *161*

THE ALTERATION OF PROTEIN PROFILES IN CURCUMIN SOLID DISPERSION (CSD) AGAINST A DEXAMETHASONE-INDUCED DEPRESSION MODEL *161*

Ponthip Cheenkhwan[1,6], Suchiwa Pan-on[2], Waree Tiyaboonchai[3,6], Samur Thanoi[4], Sawanya Charoenlappanit[5], Sittiruk Roytraku[5], Jureepon Roboon[1,6], and Sutisa Nudmamud-Thanoi[1,6]* 161

P-049: *163*

D-GALACTOSE INDUCES SENESCENCE AND FUNCTIONAL IMPAIRMENT IN HUMAN MICROGLIAL CELL LINE THROUGH RAGE SIGNALING *163*

Poramet Klangmongkon[1], Patit Choksawangkarn[2], Monruedee Sukprasansap[3], Tewin Tencomnao[4], Sunhapas Soodvilai[1,5], Alisa Damnernsawad[6], Benjamin Ongnok[1,7,*] 163

P-050: *165*

ADMINISTRATION OF HIGH-DOSE TACROLIMUS IMPAIRED HIPPOCAMPAL SYNAPTIC PLASTICITY AND HIPPOCAMPAL-DEPENDENT MEMORY IN MALE WISTAR RATS *165*

Houzhi Cheng [1,2,3,4], Patcharapong Pantiya[2,3], Hiranya Pintana[2,3], Titikorn Chunchai[2,3], Nipon Chattipakorn[2,3,4], Siriporn C Chattipakorn[2,3,5 *] 165

P-051: *167*

LYSOSOMAL STRESS FLUORESCENCE REPORTER (LSFR): A NEW METHOD FOR DETECTION AND QUANTITATION OF LYSOSOMAL STRESS RESPONSE IN NEURONS *167*

Narinporn Tresteankij, Tai Chaiamarit, Tanida Chokpanuwat, and Benjamin Ongnok* 167

P-052: *168*

EVALUATION OF CUE-BASED PROTOCOL IMPLEMENTATIONS AND SEMANTIC FEEDBACK IN MOTOR IMAGERY-BASED BRAIN-COMPUTER INTERFACE EXPERIMENTS *168*

H.T Nguyen[1], Q.C Pham[3], H.K.L Nguyen[2^], T.D.M Nguyen[4], and T.T.H Ha[1*] 168

P-053: *170*

A SMALL-MOLECULE NATURAL PRODUCT ATTENUATES Aβ42-INDUCED NEUROINFLAMMATION VIA IDO-1 INHIBITION IN SH-SY5Y CELLS *170*

Munnutchaya Tarmtiranont [1], Kornkanok Promthep[1], Sujira Mukda, Banthit Chetsawang[1], Jiraporn Panmanee[1*] 170

P-054: *176*

INULIN EXERTS NEUROPROTECTIVE EFFECTS *176*

IN D-GALACTOSE-INDUCED AGED RATS *176*

Yanmei Huang[1,2,3], Chanisa Thonusin[2,3,4], Nipon Chattipakorn[2,3,4,5], Siriporn C Chattipakorn[2,3,6*] 176

P-055: *177*

REGULATORY MECHANISMS OF MEMBRANE TRAFFICKING TRANSCRIPT LOCALIZATION AT THE DROSOPHILA NEUROMUSCULAR SYNAPSE *177*

Tai Chaiamarit[1,3*], Danail Stoychev[2,3], Francesca Robertson[3,4], Jeffrey Y Lee[2,3], Ilan Davis[2,3] 177

P-056: *178*

AN IMPACT OF SLEEP QUALITY ON RISKY-DECISION MAKING BEHAVIORS AND ITS NEURAL ACTIVITY IN THAI HEALTHY ADOLESCENTS AND ADULTS: AN EVENT-RELATED POTENTIAL STUDY *178*

Mananya Potima[1*], Vorasith Siripornpanich[1] 178

P-057: *179*

A PILOT STUDY OF INATTENTIONAL BLINDNESS: THE UNDERSTANDING OF NEURAL ACTIVITY AND BEHAVIORS OF THE ATTENTIONAL NETWORK IN HEALTHY THAI FEMALE ADULTS *179*

Kurisara Boonyakitmaitree[1*], Mananya Potima[1], Prao Pongpipat[1], Patcharapha Poonthawatsanti[1], Samuel T. Ward[2], and Vorasith Siripornpanich[1] 179

P-058: *179*

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 *179*

Chattraporn Nantawanichakorn[1], Hathaitip Sritanaudomchai[2], Kovit Pattanapanyasat[3], Dungdol Narasirilrk[4], Jinnipa Matchwong[4], Sukonthar Ngampramuan[1*] 180

P-059: *182*

ELECTROPHYSIOLOGICAL MARKERS OF INHIBITORY CONTROL USING AN EMOTIONAL GO/NO-GO PARADIGM IN HEALTHY ADULTS: A PILOT FOR FUTURE MDD STUDIES *182*

Fahsai Kaewpikul[1], Jiraporn Panmanee[1], Sukonthar Ngampramuan[1], Vorasith Siripornpanich[1*] 182

P-060: *183*

INHIBITION OF SPHINGOSINE KINASE 1 BY PF543 IMPROVES AIRWAY HYPERREACTIVITY AND NEUROCOGNITIVE OUTCOMES IN A MURINE MODEL OF BRONCHOPULMONARY DYSPLASIA *183*

Tara Sudhadevi[1], Prathima Basa[2], Ian Adams[2] and Anantha Harijith[3] 183

P-061: *184*

TOCOTRIENOL-RICH FRACTION FROM PALM OIL ATTENUATES MOTOR, LEARNING AND MEMORY DEFICITS IN A ZEBRAFISH MODEL OF PARKINSON'S DISEASE *184*

Thenmoly Damodaran[1*] and Dhanveer Ahamed Bathurutheen [2] 184

P-062: *185*

ESTABLISHING IN VITRO MODELS OF ASTROCYTE AGING FOR TESTING ANTI-AGING EFFECTS OF FRACTIONATED CELASTRUS PANICULATUS SEED EXTRACTS *185*

Phossawee Kongkaew[1*], Siriporn Chamniansawat[1] 185

P-063: *186*

MUSCLE-DERIVED APELIN MEDIATES EXERCISE-INDUCED ANTIDEPRESSANT EFFECTS AND HIPPOCAMPAL NEUROPLASTICITY *186*

Jiasui Yu[1,2], Suk-yu Yau[1,2*] 186

P-064: *187*

HUMAN MICROGLIAL RESPONSES TO RESVERATROL EXPOSURE: INSIGHTS FROM MORPHOLOGICAL IMAGING AND PROTEOMIC APPROACHES *187*

Ekkaphot Khongkla[1,*], Kornkanok Promthep[1], Pipob Suwanchaikasem[2], Fikri Aminullah[3], Rafi Aliefiyanto[3], Dana Mohammad Khader AlNajmi[4], Farouk Raed[4], Maral Daneshpazhouh[4], Banthit Chetsawang[1] 187

P-065: *188*

GENETIC ASSOCIATION OF THE SLC6A4 5-HTTLPR-STIN2 HAPLOTYPE WITH SUICIDAL BEHAVIOR: A SYSTEMATIC REVIEW AND META-ANALYSIS *188*

Sulaifan Waehama[1], Nathorn Chaiyakunapruk[2], Ratree Sawangjit[3], Benjamard Thaweethee-Sukjai[4], Samur Thanoi[5], Jureepon Roboon[7,8], Gavin P. Reynolds[6,7], Sutisa Nudmamud-Thanoi[7,8*] 188

P-066: *190*

NON-INVASIVE ASSESSMENT OF AGE-RELATED SWEAT 3-HYDROXYANTHRANILIC ACID LEVELS: IMPLICATIONS FOR EARLY DETECTION OF COGNITIVE DECLINE *190*

Yaowapa Trangan[1,2], Kanlaya Prapainop Katewongsa[3], Preeyanuch Manohong[3], Rungrat Palakai[2,4], Sirinapa Mysook[5,6], Banthit Chetsawang[1], Piyawat Katewongsa[2*] 190

P-067: *191*

THE EFFECT OF FINE PARTICULATE MATTER 2.5 (PM2.5) ON NEURITE DEVELOPMENT IN N2A NEUROBLASTOMA CELLS *191*

Napol Kaewkascholkul[1*], Kunlaya Somboonwiwat[1], Fareda Sapphot[2], Kanokporn Wantem[2] 191

P-068: *192*

A HUMAN STEM CELL-DERIVED BIOENGINEERED PLATFORM WITH DEFINED CIRCUIT ARCHITECTURE FOR SYNAPTIC CONNECTIVITY ANALYSIS *192*

Pacharaporn Suklai[1,2,3*], Andrea Serio[1,2] 192

P-069: *194*

EXPLORING THE LINK BETWEEN NIGHTMARES, SLEEP PARAMETERS, AND PREFRONTAL CORTEX DYSFUNCTION IN MILD COGNITIVE IMPAIRMENT: A PILOT FEASIBILITY STUDY *194*

Kotchapit Maksri[1], Yaowapa Trangan[1], Banthit Chetsawang[1], Siraprapa Boobphahom[1], Vorasith Siripornpanich[1*] 194

P-070: *195*

COMPUTATIONAL AND EXPERIMENTAL STRATEGIES FOR DEVELOPING NOVEL BUTYRYLCHOLINESTERASE INHIBITORS FOR TREATMENT OF ALZHEIMER'S DISEASE *195*

Ankit Ganeshpurkar [1, 2*], Sushil Kumar Singh [2*] 195

P-071: *197*

ASARININ AS A POTENTIAL DUAL OREXIN RECEPTOR ANTAGONIST FOR THE TREATMENT OF INSOMNIA: THE STUDY OF OREXIN-2 RECEPTOR (ORX2R) DOWNSTREAM SIGNALING CASCADES IN ORX2R-OVEREXPRESSING CELL *197*

Fahsai Thaion[1], Jiraporn Panmanee[1], Narisorn Kitiyanant[2], Kornkanok Promthep[1], Chonnicha Subkod[1], Banthit Chetsawang[1*] 197

P-072: *205*

KV1.3 CHANNEL INHIBITION PRESERVES NEURONAL INTEGRITY AND PROMOTES FUNCTIONAL RECOVERY AFTER INTRACEREBRAL HEMORRHAGE *205*

Wachirapong Saleeon[1], Namphung Thongta[1], Anchalee Vattarakorn[1], Yingrak boondam[2], Wattana Watanapa[1], Sompol Tapechum[1], Narawut Pakaprot[1*] 205

P-073: *206*

EFFECT OF HOME-BASED, LOW-INTENSITY EXERGAMING ON COGNITIVE FUNCTION IN OLDER ADULTS WITH MILD COGNITIVE IMPAIRMENT *206*

Sirintip Kumfu[1], Sirinun Boripantakul[1], Piangkwan Sa-nguanmoo[1], Siriporn C Chattipakorn[2,3], Somporn Sungkarat[1]* 206

P-074: *207*

ENGINEERED EXOSOMES FROM HUMAN EXFOLIATED DECIDUOUS TEETH (SHED) EXPRESSING RVG PEPTIDE ATTENUATE NEURONAL INJURY AND PROMOTE FUNCTIONAL RECOVERY AFTER ISCHEMIC STROKE IN RATS *207*

Sukonthar Ngampramuan[1]*, Anyapat Atipimonpat[2], Hathaitip Sritanaudomchai[3], Pakpoom Subsoontorn[2], Waracharee Srifa[2], Pornprom Surakul[4], Chattraporn Nantawanichakorn[1], Kovit Pattanapanyasat[5]........ 207

P-075:........ *208*

TIME-DEPENDENT ANALYSIS OF PAIN-ASSOCIATED PROTEIN LOCALIZATION IN THE TIDEMARK OF CFA-INDUCED TEMPOROMANDIBULAR OSTEOARTHRITIS IN MICE........ *208*

Lattanawan Inchaisri[1], Aree Wanasuntronwong[2*], and Onrawee Khongsombat[1*]........ 208

P-076:........ *209*

FENDOSAL ATTENUATES OXYGEN-GLUCOSE DEPRIVATION/REPERFUSION-INDUCED UPREGULATION OF RHEB AND MTOR IN HIPPOCAMPAL NEURONAL CELLS........ *209*

Kornkanok Promthep[1], Chonnicha Subkod[1], Theptharin Charuraksa[2], Pathidta Chodewaratham[1], Patlada Tangweerasing[1], Tanya Prasertporn[1], Jiraporn Panmanee[1], Narisorn Kitiyanant[3], Surapon Piboonpocanun[3], Sujira Mukda[1]*........ 209

TNS28 AWARDS........ *218*

ACKNOWLEDGEMENTS........ *220*

AUTHOR INDEX........ *222*

INDEX........ *226*

Welcome Message from the Chairman of Center of Multidisciplinary Technology for Advanced Medicine (CMUTEAM)

Professor Dumnoensun Pruksakorn, MD, PhD

Vice Dean for Research, Faculty of Medicine, Chiang Mai University

It is my great pleasure to welcome all participants to the 28th Annual Thai Neuroscience Society Conference (TNS28), held in the vibrant city of Chiang Mai. On behalf of the organizing committee, I would like to express our deep appreciation to the Thai Neuroscience Society (TNS) for their support in bringing this important international neuroscience event to Chiang Mai, as well as to all members of the organizing committee, particularly CMUTEAM and the Department of Pharmacology, Faculty of Medicine, Chiang Mai University, for their dedication and hard work in making this conference possible.

This year's conference, themed **"Neuroplasticity Across the Lifespan: Advancing Neuroplasticity Research through Cutting-Edge Methodologies,"** provides an important opportunity for researchers, clinicians, and students from many fields to come together, share their knowledge, and explore how new discoveries can lead to better understanding and treatment of brain disorders.

TNS28 also emphasizes the importance of fostering international collaboration, bringing together participants from all over the world. By creating a platform for dialogue, knowledge exchange, and networking, the conference strengthens partnerships and advances global neuroscience research.

Beyond scientific discussion, TNS28 offers educational and outreach activities, including the TNS Neuro Quiz 2025 and the SKT Workshop on Elderly Memory and Health Healing, reflecting the conference's commitment to sharing neuroscience knowledge with the wider community.

On behalf of the organizing committee, I wish you an inspiring and productive meeting filled with meaningful discussions, collaborations, and shared vision toward advancing brain health and neuroscience in Thailand and beyond.

Welcome to TNS28, and welcome to Chiang Mai.

Welcome Remarks from the President of Thai Neuroscience Society

Professor Supin Chompoopong, PhD

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

Distinguished Speakers, Attendees, and Esteemed Guests of the TNS28 Conference

On behalf of the Thai Neuroscience Society (TNS) and the Conference Committee, it's my great pleasure to welcome you to the 28th Thai Neuroscience Society Conference (TNS28) that is co-hosted by Faculty of Medicine, Chiang Mai University. TNS28 is organized as the international conference in conjunction with the IBRO-APRC supported Associate school by Center of Multidisciplinary Technology for Advanced Medicine (CMUTEAM) during October 27-November 2,2025.

TNS28 at the **Kantary hills hotel, October 29 - 31, 2025**, unveils its compelling theme: **Neuroplasticity Across the Lifespan: Advancing Neuroplasticity research through cutting-edge methodologies.**

Under this theme, TNS28 designed to bring together neuroscience professionals for collaboration and idea exchange. It highlights the goal of fostering innovation and partnership among leading experts, researchers, and clinicians in the field of neuroscience, genetics, neurophysiology to explore these transformative discoveries. By integrating perspectives from small animal models, primate studies, and human clinical research. This conference will provide a comprehensive platform to discuss the future of neuroplasticity and its implications for both basic science and novel neurotherapeutics.

In addition, TNS28 would like to share the basis of neuroscience knowledge to the interested groups with the special activities: 1) TNS Neuro Quiz 2025, which is the second year of competition (high school level) engages young minds in neuroscience, and 2) SKT Workshop: The Elderly Memory and Health Healing.

Finally, I extend my warmest wishes to all attendees for a fruitful and inspiring conference experience. Thank you for joining us, we look forward to your participation in making TNS28 a resounding success.

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

Luca Lo Piccolo, PhD

Functional Genomics Unit at CMUTEAM, Faculty of Medicine, Chiang Mai University

It is a great honor to welcome all participants to the 28th Annual Thai Neuroscience Society Conference (TNS28), held in the beautiful city of Chiang Mai. On behalf of the organizing and scientific committees, I would like to express our sincere appreciation to the Faculty of Medicine, Chiang Mai University, and the Thai Neuroscience Society for hosting this important international gathering and for welcoming TNS to Northern Thailand.

This year, TNS28 explores the theme “Neuroplasticity Across the Lifespan: Advancing Neuroplasticity Research through Cutting-Edge Methodologies”, which addresses one of the most dynamic and relevant areas in neuroscience. Understanding how the brain adapts and reorganizes itself not only advances fundamental science but also informs education and clinical approaches, offering opportunities to improve brain health across all ages.

A central spirit of TNS28 is the fostering of a supportive community for early-career researchers. Engaging and encouraging these emerging scientists is important because they will play a key role in addressing the evolving challenges in neuroscience and brain health. By providing opportunities to share their work, learn from experienced researchers, and build professional networks, TNS28 helps equip the next generation with the skills, knowledge, and connections needed to develop their research careers. Their fresh perspectives and enthusiasm contribute to a dynamic scientific environment, supporting the continued growth and sustainability of neuroscience in our region and beyond.

The international character of TNS28 is one of its greatest strengths. While the conference draws participants from across the Asia-Pacific region, it also welcomes attendees and contributors from overseas, creating a platform for exchange of knowledge, collaboration, and mentorship.

We are especially grateful to the reviewers, who dedicated their time and expertise to carefully evaluate all submissions and ensure the scientific rigor and quality of this conference. Special acknowledgment is also extended to the Evaluation Committee, responsible for selecting the four outstanding abstracts for oral presentations and three abstracts for flash talks, highlighting the most innovative and impactful research among our participants.

We hope that TNS28 will be not only a forum for sharing scientific discoveries but also an opportunity to build professional relationships, learn from colleagues, and strengthen the regional and global neuroscience community.

Welcome to Chiang Mai, and welcome to TNS28.

Welcome Message from the TNS28 Organizing Committee

Salinee Jantrapirom, PhD

Coordinator of TNS28
Faculty of Medicine, Chiang Mai University

It is with great pleasure that I extend a warm welcome to all participants of the 28th Annual Thai Neuroscience Society Conference (TNS28), held this year in Chiang Mai. Organizing this event has been a collective endeavor that reflects the strong spirit of collaboration and unity within our national neuroscience community.

Bringing TNS to Chiang Mai represents more than a change of venue, it is a celebration of the teamwork, dedication, and shared vision that connect neuroscientists across Thailand. From the initial planning stages to the final program, this conference has been made possible through the contributions of many individuals and institutions who share a commitment to advancing neuroscience research and education in our country. In particular, we would like to acknowledge the Faculty of Medicine, Chiang Mai University (MedCMU), the Center of Multidisciplinary Technology for Advanced Medicine (CMUTEAM), and the Department of Pharmacology, Faculty of Medicine, Chiang Mai University, whose generous support, facilities, and guidance have been instrumental in making TNS28 a success.

We would also like to express our heartfelt gratitude to our sponsors and partners for their generous support, to our reviewers for their thoughtful evaluation of abstracts, and to the many volunteers who have worked tirelessly to ensure the success of this conference. Their collective effort exemplifies the collaborative ethos that defines our society.

TNS28 also serves as an important platform for strengthening national neuroscience networking. It brings together researchers, clinicians, and students from diverse disciplines, fostering dialogue, collaboration, and new partnerships that will continue to enrich our scientific community long after the conference concludes.

On behalf of the organizing committee, I wish all participants an inspiring and rewarding experience at TNS28. May this gathering spark new ideas, friendships, and collaborations that drive Thai neuroscience toward an even brighter future.

Welcome to Chiang Mai, and welcome to TNS28.

TNS28 Conference Information and Organizing Committee

TNS28 Conference Information

The Thai Neuroscience Society (TNS) and Faculty of Medicine, Chiang Mai University are excited to announce that **"The 28th Annual Thai Neuroscience Society Conference**, TNS28" in 2025 will be organized as the international conference in conjunction with the IBRO – APRC supported Associate school, 2025 in Chiang Mai (October 27-November 2,2025).

TNS28 will be hosted at the **Kantary hills hotel, Chiang Mai, Thailand, from October 29 - 31, 2025**, the TNS28 unveils its compelling theme: **Neuroplasticity Across the Lifespan: Advancing Neuroplasticity research through cutting-edge methodologies.**

TNS28 warmly welcomes leading experts, researchers, and professionals in the field of neuroscience for a collaborative gathering where ideas flourish and collaboration thrives. In addition, TNS28 would like to share the basis of neuroscience knowledge to the interested groups with the special activities: 1) TNS Neuro Quiz 2025, which is the second year of competition (high school level), and 2) SKT Workshop: The Elderly Memory and Health Healing.

Neuroplasticity is a defining feature of the brain, enabling learning, memory, recovery from injury, and adaptation throughout life. As neuroscience progresses, our understanding of how plasticity shapes brain function – from development to aging and disease – continues to evolve. In 2025, we find ourselves at the intersection of groundbreaking discovery and therapeutic innovation, where cutting-edge research is unlocking new possibilities for brain health and the treatment of neurodegenerative diseases.

A landmark achievement in 2024 – the completion of the *Drosophila* brain connectome – has provided an unprecedented view of neural circuit organization, revealing how structural and functional plasticity are interconnected. This milestone fuels our excitement for the eventual mapping of the human brain connectome. At the same time, the rise of omics technologies, such as single-cell transcriptomics and epigenomics, is revolutionizing our understanding of the molecular mechanisms driving neuroplasticity across the lifespan.

Breakthroughs in gene and cell therapies are also reshaping our approach to previously incurable neurodegenerative diseases, demonstrating that the brain's adaptive potential may extend beyond what was once imagined. Advances in neuropharmacology, glial biology, and regenerative medicine are further enhancing our ability to modulate and restore brain function.

The TNS28 Conference on Neuroplasticity Across the Lifespan will bring together leading experts in neuroscience, genetics, neurophysiology, and clinical research to explore these transformative discoveries. By integrating perspectives from small animal models, primate studies, and human clinical research, this conference will provide a comprehensive platform to discuss the future of neuroplasticity and its implications for both basic science and novel neurotherapeutics.

Join us at TNS28 to expand your network in neuroscience and stay updated on the latest discoveries in the field. Together, let's contribute to advancing neurobiology and shaping the future of brain health!

Overview

Date	**Oct 29 (Wed) – Oct 31 (Fri), 2025**
Venue	**Kantary hills hotel, Chiang Mai, Thailand**
Theme	"**Neuroplasticity Across the Lifespan: Advancing Neuroplasticity research through cutting-edge methodologies**"
Official Language	**English**
Organized by	**Thai Neuroscience Society** and **Faculty of Medicine, Chiang Mai University**
Hosted by	**Faculty of Medicine, Chiang Mai University**
Supported by	**International Brain Research Organization - Asia Pacific Regional Committee (IBRO-APRC)** **Faculty of Medicine, Chiang Mai University** **Thai Neuroscience Society (TNS)**

TNS28 Organizing Committee

Dumnoenson Pruksakorn	Chair of Organizing committee
Supin Chompoopong	Co-Chair of Organizing committee
Luca Lo Piccolo	Vice-Chair of Organizing committee & Chair of Scientific committee
Akkradate Siriphorn	Co-Chair of Scientific committee
Chairat Turbpiboon	Vice-Chair of Scientific committee
Chailerd Pichitpornchai	Chair of Special activity

Committee from Chiang Mai University

Supanimit Teekachunhatein	Advisory committee
Dumnoenson Pruksakorn	Chair
Luca Lo Piccolo	Co-Chair
Salinee Jantrapirom	Ranchana Yeewa
Wasinee Wongkummool	Phatcharida Jantaree
Natrujee Wiwattanadittakul	Ruedeemars Yubolphan

Punate Weerateerangkul	Rungsinee Phongpradist
Jannapas Tharavichikun	Pathacha Suksakit
Yuparad Kongnak	Thunpitcha Meesawat
Natsinee U-on	Siwat Poompoung

Committee from Thai Neuroscience Society (TNS)

TNS Advisory committee
Pavich Thongroj
Kanokwan Tiloksakulchai
Piyarat Govitrapong
Sukumal Chongthammakun

TNS committee (2025-2027)

Supin Chompoopong	President
Chailerd Pichitpornchai	Vice-President & Special Activity Committee
Sutisa Thanoi	Vice-President
Akkradate Siriporn	Scientific committee
Chairat Turbpiboon	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
Amornpan Sereemaspun	Deputy Special Activity Committee
Prateep Amornruttanapun	Deputy Special Activity Committee
Kulathida Chaithirayanon	Treasurer
Manussabhorn Phatsara	Committee & Public Relations

List of Reviewers

International Reviewers

Eve Mon Oo
Paul Klosen
Shi-Qing Cai
Yuki Fujita
Zaw Myo Hein

Local Reviewers

Adisorn Ratanayotha
Akkradate Siriphorn
Anchalee Prasansuklab
Banthit Chetsawang
Chairat Turbpaiboon
James Michael Brimson
Krai Meemon
Kulathida Chaithirayanon
Luca Lo Piccolo
Manussabhorn Phatsara
Narawut Pakaprot
Nithi Asavapanumas
Onrawee Khongsombat
Prateep Amonruttanapun
Rujapope Sutiwisesak
Salinee Jantrapirom
Sujira Mukda
Sukumal Chongthammakun
Supin Chompoopong
Sutisa Thanoi
Thawornchai Limjindaporn
Utcharaporn Kamsrijai
Vipavadee Chaisuksunt

SPECIAL PROGRAMS

TNS Neuro Quiz 2025

Overview:

The Thai Neuroscience Society (TNS) Neuro Quiz 2025 is an intellectually stimulating competition aimed at inspiring bright young minds – our regional country's future leaders – by introducing them to the field of neuroscience. This event promotes active learning through mechanisms that require both active recall and the application of knowledge, thereby enhancing comprehension and critical thinking skills.

The successful completion of quiz rounds fosters a sense of achievement, increasing participants' confidence and motivation. Moreover, the inclusion of scenario-based questions encourages the practical application of theoretical knowledge, allowing students to translate neuroscience concepts into real-world contexts.

The quiz competition will consist of multiple rounds: Elimination Round I (online), Semi-Final Round or Elimination Round II (online), and the International Finals (onsite). Each subsequent round will increase in difficulty and complexity, providing a progressively challenging experience for participants.

The competition will culminate in the international finals, preceded by a series of exciting elimination rounds. The top 10 finalist teams will be awarded a sponsored accommodation to attend the international finals, which will be held onsite in TNS28 conference at the Kantary Hills Hotel, Chiang Mai, Thailand, on October 29, 2025.

Timeline:

Description	**Date**
First announcement details	1-15 June, 2025
Registration period	16 June - 11 July, 2025
Announcement of eligible teams and program details	14 July, 2025
Elimination Round I – Online MCQ test via AutoProctor	**20 July, 2025**(1.00 pm - 3.00 pm, GMT+7, Thailand Time)
Announcement of the Top 30 teams advancing to the Semi-Finals	27 July, 2025

Description	Date
Elimination Round II (Semi-Finals) – Online quiz via Kahoot	**10 August, 2025**(1.00 pm - 3.00 pm, GMT+7, Thailand Time)
Announcement of the Top 10 finalist teams advancing to the International Finals	17 August, 2025
International Finals (onsite) in TNS28 conference at Kantary Hills Hotel, Chiang Mai, Thailand	**29 October, 2025**(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 three primary student members.
5. Each school can register a maximum of two teams, designated as Team A and Team B.

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 three 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/7QYUjGTbdUxMnYHcA.
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.

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 three sequential rounds:

1. **Elimination Round I** – Conducted online through **Zoom meeting** and **AutoProctor** using multiple-choice questions (MCQs) to identify the Top 30 teams according to their highest scores.

2. **Elimination Round II** – Conducted online via **Zoom meeting** and **Kahoot** to determine the Top 10 finalist teams according to their highest scores.

3. **International Finals** – Conducted onsite at Kantary Hills Hotel, Chiang Mai, Thailand, to select the winning team.

Elimination Rounds (online):

Rules

1. All registered participants are required to join the Zoom meeting using their unique team ID followed by their full registered name. The unique team ID will be announced on July 14, 2025.
2. Participants must join within the scheduled time. Late arrivals (over 5 minutes) will be disqualified.
3. **AutoProctor** 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. Participants must refrain from opening or switching to other tabs or windows during the quiz and must avoid any form of communication with others.
6. If the **AI system** detects any dishonest behavior, such as switching tabs, altering conversations, or misrepresenting participants in pictures, the team will forfeit their scores.
7. No external assistance is allowed. No other person may participate or help in any forms.
8. 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. All participants are required to sign in to **AutoProctor** using their **registered email** and complete the demo test on the platform (demo test link) before the Elimination Round I.
2. 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.
3. Participants will be evaluated through multiple-choice questions (MCQs) in Elimination Round I (conducted online via **AutoProctor**) to identify the Top 3 0 teams.
4. Top 30 teams will be evaluated through scenario-based problem-solving questions in Elimination Round II (administered through Kahoot) to determine the Top 10 finalist teams.

5. Each team member must complete the **individual examination independently.**

6. All participants are required to use two devices: a **PC or Mac computer** to complete the quiz (with microphone and camera on) and a **second device**, such as a mobile phone, for real-time proctoring via Zoom, with the camera on throughout the session.

7. 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.

8. Please verify that your internet speed is sufficient.

9. The video conference will be held via Zoom meeting; ensure that the Zoom application is installed on your device beforehand.

10. Participants must access the online quiz via **AutoProctor** and ensure they are signed in with their **registered email** prior to logging in.

11. The TNS Neuro Quiz organizers will provide the quiz link during the Zoom session on July 20, 2025 (Elimination Round I)

International Final Round (onsite):

Rules

1. The team's composition must only include an accompanying teacher (coach) and 1-3 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. Participants will be evaluated through various types of questions onsite (administered through Kahoot) in the TNS28 conference on October 29, 2025, at Kantary Hills Hotel, Chiang Mai, Thailand.

2. All participants are required to use **only one tablet per team** to submit answers via Kahoot application.

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. Scoring is based entirely on the Kahoot system. No manual adjustments will be made.

7. Final rankings are based on the total score, which incorporates accuracy and speed. Faster responses earn higher rank, based on Kahoot's scoring algorithm.
8. Incorrect answers receive no points, regardless of response time. No points will be deducted for wrong answers.
9. Winners will be determined by the highest total score.
10. In the event of a tie, there will be an additional question to determine the winner.

Evaluation and Scoring Criteria:

Elimination Rounds (online)

1. For Elimination Round I, scoring is based on the number of correct answers submitted within the allotted time.
2. For Elimination Round II, scoring is based entirely on the Kahoot system. No manual adjustments will be made.
3. Final rankings are based on the total score, which incorporates accuracy and speed. Faster responses earn higher rank, based on Kahoot's scoring algorithm.
4. Incorrect answers will receive no points, regardless of response time. No points will be deducted for wrong answers.
5. The **team's ranking** will be determined by the combined scores of all team members. Winners will be determined by the highest total team score.
6. In the event of a tie, there will be an additional question to determine the winner.
7. Only the Top 30 teams with the highest total scores in Round I will advance to Round II (SemiFinal Round).
8. Only the top 10 finalist teams with the highest total scores in Round II will advance to the

International Final Round.

9. The official results of Elimination Rounds I and II will be communicated to the coach via email and published on the official website. Participants are expected to regularly check their email for updates and announcements.

International Final Rounds (onsite)

1. Scoring is based entirely on the Kahoot system. No manual adjustments will be made.
2. Final rankings are based on the total score, which incorporates accuracy and speed. Faster responses earn higher rank, based on Kahoot's scoring algorithm.
3. Incorrect answers will receive no points, regardless of response time. No points will be deducted for wrong answers.
4. The team's ranking will be determined by the total team scores. Winners will be determined by the highest total team score.
5. In the event of a tie, there will be an additional question to determine the winner.

6. The official results of the International Final Round will be announced at the TNS Neuro Quiz 2025 Award Ceremony in TNS28 conference on October 29, 2025.

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 30 teams advancing to Elimination Round II (Semi-Finals) will receive Certificates of Runner-Up Finalist in the form of an e-certificate.
5. 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 TNS28 Conference (October 29, 2025). Further details regarding accommodation will be announced prior to final round confirmation.

TNS Neuro Quiz 2025 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

TNS NEURO QUIZ 2025

Championship Award

- TNS Neuro Quiz Award Trophy
- Cash Prize: 40,000 Thai Baht
- Certificates for all team members
- Special gift vouchers from sponsors

REGISTRATION FEE
500 Thai Baht (THB) / team

IMPORTANT DATE

16 June-11 July, 2025 - Registration starts
14 July, 2025 - Announcement of eligible teams and program details
20 July, 2025 - Online Elimination Round I (MCQ test)
27 July, 2025 - Announcement of the Top 30 teams
10 August, 2025 - Online Elimination Round II (Kahoot)
17 August, 2025 - Announcement of the Top 10 teams
29 October, 2025 - International Finals (Onsite) at Kantary Hills Hotel, Chiang Mai, Thailand

CRITERIA

- High school students equivalent to Grades 10-12 in Thailand and International schools
- 3 students + 1 coach, assigned by the school principal or director
- A maximum of 2 teams per school, identified as Team A and Team B

tns.neuro.quiz.info@gmail.com

SPONSOR

BRAND'S Essence of Chicken

REGISTRATION

INFORMATION

Announcement: Top 30 Qualified Teams – TNS Neuro Quiz 2025

The TNS Neuro Quiz 2025 Organizing Committee is pleased to announce the **Top 30 qualified teams** that have successfully advanced from the Elimination Round I and will proceed to the next stage of the competition. The teams are listed in alphabetical order of Team ID as follows:

APW-A	***Angthong Patthamarot Wittayakhom School***	
	Harutai Antaisong	
	Wimutra Sarai, Pattama Sagarasaeranee, Perapatta Assadathorn	
BD-A	***Bodindecha (Sing Singhaseni) School***	
	Chonticha Jitsukon	
	Nataphat Treephopnakkul, Jaruda Jaruvijit, Patthanun Chantaracha	
ICS-A	***International Community School***	
	Narada Ewing	
	Kalyarak Supthavichaiyakul, Pimmada Lerdsinpakdee, Prinn Chongkititisakul	
IDS-A	***Islamic Sciences Demonstration School***	
	Kofsoh Wani	
	Muhammadrizki Yasing, Muhammatcharif Samae, Nattanun Natthakornsakul	
IDS-B	***Islamic Sciences Demonstration School***	
	Kofsoh Wani	
	Anwa madadam, Alhuda Srisang, Masudee Damsa	
KVI-A	***Kamnoetvidya Science Academy***	
	Tanawan Leeboonngam	
	Krittaphat Saicheua, Pann Nithiuthai, Phacharaphol Pangjunant	
MD-A	***Materdei School***	
	Natthamon Suphagan	
	Ravipreeya Ratprasartporn, Nattamon Debavalya, Jaifah Nanatanapramoth	
MUI-A	***Mahidol University International Demonstration School***	
	Balachander Nagaraj	
	Chutchaya Kuwatana, Kanan Nasila, Purimphat Khornaiyasruang	

MWI-A	*Mahidol Wittayanusorn School*	
	Sorachai Sae-lim	
	Panravi Tongpun, Sutthikarn Trachadamrong, Thanyathorn Leephong	
MWI-B	*Mahidol Wittayanusorn School*	
	Sorachai Sae-lim	
	Nutthanun Janyongsak, Kodchamon Sriruenthong, Noppawut Chayakul	
NT-A	*The Newton Sixth Form*	
	Yanisa Wassanatip	
	Marisa Ise, Peeranutt Krungkaew, Naratorn Kunanopparat	
NT-B	*The Newton Sixth Form*	
	Yanisa Wassanatip	
	Ruetaipat Poobrasert, Thannicha Lampanit, KaE Pinpairoh Kitamura	
PHS-A	*Panyarat High School*	
	Luke Ruedisueli	
	Chayanat Inthasakubon, Chakrapong Watanajiraj, Alif Sama	
PHS-B	*Panyarat High School*	
	Luke Ruedisueli	
	Orach Wongoungern, Meekhun Thamprapasasadon, Kanisorn Wongphichayawisan	
PM-A	*Wat Pra Sri Mahathat Secondary Demonstration School, Phranakhon Rajabhat University*	
	Suphasikant Lobbumrung	
	Nutt Phatkulthanut, Polnaphong Danuthonthan, Pratchaya Pithaworapak	
PPT-A	*Princess Chulabhorn Science High School Pathum Thani*	
	Sunisa Kongkalai	
	Pattarapol Boonsom, Hirunyasuth Stidpongpaiboon, Kantapitch Wanarat	
PPT-B	*Princess Chulabhorn Science High School Pathum Thani*	
	Sunisa Kongkalai	
	Nutt Punpulnithikul, Masiya Wanichpongpun, Chavapon Rungrittipanyo	
RYW-A	*Rayongwittayakom School*	
	Maneerat Suwanwaree	
	Ausma Burakorn, Thanika Somvanapanich, Thanakrit Sangprachoom	

SIS-B	*Singapore International School of Bangkok*	
	Victoria Willett	
	Piyalux Ngamlertdanai, Phupetch Trakarnvichit, Proud Prompattanapakdee	
SKR-A	*Suankularb Wittayalai Rangsit School*	
	Wassamol Chokkajidsamphan	
	Jiruchaya Chaisuriyaphun, Kunyanat Sangawong, Ruethaikan Junsuk	
SKR-B	*Suankularb Wittayalai Rangsit School*	
	Pratchaya Ngasit	
	Phiantankhun Phianpailoon, Nuttakit Dittakasorn, Krittipop Thanyakitpaiboon	
SNR-A	*Suksanari School*	
	Pantita Kannika	
	Similan Surarith, Sawidchaya Dangrungruaeng, Rina Boonyanusiri	
SNR-B	*Suksanari School*	
	Pantita Kannika	
	Achirayapat Kajornsaeng, Boonyisa Wasupornrujee, Phakjira Janprasit	
SPS-A	*Srinakharinwirot University Prasarnmit Demonstration School*	
	Thanalawan Pealkha	
	Kanyada Jaturapornprom, Thunyapat Prasittipong, Rapirat Supaiboon	
TUN-A	*Triamudomsuksanomklao School*	
	Natthapong Sakunkitphokhanon	
	Ammares Auncharoen, Thannaphat Nareumanphokin, Kietnumchai Suksamanphanich	
UP-A	*Udonpittayanukoon School*	
	Jatuput Lokayut	
	Juthanon Srisamer, Vichcharat Chaichak, Ratthee Siludom	
UP-B	*Udonpittayanukoon School*	
	Wanassanan Donmuensri	
	Nuttha Dunbodee, Chanoknan Somboonsup, Kornchanok Guayapai	
VCI-B	*Varee Chiang Mai International School*	
	Mohamed Aden	
	Dayeon Paek, Sen Hei Leung, Ziyu Tang	
YRC-A	*Yupparaj Wittayalai School*	
	Sarayoot Viriyakunanant	
	Napat Jeenarach, Paksaran Chantra, Kantathi Jaikaew	

YW-A	*Hatyaiwittayalai School*	
	Mariam Watthanard	
	Thanaporn Salika, Monnarat Binlee, Chayanit Kantangkul	

We congratulate all advancing teams for their exceptional performance and commend all participants for their passion and commitment to neuroscience.

Further details regarding the Elimination Round II (Semi-Final Round) will be communicated to team participants via email. Please stay tuned and check your inbox regularly.

Digitally signed by 2709e9a3-882d-4d29-9aa5-ccc45e1e989a
Date: 2025.07.24 12:23:43 +07'00'

Assoc.Prof.Dr. Akkradate Siriphorn	Assoc.Prof.Dr. Chailerd Pichitpornchai	Dr. Prateep Amonruttanapun
Committee of the TNS Neuro Quiz 2025	Chair of the TNS Neuro Quiz 2025	Committee of the TNS Neuro Quiz 2025

Announcement: Top 10 Finalist Teams – TNS Neuro Quiz 2025

The TNS Neuro Quiz 2025 Organizing Committee is pleased to announce the **Top 10 Finalist teams** for the International Final Round (onsite), to be held at Kantary Hills Hotel, Chiang Mai, Thailand, on October 29, 2025. The teams are listed in alphabetical order of Team ID as follows:

ICS-A ***International Community School***
Narada Ewing
Kalyarak Supthavichaiyakul, Pimmada Lerdsinpakdee, Prinn Chongkitisakul

IDS-A ***Islamic Sciences Demonstration School***
Kofsoh Wani
Muhammadrizki Yasing, Muhammatcharif Samae, Nattanun Natthakornsakul

KVI-A ***Kamnoetvidya Science Academy***
Tanawan Leeboonngam
Krittaphat Saicheua, Pann Nithiuthai, Phacharaphol Pangjunant

MD-A ***Materdei School***
Natthamon Suphagan
Ravipreeya Ratprasartporn, Nattamon Debavalya, Jaifah Nanatanapramoth

MWI-A ***Mahidol Wittayanusorn School***
Sorachai Sae-lim
Panravi Tongpun, Sutthikarn Trachadamrong, Thanyathorn Leephong

NT-A ***The Newton Sixth Form***
Yanisa Wassanatip
Marisa Ise, Peeranutt Krungkaew, Naratorn Kunanopparat

PM-A ***Wat Pra Sri Mahathat Secondary Demonstration School, Phranakhon Rajabhat University***
Suphasikant Lobbumrung
Nutt Phatkulthanut, Polnaphong Danuthonthan, Pratchaya Pithaworapak

SIS-B ***Singapore International School of Bangkok***
Victoria Willett
Piyalux Ngamlertdanai, Phupetch Trakarnvichit, Proud Prompattanapakdee

SKR-A ***Suankularb Wittayalai Rangsit School***
Wassamol Chokkajidsamphan
Jiruchaya Chaisuriyaphun, Supakarn Kumfoo, Ruethaikan Junsuk

SKR-B ***Suankularb Wittayalai Rangsit School***
Pratchaya Ngasit
Phiantankhun Phianpailoon, Nuttakit Dittakasorn, Krittipop Thanyakitpaiboon

We congratulate all advancing teams for their exceptional performance and commend all participants for their passion and commitment to neuroscience.

Further details regarding the program schedule and information for the International Final Round will be sent to all qualifying teams via email by late August 2025. Please check your inbox regularly.

A. Siriphorn
Assoc.Prof.Dr. Akkradate Siriphorn
Committee of the TNS Neuro Quiz 2025

Chailerd Pichitpornchai
Assoc.Prof.Dr. Chailerd Pichitpornchai
Chair of the TNS Neuro Quiz 2025

Prateep
Dr. Prateep Amonruttanapun
Committee of the TNS Neuro Quiz 2025

SKT Course & Workshop: The Elderly Memory and Health Healing

Abstract

SKT Workshops, intensive learning and practical skills development in a 3 hours, stimulate exchange of the scientific knowledges and offering training in SKT 1,3,7,8 technics to enhance the elderly memory and health problems healing. The workshops organized by TNS and the founder of SKT Meditation Healing Exercises 1-8, Prof. Dr. Somporn Kantharadussadee Triamchaisri and leading neuroscientists, Prof. Dr. Supin Chompoopong, Emeritus Professor Dr. Sukumal Chongthamakun, Prof. Dr. Banthit Chetsawang, Assoc. Prof. Dr. Chailerd Pichitpornchai and Assoc. Prof. Dr. Dr. Sujira Mukda, Assoc. Prof. Dr. Phakkarawat Sittiprapaporn, Assoc. Prof. Oriana Tio Parachita Nainggolan in their fields.

SKT Workshops are scientific and health healing based SKT meditation innovation transferring via training, discussion, evidence based, and experiences sharing. The workshop embraces the SKT concepts of self- healing, caregivers supporting anytime, anywhere, any one's, any religions, and any settings. Normal daily life activities, medications, and /or food supplements consumption are not changes. The SKT Workshops takes place on site event only.

SKT Practical workshops provide training in 4 technics of SKT Meditation Healing Exercises and promote the transfer of healing modalities between participants and trainers.

Benefits of SKT Workshops are enhance self-healing, up skills and re skills the elderly memory improvement, intensive practicing in a supportive training environment.

Activities

A1. Health examination & Welcome drink

A2. Benefits of SKT Meditation Healing Exercises

A3. Reducing stresses hormone, adjustments of sickness substances, promoting happiness substances, Improving and healing memory substances

A4. Practicing SKT 1: Fit and firm brain cells and memory

A5. Practicing SKT 3: Fit and firm body and mind

A6. Reviewing and Practicing SKT 1 and SKT 3 (Brand relaxation corner)

A7. Practicing SKT 7: Fit and firm cardiovascular system

A8. Practicing SKT 8: Fit and firm brain potential

A9. Reviewing and Practicing SKT 7 and 8 (Brand relaxation corner)

A10. Discussion & Evaluation

Advisory

Professor Dr. Supin Chompoopong, TNS President 2025-2026

Speakers & Trainers

Professor Dr. Somporn Kantharadussadee Triamchaisri (Dr. SKT)

Professor Dr. Banthit Chetsawang

Assoc. Prof. Dr. Chailerd Pichitpornchai

Assoc. Prof. Dr. Sujira Mukda

Assoc. Prof. Dr. Phakkharawat Sittiprapaporn

Assoc. Prof. Oriana Tio Parahita Nainggolan

Registration

All participants can register by completing the online registration form. Registration will close on October 28 2025, 16.00 GMT+7. Participating in the workshop fee is 1,000 THB. For 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 on October 29, 2025 (2:00 pm-5:00 pm)

English language on October 31,2025 (9:00am-12:00 am)

Organization

Thai Neuroscience Society

Workshops supported by Brand Thailand

SKT Course & Workshop: The Elderly Memory and Health Healing

Kantary Hills Hotel, Chiang Mai, Thailand

ACTIVITIES

- A1. Health examination & Welcome drink
- A2. Benefits of SKT Meditation Healing Exercises
- A3. Reducing stresses hormone, Adjustments of sickness substances, promoting happiness substances, Improving and healing memory substances
- A4. Practicing SKT 1: Fit and firm brain cells and memory
- A5. Practicing SKT 3: Fit and firm body and mind
- A6. Reviewing and Practicing SKT 1 and SKT 3 (BRAND'S relaxation corner)
- A7. Practicing SKT 7: Fit and firm cardiovascular system
- A8. Practicing SKT 8: Fit and firm brain potential
- A9. Reviewing and Practicing SKT 7 and 8 (BRAND'S relaxation corner)
- A10. Discussion & Evaluation

ADVISORY

Professor Dr. Supin Chompoopong
TNS President 2025-2027

Emeritus Professor Dr. Sukumal Chongthammakun
TNS Former President

LANGUAGES

Thai language on **October 29, 2025** (2:00 pm-5:00 pm)

English language on **October 31, 2025** (9:00 am-12:00 pm)

SPEAKERS & TRAINERS

Professor Dr. Somporn Kantharadussadee Triamchaisri (Dr. SKT)
Professor Dr. Banthit Chetsawang
Assoc. Prof. Dr. Chailerd Pichitpornchai
Assoc. Prof. Dr. Sujira Mukda
Assoc. Prof. Dr. Phakkharawat Sittiprapaporn
Assoc. Prof. Oriana Tio Parahita Nainggolan

Registration

All participants can register by completing the online registration form.
Registration will close on October 28, 2025, 16.00 GMT+7.
Participating in the workshop fee is 1,000 THB.
For questions please send an email to meditationskt@gmail.com.

QR Code ลงทะเบียน

QR Code รายละเอียดงาน

IBRO-APRC Supported Associate School 2025

Winter School on Neuroplasticity Across the Lifespan

Tentative Program (27 OCT - 2 NOV 2025)

RB0410, 4th Floor, Multidisciplinary Building, Faculty of Medicine, Chiang Mai University , Chiang Mai 50200 , THAILAND

No.	ID	Position	First Name	Last Name	Email	Gender	Nationality
1	25APRC6-8785199052	Assistant Professor	Anuradha	Sharma	s.anuradha21@ymail.com	Woman	India
2	25APRC6-3665165032	Assistant Professor	Manoj	Dandekar	manoj.dandekar@niperhyd.ac.in	Man	India
3	25APRC6-2346347899	Associate Professor	Vikas Kumar	Tiwari	vikas.kumar.tiwari.r4@dc.tohoku.ac.jp	Man	India
4	25APRC6-1677445634	Dr.	Raj	Katariya	rajkatariya4@gmail.com	Man	India
5	25APRC6-2763298955	Assistant Professor	Ankit	Ganeshpurkar	ankitganeshpurkar@gmail.com	Man	India
6	25APRC6-9177066098	Lecturer	Thenmoly	Damodaran	dthenmoly@yahoo.com	Woman	Malaysia
7	25APRC6-4599583594	Assistant Professor	Divya	Soni	divyapharma020@gmail.com	Woman	India
8	25APRC6-1869468412	PhD Student	Manisha	Suri	manishasuri9015@gmail.com	Woman	India
9	25APRC6-2297568581	Other	Tara	Sudhadevi	chandran.tara@gmail.com	Woman	India
10	25APRC6-1884307765	Postdoc	Jiasui	Yu	yujiasuijs@163.com	Man	Hong Kong
11	25APRC6-1068361799	Dr.	Rabia	Akram	rabiaakram368@gmail.com	Woman	Pakistan
12	25APRC6-3575292180	Research Assistant	Long Hoang Kim	Nguyen	hoangkimlong.hcmiu@gmail.com	Woman	Vietnam
13	25APRC6-5966287930	Postdoc	Muhammad	Jahangir	jahangir@zju.edu.cn	Man	Pakistan
14	25APRC6-3848382278	Lecturer	Suman	Pokhrel	pokhrelisuman@gmail.com	Man	Nepal
15	25APRC6-3322742688	Master Student	Titaree	Yamsri	titareeyamsri@gmail.com	Woman	Thailand
16	25APRC6-2418206055	Assistant Professor	Jakkrit	Nukitram	jakkritnu@kku.ac.th	Man	Thailand
17	25APRC6-0270581727	Assistant Professor	Yoottana	Janthakhin	yoottana.ja@go.buu.ac.th	Man	Thailand
18	25APRC6-4761671144	PhD Student	Nutthida	Phianchana	nutthida.phian@gmail.com	Woman	Thailand

19	25APRC6-5406553730	Assistant Professor	Anupong Thongklam	Songsaad	Anupong.son@mahidol.ac.th	Man	Thailand
20	25APRC6-8643356732	Lecturer	Phetcharat	Boonruamkaew	pphetcharat.bo@gmail.com	Woman	Thailand
21	25APRC6-4088264704	Dr.	Yaowapa	Trangan	am.yaowapa00@gmail.com	Woman	Thailand
22	25APRC6-4424831856	PhD Student	Sulaifan	Waehama	Sulaifanw66@nu.ac.th	Man	Thailand
23	25APRC6-6010449221	PhD Student	Mananya	Potima	mananya.ptm@gmail.com	Woman	Thailand
24	25APRC6-0797923365	PhD Student	Sudichhya	Tamrakar	suditamrakar@gmail.com	Woman	Nepal
25	25APRC6-1594121933	Master Student	HANWEN	MI	hanwen.mi@student.mahidol.edu	Man	China
26	25APRC6-8521178707	PhD Student	Siripaporn	Kesyou	siripapornk62@nu.ac.th	Woman	Thailand
27	25APRC6-6945024158	PhD Student	Plaiyfah	Janthueng	plaiyfahj66@nu.ac.th	Woman	Thailand
28	25APRC6-9760923144	PhD Student	Wanfrutkon	Waehama	wanfrutkonw65@nu.ac.th	Man	Thailand
29	25APRC6-4478129836	PhD Student	Yanmei	Huang	yanmei_huang@cmu.ac.th	Woman	China
30	25APRC6-5957239312	PhD Student	Houzhi	Cheng	houzhi_cheng@cmu.ac.th	Man	China
31	25APRC6-1118441801	Master Student	Poramet	Klangmongkon	nst.porakl@gmail.com	Man	Thai
32	25APRC6-6365914931	BSc	Napasakorn	Verasaksuriya	pinyeon609@gmail.com	Woman	Thai
33	25APRC6-1367126953	Bachelor Student	Narinporn	Tresteankij	narinporn.trs@gmail.com	Woman	Thai
34	25APRC6-1513190996	Master Student	Phossawee	Kongkaew	garfield29phos@gmail.com	Man	Thai
35	25APRC6-4496912161	PhD student	Ponthip	Cheenkhwan	ponthip_o@hotmail.com	Woman	Thai
36	25APRC6-7903368447	PhD Student	Puncharatsm	Pannin	Ppuncharatsm@gmail.com	Woman	Thai
37	25APRC6-1434971384	Master Student	Kotchapit	Maksri	kotchapitmaksri@gmail.com	Woman	Thai
38	25APRC6-0414763504	Master Student	Fahsai	Kaewpikul	fahsai.kaewp@gmail.com	Woman	Thai
39	25APRC6-4898608079	PhD Student	Wongsakorn	Siripun	wsiripant@gmail.com	Man	Thai
40	25APRC6-2280620418	PhD Student	Vishal	Kumar	vishalrajput0408@gmail.com	Man	India

Monday 27-10-2025 (DAY 1)			
Time		**Session**	**Room**
8.00	**8.30**	**Registration** 4th Floor, Multidisciplinary Building, Faculty of Medicine, Chiang Mai University	**RB0410**
8.30	**9.00**	**Opening remark** Prof. Dumnoensun Pruksakorn, Associate Dean in Research, Innovation and International Affairs Assoc. Prof. Saranyapin Potikanond, Head of Pharmacology Department	**RB0410**
9.00	**9.50**	**Neuroplasticity in Action: Pediatric Case Studies and Brain Reorganization** Assoc. Prof. Natrujee Wiwattanadittakul Neurology Division, Department of Pediatrics, Faculty of Medicine, Chiang Mai University	**RB0410**
9.50	**10.10**	**Coffee break**	
10.10	**11.00**	**Acquired epileptogenesis and Neuroplasticity** Assoc. Prof. Atiwat Soontornpun Neurology Division, Department of Internal Medicine, Faculty of Medicine, Chiang Mai University	**RB0410**
11.00	**11.50**	**iPSC-derived motor neurons (iMNs): From disease modeling to therapy discovery** Dr.Wasinee Wongkummool Center of Multidisciplinary for Advanced Medicine, Faculty of Medicine, Chiang Mai University	**RB0410**
12.00	**13.00**	**Lunch**	
13.00	**13.50**	**Emerging Pharmacotherapy in Neurodegenerative diseases** Assist. Prof. Jannapas Tharavichitkun Department of Pharmaceutical care, Faculty of Pharmacy, Chiang Mai University	**RB0410**
14.00	**14.50**	**Leveraging Nature, Crafting Advanced Herbal Formulation for Neurological Wellness** Assoc. Prof.Chuda Chittasupho Department of Pharmaceutical sciences, Faculty of Pharmacy, Chiang Mai University	**RB0410**
14.50	**15.00**	**Coffee break**	
15.00	**15.50**	**Multi omics approaches to understand neuronal plasticity** Dr. Ranchana Yeewa Center of Multidisciplinary for Advanced Medicine, Faculty of Medicine, Chiang Mai University	**RB0410**
15.50	**16.00**	**Summary of Day 1**	**RB0410**

Tuesday 28-10-2025 (DAY 2)			
Time		**Session**	**Room**
8.30	**9.00**	**Registration/Introduction**	**RB0410**
9.00	**9.50**	**Epigenetic regulation of Neuronal Function and Plasticity in *Drosophila melanogaster*** Dr. Luca Lo Piccolo Center of Multidisciplinary for Advanced Medicine, Faculty of Medicine, Chiang Mai University	**RB0410**
9.50	**10.40**	***Drosophila* in Drug Discovery** Dr. Salinee Jantrapirom Department of Pharmacology, Faculty of Medicine, Chiang Mai University	**RB0410**
10.40	**11.00**	**Coffee break**	
11.00	**12.00**	**Lab 1: *Drosophila*'s Neuromuscular Junction (NMJ) dissection** Dr. Luca Lo Piccolo and team	**MD-2, CMUTEAM**
12.00	**13.00**	**Lunch**	
13.00	**13.50**	**Choices in Decline: What Monkeys Reveal about Decision-Making** Dr. Lalitta Suriya-Arunroj Faculty of Medicine, Chulalongkorn University	**RB0410**
13.50	**14.40**	**Lab 2: Non-human primate neurobehavioral study** Dr. Lalitta Suriya-Arunroj and team	**RB0410**
14.40	**15.00**	**Coffee break**	
15.00	**15.50**	**Tracking Cognitive Function in Rodents Through Maze-Based Behavioral Models** Dr. Ruedeemars Yubolphan Department of Pharmacology, Faculty of Medicine, Chiang Mai University	**RB0410**
15.50	**16.00**	**Summary of Day 2**	**RB0410**

Wednesday 29-10-2025 (DAY 3)			
Time		**Session**	**Room**
8.30	**9.00**	**Registration/Introduction**	**RB0410**
9.00	**9.50**	**Exploring Cellular and Molecular Mechanisms in Neurodegenerative diseases using iPSC neurons** Dr. Nithi Asavapanumas Chakri Naruebodindra Medical Institute, Faculty of Medicine, Ramathibodi Hospital University	**RB0410**

9.50	**10.40**	**Knowing the Unknown: Parkinson's Hidden Face** Prof. Wael Mohamad BMS, KOM, International Islamic University Malaysia	**RB0410**
10.40	**11.00**	**Coffee break**	
11.00	**11.50**	**Sleeping and the Aging Brain: The Bidirectional Relationship** Assoc. Prof. Vorasith Siripornpanich Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University	**RB0410**
11.50	**13.00**	**Lunch at Kantary Hotel**	
13.00	**17.00**	**The 28th Annual Conference of Thai Neuroscience Society (TNS28)**	
17.30	**20.00**	**Welcome dinner at Amataros Sourdough Pizza** **** For Invited speakers, IBRO lecturers, invited TNS committee, TNS28 organizing committee, IBRO students***	

Saturday 1-11-2025 (DAY 4)			
Time		**Session**	**Room**
8.30	**9.00**	**Registration/Introduction**	**RB0410**
9.00	**9.50**	**Genetically Engineered *Drosophila melanogaster* as a model for Neurological diseases** Assoc. Prof. Hideki Yoshida Department of Applied Biology, Kyoto Institute of Technology, Japan	**RB0410**
9.50	**10.40**	**Lab 3: Drosophila Transgenesis and NMJ visualization by confocal microscope** Assoc. Prof. Hideki Yoshida and team	**MD-2, CMUTEAM**
10.40	**11.00**	**Coffee break**	
11.00	**12.00**	**Channelopathies of the Brain: Mechanisms, Disorders, and Therapeutic Insights** Dr. Arnaud Monteil Institute of Functional Genomics, Montpellier, France and Siriraj Hospital, Mahidol University	**RB0410**
12.00	**13.00**	**Lunch**	
13.00	**13.50**	**Live imaging of glia-neuron plasticity: from synaptic phagocytosis to developmental erythrophagocytosis** Dr. Ryuta Koyama National Institute of Neuroscience, National Center of Neurology and Psychiatry, Japan	**RB0410**

13.50	**14.40**	**Nanopipette biosensors for cell and single molecule analysis: Scanning Ion Conductance Microscopy** Prof. Yuri Korchev Faculty of Medicine, Imperial College London, England	**RB0410**
14.40	**15.00**	**Coffee break**	
15.00	**15.50**	**Lab 4: Confocal Microscopy Q&A** **Dr.Luca and team**	**CMUTEAM**
15.50	**16.10**	**Closing Remark**	**0410**

Sunday 2-11-2025 (DAY 5)		
Time		**SessionRoom**
8.30	**17.00**	**Chiang Mai Free style**

IBRO-APRC Supported Associate School 2025

"Winter School on Neuroplasticity Across the Lifespan"

Organizing Committee

Salinee Jantrapirom, B.Pharm, PhD

Chair, Organizing Committee

Department of Pharmacology Faculty of Medicine Chiang Mai University

Luca Lo Piccolo, PhD

Center of Multidisciplinary for Advanced Medicine, Faculty of Medicine, Chiang Mai University

Supanimit Theekachunhatien, MD, PhD

Professor in Pharmacology, Faculty of Medicine, Chiang Mai University

Ranchana Yeewa, PhD

Center of Multidisciplinary for Advanced Medicine, Faculty of Medicine, Chiang Mai University

Wasinee Wongkummool, PhD

Center of Multidisciplinary for Advanced Medicine, Faculty of Medicine, Chiang Mai University

Ruedeemars Yubolphan, PhD

Department of Pharmacology Faculty of Medicine Chiang Mai University

Natrujee Wiwattanadittakul, MD

Neurology Division, Department of Pediatrics, Faculty of Medicine, Chiang Mai University

Rungsinee Phongpradist, B.Pharm, PhD

Department of Pharmaceutical sciences, Faculty of Pharmacy, Chiang Mai University

Jannapas Tharavichikun, Higher Grad. Dip. in Pharm.

Department of Pharmaceutical care, Faculty of Pharmacy, Chiang Mai University

Phatcharida Jantaree, PhD

Department of Biochemistry, Faculty of Medicine, Chiang Mai University

Natsinee U-On, MSc

Center of Multidisciplinary for Advanced Medicine, Faculty of Medicine, Chiang Mai University

Chansunee Panto, BSc

Center of Multidisciplinary for Advanced Medicine, Faculty of Medicine, Chiang Mai University

Puttachat Poound, BSc

Center of Multidisciplinary for Advanced Medicine, Faculty of Medicine, Chiang Mai University

The 28th Thai Neuroscience Society Annual Conference 2025

IBRO-APRC Supported Associate School 2025

“Winter School on Neuroplasticity Across the Lifespan”

27 OCT - 2 NOV 2025

Faculty of Medicine, Chiang Mai University,

Chiang Mai 50200, THAILAND

IBRO-APRC Supported Associate School 2025
“Winter School on Neuroplasticity Across the Lifespan”
Invited Lecturers

Dr. Luca Lo Piccolo

Center of Multidisciplinary for Advanced Medicine, Faculty of Medicine, Chiang Mai University

Assoc. Prof. Natrujee Wiwattanadittakul

Neurology Division, Department of Pediatrics, Faculty of Medicine, Chiang Mai University

Assoc. Prof. Atiwat Soontornpun

Neurology Division, Department of Internal Medicine, Faculty of Medicine, Chiang Mai University

Assist. Prof. Jannapas Tharavichitkun

Department of Pharmaceutical care, Faculty of Pharmacy, Chiang Mai University

Assoc. Prof. Chuda Chittasupho

Department of Pharmaceutical sciences, Faculty of Pharmacy, Chiang Mai University

Dr. Ruedeemars Yubolphan

Department of Pharmacology Faculty of Medicine Chiang Mai University

Dr. Salinee Jantrapirom

Department of Pharmacology Faculty of Medicine Chiang Mai University

Dr. Ranchana Yeewa

Center of Multidisciplinary for Advanced Medicine, Faculty of Medicine, Chiang Mai University

Dr. Nithi Asavapanumas

Chakri Naruebodindra Medical Institute, Faculty of Medicine, Ramathibodi Hospital University

Dr. Lalitta Suriya-Arunroj

Faculty of Medicine, Chulalongkorn University

Prof. Wael Mohamad

BMS, KOM, International Islamic University Malaysia

Dr. Wasinee Wongkummool

Center of Multidisciplinary for Advanced Medicine, Faculty of Medicine, Chiang Mai University

Dr. Ryuta Koyama

National Institute of Neuroscience, National Center of Neurology and Psychiatry, Japan

Assoc. Prof. Vorasith Siripornpanich

Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University

Assoc. Prof. Hideki Yoshida

Department of Applied Biology, Kyoto Institute of Technology, Japan

Dr. Arnaud Monteil

Institute of Functional Genomics, Montpellier, France and Siriraj Hospital, Mahidol University

Prof. Yuri Korchev

Faculty of Medicine, Imperial College London, England

IBRO Supported Associate school 2025
School Title: Winter School on Neuroplasticity Across the Lifespan: Development, Aging, and Neurodegeneration
School Venue: Faculty of Medicine, Chiang Mai University

Organiser Details
• Title: Dr.
• Name: Dr.Salinee Jantrapirom
• Gender * Female
• Organiser E-mail (Give one only) * salinee.jan@cmu.ac.th
• Current Professional Title *-
• Professional Affiliation * Faculty of Medicine, Chiang Mai University
• City: Chiang Mai
• Country: Thailand

Previously organized Schools and/or Events
1) Biomedical conference and Drosophila workshop supported by the Japan Society of the Promotion of Science (JSPS) core-to-core program and NRCT, Chiang Mai, Thailand, December 2016
2) The 41st annual meeting of Pharmacological and Therapeutic Society of Thailand, Chiang Mai Thailand, February 2019
3) Kyoto Institute of Technology-ASEAN International Symposium for Agricultural Biomedical Research Network, Chiang Mai, Thailand, January 2025
Region: Asia-Pacific

School duration: 7 days
Proposed Start date: 27/10/2025
Proposed End date: 2/11/2025
In what format will the school take place? Hybrid
Funding limits:
Associate Schools: up to 20,000 USD
Support request for: Associate School

AWARD

Honorary Membership
Professor Dr Prasop Ratanakorn's Lecture
Professor Dr Roongtam Ladpli's Lecture

2025 TNS Honorary Membership

Professor Dr. Siriporn Chattipakorn

Professor of Neuroscience and Oral Biology
Faculties of Medicine and Dentistry, Chiang Mai University

It is with great pleasure that the Thai Neuroscience Society (TNS) announces the appointment of Professor Dr. Siriporn Chattipakorn, Professor of Neuroscience and Oral Biology, as an Honorary Member of our esteemed society.

Professor Dr. Siriporn Chattipakorn is an internationally recognized neuroscientist whose exceptional academic and research achievements have profoundly advanced neuroscience in Thailand and beyond. She serves as Professor in the Department of Oral Biology and Diagnostic Sciences, Faculty of Dentistry, and heads the Neurophysiology Unit at the Cardiac Electrophysiology Research and Training (CERT) Center, Faculty of Medicine, Chiang Mai University.

Professor Siriporn obtained her D.D.S. from Chiang Mai University and her Ph.D. in Oral Biology (Neuroscience) from the University of North Carolina at Chapel Hill, followed by postdoctoral training in Physiology and Biophysics at the University of Alabama at Birmingham.

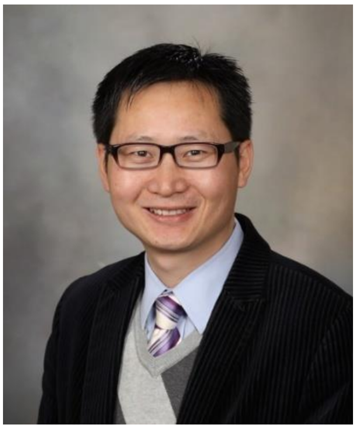
Her pioneering research in neurophysiology, particularly on the impact of metabolic syndrome, diabetes, thalassemia, and chemotherapy on brain function, has made significant scientific contributions, spanning from mitochondrial studies to clinical applications. She has published over 520 peer-reviewed papers and authored 14 textbooks, making her one of Thailand’s most prolific neuroscientists.

Her numerous honors include the Outstanding Scientist Award (2025), Dushdi Mala Medal in Medical Science (2022) bestowed by His Majesty the King of Thailand, and multiple awards from the National Research Council of Thailand and the Thailand Research Fund.

In recognition of her lifelong dedication, leadership, and groundbreaking research that continues to inspire neuroscientists worldwide, the Thai Neuroscience Society proudly names Professor Dr. Siriporn Chattipakorn as an Honorary Member.

Congratulations to Professor Dr. Siriporn Chattipakorn on this well-deserved honor.

Given on the 29th day of October, 2025.



Prof. Long-Jun Wu, PhD

University of Texas Health Science Center at Houston

e-mail: longjun.wu@uth.tmc.edu

Professor Dr Prasop Ratanakorn's Lecture

Rod-shaped microglia regulate neuronal activity in TDP-43 neurodegeneration

Abstract:
Microglia, the principal immune cells of the central nervous system, have emerged as important players in sensing and regulating neuronal activity. While microglial activation is a hallmark in neurodegeneration, the specific role of microglia in disease-related cortical excitability remains unknown. Utilizing multichannel probe recordings and longitudinal in vivo calcium imaging, we observed neuronal hyperactivity at the initial stage of disease progression in a mouse model of TAR DNA-binding protein 43 (TDP-43) neurodegeneration (rNLS8, regulated nuclear localization sequence-deleted human TDP-43 transgenic mouse model). Spatial and single-cell RNA sequencing revealed a specific subpopulation of microglia, rod-shaped microglia, with a distinct morphology and direct response to cortical hyperactivity. Rod-shaped microglia predominantly interacted with neuronal dendrites and remodeled excitatory synaptic inputs to attenuate motor cortical hyperactivity. Triggering receptor expressed on myeloid cells 2 (TREM2) deficiency led to a marked reduction of rod-shaped microglia accompanied by increased neuronal activity in rNLS8 mice. Together, our results suggest that rod-shaped microglia play a neuroprotective role by attenuating cortical hyperexcitability in TDP-43-related neurodegeneration

Keywords: TDP-43; TREM2; amyotrophic lateral sclerosis; neuroimmunology; rod-shaped microglia

Dr. Long-Jun Wu earned his Ph.D. in Neurobiology from the University of Science and Technology of China in 2004, followed by postdoctoral training at the University of Toronto (2004–2008) and Harvard Medical School (2008–2011). He currently serves as the C. Harold and Lorine G. Wallace Distinguished University Chair, Professor, and Founding Director of the Center for Neuroimmunology and Glial Biology, University of Texas Health Science Center at Houston. Prior to this, he was a Professor of Neurology and Associate Director of the Neuroscience Ph.D. Program at the Mayo Clinic. Dr. Wu's research focuses on neuroimmune interactions, with particular emphasis on the function of microglia in normal and diseased brains. His laboratory integrates cellular, molecular, and systems neuroscience approaches to investigate how immune signaling shapes neural circuits and contributes to neurological disorders. Dr. Wu has published over 200 peer-reviewed articles in leading journals including *Nature Neuroscience*, *Neuron*, *Immunity*, *Journal of Clinical Investigation*, *Nature Communications*, *etc*. His work has been cited more than 20,000 times, with an h-index of 76. Dr Wu received the Teacher of the Year Award at the Mayo Clinic (2020), the NINDS Outstanding Investigator Award (2023), and UT System STARs Award (2024). He is a Senior Editor for *Neuroscience*, *Molecular Brain*, *Neuroscience Bulletin*, and *Cell & Bioscience*, and sits on the editorial boards of *Glia*, *Brain, Behavior, and Immunity*, *Molecular Pain*, and several other journals.

Prof. Ingrid Y Liu, Ph.D.

President, Tzu Chi University
Professor, Institute of Medical Sciences, College of Medicine,
Hualien, Taiwan

Email: ycliu@gms.tcu.edu.tw

Professor Dr Roongtam Ladpli's Lecture

From Sleep to Synapse: Neuroplasticity Impairment via Actin Depolymerization in REM Sleep-Deprived Mice

Abstract:
Sleep deprivation (SD) impairs health and cognition, with rapid eye movement (REM) sleep essential for fear memory consolidation and synaptic integrity. The mechanisms of REM-SD–induced memory loss remain unclear. Using contextual fear conditioning (CFC) in mice, we examined how 4 hours of REM-SD (ZT0–ZT4) after training affects memory consolidation, reconsolidation, and remote memory. REM-SD impaired all memory phases and induced hippocampal presynaptic and postsynaptic dysfunction, as shown by electrophysiology. Protein analyses revealed decreased phosphorylated synapsin (p-Synapsin), CaMKII, and PSD95, alongside increased gelsolin (GSN), BDNF, and AKT, with marked F-actin loss. Intrahippocampal GSN siRNA reversed consolidation and reconsolidation deficits but not remote memory impairment. These results indicate that REM-SD upregulates GSN, promoting actin depolymerization and synaptic destabilization, thereby disrupting memory processes. GSN emerges as a promising molecular target for treating cognitive deficits linked to sleep disruption.

Keywords: Sleep Deprivation, Synaptic Plasticity, Gelsolin, Actin Depolymerization, Fear memory

Dr. Ingrid Y. Liu is a Full Professor at the Institute of Medical Sciences, Faculty of Medicine, Tzu Chi University (TCU), and currently serves as the President of 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*, *Frontiers in Cellular Neuroscience*, *Neuroscience Letters*, *PLOS ONE*, *Brain Research*, *Marine Drugs*, and *BMC Medical Genetics*.

SCIENTIFIC PROGRAM

Plenary Lecture
Special Lecture

Prof. Siriporn Chattipakorn, DDS, PhD

Neurophysiology Unit, Cardiac Electrophysiology Research and Training Center, Faculty of Medicine, Chiang Mai University

Department of Oral Biology and Diagnostic Sciences, Faculty of Dentistry, Chiang Mai University

E-mail: scchattipakorn@gmail.com, siriporn.c@cmu.ac.th

Plenary Lecture

Aging, Obesity, and Cognitive Impairment: Mechanistic Insights and Non-Pharmacological Approaches

Abstract:

Aging and obesity are major contributors to cognitive decline, driven by overlapping mechanisms such as metabolic dysfunction and chronic inflammation. Experimental models using D-galactose or high-fat diets demonstrate that these conditions lead to brain aging through blood-brain barrier disruption, mitochondrial dysfunction, neuroinflammation, and Alzheimer-like pathology. Non-pharmacological interventions – particularly caloric restriction and physical exercise – have proven effective in mitigating these effects by enhancing neuro-metabolism and neurotrophic factor production. In obesity-induced brain aging, both short- and long-term interventions improve cognitive function, with short-term regimens followed by weight maintenance offering a practical approach. However, in D-galactose-induced aging, only long-term interventions yield cognitive benefits. These findings underscore the need for sustained lifestyle modifications to preserve brain health and reduce the impact of age-related neurodegenerative diseases.

Keywords: Brain aging; Obesity; Caloric restriction; Exercise; Cognitive impairment

Prof. Dr. Siriporn Chattipakorn is a Professor at the Faculty of Dentistry, Chiang Mai University (CMU). She serves as Head of the Neurophysiology Unit at the Center of Excellence for Cardiac Electrophysiology and is a co-founder of the Cardiac Electrophysiology Research and Training Center, Faculty of Medicine, CMU. Her research focuses on the mechanistic understanding of neurodegeneration, aiming to identify novel therapeutic strategies to treat these conditions. She has received numerous prestigious national and international awards, including the Outstanding Scientist Award (2025) from the Foundation for the Promotion of Science and Technology under the Patronage of H.M. the King, Distinguished Research Professor Awards (2023, 2017) from the National Research Council of Thailand (NRCT), and the Dushdi Mala Medal in Medical Science (2022) from His Majesty the King of Thailand. Additional accolades include the Thailand Best Researcher Award in Biomedical Sciences (2017), TRF–OHEC Clarivate Analytics Research Excellence Award (2017), and TRF Senior Research Scholar Awards (2017, 2020). Other honors comprise the TRF Best Research Project of the Year (2014), Gold

Elephant Awards (2014, 2022) from CMU, the TRF–CHE–Scopus Researcher Award (2012), and the Outstanding Visiting Scholar Award (2001) from the University of Alabama at Birmingham. She also received Fellowship Awards from the American Heart Association and the American Epilepsy Society. Prof. Chattipakorn has authored over 520 peer-reviewed publications and 13 international textbooks, and serves on editorial and reviewer boards of several leading journals in neuroscience and endocrinology

Prof. Vorasuk Shotelersuk, MD

Chulalongkorn University, Bangkok, Thailand

e-mail: vorasuk.s@chula.ac.th

Plenary Lecture

Advanced DNA Sequencing Technologies in Neuroscience and Human Health

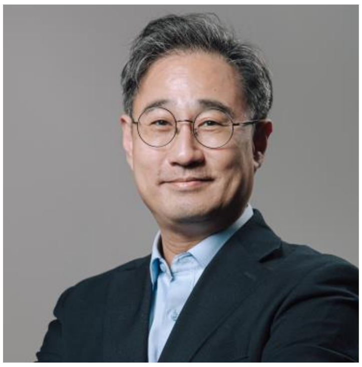
Abstract:

Understanding how the human brain works remains one of the greatest frontiers of science. Achieving this goal requires collaborative efforts across multiple disciplines. One key contribution comes from studying patients with rare, single-gene disorders – nature's own experiments that reveal the molecular basis of disease. Advances in DNA sequencing technologies have revolutionized our ability to identify genetic etiologies of such disorders. These discoveries not only elucidate disease mechanisms but also provide unique insights into normal physiology. Our team has leveraged these technologies to identify several new human disease genes, including those responsible for benign adult familial myoclonic epilepsy (BAFME) types 4 and 8, caused by pathogenic variants in *YEATS2* and *RAI1*. Beyond deepening our understanding of the brain, advanced sequencing offers broader applications in public health. For example, newborn screening using genomic sequencing has the potential to enhance traditional biochemical approaches, paving the way for earlier and more precise interventions. Looking forward, these technologies hold promise for promoting longevity and lifelong health. To this end, our team has launched the LIFE-Seq project, which applies long-read whole-genome sequencing to improve human health across the lifespan.

Keywords: Genetics, DNA sequencing, Rare disease, newborn screening

Prof. Vorasuk Shotelersuk earned his MD with First Class Honours from Chulalongkorn University in Bangkok, Thailand, in 1992. He completed his pediatric residency training in 1996 and went on to become a visiting associate at the NICHD, as well as a genetics fellow at the NHGRI, NIH, USA. In 1999, he was awarded a Diploma from the American Board of Medical Genetics. Currently, he holds the position of Associate Dean for Research Affairs and serves as the founding and current Director of the Center of Excellence for Medical Genomics at the Faculty of Medicine, Chulalongkorn University. He also holds the esteemed title of founding president of the Thai Society of Human Genetics. Professor Shotelersuk's extensive contributions to the field are reflected in his publication of over 290 articles indexed in PubMed, along with an H-index of 45. His dedication and expertise have earned him a plethora of awards, including the UK MRC's Newton Prize and Thailand's Outstanding Researcher and Scientist Awards. He has also been honored with the Outstanding Pediatrician Award from both the Pediatric Society of Thailand and the

Royal College of Pediatricians of Thailand, as well as the Outstanding Physician Award from the Medical Association of Thailand. He has also been appointed to the World Health Organization (WHO) Technical Advisory Group on Genomics.



Prof. Daesoo Kim, PhD

Dept. Brain & Cognitive Sciences, KAIST

e-mail: daesoo@kaist.ac.kr

Plenary Lecture

Neuroscience Takes AI to tango

Abstract:
Neuroscience and artificial intelligence (AI) are no longer in a unidirectional relationship. Instead, they are engaging in a dynamic, reciprocal partnership – like a tango – where each field informs and advances the other. Understanding the complexity of life, especially brain functions and behavior, has long been constrained by the limitations of conventional experimental approaches, which often require scaling down multidimensional data, leading to critical information loss. To overcome these challenges, our research introduces AI-powered systems that offer unprecedented precision and scalability in behavioral neuroscience:

-AVATAR enables real-time 3D motion capture and behavioral classification, allowing the detection of disease phenotypes (e.g., depression, Parkinson's disease, Alzheimer's disease) with accuracy exceeding human observers.
-MATER employs a Transformer-based architecture for pose estimation and action classification through self-supervised learning.
-BehaVERT, a motion language model, are repurposed to interpret motion data as linguistic sequences, enabling sophisticated behavior analysis and disease prediction.

Beyond analysis, AI is integrated into closed-loop brain stimulation systems, enabling real-time feedback experiments that link specific neural circuits to observable behaviors. One notable application is the AI-driven prediction of individual susceptibility or resilience to future stress or disease, showcasing the system's predictive power beyond current behavioral states. Together, these innovations highlight a paradigm shift where AI does not merely assist but transforms neuroscience. This mutual advancement between brain science and AI signals a new era of integrative intelligence.

Keywords: Artificial intelligence, Behavioral analysis, Motion-based prediction

Daesoo Kim is a Dean of the College of the Life Sciences & Bioengineering at KAIST (Korea Advanced Institute of Science and Technology). He is also the Director of the KAIST-Formosa Bio R&D Center and the KAIST-Wonjin Cell Therapy Center. His research focuses on the neural basis of innate behavior, and neurogenerative disorders, with a strong emphasis on integrating cutting-edge neural circuit analysis tools and **AI technologies** into neuroscience. Dr. Kim is internationally recognized for his interdisciplinary approach, combining genetic models, real-time behavior analysis, and neural circuit manipulation. He is also a co-founder of NeuroTobe, a biotechnology company developing novel therapeutics for brain diseases.

Arnaud Monteil, PhD

University of Montpellier, France

e-mail: Arnaud.monteil@cnrs.fr

Special Lecture

Paving the path to therapies for genetic diseases linked to the NALCN channelosome

Abstract:
The excitability of neurons is tightly dependent on their ion channel repertoire, with the leak sodium channel NALCN playing a key role in maintaining resting membrane potential and regulating electrical activity. Studies in animal models highlight NALCN's involvement in essential functions such as respiratory and circadian rhythms, sleep, locomotion, and pain perception. Increasing evidence links NALCN dysfunction to severe neurodevelopmental disorders, characterized by a large panel of symptoms that include developmental delay, facial dysmorphisms, seizures, and premature death. Currently, no therapies exist for these patients. Our research addresses this gap by identifying therapeutic options through innovative *in vitro* and *in vivo* models of NALCN-related diseases. This work is driven by a strong collaboration with clinicians and patient families worldwide.

Keywords: Neuronal excitability, Sodium leak channel, NALCN, Pathogenic variants, Neurodevelopmental disorders, Therapies

Arnaud Monteil earned his Bachelor's degree in Biological Sciences from the University of Montpellier, France, in 1989. He continued his graduate studies in Molecular Biology and Biochemistry at the same institution, completing his PhD in 2000 under the supervision of Dr. Joël Nargeot. His doctoral research led to the cloning and characterization of the then-unidentified T-type voltage-gated calcium channels, resulting in several impactful publications on ion channels and cellular excitability.
Following his PhD, Dr. Monteil joined Prof. Richard J. Miller's laboratory, first at the University of Chicago and later at Northwestern University, as a postdoctoral fellow. There, he identified and cloned the novel four-domain ion channel NALCN. In 2001, he returned to France as a permanent researcher at the National Center for Scientific Research (CNRS).
Since then, Dr. Monteil has led research within the *Ion Channels in Neuronal Excitability and Diseases* team at the Institute of Functional Genomics (IGF) in Montpellier (igf.cnrs.fr). His work focuses on ion channel mutations associated with complex neurological disorders, combining molecular and electrophysiological approaches in vitro (cell lines,

primary cultures) and in vivo (animal models). He also explores novel molecules that modulate ion channel activity for potential therapeutic applications.
Dr. Monteil currently coordinates the European **RestoreLeak** consortium (2022–2026), dedicated to neurodevelopmental disorders linked to NALCN dysfunction. He has also directed two **Hubert Curien** exchange programs (2013–2014, 2020–2021) in collaboration with Dr. Narawut Pakaprot at Mahidol University, Thailand, where he was appointed **Adjunct Professor** in 2023.
In addition to his research, Dr. Monteil has directed the **Viral Vector Production Facility** at Biocampus Montpellier since 2008. He has served as **President of the Ion Channels Society** since 2014 and, since 2023, has chaired the **Scientific Committee of the Channeling Hope Foundation**. He is also an elected member of the **CNRS National Committee (CoNRS)**, Subcommittee #24 (*Physiology, Pathophysiology, and Biology of Cancer*), serving from 2016 to 2025.

Assoc. Prof. Wael Mohamed, MD, PhD

Basic Medical Science Department, Kulliyyah of Medicine, International Islamic University Malaysia (IIUM), Kuantan, Pahang, Malaysia

e-mail: waelmohamed@iium.edu.my

Special Lecture

Knowing the Unknown: Parkinson's Hidden Face

Abstract:

Despite tremendous progress in Parkinson's disease (PD) research, the underlying mechanisms driving its onset, heterogeneity, and progression remain only partially understood. The lack of disease-modifying therapies and limited insights into early molecular events continue to challenge the development of effective interventions. Traditional animal models, while invaluable, often fail to capture the full complexity of human PD pathology and its gene–environment interactions. To address these limitations, the zebrafish (ZF) has emerged as a powerful and complementary model for exploring PD's "hidden face. "The ZF offers unique advantages including optical transparency, rapid development, and conserved dopaminergic pathways comparable to humans. Its suitability for genetic manipulation, live imaging, and high-throughput screening makes it an exceptional platform for investigating neurodegenerative processes in real time. Using the ZF model, researchers can visualize dopaminergic neuronal loss, assess mitochondrial dysfunction, and study oxidative stress mechanisms underlying PD. Furthermore, ZF allows evaluation of environmental toxins, gene mutations, and potential neuroprotective compounds with unparalleled precision and efficiency. Dr. Wael Mohamed's work leverages the zebrafish model to uncover early pathogenic events and identify neuroprotective strategies that align with Healthy Longevity Medicine and Precision Geromedicine. His studies link molecular mechanisms to behavioral outcomes, bridging basic and translational neuroscience. By combining multi-omics, environmental analysis, and advanced imaging in zebrafish, his research aims to illuminate the unseen aspects of PD and open new therapeutic avenues. Unveiling this "hidden face" holds promise for transforming PD management and improving brain health across the lifespan.

Keywords: ZF, PD, Gero-science, Behavioral

Dr. Wael Mohamed is a physician neuroscientist at the International Islamic University Malaysia (IIUM), where he serves as Associate Professor in the Department of Basic Medical Sciences. His research focuses on the neurobiology of neurodegenerative disorders, particularly Parkinson's and Alzheimer's diseases, with an emphasis on how genetic, molecular, and environmental factors interact to influence disease onset and progression. Dr. Mohamed is the Founder and Chair of the AfrAbia Parkinson's Disease Genomic Consortium (APDGC), a collaborative initiative that unites researchers across Africa, and the Middle East, to explore the genetic diversity of PD in underrepresented populations. His work bridges neurogenetics, toxicology, and precision medicine, aiming to uncover novel biomarkers and therapeutic targets. He has also pioneered research using zebrafish model to investigate molecular pathways and environmental toxins implicated in PD, contributing to a deeper understanding of oxidative stress, mitochondrial dysfunction, and neuroprotection. Dr. Mohamed's studies on Edible Bird's Nest (EBN) and its neuroprotective effects have further advanced the integration of traditional bioactives into modern neuropharmacology. Beyond research, Dr. Mohamed is active in international neuroscience education and advocacy, serving in leadership programs under the American Academy of Neurology (AAN) and contributing to global initiatives on Healthy Longevity Medicine and Precision Geromedicine. His career reflects a dedication to advancing brain health through collaborative, cross-disciplinary, and culturally inclusive neuroscience that bridges basic science with translational outcomes.

Assoc. Prof. Vorasith Siripornpanich, M.D., Ph.D.

Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University

e-mail: vorasith.sir@mahidol.ac.th

Special Lecture

Mind at Rest, Brain in Motion: Sleep and Synaptic Plasticity

Abstract:

The human brain is a highly dynamic organ that constantly undergoes structural and functional modifications. It can reorganize its networks in response to appropriate and continuous stimulation. At the synaptic level, structural changes may occur within a single day. According to the synaptic homeostasis hypothesis, synaptic strengthening occurs during wakefulness following learning and experience, whereas synaptic downscaling takes place during sleep. These cyclic modifications form the foundation of many theories on the role of sleep in brain function, particularly in memory consolidation. The consolidation of long-term memories occurs mainly during N3 sleep, especially for declarative memory, through coordinated communication between cortical and hippocampal regions. Such bidirectional interactions lead to the establishment of long-term memory circuits and are consistent with the cortical synchronization characteristic of N3 sleep. In conclusion, adequate sleep enhances overall brain function and plays a critical role in regulating synaptic plasticity in accordance with experiences and learning accumulated during wakefulness.

Keywords: Brain plasticity, Sleep, Synaptic homeostasis

Dr.Vorasith Siripornpanich is an Associate Professor in Neuroscience and Medical Specialist in Pediatrics, Pediatric Neurology, and Sleep Medicine. He currently serves as the Head of the Research Center for Neuroscience, Institute of Molecular Biosciences (MB), Mahidol University. His laboratories include the Brain Electrophysiology Laboratory, which focuses on the use of EEG and event-related potentials (ERPs) as research tools for studying human cognition, and MB Hypnos, dedicated to sleep research. His research interests involve investigating factors that promote human cognitive function, as well as exploring cellular and molecular mechanisms underlying neurological and psychiatric disorders. Beyond his role as a neuroscientist, he also serves as a medical consultant for several government and private hospitals in Bangkok.

Ranchana Yeewa, PhD

Center of Multidisciplinary Technology for Advanced Medicine (CMUTEAM), Faculty of Medicine, Chiang Mai University

e-mail: ranchana.y@cmu.ac.th

Special Lecture

Epigenetic–Metabolic Crossroads in Neuroplasticity and Brain Aging

Abstract:
Epigenetic and metabolic networks collaborate to regulate neuronal plasticity and resilience, yet their interconnection remains an emerging area of understanding in brain aging. Histone acetylation, a dynamic epigenetic mark that responds to cellular metabolism, integrates signals from energy flux and nutrient levels to fine-tune transcriptional programs vital for synaptic function and neuronal survival. In this study, we investigated how epigenetic readers of histone acetylation associate chromatin states with metabolic pathways that impact neuroplasticity and longevity. Using *Drosophila melanogaster* as a genetically tractable model, we demonstrated that suppression of YEATS-domain containing acetylation readers – ENL/AF9 homologs – extended lifespan, preserved locomotor ability, and maintained better sleep in aged individuals. Integrative transcriptomic, metabolomic and lipidomics analyses revealed coordinated alterations of pathways involved in fatty acid oxidation, pyruvate metabolism, and amino acid metabolism, many of which converge on the tricarboxylic acid cycle. These changes were accompanied by enhanced tolerance to oxidative stress in aging flies, implying adaptive neuroprotection through coordinated epigenetic–metabolic regulation. Collectively, these findings identify histone acetylation readers as key molecular nodes linking chromatin states with energy metabolism and reveal potential mechanisms by which epigenetic modulation could reshape metabolic cues to sustain neuronal resilience. Targeting these YEATS family proteins may offer new therapeutic opportunities to maintain neuroplasticity and promote healthy brain aging.

Keywords: Aging, Metabolism, Epigenetics, Omics, *Drosophila melanogaster*

Dr. Ranchana Yeewa is a researcher affiliated with the Faculty of Medicine, Chiang Mai University, Thailand. Her work focuses on functional genetics, epigenetics, and multi-omics approaches using *Drosophila melanogaster* as a model organism. She mainly contributes to projects exploring how epigenetic regulators – particularly histone acetylation readers such as YEATS-domain proteins (ENL/AF9) – influence metabolism, neuronal health, and aging. Currently, her research attempts to integrate transcriptomics, metabolomics, lipidomics, and proteomics to uncover mechanisms linking chromatin regulation with metabolic adaptation and longevity.

Ha Thi Thanh Huong, PhD

School of Biomedical Engineering,
International University,
Vietnam National University Ho Chi Minh City

htthuong@gmail.com

Special Lecture

Comprehensive AI in Alzheimer's: From Early Diagnosis to Progression Insights Across Multiple Modalities

Abstract:

Alzheimer's disease demands approaches that connect early detection and progression modeling, across data types. Our group integrates artificial intelligence with imaging, molecular, behavioral, and speech signals to address AD and MCI. For diagnostic modeling, an ensemble combining deep learning and traditional machine learning classifies AD, MCI, and cognitively normal subjects with explainability, achieving an AUC of 96% on ADNI data. We also systematically compared three computational paradigms on ADNI data: statistical methods using FreeSurfer-derived features achieved 91% accuracy, region-based ResNet18 reached 73-74% accuracy, and sequence-based DeiT achieved 76% accuracy. To support clinical feasibility, a cloud-based, serverless system with Neural Architecture Search automates MRI processing and multi-class diagnosis at low cost, achieving 86.7% accuracy in classifying CN, MCI, and AD. We are also incorporating voice and linguistic features to enhance AI-based detection of AD and MCI, developing automatic speech-recognition and feature-based classification models that achieved 93.7% accuracy. To characterize disease progression, clustering analyses integrating MRI, plasma proteins, and cognitive measures reveal heterogeneous MCI subtypes and prognostic biomarkers. Our ongoing work focuses on two areas: using p-Tau, genetic, and MRI data to investigate molecular imaging relationships, and integrating OCT, fundus, and MRI for eye and brain research in Alzheimer's disease. Together, these projects form a comprehensive AI program that connects detection and prediction across modalities, advancing precision and accessibility in Alzheimer's care in Vietnam.

Keywords: Alzheimer's disease, Multimodal data integration, EEG analysis, Disease progression prediction

Dr. Ha Thi Thanh Huong is a Lecturer at the School of Biomedical Engineering, International University – Vietnam National University Ho Chi Minh City, where she chairs the Department of Tissue Engineering and Biomedical Engineering and directs the Brain Health Laboratory. She is a member of the IBRO Early Career Committee and serves on the National Academies' Workshop Planning Committee on Engaging Scientists in Shared Responsible Innovation in Neuroscience in Southeast Asia. Her research focuses on applying artificial intelligence and neuroengineering approaches to address Alzheimer's disease and mental health challenges in Vietnam. Her work encompasses neuroimaging analysis, EEG-based interventions, molecular biomarker development, and brain-computer interfaces. Dr. Ha has received numerous recognitions including the L'Oréal-UNESCO For Women in Science Award (2022), Vietnam's Golden Globe Science and

Technology Award (2023), Women of the Future Awards Southeast Asia (2023), and the IBRO Early Career Award (2020). She was honored as an Outstanding Young Citizen of Ho Chi Minh City (2023) and received the National Outstanding Teacher Award (2023). She has published over 30 peer-reviewed papers in international journals including Frontiers in Molecular Neuroscience, IBRO Neuroscience Reports, and Brain Multiphysics, and has presented at numerous conferences including the Asian Society Against Dementia Congress and IBRO symposia.

Prof. Sutisa Nudmamud-Thanoi, PhD

Department of Anatomy, Faculty of Medical Science and Centre of Excellence in Medical Biotechnology, Naresuan University, Thailand

e-mail: sutisat@nu.ac.th

Special Lecture

Proteomic Insights into Cognitive Performance: Age-Dependent Sex Differences in Healthy Thai Subjects

Abstract:

Sex differences in cognitive function arise from complex interactions among genetic, hormonal, and environmental factors. These differences are not static but evolve across the lifespan, reflecting age-related shifts in neural plasticity and neurotransmission. Despite growing interest, the proteomic associations of age-dependent cognitive sex differences remain insufficiently characterized. To address these gaps, we aimed to examine age-dependent sex differences in cognitive performance in healthy Thai subjects using the Wisconsin Card Sorting Test (WCST) and to identify associated molecular pathways through serum-based proteomic and bioinformatic analyses. Sex differences in cognitive performance were observed in the WCST, specifically in the percentage of total errors, a measure of non-specific cognitive impairment, with males outperforming females. This difference was age-dependent, becoming more pronounced in participants over 60 years of age. Proteomic analysis revealed sex-specific expression of individual proteins and protein complexes associated with potential N-methyl-D-aspartate receptor (NMDAR)– mediated excitotoxicity, with the NMDAR complex being enriched exclusively in elderly female samples, corresponding to their cognitive performance. Education level showed a modulatory effect, with sex differences evident in the primary education group but attenuated in higher education groups, with a cholinergic–estrogen interaction proposed as a potential underlying mechanism. These findings provide preliminary evidence that healthy Thai females may be more susceptible to NMDAR-mediated neurotoxicity, with serum enrichment of the NMDAR protein complex emerging as a potential indicator of cognitive aging in healthy Thai females. The observed influence of education points to a possible interaction between environmental context and sex-specific neurobiological mechanisms shaping cognitive outcomes.

Keywords: Sex difference, cognition, aging, proteomics, Wisconsin Card Sorting Test (WCST)

Sutisa Thanoi (**Sutisa Nudmamud-Thanoi)** is a Professor of Anatomy in the Faculty of Medical Science at Naresuan University. She is now also working as a director of the Excellence Centre in Medical Biotechnology at Naresuan University. She has been appointed a member of the Expert Committee on Drug Dependence (ECDD), World Health Organization (WHO), since 2017. Currently, Sutisa is the president of the Anatomy Association of Thailand (AAT) and the vice-president of the Thai Neuroscience Society

(TNS). Sutisa completed her PhD in Neuroscience at the Department of Biomedical Science, Sheffield University, UK. Her research interests focus on neurobiology in psychiatric diseases (such as schizophrenia, bipolar disorder and major depressive disorder) and drug dependence (including methamphetamine, ecstasy, pseudoephedrine, and dextromethorphan). She has also researched the potential therapeutics of natural products for brain health in both animal and human studies. Apart from the alternative treatment study for psychiatric disorders, she has also researched the genetic variation studies in psychiatric disorders, such as major depressive disorder or drug addiction. She has received several research grants funded by national and international organisations. She was awarded funding from the International Brain Research Organization (IBRO) for organising Associate Schools of Neuroscience in 2016 and 2023. The main objective of these meetings was to provide a platform for postgraduate students to expand their knowledge of neuroscience. She has now established the Frontier Research Cluster in Brain Health at Naresuan University, aiming *to* advance research on the mechanisms of psychiatric neurological disorders and to explore the therapeutic potential of natural products through in vitro, in vivo, and human studies.

Catleya Rojviriya, PhD

Synchrotron Light Research Institute (Public Organization)

catleya@slri.or.th

Special Lecture

Super High-Resolution Tomography Imaging in Neuroscience

Abstract

Understanding how billions of neurons form complex networks remains a central challenge in neuroscience. The human brain connectome, composed of vast neuronal pathways and synaptic connections, underlies all cognitive and behavioral functions. High-resolution mapping of these networks is therefore vital for advancing studies of brain organization, development, and disease. This work demonstrates the application of synchrotron-based X-ray tomographic microscopy (XTM) for non-destructive, three-dimensional imaging of brain tissue at sub-micron resolution. Utilizing the Siam Photon Source at the Synchrotron Light Research Institute, imaging conditions were optimized to visualize fine neuronal architectures – including cell bodies, dendrites, and axonal projections – within intact brain tissue blocks. The system achieves 0.5 μm spatial resolution with rapid acquisition and excellent contrast, eliminating the need for serial slicing or heavy staining.This approach bridges the gap between light and electron microscopy, providing a powerful tool for mesoscale connectome mapping. By integrating synchrotron tomography with neuroanatomical and computational analyses, it enables high-fidelity 3D reconstruction of neuronal networks. The development of this imaging capability establishes a foundation for Thailand's first high-resolution brain imaging platform, strengthening international collaborations and supporting future advances in brain connectivity research, diagnostics, and neurotechnology.

Keywords: Brain Connectome; Synchrotron X-ray Tomography; High-Resolution Neuroimaging; Neuronal Microstructure; Non-Destructive 3D Imaging

Dr. Catleya Rojviriya is the Head Scientist of the Tomography and Imaging Section at the Synchrotron Light Research Institute (SLRI), Thailand. Her research focuses on applying synchrotron-based X-ray tomography to achieve high-resolution, three-dimensional imaging for neuroscience applications. With expertise in advanced imaging technologies, she leads national efforts to enhance Thailand's capability in brain microstructure visualization and connectome mapping. Her current project employs upgraded synchrotron beamline systems to enable non-destructive, sub-micron imaging of brain tissue, bridging a critical gap in Thailand's neuroimaging landscape. In collaboration with international partners, including the Shanghai Advanced Research Institute, her work contributes to the development of advanced tools for microscale brain mapping, neurodiagnostics, and cognitive science research.

Prof. Banthit Chetsawang, PhD

Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Salaya, Nakhon Pathom 73170, Thailand

banthit.che@mahidol.ac.th

Special Lecture

The Regulation of Kynurenine Pathway: A Potential Targeting to Prevent Neurodegeneration in Methamphetamine-Induced Toxicity Model

Abstract

Methamphetamine (METH) is a psychostimulant drug whose effects on the toxicity of mammalian brains have been continually reported to induce neurotoxicity. Chronic or excessive use of METH has a profound potential for cognitive impairment and deterioration of brain function. For instance, prolonged use of methamphetamine causes dopamine accumulation in the brain, leading to dopamine auto-oxidation, which generates reactive oxygen species (ROS) and free radicals, resulting in mitochondrial damage and oxidative stress. Moreover, several findings consistently demonstrate that METH contributes to brain inflammation by persisting microglial activation. The activation of microglial cells was observed, resulting in neuroinflammation and an increase in the production of pro-inflammatory cytokines. Our recent study found elevated levels of inflammatory agents in the blood of long-term methamphetamine users, which correlated with cognitive impairments. However, the underlying mechanisms by which elevated inflammatory agents induce neuronal degeneration and cognitive decline remain unclear. Several studies in animal models and human patients suggest that inflammation-associated brain dysfunction may involve increased tryptophan metabolism. Tryptophan is converted to kynurenine (KYN) by the enzyme indoleamine-2,3-dioxygenase-1 (IDO1). Elevated KYN levels have been associated with neurodegenerative disorders such as Alzheimer's disease (AD), highlighting the importance of investigating IDO1 activity. Our current study aims to identify molecules or compounds that can inhibit IDO1 activity, providing critical insights into the pathways by which neuroinflammation contributes to neuronal degeneration and subsequent cognitive impairments. Moreover, such knowledge could support the development of novel therapeutic agents that mitigate inflammation-induced brain dysfunction and neurodegeneration

Keywords: Neurodegeneration, Kynurenine Pathway, Methamphetamine, Neuroinflammation, Indoleamine-2,3-dioxygenase-1 (IDO1)

Prof. Dr. Banthit Chetsawang is a professor in neuroscience at Mahidol University. He has been a faculty member of the graduate program in neuroscience (an international program) for over 35 years. The main area of his research interests focuses on neurodegeneration, neuroprotection, and neurorestoration, particularly the death and survival signaling in neuronal cells, as well as the protective roles of melatonin in neurodegeneration. Moreover, the neurotoxic effects of drug abuse, such as methamphetamine, on neurodegeneration, neuroinflammation, and cognitive impairment are also the aim of his study. His research is conducted in cellular models, animal models, and humans to study the mechanisms underlying neurodegeneration, ranging from the molecular level to neurobehavioral processes. His current research focuses on novel drug discovery and the development of therapeutic agents or chemical compounds that mitigate inflammation-induced brain dysfunction and neurodegeneration.

Prof. Toshihide Yamashita, MD, PhD

Department of Molecular Neuroscience, Graduate School of Medicine, The University of Osaka

e-mail: yamashita@molneu.med.osaka-u.ac.jp

Special Lecture

Development of therapeutic strategies to repair neuronal network for the central nervous system diseases

Abstract

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

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

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

Keywords: axon regeneration, immune regulation, neurodegenerative disease, spinal cord injurys

In the field of central nervous regeneration inhibition signaling, **Yamashita** has discovered novel regeneration inhibitors, identified their receptors, elucidated intracellular key signals, and successfully repaired damaged axons *in vivo*. These basic research results have had a great impact by answering the question of why the central nervous circuits are difficult to repair once damaged. Regarding his achievements thus far, Yamashita has received Ameritec Prize (US), for the first time in Japan in 2005. The prize is awarded to researchers in the field of nerve regeneration. He also received the Japan Society for the Promotion of Science Award in 2010, Osaka Science Award in 2011, and the Science and Technology Award from the Ministry of Education, Culture, Sports, Science and Technology in 2014. A drug developed by Yamashita based on the basic research results is currently undergoing clinical trials in Japan, the United States and Canada.

Assoc. Prof. Hideki Yoshida, PhD

Department of Applied Biology, Kyoto Institute of technology

e-mail: hyoshida@kit.ac.jp

Special Lecture

Diversity of molecular mechanisms underlying mRNA localization

Abstract

mRNA localization followed by local translation is a widely conserved mechanism from bacteria to humans for providing enough proteins at sites where they are required in the cells, which is indispensable for crucial biological processes, including neurological events. The general model for mRNA localization mechanism shows that an RNA-binding protein binds to the secondary structure in the 3' untranslated region of mRNA, and the complex is transported along the cytoskeleton by a motor protein. However, the model was suggested based on limited data, and as research into various localized mRNA has been performed, several mechanisms distinct from the proposed model have emerged.

In the early *Drosophila* embryos, it had been reported that a large number of localized mRNAs show a wide variety of localization patterns. We have revealed the non-canonical localization mechanism by investigating the molecular mechanism of the RNAs showing the same localization pattern.

Keywords: mRNA localization, *Drosophila*, nascent polypeptide chain, polyproline

Dr. Hideki Yoshida is an Associate Professor in Department of Applied Biology, Kyoto Institute of Technology. He has been studying mRNA localization underlying local translation related to synaptic plasticity using Drosophila melanogaster. He has published in over 90 international journals, including Cell.

Nithi Asavapanumas, MD, PhD

Chakri Naruebodindra Medical Institute, Faculty of Medicine
Ramathibodi Hospital, Mahidol University

e-mail: nithi.asa@mahidol.ac.th

Special Lecture

Induced Pluripotent Stem Cells (iPSCs)-Derived Brain Models in the Study of Neurodevelopment and Neurodegeneration

Abstract

Neurodegenerative disorders are characterized by progressive neuronal loss driven by multiple cellular dysfunctions, including lysosomal impairment, mitochondrial dysfunction, and disrupted protein homeostasis. Gaucher disease is a lysosomal storage disorder caused by mutations in the *GBA1* gene, resulting in a deficiency of the enzyme glucocerebrosidase (GCase) and accumulation of glucosylceramide within lysosomes. In the neuronopathic forms of Gaucher disease, this lysosomal dysfunction disrupts neuronal homeostasis, impairs autophagy, and contributes to synaptic degeneration and neuroinflammation. Moreover, *GBA1* mutations are recognized as the most common genetic risk factor for Parkinson's disease, linking lysosomal dysfunction in Gaucher disease to α-synuclein aggregation and dopaminergic neurodegeneration. Although pathological features have been identified in postmortem brain tissue, understanding the disease mechanisms and developing effective therapeutic strategies remains limited due to the inaccessibility of live human neurons. To overcome this challenge, our group utilizes patient-derived induced pluripotent stem cells (iPSCs) as a translational platform, enabling the generation of disease-relevant neural cell types with patient-specific genetic backgrounds. The iPSCs generated from the peripheral blood of patients with Gaucher disease were differentiated into neuronal cells and compared with those from healthy controls. Using advanced live-cell imaging and single-cell transcriptomic profiling, we observed increased lysosomal vesicle accumulation, reduced lysosomal motility, enhanced autophagic activity, and disturbed calcium homeostasis in Gaucher iPSC-derived neurons. These findings demonstrate that iPSC-based models effectively recapitulate patient-specific neuropathological phenotypes, providing a powerful system for dissecting disease mechanisms and evaluating targeted therapeutic strategies in lysosomal storage disorders and related neurodegenerative diseases.

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

Dr. Nithi Asavapanumas is a lecturer at the Chakri Naruebodindra Medical Institute, Faculty of Medicine Ramathibodi Hospital, Mahidol University, Thailand. His research focuses on translational neuroscience, specifically disease modeling using patient-derived induced pluripotent stem cells (iPSCs) to investigate lysosomal and mitochondrial dysfunction in neurodegenerative disorders such as Gaucher disease. He has extensive experience in cellular and molecular neurobiology, lysosomal biology, and mechanisms related to neurodegeneration, employing advanced live-cell imaging techniques to study dynamic cellular processes in real time.

Hiranya Pintana, PhD

Neurophysiology Unit, Cardiac Electrophysiology Research and Training Center, Faculty of Medicine, Chiang Mai University, Thailand

hiranya.pintana@gmail.com,
hiranya.pintana@cmu.ac.th

Special Lecture

The Impact of 5-Alpha-Reductase Inhibition on Anxiety- and Depression-Like Behaviors in Obese Insulin Resistant or Early Senescent Male Rats

Abstract

Finasteride (FIN), a 5-alpha reductase inhibitor (5-ARI), is commonly used to treat benign prostatic hyperplasia and androgenic alopecia, which has been linked to depression or anxiety in users. The impacts of FIN on anxiety/depression-like behaviors in aging or obese-conditions remain unclear. This study aims to explore whether FIN exacerbates or alleviates anxiety- and depression-like behaviors associated with metabolic disturbances, inflammation and oxidative stress, in obese or D-galactose-induced senescent rats.

In this study, fifty-six males Wistar, were fed either a normal (ND; n=40) or high-fat diet (HD; n=16) for 18 weeks. Sixteen ND-fed rats were treated with D-galactose (150 mg/kg/day; subcutaneously) for 18 weeks. At 13 weeks, rats in each group were divided to receive either drinking water as control or FIN orally at 5 mg/kg/day for 6 weeks. At 18 weeks, anxiety/depression-like behaviors were determined. Blood was collected for biochemical analysis. After euthanasia, the brains were removed to examine brain inflammation, oxidative stress, brain metabolites, and microglial complexity.

We observed that both obese-rats and D-galactose-treated rats exhibited metabolic disturbance, increased inflammation, oxidative stress, neurotoxic metabolites, decreased microglial complexity, and worsening anxiety/depression-like behaviors. In obese rats, FIN alleviated depression-like behavior but did not improve metabolic disturbances or anxiety-like behavior. In D-galactose-treated rats, FIN improved metabolic parameters, anxiety- and depression-like behaviors. Across both models, FIN reduced inflammation, oxidative stress, and neurotoxic metabolites, while enhancing microglial complexity.

Conclusion, FIN reduces depression and brain pathology in both models. This study highlights FIN's potential therapeutic effects on depression in these models.

Keywords: Finasteride; Anxiety; Depression; Ageing; Obesity

Dr. Hiranya Pintana is a researcher at the neurophysiology unit, Cardiac Electrophysiology Research and Training (CERT) Center, Faculty of Medicine, Chiang Mai University, Thailand. My research focuses on the association between metabolic impairment and neurological complications including cognitive dysfunction and mood disorders using western blot, immunohistochemistry, ex vivo electrophysiology (extracellular recordings in brain slices), and cognitive/behavioral testing in rodents. I also evaluate therapeutic strategies, including hormone-replacement therapies and antidiabetic agents to mitigate metabolic disturbances and associated neurobehavioral deficits. Currently, I have been investigating how metabolic dysregulation drives anxiety and depression in the contexts of obesity, aging, and altered male reproductive hormones, and evaluates the effects of 5-α-reductase inhibitors (5-ARIs) and cell-death pathway inhibitors in these settings.

Salinee Jantrapirom, PhD

Department of Pharmacology, Faculty of Medicine,
Chiang Mai University

e-mail: salinee.jan@cmu.ac.th

Special Lecture

UBQLN2-Associated Neurodegeneration: Molecular Mechanisms and Therapeutic Perspectives

Abstract

Ubiquilin-2 (UBQLN2) coordinates protein and RNA quality control in neurons, and its dysfunction contributes to a broad spectrum of neurodegenerative proteinopathies. Although UBQLN2 mutations are found in a subset of amyotrophic lateral sclerosis–frontotemporal dementia (ALS–FTD) cases, UBQLN2-related pathology is frequently observed in the absence of coding variants, underscoring dosage- and context-dependent vulnerabilities. Here, we evaluate a proteostasis-centered therapeutic strategy that enhances protein degradation by mitigating cellular stress. Because endoplasmic reticulum (ER) stress is a hallmark of UBQLN2 dysfunction, we targeted an ER-resident regulator of the unfolded protein response. Across complementary cellular and animal models of UBQLN2 dysfunctions, this intervention attenuated ER-stress signaling, improved neuronal viability, and ameliorated neurobehavioral deficits associated with UBQLN2 dysfunction. These findings demonstrate that relieving ER stress can rebalance proteostasis and modify disease-relevant phenotypes in UBQLN2-associated neurodegeneration.

Keywords: Ubiquilin-2, proteostasis, ER-stress, neurodegeneration

Dr. Salinee Jantrapirom is a Lecturer in the Department of Pharmacology, Faculty of Medicine, Chiang Mai University. She earned her degree in Biotechnology from the Kyoto Institute of Technology, where she began applying Drosophila melanogaster to biomedical research. A pioneer in building Drosophila research capacity in Thailand, she uses transgenic fly models to dissect mechanisms of human neurological disease and accelerate therapeutic discovery. Her research focuses on restoring neuronal proteostasis and strengthening cellular defenses against chronic stress, with the long-term goal of translating mechanistic insights into disease-modifying interventions for currently incurable neurological disorders.

Assoc. Prof. Narawut Pakaprot, MD, PhD

Department of Physiology
2 Srisavarindhira Brd. 13th Floor, Wanglang Road, Siriraj Subdistrict, Bangkoknoi District, Bangkok, Thailand 10700

e-mail: narawut.pak@mahidol.ac.th

Special Lecture

In Search for the Molecular Target of *Centella Asiatica* Extract

Abstract

Centella asiatica (L.) Urban, a medicinal plant known for its neuroprotective and memory-enhancing properties, has been widely used in traditional medicine. Yet, its precise molecular targets remain incompletely characterized. Our integrative research investigates the mechanisms by which the standardized extract ECa 233 and its principal triterpenoid glycosides – madecassoside (MDS) and asiaticoside (ASS) – enhance neuronal and synaptic functions. Behavioral and electrophysiological studies in rodents revealed that ECa 233 facilitates hippocampal long-term potentiation (LTP) and improves learning and memory performance, associated with the upregulation of synaptic plasticity–related genes including BDNF, CREB, and TrkB. Complementary proteomic and mitochondrial analyses demonstrated that both ECa 233 and the MDS + ASS mixture increased mitochondrial respiration, ATP production, and the expression of synaptic proteins such as synaptophysin and SNAP25. Pathway enrichment analyses identified the Akt–mTOR cascade as a central signaling axis mediating these effects, linking enhanced mitochondrial metabolism to synaptic plasticity. Interestingly, while the MDS + ASS mixture produced stronger mitochondrial responses, ECa 233 elicited greater hippocampal synaptic activation, suggesting that minor constituents in the extract synergize with the major triterpenoids to optimize neurobiological efficacy. Together, these findings indicate that C. asiatica exerts its cognitive-enhancing effects by targeting convergent molecular pathways regulating mitochondrial bioenergetics, protein synthesis, and synaptic remodeling. This mechanistic understanding supports the potential of C. asiatica–derived compounds as candidates for the prevention and treatment of cognitive impairment and neurodegenerative disorders.

Keywords: Centella asiatica, learning and memory, synaptic plasticity, proteomic study, mitochondrial function

Dr. Narawut Pakaprot is a neuroscientist and medical doctor specializing in neurophysiology. He currently serves as a faculty member in the Department of Physiology, Faculty of Medicine Siriraj Hospital, Mahidol University, Thailand. His research focuses on the cellular and molecular mechanisms underlying learning, memory, and cognitive plasticity, with particular interest in the electrophysiological and molecular correlates of synaptic function. Dr. Pakaprot leads a multidisciplinary laboratory that integrates electrophysiology, proteomics, and behavioral neuroscience to investigate natural compounds with neuroprotective potential, notably Centella asiatica extract (ECa 233) and its active triterpenoid constituents. His recent work, published in Scientific

Reports (2025), elucidated the role of mitochondrial bioenergetics and Akt–mTOR signaling in mediating the synaptic effects of ECa 233, providing a mechanistic framework for its cognitive-enhancing properties. His long-term goal is to bridge molecular neurobiology with translational medicine to advance therapeutic strategies for neurodegenerative and cognitive disorders.

Prof. Ishwar Parhar, PhD

Center Initiative for Training International Researchers (CITIR), University of Toyama, Japan

e-mail: ishwar@ctg.u-toyama.ac.jp

Special Lecture

Neural Plasticity in Reproduction and Social Behaviors

Abstract

Neural plasticity—the brain's ability to reorganize its structure, function, and connections—plays a vital role in regulating reproductive and social behaviors across vertebrates. Hormonal signals such as testosterone and estrogen interact with neural circuits involving the olfactory system, preoptic area, habenula, and raphe nucleus to mediate adaptive changes in mating, dominance, and emotion. The olfactory system, via pheromonal cues, influences hypothalamic GnRH neurons and limbic circuits essential for partner recognition and reproductive readiness. The preoptic area serves as a central hub where kisspeptin neurons activate gonadotropin-releasing hormone (GnRH) secretion, initiating reproductive cascades. Conversely, gonadotropin-inhibitory hormone (GnIH, in non-mammalian vertebrates) suppresses this axis during stress or social subordination, maintaining synchronization between reproductive activity and environmental cues. The habenula links limbic emotional centers to midbrain monoaminergic nuclei and regulates aversive learning, fear, and dominance through modulation of serotonin release from the raphe nucleus. Heightened habenular activity during social defeat and fear alters serotonergic tone, reshaping affiliative behaviors. Concurrently, serotonin from the raphe nucleus helps maintain emotional stability and facilitates adaptive social decision-making. Collectively, these interconnected systems demonstrate remarkable plasticity, allowing organisms to fine-tune reproductive and social behaviors in response to internal states and external stimuli. By linking sensory input, hormonal feedback, and neural remodeling within the habenula–preoptic–raphe axis, the brain dynamically adapts behavior to environmental and social cues, demonstrating the neurobiological basis of fertility, emotion, and social adaptation across species.

Keywords: neural plasticity; reproduction; social behavior; kisspeptin; habenula

Prof. Ishwar S. Parhar is currently a Distinguished Professor and Chief Director of the Center Initiative for Training International Researchers (CITIR) at the University of Toyama, Japan. He obtained his Ph.D. in Neuroendocrinology from the National University of Singapore and completed postdoctoral training at The Rockefeller University, New York. A distinguished neuroendocrinologist with an academic career spanning over four decades across Malaysia and Japan, he previously served as Professor of Neuroscience and Founding Director of the Brain Research Institute, Monash University Malaysia (BRIMS). Prof. Parhar serves as President of the Asian Oceania Society for Comparative Endocrinology (AOSCE) and as an Executive Member of the International Federation of Comparative Endocrinology Societies (IFCES). His outstanding contributions to science have been recognized through numerous awards, including the Top Scientist Award from the Malaysian Government and the Hind Rattan Award from India. He has delivered many keynotes and plenary lectures, including the prestigious Sir John Monash Lecture, and serves on governmental scientific advisory boards and editorial boards of several leading journals. Prof. Parhar has published more than 270 peer-reviewed papers and book chapters in high-impact journals such as *Nature, PNAS, Molecular Psychiatry, Scientific Reports, and Biological Reviews.* His pioneering research on gonadotropin-releasing hormone (GnRH), kisspeptin, and gonadotropin-inhibitory hormone (GnIH) in non-mammalian vertebrates has profoundly advanced understanding of how neural circuits integrate hormonal, sensory, and social cues to regulate reproduction, social behavior, aversive memory, and aversive emotions.

Lalitta Suriya-Arunroj, Dr. rer. nat.

75/154 Sap Rd., Si Phraya, Bang Rak, Bangkok 10500

E-mail: Lalitta.s@chula.ac.th

Special Lecture

Cognition, Clocks, and Computation: Cognitive Decline and Epigenetic Aging in Monkeys - Bridging Computational and Translational Neuroscience

Abstract:

Aging is the leading risk factor for cognitive decline, yet translational barriers limit the development of effective human anti-aging therapeutics. Non-human primates (NHPs) provide a critical bridge between rodent and human studies due to their evolutionary proximity, similar lifespan trajectories, and complex cognition. They recapitulate human aging across behavioral, neurobiological, and molecular dimensions, offering a uniquely powerful model for elucidating the mechanisms underlying cognitive decline.

Recent advances in epigenetic clocks – biomarkers derived from DNA methylation patterns – have revolutionized aging research. These clocks accurately estimate biological age across primate species using unified mathematical models and offer cost-effective, time-efficient surrogate endpoints for evaluating interventions targeting healthspan and lifespan. When combined with cognitive and neurophysiological assessments, epigenetic clocks allow precise quantification of how molecular aging correlates with functional changes in cognition.

Computational models of cognition further link molecular and neural aging by describing how epigenetic alterations may disrupt value-based choice computations in prefrontal circuits. By merging behavioral, molecular, and computational data, this integrative framework aims to identify biomarkers predicting cognitive resilience or decline.

Ultimately, bridging cognition, clocks, and computation in NHPs advances translational neuroscience uncovering the mechanistic pathways through which molecular aging shapes the aging mind – laying the foundation for predictive diagnostics and targeted interventions to extend not only human lifespan, but *mindspan*.

Keywords: Non-human primates (NHPs), Epigenetic clocks, Cognitive decline, Computational neuroscience, Translational aging research

Dr. Lalitta Suriya-Arunroj, a lecturer affiliated at Research Affairs, Faculty of Medicine, Chulalongkorn University, Bangkok, specialized in cognitive neuroscience. My research currently focuses on the trajectories of cognitive development and decline across the lifespan. I have experience designing behavioral and cognitive paradigms for both humans and non-human primates, as well as conducting primate neuroelectrophysiological studies. I am particularly fascinated by the neural mechanisms underlying decision-making and how these processes become disrupted in aging or dysfunction.

Assist. Prof. Amornpun Sereemaspun, MD, PhD

Center of Excellence in Nanomedicine,
Department of Anatomy, Faculty of Medicine,
Chulalongkorn University, Bangkok, Thailand

E-mail: amornpun.s@chula.ac.th

Special Lecture

Emerging Trends of Microbiome-related products used for Gut-Brain Axis: Cutting-Edge Clinical Application

Abstract:

The concept of the gut-brain axis and its implications on mental health and overall well-being have been gaining increasing attention in recent years. An emerging trend in this field involves the utilization of microbiome-related products to modulate the gut microbiota and ultimately impact brain function. This knowledge delves into the cutting-edge clinical applications of such products, exploring their potential in revolutionizing the treatment of neurological and psychiatric disorders.

Drawing from recent advancements in neuroscience and microbiology, this presentation will delve into the intricate relationship between the gut microbiota and the central nervous system. Through a critical review of current research findings, we will shed light on the mechanisms through which microbiome-related products exert their effects on the gut-brain axis, influencing neuroinflammation, neurotransmitter levels, and synaptic plasticity.

Moreover, this presentation will discuss the clinical implications of these findings, highlighting the potential of microbiome-based interventions in managing conditions such as depression, anxiety, and neurodegenerative diseases. Presenter will give an example of the gut-brain axis in Alzheimer's disease treatment via probiotics in our recent publication. By elucidating the link between gut health and brain function, this presentation aims to pave the way for novel therapeutic strategies that target the microbiome-related products that are commercially available in this current era.

Keywords: Gut–brain axis, Microbiome-based therapy, Probiotics, Neuroinflammation

Assistant Professor Dr. Amornpun Sereemaspun is a faculty member in the Department of Anatomy, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand. His multidisciplinary research encompasses nanomedicine, molecular diagnostics, neuroscience, and regenerative medicine, with a particular emphasis on epigenetic biomarkers, nanoparticle-based therapeutics, and the gut–brain axis. His work focuses on translational toxicology at the cellular and genetic levels, integrating nanotechnology to elucidate disease mechanisms and therapeutic responses. Dr. Amornpun has published extensively in international peer-reviewed journals and actively promotes biomedical innovation through interdisciplinary collaboration. He also serves as Director of the Ph.D. Program in Biomedical Science (Interdisciplinary Program) at Chulalongkorn University, where he advances research training and fosters translational impact in biomedical sciences.

POSTER PRESENTATION

P-001:

LCN2/LCN2R SIGNALING IMPAIRS MITOCHONDRIAL BIOENERGETICS TO PROMOTE NEURONAL DEATH IN HEPATIC ENCEPHALOPATHY

Ching-Yi Tsai[1*]

[1]*Institute for Translational Research in Biomedicine, Kaohsiung Chang Gung Memorial Hospital, Kaohsiung 83301, Taiwan*
**Corresponding author. Email: cytsai@cgmh.org.tw*

Abstract

Background: Hepatic encephalopathy (HE) is a serious neuropsychiatric disorder caused by liver failure, often leading to irreversible brain dysfunction and poor prognosis. Our previous work demonstrated that neuronal death in the rostral ventrolateral medulla, a key neural substrate that maintains blood pressure and sympathetic vasomotor tone, leading to baroreflex dysregulation, is causally related to death in a mouse model of HE. Furthermore, mRNA analysis of astrocytes purified from mouse brainstem revealed a significant increase in lipocalin-2 (Lcn2) gene expression in experimental HE.

Aim(s): The present study was to delineate the role of astrocyte-secreted Lcn2 in the pathogenesis of HE.

Methods: In this study, an azoxymethane (AOM; 100 µg/g, i.p.)-induced acute liver failure model in C57BL/6 mice was used to mimic HE. Primary neuronal and astrocyte cultures prepared from postnatal day 1 mouse pups were employed for mechanistic in vitro studies.

Results: We demonstrated that Lcn2 receptors (Lcn2R) are present in neurons. Furthermore, JC-1 staining revealed a decrease in mitochondrial membrane potential during HE, accompanied by an increase in apoptotic cell death. Intracerebroventricular infusion of Lcn2-neutralizing antibody significantly prolonged the survival of HE mice and reduced apoptotic cell death in the brain. In vitro knockdown studies further confirmed that Lcn2 exerts neurotoxic effects through Lcn2R in neurons. Mechanistically, Lcn2 activates the MEK/ERK signaling cascade, leading to upregulation of pyruvate dehydrogenase kinases (PDK1 and PDK3), enhanced phosphorylation and inhibition of pyruvate dehydrogenase (PDH), and subsequent reduction in mitochondrial ATP production.

Conclusion: Our findings suggest that Lcn2/Lcn2R signaling contributes to HE-associated neuronal death by disrupting mitochondrial energy metabolism. Specifically, Lcn2 impairs PDH activity and mitochondrial ATP production via MEK/ERK-mediated PDK upregulation, identifying a novel astrocyte-to-neuron signaling pathway that drives fatal outcomes in HE.

Keywords: hepatic encephalopathy, Lcn2, MAPK pathway, pyruvate dehydrogenase, mitochondrial dysfunction

----

P-002:

4-METHYLUMBELLIFERONE REVERSES AGE-RELATED CHANGES IN NEUROPLASTICITY AND NEUROINFLAMMATION

Anda Cimpean[1,2], Jana Dubisova[1], Pavla Jendelova[1], James W. Fawcett[1,3], Jessica C.F. Kwok[1,4]

[1] *Institute of Experimental Medicine, Czech Academy of Science, Prague, Czechia*
[2] *Second Faculty of Medicine, Charles University, Prague, Czechia*
[3] *Department of Clinical Neurosciences, John Van Geest Centre for Brain Repair, University of Cambridge, Cambridge,*
United Kingdom
[4] *Faculty of Biological Sciences, University of Leeds, Leeds, United Kingdom*
**Corresponding author. Email: anda.cimpean@iem.cas.cz*

Abstract

Background: Aging is accompanied by a reduction in neural plasticity that substantially impacts quality of life. Two major factors contributing to this decline are the progressive accumulation of perineuronal nets (PNNs) and increased neuroinflammation. Disruption of PNNs is a recognized strategy to enhance plasticity, and 4-methylumbelliferone (4-MU), an FDA-approved drug that inhibits hyaluronan synthesis – the backbone of PNNs – has been proposed to disrupt PNNs and enhance plasticity and memory in a young rodent model. Furthermore, 4-MU is also well known for being an effective immune response regulator.

Aim(s): Here, we investigated the effects of oral 4-MU administration for six months in aged mice (20–22 months) to assess its safety, impact on PNNs, memory, and neuroinflammation.

Methods: Mice aged 14–16 months received chow supplemented with 5% (w/w) 4-MU (~6.5 mg/g/day) or standard chow for six months. Additionally, a group of 10-month-old mice was included as a young control to assess the effects of aging. At the end of the treatment, animals were weighed, blood collected, and behavioral tests assessed memory and motor function. Immunohistochemistry was performed to analyze PNNs, astrocytes, microglia, and peripheral immune cell infiltration in the somatosensory cortex, corpus callosum, and hippocampus.

Results: Qualitative intensity measurements revealed an age-related increase in PNNs, which was effectively reduced by 4-MU treatment to levels observed in 10-month-old mice, restoring youthful levels of recognition-memory performance. Moreover, aging-associated neuroinflammation, indicated by astrocytic and microglial activation and peripheral immune cell infiltration in the somatosensory cortex, corpus callosum, and hippocampus, was also normalized to young controls. Long-term oral 4-MU was well tolerated with no detectable serious side effects.

Conclusion: These findings indicate that 4-MU safely reduces both PNNs and neuroinflammaging in aged mice, two effects that act synergistically to enhance plasticity and improve recognition memory, supporting its potential as a treatment for age-related cognitive decline.

Keywords: 4-methylumbelliferone, perineuronal nets, neuroinflammaging, plasticity, aging

---

P-003:

PROBUCOL AND RIFAMPICIN AMELIORATE 3-NITROPROPIONIC ACID-INDUCED HUNTINGTON'S DISEASE BY DOWNREGULATING HMGB1/TLR4/NF-κB SIGNALING PATHWAY

Vishal Kumar[1], Puneet Kumar[1*]

[1]Department of Pharmacology, Central University of Punjab, Bathinda, Punjab, India, 151401
**Corresponding author. Email: punnubansal79@gmail.com*

Abstract:

Background: Huntington's disease (HD) is an inherited movement disorder. 3-nitropropionic acid (3-NP) is well known to induce HD in experimental animals. Probucol and rifampicin have anti-inflammatory and antioxidant effects.

Aim: The current study is designed to investigate the involvement of high mobility group box-1 (HMGB1) in neuroinflammation and the assessment of the neuroprotective potential of probucol and rifampicin against HD.

Methods: The present investigation employed either sex of Wistar rats (aged 3-6 months) weighing 180-220 gm. The 3-NP (10 mg/kg/i.p), probucol (10, 20, and 40 mg/kg p.o), and rifampicin (20, 40, and 80 mg/kg i.p) were administered for 21 days. The study was assessed in terms of percentage change in body weight, behavioral assessments including narrow beam walk (NBW), open field test (OFT), and rotarod activity, western blotting {HMGB1 and nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB)}, reverse transcription polymerase chain reaction (RT-PCR) (Bax, Bcl, and caspase-3), immunohistochemistry (IHC) pTLR4 (phosphorylated toll like receptor 4) and GFAP (glial fibrillary acidic protein), and apoptotic markers (caspase-3, Bax, and Bcl-2), respectively. Hematoxylin and eosin (H&E) staining was performed to investigate neuronal damage.

Results: Administration of probucol (20 and 40 mg/kg/p.o.) and rifampicin (40 and 80 mg/kg/i.p.) significantly ameliorates 3-NP (10 mg/kg/p.o.)-induced behavioral and biochemical alterations. Furthermore, probucol and rifampicin significantly ameliorate the 3-NP-induced increased level of inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), Interleukin 1-beta (IL-1β), and interleukin-6 (IL-6). The results of the Western blot analysis demonstrated that the probucol and rifampicin preferentially inhibit the activity of the HMGB1/TLR4/NF-κB axis. In addition, RT-PCR analysis showed that probucol and rifampicin substantially decreased the 3-NP-caused elevations in Bcl-2 and caspase-3 and Bax mRNA expression. Thus, probucol and rifampicin considerably reduce the brain neuronal damage caused by 3-NP.

Conclusion: Probucol (20 and 40 mg/kg/p.o.) and rifampicin (40 and 80 mg/kg/i.p.) provide neuroprotection via the inhibition of the HMGB1/TLR4/NF-κB axis, thereby suppressing the downstream HMGB1 signaling pathway. Hence, findings suggest that probucol and rifampicin may have the ability to mitigate HD-associated symptoms via the HMGB1/TLR4/NF-κB pathway.

Keywords: 3-nitropropionic acid, HMGB1/TLR4/NF-κB, probucol, rifampicin, neuroinflammation

---

P-004:

IMPLICATION OF COLORED RICE TREATMENT ON MICROGLIAL RESPONSE AND CHOLINERGIC DYSFUNCTION UNDER MILD CEREBRAL HYPOPERFUSION IN MICE

Danuyada Wattanaumadechakul[1,2], Chairat Turbpaiboon[1], Panapat Uawithya[3], Nanthanit Pholphana[4], Nuchanart Rangkadilok[4], Supin Chompoopong[1*]

[1]Department of Anatomy, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand 10700
[2]Faculty of Medicine, Praboromarajchanok Institute, Ministry of Public Health, Nonthaburi, Thailand 11000
[3]Department of Physiology, Faculty of Medicine Siriraj hospital, Mahidol University, Bangkok, Thailand 10700
[4]Laboratory of Pharmacology, Chulabhorn Research Institute, Bangkok, Thailand 10210
**Corresponding author. Email: supin.cho@mahidol.ac.th*

Abstract

Background: Cerebral hypoperfusion is a major risk factor for vascular cognitive impairment. Under ischemic conditions and environmental cues, microglia can shift between M1 pro-inflammatory and M2 anti-inflammatory phenotypes. Both phenotypes contribute to the pathogenesis of neurodegenerative disorder for maintaining tissue homeostasis. Colored rice varieties, such as black glutinous (BR) and Riceberry (RR), are rich in anthocyanins with reported neuroprotective properties.

Aim: To investigate whether supplementation with colored rice could mitigate cognitive impairment by modulating microglial activation and cholinergic function in a mouse model of mild cerebral hypoperfusion.

Methods: Sixty male mice received WR, RR or BR (12 mg/kg) via oral gavage for 15 days. After 10 days of treatment, mice were randomly assigned to either mild bilateral common carotid artery occlusion or sham surgery.

Results: BR reduced TNF-α and IL-1β while enhanced IL-10 more effectively than RR and WR. The qRT-PCR showed that arterial occlusion induced M1 mRNA (iNos, CD32, CCL2), whereas BR promoted M2 markers (CD206, Arg-1, YM-1) with elevated IL-10. Histopathology revealed reduced neuronal cell deaths in the nucleus basalis of Meynert (NBM), nucleus accumbens (NuAc) and hippocampus, correlating with improved memory in Morris water maze and trace fear conditioning tests in BR and RR groups. Both groups also decreased neuronal loss ($p < 0.05$), increased ERK1/2 ($p < 0.001$) related to long term memory potential and alleviated cholinergic dysfunction and white matter injury. These effects were evidenced by elevated ChAT expression in the NBM and NuAc, and increased myelin basic protein in the brain. Microglial activation (Iba1) was also reduced, particularly in BR group.

Conclusion: Colored rice exerts neuroprotective effects under mild cerebral hypoperfusion by modulating microglia phenotypes and cholinergic dysfunction. These findings suggest its potential as a dietary intervention against vascular cognitive decline.

Keywords: Neuroinflammation, Colored Rice, Microglia Activation, TNF-α, Vascular Cognitive Impairment

---

P-005:

TREATMENT WITH *CAULERPA LENTILLIFERA* ATTENUATED ALPHA-SYNUCLEIN ACCUMULATION IN MPP-INDUCED PARKINSON'S-LIKE MODEL

Darunee Rodma[1,5], Chairat Turbpaiboon[2], Rungsarit Sunan[1,5], Ladawan Khowawisetsut[3] Krai Meemon[4], Prasert Sobhon[4], Supin Chompoopong[2], Morakot Sroyraya[4*]

[1]*Molecular Medicine Graduate Program, Faculty of Science, Mahidol University, Bangkok, Thailand 10400.*
[2]*Department of Anatomy, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand 10700.*
[3]*Department of Parasitology, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand 10700.*
[4]*Department of Anatomy, Faculty of Science, Mahidol University, Bangkok, Thailand 10400.*
[5]*Division of Basic and Medical Sciences, Faculty of Allied Health Sciences, Pathumthani University, Pathum Thani, Thailand 12000.*
**Corresponding author. Email: morakot.sry@mahidol.edu*

Abstract

Background: Research has increasingly focused on identifying and treating the preclinical phase of Parkinson's disease (PD) using biomarkers such as alpha-synuclein. Although α-synuclein is primarily expressed in neuronal cells and located in the cytoplasm, it is also detectable in body fluids, including cerebrospinal fluid (CSF) and blood. *Caulerpa lentillifera* (sea grape) has been widely used in Thailand's pharmaceutical industry and healthcare products. Sea grape extracts in ethanol fraction (CLET) have been shown to possess a high antioxidant capacity and low toxicity.

Aim(s): This study aimed to determine whether CLET could decrease alpha synuclein in neuronal cells as a targeting therapy for PD.

Methods: Both *in vitro*, the SH-SY5Y DAergic neurons incubated with the neurotoxin, MPP+ and CLET, and *in vivo*, mice orally administered with 50 mg/kg CLET for 17 days and with MPTP daily (30 mg/kg, 12-hour intervals) during the last 7 days of treatment were studied.

Results: The impressive analysis with Western blot demonstrated that CLET treatment resulted in a significant reduction in the expression of alpha-synuclein in both models. *In vitro* study, CLET significantly reduced MPP+-mediated cell death and apoptosis in SH-SY5Y neuronal cells by MTT assay and alleviated the mitochondrial membrane potential (MMP) by JC-10 flow cytometry. CLET significantly elevated the nuclear translocation of Nrf2 and its downstream antioxidant genes, including Heme oxygenase 1 (HO-1) and Glutathione-S-Transferase (GST) and also restored glutathione levels. For *in vivo* study, CLET-treated mice showed significantly reduced loss of dopaminergic neurons in the substantia nigra. Moreover, it was revealed that CLET attenuated motor behavioral deficits by the open field, rotarod, and narrow beam tests.

Conclusion: CLET decreases alpha-synuclein aggregation and scavenges the MPP+/MPTP-induced toxicity by activating the Nrf2-ARE pathway.

Keywords: *Caulerpa lentillifera,* antioxidants, alpha synuclein, Nrf2, MPP+, MPTP

P-006:

HALYMENIA DURVILLEI EXTRACT MITIGATES NEUROINFLAMMATORY CYTOKINES IN VASCULAR COGNITIVE IMPAIRMENT MICE

Rungsarit Sunan[1,2], Chairat Turbpaiboon[3], Darunee Rodma[1,2], Ladawan Khowawisetsut[4], Morakot Sroyraya[5], Krai Meemon[5], Prasert Sobhon[5], Supin Chompoopong[3*]

[1]Molecular Medicine Graduate Program, Faculty of Science, Mahidol University, Bangkok 10400, Thailand.
[2]Faculty of Allied Health Sciences, Pathumthani University, Pathum Thani 12000, Thailand.
[3]Department of Anatomy, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok 10700, Thailand.
[4]Department of Parasitology, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok 10700, Thailand.
[5]Department of Anatomy, Faculty of Science, Mahidol University, Bangkok 10400, Thailand.
**Corresponding author. Email: supin.cho@mahidol.ac.th*

Abstract

Background: Marine red seaweeds have emerged as a rich source of anti-inflammatory compounds with neuroprotective potential. Among these, *Halymenia durvillei*, widely distributed in Southeast Asia including Thailand, has attracted increasing attention for its therapeutic potential. Previous studies have shown that the ethanol extract of *H.durvillei* (HDE) contains a high proportion of n-hexadecanoic acid, also known as palmitic acid (79.65%), as determined by GC-MS analysis. Palmitic acid has been reported to act as an anti-inflammatory agent by inhibiting phospholipase A2, a key initiator of the inflammatory process in neuronal cells.

Aim(s): To investigate whether ethanol extracts of *H.durvillei* (HDE) could mitigate neuroinflammatory cytokines in a mouse model of vascular cognitive impairment (VCI)

Methods: A cerebral hypoperfusion-induced ischemia and hypoxia was performed in the modified bilateral common carotid artery occlusion (mBCCAO) mouse model. Mice were treated with HDE (25 mg/kg, 28 days, oral gavage) or with pure oleanolic acid (OA) as a standard control (n = 9 per group).

Results: HDE showed no toxic or systemic effects, particularly in the liver and kidneys. In the mBCCAO mouse model, progressive loss of hippocampal CA1 and CA3 pyramidal neurons were observed, which were significantly attenuated by HDE, similar to pure oleanolic acid (OA), leading to improve cognitive performance in the Morris water maze test ($p < 0.001$). HDE attenuated neuroinflammation significantly by decreasing proinflammatory cytokines TNF-α and IL-1β and increasing anti-inflammatory IL-10, as determined by ELISA (all $p < 0.001$). Mechanistically, HDE exerted its effects by suppressing JNK signaling, NF-κB activation, iNOS expression, and NO production, as demonstrated by Western blot, RT-qPCR, and Griess assay ($p < 0.001$). Data were analyzed using one-way ANOVA followed by post hoc multiple comparison with Tukey's test.

Conclusion: HDE mitigates neuroinflammatory cytokines and ameliorates VCI in mice.

Keywords: Neuroinflammation, Red Seaweed, *Halymenia Durvillei*, Vascular Cognitive Impairment

---

P-007:

DEVELOPMENT OF A SELF-EMULSIFYING FORMULATION OF *HUPERZIA CARINATA* EXTRACT FOR IMPROVING HUPERZINE A SOLUBILITY AND EVALUATING EFFICACY IN AN MPTP-INDUCED PARKINSONIAN MURINE MODEL

Namfa Sermkaew[1,2], Nuttapon Songnaka[1,2], Wuttipong Marsraksa[3], Amit Jaisi[1], Yosita Yongyuen[1], Pannatorn Buntha[1], Nadiah Syafiqah Nor Azman[4], Juntratip Jomrit[1], and Phetcharat Boonruamkaew[1,*]

[1]*School of Pharmacy, Walailak University, Nakhon Si Thammarat, Thailand 80160*
[2]*Drug and Cosmetics Excellence Center, Walailak University, Nakhon Si Thammarat, Thailand 80160*
[3]*Faculty of Medicine, Vongchavalitkul University, Nakhon Ratchasima, Thailand 30000*
[4] *Faculty of Pharmacy, Lincoln University college, Selangor, Malaysia 47301*
**Corresponding author. Email: phetcharat.bo@wu.ac.th or pairwaka@gmail.com*

Abstract

Background: Huperzine A (HupA), a sesquiterpene alkaloid first isolated from *Huperzia serrata*, is used in China as an anti-Alzheimer's drug and as a dietary supplement in the United States. In Thailand, *Huperzia carinata* (*H. carinata*) represents the richest natural source of HupA (Fatima et al., 2025). Despite its promising therapeutic potential, HupA suffers from poor aqueous solubility and low oral bioavailability. Self-emulsifying drug-delivery systems (SEDDS) provide an effective strategy to overcome these limitations by forming stable oil-in-water emulsions within gastrointestinal fluids, thereby enhancing solubility and absorption (Gursoy and Benita, 2004). Although HupA exhibits antioxidant, anti-inflammatory, and acetylcholinesterase-inhibitory activities in Alzheimer's disease, its neuroprotective effects in Parkinson's disease (PD) – particularly when delivered via SEDDS – remain insufficiently characterized.

Aims: To develop a SEDDS incorporating *H. carinata* extract to enhance HupA solubility and to evaluate its therapeutic efficacy in a murine model of PD.

Methods: The SEDDS formulation was optimized through HupA solubility testing, excipient selection, droplet-size analysis, and *in vitro* HupA dissolution studies. The optimized formulation was evaluated in male C57BL/6 mice (3 months old; n = 8 per group; N = 72) with MPTP-induced Parkinsonian symptoms (20 mg/kg body weight, four injections at 2-h intervals). Motor coordination and balance were assessed using the rotarod test.

Results: Oleic acid, Cremophor® RH40, and propylene glycol (PG) were identified as optimal excipients, yielding the F4 formulation (among nine candidates) with droplet sizes < 300 nm, rapid emulsification, and clear nanoemulsion characteristics suitable for lymphatic absorption. *In vivo*, SEDDS containing *H. carinata* extract (0.75 and 1.00 μg/kg body weight) significantly improved motor performance compared with the unformulated extract + MPTP ($p < 0.01$) and vehicle + MPTP groups ($p < 0.001$) after seven days.

Conclusion: The *H. carinata*-based SEDDS exhibited superior emulsification and notable *in vivo* neurofunctional recovery. Further clinical investigations are warranted to substantiate its therapeutic potential in PD.

Keywords: Huperzine A; MPTP; murine; Parkinson's disease; SEDDS

P-008:

ADOLESCENCE AS A WINDOW FOR RECALIBRATION: PUBERTAL STRESS MODULATES BEHAVIORAL AND MICROBIOTA CONSEQUENCES OF EARLY-LIFE STRESS

Muhammad Jahangir[1], Dai Wenjing[1,2], Li Tao[1], Guo Wan-Jun*[1]

[1]*Affiliated Mental Health Centre & Hangzhou Seventh People's Hospital, Zhejiang University School of Medicine, Hangzhou, Zhejiang, China.*
[2]*Wenzhou Medical University, Wenzhou, Zhejiang, China.*
**Corresponding author. Email:* guowjcn@zju.edu.cn

Abstract

Background: Early-life adversity induces persistent neurodevelopmental disruptions that increase stress susceptibility later in life. Adolescence is hypothesized to serve as a critical period for recalibrating these effects, potentially restoring normative brain function. However, empirical evidence remains limited.

Aim(s): Therefore, our study aims to investigate the longitudinal effects of early-life adversity and examine whether disruptions during pubertal development alter stress-related outcomes in adulthood.

Methods: We employed a longitudinal rat model to investigate the combined effects of early-life maternal separation (MS) and adolescent sleep deprivation (SDP) on behavior and the gut-brain axis. Pups underwent MS (PND4-16, 2h/day, 3x daily) followed by four experimental conditions in adolescence: (1) control (no stress), (2) MS-only, (3) normal rearing + sleep deprivation (NSDP), and (4) MS + sleep deprivation (MS-SDP). Behavioral assessments: fear conditioning test and anxiety-like behavior assessment (open field test) were performed after stress exposure to evaluate cognitive and affective outcomes. The gut microbiota composition was analyzed (16S ribosomal RNA (rRNA) sequencing of gut luminal contents) to investigate potential disruptions to the gut-brain axis.

Results: Our findings show that both sleep-deprived groups (NSDP and MS-SDP) spent significantly less time in the periphery and exhibited increased locomotor speed in the open field test (OFT) compared to controls ($p < 0.05$), indicating emotional dysregulation induced by stress during adolescence. In contrast, rats subjected only to maternal separation in early life but maintained in a stable adolescent environment (MS group) did not differ significantly from controls ($p > 0.05$), suggesting that a stable adolescent environment may mitigate the long-term emotional effects of early-life adversity. Furthermore, in the fear conditioning test, only the MS-SDP group displayed a significantly lower freezing duration ($p < 0.05$) compared to all other groups, indicating that the combined exposure to early-life adversity and adolescent sleep deprivation specifically impairs fear memory. Microbiota profiling, using 16S rRNA sequencing, revealed distinct compositional differences among the groups. Notably, Bacteroides and *Heminiphilus* were enriched in the MS-SDP and NSDP groups, while their abundance was markedly reduced in the MS group. Additionally, *Parabacteroides* and *Muribaculum* levels were substantially decreased in the MS group. These microbial shifts further underscore the differential impact of early and adolescent life stressors on gut microbiota composition.

Conclusions: Our findings indicate that adolescent sleep deprivation leads to emotional dysregulation independent of early-life stress. However, the combination of

early-life adversity and adolescent sleep disruption (MS-SDP) not only exacerbates emotional dysregulation but also uniquely impairs fear memory. Notably, a stable adolescent environment appears to buffer theadverse effects of early-life stress. Gut microbiota profiling revealed distinct compositional shifts: both the MS-SDP and NSDP groups exhibited increased abundance of Bacteroides and *Heminiphilus*. In contrast, the MS-only group showed decreased levels of Parabacteroides and *Muribaculum*. These findings highlight adolescence as a critical period where environmental interventions can modulate neurobehavioral and microbiome consequences of early-life stress.

Keywords: Early life adversity, adolescence, gut microbiome.

P-009:

DECIPHERING THE ROLE OF AURANOFIN-LOADED NANOPARTICLES AGAINST ANIMAL MODEL OF PARKINSON'S DISEASE: VIA GSK-3β/ NRF2/HO-1 SIGNALING PATHWAYS

Divya Soni[1], Yogesh Garg[2], Shubham Upadhayay[1], Amit Bhatia[2] and Puneet Kumar[1]*

[1]*Department of Pharmacology, Central University of Punjab, Ghudda, Bathinda, Punjab, India, 151401*
[2]*Department of Pharmaceutical Sciences and Technology, Maharaja Ranjit Singh Technical University, Bathinda, Punjab, India-151001*
**Corresponding author. Email: Puneet.bansal@cup.edu.in*

Abstract

Background: The current therapies for Parkinson's disease (PD) provide only symptomatic treatment and are unable to cure the disease completely. The auranofin (AUF) is approved by the FDA for the treatment of rheumatoid arthritis due to its strong anti-inflammatory properties. Several studies also evidence that it modulates Nrf2 signalling. Therefore, the researchers have developed Auranofin hybrid nanoparticles (AUFHNPs) to increase the blood-brain barrier (BBB) penetration of AUF.

Aim: To study the neuroprotective impact of AUFHNPs against rotenone-induced PD using SH-SY5Y cells and Wistar rats.

Methods: AUFHNPs were prepared using ethanol injection method and characterized for various physicochemical properties. Then, an *in vitro* study was conducted on SH-SY5Y following MTTAO/EB assay, RT-PCR, and western blot analysis. Further, an *in vivo* study was conducted on rats (n=10) and pretreated with LiCl (50 mg/kg, i.p.) and Snpp (40 μM/kg, i.p.) as a GSK-3β and HO-1 modulator respectively, in combination with AUF. However, AUF and AUFHNPs (5 & 10 mg/kg) were administered orally, followed by rotenone (1.5 mg/kg, s.c.) for 28 days. The behavioral parameters (rotarod, catalepsy, open field test) were evaluated on the 27^{th} and 28^{th}. On the 29^{th} day, animals were sacrificed, and brains were isolated for biochemical, apoptotic (caspase 3&9), inflammatory (IL-1β, TNF-α), histopathology, immunohistochemistry, and western blot analysis.

Results: The particle size and zeta potential were 284.95 nm, and 42.2±4.3 mV, respectively. Spherical shape NPs were observed under FE-SEM analysis and followed the pattern of first-order release kinetics. The *in vitro* study showed that AUF significantly restored rotenone-induced neurotoxicity by increasing % cell viability. While promoted GSK-3β-mediated Nrf2/HO-1 expression by inhibiting ubiquitination and proteasomal degradation of Nrf2, which allows nuclear translocation and antioxidant expression. Combining LiCl with AUF increased ($P<0.01$) its effect than AUF alone, due to inhibitory action of LiCl over GSK-3β. Administration of AUFHNPs in rats significantly restored motor activity ($P<0.001$), reduced histopathological changes, and phosphorylated GSK-3β ($P<0.01$) to increase expression of Nrf2/HO-1

Conclusion: The study concludes that developing AUFHNPs could increase AUF's bioavailability in the rat brain and exerts neuroprotection via modulation of GSK-3β/Nrf2/HO-1 signaling pathways.

Keywords: Parkinson's disease, Rotenone, Auranofin, Nanoparticles, Neuroprotection

---

P-010:

LIRAGLUTIDE IMPROVES COGNITIVE DEFICITS IN TYPE 2 DIABETIC MICE

Yoottana Janthakhin[1*], Theerawat Khunsanthia[3], Sirikran Juntapremjit[2], and Sutin Kingtong[3]

[1]Department of Research and Applied Psychology, Faculty of Education, Burapha University, Chonburi 20131, Thailand
[2]Department of Learning Management, Faculty of Education, Burapha University, Chonburi 20131, Thailand
[3]Department of Biology, Faculty of Science, Burapha University, Chonburi 20131, Thailand
** Corresponding author: E-mail: yoottana.ja@buu.ac.th*

Abstract

Background: Diabetes-associated cognitive dysfunction has gained attention for its deleterious impacts on people with diabetes. However, clinical interventions for preventing these conditions are still lacking. Glucagon-like peptide-1 (GLP-1) receptor agonist has been shown to exert neuroprotective effects in preclinical studies.

Aim(s): This study aimed to investigate the effects of GLP-1 receptor agonist, liraglutide, on cognitive functions in a diabetic mouse model.

Methods: 15 eight-week-old male C57BL/6N mice were fed high-fat diet for four weeks, followed by a single intraperitoneal injection of streptozotocin (100 mg/kg). Ten Diabetic mice were then treated with liraglutide (0.2 mg/kg daily intraperitoneally) for six weeks, while five diabetic control mice were received injections of normal saline for the same duration. Mice underwent the novel object recognition test (NORT) and the novel object location recognition test (NOLT) to evaluate the cognitive functions. The open-field test and forced-swim test were performed to assess the affective functions. Fasting blood glucose (FBG) and plasma Aβ1-42 levels were also investigated.

Results: We found that liraglutide effectively decreased the FBG levels in diabetic mice ($p < 0.001$). Moreover, cognitive functions were also improved in diabetic mice treated with liraglutide in both NORT ($p < 0.05$) and NOLT ($p < 0.001$). However, the treatment with liraglutide did not affect anxiety-like behavior and depressive-like behavior observed in diabetic mice ($p > 0.05$). In addition, liraglutide treatment was capable of normalizing plasma Aβ1-42 levels in diabetic mice ($p < 0.05$).

Conclusion: Our findings encourage the use of GLP-1 receptor agonist liraglutide to prevent cognitive impairments associated with diabetes conditions.

Keyword: GLP-1 receptor agonist, Liraglutide, Diabetes mellitus, Cognitive deficits

---

P-011:

ERβ-SELECTIVE MODULATION AND NEUROPLASTICITY ENHANCEMENT BY TINOSPORA CORDIFOLIA IN A PERIMENOPAUSAL CONTEXT

Anuradha Sharma[1,*], Anuj Babbar[1]

[1]*Department of Molecular Biology and Genetic Engineering, School of Bioengineering and Biosciences, Lovely Professional University, Punjab, India, 144411*
**Corresponding author. Email: s.anuradha21@ymail.com, anuradha.28927@lpu.co.in*

Abstract

Background: Neuroplasticity and cognition are significantly impacted due to fluctuating levels of estrogens during perimenopausal phase, clinically being managed by Hormone Replacement Therapy (HRT). However, long term HRT is associated with risks necessitating the alternatives like natural Selective estrogen receptor modulators (SERMs) as safer and tissue specific approach. *T. cordifolia* is well known herb to possess neuroprotective effect, however, its SERM-like potential is poorly explored.

Aim(s): This research aimed to identify phytocompounds of *T. cordifolia* that exhibit potential SERM activity and validate the receptor expression modulation activity in an *in vitro* model system.

Methods: By utilizing a two-phase methodology, we screened different *T. cordifolia* compounds for their docking with estrogen receptors using AutoDock tools and assessed their ADME criteria. Further, the effect of selected phytocompounds and butanol extract of *T. cordifolia* (B-TCE) was studied on ER α and β receptors in *in vitro* using MCF-7 and Neuro2A (N2A) cell lines. MTT assay was used to select the treatment dose. Further, morphological studies and immunostaining was performed to study the expression of these receptors.

Results: Molecular docking revealed high docking scores for Berberine (-7.213 and -8.681 kcal/mol) and Magnoflorine (-7.323 and -6.630 kcal/mol) with ERβ and ERα, respectively among total 31 screened compounds of *T. cordifolia*. Based upon their docking scores, ADMET properties and ability to cross the Blood Brain Barrier (BBB), and their reported presence in B-TCE, these two compounds alongwith B-TCE were selected for further evaluation. Treatment with Berberine (10 μg/mL), Magnoflorine (20 μg/mL) and B-TCE (35 μg/mL) exhibited significantly enhanced morphology (increased number and length of processes) as well as upregulated the ERβ expression ($p \leq 0.05$) as compared to untreated control suggesting the ERβ specific SERM-like potential of these *T. cordifolia* based constituents.

Conclusion: The findings of current study suggest that Berberine, Magonoflorine and B-TCE may serve as therapeutic alternatives against HRT for post-menopausal cognitive and plasticity impairments, however, the validation of these findings in *in vivo* and clinical conditions is warranted.

Keywords: SERMs, Estrogen receptor, T. cordifolia, Cognitive decline, Berberine, Magnoflorine

---

P-012:

PHARMACOLOGICAL EVALUATION OF NEUROMEDIN B IN SEPSIS-INDUCED COGNITIVE DYSFUNCTION IN MOUSE MODEL

Manisha Suri[1] and Anjana Bali[1*]

[1]*Department of Pharmacology, Central University of Punjab, Bathinda, Punjab, India, 151401*
**Corresponding author. Email: anjana.bali@cup.edu.in*

Abstract:

Background: Sepsis-associated encephalopathy (SAE) is a Sepsis-associated encephalopathy (SAE) remains a critical neurological complication of sepsis, leading to long-term cognitive impairment. Neuromedin B (NMB) is a neuropeptide with neuromodulatory and neuroprotective properties.

Aim(s): The current study is designed to explore the therapeutic potential of NMB in sepsis-induced cerebral injury and cognitive decline in mice.

Methods: Swiss albino mice were subjected to cecal ligation and puncture (CLP) surgery for induction of sepsis (n=8). Following CLP induction, different pharmacological agents were administrated to animals including NMB at dose of 16, 32, and 64 nmol/kg/i.p. along with the FAK inhibitor (PND-1168-30 mg/kg/i.p.) and the Src inhibitor (SU-6656-4 mg/kg/i.p.) for six consecutive days. Different behavioural parameters including open field test (OFT), inhibitory avoidance test (IAT), novel object recognition test (NORT), morris water maze (MWM) were performed between days 7 - 12, followed by biochemicals analysis of malondialdehyde (MDA), superoxide dismutase (SOD), & reduced glutathione test (GSH) and ELISA of synaptophysin, tumor necrosis factor- α (TNF-α), interleukin-1β (IL-1β), caspase 3, Bcl-2, S100B and neuron specific enolase (NSE) along with brain water content. Furthermore, mRNA and protein expression (pFak & pSrc) were assessed through qRT-PCR and western blotting, respectively. Additionally, structural changes were assessed through Nissl's and immunohistochemistry staining (IHC).

Results: Administration of NMB (16, 32, and 64 nmol/kg/i.p.) potentiates memory and locomotor activity in a dose-dependent manner. Conversely, PND-1168 30 mg/kg/i.p. and SU-6656 4 mg/kg/i.p. mitigate NMB's efficacy by inducing cognitive deficits in CLP mice. Biochemical studies demonstrated, NMB rescued CLP-altered redox balance via modulating SOD, GSH, and MDA levels. Additionally, NMB significantly downregulated pro-inflammatory cytokines (TNF-α, IL-1β), apoptotic marker caspase-3, and neuronal injury markers (S100B and NSE), while upregulating the anti-apoptotic protein Bcl-2. In addition, NMB was able to increase synaptophysin and decrease brain water content in CLP-subjected mice. However, qRT-PCR and western blotting revealed that NMB significantly ameliorates the mRNA and protein expression (pFak, pSrc) in a dose-dependent manner. In addition, IHC staining demonstrated a significant reduction in glial fibrillary acidic protein (GFAP) expression. Further NMB significantly improved CLP-induced morphological alterations assessed by Nissl, s staining. All data was expressed in mean ± SD and analyzed by one way ANOVA followed by Tukey's post-hoc test and two way ANOVA followed by Bonferroni's post-hoc test for multiple comparisons (n=8).

Conclusion: NMB exhibit the potential to mitigate the CLP-induced cerebral injury and cognitive decline in mice.

Keywords: Cerebral injury, cognitive impairment, inflammation, neuromedin B, sepsis associated encephalopathy.

P-013:

Withdrawn

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[1,2*], Pawanrat Chalorak[3], Darunee Rodma[1,2], Rungsarit Sunan[1,2], Apinya Sayinta[1,4], and Krai Meemon[4*]

[1]*Division of Basic and Medical Sciences, Faculty of Allied Health Sciences, Pathumthani University, Mueang Pathum Thani, Pathum Thani, 12000, Thailand*
[2]*Molecular Medicine Program, Multidisciplinary Unit, Faculty of Science, Mahidol University, Ratchathewi, Bangkok, 10400, Thailand*
[3]*Department of Radiological Technology and Medical Physics, Faculty of Allied Health Sciences, Chulalongkorn University, Pathumwan, Bangkok, 10330, Thailand*
[4]*Department of Anatomy, Faculty of Science, Mahidol University, Ratchathewi, Bangkok, 10400, Thailand* [4]
**Corresponding author. E-mail: krai.mee@mahidol.ac.th, promtang.sukrit@gmail.com*

Abstract

Background: Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by dopaminergic (DAergic) neuronal loss and oxidative stress. The marine-derived compound 2-Butoxytetrahydrofuran (2-BTHF), isolated from *Holothuria scabra*, has been reported to exhibit neuroprotective properties in *Caenorhabditis elegans* (*C. elegans*), with its conserved molecular pathways serving as a valuable model for investigating PD-related neuromolecular mechanisms.

Aim: This study aimed to characterize the neuroassociated pathways modulated by 200 µM 2-BTHF treatment in a 50 mM 6-hydroxydopamine (6-OHDA)-induced *C. elegans* PD model.

Methods: Transcriptome-wide RNA sequencing combined with Metascape-based bioinformatics analysis was employed to identify genetic and pathway associations treated by 2-BTHF in 6-OHDA-mediated PD.

Results: Differentially expressed genes (DEGs) were significantly enriched in neurobiological processes, including post-translational modification (protein arginine methyltransferases), cilium organization, synaptic activity, neuronal regulation, neuromuscular transmission, locomotion, and signal transduction. KEGG analysis revealed modulation of pathways associated with synaptic function, glutathione and drug metabolism, neutrophil extracellular trap formation, apoptosis, and necroptosis. Molecular Complex Detection (MCODE) clustering of protein-protein interaction (PPI) networks highlighted mainly five key complexes: major sperm proteins (*msp,* MCODE score 6.5), potassium channels (*twk,* MCODE score 1.5) linked to behavior, mechanosensory proteins (*poml* and *mec,* MCODE score 1.0), ciliogenesis-related proteins (*mks* and *tmem,* MCODE score 1.0), and C-type lectin-like proteins (*clec,* MCODE score 1.0) associated with feeding behavior.

Conclusion: These findings offer new insights into the molecular mechanisms by which 2-BTHF alleviates neurodegeneration in 6-OHDA-induced *C. elegans* PD models. By modulating pathways associated with synaptic function, neuronal signaling, behavior, and oxidative stress, 2-BTHF shows promise as a neurorestorative candidate.

Nonetheless, further studies in mammalian systems are required to validate these effects and assess their translational potential for PD therapy.

Keywords: Metascape, Transcriptomics, 2-BTHF, *C. elegans*, Parkinson’s disease

---

P-015:

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

Tiwari VK[1,2], Honda Y[1], Ogawa K[1], Nakamura S[1], Tsutsui KI[1]

[1] Department of Physiology, Shri Gorakshnath Medical College Hospital and Research Centre, Gorakhpur, India.
[2] Department of Systems Neuroscience, NeuroGlobal Program, Graduate School of Medicine, Tohoku University, Sendai, Japan.

Abstract

Background: Motor impairment, a common and severe consequence of neurological incidents like strokes, demands precise tools for both diagnosis and forecasting recovery.

Aim(s): This research aimed to identify and characterize a new brain activity signature, elicited by brief magnetic stimulation single-pulse TMS (spTMS) of the motor cortex in primates, to assess its utility in evaluating and predicting the course of motor paralysis.

Methods: Our study involved primate subjects, placing electrodes directly on the surface of their brains to record electrical (electrocorticography, ECoG) and muscle activities (motor evoked potential, MEP). We applied single-pulse TMS to the primary motor area and carefully analyzed the resulting brain and limb signals. Statistical methods, including correlation analyses, were used to quantify the associations between the observed neural patterns and physiological responses.

Results: Our investigations have uncovered distinct and consistent brain signals, which we termed the P5 wave, consistently appearing around 5 milliseconds after the TMS pulse. This P5 wave showed a significant positive correlation with the amplitude of the MEPs (P=0.0023, R2=0.7089), indicating its strong link to the excitability of motor pathways.

Crucially, this signal was largely unaffected by direct muscle activity. Furthermore, its strength and frequency of occurrence changed when repetitive TMS (rTMS) was applied, suggesting its involvement in cortical adaptability.

Conclusion: The P5 wave identified in this study represents a dependable and robust indicator of cortical excitability following spTMS. Its independence from peripheral muscle activity and strong correlation with motor output reflect its substantial promise for diagnose and prognosing individual motor recovery.

P-016:

EVENT-RELATED POTENTIAL BLIND SOURCE SEPARATION USING RECURRENT NEURAL NETWORK: EVALUATION ON SIMULATED DATA

Jamie A. O'Reilly [1]

[1]School of International & Interdisciplinary Engineering Programs, School of Engineering, King Mongkut's Institute of Technology Ladkrabang, Bangkok 10520, Thailand
**Corresponding author. Email: jamie.or@kmitl.ac.uk*

Abstract

Background: Event-related potential (ERP) waveforms reflect spatiotemporal summation of potential differences arising from neurophysiological current sources in the brain. Separating ERPs into underlying source waveforms and their scalp distributions is therefore potentially valuable for analyzing neurophysiology associated with psychophysiological events. However, ground-truth sources are unknown when dealing with real ERP data.

Aim(s): This study aimed to evaluate recurrent neural network (RNN) blind source separation (BSS) of ERP waveforms using simulated data with known ground-truth.

Methods: Simulated ERP waveforms were generated using seven simulated source waveforms and scalp distributions. The epoch timespan was from −0.2 to 0.8 s. Scalp distributions were generated by normalizing instantaneous electroencephalography (EEG) amplitudes from 28 electrodes to their absolute maximum. Source waveforms were simulated using Hann-windowed functions with 0.2 s duration and equidistant peak latencies from 0.1 and 0.7 s. Scalp projections from seven sources were summed to simulate a noiseless ERP waveform. Adding random 1/f noise generated a set of 35 simulated ERP waveforms for RNN-BSS. Two simulations were evaluated: one with positive-going source waveforms, and another with negative-going source waveforms; designed to explore RNN source signal rectification. The source waveforms and scalp distributions extracted from applying RNN-BSS to simulated data were compared with the known ground-truth using Pearson's correlation coefficient.

Results: Source waveforms and scalp distributions extracted by RNN-BSS were highly correlated with their ground-truth counterparts. However, where scalp distributions of ground-truth sources correlated highly, they were not separated perfectly by the RNN. Negative-going simulations were reconstructed by the RNN effectively inverting their scalp distributions.

Conclusion: Overall, these results demonstrate that RNN-BSS is reasonably effective when applied to simulated data from seven sources with 28-channel ERP waveforms. Further evaluations are required to determine the limits of source separation when scalp distributions are highly correlated, and the influence of number of electrodes in RNN-BSS.

Keywords: Event-related potential, blind source separation, recurrent neural network, cognitive neuroscience, simulations

----

P-017:

PROLONGED P1 LATENCY IN SIMULTANEOUS AUDITORY-SOMATOSENSORY INTEGRATION AND THE RELATIONSHIP WITH SENSORY BEHAVIORAL PERFORMANCE IN CHILDREN WITH AUTISM SPECTRUM DISORDER

Nutthida Phianchana[1], Timothy W. Budd[2], Vorasith Siripornpanich[1]*

[1]*Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom, Thailand, 73170*
[2]*School of Psychology, University of Newcastle, Ourimbah, New South Wales, Australia, 2258*
*Corresponding author.
Email: vorasith.sir@mahidol.ac.th

Abstract

Background: Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by both hypersensitivity and hyposensitivity to sensory modalities such as sound and touch. P1 is an early sensory response to external stimuli that has been studied in event-related potential (ERP) experiments on perceptual processing in individuals with ASD, but the possibility of P1 as a neural marker for sensory deficits in ASD remains unclear.

Aim(s): The purpose of this study is to investigate the neural responses to sensory stimuli, as measured by P1 component to auditory-somatosensory integration, and the relationship between P1 component and behavioral assessment in children with ASD.

Methods: Twenty-four children aged 6-–12 years (12 children with ASD, 12 age-matched typically developing children) participated in this study. The ERP recording was conducted during passive stimuli presentation under 3 conditions: isolated auditory stimulation, isolated somatosensory stimulation, and simultaneous auditory-somatosensory stimulation while participants watched a silent cartoon.

Results: Results indicated the different neural responses that children with ASD showed significantly prolonged P1 latency in response to only simultaneous auditory-somatosensory integration over frontal and central brain regions. Among entire participants, the relationship between P1 latency over Cz in simultaneous auditory-somatosensory integration and behavioral assessment via Child Sensory profile-2 (CSP-2) in auditory processing (r = 0.524, p = 0.045) and conduct associated with sensory processing subscale (r = 0.521, p = 0.046) was positively significant by using Pearson correlation.

Conclusion: The longer latency of P1 over central brain area, particularly simultaneous auditory-somatosensory integration, was associated with higher raw score of CSP-2, the more sensory dysfunction in daily living of children. Our study suggested that delayed neural transmission can be seen as P1 latency changes associated with sensory issues in autism. These findings may provide new insights into the assessment of clinical severity of ASD and sensory difficulties related to autistic behaviors.

Keywords: Autism spectrum disorder, auditory, somatosensory, ERP, P1 wave

---

Background

Autism spectrum disorder (ASD) is a neurodevelopmental disorder defined by impairments in social communication skills, and restrictive or stereotyped pattern of behaviors (American Psychiatric Association, 2013). In addition, sensory differences are commonly observed through autistic children's sensory-related behaviors such as staring at visual details in objects, holding hands over ears to block loud noises, and showing heightened sensitivity to sound and touch (Tomcheck and Dunn, 2007; Cascio et al., 2016; Little et al., 2018; Schulz and Stevenson, 2019; McKernan et al., 2020). Moreover, sensory processing problems can be found not only in ASD children but children with other developmental problems such as attention deficit hyperactivity disorder (ADHD) or even in healthy children (Little et al., 2017; Little et al., 2018). Therefore, understanding of sensory function in ASD has become an important focus among clinical practitioners and neuroscientists. Numerous studies using neuroimaging and neurophysiological techniques have been used to investigate autism and autistic individual's behaviors. However, differences of ERP characteristics in ASD individuals have also been reported, but it is still inconclusive. Currently, there is a lack of studies that focus on various kinds of sensory domains, especially multisensory integration and symptom severity in children with autism.

Multisensory integration (MSI) is associated with the brain's ability to integrate information from the external world by processing input from two or more sensory modalities. The integration of information across the different sensory modalities plays a fundamental role in building higher-order differences, leading to healthy cognitive abilities (Baum et al., 2015). Consistent with sensory research, the auditory system is crucial in humans as it provides the sensory input necessary for speech recognition, while unusual sensory responses, especially within the auditory domain, may lead to the significant impairments in the language system found in individuals with ASD. Within the tactile system, the sense of touch plays a key role in forming human relationships (Ayres, 1964). Thus, the ability of the brain to integrate all sensory systems may provide the actions of behaviors that usually occur across modalities and reflect thresholds and self-regulation strategies (Dunn, 2014).

The event-related potential (ERP) are electrophysiological responses derived from EEG signals that are time-locked to a specific event or stimulus. The events that are designed to elicit ERPs cover a large variety of sensory, motor, and cognitive functions. In sensory systems, the ERP components – P1 (a positive peak) and N1 (a negative peak around 100 ms after stimulus onset) – are associated with sensory modalities specific to specific brain regions. The auditory P1 wave is thought to have originated from Heschl's gyrus and the temporal cortex, while the N1 wave is associated with the primary or secondary auditory cortex (Modi & Sahin, 2017). The ERP parameters commonly used for comparison consist of the latency (duration stimulus onset to the peak of ERP wave presented in milliseconds) and the amplitude (the height of ERP wave presented in microvolts). Several studies on auditory ERPs have reported that children with ASD demonstrated lower amplitudes of the P1 and N1 components than children with typical development (Bruneau et al., 1999; Stroganova et al., 2013). Based on electrophysiological methods, the integration of multisensory processing of auditory and somatosensory information reported the appearance of MSI in typically developing (TD) children, but not in ASD children aged 6-16 years (Russo et al., 2010).

The aim of the current study is to examine the neural responses to sensory stimuli, as measured by the early sensory P1 component of auditory-somatosensory integration, and to explore the relationship between these neural responses and sensory behavioral performance in children with ASD. The research study is expected to observe differences in early ERP waveform among sensory stimuli (unisensory and multisensory integration conditions) in children with ASD compared to children with typical development. Using electrophysiology ERP methods in this study may lead to a better understanding of how autistic brains work particularly in terms of multisensory processing and further early intervention and appropriate treatment of autism at an early age.

Materials and Methods

Participants

The total number of participants, aged between 6 and 12 years, was 24: 12 children with autism spectrum disorder (ASD) and 12 typically developing (TD) children. ASD participants were recruited from the Rajanagarindra Institute of Child Development (RICD) and OT Freeland Education Center in Chiang Mai. All ASD subjects were confirmed by an experienced developmental-behavioral pediatrician using both the Autism Diagnostic Observation Schedule (ADOS) (Lord et al., 2000) and the Autism Diagnostic Interview-Revised (ADI-R) (Lord, Rutter and Couteur, 1994) in accordance with the DSM-5 (American Psychiatric Association, 2013) criteria. In addition, only autistic children who had scores in mild and moderate severity levels on the Autism Treatment Evaluation Checklist (ATEC) (Rimland and Edelson, 1999) were accepted. Individuals with ASD who had any of the following medical profile following conditions: genetic syndrome and neurodegenerative disorders associated with autism features (e.g. tuberous sclerosis, Rett's syndrome, childhood disintegrative disorder), epilepsy, taking Benzodiazepines or antipsychotic drugs or antihistamine drugs, impaired gross motor function, showing scalp inflammation, head injury were excluded from this study. For TD controls, individuals typically developing were recruited from Dara Academy, Chiang Mai without previous diagnosis of ASD. The participants in this study were recruited on both genders and had normal vision and hearing. They were able to cooperate in the experimental session.

Informed parental consent was given written and participating children were provided for informed verbal assent. All procedures in this study were performed in accordance with the 1964 Helsinki Declaration and its later amendments or comparable human ethical standards. This project was also approved for human ethics by the Mahidol University Central Institutional Review Board (MU-CIRB) and the Ethical Review Committee for Research Involving Human Research Subjects, Department of Mental Health, Ministry of Public Health, Thailand with the approval number COA No. MU-CIRB 2020/006.1301 and DMH.IRB.COA 020/2563 respectively.

Behavioral assessments

All participants were evaluated for their preferences of using hand by the Edinburgh Handedness Inventory, a questionnaire for assessment of person's hand dominance (Oldfield,1971). Only right handed children were included in this study. By using interview information, the Vineland Adaptive Behavior Scales 2nd edition (VABS-2), a semi-structure interview with parent or caregiver was used for evaluating adaptive behavior of participants (Sparrow et al., 2005). Additionally, the Child Sensory Profile 2 (CSP-2) was completed by the primary caregiver in order to evaluate a child's sensory processing patterns in the context of everyday life (Dunn, 2014). In this study, children's raw scores obtained from the CSP-2 were chosen for finding out what sensory systems the child might have difficulty processing, as well as adapting to sensory stimuli in the world environment or participating in activities of daily life.

Stimuli

Two groups of stimuli; auditory and somatosensory stimuli were presented via headphones and vibrotactile stimulator respectively by using Presentation software (Version, 20.2 Neurobiological Systems Inc., Albany, CA, USA) during event-related potential (ERP) recording. Auditory stimuli of tones at 80 dB SPL were presented binaurally through headphones (Sennheiser HD 280 Pro, Germany) as described in previous research (Timora and Budd., 2018). Somatosensory stimuli were delivered to the middle fingers of the participants by using tactors (Dancer Design, St. Helens, UK), which were attached by double-sided adhesive rings and surgifix for fingers. The participants were asked to wear headphones and attach tactors on middle fingers of both hands in the process of ERP recording.

Electroencephalographic acquisition

EEG was recorded with a set of 32 channels waveguard EEG cap designed with high quality of Ag/AgCl electrode including soft silicone electrode cups, using the eego™ sports system (ANT Neuro BV, Enschede, Netherlands). The CPZ electrode was placed as a reference position and GND as an additional ground electrode located at AFZ position following the international 10-20 system. Due to contact between the EEG cap and the participant's head, EEG conductive gel (OneStep Cleargel, H+H Medizinprodukte GbR, Germany) was used via a Monoject curved tip syringe. However, the attachment of horizontal and vertical electrooculography (hEOG and vEOG) were not applied due to the limitation of EOG recording in young children. The EEG signals were collected using an eego™ amplifier with a sampling rate of 1024 Hz. The online montage was set at a common average reference. All electrode impedances were kept below 20 kOhms during EEG/ERP recording. The pre-recording filter was set for band-pass filtered at 0.1 to 100 Hz. The notch filter was opened at 50 Hz. Analog-to-digital (A/D) rate was set at 500 Hz.

Experimental procedure

The ERP recording was performed during passive stimuli presentation while participants watched a silent cartoon without required any response. There were 3 stimulus conditions among a total of 3000 trials of ERP paradigm following: isolated auditory stimuli; AUD (1000), isolated somatosensory stimuli; TAC (1000), and simultaneous auditory-somatosensory stimuli or multisensory integration; MSI (1000). Both sensory modalities (auditory and somatosensory) were presented non-simultaneous (auditory or somatosensory stimulation: unisensory) as well as simultaneous (auditory and somatosensory stimulation: multisensory integration) by designed random of the stimulus onset asynchrony (SOA) between 50 ms and 550 ms. The whole session of the experiment was 30 minutes.

Data and Statistical Analysis

The SPSS statistical package version 26 (IBM Corp., 2022) was used for data analysis. The behavioral data were analyzed statistically by using an independent sample t-test to compare the differences between ASD and TD groups, after the test of normal distribution of data. The data of age, handedness, and all behavioral assessments were displayed as demographic data (Table 1). In this study, the target ERP waveform was focused on early ERP waveform including P1 within the conditions of stimuli presentation. The ERP data acquired during the ERP paradigm was analyzed off-line processing with Brain Vision Analyzer 2 software (Brain Products, GmbH). The ERP waveforms were collected based on their peak amplitude and latency according to grand average over midline electrodes, focusing on Fz, Cz, and Pz electrode sites (Figure 1). The determination of time windows ranged from 30-100 ms post-stimulus for the P1 wave of data analysis in children. The sampling rate conversion was based on spline interpolation and changed into 512 Hz. The ERP signals were re-referenced to the average references for all analyses. The Zero-phase shift IIR Butterworth filters were applied to the continuous data. Band pass filters were set for low cutoff at 0.3 Hz. and high cutoff at 30 Hz. with time constant 0.5, order 4 (24 dB/octave). The electrode channels FP1 & FP2 were used as ocular correction base on Gratton & Coles Method (Gratton et al., 1983). The segmentation of ERP data was processed into epoch files of 700 ms length, which started 200 ms before the stimulus onset and ended 500 ms after the stimulus presentation. Importantly, due to correct distortions in ERP waveforms when the SOA duration was less than the ERP response, the function in Brain Vision Analyzer 2 that allow overlapped segments for ERP analysis was applied. The baseline correction was included to all ERP epochs with the 100 ms preceding stimulus onset, end at 0 ms. The automatic inspection was used as the artifact rejection criterion for removing eye blinks and muscle movement in the initial raw ERP data, with a maximal allowed difference of values in intervals: 200 µV. After that, the ERP data that artifact checked by gradient > 50 µV /ms and amplitude at ± 150 µV were rejected. The artifact-free ERP segments were averaged in each stimulus condition. The number of averaged segments were accepted at greater than 70% of trials.

All peak amplitudes and latencies of P1, the dependent variables were statistically tested by the Kolmogorov-Smirnov test for normal distribution. Three-way MANOVA was used to analyze of the ERP responses on P1 using 2 Groups (ASD, TD) x 3 Conditions (AUD, TAC, MSI) x 3 Electrode sites (Fz, Cz, Pz). However, after screening data for outliers, the design was unbalanced for sample size so that significant Box's M test was found, but the Bartlett's test of sphericity was statistically significant which indicated the relationship between amplitude and latency of ERP components. Thus, only Pillai's trace criterion was used in this study for MANOVA analysis. Moreover, to investigate the relationship of P1 component and behavioral assessment, the Pearson correlation (*r*) was used for analysis across entire participants.

Table 1: Demographic data of participants (Mean ± SD)

Characteristic	**ASD (N=12)**	**TD (N=12)**	**T value**	**P value**
Gender ratio (male/female)	9/3	7/5	N/A	N/A
Handedness (right/left)	12/0	12/0	N/A	N/A
Age (months)	125.67 ± 14.93	111.33 ± 21.67	1.887	0.072
VABS-2 (Standard scores)				
Communication	86.08 ± 17.37	93.25 ± 13.57	-1.126	0.272
Daily living skills	100.08 ± 20.39	105.83 ± 12.91	-0.825	0.420
Socialization	88.17 ± 14.67	87.58 ± 7.89	0.121	0.905
Sum of domain standard scores	274.33 ± 49.85	302.58 ± 34.17	-1.619	0.120
Adaptive behavior composite	90.92 ± 18.64	94.83 ± 9.97	-0.642	0.530
CSP-2 (Raw scores) Quadrant				

Seeking/Seeker	41.67 + 9.28	38.33 + 13.66	0.699	0.492
Avoiding/Avoider	46.75 + 12.91	39.42 + 12.97	1.389	0.179
Sensitivity/Sensor	44.58 + 11.49	37.58 + 16.28	1.217	0.237
Registration/Bystander	47.17 + 10.66	40.83 + 17.14	1.087	0.289
Characteristic	**ASD**	**TD**	**T value**	**P value**
Sensory Section				
Auditory processing	21.50 + 5.99	15.92 + 5.32	2.414	0.025*
Visual processing	13.58 + 5.11	12.92 + 4.72	0.332	0.743
Touch processing	18.33 + 7.76	16.83 + 9.54	0.422	0.677
Movement processing	15.25 + 3.17	15.25 + 6.45	0.000	1.000
Body position processing	17.50 + 5.47	13.67 + 5.94	1.644	0.114
Oral sensory processing	25.42 + 10.16	23.50 + 10.88	0.446	0.660
Behavioral section				
Conduct associated with sensory processing	21.50 + 4.56	18.50 + 7.08	1.234	0.230
Social emotional responses associated with sensory processing	32.25 + 8.57	28.67 + 11.60	0.861	0.399
Attentional responses associated with sensory processing	24.33 + 7.13	19.92 + 8.12	1.416	0.171

*Significant difference, *p*-value < 0.05

Results

Descriptive statistics of peak amplitudes and latencies of the P1 between ASD and TD group were displayed in Table 2.

Table 2: Peak amplitudes (μV) and latencies (ms) of P1 at electrode sites for each condition (Mean + SD)

ERP	Condition	ASD		TD	
		Amplitude (μV)	**Latency (ms)**	**Amplitude (μV)**	**Latency (ms)**
P1 *Note*: None of P1 peaks appeared within time window of interest at Pz	**Isolated auditory stimuli: AUD**				
	Fz	1.33 + 0.54	76.82 + 7.03	1.33 + 0.69	77.15 + 4.45
	Cz	0.60 + 0.50	74.87 + 10.90	0.53 + 0.34	73.44 + 14.71
	Isolated somatosensory stimuli: TAC				
	Fz	0.24 + 0.39	36.89 + 2.28	-0.08 + 0.39	37.11 + 2.26
	Cz	0.20 + 0.49	44.70 + 13.58	0.12 + 0.27	36.72 + 3.88
	Multisensory integration: MSI				
	Fz	1.42 + 1.00	86.50 + 2.70*	1.72 + 1.56	72.54 + 17.77*
	Cz	1.31 + 0.72	84.82 + 4.35*	1.27 + 1.16	62.99 + 23.71*

*Significance level was based on Bonferroni corrected values, *p*-value < 0.05

Group difference on the P1

There was an interaction effect between Group and Condition in influencing the response of P1 wave (Pillai's Trace = 0.072, $F(4, 300) = 2.813$, $p = 0.026$). Following the interaction of Group x Condition, a simple main effect was conducted. Pairwise comparison followed by a univariate *F*-test indicated that the significant difference was found between children with ASD and TD only in the P1 latency. The P1 latency was affected by the condition of stimuli. It revealed that the P1 latency was significantly longer in the ASD than TD groups particularly in MSI ($F(1, 150) = 16.717$, $p = 0.000$), but not other conditions. The significant differences of P1 latencies were shown at Fz and Cz electrodes (Figure 2). The topographical brain mapping of grand averaged ERPs to simultaneous auditory-somatosensory integration condition or MSI for each group of participants were shown in Figure 3.

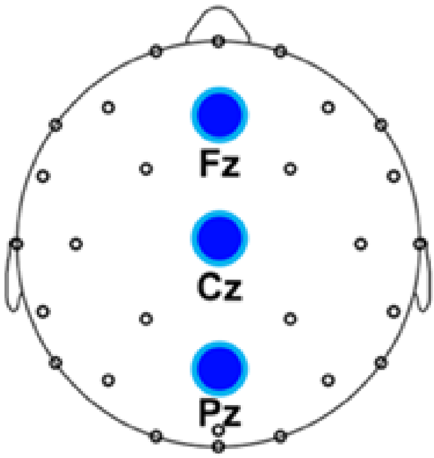


Figure 1: Midline electrodes

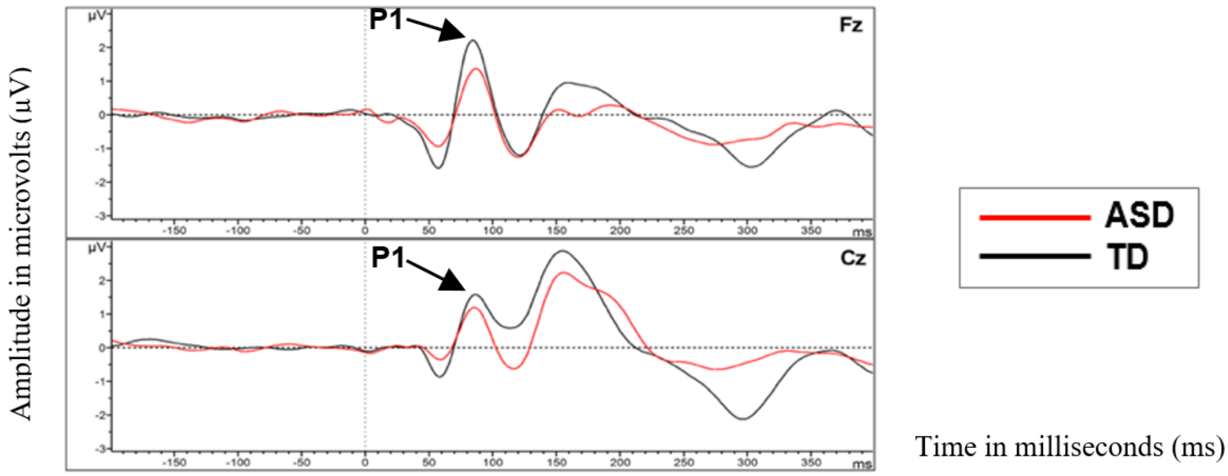


Figure 2: The grand average ERP waveforms in simultaneous auditory-somatosensory integration or MSI at Fz and Cz electrodes.

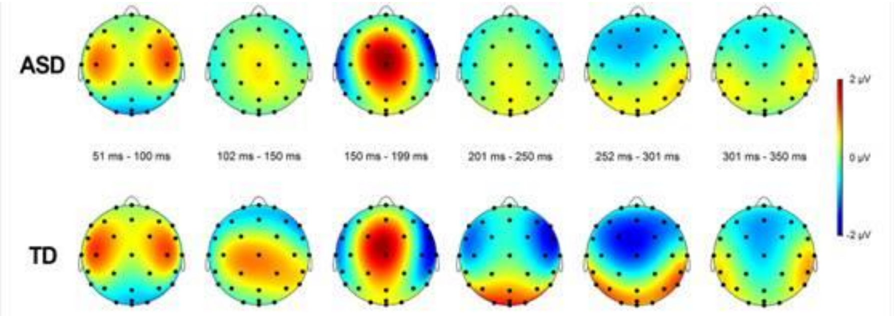


Figure 3: Topographical brain mapping of grand averaged ERPs in simultaneous auditory-somatosensory integration are displayed in 50 intervals from 50-350 ms for children with ASD and TD.

The relationships between P1 latency of simultaneous auditory-somatosensory integration condition over Cz electrode site and sensory behavioral performance

If P1 peak amplitude and latency was not occurred in the grand averaged range, the data was not used for analysis. Thus, the remaining participants per group were 7 ASD and 8 TD for MSI condition. The significant positive correlation was found between P1 latency over Cz for simultaneous auditory-somatosensory integration or MSI and the raw scores of CSP-2 particularly auditory processing in the sensory section (r = 0.524, p = 0.045) (Figure 4). In addition, the correlation between the P1 latency over Cz in simultaneous auditory-somatosensory integration and CSP-2 raw scores in conduct associated with sensory processing subscale in behavioral section was positively significant (r = 0.521, p = 0.046) (Figure 5), while there was no any significant correlation with other CSP-2 quadrant section. For the interpretive of behavioral assessment via CSP-2 questionnaires, the higher raw score of CSP-2, the more sensory dysfunction was associated with the longer latency of P1 in the simultaneous auditory-somatosensory integration condition over Cz electrode across all participants.

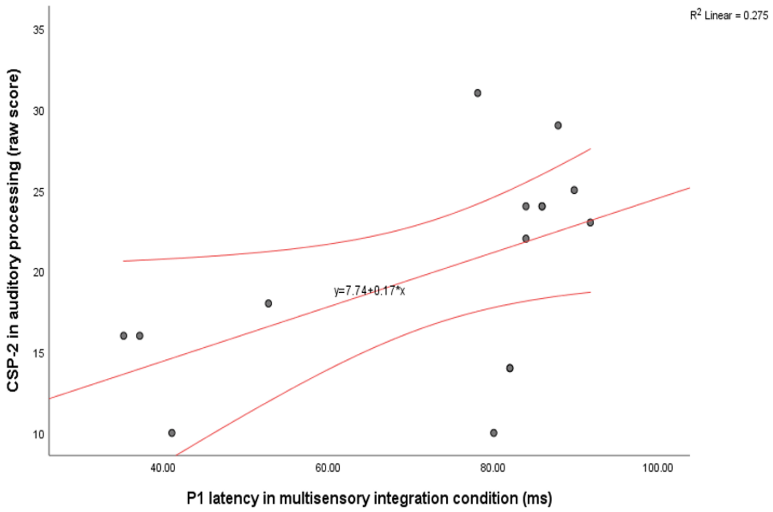


Figure 4: Scatter plot displaying the relationship between P1 latency over Cz in MSI and CSP-2 scores in auditory processing.

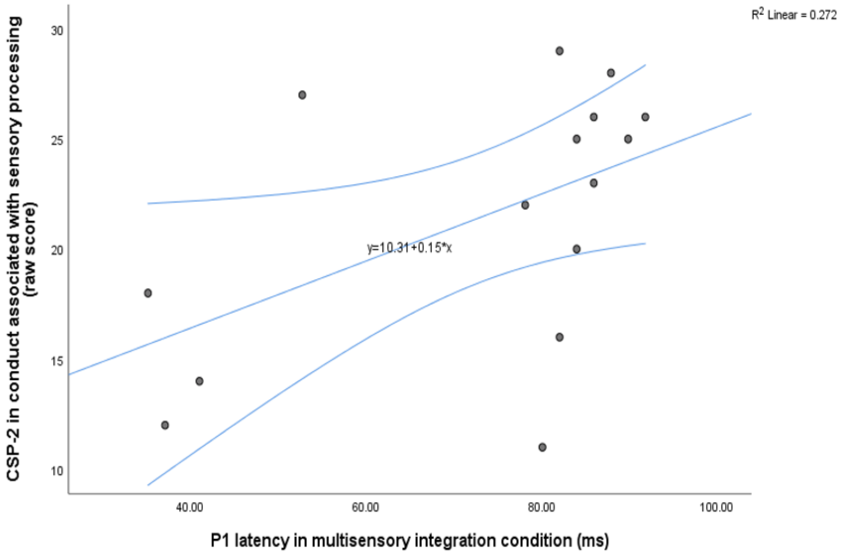


Figure 5: Scatter plot displaying the relationship between P1 latency over Cz in MSI and CSP-2 scores in conduct associated with sensory processing.

Discussion and Conclusion

Our ERP findings showed the significant group differences for early sensory ERP as measured by the P1 over fronto-central electrodes particularly simultaneous auditory-somatosensory integration or MSI condition. Only the relationship between P1 latency over Cz in MSI and behavioral assessment via CSP-2 were significantly found among entire participants. Our current findings indicated that children with ASD showed only delayed P1 latency respond to auditory and somatosensory stimuli occurred simultaneously over frontal and central brain regions, while unisensory condition (isolated auditory stimulation and isolated somatosensory stimulation) did not show the differences between ASD and TD children. By using event-related potentials (ERPs), the neural processing of MSI can elicit the unique neural response not equal to unisensory stimulation (Foxe et al., 2000). Consistent with Fox et al. (2010), according to the maturation of auditory temporal integration study, from visual inspection results, the topographical brain distribution of the P1 in children were found maximal at fronto-central site. Interestingly, P1 latency differences between ASD and TD group have also been observed that ASD individuals had slower P1 response to 80 dB sounds over the right hemisphere in recent study (Dwyer et al., 2021). Nevertheless, our research study only focused on the neural activities over midline electrodes. Based on the first research evidence of MSI with a passive auditory-somatosensory stimulation paradigm in typically developing school-aged children, our results have shown the congruent midline electrode sites particularly Cz electrode that significant MSI occurred and found differences of early sensory ERP components, however, no behavioral data have done in that first study of MSI in TD children (Brett-Green et al., 2008). Additionally, among entire children in this study, we found the relationships between P1 latency over Cz in simultaneous auditory-somatosensory stimulation and auditory processing section including conduct associated with sensory processing section on behavioral measures by CSP-2. Similar to previous studies, but different version of sensory profile evaluation, the weak P1 and N2 responses to standard tones during auditory oddball paradigm were related to more severe sensory seeking behaviors in children with autism (Donkers et al., 2015). Taken together, Donkers and colleagues (2015) have also suggested that the significant interaction of P1 and N2 could predict hyperresponsiveness behaviors in individuals with ASD. The neural response disturbance of sensory processing in ASD children may be modulated by external stimuli in bottom-up processes as well as top-down attentional processes (Donkers et al., 2015; Crasta et al., 2021).

The research studies of cortical brain responses to sensory stimuli have also reported the changes of early sensory ERP component not only for auditory or somatosensory stimulation but also visual stimulation in children with ASD. Our previous study on visual evoked potential (VEP) in preschooler with ASD has reported the relationship between P100 latency and VABS-2 scores in the interpersonal relationship subdomain and a slower N145 response was associated with a higher score of autism severity level in sensory/cognitive awareness subdomain by using ATEC (Sayorwan et al., 2018). However, the early sensory ERP component in VEP task identified as N75 and P100 did not found the significant differences between ASD and TD children, but the repetitive pattern-reversal VEP could demonstrate the slower speed of neural response marked as N145 in ASD (Sayorwan et al., 2018). It might be explained by the atypical intrinsic brain connectivity in autism (Shou et al., 2017) and lack of functional connectivity during VEP and hypersensitivity to stimulation with concurrent reduced functional connectivity between hemispheres particularly sensory cortices in ASD children (Isler et al., 2010). Noteworthy, for the alteration of early sensory ERP components (i.e., Na, P1, C1, and N1 waves) in term of latency for auditory, visual, and tactile processing, intermodal attention was also impact on early cortical response and thalamocortical processing (Karns and Knight, 2009). As mentioned above from our previous study (Sayorwan et al., 2018), the delayed neural response was found in N145 wave that followed behind P100 wave, however, in this study, the delayed P1 latency observed in ASD children specialized for MSI condition may suggest the different neural processing between MSI and unisensory neural mechanisms because we found the longer of P1 latency when MSI condition was presented. Thus, by using the different condition of sensory modalities, the comparison between unisensory and MSI condition can be found the slower of neural speed with the prolonged early sensory ERP component in ASD children particularly the integration of several types of stimuli might bring out or show the modulation of brain's abilities for MSI too early.

For the limitation of our ERP study, sample size in current study was small, resulting in not enough statistical power. There is still a gap and lack of neural activity information in severe levels of ASD children because low-functioning children with ASD were not included in this study. Moreover, the clinical and behavioral performance scores should be concerned as covariate for further correlation analysis.

In conclusion, children with ASD showed delayed neural transmission, indicating reduced neural processing speed during simultaneous auditory-somatosensory integration. This suggests that when multiple sensory inputs are combined, the neural response, as indexed by the P1, is altered. The delayed P1 latency may reflect alterations in neural activity that help explain behavioral patterns in children with ASD, such as over- or under-responsiveness to sensory input. Understanding these neural mechanisms could contribute to developing appropriate interventions and environmental supports for autistic individuals. The research findings might be useful for further studies in various kinds of sensory modalities in which multisensory integration may shed light on the evaluation of the ASD symptoms. Moreover, these findings may provide new insights into the assessment of clinical severity in children with autism spectrum disorder and suggest that sensory difficulties could underlie some of the common behaviors observed in autistic individuals.

Acknowledgements

This research study was partially supported by Graduate Studies of Mahidol University Alumni Association, Mahidol University, Thailand. We would like to thank the entire participants and generous cooperation from all parents and caregivers in this study.

References

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders (5th ed.)*. American Psychiatric Association

Ayres, A. J. (1964). Tactile Functions. Their Relation to Hyperactive and Perceptual Motor Behavior. *Am J Occup Ther*, *18*, 6-11. https://www.ncbi.nlm.nih.gov/pubmed/14116444

Baum, S. H., Stevenson, R. A., & Wallace, M. T. (2015). Behavioral, perceptual, and neural alterations in sensory and multisensory function in autism spectrum disorder. *Prog Neurobiol*, *134*, 140-160. https://doi.org/10.1016/j.pneurobio.2015.09.007

Brett-Green, B. A., Miller, L. J., Gavin, W. J., & Davies, P. L. (2008). Multisensory integration in children: A preliminary ERP study. *Brain Research*, *1242*, 283-290. https://doi.org/https://doi.org/10.1016/j.brainres.2008.03.090

Bruneau, N., Roux, S., Adrien, J. L., & Barthelemy, C. (1999). Auditory associative cortex dysfunction in children with autism: evidence from late auditory evoked potentials (N1 wave-T complex). *Clin Neurophysiol*, *110*(11), 1927-1934. https://www.ncbi.nlm.nih.gov/pubmed/10576489

Cascio, C. J., Woynaroski, T., Baranek, G. T., & Wallace, M. T. (2016). Toward an interdisciplinary approach to understanding sensory function in autism spectrum disorder. *Autism Res*, *9*(9), 920-925. https://doi.org/10.1002/aur.1612

Crasta, J. E., Gavin, W. J., & Davies, P. L. (2021). Expanding our understanding of sensory gating in children with autism spectrum disorders. *Clin Neurophysiol*, *132*(1), 180-190. https://doi.org/10.1016/j.clinph.2020.09.020

Donkers, F. C. L., Schipul, S. E., Baranek, G. T., Cleary, K. M., Willoughby, M. T., Evans, A. M., Bulluck, J. C., Lovmo, J. E., & Belger, A. (2015). Attenuated Auditory Event-Related Potentials and Associations with Atypical Sensory Response Patterns in Children with Autism [Article]. *Journal of Autism and Developmental Disorders*, *45*(2), 506-523. https://doi.org/10.1007/s10803-013-1948-y

Dunn, W. (Ed.). (2014). *Sensory profile-2*. Pearson Publishing.

Dwyer, P., De Meo-Monteil, R., Saron, C. D., & Rivera, S. M. (2021). Effects of age on loudness-dependent auditory ERPs in young autistic and typically-developing children. *Neuropsychologia*, *156*, 107837. https://doi.org/https://doi.org/10.1016/j.neuropsychologia.2021.107837

Fox, A. M., Anderson, M., Reid, C., Smith, T., & Bishop, D. V. (2010). Maturation of auditory temporal integration and inhibition assessed with event-related potentials (ERPs). *BMC Neurosci*, *11*, 49. https://doi.org/10.1186/1471-2202-11-49

Foxe, J. J., Morocz, I. A., Murray, M. M., Higgins, B. A., Javitt, D. C., & Schroeder, C. E. (2000). Multisensory auditory-somatosensory interactions in early cortical processing revealed by high-density electrical mapping. *Brain Res Cogn Brain Res*, *10*(1-2), 77-83. https://doi.org/10.1016/s0926-6410(00)00024-0

Gratton, G., Coles, M. G., & Donchin, E. (1983). A new method for off-line removal of ocular artifact. *Electroencephalogr Clin Neurophysiol*, *55*(4), 468-484. https://doi.org/10.1016/0013-4694(83)90135-9

Isler, J. R., Martien, K. M., Grieve, P. G., Stark, R. I., & Herbert, M. R. (2010). Reduced functional connectivity in visual evoked potentials in children with autism spectrum disorder. *Clinical Neurophysiology*, *121*(12), 2035-2043. https://doi.org/https://doi.org/10.1016/j.clinph.2010.05.004

Karns, C. M., & Knight, R. T. (2009). Intermodal auditory, visual, and tactile attention modulates early stages of neural processing. *J Cogn Neurosci*, *21*(4), 669-683. https://doi.org/10.1162/jocn.2009.21037

Little, L. M., Dean, E., Tomchek, S., & Dunn, W. (2018). Sensory Processing Patterns in Autism, Attention Deficit Hyperactivity Disorder, and Typical Development. *Phys Occup Ther Pediatr*, *38*(3), 243-254. https://doi.org/10.1080/01942638.2017.1390809

Little, L. M., Dean, E., Tomchek, S. D., & Dunn, W. (2017). Classifying sensory profiles of children in the general population. *Child Care Health Dev*, *43*(1), 81-88. https://doi.org/10.1111/cch.12391

Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Jr., Leventhal, B. L., DiLavore, P. C., Pickles, A., & Rutter, M. (2000). The autism diagnostic observation schedule-generic: a standard measure of social and communication deficits associated with the spectrum of autism. *J Autism Dev Disord*, *30*(3), 205-223. https://www.ncbi.nlm.nih.gov/pubmed/11055457

Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism Diagnostic Interview-Revised: a revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *J Autism Dev Disord*, *24*(5), 659-685. https://doi.org/10.1007/BF02172145

McKernan, E. P., Wu, Y., & Russo, N. (2020). Sensory Overresponsivity as a Predictor of Amplitude Discrimination Performance in Youth with ASD. *J Autism Dev Disord*, *50*(9), 3140-3148. https://doi.org/10.1007/s10803-019-04013-0

Modi, M. E., & Sahin, M. (2017). Translational use of event-related potentials to assess circuit integrity in ASD. *Nat Rev Neurol*, *13*(3), 160-170. https://doi.org/10.1038/nrneurol.2017.15

Oldfield, R. C. (1971). The assessment and analysis of handedness: the Edinburgh inventory. *Neuropsychologia*, *9*(1), 97-113. http://www.ncbi.nlm.nih.gov/pubmed/5146491

Rimland, B., & Edelson, S. M. (1999). *Autism Treatment Evaluation Checklist (ATEC)* https://autism.org/autism-treatment-evaluation-checklist/

Russo, N., Foxe, J. J., Brandwein, A. B., Altschuler, T., Gomes, H., & Molholm, S. (2010). Multisensory processing in children with autism: high-density electrical mapping of auditory-somatosensory integration. *Autism Res*, *3*(5), 253-267. https://doi.org/10.1002/aur.152

Sayorwan, W., Phianchana, N., Permpoonputtana, K., & Siripornpanich, V. (2018). A Study of the Correlation between VEP and Clinical Severity in Children with Autism Spectrum Disorder. *Autism Res Treat*, *2018*, 5093016. https://doi.org/10.1155/2018/5093016

Schulz, S. E., & Stevenson, R. A. (2019). Sensory hypersensitivity predicts repetitive behaviours in autistic and typically-developing children. *Autism*, *23*(4), 1028-1041. https://doi.org/10.1177/1362361318774559

Shou, G., Mosconi, M. W., Wang, J., Ethridge, L. E., Sweeney, J. A., & Ding, L. (2017). Electrophysiological signatures of atypical intrinsic brain connectivity networks in autism [Article]. *Journal of Neural Engineering*, *14*(4), Article 046010. https://doi.org/10.1088/1741-2552/aa6b6b

Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland Adaptive Behavior Scales, Second Edition, Survey Forms Manual*. NCS Pearson, Inc.

Stroganova, T. A., Kozunov, V. V., Posikera, I. N., Galuta, I. A., Gratchev, V. V., & Orekhova, E. V. (2013). Abnormal pre-attentive arousal in young children with autism spectrum disorder contributes to their atypical auditory behavior: an ERP study. *PLoS One*, *8*(7), e69100. https://doi.org/10.1371/journal.pone.0069100

Timora, J. R., & Budd, T. W. (2018). Steady-State EEG and Psychophysical Measures of Multisensory Integration to Cross-Modally Synchronous and Asynchronous Acoustic and Vibrotactile Amplitude Modulation Rate. *Multisens Res*, *31*(5), 391-418. https://doi.org/10.1163/22134808-00002549

Tomchek, S. D., & Dunn, W. (2007). Sensory processing in children with and without autism: a comparative study using the short sensory profile. *Am J Occup Ther*, *61*(2), 190-200. https://doi.org/10.5014/ajot.61.2.190

P-018:

EXTRACTS DERIVED FROM MULBERRY LEAVES, COMBINED WITH BUTTERFLY PEA FLOWERS SYNERGISTICALLY ENHANCE REM SLEEP AND COGNITIVE PERFORMANCE THROUGH A $GABA_A$ RECEPTOR-DEPENDENT MECHANISM IN RATS

Jakkrit Nukitram[1, 2, 3, *], Jintanaporn Wattanathorn[3, 4]

[1]*Department of Biology, Faculty of Science, Khon Kaen University, Khon Kaen 40002, Thailand*
[2]*Electrophysiology Laboratory (ElephLab), Khon Kaen University, Khon Kaen 40002, Thailand*
[3]*Human High Performance and Health Promotion (HHP&HP) Research Institute, Khon Kaen University, Khon Kaen 40002, Thailand*
[4]*Department of Physiology, Faculty of Medicine, Khon Kaen University, Khon Kaen 40002, Thailand*
**Corresponding author. Email: jakkritnu@kku.ac.th*

Abstract

Background: The neuropharmacological benefits for sleep quality and mental health from the extracts of *Morus alba* L. leaves (MA) and *Clitoria ternatea* L. flowers (CT) have been revealed previously. However, due to synergistic interactions of polyherbal ingredients, the positive effects of MA mixed with CT still need to be elucidated, and the possible mechanisms of this mixture through the $GABA_A$ receptor remain to be examined.

Aim: To examine the synergistic effects of MA mixed with CT on the modulation of sleep and cognitive function.

Methods: MA and CT were combined in seven combinations: 0MA1CT, 1MA0CT, 1MA1CT, 1MA2CT, 1MA3CT, 2MA1CT, and 3MA1CT to initially evaluate their antioxidant capacity and GABA-promoting activity through *in vitro* assessment. Later, an *in vivo* study was performed to examine their sedative-hypnotic effects by monitoring their electrophysiological responses. Specifically, electroencephalographical and electromyographical oscillations of male Wistar rats (n = 6/group) were recorded for 3 hours after oral administration of the treated solution (500 mg/kg MA, CT, and 3MA1CT). Cognitive performance was then measured using the novel object recognition task. Bicuculline methiodide (2 mg/kg), a $GABA_A$ receptor antagonist, was pre-administered to explore the potential mechanism of the herbal ingredient. Sleep parameters included sleep latency to non-rapid eye movement (NREM) and rapid eye movement (REM) sleep, the percentage of sleep/wake stage durations, the number of bouts, and the average duration of bouts in each sleep/wake cycle stage.

Results: 3MA1CT demonstrated the highest *in vitro* antioxidant capacity and GABA-promoting activity. *In vivo* results also showed that 3MA1CT had positive effects on sleep and cognitive function, outperforming either 500 mg/kg MA or CT alone. Notably, most of these effects, including sedative-hypnotic parameters such as reduced latency to REM sleep, decreased wake time, increased REM sleep duration, more REM sleep bouts, and improved cognitive function, were reversed by pretreatment with bicuculline methiodide. This

suggests that 3MA1CT mediates its effects through a $GABA_A$ receptor-dependent mechanism.

Conclusion: Overall, the benefits of 3MA1CT-based polyherbal drugs, which target $GABA_A$ receptor preferentially, could lead to developing 3MA1CT as an alternative treatment for neuropharmacological conditions related to $GABA_A$ receptor dysfunction.

Keywords: Butterfly pea flowers; Cognitive function; $GABA_A$ receptors; Mulberry leaves; REM sleep

----

P-019:

IRON OVERLOAD IMPAIRS MOTOR COORDINATION AND TENDS TO AFFECT RECOGNITION MEMORY IN b-THALASSEMIA MICE

Benjaporn Kiatpakdee[1], Pornthip Chaichompoo[2], Paranee Yatmark[3], Parin Suwannaprapha[3], Rujapope Sutiwisesak[4], Jim Valodas[5], Saovaros Svasti[1*]

[1]Thalassemia Research Center, Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom 73170
[2]Department of Pathobiology, Faculty of Science, Mahidol University, Bangkok 10400
[3]Department of Pre-Clinic and Applied Animal Science, Faculty of Veterinary Science, Mahidol University, Nakhon Pathom 73170
[4]Department of Physiology, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok 10700
[5]Center for Cancer Research, Hudson Institute of Medical Research, Melbourne, Australia 3168
**Corresponding author. Email: saovaros.sva@mahidol.ac.th*

Abstract

Background: β-Thalassemia is a common genetic disorder in Thailand, characterized by defective β-globin synthesis leading to chronic anemia and systemic complications. Iron overload, resulting from repeated transfusions and increased intestinal absorption, contributes to reduced physical activity due to cardiac dysfunction and muscle atrophy. Some patients also develop early cognitive decline following chronic iron exposure. However, thalassemia patients exhibit varying degrees of iron overload, and its impact on both motor and cognitive functions remains unclear. Due to limitations in human studies, we employed a β-globin knockout (BKO) mouse model, which replicates the thalassemic phenotype and allows for controlled, dose-dependent iron administration. This enables focused investigation of iron's functional effects on movement and memory.

Aims: To examine the effects of varying degrees of iron overload on muscle strength, motor performance, and short-term memory in a β-thalassemia mouse model.

Methods: Female wild-type (WT) and β-thalassemia (BKO) mice (age 2 months) were treated with different doses of iron dextran. At 6 months, mice were tested using grip strength (muscle strength), open field (locomotion), rotarod (balance and coordination), and novel object recognition (NOR; short-term memory).

Results: Iron-overloaded BKO mice exhibited significantly reduced grip strength, locomotion, and rotarod performance compared to non-overloaded controls ($p < 0.05$). In the NOR test, BKO mice treated with low-dose iron showed a trend toward reduced discrimination index and recognition percentage, suggesting early memory impairment.

Conclusion: Iron overload induces dose-dependent deficits in both motor and cognitive functions in β-thalassemia mice. These findings suggest a pathological role of iron in thalassemia-related myopathy and potential neurotoxicity, warranting further investigation into iron's effects on the neuromuscular and central nervous systems.

This study was approved by the Institute of Molecular Biosciences Animal Care and Use Committee, Mahidol University (IMB-ACUC), approval number IMB-ACUC 2023/015.Funding: This research project is supported by Mahidol University (MU's Strategic Research Fund): fiscal year 2024 (MU-SRF-ST-04A/67) and Basic Research Fund: fiscal year 2025 (FFMY-212/2568).

Keywords: β-thalassemia, iron overload, motor function, cognitive impairment, mouse model

P-020:

IRON OVERLOAD INDUCES BLOOD-BRAIN BARRIER DAMAGE AND HIPPOCAMPAL NEURONAL LOSS CONTRIBUTING TO LEARNING AND MEMORY IMPAIRMENT IN β-THALASSEMIA MICE

Parinda Jamrus[1], Nuttanan Pholngam[1,2], Benjaporn Kiatpakdee[1], Chinarat Changsangfa[3], Jim Vadolas[4,5], Sukonthar Ngampramuan[6], Pornthip Chaichompoo[7], Saovaros Svasti[1,8,*]

[1]*Thalassemia Research Center, Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom, Thailand, 73170.*
[2]*Graduate Program in Molecular Medicine, Faculty of Science, Mahidol University, Bangkok, Thailand, 10400.*
[3]*Office of Research and Innovation Affair, Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom, Thailand, 73170.*
[4]*Centre for Cancer Research, Hudson Institute of Medical Research, Melbourne, Australia, 3168.*
[5]*Department of Molecular and Translational Science, Monash University, Melbourne, Australia, 3004.*
[6]*Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom, Thailand, 73170.*
[7]*Department of Pathobiology, Faculty of Science, Mahidol University, Bangkok, Thailand, 10400.*
[8]*Department of Biochemistry, Faculty of Science, Mahidol University, Bangkok, Thailand, 10400.*
**Corresponding author. Email: saovaros.sva@mahidol.ac.th, stssv@yahoo.com*

Abstract

Background: β-Thalassemia is an autosomal recessive disorder caused by reduced or absent β-globin synthesis, leading to ineffective erythropoiesis, severe anemia, and related complications. Iron overload, a major complication of the disease, has been associated with an earlier onset of cerebrovascular disease and cognitive impairment in β-thalassemia patients. However, the mechanisms of iron overload contributing to cerebrovascular pathology and memory decline remain unclear.

Aim(s): This study aims to investigate the effects of iron overload on blood-brain barrier (BBB) disruption contributing to brain pathology, particularly CA3 hippocampal pyramidal neuron damage, and associated learning and memory impairments in β-thalassemia (BKO) mice.

Methods: Three-month-old female BKO and wild-type (WT) mice were intraperitoneally injected with iron dextran (200 mg) to induce iron overload. At 12 months of age, mice were assessed for learning and memory performance using the Morris water maze test. Brain histopathology and BBB structure were examined using hematoxylin and eosin, Prussian blue, Nissl, and immunofluorescent staining.

Results: BKO mice that received iron dextran (BKO+iron) exhibited learning and memory impairments compared to both WT and untreated BKO mice. In BKO+iron mice, BBB disruption was evidenced by reduced expression of zonula occludens-1 (ZO-1), which correlated with increased iron accumulation in the endothelium of cerebral blood vessels. An increased number of astrocytes and microglia was also observed. Decreased living neurons were observed in the CA3 region of BKO+iron mice compared to both WT and untreated BKO mice. Furthermore, the number of CA3 neurons was significantly correlated with both cognitive impairment and iron accumulation at the BBB.

Conclusion: These findings suggest that iron overload contributes to BBB dysfunction and hippocampal neurodegeneration, leading to memory impairment. Therefore, iron

chelation therapy and prognosis in cognitive impairment need to be a concern to improve the quality of life for the patients.

This study was approved by the Institute of Molecular Biosciences Animal Care and Use Committee, Mahidol University (IMB-ACUC), approval number IMB-ACUC 2021/020 and IMB-ACUC 2022/014. Funding: This work was supported by Mahidol University; Postdoctoral Fellowship: Fiscal Year 2025 (MU-PD_2025_27) and Basic Research Fund: fiscal year 2025 (FFMY-212/2568).

Keywords: Blood-brain barrier, Memory impairment, Brain pathology, Iron overload, β-Thalassemia

---

P-021:
EFFECTS OF COVID-19 INFECTION ON CARDIAC AUTONOMIC FUNCTION AND COGNITIVE FUNCTION

Suman Pokhrel[1*], Dilip Thakur[2], Amrendra Jha[2], Ashata Dahal[3]

[1]Nepal Medical College Teaching Hospital, Kathmandu 44600.
[2]B.P. Koirala Institute of Health Sciences, Dharan 56700.
[3]Oxford University Clinical Research Unit, Lalitpur 44600.
**Corresponding author. Email: pokhrelisuman@gmail.com*

Abstract

Background: The coronavirus disease 2019 (COVID-19) which is caused by a coronavirus (SARS-CoV-2) rapidly evolved into a pandemic causing morbidity and mortality worldwide. Currently, no literature exists demonstrating the long-term cardiac autonomic and cognitive changes in COVID-19 recovered patients specifically within the Nepalese population

Aims: We aimed at assessing cardiac autonomic and cognitive function in patients having history of COVID infection.

Methods: Our study was an observational cross-sectional comparative study that comprised two groups, Group 1 consisted COVID-19 recovered patients (N=60) and Group 2 had age and sex matched healthy individuals (N=60). Anthropometric, cardio-respiratory, Heart Rate Variability (HRV) and Cognitive Scores of consented participants were recorded/measured.

Results: Our study found significant autonomic changes in individuals recovered from COVID-19. Frequency domain HRV measures showed increased sympathetic activity and reduced parasympathetic tone and time domain measures showed overall decreased HRV. Cognitive assessments revealed significant impairments in attention (5.1 ± 0.7 vs 5.5 ± 0.4, P=0.012) and delayed recall (3.5 ± 1.0 vs 4.1 ± 0.8, P=0.005) while other domains were unaffected. The COVID-19 group had lower MoCA scores than controls (26.2 ± 2.0 vs. 27.1 ± 1.8), the difference was not statistically significant, indicating only mild cognitive decline.

Conclusion: COVID-19 recovered individuals has significant autonomic dysfunction as evident by reduced HRV and sympathetic dominance, alongside mild but persistent cognitive decline in attention and memory domain. Our findings focus the need for ongoing monitoring of autonomic and cognitive function in COVID-19 recovered individuals to help them lead a quality life.

Keywords: COVID-19, Cardiac Autonomic Function, Cognitive Function, Heart Rate Variability

---

P-022:

MECHANICAL ALLODYNIA AND DRG PLASTICITY ARE MODULATED BY ANDROGRAPHOLIDE AFTER SCIATIC NERVE INJURY IN RATS

Adisorn Ratanayotha[1,2], Kanyaratana Bamrungsuk[3], Napatsorn Jongapirattanakul[1], Bhornluck Paepetch Suato[1], Chairat Turbpaiboon[1], Supin Chompoopong[1]

[1]*Laboratory of Neuroscience, Department of Anatomy, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkoknoi, Bangkok, 10700, Thailand*

[2]*Laboratory of Integrative Physiology, Department of Physiology, Graduate School of Medicine, The University fo Osaka, Suita, Osaka, 565-0871, Japan*

[3]*Department of Basic Medical Science, Faculty of Medicine Vajira Hospital, Navamindradhiraj University, Dusit, Bangkok, 10300, Thailand*

Abstract

Background: Chronic pain is a discomfort sensation that usually arises from nerve injury, an exaggerated pain response to noxious stimuli, and allodynia, in which the nociceptive responses occur to ordinarily non-painful stimuli. Dorsal root ganglia (DRG), which contain the somata of primary sensory neurons, have increasingly been considered as novel targets for clinical neural interfaces, both for neuroprosthetic and pain applications. Current therapies are limited in efficacy and tolerability. Andrographolide, a diterpenoid lactone derived from *Andrographis paniculata* (Fah Talai Jone), has demonstrated good blood-brain barrier permeability and anti-inflammatory. However, its impact on DRG integrity and neuropathic pain remains unclear.

Aim: To investigate whether systemic andrographolide alleviates mechanical allodynia and DRG neuronal plasticity in a rat model of sciatic nerve injury (SNI).

Methods: Sixteen male Sprague-Dawley rats were divided into Sham, untreated SNI, and andrographolide-treated SNI groups. SNI was induced using stepwise transection with fibrin glue repair. Andrographolide (1 mg/kg) was administered intraperitoneally three times per week for two weeks. Mechanical allodynia was evaluated using von Frey filaments at baseline and day 7 to day 42. On day 42, L4-L6 DRGs were processed for cresyl violet staining and morphometric analyses (somatic area, nuclear area, and Feret diameter).

Results: Untreated SNI rats showed persistent mechanical allodynia and significant reductions in DRG soma and nuclear size, accompanied by a leftward shift toward small nociceptive neurons. Andrographolide significantly increased paw withdrawal thresholds, indicating a reduction in allodynia and pain from day 28 to day 42, preserved normal neuronal morphology, and restored DRG morphometric parameters toward Sham values. Frequency analysis revealed that andrographolide maintained medium- and large-diameter neuronal populations, indicating preservation of non-nociceptive pathways that contribute to pain inhibition.

Conclusion: Andrographolide attenuates mechanical allodynia and preserves DRG neuronal structure after sciatic nerve injury, indicating dual anti-nociceptive and neuroprotective effects. These findings highlight the therapeutic potential of andrographolide in modulating peripheral neuroplasticity and advancing strategies for neuropathic pain management.

Keywords: andrographolide; mechanical allodynia; sciatic nerve injury; dorsal root ganglion; neuroprotection

---

P-023:

NEW INSIGHTS INTO RECESSIVE ATAXIA: *CA8* AND *MAG* SPLICE DONOR MUTATIONS IN PAKISTANI FAMILIES

Rabia Akram[1,2], Tehreem Iman[1], Shahid Mahmood Baig[3], Stephanie Efthymiou[2], Henry Houlden[2], Ghulam Hussain[1*]

[1]Neurochemicalbiology and Genetics Laboratory (NGL), Department of Physiology, Faculty of Life Sciences, Government College University, Faisalabad, Pakistan, 38000.
[2]Department of Neuromuscular Disorders, UCL Queen Square Institute of Neurology, WC1N 3BG, University College London, London, United Kingdom.
[3]Human Molecular Genetics, Department of Biological and Biomedical Sciences, Aga Khan University, Karachi, 74800.

Abstract

Background: Cerebellar ataxia is characterized by uncoordinated walking, truncal instability, body or head tremors, uncontrolled hand coordination, dysarthria, and abnormal eye movements.

Aim: This study aims to provide further insight into the clinical and molecular manifestations of cerebellar ataxia, poor intellectual development, and dysequilibrium syndrome (CAMRQ) as well as spastic paraplegia 75 (SPG75).

Methods: Using whole-exome sequencing, we investigated two Pakistani families in which six individuals exhibited clinical signs of recessive ataxia. Sanger sequencing was employed to conduct segregation analysis on available family members, and a minigene splicing assay was utilized to assess the impact of the splicing variant.

Results: We identified a novel homozygous splice donor variant in *MAG* and *CA8*. We report a genetic variation in *MAG* (c.46+1G>T) in a sibship presenting with the phenotype of spastic paraplegia 75 (SPG75) and a variation in *CA8* (c.738+1G>A) associated with CAMRQ. Only a limited number of *MAG* and *CA8* variants have been documented to date.

Conclusion: The present study introduces a novel mutation in *MAG* and *CA8*, the first reported from the Pakistani population, thereby broadening the spectrum of these variants.

Keywords: Cerebellar Ataxias; *MAG; CA8*; Whole exome sequencing; Pakistan

---

P-024:

LIVER AND NEURONAL DEVELOPMENTAL CHANGES IN ZIKV-INFECTED CHICK EMBRYOS

Wongsakorn Siripan[1], Pornkanok Nimnoi[1], Chairat Turbpaiboon[2], Boonrat Tassaneetrithep[3], Supin Chompoopong[2*]

[1]PhD Graduate Program in Molecular Medicine, Faculty of Science, Mahidol University, Bangkok 10400, Thailand.
[2]Department of Anatomy, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok 10700, Thailand.
[3]Center of Research Excellence in Immunoregulation, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok 10700, Thailand.
**Corresponding author. Email: supin.cho@mahidol.ac.th*

Abstract

Background: ZIKV infection in chicken embryos leads to developmental issues in the brain and liver, causing reduced brain size, neuronal cell death, and liver inflammation. The virus targets neural progenitor cells (NPCs) in the brain, disrupting their development and decreasing cell proliferation. Due to the devastating consequences of microcephaly, most of the studies on the effects of ZIKV in fetuses have been focused on the neural development, and there are very few reports about its effects on other systems.

Aim(s): To investigate whether the inoculation of ZIKV in chicken embryos could suppress fetal brain development and induce inflammatory responses in the livers.

Methods: Fertilized chicken eggs were inoculated via the yolk sac at embryonic day 2 with 10^2 and 10^4 FFU/ml of ZIKV and incubated until embryonic days 6, 9, and 14. Brain and liver tissues were collected for histopathological evaluation using hematoxylin and eosin staining and viral antigen detection.

Results: ZIKV-infected embryos exhibited growth retardation and reduced brain size, accompanied by neuronal cell death and evidence of viral antigen expression in the brain by day 14. Liver tissues showed marked inflammatory infiltration and structural disruption, indicating hepatic injury. Notably, infection at even low viral doses resulted in significant liver pathology, suggesting that both viral replication and host immune responses contribute to tissue damage.

Conclusion: This study demonstrates that ZIKV infection in chicken embryos impairs both brain and liver development, highlighting the susceptibility of multiple fetal organs to ZIKV-induced pathology. The chicken embryo model offers a valuable platform for investigating flavivirus-induced developmental disorders and for testing potential antiviral therapies.

Keywords: Fetal development, ZIKV infection, fetal liver injury, liver inflammation

---

P-025:

HIPPOCAMPAL NEURODEGENERATION AND COGNITIVE IMPAIRMENT IN AGING β-THALASSEMIA MICE

Nuttanan Pholngam[1,2], Parinda Jamrus[2,3], Kittikun Viwatpinyo[4,5], Benjaporn Kiatpakdee[2], Jim Vadolas[6,7], Pornthip Chaichompoo[3], Sukonthar Ngampramuan[4]*, Saovaros Svasti[2,8]*

[1]Graduate Program in Molecular Medicine, Faculty of Science, Mahidol University, Bangkok, Thailand, 10400; [2]Thalassemia Research Center, Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom, Thailand, 73170.
[3]Department of Pathobiology, Faculty of Science, Mahidol University, Bangkok, Thailand, 10400;
[4]Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom, Thailand, 10400;
[5]Department of Medical Science, School of Medicine, Walailak University, Nakhonsithammarat, Thailand, 80160.
[6]Centre for Cancer Research, Hudson Institute of Medical Research, Melbourne, Australia, 3168.
[7]Department of Molecular and Translational Science, Monash University, Melbourne, Australia, 3004.
[8]Department of Biochemistry, Faculty of Science, Mahidol University, Bangkok, Thailand 10400.
**Corresponding author. Email: sukonthar.nga@mahidol.ac.th; saovaros.sva@mahidol.ac.th*

Abstract

Background: Cognitive impairment is a major concern in aging populations, where anemia and iron overload are increasingly recognized as risk factors contributing to neurodegeneration. β-thalassemia, a group of inherited blood disorders characterized by reduced or absent hemoglobin production, provides a model in which chronic anemia and iron overload coexist throughout life and may combine impact on brain.

Aims: To investigate cognitive function and hippocampal neuronal integrity in aging β-thalassemia knockout (BKO) mice, and to determine the relationship between anemia, iron overload, and cognitive decline.

Methods: Cognitive function was assessed in 22-month-old BKO and age- and sex-matched wild-type (WT) mice using the Morris Water Maze. Brains were collected for histopathological analysis. Hippocampal neuronal numbers were examined by Nissl staining. Hemoglobin and liver tissue iron were measured. Correlation analysis was performed to evaluate factors inducing cognitive impairment.

Results: BKO mice showed no difference in latency and distance to target after training and spent similar time in each quadrant during probe trial, whereas WT mice exhibited improvements in latency and distance to target and showed significantly increased percentage of time in target quadrant, suggesting deficits in hippocampal-dependent spatial learning and memory in BKO mice. Nissl staining revealed decreased number of living neurons in hippocampal CA3 region compared with WT mice. Notably, living neuronal counts were significantly correlated with behavioral performance, indicating that hippocampal neuronal loss underlies the observed cognitive impairment. Moreover, BKO mice exhibited reduced hemoglobin and elevated liver tissue iron. Spearman's rho correlation further demonstrated significant associations between behavioral performance with anemia and iron overload, underscoring their combined role in driving neurocognitive decline.

Conclusion: Aging BKO mice exhibit hippocampal neuronal loss and cognitive impairment. Chronic anemia and iron overload may synergistically induce hippocampal neurodegeneration. This aging BKO mouse thus serves as a model for dissecting mechanisms of iron- and anemia-related brain dysfunction.

Acknowledgement: *This study was approved by the Institute of Molecular Biosciences Animal Care and Use Committee, Mahidol University (IMB-ACUC), approval number IMB-ACUC 2020/003 and IMB-ACUC 2021/020. Funding: This work was supported by Mahidol University (MRC-MGR 01/2565 and Basic Research Fund: fiscal year 2022; BRF1-077/2565); National Research Council of Thailand (NRCT) and Mahidol University (N42A650349) and the Royal Golden Jubilee PhD Research Scholarship, the National Research Council of Thailand (NRCT) and the Thailand Research Fund (TRF) (Grant no. PHD/0054/2561, Code no. 4.U.MU/61/C.1.O.XX). NP was supported by the Royal Golden Jubilee PhD Research Scholarship, the National Research Council of Thailand (NRCT) and the Thailand Research Fund (TRF) (Grant no. PHD/0054/2561, Code no. 4.U.MU/61/C.1.O.XX).*

Keywords: β-thalassemia, Cognitive impairment, Hippocampus, Behavioral test, Aging

----

P-026:

REPEATED LEARNING IMPROVE COGNITIVE FUNCTION IN β-THALASSEMIA MICE

Parinda Jamrus[1], Nuttanan Pholngam[1,2], Benjaporn Kiatpakdee[1], Chinarat Changsangfa[3], Suparada Aiemongkot[1], Jim Vadolas[4,5], Sukonthar Ngampramuan[6], Pornthip Chaichompoo[7], Saovaros Svasti[1,8,*]

[1]Thalassemia Research Center, Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom, Thailand, 73170.
[2]Graduate Program in Molecular Medicine, Faculty of Science, Mahidol University, Bangkok, Thailand, 10400.
[3]Office of Research and Innovation Affair, Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom, Thailand, 73170.
[4]Centre for Cancer Research, Hudson Institute of Medical Research, Melbourne, Australia, 3168.
[5]Department of Molecular and Translational Science, Monash University, Melbourne, Australia, 3004.
[6]Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom, Thailand, 73170.
[7]Department of Pathobiology, Faculty of Science, Mahidol University, Bangkok, Thailand, 10400.
[8]Department of Biochemistry, Faculty of Science, Mahidol University, Bangkok, Thailand, 10400.
**Corresponding author. Email: saovaros.sva@mahidol.ac.th, stssv@yahoo.com*

Abstract

Background: Mild cognitive impairment affects over 70.2% of adult thalassemia patients and is associated with iron overload. However, the impact of aging and chronic iron overload on cognitive decline in β-thalassemia remains unclear. Additionally, the potential of repeated learning as an intervention strategy to improve cognitive function in β-thalassemia remains unexplored.

Aim(s): This study investigated the impact of aging and chronic iron overload on the onset of cognitive dysfunction in β-thalassemia knockout (BKO) and wild-type (WT) mice, and aimed to evaluate the potential of repeated learning training as a strategy to improve cognitive function in β-thalassemia.

Methods: A total of 173 mice, 78 BKO and 95 WT, were divided into 14 groups (10–15 mice per group) and assessed at three age points: 6, 12, and 22 months, with or without iron overload induced by intraperitoneal injection of iron dextran at 3 months of age. Locomotion was evaluated using the Open Field test, while spatial learning and memory were assessed using the Morris Water Maze. Single-trial learning (cross-sectional) groups were tested once at each age point, whereas repeated learning (longitudinal) groups were tested at 6, 10, and 12 months.

Results: BKO mice showed reduced locomotor activity with increasing age and iron overload, independent of learning frequency. Learning and memory impairments were observed at 22 months in non-overloaded BKO mice, but emerged earlier in iron-overloaded groups, at 6 months in BKO and 12 months in WT mice. Notably, repeated

learning sessions significantly improved memory performance, even in 12-month-old iron-overloaded BKO mice.

Conclusion: These findings demonstrate that aging and iron overload accelerate cognitive decline in β-thalassemia. However, repeated cognitive training can remarkably improve memory performance despite iron-induced neurodegeneration. This highlights the therapeutic potential of behavioral interventions in managing cognitive decline and improving the quality of life in individuals with β-thalassemia.

This study was approved by the Institute of Molecular Biosciences Animal Care and Use Committee, Mahidol University (IMB-ACUC), approval number IMB-ACUC 2021/020 and IMB-ACUC 2022/014. Funding: This work was supported by Mahidol University; Postdoctoral Fellowship: Fiscal Year 2025 (MU-PD_2025_27) and Basic Research Fund: fiscal year 2025 (FFMY-212/2568).

Keywords: Cognitive impairment, Memory improvement, Longitudinal study, Iron overload, β-Thalassemia

----

P-027:

NEUROPSYCHIATRIC MANIFESTATIONS IN CADASIL: A 10-YEAR RETROSPECTIVE STUDY AT SIRIRAJ HOSPITAL

Chatchawan Rattanabannakit[1]*, Suprakit Jiraratwattana[2]

[1] *Division of Neurology, Department of Medicine, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok 10700, Thailand*
[2] *Division of Neurology, Department of Medicine, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok 10700, Thailand*
**Corresponding author. Email: chatchawan.rat@mahidol.ac.th*

Abstract

Background: Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) is a rare, hereditary cerebrovascular disorder caused by *NOTCH3* mutations. In addition to ischemic stroke, CADASIL frequently presents with neuropsychiatric symptoms. However, data from Asian populations remains limited.

Aim(s): To retrospectively characterize neuropsychiatric profiles in genetically confirmed CADASIL patients treated at Siriraj Hospital.

Methods: A retrospective chart review was conducted between 2015 and 2024 in patients with confirmed *NOTCH3* mutations. Demographic, clinical, neuropsychological, and neuroimaging data were analyzed descriptively.

Results: Fourteen patients (9 female, 5 male) were included. All carried heterozygous *NOTCH3* mutations: exon 11 (71.4%), exon 23 (14.3%), exon 6 (7.1%), exon 4 (7.1%). Median age at onset was 55.5 years (IQR 50.0–63.3); median disease duration of follow-up was 8 years (IQR 3.8–11.3). Stroke occurred in 12 patients (85.7%), and cognitive decline in 8 (57.1%). The mean interval from onset of the first symptoms and cognitive symptoms to genetic confirmation was 6 years(SD ±7.2) and 1.9 years (SD ±1.6), respectively. Memory impairment predominated (63.4%), followed by executive dysfunction (14.3%); language, attention, visuospatial, and social cognition deficits were less frequent (7.1% each). Mood disorders were identified in 6 patients (42.9%): depression in 3, irritability in 2, and dysregulated mood with aggression in 1. At baseline, median scores of TMSE was 26 and MoCA 19.5. Imaging revealed Fazekas grade 3 white-matter hyperintensity in 78.6% and cerebral microbleeds in 92.9%.

Conclusion: Cognitive decline and mood disorders are common neuropsychiatric manifestations in CADASIL. Memory impairment was the most prominent cognitive deficit, and depression the most frequent mood disturbance. Systematic neuropsychiatric evaluation and multicenter studies are needed to further delineate the clinical spectrum in Asian populations.

Keywords: CADASIL, neuropsychiatry, cognitive impairment, mood disorder, NOTCH3

P-028:

FENDOSAL SELECTIVELY MODULATES CYTOKINE SECRETION IN ASTROCYTES UNDER ISCHEMIA-REPERFUSION CONDITIONS

Sudichhya Tamrakar[1], Kornkanok Promthep[1], Sovaritthon Chansaengsee[1,2], Jiraporn Panmanee[1], Narisorn Kitiyanant[3], Surapon Piboonpocanun[3], Sujira Mukda[1]*

[1]*Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Salaya, Nakornpathom 73170 Thailand*
[2]*Department of Education, Faculty of Social Sciences and Humanities, Mahidol University, Salaya, Nakornpathom 73170 Thailand*
[3]*Center for Advanced Therapeutics, Institute of Molecular Biosciences, Mahidol University, Salaya, Nakornpathom 73170 Thailand*

*Corresponding author. Email: sujira.muk@mahidol.ac.th

Abstract

Background: Stroke is a leading cause of mortality and disability worldwide, with ischemic stroke accounting for approximately 85% of cases. Neuroinflammation, characterized by activated astrocytes releasing pro-inflammatory cytokines, constitutes a critical pathological hallmark that can exacerbate secondary neuronal injury. Fendosal, a salicylate-derived nonsteroidal anti-inflammatory drug (NSAID), demonstrates anti-inflammatory efficacy in chronic inflammation models. However, its effects on astrocyte-mediated inflammatory responses during ischemic stroke remain unexplored.

Aim(s): This study investigated the effects of fendosal on pro-inflammatory cytokine secretion in astrocytes subjected to oxygen-glucose deprivation (OGD) and re-oxygenation (OGD/R) conditions, mimicking ischemic stroke pathophysiology.

Methods: The mouse astrocyte cell line C8-D1A was subjected to 3-hour OGD followed by 24-hour OGD/R. Cells were treated with 1 nM or 5 nM Fendosal during the re-oxygenation phase. Pro-inflammatory cytokines (IL-1β, IL-6, TNF-α) concentrations in conditioned medium were quantified using ELISA.

Results: IL-6 levels were significantly reduced during OGD compared to control, rebounded upon re-oxygenation, and were further enhanced by fendosal treatment ($p < 0.05$ vs. OGD/R). Both IL-1β and TNF-α showed a significant increase during OGD ($p < 0.0001$ vs. control) but returned to baseline levels following re-oxygenation. Since IL-1β and TNF-α had already normalized during re-oxygenation, Fendosal treatment produced no additional significant changes in these cytokines.

Conclusion: Fendosal demonstrated context- and timing-dependent effects on cytokine expression in astrocytes under OGD/R conditions, enhancing IL-6 while having no observable impact on already-normalized IL-1β and TNF-α levels. These findings highlight the complex, selective nature of NSAID actions in a neuroinflammatory environment and suggest that therapeutic timing may be critical for NSAID intervention in stroke treatment.

Keywords: ischemic stroke, neuroinflammation, fendosal, astrocytes, oxygen-glucose deprivation

---

Background

Stroke represents one of the leading causes of mortality and disability worldwide, constituting a severe cerebrovascular condition characterized by reduced blood supply to the brain, which leads to numerous neurological impairments (Feigin et al., 2022; Warlow et al., 2003). Ischemic stroke, which occurs when blood flow to the brain becomes obstructed, accounts for approximately 85% of all stroke cases globally (Pu et al., 2023; Saini et al., 2021). The reduction in cerebral blood flow deprives neural tissue of essential oxygen and glucose, initiating a complex cascade of detrimental molecular and cellular events, including oxidative stress, excitotoxity, calcium dysregulation, blood-brain barrier (BBB) disruption, and neuroinflammation (Kuriakose & Xiao, 2020).

Neuroinflammation is a critical pathological hallmark of ischemic stroke, triggered by the release of damage-associated molecular patterns (DAMPs) from necrotic cells, cellular debris, and reactive oxygen species generated during the ischemic cascade (Lei et al., 2025). While neuroinflammation initially serves as a protective function by clearing debris and promoting tissue repair, excessive and sustained inflammatory responses aggravate reperfusion injury, enhance oxidative stress, disrupt BBB integrity, and worsen neuronal damage, ultimately contributing to stroke progression and poor clinical outcomes (Cao et al., 2023).

Activated glial cells, particularly microglia and astrocytes, serve as central players of the neuroinflammatory response through the secretion of pro-inflammatory cytokines, including interleukin-1β (IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-α (TNF-α) (Tuttolomondo et al., 2008). These mediators disrupt the BBB integrity, promote infiltration of peripheral immune cells, and amplify secondary neuronal injury cascades (Hudome et al., 1997; Ishikawa et al., 2004; Matsuo et al., 1994; Tuttolomondo et al., 2008). Among glial populations, astrocytes play a particularly dynamic role and phase-dependent responses to ischemic injury (Cekanaviciute et al., 2014; Luo, 2022). In the early stages, astrocytes may provide neuroprotective support through anti-inflammatory signaling and metabolic assistance. However, in later phases, reactive astrocytes can intensify neuroinflammatory response by producing high levels of pro-inflammatory mediators, potentially impairing neuronal function and limiting neuroplasticity (Kim et al., 2018; Linnerbauer et al., 2020; Ye et al., 2015). These dual roles place astrocytes as both critical neuroprotectors and potential drivers of secondary injury in ischemic stroke pathophysiology. As such, therapeutic strategies for ischemic stroke increasingly focus on modulating astrocyte-mediated inflammation to optimize the balance between beneficial and detrimental inflammatory responses.

Fendosal (5-(4,5-dihydro-2-phenylbenz[e]indol-3-yl)salicylic acid), also known as HP129, is a salicylate-derived nonsteroidal anti-inflammatory drug (NSAID) with demonstrated potent anti-inflammatory efficacy in experimental models of chronic inflammation (Wills et al., 1985). Its primary mechanism involves non-selective inhibition of cyclooxygenase (COX) enzymes, specifically COX-1 and COX-2, which are crucial for the biosynthesis of prostaglandins.(Bloomfield et al., 1978; Forbes et al., 1984; Lassman et al., 1978). NSAID effects on cytokine expression can be highly context-dependent and may involve COX-independent pathways. The pharmacological effects of NSAIDs, including their impact on cytokine production, can vary significantly depending on cellular context, concentration, and timing of exposure, with some studies reporting paradoxical pro-inflammatory effects under specific conditions (Harizi et al., 2010). However, Fendosal's potential effects on astrocytic inflammatory responses within the context of ischemic stroke pathophysiology remain largely unexplored.

Given the pivotal role of astrocytes in orchestrating neuroinflammation during ischemic stroke and the complex context-dependent nature of NSAID pharmacology, this study aims to investigate the effects of Fendosal on pro-inflammatory cytokine expression in astrocytes subjected to oxygen-glucose deprivation (OGD) and reperfusion (OGD/R). By examining these effects, we seek to elucidate whether fendosal can modulate astrocyte-mediated inflammatory responses in a temporally dependent manner and potentially contribute to therapeutic strategies aimed at mitigating stroke-associated neuroinflammatory injury.

Materials and Methods

Cell culture

The mouse astrocyte cell line C8-D1A (ATCC® CRL-2541™, Manassas, VA, USA), originally derived from the cerebral cortex of C57BL/6 mice, was used in this study. Cells were maintained in Dulbecco's Modified Eagle Medium (DMEM; Thermo Fisher Scientific, Waltham, MA, USA) supplemented with 10% heat-inactivated fetal bovine serum (FBS), 1% GlutaMAX™, and 1% penicillin/streptomycin and maintained at 37°C in a humidified atmosphere containing 5% CO_2. Cells were passaged upon reaching 80–90% confluency to ensure they remain in the exponential growth phase.

Oxygen-glucose deprivation (OGD) and re-oxygenation (OGD/R)

In vitro ischemic conditions were mimicked using oxygen-glucose deprivation (OGD) followed by re-oxygenation (OGD/R) protocols, as previously described (Mukda et al., 2019). Hypoxic conditions were generated using a Forma Series 3 Water Jacketed CO_2 Incubator (Thermo Fisher Scientific, Waltham, MA, USA) configured for tri-gas mixing. Glucose-free DMEM was pre-equilibrated by continuous gassing with 5% CO_2, 1% O_2, and 94% N_2 for a minimum of 15 hours prior to use.

For the OGD experiment, culture medium was removed, and C8-D1A cells were washed once with pre-warmed (37°C) phosphate-buffered saline (PBS). Cells were then exposed to pre-equilibrated glucose-free DMEM under hypoxic conditions (5% CO_2, 1% O_2, and 94% N_2) for 3 hours at 37°C. Normoxic control cultures were maintained in standard DMEM under normal atmospheric conditions (5% CO_2, 95% air) at 37 °C. Following OGD exposure, cells were returned to standard culture conditions (37°C, 5% CO_2, 95% air, humidified atmosphere) with glucose-containing DMEM in the presence or absence of 1 nM or 5 nM fendosal (CAS no. 53597-27-6, TargetMol, MA, USA) for 24 hours to simulate re-oxygenation, as shown in Figure 1. These concentrations of fendosal (1 nM and 5 nM) were selected based on preliminary dose-optimization studies using MTT cell viability assays. These concentrations were chosen because they did not significantly affect C8-D1A astrocyte viability (data not shown). Conditioned medium was collected at two time points: immediately following the 3-hour OGD period and after the 24-hour re-oxygenation phase for subsequent enzyme-linked immunosorbent assay (ELISA) analysis.

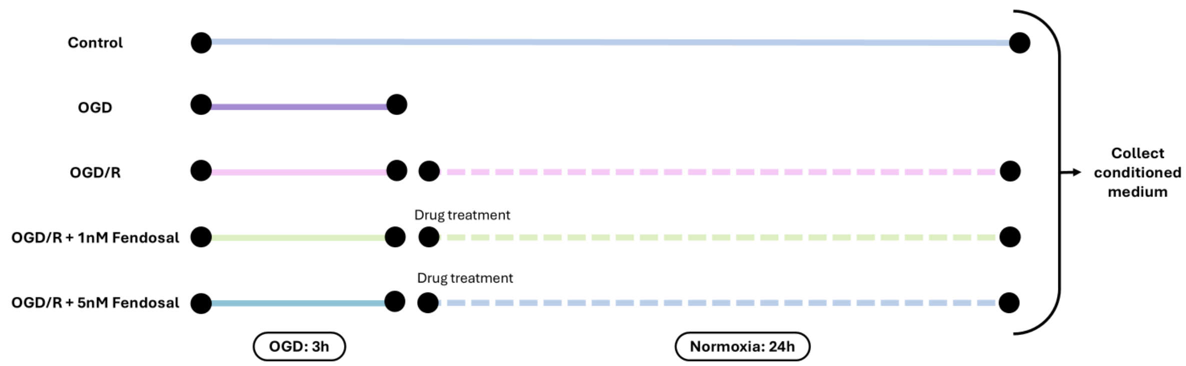


Figure 1. Schematic representation of the experimental setup.

Enzyme-Linked Immunosorbent Assay (ELISA) for cytokine quantification

Pro-inflammatory cytokine concentrations in conditioned medium were quantified using commercially available ELISA kits according to the manufacturer's protocols. Interleukin-1β (IL-1β) levels were measured using the Mouse IL-1β/IL-1F2 DuoSet® ELISA kit (R&D Systems, MN, USA). Interleukin-6 (IL-6) concentrations were determined with the Mouse IL-6 ELISA Kit (Invitrogen™, Thermo Fisher Scientific, Waltham, MA, USA). Tumor necrosis factor-alpha (TNF-α) levels were assessed using the Mouse TNF ELISA Set BD OptEIA™ (BD Biosciences, NJ, USA).

Statistical Analysis

Data are presented as mean ± standard error of the mean (SEM) from six independent experiments. Statistical comparisons were performed using one-way analysis of variance (ANOVA) followed by Tukey's multiple comparison post hoc test. Statistical significance was defined as $p < 0.05$.

Results

OGD/R modulates pro-inflammatory cytokine expression in astrocytes

The expression profiles of proinflammatory cytokines (IL-6, IL-1β, and TNF-α) in C8-D1A astrocytes were evaluated following 3-hour OGD exposure and subsequent 24-hour re-oxygenation. IL-6 expression exhibited a distinct pattern characterized by significant suppression during OGD compared to normoxic controls (Figure 2A). Re-oxygenation partially restored IL-6 levels, while fendosal treatment (5 nM) during the re-oxygenation phase significantly enhanced IL-6 secretion above OGD/R levels alone. Conversely, IL-1β demonstrated an opposite response pattern (Figure 2B). OGD exposure significantly elevated IL-1β expression compared to control conditions. However, re-oxygenation effectively normalized IL-1β levels, returning them to baseline values. Fendosal treatment did not produce additional significant changes in IL-1β expression during the re-oxygenation phase. TNF-α expression showed marked upregulation following OGD exposure compared to normoxic controls (Figure 2C). Similar to IL-1β, re-oxygenation restored TNF-α levels to near-baseline values. Fendosal treatment resulted in a modest, non-significant increase in TNF-α expression during re-oxygenation compared to the OGD/R group alone.

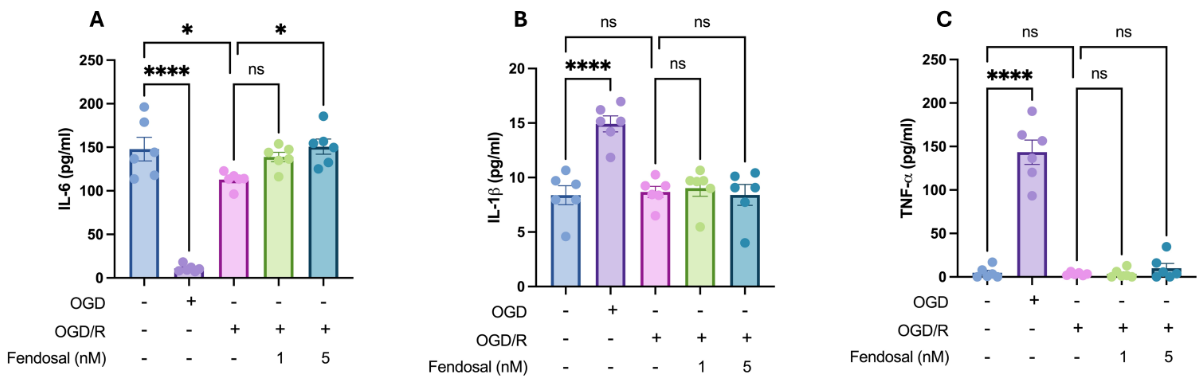


Figure 2. Expression levels of pro-inflammatory cytokines in C8-D1A astrocytes following OGD/R treatment. Concentration of (A) IL-6, (B) IL-1β, and (C) TNF-α in conditioned medium measured by ELISA following 3-hour OGD exposure and 24-hour re-oxygenation with or without 1 nM or 5 nM fendosal. Data represent mean ± SEM from six independent experiments (n = 6). Statistical analysis was performed using one-way ANOVA followed

by Tukey's multiple comparison post hoc test. * $p < 0.05$, **** $p < 0.0001$ compared to indicated groups.

Discussion and Conclusion

The present findings contribute to the growing understanding of astrocyte-mediated inflammatory responses following ischemic injury and reperfusion. Cytokine analysis in C8-D1A astrocytes revealed distinct, phase-dependent responses under OGD/R conditions, demonstrating the complex nature of neuroinflammatory signaling in stroke pathophysiology.

Our results demonstrated a profound reduction in IL-6 levels during OGD, followed by recovery upon reperfusion and further elevation with fendosal treatment. The initial decrease, while seemingly counterintuitive, may be attributed to severe metabolic stress and energy deprivation during the ischemic phase, which could impair the cellular machinery required for robust cytokine synthesis and secretion (Dziurla et al., 2010). The subsequent increase with reperfusion is consistent with the well-documented inflammatory cascade triggered by reperfusion injury, where reintroduction of oxygen and glucose fuels the inflammatory response(Abuirmeileh et al., 2012; Langdale et al., 2008). The most notable observation relates to the effect of fendosal. At a 5 nM concentration, fendosal amplified reperfusion-induced IL-6 expression to a level significantly higher than the OGD/R group. This outcome is surprising given that fendosal is an NSAID, a class of drugs typically associated with anti-inflammatory effects. However, NSAIDs' effects on cytokine expression are not always inhibitory and can be highly context dependent. Similar paradoxical increases in IL-6 secretion have been observed in other NSAIDs like aspirin, demonstrating that drug effects depend critically on cellular context, concentration, and exposure timing(Antunes et al., 2019). In contrast to the complex dynamics of IL-6, IL-1β, and TNF-α responses were more predictable. Both cytokines showed significant upregulation during OGD, followed by normalization during reperfusion, returning to near-baseline levels. Under these conditions, where IL-1β and TNF-α had already been effectively normalized by the re-oxygenation process itself, fendosal treatment produced no additional significant changes. This lack of observable effect likely reflects the fact that these cytokines had already returned to control levels during re-oxygenation, providing limited opportunity for further modulation rather than indicating a lack of drug activity *per se*.

The elevated IL-6 following reperfusion is particularly intriguing. The mechanism behind the elevation is complex and may involve off-target effects on transcription factors, modulation of alternative inflammatory pathways, or activation of astrocyte-specific signaling cascades during reperfusion (Clayton & Liddelow, 2025; Sofroniew, 2020). The elevation likely represents a compensatory and potentially neuroprotective mechanism, rooted in its dual pro- and anti-inflammatory roles (Erta et al., 2012). Elevated IL-6 activates intracellular signaling pathways, particularly the JAK/STAT3 pathway, which promotes neurogenesis, gliogenesis, astrogliosis, and angiogenesis, all of which are critical for brain repair and restoration of homeostasis following ischemic insults (Fang et al., 2013; Jung et al., 2011). IL-6 therefore exhibits neurotrophic-like functions beyond its classical pro-inflammatory role. Importantly, IL-6's effects are context-dependent, governed by the balance between classic signaling (via membrane-bound IL-6 receptor) which tends to be neuroprotective, and trans-signaling (via soluble IL-6 receptor) which is often pro-inflammatory (Rose-John, 2012).Overall, the elevation of IL-6 post-stroke can be best understood as a multifaceted compensatory response that mediates neuroprotection and repair while modulating inflammation. Understanding these mechanisms could provide insights into both therapeutic applications and potential limitations of NSAIDs in stroke treatment.

In conclusion, this study reveals that fendosal effects on cytokine expression in astrocytes following OGD/R are both timing- and context-dependent. The enhancement of IL-6 expression demonstrates fendosal's capacity to modulate inflammatory signaling when pathways remain active, while the lack of observable changes in IL-1β and TNF-α likely reflects the already-normalized status of these cytokines during the re-oxygenation phase rather than drug inefficacy. These findings suggest that the therapeutic window and target selection for NSAID intervention in stroke may be critical factors requiring further investigation. Future studies should examine alternative treatment timings, dose-response relationships, and the molecular pathways underlying these context-dependent effects to optimize Fendosal's therapeutic potential in stroke therapy.

Acknowledgements

This research was supported by Mahidol University through the Fundamental Fund for fiscal year 2025 (FF-211/2568), funded by the National Science Research and Innovation Fund (NSRF), awarded to SM. ST was supported by a Mahidol University Ph.D. Scholarship (2022).

References

Abuirmeileh, A., Harkavyi, A., Rampersaud, N., Lever, R., Tadross, J. A., Bloom, S. R., & Whitton, P. S. (2012). Exendin-4 treatment enhances L-DOPA evoked release of striatal dopamine and decreases dyskinetic movements in the 6-hydoxydopamine lesioned rat. *J Pharm Pharmacol*, *64*(5), 637-643. https://doi.org/10.1111/j.2042-7158.2011.01394.x

Antunes, D. M., Rodrigues, M., Guimaraes, D. M., Duarte, C. M. E., Miguita, L., Correa, L., APL, D. E. O., Fernandes, K. P. S., & Nunes, F. D. (2019). Nonsteroidal Anti-inflammatory Drugs Modulate Gene Expression of Inflammatory Mediators in Oral Squamous Cell Carcinoma. *Anticancer Res*, *39*(5), 2385-2394. https://doi.org/10.21873/anticanres.13356

Bloomfield, S. S., Barden, T. P., & Mitchell, J. (1978). Fendosal and aspirin in postpartum uterine pain. *Clin Pharmacol Ther*, *23*(4), 390-396. https://doi.org/10.1002/cpt1978234390

Cao, Y., Yue, X., Jia, M., & Wang, J. (2023). Neuroinflammation and anti-inflammatory therapy for ischemic stroke. *Heliyon*, *9*(7), e17986. https://doi.org/10.1016/j.heliyon.2023.e17986

Cekanaviciute, E., Fathali, N., Doyle, K. P., Williams, A. M., Han, J., & Buckwalter, M. S. (2014). Astrocytic transforming growth factor-beta signaling reduces subacute neuroinflammation after stroke in mice. *Glia*, *62*(8), 1227-1240.

Dziurla, R., Gaber, T., Fangradt, M., Hahne, M., Tripmacher, R., Kolar, P., Spies, C. M., Burmester, G. R., & Buttgereit, F. (2010). Effects of hypoxia and/or lack of glucose on cellular energy metabolism and cytokine production in stimulated human CD4+ T lymphocytes. *Immunol Lett*, *131*(1), 97-105. https://doi.org/10.1016/j.imlet.2010.02.008

Feigin, V. L., Brainin, M., Norrving, B., Martins, S., Sacco, R. L., Hacke, W., Fisher, M., Pandian, J., & Lindsay, P. (2022). World Stroke Organization (WSO): Global Stroke Fact Sheet 2022. *Int J Stroke*, *17*(1), 18-29. https://doi.org/10.1177/17474930211065917

Forbes, J. A., Barkaszi, B. A., Ragland, R. N., & Hankle, J. J. (1984). Analgesic effect of fendosal, ibuprofen and aspirin in postoperative oral surgery pain. *Pharmacotherapy*, *4*(6), 385-391. https://doi.org/10.1002/j.1875-9114.1984.tb03401.x

Hudome, S., Palmer, C., Roberts, R. L., Mauger, D., Housman, C., & Towfighi, J. (1997). The role of neutrophils in the production of hypoxic-ischemic brain injury in the neonatal rat. *Pediatr Res*, *41*(5), 607-616. https://doi.org/10.1203/00006450-199705000-00002

Ishikawa, M., Zhang, J. H., Nanda, A., & Granger, D. N. (2004). Inflammatory responses to ischemia and reperfusion in the cerebral microcirculation. *FBL*, *9*(2), 1339-1347. https://doi.org/10.2741/1330

Kim, J. W., Park, M. S., Kim, J. T., Kang, H. J., Bae, K. Y., Kim, S. W., Shin, M. G., Cho, K. H., & Kim, J. M. (2018). The Impact of Tumor Necrosis Factor-alpha and Interleukin-1beta Levels and Polymorphisms on Long-Term Stroke Outcomes. *Eur Neurol*, *79*(1-2), 38-44. https://doi.org/10.1159/000484599

Kuriakose, D., & Xiao, Z. (2020). Pathophysiology and Treatment of Stroke: Present Status and Future Perspectives. *Int J Mol Sci*, *21*(20). https://doi.org/10.3390/ijms21207609

Langdale, L. A., Hoagland, V., Benz, W., Riehle, K. J., Campbell, J. S., Liggitt, D. H., & Fausto, N. (2008). Suppressor of cytokine signaling expression with increasing severity of murine hepatic ischemia-reperfusion injury. *J Hepatol*, *49*(2), 198-206. https://doi.org/10.1016/j.jhep.2008.03.014

Lassman, H. B., Wilker, J. C., Anderson, V. B., Agnew, M. N., Allen, R. C., & Novick, W. J., Jr. (1978). Fendosal (HP 129): a potent anti-inflammatory and analgesic compound. *Agents Actions*, *8*(3), 209-217. https://doi.org/10.1007/BF01966606

Lei, W., Zhuang, H., Huang, W., & Sun, J. (2025). Neuroinflammation and energy metabolism: a dual perspective on ischemic stroke. *J Transl Med*, *23*(1), 413. https://doi.org/10.1186/s12967-025-06440-3

Linnerbauer, M., Wheeler, M. A., & Quintana, F. J. (2020). Astrocyte Crosstalk in CNS Inflammation. *Neuron*, *108*(4), 608-622. https://doi.org/10.1016/j.neuron.2020.08.012

Luo, J. (2022). TGF-β as a Key Modulator of Astrocyte Reactivity: Disease Relevance and Therapeutic Implications. *Biomedicines*, *10*(5). https://doi.org/10.3390/biomedicines10051206

Matsuo, Y., Onodera, H., Shiga, Y., Nakamura, M., Ninomiya, M., Kihara, T., & Kogure, K. (1994). Correlation between myeloperoxidase-quantified neutrophil accumulation and ischemic brain injury in the rat. Effects of neutrophil depletion. *Stroke*, *25*(7), 1469-1475. https://doi.org/doi:10.1161/01.STR.25.7.1469

Mukda, S., Tsai, C. Y., Leu, S., Yang, J. L., & Chan, S. H. H. (2019). Pinin protects astrocytes from cell death after acute ischemic stroke via maintenance of mitochondrial anti-apoptotic and bioenergetics functions. *J Biomed Sci*, *26*(1), 43. https://doi.org/10.1186/s12929-019-0538-5

Pu, L., Wang, L., Zhang, R., Zhao, T., Jiang, Y., & Han, L. (2023). Projected Global Trends in Ischemic Stroke Incidence, Deaths and Disability-Adjusted Life Years From 2020 to 2030. *Stroke*, *54*(5), 1330-1339. https://doi.org/10.1161/strokeaha.122.040073

Saini, V., Guada, L., & Yavagal, D. R. (2021). Global Epidemiology of Stroke and Access to Acute Ischemic Stroke Interventions. *Neurology*, *97*(20 Suppl 2), S6-s16. https://doi.org/10.1212/wnl.0000000000012781

Tuttolomondo, A., Di Raimondo, D., di Sciacca, R., Pinto, A., & Licata, G. (2008). Inflammatory cytokines in acute ischemic stroke. *Curr Pharm Des*, *14*(33), 3574-3589. https://doi.org/10.2174/138161208786848739

Warlow, C., Sudlow, C., Dennis, M., Wardlaw, J., & Sandercock, P. (2003). Stroke. *The Lancet*, *362*(9391), 1211-1224. https://doi.org/https://doi.org/10.1016/S0140-6736(03)14544-8

Wills, R. J., Velagapudi, R. B., Puri, S. K., & Yakatan, G. J. (1985). The effect of food and antacid on the absorption of fendosal. *Biopharm Drug Dispos*, *6*(1), 43-50. https://doi.org/10.1002/bdd.2510060106

Ye, L., Yang, Y., Zhang, X., Cai, P., Li, R., Chen, D., Wei, X., Zhang, X., Xu, H., Xiao, J., Li, X., Lin, L., & Zhang, H. (2015). The Role of bFGF in the Excessive Activation of Astrocytes Is Related to the Inhibition of TLR4/NFκB Signals. *Int J Mol Sci*, *17*(1). https://doi.org/10.3390/ijms17010037

P-029:

OXYBERBERINE ATTENUATES VASCULAR DEMENTIA VIA MODULATION OF RHO-KINASE SIGNALING IN A RAT MODEL OF CHRONIC CEREBRAL HYPOPERFUSION

Manoj P. Dandekar[1,*], Harapriya Baral[1], Simranjit Kaur[1], Arbaz Sujat Shaikh[2], K. Venkata Rao[2]

[1]*Department of Pharmacology and Toxicology, National Institute of Pharmaceutical Education and Research, Hyderabad, Telangana, 500037, India*
[2]*Department of Medicinal Chemistry, National Institute of Pharmaceutical Education and Research, Hyderabad, Telangana, 500037, India.*
**Corresponding author: manoj.dandekar@niperhyd.ac.in*

Abstract

Background: Chronic cerebral hypoperfusion induced vascular dementia is a leading cause of dementia after Alzheimer's disease. Oxyberberine is a key derivative of berberine, known for exerting profound beneficial effects in several neurological impairments. ROCK inhibitors have also been shown to have beneficial effects on neurological diseases.

Aim: To investigate the effects of oxyberberine in the SH-SY5Y neuroblastoma cell line and a vascular dementia rat model.

Methods: The MTT assay was performed in SH-SY5Y cells. Vascular dementia was induced in Sprague-Dawley rats by bilateral common carotid artery occlusion (BCCAO) surgery. The neurobehavioral changes were assessed using novel object recognition test (NORT) and Morris water maze (MWM), and elevated plus maze (EPM) assays. Oxyberberine treatment was given for 28 days, and blood and brains were obtained for molecular studies.

Results: In the molecular docking study, oxyberberine aligned comfortably at the active site of ROCK-1 and ROCK-2 and exhibited key hydrogen bond interactions with Lys105 and Lys121, respectively. Netarsudil showed hydrogen bond interaction with Lys105 and Asp202 and salt interaction with Asp160 at ROCK-1 and Lys121 at ROCK-2. The interactions at the active site of ROCK-1 and ROCK-2 were comparable with the oxyberberine, drug (netarsudil), and co-crystal. Rats underwent BCCAO surgery showed anxiogenic-like phenotype in EPM, and dramatic cognitive deficits in NORT and MWM assays. The administration of oxyberberine (50 and 100 mg/kg/day, peroral) or berberine (50 mg/kg/day, peroral) restored BCCAO-generated cognitive deficits. In molecular analysis, oxyberberine showed increased levels of hippocampal plasticity-related proteins synaptophysin and post-synaptic density protein (PSD-95) by inhibiting Rho-kinase signaling. Oxyberberine also modulated the levels of antioxidant enzymes and ROCK-1 and ROCK-2 expression.

Conclusion: These results suggest the potential of oxyberberine in reversing BCCAO-generated vascular dementia via modulation of Rho-kinase signaling pathway.

Keywords: Oxyberberine, Vascular dementia, ROCK-1, SH-SY5Y, BCCAO

P-030:

SCANNING ION-CONDUCTANCE MICROSCOPY FOR STUDYING β-AMYLOID AGGREGATE FORMATION ON LIVING CELL SURFACES

Vasilii S. Kolmogorov[1], Vangene Tay[2], Petr V. Gorelkin[1,3*]

[1]*National University of Science and Technology "MISIS", Moscow, 119049, Russian Federation*
[2]*Hi-Tech Instruments Sdn. Bhd., Selangor, 47120, Malaysia*
[3]*ICAPPIC Limited, London, NW10 6TD, United Kingdom*
**Corresponding author. Email: pg@icappicl.com*

Abstract

Background: Alzheimer's disease, the most common form of dementia, is characterized by progressive memory loss and cognitive decline. A central pathological factor is β-amyloid (Aβ) aggregation on neuronal membranes, which disrupts cellular function through calcium imbalance, oxidative stress, and membrane permeabilization. These aggregates activate kinase signaling pathways (such as p38 MAPK and Rho GTPases) and alter ion channel activity, leading to cytoskeletal remodeling, reduced membrane potential, and synaptic loss. Although changes in cellular stiffness (Young's modulus) after Aβ exposure have been reported, the link between amyloid aggregation and local mechanical properties of living cells remains unresolved.

Aim(s): To investigate how β-amyloid 1-42 (Aβ42) aggregates affect neuronal cell mechanics and correlate aggregate formation with cytoskeletal remodeling and membrane integrity.

Methods: Scanning ion-conductance microscopy (SICM) was used for topography and Young's modulus mapping of SH-SY5Y neuroblastoma cells. Correlative confocal microscopy with fluorescent Aβ42 (FAM-Aβ42) visualized aggregates, and membrane integrity was assessed with lipid dye DiD. Aβ peptides were prepared as 2.5 mM solutions in anhydrous DMSO and incubated for 1 hour at room temperature to ensure complete dissolution.

Results: Aβ42 exposure increased cell stiffness, while localized aggregates corresponded to reduced modulus and pore-like disruptions. Correlative confocal imaging confirmed aggregate formation with associated actin cytoskeleton remodeling.

Conclusion: Aβ42 aggregates drive cytotoxicity through stiffening, pore formation, and cytoskeletal disruption. Correlative SICM offers a multimodal approach for studying amyloid-induced dysfunction in living cells, providing insights into mechanisms of early neurodegeneration.

Keywords: Alzheimer's disease; β-amyloid aggregation; Scanning ion-conductance microscopy; Neurodegeneration, Young's modulus

---

P-031:

THERAPEUTIC TARGETING OF CENTRAL AGMATINE SIGNALING FOR INTERVENTION OF HUNTINGTON'S DISEASE

Raj Katariya[1], Brijesh Taksande[2*]

[1]Division of Neuroscience, Smt. Kishoritai Bhoyar College of Pharmacy, New Kamptee, Nagpur-441 002
[2]Division of Neuroscience, Smt. Kishoritai Bhoyar College of Pharmacy, New Kamptee, Nagpur-441 002
**Corresponding Author Email: brijeshtaksande@gmail.com*

Abstract

Background: Huntington's disease (HD) is a fatal, inherited progressive neurodegenerative movement disorder characterized by a triad of motor incoordination, cognitive decline, and neuropsychiatric symptoms with subsequent loss of neurons in the striatum. Despite the knowledge of the exact cause and substantial efforts of the multidisciplinary scientific community, a definitive cure for this devastating disorder remains elusive. Recent studies suggest that central agmatine signaling may represent a novel therapeutic target for the management of HD. Agmatine, a biogenic amine derived from the decarboxylation of L-arginine, has demonstrated promising pharmacological actions in neurological disorders.

Aim(s): To investigate the therapeutic potential of targeting central agmatine signaling in the 3-Nitropropionic acid (3-NP) rat model of HD.

Methods: Rats were randomly assigned to five experimental groups (n = 15 per group). The 3-NP group received intraperitoneal injections of 3-NP (10 mg/kg) on alternate days. Starting from day 10, rats in the treatment group were administered agmatine (5 - 20 µg/rat) via intrastriatal injections daily until day 21 of the experimental protocol. Control rats received equivalent volumes of artificial cerebrospinal fluid (aCSF) and saline. All animals were housed under standard laboratory conditions with free access to food and water, and behavioral as well as biochemical assessments were performed at defined time points.

Results: The findings of the current investigation revealed that repeated intrastriatal agmatine injection significantly restored behavioral abnormalities such as motor deficits, cognitive dysfunction, anxiety, and depression-like behavior (mean ± SEM, n = 15 per group) along with neurochemical dysregulation within the striatum in 3-NP-treated rats (mean ± SEM, n = 3 per group). Moreover, agmatine treatment attenuated striatal astrogliosis, reduced brain lesions, and normalized the levels of BDNF, IL-6, IL-10, and TNF-α in the striatum (mean ± SEM, n = 6 per group). Importantly, agmatine intervention was able to preserve the neuronal integrity in discrete brain areas (mean ± SEM, n = 3 per group). Notably, agmatine treatment mitigated mitochondrial swelling and restored succinate dehydrogenase enzyme levels in the striatum (mean ± SEM, n = 3 per group). Interestingly, we found that chronic 3-NP exposure was associated with reduced endogenous agmatine levels across different brain regions, signifying that targeting central agmatine signaling may offer a new therapeutic approach for mitigating the consequences of HD.

Conclusion: In conclusion, our research work provides strong preclinical evidence that targeting central agmatine signaling may confer neuroprotective effects against 3-

NP-induced neurotoxicity, warranting further exploration for clinical application in HD and its associated complications.

Keywords: Huntington's disease, Agmatine, Striatum, 3-Nitropropionic acid.

P-032:

HUMAN STEM CELL FROM APICAL PAPILLA: THE POTENTIAL ALTERNATIVE CELL SOURCE FOR NEURONAL DIFFERENTIATION

Anupong Thongklam Songsaad[1*], Tatcha Balit[2], Tarinee Chodchavanchai[3], Amarin Thongsuk[3], Nisarat Ruangsawasdi[4], Charoensri Thonabulsombat[3]

[1]Department of Anatomy, Faculty of Dentistry, Mahidol University, Bangkok, Thailand, 10400
[2]Department of Medical Science, School of Medicine, Walailak University, Nakhonsrithammarat, Thailand, 80160
[3]Department of Anatomy, Faculty of Science, Mahidol University, Bangkok, Thailand, 10400
[4]Department of Pharmacology, Faculty of Dentistry, Mahidol University, Bangkok, Thailand, 10400
*Corresponding author. Email: Anupong.son@mahidol.ac.th

Abstract

Background: To explore neurogenesis, studying via an animal model is required. However, it presented ethical considerations, donor-site morbidity, and interspecies variability. Therefore, the alternative cell source with neuronal differentiation ability should be demonstrated.

Aim(s): This study aims to demonstrate the neuronal differentiation ability of human stem cells from apical papilla (hSCAPs) as the potential alternative source to generate neuronal cells.

Methods: The hSCAPs were established from impacted human third molars of Thai patients (n = 3, 18-21 years old) and characterized by the properties of Mesenchymal stem cells (MSCs). The neuronal differentiation capacities of the characterized hSCAPs were demonstrated with 3 protocols. Firstly, these hSCAPs were administered with 2 phases of neurogenic induction medium to demonstrate 2D neuronal differentiation. Secondly, the neural stem cells (NSCs) induction was performed by forming 3D neurospheres. Finally, neurogenic maturation was triggered to investigate. Notably, the differentiated cells were further verified for their typical characteristics of neuronal cells, including observation of cell morphology, identification of Nissl substance by Cresyl violet staining, neurogenic-associated proteins/genes expression, and functional neuronal activity via intracellular calcium oscillation.

Results: The isolated cells from human apical papilla tissue were characterized as MSCs, namely hSCAPs. Under 2D neuronal induction, these hSCAPs were differentiated into neuronal-like cells, which presented the neuronal appearance, positively revealed the Nissl substance, highly expressed the neuronal-related genes, and functional neuronal activity. Furthermore, hSCAPs were aggregated into 3D neurospheres, which consisted of NSCs, highly expressed the early neuronal stage markers. Consequently, these NSCs can be matured into the functional mature neuronal cells under neurogenic maturation-inducing environment. The expression of late neuronal stage markers obviously investigated and functional neuronal activity can be detected.

Conclusion: The hSCAPs demonstrated the differentiation abilities into neuronal cells, NSCs, and functional mature neuronal cells, which serve as the potential alternative source for neuroscience research.

Keywords: Alternative cell source, Dental stem cells, Human stem cells from apical papilla, Neuronal differentiation, Neuronal cells

---

P-033:

PROTECTIVE EFFECT OF MELATONIN AGAINST OXIDATIVE STRESS IN CEREBRAL ISCHEMIC RATS

Naparat Promyoo[1], Jirakhamon Sengking[2], Mathurada Saephu[1], Chainarong Tocharus[2], Jiraporn Tocharus[1*]

[1]Department of Physiology, Faculty of Medicine, Chiang Mai University, Chiang Mai, Thailand, 50200.
[2]Department of Anatomy, Faculty of Medicine, Chiang Mai University, Chiang Mai, Thailand, 50200.
**Corresponding author. Email: jiraporn.tocharus@cmu.ac.th*

Abstract

Background: Globally, ischemic stroke is a leading cause of death, disability, and memory impairment. Cerebral ischemia occurs following the occlusion of the cerebral arteries, triggering irreversible brain injury that leads to oxidative stress and damage to lipids and proteins, thereby activating cell death, contributing to neuronal loss and neurological deficits. Recently, melatonin, a neurohormone with potent antioxidant and anti-inflammatory properties, has shown protective effects in stroke models.

Aim: This study aimed to investigate the ability of melatonin to reduce oxidative stress after permanent cerebral ischemia in rats

Methods: Male Wistar rats underwent surgery to induce cerebral ischemia. An intraluminal filament was inserted through the ECA stump and advanced into the ICA to occlude blood flow. The 24 rats were randomly divided into four groups (n = 6 per group): sham, sham + melatonin, permanent middle cerebral artery occlusion (pMCAO), and pMCAO + melatonin 10 mg/kg. After 14 days, neurological deficit scores and the rotarod test were determined. The rats were then sacrificed to remove the brain for infarct volume measurement using 2,3,5-triphenyl tetrazolium chloride (TTC) staining. Morphological alterations of neurons were examined by H&E staining. In addition, the expression of oxidative stress-related proteins was analyzed by Western blotting. Moreover, the MDA level was analyzed by thiobarbituric acid reactive substances (TBARS) assay.

Results: Our results showed that neurological deficit scores and infarct volume in the pMCAO group was significantly increased versus the sham group and decreased in the pMCAO + melatonin group compared to the pMCAO group. On the other hand, performance in the rotarod test was significantly decreased in the pMCAO versus the sham group and increased in the pMCAO + melatonin group versus the pMCAO group. Moreover, pMCAO + melatonin also reduced oxidative stress, as indicated by downregulation of oxidative stress markers such as malondialdehyde (MDA) and 4-hydroxynonenal (4HNE).

Conclusion: The results showed that melatonin can diminish the neurodegeneration induced by ischemic stroke.

Keywords: Melatonin, Oxidative stress, Cell death, Permanent cerebral ischemia

---

P-034:

PROTECTIVE EFFECT OF APOCYNIN ON COGNITIVE IMPAIRMENT IN CHRONIC CEREBRAL HYPOPERFUSION-INDUCED RATS

Mathurada Saephu[1], Phakkawat Thangwong[2], Jirakhamon Sengking[3], Naparat Promyoo[1], Chainarong Tocharus[3], Jiraporn Tocharus[1]*

[1]Department of Physiology, Faculty of Medicine, Chiang Mai University, Chiang Mai, Thailand, 50200.
[2]Department of Medical Science, School of Medicine, Walailak University, Nakhon Si Thammarat, Thailand, 80160.
[3]Department of Anatomy, Faculty of Medicine, Chiang Mai University, Chiang Mai, Thailand, 50200.

*Corresponding author. E-mail: jiraporn.tocharus@cmu.ac.th

Abstract

Background: Vascular dementia (VaD) is caused by reduced cerebral blood flow, and its pathology is closely related to Alzheimer's disease (AD). It often results from chronic cerebral hypoperfusion (CCH), which leads to oxidative stress, mitochondrial dysfunction, and induction of AD-like pathology, all of which contribute to cognitive impairment. Recent studies have demonstrated that apocynin, a compound with potent antioxidants and anti-inflammatory properties, exerts neuroprotective effects in dementia models.

Aim(s): This study aimed to evaluate the therapeutic effects of apocynin against cognitive impairment in CCH-induced rats.

Methods: CCH was induced in male Wistar rats by bilateral occlusion of the common carotid arteries (2VO). All rats were randomly assigned to five groups: Sham, Sham+Apocynin, 2VO, 2VO + Apocynin, and 2VO+N-acetylcysteine. At the end of the experiment, memory performance was evaluated using the novel object location (NOL) test. Subsequently, all rats were sacrificed, and their brains were collected to determine the levels of malondialdehyde (MDA), and subjected to western blotting of proteins related to oxidative stress.

Results: Our results showed that the discrimination index obtained from the NOL test was significantly increased in the 2VO + Apocynin group compared to the 2VO group. Furthermore, the level of MDA was significantly decreased in the 2VO + Apocynin group compared to the 2VO group. Western blot analysis also showed downregulation of oxidative stress-related proteins in the 2VO + Apocynin group compared to the 2VO group.

Conclusion: The study found that apocynin exerts neuroprotective effects and improves cognitive impairment in CCH conditions by reducing oxidative stress. These findings suggest that apocynin may serve as a promising therapeutic agent for preventing the progression of VaD.

Keywords: Apocynin, Vascular dementia, Chronic cerebral hypoperfusion, Cognitive impairment, Oxidative stress

---

Background

Dementia is a progressive condition characterized by a decline in cognitive functions. Importantly, it is not a single disease but a broad term encompassing various symptoms, including memory loss, behavioral changes, emotional disturbances, and impaired learning abilities (Arvanitakis et al. 2019). Alzheimer's disease (AD) is the most common cause of dementia. It is characterized by hallmark pathological features, including amyloid beta (Aβ) plaques, neurofibrillary tangles (NFTs), and cholinergic system dysfunction (Akhtar et al., 2024; Lee et al., 2022). However, AD is frequently accompanied by cerebrovascular dysfunction, which is strongly associated with vascular dementia (VaD). VaD, the second most prevalent form of dementia after AD, arises from reduced cerebral blood flow due to impaired vascular circulation (Iliff et al. 2013). VaD can be caused by chronic cerebral hypoperfusion (CCH), which leads to reduced blood flow to the brain (Ladecola et al. 2013). Prolonged CCH leads to a reduction in the supply of oxygen and nutrients to the brain, thereby initiating a hypoxic state that can impair neuronal metabolism and function. The resulting energy depletion impairs the function of the Na^+/K^+-ATPase pump, leading to neuronal depolarization (Rajeev et al. 2023). This depolarization activates NADPH oxidase 4 (NOX4), which generates reactive oxygen species (ROS) by transferring electrons to molecular oxygen, contributing to neuronal cell damage and cognitive impairment observed in VaD (Kimura et al. 2025, Boonpraman et al. 2023). Apocynin, a naturally occurring compound known for its potent anti-inflammatory and antioxidant effects (Heumüller et al. 2008), has been widely studied for its ability to inhibit NOXs. By reducing oxidative stress and limiting ROS overproduction (Paravicini et al. 2008, Kinkade et al. 2013), apocynin protects against oxidative damage to DNA, lipids, and proteins, thereby providing significant neuroprotection (Trumbull et al. 2011).

Materials and Methods

Animal and experimental groups

Male Wistar rats, weighing 250–300 g, were obtained from Nomura Siam International Co., Ltd. (Bangkok, Thailand). The animals were housed under controlled environmental conditions at 24 ± 1 °C with a 12-hour light/dark cycle, and had free access to standard food and water. All experimental procedures were approved by the Institutional Animal Care and Use Committee of Chiang Mai University. All rats were randomly divided into five groups: (1) sham-operated (Sham); (2) sham treated with apocynin (Sham + Apo); (3) two-vessel occlusion (2VO); (4) the 2VO treated with apocynin (2VO + Apo); (5) the 2VO treated with 2VO + N-acetylcysteine (2VO + NAC). Following 2VO or sham surgery performed on Day 0, treatment with apocynin (25 mg/kg body weight) or NAC (50 mg/kg body weight) was initiated on Day 1 and administered orally via gavage once daily for 14 consecutive days. Then, the rats were sacrificed by decapitation, and brain samples were collected for further analysis.

Two-vessel occlusion protocol

Rats underwent either a sham operation or two-vessel occlusion (2VO) surgery. Briefly, the animals were anesthetized with Zoletil (30 mg/kg) and xylazine (10 mg/kg) administered via intraperitoneal injection and then placed in the supine position on a heated pad to maintain body temperature. A midline incision was made in the neck to expose the common carotid arteries (CCAs), which were carefully separated from the vagus nerve. Both CCAs were then occluded using 5-0 silk suture loops (Fig.1). Rats in the sham group underwent the same surgical procedure, except that the arteries were not occluded.

Novel object location test

The test was conducted in an open-field arena made of black polypropylene sheets (30 cm × 30 cm × 30 cm). Two identical objects made of non-toxic plastic or metal were used. Both the objects and the arena were cleaned with 70% ethanol between trials to eliminate olfactory cues. The Novel Object Location (NOL) test consisted of three phases conducted over three consecutive days. The habituation phase, conducted on day 11, involved placing each animal individually into the empty arena for 5–10 minutes to allow acclimatization to the environment in the absence of any objects. On day 12, two identical objects were placed symmetrically at fixed locations within the arena. In the test phase on day 13, one of the objects was moved to a new location within the arena. Each animal was allowed to explore the objects, and the time spent exploring each object was recorded. Exploration was defined as the animal directing its nose toward the object at ≤2 cm or physically touching it. The discrimination index (DI) was subsequently calculated.

Determination of malondialdehyde (MDA) levels

Hippocampal tissues were homogenized in phosphate-buffered solution. The MDA level was examined via the reaction of thiobarbituric acid (TBA). The product was measured by using a microplate reader (BioTek Instruments, Inc, Winooski, VT, USA) at a wavelength of 532 nm.

Western blotting analysis

The total protein in brain tissues was determined using the Bradford assay (Bio-Rad, USA) with bovine serum albumin (BSA) as a standard. For Western blot analysis, 25 µg of protein from each sample was separated by sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) on 10–15% polyacrylamide gels to resolve proteins according to their molecular weights. The proteins were then transferred onto polyvinylidene difluoride (PVDF) membranes (Immobilon-P, Millipore, Bedford, MA, USA). To block non-specific binding, membranes were incubated in 5% nonfat milk for 1 hour at room temperature. Subsequently, membranes were incubated overnight at 4°C with the appropriate primary antibody (anti-NOX4). The following day, membranes were washed with Tris-buffered saline with 0.1% Tween 20 (TBST) and incubated with horseradish peroxidase (HRP)-conjugated secondary antibody. Protein bands were visualized using an enhanced chemiluminescence (ECL) substrate and imaged with a ChemiDoc imaging system (Aplegen Gel company, Inc., San Francisco, CA, USA). Quantitative analysis of band intensities was performed using ImageJ® software (National Institutes of Health, Bethesda, MD, USA).

Statistical analysis

All data were presented as mean ± standard error of the mean (SEM) and analyzed using one-way analysis of variance (ANOVA), followed by Tukey's post hoc test for multiple comparisons between groups. A p-value of less than 0.05 was considered statistically significant.

Results

Administration of apocynin significantly attenuated cognitive deficits induced by chronic cerebral hypoperfusion (CCH) in the 2-vessel occlusion (2VO) rat model.

Behavioral assessment using the novel object location (NOL) test demonstrated a significant improvement in the discrimination index in the 2VO + apocynin group compared to the untreated 2VO group, indicating preserved recognition memory (Fig. 2). The 2VO group showed a significantly increased MDA level compared with the sham group; on the contrary, the 2VO+apocynin and 2VO+NAC groups showed significantly decreased the MDA levels (Fig. 3). Western blot analysis further confirmed an upregulation of NADPH oxidase 4 (NOX4) in the 2VO group, consistent with enhanced ROS generation under hypoxic-ischemic conditions. Importantly, apocynin administration markedly downregulated NOX4 expression levels, indicating its inhibitory effect on NADPH oxidase activity (Fig. 4). These findings indicate that apocynin effectively mitigates oxidative stress, neuronal damage, and AD-like pathology associated with chronic cerebral hypoperfusion.

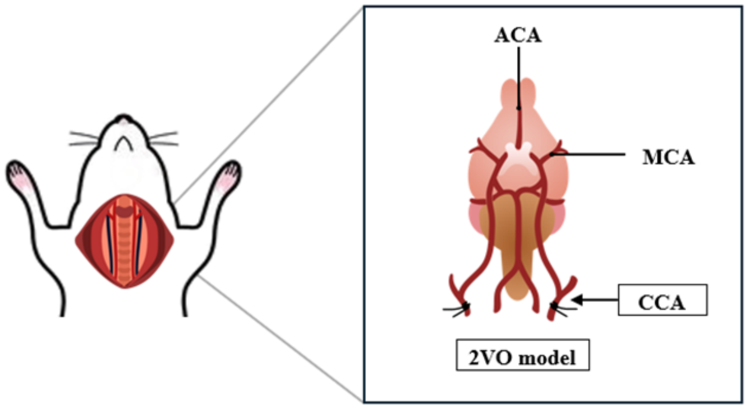
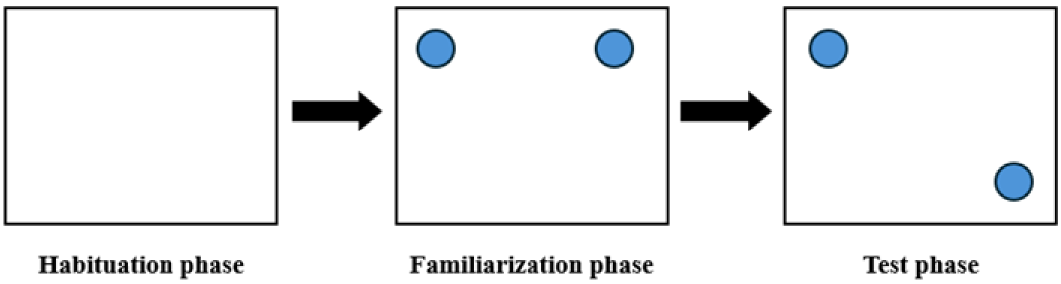


Figure 1. The two-vessel occlusion (2VO) model. ACA = Anterior cerebral artery, MCA = Middle cerebral artery, CCA = Common carotid artery.

A



B

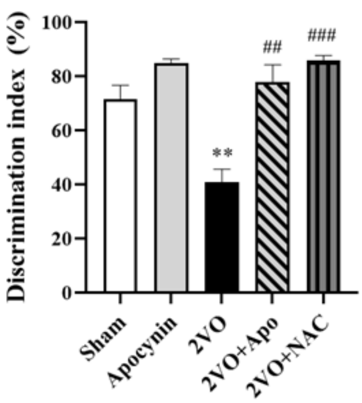


Figure 2. Apocynin alleviated cognitive impairment induced by chronic cerebral hypoperfusion. (A) The experimental protocol of NOL test. (B) Cognitive behavior was evaluated by the discrimination index. Values represented the mean ± SEM (n=3), $^{**}P <$ 0.01 versus sham group. $^{\#\#} P < 0.01$ and $^{\#\#\#} P < 0.001$ versus 2VO group.

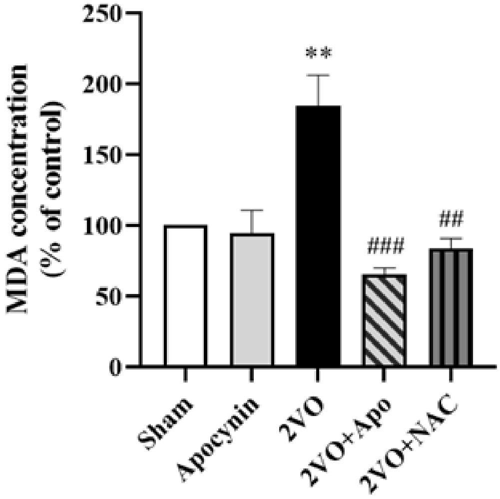


Figure 3. Apocynin suppressed the MDA level after 2VO in rats. Values represented the mean ± SEM (n=3), $^{**}P < 0.01$ versus sham group. $^{\#\#} P < 0.01$ and $^{\#\#\#} P < 0.001$ versus 2VO group.

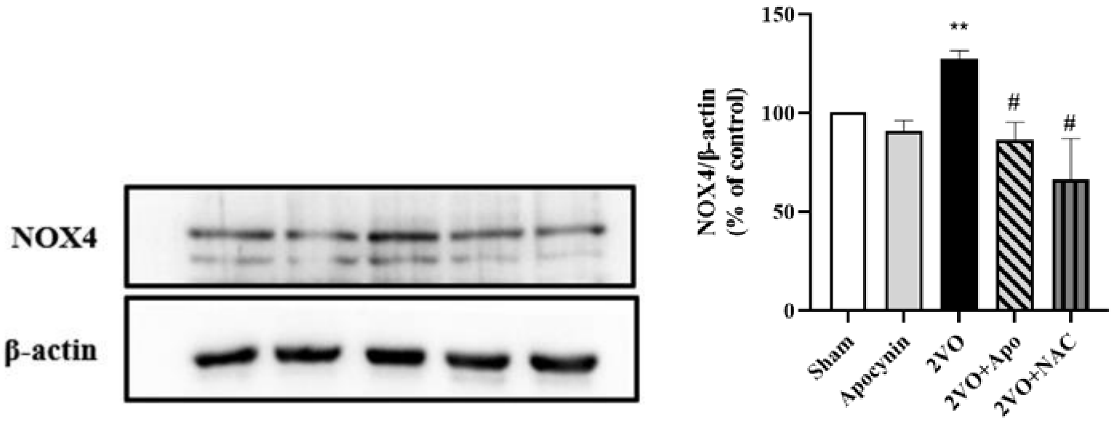


Figure 4. Apocynin downregulated NOX4 expression levels. (A) The representative Western blot analysis band of NOX4. (B) Quantification of the relative expression of NOX4. Values represented the mean ± SEM (n=3), $^{**}P < 0.01$ versus sham group. $^{\#}P <$ 0.05 versus 2VO group.

Discussion and Conclusion

The present study highlights the neuroprotective role of apocynin in alleviating cognitive deficits induced by CCH in the 2VO rat model. Apocynin treatment significantly improved cognitive performance, as evidenced by behavioral assessments. These findings align with apocynin's well-established pharmacological profile as a potent NOX inhibitor, particularly targeting NOX4, a major enzymatic source of ROS in the brain under ischemic and hypoxic conditions. Excessive ROS production following CCH is a key contributor to oxidative damage, mitochondrial dysfunction, and subsequent neuronal injury. By attenuating NOX4 expression. Compared to general antioxidants such as NAC, which exert ROS scavenging activity, apocynin offers more targeted inhibition of NOX enzymatic activity, thereby addressing the primary source of oxidative stress in ischemic and neurodegenerative contexts. This mechanistic specificity may underlie its superior efficacy in reducing neuronal oxidative damage and improving cognitive outcomes observed in this model. In summary, apocynin demonstrates a multifaceted neuroprotective mechanism in CCH-induced cognitive impairment. By inhibiting NOX4 activity, reducing oxidative stress, apocynin preserves neuronal structure and cognitive function. These findings support further investigation into apocynin as a promising therapeutic candidate for vascular dementia and potentially other neurodegenerative diseases with a vascular component.

Acknowledgements

This work was supported by Faculty of Medicine, Chiangmai University, Thailand

References

Arvanitakis, Z., Shah, R. C., & Bennett, D. A. (2019). Diagnosis and management of dementia. *JAMA, 322*(16), 1589–1599. https://doi.org/10.1001/jama.2019.4782

Akhtar, A., Singh, S., Kaushik, R., Awasthi, R., & Behl, T. (2024). Types of memory, dementia, Alzheimer's disease, and their various pathological cascades as targets for potential pharmacological drugs. *Aging Research Reviews, 96*, 102289. https://doi.org/10.1016/j.arr.2024.102289

Lee, J. H., Yang, D. S., Goulbourne, C. N., Im, E., Stavrides, P., Pensalfini, A., et al. (2022). Faulty autolysosome acidification in Alzheimer's disease mouse models induces autophagic build-up of Aβ in neurons, yielding senile plaques. *Nature Neuroscience, 25*(6), 688–701. https://doi.org/10.1038/s41593-022-01071-7

Iliff, J. J., Wang, M., Zeppenfeld, D. M., Venkataraman, A., Plog, B. A., Liao, Y., et al. (2013). Cerebral arterial pulsation drives paravascular CSF–interstitial fluid exchange in the murine brain. *Journal of Neuroscience, 33*(46), 18190–18199. https://doi.org/10.1523/JNEUROSCI.1592-13.2013

Iadecola, C. (2013). The pathobiology of vascular dementia. *Neuron, 80*(4), 844–866. https://doi.org/10.1016/j.neuron.2013.10.008

Rajeev, V., Chai, Y. L., Poh, L., Selvaraji, S., Fann, D. Y., Jo, D. G., et al. (2023). Chronic cerebral hypoperfusion: A critical feature in unravelling the etiology of vascular cognitive impairment. *Acta Neuropathologica Communications, 11*(1), 93. https://doi.org/10.1186/s40478-023-01609-2

Kimura, S., Iwata, M., Takase, H., Lo, E. H., & Arai, K. (2025). Oxidative stress and chronic cerebral hypoperfusion: An overview from preclinical rodent models. *Journal of Cerebral Blood Flow & Metabolism, 45*(3), 381–395. https://doi.org/10.1177/0271678X241234567

Boonpraman, N., & Yi, S. S. (2024). NADPH oxidase 4 (NOX4) as a biomarker and therapeutic target in neurodegenerative diseases. *Neural Regeneration Research, 19*(9), 1961–1966. https://doi.org/10.4103/1673-5374.421031

Okamoto, Y., Yamamoto, T., Kalaria, R. N., Senzaki, H., Maki, T., Hase, Y., et al. (2012). Cerebral hypoperfusion accelerates cerebral amyloid angiopathy and promotes cortical microinfarcts. *Acta Neuropathologica, 123*(3), 381–394. https://doi.org/10.1007/s00401-011-0939-5

Kitazawa, M., Yamasaki, T. R., & LaFerla, F. M. (2004). Microglia as a potential bridge between the amyloid beta-peptide and tau. *Annals of the New York Academy of Sciences, 1035*, 85–103. https://doi.org/10.1196/annals.1332.006

Wang, J. Z., Xia, Y. Y., Grundke-Iqbal, I., & Iqbal, K. (2013). Abnormal hyperphosphorylation of tau: Sites regulation, and molecular mechanism of neurofibrillary degeneration. *Journal of Alzheimer's Disease, 33*(Suppl 1), S123–S139. https://doi.org/10.3233/JAD-2012-129037

Heumüller, S., Wind, S., Barbosa-Sicard, E., Schmidt, H. H., Busse, R., & Schröder, K. (2008). Apocynin is not an inhibitor of vascular NADPH oxidases but an antioxidant. *Hypertension, 51*(2), 211–217. https://doi.org/10.1161/HYPERTENSIONAHA.107.100024

Paravicini, T. M., & Touyz, R. M. (2008). NADPH oxidases, reactive oxygen species, and hypertension: Clinical implications and therapeutic possibilities. *Diabetes Care, 31*(Suppl 2), S170–S180. https://doi.org/10.2337/dc08-s247

Kinkade, K., Streeter, J., & Miller, F. J. (2013). Inhibition of NADPH oxidase by apocynin attenuates progression of atherosclerosis. *International Journal of Molecular Sciences, 14*(8), 17017–17028. https://doi.org/10.3390/ijms140817017

Trumbull, K., McAllister, D., Gandelman, M., Fung, W., Lew, T., Brennan, L., et al. (2011). Diapocynin and apocynin administration fails to significantly extend survival in G93A SOD1 ALS mice. *Neurobiology of Disease, 45*(1), 137–144. https://doi.org/10.1016/j.nbd.2011.07.011

P-035:

RED ONION OLIGOSACCHARIDE SUPPLEMENTATION COUNTERACTS HIGH-FAT DIET-INDUCED COGNITIVE AND METABOLIC ABNORMALITIES IN RATS

Juefang Gan[1,2], Jittiporn Wongpun[3], Jirakhamon Sengking[1], Sivanan Sivasinprasasn[1], Apinun Kanpiengjai[4], Jiraporn Tocharus[5], Chainarong Tocharus[1]*

[1]*Department of Anatomy, Faculty of Medicine, Chiang Mai University, Chiang Mai, 50200, Thailand*
[2]*Department of Anatomy, Youjiang Medical University for Nationalities, Baise, 533000, Guangxi, China*
[3]*Department of Anatomy, Naresuan University, Phitsanulok, 65000, Thailand*
[4]*Division of Biochemistry and Biochemical Innovation, Department of Chemistry, Faculty of Science, Chiang Mai University, Chiang Mai, 50200, Thailand*
[5]*Department of Physiology, Faculty of Medicine, Chiang Mai University, Chiang Mai, 50200, Thailand*
Corresponding Author. E-mail: chainarong.t@cmu.ac.th

Abstract

Background: Fructooligosaccharides (FOSs) are natural prebiotics, known for their benefits in maintaining probiotic balance as well as their antioxidative and anti-inflammatory effects. Red onion is recognized for its antioxidative and anti-inflammatory properties and provides high-quality short-chain fructooligosaccharide (scFOS). ScFOSs have characteristics such as being low in calories, having low sweetness, and exhibiting strong thermal stability. They cannot be directly absorbed by the small intestine; however, they can be fermented by gut microbiota to help regulate the balance of intestinal microorganisms. However, it remains unclear whether scFOS derived from red onion exerts neuroprotective and ameliorative effects against obesity-related damage and related gut-brain pathways.

Aim: To elucidate the potential functions of scFOS from red onion on body indices and neurodegeneration in obese rats.

Methods: Thirty male Wistar rats were randomly divided into five groups: normal diet (ND), high-fat diet (HFD), high-fat diet with scFOS 500 mg/kg/day (HFD+scFOS500), high-fat diet with scFOS 1000 mg/kg/day (HFD+scFOS1000), and high-fat diet with atorvastatin 10 mg/kg/day (HFD+ATOR). Each group was fed either a normal diet or HFD for 16 weeks to induce obesity models, after obesity was established which the HFD subgroups were treated with scFOS or ATOR for 8 weeks. HFD+ATOR group served as a pharmacological control for metabolic improvement or neuroprotection. scFOS used in this study was prepared and purified from red onions (*Allium cepa var. viviparum*). Body weight (BW) and food/water intake were recorded. Cognitive function was assessed by the Morris water maze (MWM) test.

Results: Body weight and Lee's index increased markedly in HFD groups versus ND. The scFOS treatment group exhibited a significant reduction in body weight gain (19%) and Lee's index compared to the HFD control group. Spatial learning and memory impairments were partially improved in the scFOS and ATOR groups compared to rats receiving only the HFD as demonstrated by significantly longer escape latency and decreases in platform crossing. However, spatial learning and memory dysfunction were slightly moderated in the scFOS and ATOR groups versus HFD. These findings indicate

that HFD induces obesity and cognitive impairment, while scFOS shows beneficial effects in ameliorating neurodegeneration in HFD-fed rats.

Conclusion: scFOS demonstrated potential to improve body indices and attenuate neurodegenerative damage associated with chronic HFD-induced obesity via anti-inflammatory mechanisms. Further mechanistic studies are required to elucidate the gut–brain pathways through which scFOS confers neuroprotection in the context of obesity.

Keywords: high fat diet; obesity; short chain fructooligosaccharide; atorvastatin; cognitive dysfunction

----

P-036:

TCU412 EXTRACT EXERTED A PROTECTIVE EFFECT AGAINST CORTICOSTERONE-INDUCED CYTOTOXICITY IN HT22 CELLS, POTENTIALLY THROUGH MODULATION OF THE FERROPTOSIS PATHWAYS

Nhan Do Thi Thuy[1], Peeraporn Varinthra[1], Ingrid Y. Liu[1*]

[1]*Institute of Medical Sciences, Tzu Chi University, Hualien, Taiwan, 97004*
**Corresponding author. Email*: ycliu@gms.tcu.edu.tw

Abstract

Background: Depression is a globally prevalent psychiatric disorder. The limitations of conventional antidepressants, including adverse effects and treatment resistance, underscore the urgent need for novel therapeutic strategies. Corticosterone (CORT), a stress hormone implicated in depression, induces neuronal cell death, including ferroptosis. TCU412, an herbal medicine with antioxidant and neuroprotective properties, reduces mitochondrial reactive oxygen species and preserves mitochondrial integrity, making it a promising candidate for depression.

Aim(s): This study aimed to evaluate the neuroprotective potential of TCU412 extract in mitigating CORT-induced toxicity in HT22 mouse hippocampal neurons, with a specific focus on ferroptosis pathways.

Methods: HT22 cells were treated with TCU412 (0.16 - 10 µg/mL) and CORT (25 – 400 µM), after which cytotoxicity was assessed using the MTT assay. To evaluate the protective effects of TCU412, co-treatments with TCU412 and CORT were performed, and cell viability was measured via both MTT and LDH assays. Expression of ferroptosis-related markers was examined using western blotting and immunocytochemistry/immunofluorescence staining (ICC/IF).

Results: TCU412 demonstrated no cytotoxicity at concentrations of 0.16, 0.8, 4, and 10 µg/mL; however, concentrations of 20 and 100 µg/mL induced toxicity at both 24 and 48 hours. CORT at 400 µM significantly reduced cell viability after 24 hours and was selected for co-treatment experiments. Co-administration of TCU412 at 4 µg/mL provided no protection, whereas 10 µg/mL conferred measurable protection against CORT-induced cell death. Western blotting revealed that CORT treatment (400 µM) tended to reduce GPX4 expression while significantly increasing CD71 (transferrin receptor) and HO-1 expression. ICC/IF analysis showed that reactive oxygen species, CD71, HO-1, MDA, and 4-HNE levels were elevated following CORT treatment but were reduced in cells co-treated with TCU412 (10 µg/mL).

Conclusion: TCU412 may exert protective effects against CORT-induced neurotoxicity via ferroptosis pathways. Future studies will extend to assessing iron accumulation, GPX4 expression, and ferroptosis inhibitors *in vitro;* additionally, animal models of depression to evaluate neuroplastic adaptations and behavioral outcomes.

Keywords: depression, corticosterone, herbal medicine, ferroptosis

---

P-037:
ESTABLISHMENT OF A DROSOPHILA MODEL OF CHARCOT-MARIE-TOOTH TYPE 4B3

Titaree Yamsri and Hideki Yoshida[1,*]

[1]*Department of Applied Biology, Kyoto Institute of Technology, Kyoto, Japan, 606-8585.*
**Corresponding author. Email: hyoshida@kit.ac.jp*

Abstract

Background: Charcot-Marie-Tooth disease type 4B3 (CMT4B3) is a hereditary peripheral neuropathy characterized by demyelination and axonal loss, leading to progressive motor and sensory impairments. The disease is caused by variants in the SET Binding Factor 1 (*SBF1*) gene, which encodes the Myotubularin-Related Protein 5 (MTMR5). Although MTMR5 has been implicated in the suppression of autophagy in neurons, its precise function remains largely uncharacterized, and no effective therapeutic intervention is currently available.

Aim(s): To gain mechanistic insight into CMT4B3 and facilitate the identification of potential therapeutic targets, we aim to establish a genetically modified *Drosophila melanogaster* disease model.

Methods: Motor neuron-specific knockdown of the *Sbf,* a *Drosophila* ortholog of human *SBF1*, was achieved using RNA interference combined with the GAL4/UAS system. Locomotor ability was assessed in third instar larvae through the larval crawling assay. The larval neuromuscular junction (NMJ) morphology, presynaptic terminals, was visualized by immunostaining with FITC-conjugated anti-horseradish peroxidase IgG, followed by observation with a confocal microscope.

Results: Locomotor ability was significantly impaired in the motor neuron-specific *Sbf* knockdown larvae. Morphological analysis of the NMJs revealed distinct structural abnormalities, including increased numbers of both synaptic boutons and branches.

Conclusion: These results demonstrate that motor neuron-specific knockdown of *Sbf* in *Drosophila* causes locomotor defects accompanied by morphological defects of the NMJ. Further analyses will establish this model as a valuable and robust platform for genetic and pharmacological screening, and for elucidating disease mechanisms and identifying novel therapeutic strategies.

Keywords: CMT4B3, Charcot-Marie-Tooth disease, *Sbf*, *Drosophila*, neuropathy

---

P-038:

NEUROPROTECTIVE EFFECTS OF TCU 451 AGAINST AMYLOID BETA-INDUCED APOPTOSIS AND OXIDATIVE STRESS IN HT22 CELLS

Ismat Noor Paromi [1], Peeraporn Varinthra [1], Ingrid Y. Liu [1*]

[1]*Institute of Medical Sciences, Tzu Chi University, Hualien, Taiwan, 97004*
**Corresponding author, e-mail:* ycliu@gms.tcu.edu.tw

Abstract

Background: Excessive amyloid-beta (Aβ) accumulation is a hallmark of Alzheimer's disease (AD), driving neuronal apoptosis, oxidative stress, and cognitive decline. Current AD treatments are limited by side effects and lack of efficacy, highlighting the need for alternative therapies. TCU 451, a traditional Chinese herbal extract with demonstrated anti-inflammatory and antioxidant capabilities, may offer a promising natural approach for AD treatment.

Aim(s): In this study, we investigated whether TCU 451 leaves extract could protect against Aβ-induced neurotoxicity.

Methods: HT22 mouse hippocampal cells were cultured and exposed to Aβ oligomers to induce neurotoxicity. Cells were treated with TCU 451 extract (12.5–400 μg/ml) for 72 hours to evaluate cytotoxicity and neuroprotection. Cell viability was assessed by MTT assay, oxidative stress by DHE staining, and membrane integrity by LDH assay. Apoptosis was measured with TUNEL, while caspase-3/cleaved caspase-3 expression was detected by immunocytochemistry. BDNF and phosphorylated CREB (p-CREB) levels were analyzed by western blot analysis to explore mechanisms.

Results: MTT assay revealed that TCU 451 was non-toxic to HT22 cells at concentrations 12.5 to 200 μg/ml after 72 hours, while a higher dose of 400 μg/ml showed cytotoxic effects. Treatment with 50 and 200 μg/ml TCU 451 significantly improved cell viability and reduced ROS levels. At 200 μg/ml, TCU 451 also mitigated Aβ-induced membrane damage and reduced LDH leakage. TUNEL assay and immunocytochemistry demonstrated that both 50 and 200 μg/ml TCU 451 significantly reduced apoptotic cell death and suppressed the expression of caspase-3 and cleaved caspase-3. Furthermore, Aβ exposure decreased BDNF levels and showed a downward trend in p-CREB expression, while TCU 451 treatment restored BDNF expression and showed an increasing trend in p-CREB levels, indicating a potential mechanism for its neuroprotective activity.

Conclusion: TCU 451 showed protective effects against Aβ-induced toxicity in HT22 cells by reducing oxidative stress, inhibiting apoptosis, and modulating BDNF expression. However, further *in vivo* studies are required to validate these findings and clarify its therapeutic potential for AD.

Keywords: Alzheimer's disease, amyloid beta, oxidative stress, apoptosis, TCU 451

P-039:

PRUNUS DOMESTICA L. EXTRACT ATTENUATES D-GALACTOSE–INDUCED IMPAIRMENTS IN HIPPOCAMPAL NEUROGENESIS

Puncharatsm Pannin[1], Nataya Sritawan[1], Anusara Aranarochana[1], Apiwat Sirichoat[1], Jariya U Welbat[1*]

[1]*Department of Anatomy, Faculty of Medicine, Khon Kaen University, Khon Kaen 40002, Thailand*
**Corresponding author. Email: jariya@kku.ac.th*

Abstract

Background: The aging of the brain contributes significantly to both cognitive decline and disruptions in hippocampal neurogenesis. D-galactose (D-gal) exposure accelerates aging processes by promoting oxidative stress, leading to neuronal damage and impaired plasticity in animal studies. *Prunus domestica* L. (PD), also known as "Look-nai," is native to northern Thailand. Its fruits contain high levels of secondary metabolites, specifically polyphenols and flavonoids, which exhibit antioxidant activity and help to protect neural function.

Aim(s): This study investigates the protective effects of PD on hippocampal neurogenesis and the expression of key neurogenic and synaptic proteins in a D-gal–induced brain aging model.

Methods: Twelve-week-old male Sprague-Dawley rats were randomized into eight groups. The vehicle group received distilled water orally and normal saline intraperitoneally (i.p.). The D-gal group was given D-galactose (50 mg/kg, i.p.). Three groups received oral PD extract at doses of 75, 100, or 150 mg/kg, respectively. Three corresponding co-treatment groups received the same doses of PD extract along with D-gal (50 mg/kg, i.p.). Treatments were given once daily for 8 weeks. Hippocampi were collected, and the hemispheres were separated. One hemisphere was used for serial sectioning and immunostaining with nestin and Sox2, while the other was reserved for Western blot analysis of Sox2, DCX, BDNF, and PSD95.

Results: Immunofluorescence analysis showed that D-gal administration markedly reduced nestin- and Sox2-positive cells and lowered the levels of Sox2, DCX, BDNF, and PSD95, indicating impaired neurogenesis. Notably, co-treatment with PD and D-gal reversed these deficits by significantly restoring nestin and Sox2 expression and increasing the levels of Sox2, DCX, BDNF, and PSD95, thereby supporting neural stem cell activity, neuronal differentiation, and synaptic maturation.

Conclusion: PD effectively counteracts D-gal-induced hippocampal neurogenesis deficits by enhancing neural stem cell maintenance, promoting neuronal differentiation, and supporting synaptic maturation. These findings highlight its neuroprotective and pro-neurogenic potential.

Keywords: Brain aging, Hippocampal neurogenesis, *Prunus domestica* L., D-galactose

---

P-040:

PROTEOMIC INSIGHTS INTO HIPPOCAMPAL NEUROINFLAMMATION IN CUMS-INDUCED ANXIETY: THERAPEUTIC EFFECTS OF COMBINED CANNABIDIOL AND BRAHMI

Plaiyfah Janthueng[1,7], Wanfrutkon Waehama[1,7], Sawanya Charoenlappanit[2], Sittiruk Roytrakul[2], Prapapan Temkitthawon[3,4], Paweena Kaewman[5,7], Jureepon Roboon[5,7], Samur Thanoi[6], and Sutisa Nudmamud-Thanoi[2,7*]

[1] *Medical Science Graduate Program, Faculty of Medical Science, Naresuan University, Thailand*
[2] *National Center for Genetic Engineering and Biotechnology (BIOTEC), National Science and Technology Development Agency, Thailand*
[3] *Faculty of Pharmaceutical Sciences, Naresuan University, Thailand*
[4] *Center of Excellence for Natural Health Product Innovation and Center of Excellence for Innovation in Chemistry, Faculty of Pharmaceutical Sciences, Naresuan University, Thailand*
[5] *Department of Anatomy, Faculty of Medical Science, Naresuan University, Thailand*
[6] *School of Medical Sciences, University of Phayao, Thailand*
[7] *Center of Excellence in Medical Biotechnology, Naresuan University, Thailand*
* *The corresponding authors: E-mail address: sutisat@nu.ac.th*

Abstract

Background: Anxiety disorders are frequently associated with chronic stress and the most common mental disturbances. Chronic stress response disrupts the Hypothalamic-Pituitary-Adrenal axis, leading to an altered inflammatory system. Therefore, the hippocampal protein in inflammation pathway might be associated with stress-induced anxiety. Cannabidiol (CBD) is non-psychoactive component of Cannabis sativa and exerts a beneficial in anti-inflammatory. Similarly, Brahmi (or Bacopa monnieri) has a long history in Ayurvedic medicine for anti-inflammatory, anxiolytic, and memory-improving effects. Therefore, the co-administration of CBD and Brahmi may offer enhanced therapeutic benefits against neuroinflammation and anxiolytic properties.

Aim(s): This study employs proteomics analysis to identify and characterize the specific hippocampal protein targets implicated in neuroinflammation underlying chronic stress-induced anxiety and CBD-Brahmi treatment.

Methods: Male Sprague-Dawley rats (n=5-6 per group) were divided into 6 groups: control (1 group) and chronic unpredictable mild stress (CUMS) (5 groups). Rats underwent a 2-week CUMS regimen with daily stressors, followed by oral administration of water, CBD-enriched extract (2.5 & 5 mg/kg) or CBD isolate (2.5 & 5 mg/kg) mixed with Brahmi (20 mg/kg). On the final day, locomotor and anxiety-like behaviors were assessed using the open field test (OFT) and elevate plus maze test (EPM). Hippocampal proteomes were then analyzed by LC-MS/MS. The MS/MS data identified and quantified using Mascot MS/MS ions search by the NCBI protein database.

Results: Rats subjected to CUMS exhibited hyper-locomotor activity and anxiety-like behavior, which were attenuated by co-administration of CBD and Brahmi. Among the 14,096 quantified proteins, 4,243 were upregulated following CUMS. Functional enrichment analysis revealed that these proteins were primarily associated with neuroinflammatory pathways, including the cytokine-cytokine receptor interaction, p53, PI3K-Akt, and NF-kappa B signaling pathways. Notably, co-administration of CBD and

Brahmi reduced the expression of the NF-kappa B pathway proteins, such as Icam, Tnf, Tab2, Cd40, Pidd1, and Lta.

Conclusion: Collectively, this study suggests that the co-administration of CBD and Brahmi improved in anxiety and hippocampal neuroinflammation by modulating abnormally expressed proteins

Keywords: Hippocampus, Proteomics, Neuroinflammation, Chronic Unpredictable Mild Stress, Anxiety

---

P-041:

NEURONAL PNN DEFICIENCY LEADS TO BRAIN STRUCTURAL ALTERATIONS AND BEHAVIORAL ABNORMALITIES IN MICE

Shin-Meng Deng[1], Sujira Mukda[2], Steve Leu[3*]

[1]*Graduate Institute of Biomedical Sciences, College of Medicine, Chang Gung University, Taoyuan 333, Taiwan*
[2]*Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Salaya Campus, Nakornpathom 73170, Thailand*
[3]*Institute for Translational Research in Biomedicine, Kaohsiung Chang Gung Memorial Hospital, Kaohsiung 833, Taiwan*
**Corresponding author. Email: leu@cgmh.org.tw*

Abstract

Background: Aberrant expression of mRNA splicing regulators and consequent dysregulation of pre-mRNA alternative splicing in genes essential for neuronal maintenance have been implicated in the pathogenesis of neurodegenerative diseases.

Aim: This study investigated whether Pnn (Pinin), an mRNA splicing regulator, contributes to the initiation of neurodegenerative processes using an inducible, neuron-specific gene depletion mouse model (Pnn-nKO).

Methods: Six-week-old male and female CaMKII-CreERT2; *Pnn*$^{flox/flox}$ mice were administered tamoxifen to induce neuronal Pnn deletion. Histopathological analyses, brain MRI, and behavioral assessments were performed to evaluate structural and functional consequences.

Results: No overt morphological alterations were detected by H&E or immunofluorescence; however, MRI revealed ventricular dilatation and hippocampal atrophy in 10-month-old Pnn-nKO mice. At 5 months, Pnn-nKO mice showed no significant behavioral differences compared with wild-type (WT) controls. By 12 months, Pnn-nKO mice exhibited neurodegeneration-like deficits, including increased anxiety-like behavior (light–dark box), reduced social novelty preference (three-chamber test), impaired spatial memory and search strategy (Barnes maze), and markedly disrupted nesting behavior.

Conclusion: The depletion of Pnn leads to ventricular dilatation, hippocampal atrophy, and progressive behavioral impairments resembling neurodegeneration. Therefore, an mRNA splicing regulator, Pnn, contributes to the initiation of neurodegenerative processes.

Keywords: Pnn, Pinin, Neurodegeneration, Mouse model, Behavior deficits

---

P-042:

EFFECT OF CAFFEINE ON AMYLOID PRECURSOR PROTEIN AND ALZHEIMER'S MARKERS IN AMYLOID BETA-TREATED SH-SY5Y NEUROBLASTOMA CELLS

Chonnicha Subkod[1], Kornkanok Promthep[1], Theptharin Charuraksa[2], Jiraporn Panmanee[1] and Narisorn Kitiyanant[3]*

[1] Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom 73170, Thailand
[2] Office of Research and Innovation Affair, Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom 73170, Thailand
[3] Center for Advanced Therapeutics (CAT), Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom 73170, Thailand
Corresponding author. E-mail: narisorn.kit@mahidol.ac.th

Abstract

Background: Alzheimer's disease (AD) is a neurodegenerative disorder characterized by the accumulation of β-amyloid (Aβ) plaques and hyperphosphorylated tau protein tangles. These two pathological hallmarks lead to generalized neuronal loss in the brain, leading to memory loss and cognitive decline. Caffeine, the world's most widely consumed psychoactive substance, has gained attention as a regulator of the processing of amyloid precursor protein (APP), a key player in the development of AD. The amyloidogenic pathways consist of the sequential cleavage of APP by beta-secretase (BACE1) and gamma-secretase, leading to the generation of various forms of Aβ peptides, particularly the aggregation-prone Aβ42. Alternatively, APP can be cleaved by α-secretase (ADAM10), which precludes Aβ formation and promotes the non-amyloidogenic pathway. Therefore, we hypothesize that caffeine may promote this non-amyloidogenic processing of APP, thereby reducing the production of toxic Aβ species.

Aim(s): To investigate the effect of caffeine on exogenous Aβ42-induced changes in AD-related protein expression levels in SH-SY5Y Cells

Methods: SH-SY5Y human neuroblastoma cells were used as an *in vitro* model. An MTT assay was performed to determine the toxicity of caffeine at concentrations of 0, 10, 50, 100, 150, and 200 μM. The cells were pretreated with caffeine for 24 hours before the assay. The caffeine concentration at 10 μM was chosen for further experiments. AD-like pathology was induced by exposing the cells to 1 μM of Aβ42 following caffeine pretreatment. The protein expression levels of p-Tau181, APP, and BACE1 were evaluated by Western blot. All experiments were performed in triplicate.

Results: Caffeine concentrations between 10μM and 150μM were found to be non-toxic to SH-SY5Y cells, while 200μM reduced cell activity. Treatment with Aβ42 significantly upregulated p-Tau181, APP, and BACE1 expression. Pretreatment with caffeine significantly attenuated these protein levels, suggesting a potential protective effect of caffeine against Aβ42-induced pathology.

Conclusion: These findings demonstrate that caffeine can modulate the dysregulation of AD-related proteins in a cellular model, suggesting its potential as a protective agent for AD.

Keywords: Caffeine, Amyloid precursor protein, Alzheimer's disease, beta-secretase, tau

---

P-043:

TCU412 AMELIORATES AMYLOID BETA-INDUCED TOXICITY IN HT22 CELLS AND MEMORY IMPAIRMENTS IN 3XTG-AD MICE

Mubashir Raza[1], Ingrid Y. Liu[1]*

[1]*Institute of Medical Sciences, Tzu Chi University, Hualien, Taiwan, 97004*
Corresponding author. Email: ycliu@gms.tcu.edu.tw

Abstract

Background: Alzheimer's disease (AD) is a neurodegenerative disease that is caused by the extracellular deposition of amyloid beta (Aβ) and intracellular accumulation of neurofibrillary tangles (tau). Aβ induces synaptic dysfunction, impairment of working memory, spatial reversal learning deficits, and causes the downregulation of neurotrophic molecules and synaptic molecules. Currently, there is no adequate cure for AD. TCU412 is a Chinese herbal medicine that has demonstrated anti-inflammatory and neuroprotective properties.

Aim(s): This study investigates therapeutic potential of TCU412 extract on Aβ-induced cytotoxicity in HT22 cells and 3xTg-AD mice.

Methods: HT22 mouse hippocampal cells were exposed to TCU412 (0.1–1000 μg/mL) for 24 hours, followed by 5 μM amyloid beta for 48 hours, with cytotoxicity assessed using an MTT assay. Neuroprotection was evaluated in six-month-old male wildtype and 3xTg AD mice across untreated, sham, and TCU412-treated (100 mg/kg) groups, using the Open Field Test, T Maze, and Morris Water Maze. Western blot analysis of brain tissues examined amyloid beta, neurotrophic marker (BDNF), downregulated proteins (TrkB, pERK, pCREB) and synaptic (PSD-95, phospho-synapsin) markers.

Results: The results demonstrated that 1 and 10 μg/ml of TCU-412 extract rescued Aβ-induced cell death in HT22 cells. Additionally, 100 mg/kg of TCU412 improved spatial working memory and spatial reversal learning deficits in the 3xTg-AD mice. TCU412 upregulated BDNF, TrkB, pERK, pCREB, PSD-95, phospho-synapsin, and decreased Aβ in the hippocampi of the 3xTg-AD mice.

Conclusion: TCU412 rescued Aβ-induced cell death in HT22 cells and mitigated spatial working memory and spatial reversal learning via upregulation of neurotrophic and synaptic molecules and clearing of Aβ in 3xTg-AD mice.

Keywords: Alzheimer’s disease, amyloid beta, neurotrophins, 3xTg-AD, spatial reversal learning

---

P-044:

NEURONAL ACTIVATION IN CORTICOSTRIATAL CIRCUITS FOLLOWING REPEATED ANODAL-TRANSCRANIAL DIRECT CURRENT STIMULATION IN MICE

Siripaporn Kesyou[1,2], Bahrie Ramadan[2], Stephanie Dumontoy[2], Sutisa Nudmamud-Thanoi[3,4*], Vincent Van Waes[2*]

[1]*Faculty of Medical Science, Medical Science graduate program, Naresuan University, Phitsanulok, 65000*
[2]*Marie et Louis Pasteur University, INSERM, UMR 1322 LINC, Besançon, F-25000*
[3]*Department of Anatomy, Faculty of Medical Science, Naresuan University, Phitsanulok, 65000*
[4]*Centre of Excellence in Medical Biotechnology, Faculty of Medical Science, Naresuan University, Phitsanulok, 65000*
**Corresponding author. Email: vincent.van_waes@univ-fcomte.fr, sutisat@nu.ac.th*

Abstract

Background: Transcranial direct current stimulation (tDCS) is a non-invasive neuromodulation technique that delivers a weak, constant electric current across the scalp to modulate cortical excitability. Clinical studies reported symptom improvements in several psychiatric disorders, including depression and substance use disorder. However, the underlying neurobiological mechanisms remain unclear.

Aim(s): To map cortical and striatal activation in mice after a single tDCS session or a repeated 10- session protocol, the latter mirroring clinical regimens.

Methods: Mice were randomly assigned to a tDCS group, in which an electrode was surgically affixed to the skull (2 mm left and 2 mm anterior to bregma) for current delivery, or to a SHAM group, which underwent the same procedure without stimulation. In the single-session protocol, mice received one anodal stimulation (0.2 mA, 20 min). In the repeated protocol, stimulation was delivered twice daily for five consecutive days (10 session total). The brain was collected 1 hour after the last stimulation, c-fos immunohistochemistry was performed across 26 cortical areas and 23 striatal sectors to map tDCS-induced activation.

Results: Following a single session, c-fos expression was restricted to cortical regions, with maximal activation in the left hemisphere beneath the electrode. Cortical activation was most pronounced in the frontal areas, including the primary and secondary motor cortices, prelimbic cortex, and cingulate cortex, whereas no striatal activation was observed. After 10 sessions, cortical activation persisted but was attenuated relative to the single-session response, suggesting an adaptive neuroplastic process. In contrast, striatal activation emerged only after repeated stimulation and was bilateral (right and left hemispheres), involving the dorsal striatum as well as the medial shell, medial core, and lateral shell of the nucleus accumbens.

Conclusion: tDCS first enhances cortical excitability, and repeated sessions progressively recruit striatal circuits. This sequential modulation of corticostriatal activity may represent a key mechanism underlying the therapeutic effects of tDCS in neuropsychiatric disorders.

Keywords: Transcranial direct current stimulation, Mice, c-fos, Neuronal activation, Corticostriatal circuit

---

P-045:

IMP-1710: REPURPOSING UBIQUITIN C-TERMINAL HYDROLASE L1 (UCHL1) INHIBITION INTO A SENESCENCE-DRIVING STRATEGY

Napasakorn Verasaksuriya[1], Nattawadee Panyain[2], Kowit Hengphasatporn[3], Yasuteru Shigeta[3], Krisada Rungsang[1], Monruedee Sukprasansap[4], Tewin Tencomnao[5], Sunhapas Soodvilai[6,7], Benjamin Ongnok[6,8,*]

[1]Biomedical Science Program, Department of Pathobiology, Faculty of Science, Mahidol University, Bangkok 10400, Thailand
[2]Department of Biochemistry, Faculty of Science, Mahidol University, Bangkok 10400, Thailand
[3]Center for Computational Sciences, University of Tsukuba, Ibaraki 305-8577, Japan
[4]Food Toxicology Unit, Institute of Nutrition, Mahidol University, Nakhon Pathom 73170, Thailand
[5]Center of Excellence on Natural Products for Neuroprotection and Anti-Ageing, Department of Clinical Chemistry, Faculty of Allied Health Sciences, Chulalongkorn University, Bangkok 10330, Thailand
[6]Department of Physiology, Faculty of Science, Mahidol University, Bangkok 10400, Thailand
[7]Research Center of Transporter Protein for Medical Innovation, Department of Physiology, Faculty of Science, Mahidol University, Bangkok 10400, Thailand
[8]Center for Neuroscience, Faculty of Science, Mahidol University, Bangkok 10400, Thailand
**Corresponding author. Email: benjamin.ong@mahidol.ac.th*

Abstract

Background: Ubiquitin C-terminal hydrolase L1 (UCHL1) is a neuron-enriched deubiquitinating enzyme essential for protein homeostasis and autophagy. While well studied in neurons, the role of UCHL1 in microglia, the brain's resident immune cells, remains unknown. Given that autophagic disruption drives cellular senescence and neurodegeneration, understanding the role of UCHL1 in microglia is crucial. IMP-1710, a novel and highly selective activity-based probe of UCHL1, enables a precise investigation of its function, yet its role in microglial biology remains unexplored.

Aim: To investigate the effects of the selective UCHL1 inhibitor, IMP-1710, on autophagic regulation and senescence phenotypes in a human microglial cell line.

Methods: Human microglia cell line, HMC-3, was treated with IMP-1710. The cell viability assay was performed, and an in-gel fluorescence study using click chemistry and molecular modeling analysis was conducted to confirm the binding mechanism of IMP-1710 and UCHL1. The senescent markers were determined by Western blot and RT-qPCR. Cell migration assay and cell cycle were also conducted. Autophagosome formation was visualized using live-cell imaging.

Results: We first confirmed that IMP-1710 binds to UCHL1 in microglial cells. Pharmacological inhibition of UCHL1 by IMP-1710 markedly upregulated autophagosome formation. The IMP-1710 treatment also modestly elevated CDKN1A and CDKN2A mRNA expression levels while suppressing cyclin D1 protein, consistent with the upregulation of senescent features. Functionally, UCHL1 inhibition triggered G2/M cell cycle arrest and reduced microglial migration. Mechanistically, IMP-1710 covalently targeted Cys90 of the UCHL1 catalytic triad, with His161 and Asp176 stabilized by hydrogen bonding, and showed stronger binding than CG341.

Conclusion: This study provides the first evidence that UCHL1 inhibition by IMP-1710 disrupts autophagy and induces senescence-like changes in human microglia. Our findings unveil the role of UCHL1 in microglial biology and establish IMP-1710 as a novel tool to probe microglial function.

Keywords: UCHL1, autophagy, cellular senescence, microglial cells, deubiquitinase enzymes

----

P-046:

COMPUTATIONAL DRUG REPURPOSING YIELDS EFFECTIVE GSK3β INHIBITORS IN HT-22 HIPPOCAMPAL CELLS

Hanwen Mi[1], Utid Suriya[2], Monruedee Sukprasansap[3], Tewin Tencomnao[4], Sunhapas Soodvilai[1,5], Benjamin Ongnok[1,6,*]

[1]*Department of Physiology, Faculty of Science, Mahidol University, 272 Rama VI Rd, Bangkok, 10400*

[2]*Department of Biochemistry, Faculty of Science, Mahidol University, 272 Rama VI Rd, Bangkok, 10400*

[3]*Food Toxicology Unit, Institute of Nutrition, Mahidol University, 999 Phutthamonthon Sai 4 Rd, Nakhon Pathom, 73170*

[4]*Center of Excellence on Natural Products for Neuroprotection and Anti-Ageing, Department of Clinical Chemistry, Faculty of Allied Health Sciences, Chulalongkorn University, 254 Phaya Thai Rd, Bangkok, 10330*

[5]*Research Center of Transporter Protein for Medical Innovation, Department of Physiology, Faculty of Science, Mahidol University, 272 Rama VI Rd, Bangkok, 10400*

[6]*Center for Neuroscience, Faculty of Science, Mahidol University, 272 Rama VI Rd, Bangkok, 10400*

**Corresponding author. Email: benjamin.ong@mahidol.ac.th*

Abstract

Background: Glycogen synthase kinase-3β (GSK3β) is a serine/threonine kinase involved in neuronal apoptosis, inflammation, and multiple neurodegenerative disorders.

Aim(s): To evaluate the efficacy of an in silico drug repurposing pipeline for identifying FDA-approved compounds capable of inhibiting GSK3β in neuronal cells.

Methods: A compound library was retrieved from the ZINC database using filters for natural products, biogenic, in vitro, named, and for sale entries. Docking-based virtual screening was performed using AutoDock Vina to rank compounds by predicted binding energy (BE). The top 200 candidates with the lowest BEs (highest predicted affinity) were clustered and subsequently docked against p38α MAP kinase to evaluate off-target effects. Compounds displaying higher BEs (weaker affinity) than the known p38 inhibitor SCIO-469 were retained to minimize promiscuity. Two hits – budesonide (BE = −9.5 kcal/mol) and cefonicid (BE = −9.3 kcal/mol) – were selected based on favorable selectivity, affordability, and commercial availability. Molecular dynamics simulations (300 ns) and Molecular Mechanics Generalized Born Surface Area (MM-GBSA) free-energy analyses confirmed stable and energetically favorable binding.

Results: In vitro validation using HT-22 hippocampal cells showed both compounds maintained >90% cell viability up to 10 µM. Western blotting revealed that budesonide exhibited a statistically significant, dose-dependent increase in GSK3β Ser9 phosphorylation across the tested concentrations, whereas cefonicid produced a comparable effect only at the higher end of the concentration range, suggesting partial inhibitory activity without overt cytotoxicity. Flow cytometry confirmed no major disturbance of the cell cycle, supporting their safety profiles.

Conclusion: The integration of *in silico* screening and in vitro assays successfully identified budesonide and cefonicid as potential GSK3β inhibitors. While enhanced Ser9 phosphorylation implies functional inhibition, further kinase activity assays and related target gene transcripts are required to confirm this mechanism. Overall, this study

highlights the translational potential of computational drug repurposing for neuroprotective drug discovery for neurological diseases.

Keywords: HT-22, GSK3β, Computational Drug Repurposing, Structure-based virtual screening, molecular docking

---

P-047:

CBD-ENRICHED HEMP EXTRACT AND CBD ISOLATE ATTENUATE STRESS-INDUCED ANXIETY VIA ENDOCANNABINOID SYSTEM MODULATION IN RATS

Wanfrutkon Waehama[1,2], Jureepon Roboon[2,3], Prapapan Temkitthawon[4,5], Samur Thanoi[6], Sawanya Charoenlappanit[7], Sittiruk Roytrakul[7], Sutisa Nudmamud-Thanoi[2,3*]

[1]Medical Science Graduate Program, Faculty of Medical Science, Naresuan University, Phitsanulok, 65000
[2]Center of Excellence in Medical Biotechnology, Naresuan University, Phitsanulok, 65000
[3]Department of Anatomy, Faculty of Medical Science, Naresuan University, Phitsanulok, 65000
[4]Faculty of Pharmaceutical Sciences, Naresuan University, Phitsanulok, 65000
[5]Center of Excellence in Cannabis Research, Faculty of Pharmaceutical Sciences and Center of Excellence for Innovation in Chemistry, Naresuan University, Phitsanulok, 65000
[6]School of Medical Sciences, University of Phayao, Phayao, 56000
[7]National Centre for Genetic Engineering and Biotechnology, National Science and Technology Development Agency, Pathum Thani, 12120
**Corresponding author. Email: sutisat@nu.ac.th*

Abstract

Background: Anxiety disorders are prevalent mental health conditions, often associated with chronic stress. Cannabidiol (CBD) has been reported to exert anxiolytic effects through modulation of the endocannabinoid system.

Aim(s): This study aimed to investigate the effects of CBD-enriched hemp extract (CBD-E) and CBD isolate (CBD-I) on anxiety-like behaviors in rats subjected to chronic unpredictable mild stress (CUMS).

Methods: Male Sprague-Dawley rats were orally administrated CBD-E or CBD-I prior to 14 days of CUMS to induce anxiety-like behaviors. Animals were assigned to six groups: control, CUMS, CUMS with CBD-E (2.5 and 5 mg/kg), and CUMS with CBD-I (2.5 and 5 mg/kg). Following stress induction, anxiety-like behaviors were assessed by the open field test (OFT) and elevated plus maze (EPM). Subsequently, hippocampal tissues were collected for proteomic analysis.

Results: The CUMS group exhibited a significant increase in closed-arm entries in the EPM compared with the control group, indicating anxiety-like behavior. Administration of CBD-I (2.5 and 5 mg/kg) significantly reduced closed-arm entries compared with the CUMS group, demonstrating an anxiolytic effect. Meanwhile, CBD-E (2.5 and 5 mg/kg) produced a decreasing trend in closed-arm entries relative to the CUMS group. Proteins in the endocannabinoid system, including cannabinoid receptor 1 (CB1R) and N-acyl phosphatidylethanolamine phospholipase D (NAPEPLD), were downregulated in the CUMS group, whereas the N-methyl-D-aspartate receptor (NMDAR) was upregulated. CBD-I (5 mg/kg) restored NAPEPLD expression, while CBD-E (5 mg/kg) restored CB1R expression. Notably, both CBD-E and CBD-I at 5 mg/kg attenuated NMDAR upregulation, indicating a compensatory effect on endocannabinoid system dysregulation.

Conclusion: These findings support the potential of CBD-E and CBD-I to alleviate stress-induced anxiety through regulation of the endocannabinoid system.

Keywords: Cannabidiol, Hemp, Anxiety, Endocannabinoid system

P-048:

THE ALTERATION OF PROTEIN PROFILES IN CURCUMIN SOLID DISPERSION (CSD) AGAINST A DEXAMETHASONE-INDUCED DEPRESSION MODEL

Ponthip Cheenkhwan[1,6], Suchiwa Pan-on[2], Waree Tiyaboonchai[3,6], Samur Thanoi[4], Sawanya Charoenlappanit[5], Sittiruk Roytraku[5], Jureepon Roboon[1,6], and Sutisa Nudmamud-Thanoi[1,6]*

[1]*Department of Anatomy, Faculty of Medical Science, Naresuan University, Phitsanulok, 65000*
[2]*Department of Cosmetic Science and Technology, Faculty of Pharmaceutical Sciences, Burapha University, Chonburi, 20131*
[3]*Department of Pharmaceutical, Faculty of Pharmaceutical Sciences, Naresuan University, Phitsanulok, 65000*
[4]*Department of Anatomy, School of Medical Sciences, University of Phayao, Phayao, Thailand 56000*
[5]*National Centre for Genetic Engineering and Biotechnology, National Science and Technology Development Agency, Pathum Thani, 12120*
[6]*Centre of Excellence in Medical Biotechnology, Naresuan University, Phitsanulok, 65000*
**Corresponding author, e-mail: sutisat@nu.ac.th*

Abstract

Background: Major depressive disorder is linked to neuroinflammation, oxidative stress, and impaired neuronal plasticity. Chronic dexamethasone administration in a rodent model of depression by inducing HPA axis dysregulation and molecular changes. Curcumin, a polyphenolic compound from Curcuma longa, exhibits antioxidant, anti-inflammatory, and neuroprotective properties and has shown antidepressant-like effects. However, its clinical efficacy is limited by poor brain permeability and rapid systemic clearance. To overcome these limitations, this study investigates the development of curcumin solid dispersion (CSD) to enhance its absorption and bioavailability in the brain, but its effects on molecular change remain unclear.

Aim(s): The study aims to explore the CSD effects on protein alteration in dexamethasone (DEX)-induced depression model.

Methods: Male Sprague-Dawley rats were divided into three groups: control, DEX, and DEX+CSD. Brain tissue was collected for proteomics analysis using the LC-MS/MS technique to determine differentially expressed proteins (DEPs). Followed by Gene Ontology (GO) annotation, KEGG pathway enrichment, and protein–protein interaction (PPI) network analysis to characterize overlapping DEPs.

Results: Proteomic analysis revealed that dexamethasone exposure increased the expression of proteins linked to neuroinflammation and apoptosis while reducing proteins associated with neuroplasticity and neurogenesis in the frontal cortex. In contrast, rats receiving CSD alongside dexamethasone showed the opposite pattern, with decreased expression of neuroinflammatory and pro-apoptotic proteins and increased expression of proteins related to neuroplasticity and neurogenesis.

Conclusion: CSD decreased the expression of proteins linked to neuroinflammation and cell apoptosis while increasing those associated with neuroplasticity and neurogenesis, suggesting its protective role in the brain.

Keywords: Proteomics, Depression, Curcumin, Dexamethasone, Frontal cortex

---

P-049:

D-GALACTOSE INDUCES SENESCENCE AND FUNCTIONAL IMPAIRMENT IN HUMAN MICROGLIAL CELL LINE THROUGH RAGE SIGNALING

Poramet Klangmongkon[1], Patit Choksawangkarn[2], Monruedee Sukprasansap[3], Tewin Tencomnao[4], Sunhapas Soodvilai[1,5], Alisa Damnernsawad[6], Benjamin Ongnok[1,7,*]

[1]*Department of Physiology, Faculty of Science, Mahidol University, Bangkok, Thailand, 10400*
[2]*Department of Biochemistry, Faculty of Science, Mahidol University, Bangkok, Thailand, 10400*
[3]*Food Toxicology Unit, Institute of Nutrition, Mahidol University, Nakhon Pathom, Thailand, 10400*
[4]*Center of Excellence on Natural Products for Neuroprotection and Anti-Ageing, Department of Clinical Chemistry, Faculty of Allied Health Sciences, Chulalongkorn University, Bangkok, Thailand, 10400*
[5]*Research Center of Transporter Protein for Medical Innovation, Department of Physiology, Faculty of Science, Mahidol University, Bangkok, Thailand, 10400*
[6]*Department of Biology, Faculty of Science, Mahidol University, Bangkok, Thailand, 10400*
[7]*Center for Neuroscience, Faculty of Science, Mahidol University, Bangkok, Thailand, 10400*
**Corresponding author. Email: benjamin.ong@mahidol.ac.th*

Abstract

Background: The progression of neurodegenerative diseases is substantially influenced by aging, as microglia undergo senescence and shift to a pro-inflammatory state that exacerbates neuroinflammation. D-galactose (D-gal) is widely used to mimic brain aging, partly via reacting non-enzymatically amino acid residues on proteins to form advanced glycation end-products (AGEs). AGEs bind to the receptor for advanced glycation end-products (RAGE), activating downstream and senescence signaling in the brain. However, the mechanisms by which D-gal induces senescence in microglia through RAGE signaling remain obscure.

Aim(s): To investigate the efficacy of D-gal in inducing senescence in the human microglial cell line HMC-3 and to explore whether RAGE signaling contributes to the emergence of senescent and pro-inflammatory phenotypes.

Methods: HMC-3 cells were treated with a concentration range of D-gal (0–40 mg/mL). Cell viability was assessed via MTT to determine IC20 and IC50. RT-qPCR and immunoblotting analyses were performed to assess the expression of senescence- and inflammation-associated genes and proteins. Cell cycle distribution was analyzed to detect arrest. Autophagosome accumulation and migratory capacity were evaluated to further characterize the senescent phenotype. Data were collected in biological triplicate.

Results: MTT assays established IC20 and IC50 values of approximately 10 and 20 mg/mL D-gal, respectively. Furthermore, upregulation of *CDKN2A*, *CDKN1A*, *TNF*, and *IL6*, along with a significant reduction in cyclin D1 protein expression, was observed in D-gal-treated cells. D-gal significantly increased the proportion of cells arrested in the G0 phase of the cell cycle, confirming cell cycle arrest. RAGE expression was also upregulated following D-gal treatment. Autophagosome accumulation was elevated in a dose- and time-dependent manners. Migration assays revealed progressively reduced motility in D-gal-treated cells.

Conclusion: The findings indicate that D-gal senescence-like changes and proinflammatory activation in human microglia through altered expression of cell cycle

regulators and pro-inflammatory genes, potentially mediated by the RAGE signaling pathway.

Keywords: Aging, Cellular senescence, Microglia, Neurodegeneration

----

P-050:

ADMINISTRATION OF HIGH-DOSE TACROLIMUS IMPAIRED HIPPOCAMPAL SYNAPTIC PLASTICITY AND HIPPOCAMPAL-DEPENDENT MEMORY IN MALE WISTAR RATS

Houzhi Cheng [1,2,3,4], Patcharapong Pantiya[2,3], Hiranya Pintana[2,3], Titikorn Chunchai[2,3], Nipon Chattipakorn[2,3,4], Siriporn C Chattipakorn[2,3,5] *

[1]Youjiang Medical University for Nationalities, Baise City, Guangxi, China, 533000
[2] Neurophysiology Unit, Cardiac Electrophysiology Research and Training Center, Faculty of Medicine, Chiang Mai University, Chiang Mai 50200, Thailand
[3] Center of Excellence in Cardiac Electrophysiology, Chiang Mai University, Chiang Mai 50200, Thailand
[4] Cardiac Electrophysiology Unit, Department of Physiology, Faculty of Medicine, Chiang Mai University, Chiang Mai 50200, Thailand
[5] Department of Oral Biology and Diagnostic Sciences, Faculty of Dentistry, Chiang Mai University, Chiang Mai 50200, Thailand
**Corresponding author: E-mail: scchattipakorn@gmail.com ; siriporn.c@cmu.ac.th*

Abstract

Background: Tacrolimus (TAC), a calcineurin inhibitor used as an immunosuppressant, has been associated with cognitive decline in organ transplant recipients. However, although synaptic plasticity is fundamental to cognitive function, its alteration following tacrolimus administration remain uninvestigated.

Aim: To investigate the effects of TAC on synaptic plasticity and cognitive function in rats.

Methods: Twenty-four-week-old male Wistar rats (600-700g) received either vehicle (olive oil, s.c.) or TAC at doses of 0.5, 1.0, or 2.0 mg/kg/day (s.c.) for 2 weeks. One day after treatment, cognitive performance was assessed using the novel object location and recognition (NOL and NOR) tests, followed by *ex vivo* long-term potentiation (LTP) recordings. Data were expressed as mean ± SEM (n = 3–5 per group) and analyzed using one-way ANOVA with LSD post hoc testing.

Results: Treatment with high-dose TAC (2.0 mg/kg) significantly reduced the field excitatory postsynaptic potential (fEPSP) slope (TAC 2.0: 108.3 ± 5.1) compared with other groups (Vehicle: 128.9 ± 4.0; TAC 0.5: 128.4 ± 3.6; TAC 1.0: 127.9 ± 5.7; $p < 0.05$), suggesting impaired synaptic plasticity. Consistently, high-dose TAC treatment also resulted in the lowest preference for the novel location (TAC 2.0: 39.0 ± 8.5) compared with other groups (Vehicle: 63.5 ± 3.1; TAC 0.5: 63.1 ± 4.7; TAC 1.0: 77.6 ± 9.7; $p < 0.05$), indicating deficits in hippocampal-dependent memory. In contrast, no significant differences in preference for the novel object were observed among groups (Vehicle: 51.9 ± 3.5; TAC 0.5: 50.3 ± 8.5; TAC 1.0: 41.3 ± 9.2; TAC 2.0: 55.2 ± 2.5; $p > 0.05$), suggesting that hippocampal-independent recognition memory remained unaffected.

Conclusion: High-dose tacrolimus (2.0 mg/kg) impaired hippocampal synaptic plasticity and hippocampal-dependent memory in rats. The findings underscore the importance of optimizing tacrolimus dosing and developing targeted strategies to mitigate neurotoxicity while preserving its immunosuppressive efficacy in clinical settings.

Abbreviations: fEPSP, field excitatory postsynaptic potential; LTP, long-term potentiation; NOL, novel object location; NOR, novel object recognition; TAC, tacrolimus.

Keywords: Tacrolimus; Calcineurin inhibitor; Cognition; Synaptic plasticity; Neurotoxicity

---

P-051:

LYSOSOMAL STRESS FLUORESCENCE REPORTER (LSFR): A NEW METHOD FOR DETECTION AND QUANTITATION OF LYSOSOMAL STRESS RESPONSE IN NEURONS

Narinporn Tresteankij, Tai Chaiamarit, Tanida Chokpanuwat, and Benjamin Ongnok*

[1]Department of Physiology, Faculty of Science, Mahidol University, Bangkok, Thailand
*Corresponding author. Email: benjamin.ong@institute.ac.th

Abstract

Lysosomal stress response is the state that lysosomes enter in response to stressors that lead to changes in intraluminal pH, size, membrane integrity, or degradative function of lysosomes. The response is regulated by Transcription Factor EB (TFEB) that binds to coordinated lysosomal expression and regulation (CLEAR) element on the promoter region and upregulates the genes-related to lysosomal biogenesis and autophagy in order to restore lysosome function. Dysfunction in lysosomal stress response is hypothesized to contribute to the pathogenesis of various diseases such as lysosomal storage diseases (LSDs), neurodegenerative diseases, and cancer. Compared to ER stress and mitochondrial stress, lysosomal stress response is overlooked or not investigated due to limitations in method of detection. Using RT-qPCR, we found a given set of genes that are highly expressed in hiPSC-derived neural progenitor & mature neural cells. Specifically, BLOC1S1, GNPTG, or SUMF1 is significantly upregulated in neural progenitor cells and neurons from patients with neuronopathic Gaucher's disease. The purpose of this study is to make a lentiviral vector called Lysosomal Stress Fluorescence Reporter (LSFR) to facilitate in detection and monitoring the lysosomal stress response in a direct and real-time manner. This is achieved by designing and construction a clear-enriched promoter region of BLOC1S1, GNPTG, or SUMF1 gene that drives mCherry expression. The LSFR vector along with the 3rd generation lentiviral packaging plasmids were transfected in HEK293T cells, and viral particles were collected for transduction of any cell type of interest.

In conclusion, the Lysosomal Stress Fluorescence Reporter (LSFR) is proven to be useful for future research and purposing a detection method involvement in lysosomal stress response. We first tested LSFR efficacy in HMC-3 microglial cells line exposed to chloroquine, a known lysosomal stressor and found that the LSFR works accordingly to lysosomal stress response in concentration-dependent manner.

Keywords: lysosomal stress response, lysosomal stress, fluorescence reporter

---

P-052:
EVALUATION OF CUE-BASED PROTOCOL IMPLEMENTATIONS AND SEMANTIC FEEDBACK IN MOTOR IMAGERY-BASED BRAIN-COMPUTER INTERFACE EXPERIMENTS

H.T Nguyen[1], Q.C Pham[3], H.K.L Nguyen[2^], T.D.M Nguyen[4], and T.T.H Ha[1*]

[1] *School of Biomedical Engineering, VNU-HCM International University, Ho Chi Minh City, Viet Nam*
[2] *School of Biotechnology, VNU-HCM International University, Ho Chi Minh City, Viet Nam*
[3] *College of Engineering & Computer Science, VinUniversity, Viet Nam*
[4] *School of Computer Science, University of Technology Sydney, Australia*
**Corresponding author. Email: htthuong@hcmiu.edu.vn*
^ lead presenter

Abstract

Background: Non-invasive Brain-Computer Interface (BCI) studies mostly center on the motor imagery (MI) concept, where multi-channel Electroencephalogram (EEG) signals are collected and characterized by patterns for different imagined tasks. Previous studies put extensive efforts into data-driven techniques to improve classification performance on benchmark datasets; however, other aspects, such as experimental factors, still lack thorough investigation.

Aim(s): This pilot study aims to evaluate the effect of different cue-based protocols and semantic feedback on within-subject MI-BCI baseline performance to better guide the experimental instructions for a specific group of users.

Methods: An Emotiv EEG headset kit integrated into the Lab-Streaming-Layer (LSL) was used for data acquisition. Three PsychoPy-based protocols were designed, namely, G1(n=4), G2 (n=4), and G3 (n=6), incorporating different visual instructions of image-cue, arrow-cue, and arrow-cue-feedback utilizing Event-Related (de)Synchronization (ERD/ERS) demonstration, respectively. Imagery data (left/right hand/foot) from 14 healthy college participants (age 20~22, five females) were collected (12 trials/task/run) and randomly allocated for each designated protocol. A processing framework was implemented using a conventional Lasso-based sparse Filter Bank Common Spatial Pattern (SFBCSP) for feature extraction/selection and Linear Discriminant Analysis (LDA) for classification to assess the baseline performance. Average ROC (5-fold cross-validation) was calculated for the upper-limb binary model of each run with different non-overlapping time segments. Statistical non-parametric tests were used for within-group and cross-group comparative analysis.

Results: In within-group analysis, average performance between run1 & run2 is as follows: G1 (52.7% & 44.8%); G2 (62.0% & 57.8%); G3 (52.5% & 67.7%) where G3 group yields significant improvement (run2 > run1, $p<0.05$), while no statistical difference has been found within the G1 or G2 group. In cross-group analysis, an average performance combining all runs of G1, G2, and G3 are 48.8%, 59.8%, and 60.1%, respectively; where it shows significant differences in G1&G2 ($p<0.05$) and G1&G3 ($p<0.05$) but not in G2&G3. In the after-run self-assessment analysis, while few elements strongly correlate with the overall performance, no significant difference was found between the image-cue and arrow-cue groups.

Conclusion: The preliminary results highlight that different instructions (arrow/image cue & feedback) may affect within-session performance between runs,

while reporting no evidence of changing the subject's psychological factors. The statistical analysis also suggests that verbal feedback with arrow-cue can enhance the model's efficacy, which can be further explained by orienting the alpha-band ERD/ERS response. Future studies may explore other human-based factors considering the motor response-ability within the larger target group of users, potentially advancing BCI application in a personalized paradigm.

Keywords: Motor imagery; Brain–computer interface; EEG; ERD/ERS; Feedback.

P-053:

A SMALL-MOLECULE NATURAL PRODUCT ATTENUATES Aβ42-INDUCED NEUROINFLAMMATION VIA IDO-1 INHIBITION IN SH-SY5Y CELLS

Munnutchaya Tarmtiranont [1], Kornkanok Promthep[1], Sujira Mukda, Banthit Chetsawang[1], Jiraporn Panmanee[1*]

[1] *Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Salaya, Nakorn Pathom 73170 Thailand*
* Corresponding author.
Email: jiraporn.pam@mahidol.ac.th

Abstract

Background: Kynurenine pathway (KP), neuroinflammation, and Alzheimer's disease (AD) are closely related to each other. Inflammation is recognized as a key contributor to AD. Indoleamine 2,3-dioxygenase 1 (IDO-1), the rate-limiting enzyme of KP, can be activated by inflammatory cytokines. Previous studies demonstrated anti-inflammatory property of Aurantiamide (Aur), and our prior computational analysis suggested that Aur may act as a potential IDO-1 inhibitor.

Aim(s): This study aims to investigate the anti-inflammatory activity and IDO-1 inhibition of Aur in neuronal-like SH-SY5Y cell line.

Methods: SH-SY5Y cells were pretreated with Aur at 1 and 10 μM for 6 hours following Aβ exposure at 1 μM for 48 hours. After incubation period, the cells were collected to examine the protein expression of IDO-1 and p53 using Western blot analysis. mRNA expression of the proinflammatory cytokines IL-1β and TNF-α were measured using qPCR.

Results: The Western blot analysis revealed that pretreatment of Aur ameliorated Aβ-induced effects in SH-SY5Y cells. Cells pretreated with Aur at both 1 and 10 μM showed a significant decrease in IDO-1 and p53 expression compared with Aβ-treated group. Consistently, qPCR demonstrated reduced IL-1β and TNF-α expression levels relative to the Aβ-treated control.

Conclusion: Aur exhibits anti-IDO-1 and anti-inflammatory effects, suggesting its potential as a therapeutic candidate for AD.

Keywords: Alzheimer's disease, Inflammation, Kynurenine pathway, Indoleamine 2,3-dioxygenase 1

Background

Neuroinflammation is one of key factors contributes to neurodegenerative diseases including AD (Kinney JW et al., 2018). AD progression closely associated with disposition of amyloid-beta (Aβ) protein, which also a pathological hallmark of the disease (Gouras GK et al., 2015). Currently, many studies have been pointing out that the KP, a tryptophan degradation pathway, is one of AD regulators through production of neurotoxic compounds including 3-hydroxykynurenine (3-HK), 3-hydroxyanthranilic acid (3-HAA), quinolinic acid (QA) and also neuroprotective compound like kynurenic acid (KA). The level of these metabolites has been found to be abnormal in AD patients (Gouras GK,Olsson TT and Hansson O, 2015). More important, an activation of IDO-1, the rate-limiting enzyme in KP, has been found to relate with neuroinflammation and Aβ accumulation (Johnson LA and Macauley SL, 2024). Increased level of IDO-1 and QA, primarily in astrocytes and microglia, have been observed alongside Aβ in AD. However, the direct effects of Aβ oligomers on KP and IDO-1 in neuronal cells remain poorly understood. This study aims to investigate the effects of Aβ oligomers toward activation of IDO-1 in neuronal-like SH-SY5Y cell lines. It has been reported that Aur, a compound found in plants, act as an anti-inflammatory agent (Yoon C-S et al., 2014). According to our prior computational analysis, the compound may possess an effect as an IDO-1 inhibitor. In this study, we investigated effects of Aβ oligomers on the expression level of IDO-1 and protective effects of Aur on regulation of pro-inflammatory cytokine expression. Our study revealed that SH-SY5Y cells pretreated with Aur followed by Aβ exposure could reduce the expression of IDO-1 and cell death regulator p53 in protein levels. Moreover, lower levels of inflammatory cytokines IL-1β and TNF-α expression in mRNA levels were observed by Aur treatment. These findings suggest that Aur can ameliorate neuronal death by suppressing IDO-1 expression and reducing the production of pro-inflammatory cytokines.

Materials and Methods

Cell culture and Aβ1-42 treatment

Aβ1-42 was prepared following the standard Aβ preparation protocol to achieve a final concentration of 1 mM and stored at -80 °C for use as a stock solution. For the experiment, Aβ1-42 was diluted in serum-free (SF) medium to a concentration of 100 µM and incubated at 37 °C for five days to induce peptide aggregation (Chinchalongporn V et al., 2018). SH-SY5Y cells were cultured in MEM/F-12 medium supplemented with 10% fetal bovine serum (FBS) and penicillin/streptomycin and incubated at 37 °C with 95% humidity and 5% CO_2 until they reach at least 80% confluency. Subsequently, cells were seeded into 60 mm petri dishes and allowed to adhere for 24 hours. After 24 hours, the cells were pretreated with Aur at 1 and 10 µM following Aβ1-42 at 1 µM, in 1% FBS medium. The cells were incubated with Aur pretreatments for 6 hours. Then, cells were treated with 1 µM Aβ1-42 and incubated for an additional 48 hours. At the end of the incubation period, the cell pellets were washed three times with phosphate-buffered saline (PBS). The collected cell pellets were used for following experiments.

Western blot

After 48 hours of treatment, human neuroblastoma SH-SY5Y cells were collected and lysed in cold RIPA buffer supplemented with 1% protease and phosphatase inhibitors. The cell suspension was sonicated for 5 seconds twice and then centrifuged at 4 °C at 12,000 rpm for 15 minutes. The supernatants were collected, and the total protein concentration was measured using Bradford's assay (Bradford MM, 1976). Proteins were separated using SDS-PAGE containing 12% polyacrylamide gels at 120 volts for 1.5 hours. Subsequently, the proteins were transferred onto PVDF membranes at 100 volts for 2 hours using a wet tank system. The membranes were incubated with blocking buffer (3-5% non-fat milk or 2-5% bovine serum albumin in Tris-buffered saline containing 0.1% Tween 20) at room temperature for 1 hour, followed by incubation with primary antibodies, including rabbit anti-IDO1 (1:1000) and mouse anti-p53 (1:1000), for 6 hours at room temperature and then at 4 °C overnight. After incubation, the membranes were washed three times for 5 minutes each with TBS-T before being incubated with secondary antibodies at room temperature for 1.5 hours. Finally, the membranes were washed three times for 5 minutes each with

TBS-T and incubated with ClarityTM Western ECL substrate. Specific protein signals were detected using the Vilber FUSION FX chemiluminescence imaging system.

Real-time polymerase chain reaction (qPCR)

After 48 hours of treatment, cells were collected and RNA isolation was performed to achieve total RNA. The purified RNA was used to perform the cDNA synthesis. Reverse transcription was performed using EPPENDORF flexlid Mastercycler nexus gradient. At the end of the cycle, cDNA was collected. The reactions were prepared in PCR strip tube, with 20 µl as a total volume, 2 µl of cDNA (50 ng) from 6 different conditions including (i) untreated control, (ii) Aβ1-42 (1 µM), (iii) Aur (1 µM), (iv) Aur (10 µM), (v) 1 µM Aur + 1 µM Aβ, (vi) 10 µM Aur + 1 µM Aβ. The samples were mixed with 10 µl of Luna® Universal qPCR Master Mix, target-specific primers for IL-1β and TNF-α, and DEPC water. Real-time PCR was performed using Eppendorf Mastercycler realplex 2. The reaction was set as follows: the initial denaturation was performed at 95 °C for 2 min followed by 40 cycles of 95 °C for 15 sec, 60 °C for 15 sec, and 68 °C for 20 sec. Each gene was normalized with GAPDH (glyceraldehyde-3-phosphate dehydrogenase). The relative mRNA expression was calculated according to the $2^{-\Delta\Delta Ct}$ protocol (Livak KJ and Schmittgen TD, 2001).

Results

Pretreatment with Aurantiamide prior to Aβ1-42 exposure can reverse the Aβ1-42-induced an increase in IDO1 and p53 expression

Expression levels of IDO-1 and p53 were examined using Western blot analysis. Treating with Aβ alone caused upregulation of expression levels for both IDO-1 and p53 compared with the untreated control. As shown in figure 1, treating with Aur alone at 1 and 10 µM did not significantly alter the basal expression of the two proteins. However, pretreatment with Aur prior to Aβ exposure ameliorated the Aβ-induced expression levels of both IDO-1 and p53. For IDO-1, at 10 µM showed a significant decrease, while P53 was significantly lowered at both 1 and 10 µM (figure 1).

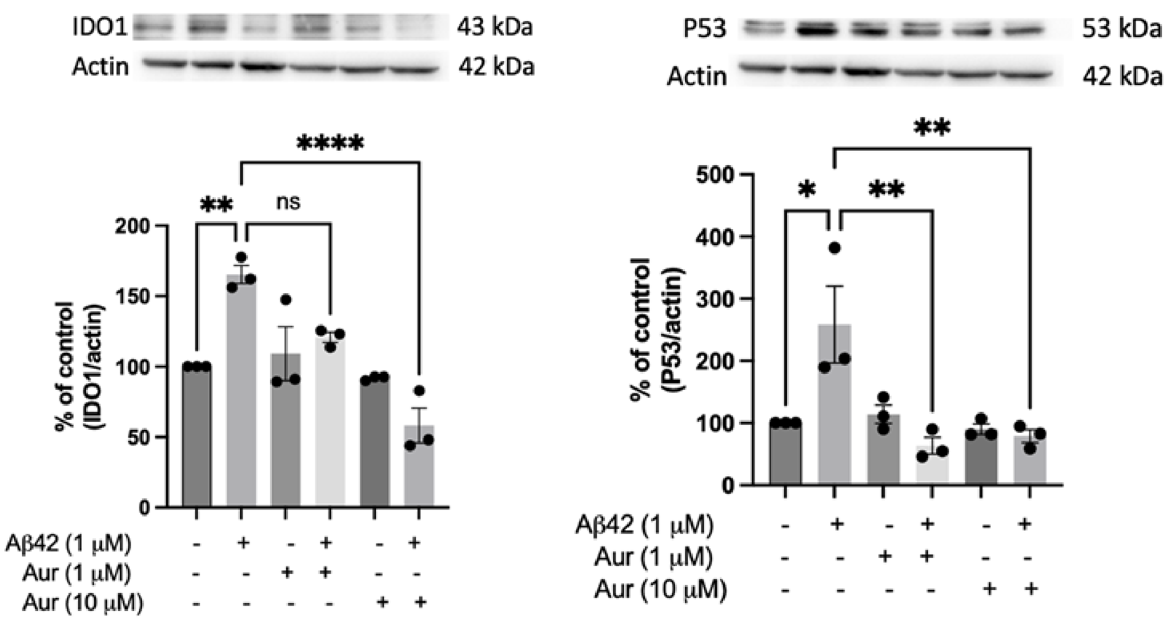


Figure 1 Effects of Aurantiamide on IDO and p53 expression in SH-SY5Y cells exposed with Aβ1-42**.**

Effects of Aurantiamide on proinflammatory cytokine expression in SH-SY5Y cells treated with Aβ1-42

IL-1β expression

qPCR analysis was performed to measure an expression of inflammatory cytokines at an mRNA level. The results revealed that treating cells with Aβ at 1 µM for 48 hours significantly increased the expression of IL-1β compared to the untreated control (figure 2). Treating with Aur alone at 1 and 10 µM did not change the basal level of IL-1β expression. However, pretreatment of Aur for 6 hours followed by Aβ exposure markedly reduced IL-1β expression compared with Aβ-treated group.

TNF-α expression

Similar to IL-1β, TNF-α expression was significantly increased following Aβ treatment for 48 hours compared to the untreated control (figure 2). Aur alone at both 1 and 10 µM did not alter the basal expression of TNF-α. However, Aur treatment at both 1 and 10 µM prior to Aβ exposure reduced the expression levels compared to Aβ-treated cells, with a more pronounced effect observed at 10 µM.

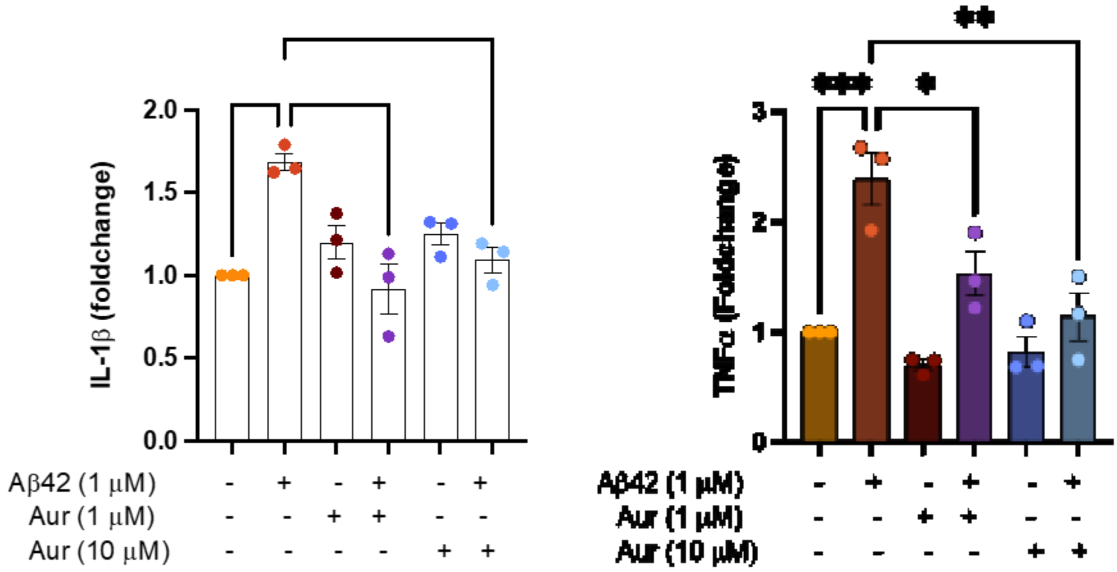


Figure 2 Effects of Aurantiamide on proinflammatory cytokines expression in SH-SY5Y cells treated with Aβ1-42

Discussion and Conclusion

Recently, several studies have pointed Aur as an anti-inflammatory agent. With our findings, Aur pretreatment, especially in higher concentration (10 µM) effectively reduced Aβ-induced increases of inflammatory cytokines IL-1β and TNF-α, in line with its inhibitory effects on IDO-1 and p53. These results suggest that Aur may disrupt the inflammatory and apoptotic signaling cascades triggered by Aβ toxicity. Moreover, because Aur pretreatment was more effective than Aur treatment alone, we propose that Aur may act upstream in the inflammatory pathway, preventing the initiation of downstream inflammatory events.

There are previous studies demonstrated that cellular stress and DNA damage caused by intracellular Aβ1-42 promote phosphorylation of p53 (Lapresa R et al., 2019). Since many apoptosis-related genes are part of p53' downstream, activation of p53 consequences in apoptotic cell death. Several studies confirmed that inflammation, or levels of inflammatory cytokines correlated with p53 expression (Shao X et al., 2020).

Therefore, the observed decrease in p53 in our study may result, at least in part, from the anti-inflammatory effects of Aur.

The anti-inflammatory effects of Aur may prime cellular defenses, so that upon exposure to Aβ1-42 peptides, inflammatory responses are not overactivated compared to normal conditions. This reduced inflammatory activity may in turn limit IDO-1 activation, leading to decreased expression of IDO-1 and lower release of inflammatory cytokines.

In this study, undifferentiated SH-SY5Y cells were employed because they provide a reproducible and well-established neuronal model for assessing amyloid-induced cytotoxicity and neuroprotective effects of test compounds. Although differentiated SH-SY5Y cells more closely resemble mature neurons, the undifferentiated form remains valuable for modeling early neurodegenerative processes and screening neuroactive agents under standardized conditions (Forster JI et al., 2016;Vulin I et al., 2025). The limitations of this study include that our results are derived solely from *in vitro* experiments using SH-SY5Y neuronal-like cells. To fully evaluate Aur's therapeutic potential, *in vivo* studies are required. Additionally, as our focus was on Aur as a "promising IDO-1 inhibitor," further investigation of KP metabolites is warranted. Measuring specific downstream metabolites would allow direct assessment of IDO-1 enzymatic activity.

Acknowledgements

This project is funded by National Research Council of Thailand (NRCT) to JP (Grant No: N42A670122)

References

Bradford MM (1976), A rapid and sensitive method for the quantitation of microgram quantities of protein utilizing the principle of protein-dye binding. Analytical Biochemistry 72:248-254.

Chinchalongporn V, Shukla M, Govitrapong P (2018), Melatonin ameliorates Aβ(42) - induced alteration of βAPP-processing secretases via the melatonin receptor through the Pin1/GSK3β/NF-κB pathway in SH-SY5Y cells. Journal of pineal research 64:e12470.

Forster JI, Köglsberger S, Trefois C, Boyd O, Baumuratov AS, Buck L, Balling R, Antony PM (2016), Characterization of Differentiated SH-SY5Y as Neuronal Screening Model Reveals Increased Oxidative Vulnerability. J Biomol Screen 21:496-509.

Gouras GK, Olsson TT, Hansson O (2015), β-Amyloid peptides and amyloid plaques in Alzheimer's disease. Neurotherapeutics 12:3-11.

Johnson LA, Macauley SL (2024), Alzheimer's and metabolism wed with IDO1. Science 385:826-827.

Kinney JW, Bemiller SM, Murtishaw AS, Leisgang AM, Salazar AM, Lamb BT (2018), Inflammation as a central mechanism in Alzheimer's disease. Alzheimer's & Dementia: Translational Research & Clinical Interventions 4:575-590.

Lapresa R, Agulla J, Sánchez-Morán I, Zamarreño R, Prieto E, Bolaños JP, Almeida A (2019), Amyloid-ß promotes neurotoxicity by Cdk5-induced p53 stabilization. Neuropharmacology 146:19-27.

Livak KJ, Schmittgen TD (2001), Analysis of relative gene expression data using real-time quantitative PCR and the 2(-Delta Delta C(T)) Method. Methods 25:402-408.

Shao X, Yang X, Shen J, Chen S, Jiang X, Wang Q, Di Q (2020), TNF-α-induced p53 activation induces apoptosis in neurological injury. J Cell Mol Med 24:6796-6803.

Vulin I, Tenji D, Teodorovic I, Kaisarevic S (2025), Undifferentiated versus retinoic acid-differentiated SH-SY5Y cells in investigation of markers of neural function in toxicological research. Toxicol Mech Methods 35:53-63.

Yoon C-S, Kim D-C, Gogo G, Kim K-S, Ko W, Sohn J, Yim JH, Kim Y-C, et al. (2014), Anti-neuroinflammatory effect of aurantiamide acetate from the marine fungus Aspergillus sp. SF-5921: Inhibition of NF-κB and MAPK pathways in lipopolysaccharide-induced mouse BV2 microglial cells. Int Immunopharmacol 23:568-574.

P-054:

INULIN EXERTS NEUROPROTECTIVE EFFECTS IN D-GALACTOSE-INDUCED AGED RATS

Yanmei Huang[1,2,3], Chanisa Thonusin[2,3,4], Nipon Chattipakorn[2,3,4,5], Siriporn C Chattipakorn[2,3,6]*

[1]*Department of Ultrasonography, The Affiliated Hospital of Youjiang Medical University for Nationalities, Baise 533000, Guangxi, China.*
[2]*Neurophysiology Unit, Cardiac Electrophysiology Research and Training Center, Faculty of Medicine, Chiang Mai University, Chiang Mai 50200, Thailand*
[3]*Center of Excellence in Cardiac Electrophysiology, Chiang Mai University, Chiang Mai 50200, Thailand*
[4]*Cardiac Electrophysiology Unit, Department of Physiology, Faculty of Medicine, Chiang Mai University, Chiang Mai 50200, Thailand*
[5]*The Academy of Science, The Royal Society of Thailand, Bangkok, Thailand*
[6]*Department of Oral Biology and Diagnostic Sciences, Faculty of Dentistry, Chiang Mai University, Chiang Mai 50200, Thailand*
**Corresponding author. Email: scchattipakorn@gmail.com*

Abstract

Background: Aging is a major cause of neurodegeneration. The prebiotic, inulin, has shown neuroprotective effects against obesity. However, its benefits to the aging brain remain unexplored.

Aim: To investigate the effects of inulin on the brain in D-galactose-induced aged rats.

Methods: All experiments were approved by Laboratory Animal Center, Chiang Mai University, Thailand (approval number: 2567/RT-0005) and conducted in accordance with the US National Research Council (2011). Male Wistar rats were housed (two per cage) under controlled temperature (22 ± 2 °C) and humidity (50-60%) on a 12:12-h light-dark cycle with *ad libitum* access to standard chow and water. Five-month-old male Wistar rats were allocated into two groups. The control group (n = 5) received normal saline for twenty weeks, while the remaining (n = 10) were subcutaneously injected with 150 mg/kg/day of D-galactose for twenty weeks to induce aging. From week nine, D-galactose-treated rats were subdivided (n = 5 per group) to receive either normal saline (D-gal group) or 2 g/kg/day of inulin orally (DI group). At the end, all rats underwent behavioral tests and were then euthanized for brain tissue collection.

Results: The D-gal group exhibited cognitive deficits, along with several molecular abnormalities in the brain. These included mitochondrial dysfunction, inflammation, cellular senescence, and apoptosis. Inulin administration could attenuate these pathologies.

Conclusion: Inulin exerted neuroprotection against the aging brain via alleviating mitochondrial dysfunction, inflammation, cellular senescence, and apoptosis. All of which potentially resulted in the amelioration of cognitive deficits.

Keywords: Aging; Brain; Cognitive impairment; Inulin

P-055:

REGULATORY MECHANISMS OF MEMBRANE TRAFFICKING TRANSCRIPT LOCALIZATION AT THE *DROSOPHILA* NEUROMUSCULAR SYNAPSE

Tai Chaiamarit[1,3*], Danail Stoychev[2,3], Francesca Robertson[3,4], Jeffrey Y Lee[2,3], Ilan Davis[2,3]

[1] *Department of Physiology, Faculty of Science, Mahidol University, Bangkok, Thailand*
[2] *School of Molecular Biosciences, College of Medical, Veterinary and Life Sciences, University of Glasgow, Glasgow, UK*
[3] *Department of Biochemistry, University of Oxford, Oxford, UK*
[4] *School of Biochemistry, University of Bristol, Bristol, UK*
**Corresponding author. Email: tai.chm@mahidol.ac.th*

Abstract

Background: Long-distance transport of mRNA to undergo localized translation in distal axons underlies synaptic development and plasticity. Highly polarized cells such as neurons rely on a tight regulation of membrane trafficking by a network of Rab GTPases and their chaperone, Nonetheless, little is known about the post-transcriptional regulation and function of mRNA that produces a membrane trafficking regulator in the periphery.

Aim(s): We aim to investigate the regulatory mechanisms of localization of *rab* mRNA by identifying the molecular motors and RNA binding proteins (RBPs) for its axonal transport and test their functional requirement in synaptic plasticity at the *Drosophila* larval neuromuscular junction (NMJ).

Methods: We use a combination of RNA single molecule *in situ* hybridization (smFISH), quantitative image analysis, and RNA genetic tagging approaches for live imaging and functional behavioral analysis.

Results: We found that transcripts of *rab11* and its chaperone *gdi* are abundant at the NMJ pre-synapse, display bi-directional movement, and colocalize with kinesin-1 and a conserved Imp RBP in motoneuron axons. Knocking down kinesin-1 and/or Imp specifically in motoneurons during the fly larval stage reduces the copy number of *rab11* mRNA at the NMJ pre-synaptic terminals and results in defects of neuromuscular synaptic plasticity and locomotion. Furthermore, we found several Rab GTPase transcripts in neurons, glia, and muscle at the NMJ.

Conclusion: mRNA localization is a generalized strategy for membrane trafficking regulation of components at the tripartite synapse. These results highlight unexplored complexity of membrane trafficking regulation at cellular protrusions of neurons and glia by localized translation of small GTPase network with potential relevance to synaptic pathophysiology.

Keywords: mRNA, axonal transport, synaptic plasticity, Drosophila, neuromuscular junction

---

P-056:

AN IMPACT OF SLEEP QUALITY ON RISKY-DECISION MAKING BEHAVIORS AND ITS NEURAL ACTIVITY IN THAI HEALTHY ADOLESCENTS AND ADULTS: AN EVENT-RELATED POTENTIAL STUDY

Mananya Potima[1*], Vorasith Siripornpanich[1]

[1]*Research Center of Neuroscience, Institute of Molecular Biosciences, Mahidol University, Salaya, Nakhon Pathom 73170, Thailand:*
**Corresponding author. Email: mananya.ptm@gmail.com*

Abstract

Background: Sleep hygiene is an essential factor for health, well-being, and longevity. However, nightlife, prolonged electronics usage, and widespread sleep deprivation have become 21st-century trends in modern life across all ages, leading to sleep quality deficits and subsequently making worse decisions during risk situations in some manner.

Aim(s): To emphasize how sleep quality impacts behaviors related to risky decision-making at both behavioral and neurophysiological levels.

Methods: Fifty-three healthy participants aged 13-45 years old were assessed for risky decision-making behaviors and their sleep information through behavioral assessments (e.g., Thai-Pittsburgh Sleep Quality Inventory; TPSQI). Their brain activity was recorded using electroencephalography (EEG) during several risky situations, including impulsivity (Delay Discounting Task; DDT), risk-taking mechanism (Modified Iowa Gambling Task; MIGT), and inhibitory control (Stroop Color and Word Test; SCWT). The EEG data were converted into the Event-Related Potentials (ERPs) technique. Then all data were further statistically analyzed with an independent T-test between good sleepers (TPSQI <5) and poor sleepers (TPSQI >5).

Results: Surprisingly, the majority of participants (73.6%) exhibited poor sleep patterns. Even though poor sleepers were prone to more impulsivity and less inhibition, no significant differences were found between good and poor sleepers at the behavioral levels (p >.05). Nevertheless, ERP data revealed significant differences between the two sleepers. Poor sleepers exhibited faster latencies in centroparietal N1, P2, N2, and P3 components, as well as lower frontoparietal P2 and P3 amplitudes during risky decision-making processes (p <.05), which is commonly found after sleep deficiency.

Conclusion: Although behavioral differences are scarcely seen among general sleepers, the alteration of neural patterns appears when making a risk decision. Hence, sleep interferes with risk decision and silently modifies neural cognition even in normal conditions. Further study should explore various occasions of poor sleep quality and quantity that affect other higher cognitive functions.

Keywords: Sleep Quality, Risky Decision-Making, Impulsivity, Risk-Taking, Event-Related Potentials

---

P-057:

A PILOT STUDY OF INATTENTIONAL BLINDNESS: THE UNDERSTANDING OF NEURAL ACTIVITY AND BEHAVIORS OF THE ATTENTIONAL NETWORK IN HEALTHY THAI FEMALE ADULTS

Kurisara Boonyakitmaitree[1*], Mananya Potima[1], Prao Pongpipat[1], Patcharapha Poonthawatsanti[1], Samuel T. Ward[2], and Vorasith Siripornpanich[1]

[1]*Research Center of Neuroscience, Institute of Molecular Biosciences, Mahidol University, Salaya, Nakorn Pathom 73170 Thailand:*
[2]*Faculty of Psychology, Chulalongkorn University, Wangmai, Pathumwan, Bangkok 10330, Thailand:*
**Corresponding author. Email: ncode.nano@gmail.com*

Abstract

Background: Selective attention is defined as the process that allows people to focus on an essential task while minimizing or tuning out others that are irrelevant. However, this advantage can also lead people to overlook important information unconsciously. This phenomenon is called inattentional blindness (IB). When IB occurs in critical situations, it can lead to serious consequences, such as car accidents or medical errors. Previous studies have revealed that various factors can affect the rate of IB, such as age, task complexity, and stimulus salience

Aim(s): This study aims to investigate the brainwave patterns in both normal attention and IB, as well as other factors that may influence the occurrence of IB.

Methods: 10 healthy female participants aged 19-35 years took part in this study. All participants performed an Adapted Attention Network Task (AANT), which was modified to induce IB by showing unexpected stimuli outside the central arrow. During AANT, EEG data were recorded and analyzed using both Quantitative Electroencephalography (qEEG) and Event-Related Potentials (ERP). After the task, participants completed an IB awareness questionnaire assessment whether they had noticed unexpected stimuli, completed forms about gaming frequency and personal background, such as academic performance, education duration, personality, and game genre preferences.

Results: At a neural level, both qEEG and ERP data showed no significance among four conditions including congruence, incongruence, congruence with IB, and incongruence with IB. Nevertheless, there were some contextual factors significantly associated with attention and inattentional blindness at a behavioral level, such as age, personality, and family type ($p < 0.05$).

Conclusion: Therefore, the effects of the contextual factors might individually modulate attentional processes, particularly under conditions of inattentional blindness.

Keywords: Attention, Attention Network Test, Inattentional Blindness, Event-Related Potentials, Quantitative Encephalography

---

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[1], Hathaitip Sritanaudomchai[2], Kovit Pattanapanyasat[3], Dungdol Narasirilrk[4], Jinnipa Matchwong[4], Sukonthar Ngampramuan[1*]

[1]*Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom, Thailand*
[2]*Department of Oral Biology, Faculty of Dentistry, Mahidol University, Bangkok, Thailand*
[3]*Siriraj Center of Research Excellence for Microparticle and Exosome in Diseases, Research Department, Faculty of Medicine Siriraj Hospital, Mahidol University, Thailand*
[4]*Experimental animals Unit, Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom, Thailand*
[*]*Corresponding author. Email: sukonthar.nga@mahidol.ac.th*

Abstract

Background: Ischemic stroke, caused by arterial occlusion and reduced cerebral perfusion, is the most common stroke subtype and a leading cause of death and disability worldwide. However, current therapeutic options remain limited. Mesenchymal stem cells (MSCs)-derived exosomes, nano-sized extracellular vesicles (EVs), play essential roles in intercellular communication and tissue repair.

Aim(s): This study investigates the therapeutic effects of exosomes derived from stem cells of human exfoliated deciduous teeth (SHED-EXOs) on neurological and motor recovery following ischemic stroke in rats.

Methods: SHED-EXOs were isolated and characterized according to the “MISEV2018” guidelines. Ischemic stroke was induced in rats by bilateral common carotid artery occlusion (BCCAO). Cerebral blood flow was evaluated using laser Doppler flowmetry. Male Sprague-Dawley rats were randomly assigned to four groups: sham, sham with SHED-EXOs (100 µg) treatment, BCCAO, and BCCAO with SHED-EXOs (100 µg) treatment. SHED-EXOs were administered intravenously (IV) 3 hours after the BCCAO procedure. Neurological impairments were assessed using the modified neurological severity score (mNSS) at 1, 7, and 14 days post-surgery. Motor function was evaluated with the grip strength test at the same time points. All experimental protocols and procedures were approved by Mahidol University-Institute Animal Care and Use Committee (COA.NO.IMB-ACUC 2024/010).

Results: SHED-EXOs were successfully isolated using differential centrifugation. Nanoparticle tracking analysis (NTA) and transmission electron microscopy (TEM) revealed exosomes ranging from 60–150 nm in size, with a modal diameter of 134 nm and a characteristic cup-shaped morphology. The expression of exosome-specific markers (CD9, CD63, and CD81) was confirmed by flow cytometry. Treatment with SHED-EXOs significantly enhanced neurological recovery and increased muscle strength in rats following ischemic stroke.

Conclusion: SHED-derived exosomes improved neurological and motor recovery, supporting their potential as a therapeutic option for ischemic stroke.

Keywords: Ischemic stroke, SHEDs-derived exosomes, Neurological recovery, Bilateral common carotid arteries occlusion, Extracellular vesicles.

P-059:

ELECTROPHYSIOLOGICAL MARKERS OF INHIBITORY CONTROL USING AN EMOTIONAL GO/NO-GO PARADIGM IN HEALTHY ADULTS: A PILOT FOR FUTURE MDD STUDIES

Fahsai Kaewpikul[1], Jiraporn Panmanee[1], Sukonthar Ngampramuan[1], Vorasith Siripornpanich[1*]

[1]Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Salaya, Nakhon Pathom 73170 Thailand
**Corresponding author. Email: vorasith.sir@mahidol.ac.th*

Abstract

Background: Major depressive disorder affects over 280 million people globally and is closely linked to suicide and non-suicidal self-injury. Thailand has seen rising rates of depression and suicide, especially among adults. Impaired inhibitory control is a key cognitive deficit in MDD. The Go/No-Go task reflects neural markers of inhibition in healthy individuals. This pilot study validates the task in a healthy sample to support a future study.

Aims: This preliminary study examines the emotional Go/No-Go paradigm as a measure of inhibitory control in healthy young adults. We hypothesize that the findings will reflect characteristic patterns of inhibition modulated by emotional facial expressions, with emotional valence influencing inhibitory processing.

Methods: Five healthy young adults, aged 19 to 39, participated in the study. Neural activity during the emotional Go/No-Go task was assessed using electroencephalography (EEG) with an event-related potential (ERP). ERP analysis was focused on the N2 and P3 components, which are strongly associated with inhibitory control.

Results: Preliminary analyses revealed significant amplitude differences in both the N2 and P3 components between No-Go and Go conditions. No-Go trials elicited significantly larger N2 amplitudes than Go trials at all midline electrodes, reflecting enhanced conflict monitoring. Likewise, P3 amplitudes were significantly higher for No-Go compared to Go trials at *Fz, FCz, Cz,* and *CPz*, consistent with increased inhibitory control processes. Additionally, P3 latency differed significantly between conditions at *CPz* and *Pz* ($p < 0.05$).

Conclusion: These pilot findings indicate that the emotional Go/No-Go paradigm captures ERP markers of inhibitory control, with increased N2 and P3 amplitudes during No-Go trials under negative emotional conditions. Despite the small sample, the protocol is practical and suitable for refinement. Future studies in larger and clinical samples may identify electrophysiological markers of impaired inhibition and suicidality.

Keywords: major depressive disorder (MDD), suicidal behaviors, non-suicidal self-injury (NSSI), electroencephalogram (EEG)

P-060:

INHIBITION OF SPHINGOSINE KINASE 1 BY PF543 IMPROVES AIRWAY HYPERREACTIVITY AND NEUROCOGNITIVE OUTCOMES IN A MURINE MODEL OF BRONCHOPULMONARY DYSPLASIA

Tara Sudhadevi[1], Prathima Basa[2], Ian Adams[2] and Anantha Harijith[3]

1 Centre for Neuroscience, Cochin University of Science and Technology, Kochi, Kerala, India 682022
2 Pediatrics, Case Western Reserve University, Cleveland, OH, United States
3 University of Arizona, Tucson, Arizona, United States

Abstract

Background: Bronchopulmonary dysplasia (BPD) is a major complication of prematurity, mainly driven by hyperoxia-induced lung injury. Beyond respiratory impairment, BPD is increasingly associated with long-term neurodevelopmental deficits. Sphingosine kinase 1 (SphK1), a key enzyme in sphingolipid metabolism, has been implicated in oxidative stress, inflammation, and tissue injury, suggesting it as a potential therapeutic target.

Aim: To investigate whether pharmacological inhibition of SphK1 by PF543 improves lung structure, function, and neurocognitive outcomes in a murine model of BPD.

Methods: C57BL/6 mice were exposed to 95% O_2 from postnatal day (PN) 3 to PN10 to induce BPD. PF543 was administered daily from the end of hyperoxia until PN28. Animals were studied at PN28 (early outcomes) and PN56 (long-term outcomes). Histological analysis (mean linear intercept, MLI) was performed to assess alveolarization. Lung function testing evaluated airway hyperreactivity, while neurobehavioral assessments measured learning and memory.

Results: Hyperoxia exposure resulted in persistent alveolar simplification, with significantly increased MLI at PN28. PF543 treatment markedly reduced MLI, indicating improved alveolarization. Functional studies demonstrated that hyperoxia caused airway hyperreactivity and impaired lung function, both of which were significantly improved with PF543 administration. Furthermore, hyperoxia-exposed mice exhibited neurocognitive and behavioral deficits, which were rescued by PF543 treatment, highlighting its impact on the lung–brain axis.

Conclusion: SphK1 inhibition by PF543 improves both pulmonary and neurocognitive outcomes in a murine BPD model. These findings position SphK1 as a promising therapeutic target for addressing the dual complications of BPD – lung dysfunction and neurodevelopmental impairment.

Keywords: Bronchopulmonary dysplasia; Sphingosine kinase 1; PF543; Hyperoxia; Neurocognition

---

P-061:

TOCOTRIENOL-RICH FRACTION FROM PALM OIL ATTENUATES MOTOR, LEARNING AND MEMORY DEFICITS IN A ZEBRAFISH MODEL OF PARKINSON'S DISEASE

Thenmoly Damodaran[1*] and Dhanveer Ahamed Bathurutheen [2]

[1] Department of Basic Medical Sciences, Faculty of Dentistry, Aimst University, 08100 Bedong, Kedah, Malaysia.
[2] Faculty of Applied Science, Aimst University, 08100 Bedong, Kedah, Malaysia
*Corresponding author. Email: thenmoly@aimst.edu.my

Abstract

Background: Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by motor impairments and non-motor symptoms, including cognitive decline. Oxidative stress and mitochondrial dysfunction are central to PD pathogenesis. Tocotrienol-rich fraction (TRF), derived from palm oil, is a potent antioxidant with reported neuroprotective properties, but its effects on motor and cognitive functions in PD models remain underexplored.

Aim(s): This study aimed to investigate the preventative neuroprotective effects of 28-days TRF supplementation on motor performance, learning, and memory in a rotenone-induced zebrafish model of PD.

Methods: Adult zebrafish (*Danio rerio*) were exposed to rotenone (5 μg/L) to induce PD-like pathology. TRF was administered orally at doses of 60, 80, and 120 mg/kg concurrently with rotenone exposure to evaluate its preventive neuroprotective effects. Motor function was assessed using the open-field test (total distance traveled, average speed, exploration rate, and mobility rate). Cognitive function was evaluated using the object discrimination test (recognition memory) and Y-maze test (spatial working memory). Behavioral outcomes were recorded and statistically analyzed.

Results: Rotenone exposure significantly reduced locomotor activity in zebrafish, as evidenced by decreased swimming speed, distance traveled, exploration, and mobility rates. TRF supplementation at all doses reversed these motor deficits. In the object discrimination test, rotenone-treated zebrafish exhibited impaired recognition memory, indicated by reduced preference for the novel object. TRF supplementation (80 and 120 mg/kg) restored recognition memory, increasing novel object preference in a dose-dependent manner. Similarly, in the Y-maze test, rotenone-treated zebrafish spent less time in the novel arm, indicating impaired spatial working memory. TRF supplementation at all doses significantly improved novel arm exploration, with greater effects observed at higher doses.

Conclusion: TRF supplementation attenuates both motor and cognitive impairments in rotenone-induced PD zebrafish. The neuroprotective effects of TRF are likely mediated through the reduction of oxidative stress and preservation of mitochondrial function, highlighting its therapeutic potential as a natural antioxidant in PD management.

Keywords: Parkinson's disease, tocotrienol-rich fraction, zebrafish, neuroprotective, rotenone

---

P-062:

ESTABLISHING *IN VITRO* MODELS OF ASTROCYTE AGING FOR TESTING ANTI-AGING EFFECTS OF FRACTIONATED *CELASTRUS PANICULATUS* SEED EXTRACTS

Phossawee Kongkaew[1*], Siriporn Chamniansawat[1]

[1] *Biomedical Sciences, Faculty of Allied Health Sciences, Burapha University, Muang Chonburi, Chonburi, 20131*
**Corresponding author. Email: garfield29phos@gmail.com*

Abstract

Background: Astrocytes undergo age-related alterations that contribute to impaired neuronal support and neurodegenerative processes. Natural compounds such as *Celastrus paniculatus* (CP) have been proposed to exert neuroprotective and anti-aging properties.

Aims: To establish in vitro models of astrocyte aging and to preliminarily evaluate the effects and cytotoxicity of CP fractions.

Methods: Primary astrocytes were subjected to either long-term culture (30 days in vitro; DIV) or oxidative stress induction with hydrogen peroxide (H_2O_2) to mimic aging conditions. Astrocytic reactivity was assessed by glial fibrillary acidic protein (GFAP) expression. To examine the safety of CP fractions, cell viability of non-induced astrocytes was evaluated using MTT assay after treatment with individual fractions.

Results: Long-term culture (30 DIV) led to a progressive increase in GFAP expression over time, without evident morphological changes. In contrast, H_2O_2-induced stress resulted in both elevated GFAP expression and marked morphological alterations, including enlarged and flattened cell bodies, pronounced cytoplasmic spreading, and vacuolization. Notably, treatment with fractionated CP seed extracts (10 µg/mL) showed no cytotoxic effects on astrocytes, with fraction 6 (a more polar fraction) showing no cytotoxicity in non-induced astrocytes (110% cell viability).

Conclusion: Both long-term culture and H_2O_2 exposure effectively induced astrocyte aging, with oxidative stress producing more pronounced morphological changes. Among the CP seed extracts, fraction 6 (F6) was non-toxic to cell, supporting its potential as a neuroprotective candidate.

Keywords: (Astrocyte aging, *Celastrus paniculatus*, GFAP)

P-063:

MUSCLE-DERIVED APELIN MEDIATES EXERCISE-INDUCED ANTIDEPRESSANT EFFECTS AND HIPPOCAMPAL NEUROPLASTICITY

Jiasui Yu[1,2], Suk-yu Yau[1,2*]

[1]*Department of Rehabilitation Sciences, Faculty of Health and Social Sciences, The Hong Kong Polytechnic University, Hung Hom, Hong Kong S.A.R., China.*
[2]*Mental Health Research Center (MHRC), The Hong Kong Polytechnic University, Hung Hom, Hong Kong S.A.R., China.*
**Corresponding author. Email: author@institute.ac.th*

Abstract

Background: Physical exercise exerts antidepressant effects, yet the underlying molecular mechanisms remain incompletely understood. Apelin, a myokine implicated in muscle atrophy, may mediate muscle–brain communication relevant to mood regulation.

Aim: To determine whether muscle-derived apelin mediates the antidepressant effects of voluntary running and to examine its role in hippocampal neurogenesis and synaptic plasticity.

Methods: Male C57BL/6J mice (6 weeks old, n = 8–10 per group) were subjected to chronic unpredictable stress (CUS) to induce depression-like behaviors, followed by 4 weeks of voluntary wheel running. Control groups included non-stressed sedentary and non-stressed running mice. Skeletal muscle-specific apelin knockout ($APLN^{fl/fl}$, $MCK\text{-}Cre^{+/-}$) mice, adeno-associated virus (AAV)-mediated apelin overexpression in muscle, and apelin receptor knockdown in ventral hippocampal glutamatergic neurons were used to assess the sufficiency and necessity of apelin in hippocampal plasticity.

Results: Voluntary running increased sucrose preference in CUS mice (76.71 ± 1.57 vs. 80.83 ± 1.57), elevated apelin mRNA in muscle (3.14 ± 0.35 vs. 1.00 ± 0.35) and enhanced hippocampal apelin protein (0.984 ± 0.168 vs. 0.476 ± 0.168). Muscle-specific apelin deletion abolished these effects, reducing sucrose preference (68.08 ± 1.67 vs. 78.45 ± 1.67) and hippocampal progenitor cells (1566 ± 123.8 vs. 1985 ± 123.8) compared with flox control mice ($APLN^{fl/fl}$, $MCK\text{-}Cre^{-/-}$). Conversely, apelin overexpression mimicked running's benefits, increasing grooming duration in the sucrose splash test (116.1 ± 9.61 vs. 82.72 ± 9.61), and hippocampal NMDA receptor phosphorylation (1.56 ± 0.13 vs. 1.00 ± 0.13). APJ knockdown in ventral hippocampal neurons decreased the positive effects of running in sucrose preference (83.97 ± 2.06 vs. 89.95 ± 2.06), hippocampal neurogenesis (1843 ± 110.8 vs. 2257 ± 110.8), and NMDA receptor phosphorylation (1.161 ± 0.124 vs. 1.532 ± 0.124).

Conclusion: Muscle-derived apelin is crucial for the antidepressant and neurogenic effects of exercise, acting via APJ-mediated hippocampal plasticity.

Keywords: Apelin, Exercise, Depression, Neurogenesis, Hippocampus

---

P-064:

HUMAN MICROGLIAL RESPONSES TO RESVERATROL EXPOSURE: INSIGHTS FROM MORPHOLOGICAL IMAGING AND PROTEOMIC APPROACHES

Ekkaphot Khongkla[1,*], Kornkanok Promthep[1], Pipob Suwanchaikasem[2], Fikri Aminullah[3], Rafi Aliefiyanto[3], Dana Mohammad Khader AlNajmi[4], Farouk Raed[4], Maral Daneshpazhouh[4], Banthit Chetsawang[1]

[1] *Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Salaya, Nakhon Pathom, Thailand*
[2] *Baiya Phytopharm Co., Ltd., Bangkok, Thailand*
[3] *Faculty of Medicine Universitas Airlangga, Surabaya, Jawa Timur, Indonesia*
[4] *College of Medicine, Ajman University, Ajman, United Arab Emirates*
**Corresponding author. Email: ekkaphot.kho@mahidol.ac.th*

Abstract

Background: Microglia are resident immune cells of the central nervous system and play critical roles in maintaining brain homeostasis and responding to pathological stimuli. Their morphology varies depending on environmental cues, with elongation of processes and branching often reflecting functional adaptations. Resveratrol, a natural activator of sirtuin-1 (SIRT1), has been proposed as a therapeutic candidate against neurodegeneration due to its broad modulatory effects on aging-related molecular pathways. However, despite substantial evidence of its protective roles, the effects of resveratrol on human microglia remain poorly characterized, particularly at the levels of morphology and global proteomic regulation.

Aim: We investigated the effects of resveratrol on the morphology and proteomic landscape of human microglial HMC3 cells.

Methods: Human microglial HMC3 cells were exposed to sublethal and lethal dose of resveratrol. The immunocytochemistry staining for IBA-1 was used for morphological tracking analysis. LC-MS/MS -based proteomic approach was conducted for unbiased protein expression profile and their biological insight.

Results: Resveratrol significantly upregulated SIRT1 expression at both mRNA and protein levels. Morphological tracking analysis revealed that resveratrol-treated microglia exhibited an elongated morphology with increased branching processes. LC-MS–based quantitative proteomics coupled with bioinformatic analysis was performed, identifying 877 proteins in total. Functional enrichment analysis indicated a predominant association with translation-related processes, suggesting that resveratrol alters protein biosynthesis pathways. Among these, 22 proteins were significantly differentially expressed, with cellular localization analysis showing pronounced enrichment in extracellular vesicles and exosomes, highlighting a potential role in cell–cell communication. Notably, upregulation of Cathepsin D, S100, and Cytochrome c oxidase at the lethal dose of resveratrol indicated possible toxicity, underscoring the importance of dosage in its cellular effects.

Conclusion: Resveratrol contributes to human microglial plasticity by modulating morphology and proteomic profiles. Nonetheless, dose-dependent induction of stress-related proteins highlights the need for careful consideration of therapeutic applications.

Keywords: Resveratrol, Human Microglia Cells, IBA-1, Proteomics, Functional Enrichment

---

P-065:

GENETIC ASSOCIATION OF THE *SLC6A4 5-HTTLPR-STIN2* HAPLOTYPE WITH SUICIDAL BEHAVIOR: A SYSTEMATIC REVIEW AND META-ANALYSIS

Sulaifan Waehama[1], Nathorn Chaiyakunapruk[2], Ratree Sawangjit[3], Benjamard Thaweethee-Sukjai[4], Samur Thanoi[5], Jureepon Roboon[7,8], Gavin P. Reynolds[6,7], Sutisa Nudmamud-Thanoi[7,8*]

[1]*Medical Science Graduate Program, Faculty of Medical Science, Naresuan University, Phitsanulok, 65000, Thailand*
[2]*Department of Pharmacotherapy, University of Utah College of Pharmacy, Salt Lake City, UT, 84112, USA*
[3]*Clinical Trials and Evidence-Based Syntheses Research Unit, Mahasarakham University, Maha Sarakham, 44150, Thailand*
[4]*School of Medicine, Mae Fah Luang University, Chiang Rai, 57100, Thailand*
[5]*School of Medical Sciences, University of Phayao, Phayao, 56000, Thailand*
[6]*Biomolecular Sciences Research Centre, Sheffield Hallam University, Howard Road, Sheffield, S1 1WB, UK*
[7]*Centre of Excellence in Medical Biotechnology, Faculty of Medical Science, Naresuan University, Phitsanulok, 65000, Thailand*
[8]*Department of Anatomy, Faculty of Medical Science, Naresuan University, Phitsanulok, 65000, Thailand*
**Corresponding author. E-mail: Sutisat@nu.ac.th*

Abstract

Background: The serotonin transporter gene (*SLC6A4)* plays a crucial role in serotonin regulation and has been implicated in the pathophysiology of suicidal behavior. Although the long (L) and short (S) alleles of the serotonin transporter-linked polymorphic region (*5-HTTLPR*) and the 12 and 10-repeat alleles of the serotonin transporter intron 2 (*STin2*) in the *SLC6A4* gene have been extensively studied individually, their combined haplotypic effect may provide greater insight into genetic risk. However, this haplotypic effect on suicidal behavior risk remains unclear.

Aim: This study aimed to clarify the association between *SLC6A4, 5-HTTLPR-STin2* haplotypes and the risk of suicidal behavior through a systematic review and meta-analysis.

Methods: A systematic search of PubMed, Scopus, and Web of Science was conducted through June 2025. Case-control studies meeting Hardy-Weinberg equilibrium in controls ($p > 0.05$) were included. Two independent reviewers screened studies, assessed quality using the Newcastle-Ottawa Scale, and extracted data. Odds ratios (ORs) with 95% confidence intervals (CIs) were pooled using fixed- or random-effects models according to heterogeneity (I^2).

Results: From 553 records, six studies were included, comprising 899 suicidal behaviors cases and 1,280 controls. Meta-analysis revealed that the L10 (OR=1.25, 95%CI: 1.04-1.51) and L12 (OR=1.20, 95%CI: 1.01-1.41) haplotypes were significantly associated with an increased risk of suicidal behavior compared with the S12 haplotype. This association was stronger among patients with major depressive disorder (MDD) or other psychiatric disorders who had attempted suicide, where both L10 (OR=1.66, 95%CI: 1.11-2.47) and L12 (OR=1.64, 95%CI: 1.18-2.26) haplotypes conferred a significantly higher risk compared to the S12 haplotype.

Conclusion: The L10 and L12 haplotypes of *SLC6A4* appear to confer an increased risk of suicidal behavior, particularly in individuals with MDD or related psychiatric disorders. These variants may serve as potential biomarkers pending further validation.

Keywords: *5-HTTLPR*, *STin2*, Suicidal Behavior, Genetic Polymorphism, Meta-Analysis

----

P-066:

NON-INVASIVE ASSESSMENT OF AGE-RELATED SWEAT 3-HYDROXYANTHRANILIC ACID LEVELS: IMPLICATIONS FOR EARLY DETECTION OF COGNITIVE DECLINE

Yaowapa Trangan[1,2], Kanlaya Prapainop Katewongsa[3], Preeyanuch Manohong[3], Rungrat Palakai[2,4], Sirinapa Mysook[5,6], Banthit Chetsawang[1], Piyawat Katewongsa[2*]

[1]Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Salaya, Nakhon Pathom, 73170, Thailand,
[2]Thailand Physical Activity Knowledge Development Centre (TPAK), Institute for Population and Social Research,
Mahidol University, Salaya, Phutthamonthon, Nakhon Pathom, 73170, Thailand
[3]Department of Biochemistry, Faculty of Science, Mahidol University, Bangkok, 10400, Thailand
[4]Ubon Ratchathani Provincial Health Office, Muang district, Ubon Ratchathani, 34000, Thailand
[5]Sisaket Provincial Public Health Office, Muang district, Sisaket, 33000, Thailand
[6]Ramathibodi School of Nursing, Faculty of Medicine Ramathibodi Hospital, Mahidol University, Bangkok, 10400,
Thailand
**Corresponding author. Email: piyawat.kat@mahidol.edu*

Abstract

Background: 3-Hydroxyanthranilic acid (3-HAA), a kynurenine pathway metabolite involved in neural, immune, and aging processes, has been linked in plasma and CSF to neurodegeneration particularly in the progression of mild cognitive impairment (MCI). Its detection in sweat may provide a novel, non-invasive biomarker for early cognitive decline.

Aim(s): To investigate age-related changes in sweat 3-HAA in pre-elderly and elderly people.

Methods: This cross-sectional study included 67 healthy Thai people aged 50–79 years from Nakhon Pathom Province. Participants with neurodegenerative diseases, or major medical conditions were excluded. Sweat was collected during physical fitness tests and analyzed for tryptophan (TRP), kynurenine (KYN), and 3-hydroxyanthranilic acid levels (3-HAA) by HPLC, with sodium measured by MP-AES for normalization. Associations were assessed using linear regression.

Results: No significant gender differences were observed in sweat levels of Na, TRP, KYN, and 3-HAA. Whereas age effects were evident: linear regression analysis indicated that sweat KYN levels showed a positive trend with age, suggesting a possible age-related increase (p=0.061). In contrast, TRP remained comparable across age (p=0.862). Moreover, age was positively associated with sweat 3-HAA levels (β = 8.282 (pmol/$\mu g\ Na$) per year, p = 0.026), indicating that 3-HAA increased with age.

Conclusion: Sweat 3-HAA levels increased with age, indicating age-related changes in the kynurenine pathway. The research can continue finding the association between an increased sweat 3-HAA level and MCI.

Keywords: 3-HAA, Age, Sweat, Elderly, Non-invasive

P-067:

THE EFFECT OF FINE PARTICULATE MATTER 2.5 (PM2.5) ON NEURITE DEVELOPMENT IN N2A NEUROBLASTOMA CELLS

Napol Kaewkascholkul[1*], Kunlaya Somboonwiwat[1], Fareda Sapphot[2], Kanokporn Wantem[2]

[1]Center of Excellence for Molecular Biology and Genomics of Shrimp, Department of Biochemistry, Faculty of Science, Chulalongkorn University, Bangkok 10330
[2]Department of Biomedical Science, Faculty of Science, Rangsit University, Pathumthani, 12000
**Corresponding author. Email: napol.k@chula.ac.th*

Abstract

Background: Particulate matter (PM) is one of the most severe air pollutions that effect the public health. Fine particulate matter (PM2.5) not only adversely impacts respiratory diseases but also affects the nervous systems, contributing to neurodegenerative diseases and neurodevelopmental disorders. Exposure to PM2.5 has been linked to impairments in neuronal development, as well as anxiety and depression-like behavior. Despite these observations, the molecular and cellular mechanisms underlying PM2.5-induced neurological impairments remain poorly understood.

Aim(s): Here, we investigated the effects of PM2.5 on neurite development in Neuro2a (N2a) neuroblastoma cells.

Methods: Cell viability assays showed a dose-dependent increase in cell death following PM2.5 exposure. The percentage of neurite-bearing cells was significantly reduced by 17.3% at 25 µg/ml of PM2.5 after one day of incubation.

Results: By day three, neurite-bearing cells were decreased by 40.4% and 43.2% at 12.5 and 25 µg/ml of PM2.5, respectively. In addition, average neurite length was significantly reduced across PM2.5-treated conditions. These findings indicate that PM2.5 inhibits neurite initiation and outgrowth during N2a differentiation. Our results provide cellular evidence that PM2.5 compromises neuronal morphogenesis, suggesting a potential mechanism by which air pollution contributes to neurodevelopmental risk.

Conclusion: Future studies will define microRNA-mediated pathways in PM2.5 neurite deficits and test whether modulating these microRNAs mitigates the effects.

Keywords: PM2.5, neurite outgrowth, Neuro2a, miRNA

P-068:

A HUMAN STEM CELL-DERIVED BIOENGINEERED PLATFORM WITH DEFINED CIRCUIT ARCHITECTURE FOR SYNAPTIC CONNECTIVITY ANALYSIS

Pacharaporn Suklai[1,2,3*], Andrea Serio[1,2]

[1]Department of Basic and Clinical Neuroscience, Institute of Psychiatry Psychology & Neuroscience, King's College London, London, UK, SE5 9RT
[2]The Francis Crick Institute, London, UK, NW1 1AT
[3]National Center for Genetic Engineering and Biotechnology (BIOTEC), NSTDA, Pathum Thani, Thailand, 12120
**Corresponding author. Email: pacharaporn.suk@biotec.or.th*

Abstract

Background: Human-induced pluripotent stem cell (iPSC) technology has enabled the generation of human neuronal models to study mechanisms underlying neurological disorders. However, conventional models often lack defined network architecture, resulting in randomly arranged connections that limited the study of specific neuronal interactions, synapse-relevant diseases, and therapeutic interventions targeting connectivity. Microfluidic platforms partially address these issues, but their enclosed design restricts accessibility, scalability, and downstream analyses.

Methods: We developed a novel, barrier-free bioengineered platform for cortical network modeling that integrates iPSC-derived cortical neurons with bioengineering approaches. Polydimethylsiloxane (PDMS)-based microgroove topographies were used to guide neurite outgrowth, with cell plating guides for precise neuronal positioning. This strategy facilitated the creation of "neuronal nodes" and the assembly of customizable, open-system cortical circuits.

Results: The platform generated neuronal nodes with 5 mm in length (extendable up to 7 mm) and assembled paired nodes spaced 3 mm apart. Incorporating a perpendicular microgroove array diverted neurite extension from the receiving node, creating an exposed, predictable site for projecting axons, supporting unidirectional circuit formation. Live imaging and functional connectivity confirmed successful neurite guidance and connection between the defined neuronal groups. Co-staining for pre- and post-synaptic markers confirmed a significant increase in synapse density at the defined connection sites (n=5 circuits), confirming physical synaptic formation between nodes. Unlike existing open or semi-open systems limited to sparse or small-scale culture, this fully accessible and scalable design enables multi-level analysis, including proteomics, linking circuit architecture to molecular phenotypes.

Conclusions: This platform provides precise control of neuronal circuit topology and quantitative analysis of synaptic connectivity. Its open, scalable design creates new opportunities for mechanistic studies of neurological disorders, offering a powerful tool for disease modeling of synaptic dysfunction, and drug testing targeting synapse formation and function.

Keywords: IPSC-derived neuronal network; bioengineered platform; synaptic formation; on-chip; in vitro modeling

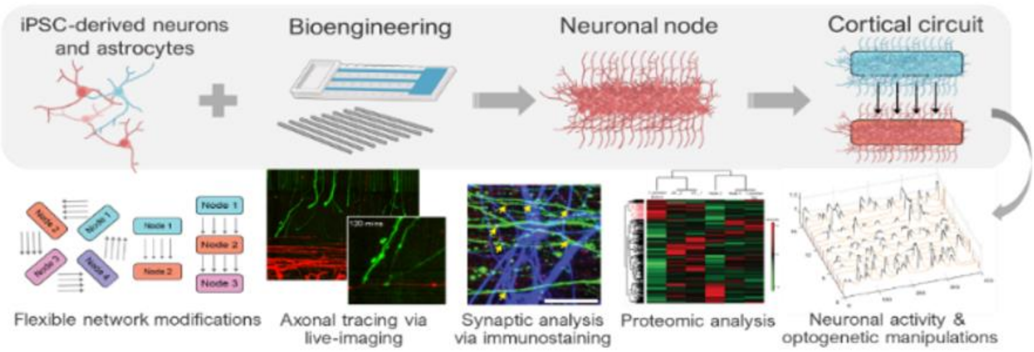


Figure 1. Engineered open-system platform for customizable cortical circuits enabling targeted analysis of synapse formation and function

P-069:

EXPLORING THE LINK BETWEEN NIGHTMARES, SLEEP PARAMETERS, AND PREFRONTAL CORTEX DYSFUNCTION IN MILD COGNITIVE IMPAIRMENT: A PILOT FEASIBILITY STUDY

Kotchapit Maksri[1], Yaowapa Trangan[1], Banthit Chetsawang[1], Siraprapa Boobphahom[1], Vorasith Siripornpanich[1*]

[1]*Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Salaya, Nakorn Pathom 73170 Thailand:*
**Corresponding author. Email: vorasith.sir@mahidol.ac.th*

Abstract

Background: Thailand is rapidly transitioning into a super-aged society, leading to the rising prevalence of dementia and related conditions. Mild cognitive impairment (MCI) represents a transitional stage between normal aging and dementia, with 10–15% of individuals progressing each year. Sleep disturbances, particularly nightmares, have emerged as potential early indicators of cognitive decline due to their association with prefrontal cortex (PFC) dysfunction. However, previous studies have been limited by retrospective reports and predominantly Western samples.

Aim: This pilot study aimed to test the feasibility of integrating nightmare and sleep questionnaires with functional near-infrared spectroscopy (fNIRS) during executive tasks in young adults, prior to application in an elderly MCI cohort.

Methods: Ten healthy young adults completed standardized assessments, including the Pittsburgh Sleep Quality Index (PSQI) and a two-week nightmare diary. Among these, five participants also performed executive function tasks while undergoing fNIRS monitoring, focusing on PFC activation patterns.

Results: Data collection is currently in progress. It is **expected** that the study will demonstrate (i) high feasibility (≥85% diary completion and ≥90% questionnaire completion), (ii) **robust task-evoked PFC activation** (HbO increase/HbR decrease), and (iii) **directional associations** whereby higher nightmare frequency/distress co-occurs with poorer PSQI scores and smaller PFC effect sizes during inhibitory control. These trends will calibrate task timing, artifact management, and sample size estimation for the MCI cohort.

Conclusion: This study is designed to validate methodological protocols for future application in the elderly population with MCI. The findings are anticipated to provide a foundation for establishing nightmares as early and culturally relevant indicators of cognitive vulnerability in aging populations.

Keywords: Mild Cognitive Impairment; Nightmares; Sleep; Prefrontal Cortex; fNIRS

P-070:

COMPUTATIONAL AND EXPERIMENTAL STRATEGIES FOR DEVELOPING NOVEL BUTYRYLCHOLINESTERASE INHIBITORS FOR TREATMENT OF ALZHEIMER'S DISEASE

Ankit Ganeshpurkar [1, 2*], Sushil Kumar Singh [2*]

[1]Department of Pharmaceutical Sciences, Dr. Harisingh Gour Vishwavidyalaya (A Central University), Sagar-470003, Madhya Pradesh, India.
[2]Department of Pharmaceutical Engineering & Technology, Indian Institute of Technology (Banaras Hindu University), Varanasi, India
**Corresponding author: Email: ankitganeshpurkar@gmail.com, sksingh.phe@iitbhu.ac.in*

Abstract

Background: Alzheimer's disease (AD) is a neurodegenerative disorder characterised by progressive cognitive and neuropsychiatric decline, such as apathy and depression. The cholinergic hypothesis implicates the loss of cholinergic function in AD progression. Butyrylcholinesterase (BChE) has emerged as a promising therapeutic target, particularly in the advanced stages of the disease.

Aim(s): To design and evaluate sulfonamide-based BChE inhibitors using machine learning (ML) and experimental methods, to address memory deficits associated with AD.

Methods: A gradient boosting classifier was used to predict potential BChE inhibitors, identifying N-phenyl-4-(phenylsulfonamido)benzamide as a lead compound. Derivatives were synthesised, characterised, and assessed through *in vitro* BChE and acetylcholinesterase (AChE) inhibition assays and blood-brain barrier (BBB) permeability by PAMPA assay. *In vivo* cognitive evaluations using the Y-maze and the Barnes maze were performed on scopolamine-induced amnesia in rats. The compounds were administered through the oral route at a dose of 5,10 and 20 mg/kg for seven consecutive days. Neurochemical assays were conducted to assess cholinergic and antioxidant activities at the molecular level, and *in silico* docking and molecular dynamics simulations were utilised to analyse binding interactions and stability of the compounds with BChE.

Results: The ML model accurately predicted BChE inhibitors. Thirty-six derivatives were synthesised and compounds **34, 37,** and **54** exhibited potent BChE inhibition with IC_{50} values in the nanomolar range and favourable blood-brain barrier permeability. The inhibitors displayed no significant AChE inhibition, indicating high selectivity for BChE. Behavioural studies showed considerable reversal of scopolamine-induced memory deficits at 20 mg/kg. At this dose, both compounds showed a reduction in primary error in the Barnes maze of more than 50%. Neurochemical assays confirmed restoration of cholinergic and antioxidant functions at the cellular level. *In silico* studies revealed stable interactions between the compounds and BChE.

Conclusions: These results demonstrate the potential of ML-assisted design for discovering selective BChE inhibitors with translational relevance for AD therapy. The lead compounds identified, particularly derivatives **34, 37,** and **54**, demonstrated potent BChE inhibition and high selectivity over AChE. These compounds exhibited excellent blood-brain barrier permeability and effectively reversed scopolamine-induced memory deficits in animal models, indicating their therapeutic potential for cognitive impairment associated with AD.

Keywords: Alzheimer's disease, Butyrylcholinesterase, Sulfonamide inhibitors, Machine learning, Cognitive impairment

---

P-071:

ASARININ AS A POTENTIAL DUAL OREXIN RECEPTOR ANTAGONIST FOR THE TREATMENT OF INSOMNIA: THE STUDY OF OREXIN-2 RECEPTOR (ORX2R) DOWNSTREAM SIGNALING CASCADES IN ORX2R-OVEREXPRESSING CELL

Fahsai Thaion[1], Jiraporn Panmanee[1], Narisorn Kitiyanant[2], Kornkanok Promthep[1], Chonnicha Subkod[1], Banthit Chetsawang[1*]

[1]Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Salaya, Nakhon Pathom, Thailand, 73170
[2]Center for Advanced Therapeutics, Institute of Molecular Biosciences, Mahidol University, Salaya, Nakhon Pathom, Thailand, 73170
*Corresponding author. E-mail: banthit.che@mahidol.ac.th

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 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 SH-SY5Y cells were established as an *in vitro* model to examine the potential mechanism of action of asarinin inhibiting ORX2R-mediated cell signaling. Phosphorylation of ERK1/2 was evaluated by Western blot analysis following receptor activation with the selective agonist HY-19320 and treatment with either asarinin or the clinically used DORA, lemborexant.

Results: Western blot analyses demonstrated that asarinin tends to reduce HY-19320-activated ERK1/2 phosphorylation. In contrast, lemborexant at 20 nM and 40 nM significantly decreased HY-19320-activated ERK1/2 phosphorylation to 84.36 ± 18.30% and 59.71 ± 18.30%, respectively. These findings suggest the therapeutic potential of asarinin as a partial DORA for insomnia. Furthermore, ORX2R activation via the Gq/PLC/PKC pathway induces calcium influx and mobilization from intracellular stores, sustaining neuronal excitation. Therefore, further studies should explore the effects of asarinin on additional ORX2R downstream pathways, such as CREB signaling and intracellular Ca^{2+} dynamics, to further evaluate its potential as a novel therapeutic agent for insomnia.

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

---

Background

The International Classification of Sleep Disorders (ICSD) defines chronic insomnia as nocturnal symptoms characterized by difficulty initiating sleep, difficulty maintaining sleep, or early morning awakening, accompanied by impaired daytime functioning such as fatigue, excessive daytime sleepiness (EDS), or reduced concentration and attention. The prevalence of insomnia in the adult population is estimated to range from 10% to 40% (Naha et al, 2024). Insomnia has been associated with a state of hyperarousal, in which wake-promoting neural systems remain active during sleep, thereby disrupting sleep initiation and maintenance (Mogavero et al, 2023). The orexin system plays a central role in regulating these wake-promoting pathways. Orexin, an excitatory neuropeptide derived from prepro-orexin in the lateral hypothalamus, comprises Orexin-A and Orexin-B, which project extensively to multiple brain regions. The orexin peptides bind to the two related G-protein-coupled receptors containing the orexin-1 (ORX1R) and the orexin-2 (ORX2R) receptors. Orexin-A binds to both receptors with similar affinity, whereas orexin-B binds to the ORX2R with higher affinity than ORX1R. The ORX2R signaling through the G_q/PLC/PKC pathway appears to be more robust than that of ORX1R. Binding to the receptor also triggers a PKC-mediated calcium influx across the plasma membrane via L-type calcium channels. Additionally, intracellular calcium stores are released through a phospholipase C (PLC)-mediated pathway. The resulting elevation in calcium levels leads to sustained neuronal excitation and maintains the stability of wakefulness. The roles of orexin support the hypothesis that dysregulation of the orexin system may contribute to the hyperarousal processes underlying insomnia.

Current therapeutic approaches for insomnia include a range of hypnotic agents, such as benzodiazepines, novel benzodiazepine receptor agonists (commonly referred to as Z compounds), and sedating antidepressants. However, these often come with neurological side effects that limit their long-term use. Recent studies have explored new drug options that target the arousal circuit through orexin (hypocretin) and orexin receptors, the dual orexin receptor antagonists (DORAs). These agents block ORX1R and ORX2R receptors, thereby increasing total sleep time primarily by promoting REM sleep while having little to no effect on or even reducing non-REM sleep. Lemborexant, a dual orexin receptor antagonist (DORA), was approved by the Thailand Food and Drug Administration (Thai FDA) in 2024 for treating adult insomnia, particularly for individuals with difficulties falling asleep or maintaining sleep (Pakjira, 2024). Nevertheless, animal experiments and clinical trials still report some adverse effects with lemborexant including drowsiness, CNS depressant effects/daytime impairment, and cataplexy-like symptoms, highlighting the need for further research to identify new molecules capable of inhibiting orexin receptors (orexin receptor antagonists). Our current *in silico* study by drug virtual screening has identified asarinin, a lignan extract from *Asarum sieboldii* Miq., as a promising candidate for a dual orexin receptor antagonist, exhibiting a higher binding affinity than lemborexant. Moreover, we expected that asarinin might have fewer clinical adverse effects when compared to current DORAs. Therefore, this study aimed to investigate the role of asarinin in inhibiting the downstream signaling cascades of the orexin-2 receptor activation in ORX2R-overexpressing cells, suggesting its potential therapeutic application.

Materials and Methods

Cell culture

Human dopaminergic neuroblastoma (SH-SY5Y) cell lines stably expressing human orexin receptor 2 (hORX2R/SH-SY5Y cells) were cultured in Dulbecco's Modified Eagle Medium/Nutrient Mixture F-12, supplemented with 10% fetal bovine serum (FBS) and 100 U/mL penicillin-streptomycin. The cells were maintained in a humidified incubator at 37°C with 5% CO_2 (Zhang et al, 2023).

Immunofluorescence staining (IF)

ORX2R-overexpressing SH-SY5Y cells generated by our research team were used to investigate orexin-2 receptor (ORX2R) downstream signaling cascades. Immunofluorescence staining was performed to check ORX2R overexpression compared with wild-type and empty vector-transfected SH-SY5Y cells. Briefly, human hORX2R/SH-SY5Y cells were seeded onto coverslips in 24-well plates and incubated for 24 h at 37 °C in a humidified incubator with 5% CO_2. Cells were fixed with 4% paraformaldehyde in PBS for 15 min at room temperature, washed three times with 1× PBS, permeabilized with 0.1% Triton X-100 in PBS for 15 min, and washed again with PBS. Non-specific binding was blocked using 1% BSA in PBS for 30 min at room temperature. Cells were then incubated with goat anti-orexin 2 receptor antibody (ab77370) for 1 h at 37 °C, washed, and incubated with fluorescence-conjugated secondary antibody for 1 h at 37 °C. Afterward, coverslips were mounted using Fluoromount-G™ with DAPI. Images were visualized using a confocal microscope.

Cytotoxicity assays

The cytotoxicity of HY-19320, asarinin, and lemborexant was evaluated using the MTT assay (3-[4,5-dimethylthiazol-2-yl]-2,5-diphenyl tetrazolium bromide). Human hORX2R/SH-SY5Y cells were seeded into 96-well plates and pre-incubated for 24 h in a humidified incubator at 37°C with 5% CO_2. Subsequently, the medium was removed, and each well was treated with 100 µl of the orexin-2 receptor agonist (HY19320) at concentrations ranging from 0 to 100 nM (0, 6.25, 12.5, 100 nM), lemborexant at concentrations ranging from 0 to 40 nM (0, 5, 10, 20, 40 nM), or asarinin at concentrations ranging from 0 to 100 µM (0, 25, 50, 100 µM) prepared in FBS-free medium. The cells were then incubated for 24 h in a humidified incubator at 37°C with 5% CO_2. Following incubation, the medium was removed, and 100 µl of MTT solution was added to each well under dark conditions, followed by a 2 h incubation. Afterward, the MTT solution was discarded, and 100 µl of DMSO was added to each well, followed by shaking for 10 minutes. Cell viability was assessed by measuring the absorbance of the color reaction at 570 nm using a microplate reader. The percentage of cell viability was calculated using the following formula (Hyun et al, 2017).

$$\text{Cell Viability (\%)} = \left[\frac{\text{OD}_{\text{Treated}} - \text{OD}_{\text{Blank}}}{\text{OD}_{\text{Control}} - \text{OD}_{\text{Blank}}}\right] \times 100$$

Orexin-2 receptor downstream signaling assay

To investigate orexin-2 receptor (ORX2R) downstream signaling, ORX2R-overexpressing SH-SY5Y cells were stimulated with the selective agonist HY-19320 and subsequently treated with asarinin or lemborexant, as a standard DORA. To investigate the optimal activation of ORX2R, ORX2R-overexpressing cells were incubated with various doses and times of HY-19320. Then, the optimal dose and time of HY-19320 activated the ORX2R were chosen to study the antagonistic effect of asarinin and Lemborexant. Cells were incubated with HY-19320 for the indicated dose and time, and then treated with asarinin (25, 50, and 100 µM) and lemborexant (10, 20, and 40 µM) for the indicated time. The control non-treated cells were incubated in FBS-free medium. The levels of ERK1/2 phosphorylation were examined by Western blot analysis, revealing the effects of asarinin on ORX2R-mediated signaling. Following treatment, the cells were harvested, and protein concentrations were determined using a BCA assay. Proteins were separated by SDS-PAGE and subsequently transferred to PVDF membranes via electrophoresis. The membranes were incubated with primary antibodies, including phospho-p44/42 MAPK (ERK1/2) (Thr202/Tyr204) and p44/42 MAPK (ERK1/2) for 2 h at room temperature. They were then incubated at 4°C overnight and washed with TBST solution. Afterward, the membranes were incubated with secondary antibodies, including Anti-rabbit IgG HRP-linked antibody and anti-biotin HRP-linked antibody, for 1.30 h at room temperature, followed by additional washes with TBST solution. The signal of protein bands was enhanced with Clarity™Western ECL Substrate (1705060, Bio-Rad) and detected by Vilber Fusion FX7 Image analyzer. (Wang et al, 2019).

Statistical analysis

All data were presented as mean ± standard error of the mean (SEM). All data were analyzed using the GraphPad Prism program (GraphPad Software, San Diego, CA, USA). In addition, statistical analysis was also undertaken using the GraphPad Prism program by A one-way analysis of variance (ANOVA) with a $P < 0.05$ for a significant difference.

Results

SH-SY5Ycell lines stably expressing human orexin receptor 2

ORX2R-overexpressing SH-SY5Y cells were generated by our research team for use in investigating orexin-2 receptor (ORX2R) downstream signaling cascades. Immunofluorescence staining **(Figure 1A)** showed that hORX2R/SH-SY5Y cells exhibited higher expression levels of human ORX2R compared with wild-type and empty vector-transfected SH-SY5Y cells. These findings were consistent with Western blot analysis **(Figure 1B)**, which demonstrated increased ORX2R protein expression in hORX2R/SH-SY5Y cells relative to wild-type and empty vector-transfected cells.

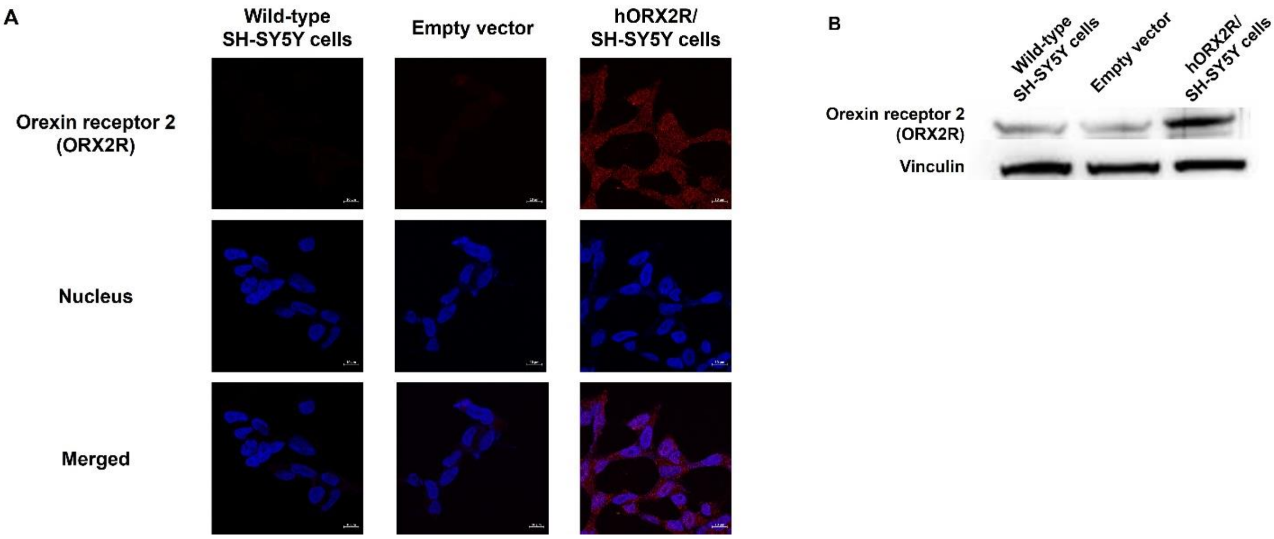


Figure 1 Immunofluorescence staining (A) and Western blot analysis (B) of hORX2R/SH-SY5Y cells stably expressing the human orexin-2 receptor compared with wild-type and empty vector-transfected SH-SY5Y cells. Red color indicated hORX2R-positive immunostaining. Cell nucleus-DAPI staining was shown in blue color. (Scale bars = 10 μm)

The effect of asarinin on cell viability in hORX2R/SH-SY5Y cells

The cytotoxic effects of HY-19320, asarinin, and lemborexant were evaluated using the MTT assay, as shown in **Figure 2**. HY-19320 at 6.25, 12.5, and 100 nM did not reduce the viability of hORX2R/SH-SY5Y cells compared to control-untreated cells. Similarly, asarinin at 25, 50 and 100 μM, and lemborexant at 5, 10, 20 and 40 nM showed no cytotoxicity in hORX2R/SH-SY5Y cells compared to control-untreated cells.

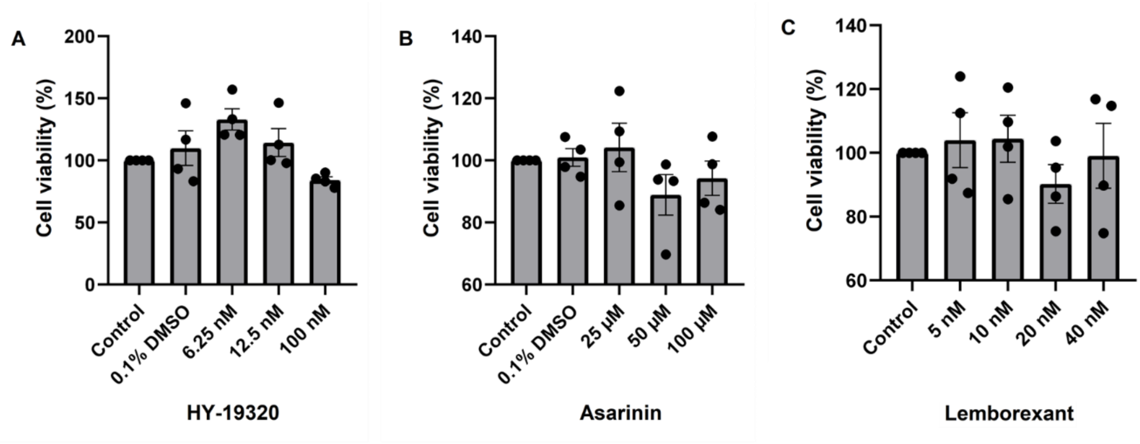


Figure 2 The cell viability of HY-19320, asarinin, and lemborexant in human hORX2R/SH-SY5Y cells after 24 h of treatment was evaluated using the MTT assay. Results are expressed as means ± S.E.M. of four independent experiments. Statistical analysis was performed using one-way ANOVA.

The effect of HY-19320 on phosphorylation of ERK1/2 in hORX2R/SH-SY5Y cells

HY-19320 increased phosphorylation of extracellular signal-regulated kinase 1/2 (ERK1/2), as shown in **Figure 3**. The results revealed that HY-19320 at 10 nM for 6 h significantly increased the phosphorylation of ERK1/2 to 400.9 ± 123.6 % of the control-untreated cells.

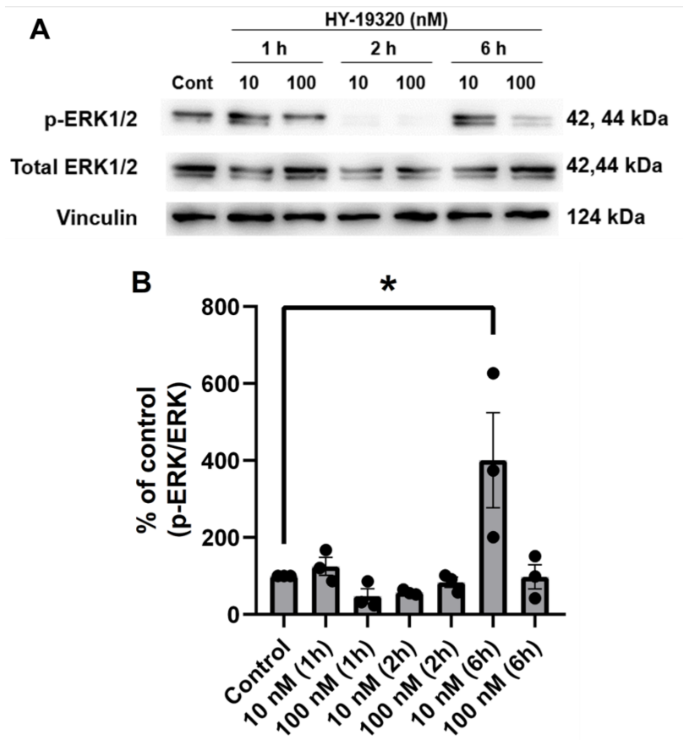


Figure 3 The immunoblot of phosphorylation of ERK1/2 in hORX2R/SH-SY5Y cells (A). Cells were treated with 10 and 100 nM HY-19320 for 1, 2, and 6 h. Protein bands were quantified by densitometry (B). Results are presented as the ratio of p-ERK1/2 to total ERK1/2. Data are expressed as means ± S.E.M. of three independent experiments and analyzed using one-way ANOVA. (*P < 0.05, compared to the control-untreated cells)

The effect of DORA on orexin-2 receptor activation in hORX2R/SH-SY5Y cells

The results of the Western blot analysis showed that 10 nM HY-19320 significantly increased the phosphorylation of ERK1/2 to 154 ± 7.67% of control-untreated cells. Lemborexant at 20 nM and 40 nM significantly decreased the phosphorylation of ERK1/2 to 84.36 ± 9.20% and 59.71 ± 15.46% respectively, compared with cells treated with 10 nM HY-19320. However, asarinin tended to decrease the phosphorylation of ERK1/2 when compared with cells treated with 10 nM HY-19320 **(Figure 4)**.

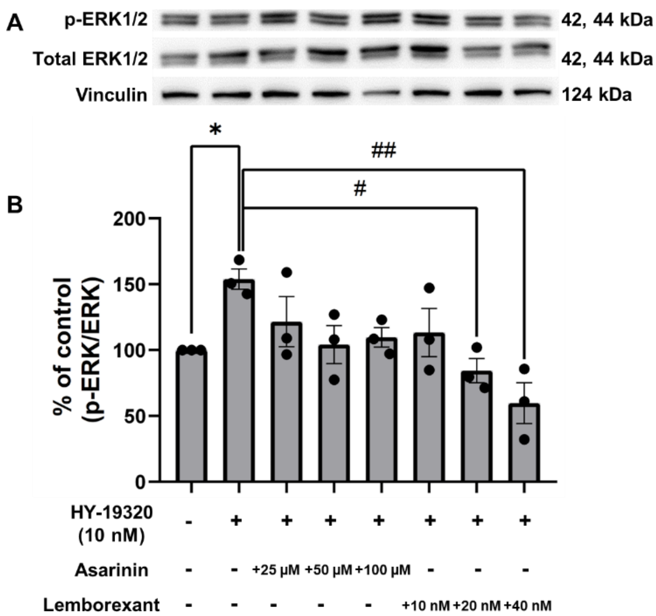


Figure 4 The effects of asarinin and lemborexant on orexin-2 receptor activation were evaluated by measuring phosphorylation of ERK1/2 in hORX2R/SH-SY5Y cells (A). Cells were treated with asarinin (25, 50, and 100 µM) or lemborexant (10, 20, and 40 nM) in combination with 10 nM HY-19320 in FBS-free medium for 6 h. (B) Results are presented as the ratio of p-ERK1/2 to total ERK1/2. Data are expressed as means ± S.E.M. of three independent experiments and analyzed using one-way ANOVA. (*P < 0.05 compared to the control-untreated cells and #P < 0.05, ##P < 0.01, compared to the HY-19320-treated cells).

Discussion and Conclusion

In this study, we investigated the role of asarinin on orexin receptors (ORXR) in a neuronal in vitro model. The results from *in silico* drug virtual screening demonstrated a potential candidate for DORAs of asarinin, with the binding free energy to ORX2R of -11.7 kcal/mol, compared to -9.8 kcal/mol for lemborexant (unpublished data). To explore the mechanism of action of asarinin on ORXR, we established ORX2R-overexpressing SH-SY5Y cells as an *in vitro* model. Immunofluorescence staining and Western blot analysis confirmed significantly higher expression of human ORX2R in these cells compared with wild-type SH-SY5Y cells. This model enabled us to investigate the activation of the orexin system via ORX2R-mediated downstream signaling cascades. Asarinin has been reported to exhibit a range of pharmacological activities, including analgesic, antiviral, anti-tuberculous, and anti-tumor effects. Previous studies have shown that asarinin alleviates gastric precancerous lesions (GPL) by inhibiting the methylnitronitrosoguanidine-induced malignant transformation of human gastric epithelial MC cells. This effect is achieved through the suppression of cell proliferation, elevation of intracellular reactive oxygen species (ROS) levels, and induction of apoptosis (Hou et al, 2020). Therefore, in the present study, the non-cytotoxic effects of asarinin were investigated. The MTT assay demonstrated that asarinin at 25-100 μM showed no cytotoxicity in hORX2R/SH-SY5Y cells.

In the present study, we demonstrated that asarinin tends to attenuate orexin-2 receptor (ORX2R) activation, represented by phosphorylation of ERK1/2 in hORX2R/SH-SY5Y cells, which suggests a partial orexin receptor antagonist. Orexin is an excitatory neuropeptide that plays a critical role in regulating the sleep–wake cycle by activating wake-promoting monoaminergic and cholinergic neurons in the hypothalamus and brainstem to sustain wakefulness during the active phase (Mogavero et al, 2023). Upon binding to their receptors, orexins activate a GPCR-mediated signaling cascade involving at least three G-protein subtypes ($G_{q/11}$, $G_{i/o}$, and G_s) or interacting with other signaling proteins such as β-arrestin. These pathways regulate various effectors, including phospholipases, ion channels, and protein kinases, which subsequently activate diverse downstream signaling cascades. One key pathway is the activation of ERK1/2 via $G_{q/11}$-coupled receptors, which stimulate phospholipase C (PLC) and protein kinase C (PKC), ultimately leading to ERK1/2 phosphorylation (Xu et al, 2013). Both orexin-1 and orexin-2 receptors contribute to sleep initiation and maintenance, with preclinical studies highlighting reciprocal interactions between them. ORX2R, in particular, has been implicated in the suppression of non-rapid eye movement (NREM) sleep, whereas both receptors contribute comparably to the suppression of rapid eye movement (REM) sleep (Carpi et al, 2024). Dysregulation of the orexin system has been proposed as a mechanism underlying the hyperarousal observed in insomnia, potentially linked to abnormally elevated orexin levels during the dark phase (Muehlan et al, 2023). Current pharmacotherapies include dual orexin receptor antagonists (DORAs), which block both ORX1R and ORX2R, thereby increasing total sleep time primarily through the promotion of REM sleep, while exerting minimal effects on NREM sleep (Gotfried et al, 2024).

Furthermore, ORX2R activation through the G_q/PLC/PKC pathway induces a PKC-mediated calcium influx across the plasma membrane via L-type calcium channels and mobilizes calcium from intracellular stores through a PLC-dependent mechanism. The resulting increase in intracellular calcium levels sustains neuronal excitation (Ohno & Sakurai, 2008). Future studies should investigate the effects of asarinin on additional ORX2R downstream pathways, such as CREB signaling and intracellular Ca^{2+} dynamics, to further evaluate its potential to inhibit orexin receptors. Additionally, the role of asarinin on sleep and pharmacological effects needs to be elucidated in an animal model.

Acknowledgements

This research project has been funded by Mahidol University (Fundamental Fund (FF-168/2567): fiscal year 2024 by National Science Research and Innovation Fund-NSRF).

References

Carpi, M., Palagini, L., Fernandes, M., Calvello, C., Geoffroy, P. A., Miniati, M., Pini, S., Gemignani, A., Mercuri, N. B., & Liguori, C. (2024). Clinical usefulness of dual orexin receptor antagonism beyond insomnia: Neurological and psychiatric comorbidities. *Neuropharmacology*, *245*, 109815. https://doi.org/10.1016/j.neuropharm.2023.109815

Gotfried, M. H., Auerbach, S. H., Dang-Vu, T. T., Mishima, K., Kumar, D., Moline, M., & Malhotra, M. (2024). Efficacy and safety of insomnia treatment with lemborexant in older adults: Analyses from three clinical trials. *Drugs & Aging*, *41*(9), 741–752. https://doi.org/10.1007/s40266-024-01135-8

Hou, Y., Hu, T., Wei, D., Gao, J., Che, D., Wang, X., Wang, C., & He, H. (2020). (-)-Asarinin inhibits mast cells activation as a Src family kinase inhibitor. *The International Journal of Biochemistry & Cell Biology*, *121*, 105701.

https://doi.org/10.1016/j.biocel.2020.105701

Mogavero, M. P., Silvani, A., Lanza, G., DelRosso, L. M., Ferini-Strambi, L., & Ferri, R. (2023). Targeting Orexin Receptors for the Treatment of Insomnia: From Physiological Mechanisms to Current Clinical Evidence and Recommendations. *Nature and Science of Sleep*, *Volume 15*, 17–38. https://doi.org/10.2147/NSS.S201994

Muehlan, C., Roch, C., Vaillant, C., & Dingemanse, J. (2023). The orexin story and orexin receptor antagonists for the treatment of insomnia. *Journal of Sleep Research*, *32*(6), e13902. https://doi.org/10.1111/jsr.13902

Naha, S., Sivaraman, M., & Sahota, P. (2024). Insomnia: A Current Review. *Missouri medicine*, *121*(1), 44–51.

Ohno, K., & Sakurai, T. (2008). Orexin neuronal circuitry: Role in the regulation of sleep and wakefulness. *Frontiers in Neuroendocrinology*, *29*(1), 70–87. https://doi.org/10.1016/j.yfrne.2007.08.001

Pakjira, W., & Sorarat, T. (2024). การใช้ยา Lemborexant ในการรักษาภาวะนอนไม่หลับ ในผู้ใหญ่. *Thai Journal of Neurology, Volume 3, NO.4.* https://thaijoneuro.com/journal/7f5746704a1f48fdb53cb208b5eb3afd

Park, H. J., Lee, K. S., Zhao, T. T., Lee, K. E., & Lee, M. K. (2017). Effects of asarinin on dopamine biosynthesis and 6-hydroxydopamine-induced cytotoxicity in PC12 cells. *Archives of Pharmacal Research*, *40*(5), 631–639. https://doi.org/10.1007/s12272-017-0908-z

Wang, L., Gu, Y., Zhang, J., & Gong, L. (2019). Effects of Sleep Deprivation (SD) on Rats via ERK1/2 Signaling Pathway. *Medical Science Monitor*, *25*, 2886–2895. https://doi.org/10.12659/MSM.913839

Xu, T.-R., Yang, Y., Ward, R., Gao, L., & Liu, Y. (2013). Orexin receptors: Multi-functional therapeutic targets for sleeping disorders, eating disorders, drug addiction, cancers and other physiological disorders. *Cellular Signalling*, *25*(12), 2413–2423. https://doi.org/10.1016/j.cellsig.2013.07.025

Zhang, S., Zhou, X., Ou, M., Fu, X., Lin, Q., Tao, X., Wang, Z., Liu, A., Li, G., Xu, Y., & Zhang, G. (2023). Berbamine promotes macrophage autophagy to clear *Mycobacterium tuberculosis* by regulating the ROS/Ca^{2+} axis. *mBio*, e00272-23. https://doi.org/10.1128/mbio.00272-23

P-072:

KV1.3 CHANNEL INHIBITION PRESERVES NEURONAL INTEGRITY AND PROMOTES FUNCTIONAL RECOVERY AFTER INTRACEREBRAL HEMORRHAGE

Wachirapong Saleeon[1], Namphung Thongta[1], Anchalee Vattarakorn[1], Yingrak boondam[2], Wattana Watanapa[1], Sompol Tapechum[1], Narawut Pakaprot[1*]

[1]Department of Physiology, Faculty of Medicine, Siriraj Hospital, Mahidol University, Bangkok, Thailand, 10700.
[2]Department of Physiology, Faculty of Pharmacy, Mahidol University, Bangkok, Thailand, 10400
**Corresponding author. Email: narawut.pak@mahidol.ac.th*

Abstract

Background: Spontaneous intracerebral hemorrhage (sICH) is a severe subtype of stroke associated with high mortality, long-term disability, and limited therapeutic strategies. Hematoma formation leads to primary brain injury by mechanical compression and secondary injury through excitotoxicity, oxidative stress, neuroinflammation, and ischemia. Microglia have been shown to play a central role in this process, with the voltage-gated potassium channel Kv1.3 highly expressed in pro-inflammatory microglia.

Aim(s): This study aimed to evaluate the therapeutic potential of PAP-1, a selective Kv1.3 inhibitor, in a collagenase-induced ICH mouse model

Methods: Adult male mice aged 7–8 weeks (n = 3 groups) were randomly assigned to sham, vehicle, or PAP-1 treatment groups. Following ICH induction, PAP-1 (40 mg/kg) or vehicle was intraperitoneally administered daily for up to 7 days. Pathological evaluations included survival rate, body weight, hematoma volume (H&E staining), and neuronal survival (NeuN immunostaining). The Kv1.3 expression and microglial phenotypes ($CD16/32^+$ for M1, $CD206^+$ for M2) were assessed. Functional recovery was evaluated using the modified neurological severity score (mNSS), cylinder test, corner turn test, and open field test.

Results: PAP-1 significantly improved 7-day survival ($p < 0.05$) and reduced weight loss compared with the vehicle control animals. The hematoma volume decreased, and preservation of NeuN-positive neurons was observed in the perihematomal region of PAP-1 animals. PAP-1 treatment suppressed Kv1.3 expression, decreased M1-like microglia, increased M2-like microglia, and elevated the M2/M1 ratio ($p < 0.05$), suggesting a shift toward a reparative phenotype. Functionally, PAP-1–treated mice showed improved neurological outcomes, including lower mNSS scores, greater forelimb use symmetry, reduced turning bias, and enhanced locomotor activity.

Conclusion: Kv1.3 inhibition with PAP-1 confers neuroprotection after ICH by limiting microglia-mediated neuroinflammation, preserving neuronal integrity, and promoting functional recovery. These findings highlight Kv1.3 as a promising therapeutic target for hemorrhagic stroke.

Keywords: Intracerebral hemorrhage, Kv1.3, microglia, PAP-1, neuroinflammation

----

P-073:

EFFECT OF HOME-BASED, LOW-INTENSITY EXERGAMING ON COGNITIVE FUNCTION IN OLDER ADULTS WITH MILD COGNITIVE IMPAIRMENT

Sirintip Kumfu[1], Sirinun Boripantakul[1], Piangkwan Sa-nguanmoo[1], Siriporn C Chattipakorn[2,3], Somporn Sungkarat[1]*

[1]*Department of Physical Therapy, Faculty of Associated Medical Sciences, Chiang Mai University, Chiang Mai, Thailand, 50200*
[2]*Neurophysiology Unit, Cardiac Electrophysiology Research and Training Center, Faculty of Medicine, Chiang Mai University, Chiang Mai, Thailand, 50200*
[3]*Department of Oral Biology and Diagnostic Sciences, Faculty of Dentistry, Chiang Mai University, Chiang Mai, Thailand, 50200*
**Corresponding author. Email: somporn.sungkarat@cmu.ac.th*

Abstract

Background: Older adults with mild cognitive impairment (MCI) experience greater cognitive decline than those without MCI. Although moderate-to-vigorous exercise benefits cognition, many older adults cannot engage at this intensity due to health conditions. Low-intensity exercise may offer a practical alternative; however, evidence for its cognitive effects is limited. Moreover, the effects of low-intensity exercise combined with cognitive training through exergames (low-intensity exergames) have not yet been investigated in this population.

Aim: To examine the effect of a home-based, low-intensity exergame on cognitive and physical functions in older adults with MCI.

Methods: Participants aged ≥60 years with MCI, based on DSM-5 criteria, were randomized to an exercise or control group. Sixty-four older adults with MCI were randomized to an exercise group (n=32, 68.3±4.6 years) or a control group (n=32, 68.1±5.4 years). The exercise group performed a home-based, low-intensity exergame combining cognitive (memory, attention, executive function) and physical training. Exercise sessions lasted 50 minutes, three times per week, for 12 weeks. The control group received no intervention. Cognitive function was assessed using the Alzheimer's Disease Assessment Scale–Cognitive Subscale, Montreal Cognitive Assessment, Trail Making Test, and Logical Memory Test, evaluating global cognition, attention and executive function, and memory, respectively. Physical function was assessed with the Timed Up and Go and 30-Second Sit-to-Stand test.

Results: After 12 weeks, the exercise group demonstrated significantly greater improvements in executive function and global cognitive function, as revealed by ANCOVA, compared to controls ($p<0.05$). However, no significant differences between groups were observed in physical performance.

Conclusion: Home-based, low-intensity exergames offer a promising approach to support cognitive health and potentially delay cognitive decline in older adults with MCI. Cognitive improvements may result from the simultaneous engagement of physical and cognitive processes, a key feature of exergame training proposed to promote neuroplasticity. Further investigations are still needed.

Keywords: Low-intensity; Exergame; MCI; Home-based exercise; Cognition

---

P-074:

ENGINEERED EXOSOMES FROM HUMAN EXFOLIATED DECIDUOUS TEETH (SHED) EXPRESSING RVG PEPTIDE ATTENUATE NEURONAL INJURY AND PROMOTE FUNCTIONAL RECOVERY AFTER ISCHEMIC STROKE IN RATS

Sukonthar Ngampramuan[1]*, Anyapat Atipimonpat[2], Hathaitip Sritanaudomchai[3], Pakpoom Subsoontorn[2], Waracharee Srifa[2], Pornprom Surakul[4], Chattraporn Nantawanichakorn[1], Kovit Pattanapanyasat[5]

[1] Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom 73170, Thailand
[2] Department of Biochemistry, Faculty of Medical Science, Naresuan University 65000, Thailand
[3] Department of Oral Biology, Faculty of Dentistry, Mahidol University, Bangkok 10400, Thailand
[4] Faculty of Allied Health Sciences, Burapha University, Chonburi 20131, Thailand.
[5] Siriraj Center of Research Excellence for Microparticle and Exosome in Diseases, Research
**Corresponding author. Email: sukonthar.nga@mahidol.ac.th*

Abstract

Background: Exosomes derived from mesenchymal stem cells offer a promising cell-free therapeutic strategy for neurological disorders. Among them, stem cells from human exfoliated deciduous teeth (SHED) represent an accessible and highly transducible source. Engineering exosomes with neuronal-targeting ligands may further enhance their therapeutic potential in ischemic stroke.

Aim(s): To develop engineered SHED-derived exosomes expressing the rabies virus glycoprotein (RVG) peptide for neuronal targeting and to evaluate their therapeutic effects in a rat model of ischemic stroke.

Methods: Plasmid vectors encoding RVG fused to Lamp2b (pPSNU134) with reporter and selection markers were constructed, and SHED were transduced with lentiviral vectors to generate producer cell banks. Exosomes from engineered (SHED-RVG) and non-engineered (SHED-WT) SHED were isolated by differential centrifugation/ultrafiltration and characterized under MISEV2018 guidelines using TEM, NTA, and Western blotting. Adult male Sprague–Dawley rats underwent permanent bilateral common carotid artery occlusion (BCCAO) and were randomized into four groups: sham, BCCAO, BCCAO + SHED-WT exosomes, and BCCAO + SHED-RVG exosomes (100 µg, IV; n = 7 each). Neurological outcomes were assessed on days 1, 7, and 14 using the mNSS and beam balance, followed by histopathological examination of cortical and hippocampal regions.

Results: Both SHED-WT and SHED-RVG exosomes exhibited typical cup-shaped morphology (50–150 nm). BCCAO rats displayed severe neurological deficits and neuronal injury. Treatment with SHED exosomes improved neurological recovery and reduced neuronal damage compared with untreated BCCAO rats. SHED- RVG exosomes provided superior restoration of proprioceptive and reflex functions at day 14 and showed a trend toward enhanced neuroprotection relative to SHED-WT exosomes.

Conclusion: Exosomes derived from SHED, particularly those engineered with RVG peptide for neuronal targeting, effectively attenuate neuronal injury and enhance functional recovery after ischemic stroke. These findings support RVG-SHED exosomes as a promising next-generation therapeutic platform for stroke.

Keywords: Exosomes; SHED; RVG peptide; Ischemic stroke; Neuroprotection

---

P-075:

TIME-DEPENDENT ANALYSIS OF PAIN-ASSOCIATED PROTEIN LOCALIZATION IN THE TIDEMARK OF CFA-INDUCED TEMPOROMANDIBULAR OSTEOARTHRITIS IN MICE

Lattanawan Inchaisri[1], Aree Wanasuntronwong[2*], and Onrawee Khongsombat[1*]

[1]Department of Physiology, Faculty of Medical Science, Naresuan University, Phitsanulok 65000
[2]Department of Oral Biology, Faculty of Dentistry, Mahidol University, Bangkok 10400
**Corresponding author. Email: aree.wan@mahidol.ac.th, onraweek@nu.ac.th*

Abstract

Background: Temporomandibular osteoarthritis (TMJ-OA) is a progressive joint disorder caused by unresolved tissue damage, leading to deformity and pain. Mechanical overload is a key trigger, especially at the tide mark, which is prone to degeneration. The mechanisms underlying TMJ-OA pain remain unclear, and further study may aid in developing effective treatments.

Aim(s): This study aims to examine the expression profiles and functional roles of pain-associated proteins such as nerve growth factor (NGF), vascular endothelial growth factor (VEGF), and alpha1 adrenergic receptor (α1AR) throughout the development of TMJ-OA.

Methods: Adult male mice were divided into three groups: sham, complete Freund's adjuvant (CFA), and CFA + ibuprofen 140 mg/kg (CFA+IBU), each with four subgroups on days 7, 14, 21, and 28. TMJ-OA was induced by CFA injection into the right TMJ. The CFA+IBU group received ibuprofen orally once daily. Tissues were collected on days 7, 14, 21, and 28 post-injection. Immunofluorescence staining of right TMJs was performed to evaluate NGF, VEGF, and α1AR expression in chondrocytes at the boundary between uncalcified and calcified cartilage (tide mark), a critical region for joint degeneration. Data were analyzed by signal intensity.

Results: CFA significantly increased α1AR on days 7, 14, 21, and 28, VEGF on days 14 and 21, and NGF on day 21 compared with the sham group. IBU reduced the effects of CFA, resulting in decreased VEGF on day 14, NGF on day 21, and α1AR on days 21 and 28 compared with the CFA group. Over time, CFA increased NGF, VEGF, and α1AR on days 14, 21, and 28 compared with day 7, while IBU reduced the effects of CFA, resulting in decreased NGF on day 28 compared with day 14.

Conclusion: CFA increased NGF, VEGF, and α1AR over time, reflecting TMJ-OA progression, whereas IBU suppressed their expression, indicating anti-inflammatory effects.

Keywords: Temporomandibular joint osteoarthritis, Complete Freund's adjuvant, Nerve growth factor, Vascular endothelial growth factor, α1-adrenagic receptor

---

P-076:

FENDOSAL ATTENUATES OXYGEN-GLUCOSE DEPRIVATION/REPERFUSION-INDUCED UPREGULATION OF RHEB AND MTOR IN HIPPOCAMPAL NEURONAL CELLS

Kornkanok Promthep[1], Chonnicha Subkod[1], Theptharin Charuraksa[2], Pathidta Chodewaratham[1], Patlada Tangweerasing[1], Tanya Prasertporn[1], Jiraporn Panmanee[1], Narisorn Kitiyanant[3], Surapon Piboonpocanun[3], Sujira Mukda[1]*

[1]*Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Salaya, Nakhon Pathom 73170 Thailand*
[2]*Office of Research and Innovation Affair, Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom 73170 Thailand*
[3]*Center for Advanced Therapeutics (CAT), Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom 73170 Thailand*
*Corresponding author. Email: sujira.muk@mahidol.ac.th

Abstract

Background: Ischemic stroke remains a leading cause of mortality and neurological disability worldwide. The Ras homolog enriched in brain (Rheb)-mechanistic target of rapamycin complex 1 (mTORC1) signaling pathway plays a critical role in regulating autophagy and cellular metabolism in neurons. Dysregulation of this pathway contributes to neuronal injury following ischemic insults. Fendosal, a non-steroidal anti-inflammatory drug with potent anti-inflammatory properties, may offer neuroprotection through mechanisms beyond inflammation modulation.

Aim(s): This study investigated the effects of fendosal on Rheb and mTOR protein expression in hippocampal neuronal cells subjected to oxygen-glucose deprivation followed by reperfusion (OGD/R), an *in vitro* model of ischemic stroke.

Methods: Mouse HT22 hippocampal neuronal cells were exposed to 6 hours of oxygen-glucose deprivation followed by 18 hours of reoxygenation with or without fendosal treatment. Protein expression levels of Rheb and mTOR were determined by Western blot analysis. Data were analyzed using one-way ANOVA followed by Tukey's *post hoc* test.

Results: OGD/R significantly increased both Rheb and mTOR protein expression compared to normoxic control cells ($p < 0.05$), indicating activation of the Rheb-mTORC1 signaling pathway in response to hypoxic-ischemic stress. Fendosal treatment significantly attenuated the OGD/R-induced upregulation of both Rheb and mTOR proteins ($p < 0.05$), suggesting modulation of the Rheb-mTORC1 axis under ischemic conditions.

Conclusion: Fendosal attenuates OGD/R-induced upregulation of Rheb and mTOR protein expression in hippocampal neurons, indicating that this anti-inflammatory drug modulates the Rheb-mTORC1 signaling pathway under ischemic stress. These findings reveal a novel mechanism by which fendosal may exert neuroprotective effects through regulation of autophagy-related pathways, warranting further investigation in *in vivo* stroke models.

Keywords: Fendosal, Rheb, mTORC1, Autophagy, Ischemic stroke

Background

Ischemic stroke represents a major public health burden, ranking among the leading cause of mortality and long-term neurological disability worldwide. It results from the obstruction of cerebral blood vessels, which causes a reduction in blood flow and deprives brain tissue of oxygen and essential nutrients, particularly glucose. This deprivation initiates a cascade of detrimental cellular events, including metabolic failure, energy depletion, oxidative stress, neuroinflammation, and activation of multiple programmed cell death pathways, which collectively contribute to irreversible neuronal injury and functional deficits (Mao et al., 2022; Qin et al., 2022). The heightened vulnerability of neurons to ischemic stress underscores the urgent need for understanding the underlying cellular mechanisms and identifying novel neuroprotective strategies.

Autophagy is a highly conserved intracellular process essential for maintaining cellular homeostasis through the lysosomal degradation and recycling of damaged organelles, misfolded proteins, and macromolecular aggregates. This process plays a pivotal role in neurons, which are particularly vulnerable to ischemic injury due to their high metabolic demands and limited regenerative capacity. Dysregulation of autophagy has been implicated in various neurological diseases, including ischemic stroke, where it may act as a double-edged sword – promoting cell survival under mild stress but contributing to cell death when excessively or insufficiently activated (Lu et al., 2022; Wang et al., 2018). This dual nature underscores the complexity of targeting autophagy for therapeutic intervention.

Central to the regulation of autophagy is the mechanistic target of rapamycin complex 1 (mTORC1), a serine/threonine kinase complex that integrates signals from nutrients, growth factors, and cellular energy status to control cell growth, metabolism, and autophagic flux (Wang et al., 2022). mTORC1 activity is tightly regulated by upstream signals, notably the Ras homolog enriched in brain (Rheb) protein, a small GTPase that directly binds to and activates mTORC1 under nutrient-sufficient conditions. In the central nervous system, Rheb is fundamental in promoting neuronal growth, synaptic plasticity, axonal regeneration, and moderation of autophagy in response to metabolic perturbations (Panwar et al., 2023). Dysregulation of the Rheb-mTORC1 signaling has been implicated in numerous pathological conditions, including ischemic stroke, where disrupted autophagy and metabolic homeostasis contribute to the progression of neuronal injury (Ajoolabady et al., 2021; Villa-Gonzalez et al., 2022). Given its central role in orchestrating neuronal homeostasis and ischemic pathophysiology, the Rheb-mTORC1 axis represents an attractive therapeutic target for stroke intervention.

Pharmacological modulation of the Rheb-mTORC1 pathway offers potential for restoring the delicate balance between autophagy and cell survival, thereby conferring neuroprotection against ischemic damage. Conventional mTOR inhibitors such as rapamycin effectively suppress mTORC1, but have limitations due to off-target effects and adverse consequences such as insulin resistance arising from mTORC2 inhibition (Harrison et al., 2009; Lamming et al., 2012). Thus, there is a growing interest in discovering selective modulators of upstream regulators like Rheb that can fine-tune mTORC1 activity while minimizing off-target complications (Ajoolabady et al., 2021; Szwed et al., 2021).

The bidirectional relationship between autophagy and inflammation is well established and particularly relevant to stroke pathophysiology. Autophagy regulates inflammatory responses through multiple mechanisms, including modulation of immune cell development and function, clearance of damaged cellular constituents, degradation of inflammasome components, and control of pro-inflammatory cytokine secretion such as interleukin-1β (IL-1β) and interleukin-18 (IL-18) (Qian et al., 2017). Functional autophagy constrains excessive inflammation by eliminating potential activators of innate immune signaling and attenuating nuclear factor-κB (NF-κB) pathway activation. Conversely, impaired or dysregulated autophagy amplifies inflammatory cascades, leading to heightened production of inflammatory mediators and exacerbation of tissue injury. Furthermore, inflammatory signals reciprocally influence autophagic flux, establishing dynamic feedback loops that are essential for cellular adaptation to stress. Disruption of this autophagy-inflammation interplay contributes significantly to the pathogenesis of neurodegenerative and inflammatory disorders, including ischemic stroke, where the crosstalk between these processes profoundly influences neuronal fate (Netea-Maier et al., 2016; Wang et al., 2018).

Fendosal, also known as HP129, is a non-narcotic analgesic agent belonging to the non-steroidal anti-inflammatory drug (NSAID) class, with demonstrated potent anti-inflammatory and analgesic properties with a favorable safety profile (National Center for Biotechnology Information, 2025). Preclinical investigations have demonstrated that fendosal exhibits anti-inflammatory efficacy 1.4-fold superior to aspirin in acute inflammation models and remarkable 6.9- to 9.5-fold greater activity compared to aspirin in chronic inflammation paradigms, such as adjuvant-induced polyarthritis in rodents (Lassman et al., 1978). Notably, fendosal demonstrates prolonged analgesic duration and substantially reduced gastrointestinal irritation relative to conventional NSAIDs including aspirin (Lassman et al., 1978), making it an attractive candidate for both acute and chronic inflammatory conditions.

Although fendosal has not been directly studied for effects on autophagy or mTOR signaling, several lines of evidence support exploration of this mechanism. NSAIDs including aspirin and celecoxib modulate autophagic processes and mTORC1 activity (Chen et al., 2022; Fu et al., 2020). Additionally, established crosstalk between NF-κB and mTORC1 suggests that potent anti-inflammatory agents may indirectly influence autophagy (Li et al., 2019). Based on these considerations, we hypothesized that fendosal may modulate the Rheb-mTORC1 signaling pathway under ischemic stress conditions. The present study was designed to investigate whether fendosal influences Rheb and mTOR protein expression in hippocampal neuronal cells subjected to oxygen-glucose deprivation followed by reperfusion (OGD/R), an established *in vitro* model of ischemic stroke. Characterization of fendosal's effects on this pathway may provide insights into potential mechanisms beyond its established anti-inflammatory properties and inform future investigations into its neuroprotective potential following cerebral ischemia.

Materials and Methods

Cell culture

The mouse (*Mus musculus*) HT22 hippocampal neuronal cell line (#SCC129) was procured from Sigma-Aldrich (Merck KGaA, Darmstadt, Germany). Cells were maintained in Dulbecco's Modified Eagle's Medium (DMEM; Thermo Fisher Scientific, Waltham, MA, USA) supplemented with 10% (v/v) fetal bovine serum (FBS) and 1% (v/v) penicillin-streptomycin antibiotic solution. Cultures were maintained in a humidified incubator at 37 °C with 5% CO_2 atmosphere and passaged upon reaching 80-90% confluency using standard trypsinization protocols.

Oxygen-glucose deprivation/Reperfusion (OGD/R)

To mimic the hypoxic-ischemic condition characteristic of severe ischemic stroke *in vitro*, oxygen-glucose deprivation (OGD) and re-oxygenation (OGDR) paradigm were employed. HT22 cells were seeded in 60-mm petri dishes until reached 80% confluence. Prior to OGD induction, the culture medium was aspirated and cells were washed twice with pre-warmed (37 °C) phosphate-buffered saline (PBS) to remove residual glucose. OGD was initiated by replacing the medium with pre-equilibrated glucose-free DMEM and transferring cultures to a modular hypoxic chamber (or hypoxia incubator) maintained at 37 °C with a gas mixture of 1% O_2, 5% CO_2, and 94% N_2 for 6 hours. Parallel normoxic control cultures were maintained in complete DMEM under standard culture conditions (37 °C, 95% air/5% CO_2). Following the OGD exposure period, reperfusion was simulated by replacing the glucose-free medium with complete culture medium and returning the cells to normoxic conditions (37 °C, 95% air/5% CO_2) for the 18 hours. Cells were harvested at predetermined time points following OGD exposure or OGD/R for subsequent biochemical analyses.

Western blotting

Cell pellets, as reported previously (Mukda et al., 2019), were denatured in loading buffer (125 mM Tris-HCl pH 6.8, 4% SDS, 20% glycerol, 4% β-mercaptoethanol, and 0.02% bromophenol blue) at 95 °C for 5 minutes. The protein concentration was determined by using Pierce™ BCA protein assay kit (Thermo Fisher Scientific, Waltham, MA, USA) according to the manufacturer's protocol. An equal amount of protein sample was denatured in loading buffer at 95℃ for 5 min and loaded onto 8% or 15% SDS-PAGE gels and electrophoretically transferred to a polyvinylidene difluoride (PVDF) membrane. The Rheb and mTOR antibodies were used to examine each target protein level in cell lysate. Species-specific horseradish peroxidase (HRP)-conjugated secondary antibodies (CST, MA, USA) were applied to recognize the primary antibodies and visualize them through Immobilon Forte Western HRP substrate (MerckMillipore, Burlington, MA, USA) and exposed to medical X-ray film (Fujifilm, Minato City, Tokyo, Japan). The immunoblot bands were quantified using ImageJ Software (version 1.53a, National Institutes of Health, Bethesda, MD, USA). Target protein expression levels were normalized to the corresponding β-actin loading control and expressed as fold change relative to control groups.

Statistical analysis

All experiments were performed in four independent biological replicates. Data are presented as mean ± standard error of the mean (SEM). Statistical significance was assessed using one-way analysis of variance (ANOVA) followed by Tukey's post hoc multiple comparison test. The significance was considered when the p-value was less than 0.05.

Results

Fendosal Attenuates OGD/R-Induced Upregulation of Rheb and mTORC1 in HT22 Hippocampal Neurons

To investigate the regulatory role of fendosal on the Rheb-mTORC1 signaling axis under ischemic conditions, the protein expression levels of Rheb and mTOR in HT22 hippocampal neuronal cells subjected to OGD followed by OGD/R were examined. Cells were exposed to 6 hours of OGD followed by 18 hours of reoxygenation in the presence or absence of 1 nM or 5 nM fendosal treatment.

Western blot analysis revealed that OGD/R significantly increased Rheb protein expression compared to normoxic control cells (Figure 1A; $p < 0.05$), indicating activation of this upstream regulator of mTORC1 in response to hypoxic-ischemic stress. Notably, both 1 nM and 5 nM fendosal treatment significantly attenuated the OGD/R-induced elevation in Rheb expression (Figure 1A; $p < 0.05$ and $p < 0.05$, respectively), suggesting that fendosal modulates upstream components of the mTORC1 pathway.

Similarly, mTOR protein expression was markedly upregulated following OGD/R compared to control conditions (Figure 1B; $p < 0.05$). This elevation in total mTOR protein is consistent with cellular stress responses aimed at regulating autophagy and metabolic homeostasis. Treatment with 1 nM fendosal significantly suppressed the OGD/R-induced increase in mTOR expression (Figure 1B; $p < 0.05$). These findings indicate that fendosal exerts inhibitory effects on both Rheb and mTOR protein expression under ischemic conditions, potentially modulating mTORC1 pathway activity.

The observation that fendosal attenuates the upregulation of both Rheb and mTOR following OGD/R suggests that this NSAID may influence autophagy regulation and cellular stress responses through modulation of the Rheb-mTORC1 signaling axis. These results support the hypothesis that fendosal's neuroprotective effects may extend beyond its established anti-inflammatory properties to include regulation of key metabolic and survival pathways in neurons subjected to ischemic injury.

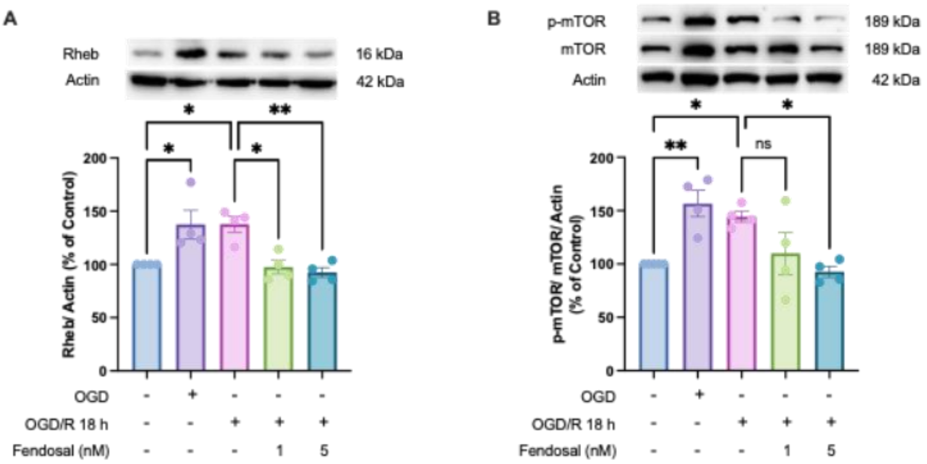


Figure 1. Fendosal attenuates OGD/R-induced upregulation of Ras homolog enriched in brain (Rheb) and mechanistic target of rapamycin (mTOR) protein expression in HT22 hippocampal neuronal cells.

HT22 cells were subjected to 6 hours of oxygen-glucose deprivation (OGD) followed by 18 hours of reoxygenation (OGD/R) with or without 1 nM and 5 nM fendosal. Western blot analysis showing protein expression of (A) Rheb and (B) mTOR across experimental groups. Densitometric quantification of Rheb protein expression normalized to β-actin. Data are presented as mean ± SEM from four independent experiments ($n = 4$). Statistical significance was determined by one-way ANOVA followed by Tukey's post hoc test. *$p < 0.05$, **$p < 0.01$ compared to indicated groups.

Discussion and Conclusion

The present study demonstrates that fendosal attenuates the upregulation of Rheb and mTOR protein expression in hippocampal neuronal cells subjected to OGD followed by OGD/R. Our Western blot analysis revealed that OGD/R significantly increased both

Rheb and mTOR protein levels, indicating potential activation of the Rheb-mTORC1 signaling pathway in response to hypoxic-ischemic stress. Importantly, fendosal treatment significantly suppressed this OGD/R-induced upregulation of both proteins, suggesting that this non-steroidal anti-inflammatory drug modulates the Rheb-mTORC1 axis under ischemic conditions. These findings provide preliminary evidence that fendosal may influence autophagy-related signaling pathways beyond its established anti-inflammatory mechanisms, though functional validation is required to confirm these implications.

The Rheb-mTORC1 pathway serves as a critical regulator of autophagy and cellular metabolism in neurons (Panwar et al., 2023; Wang et al., 2022). Under normal conditions, Rheb directly activates mTORC1, which suppresses autophagy and promotes cell growth. However, during ischemic insults, dysregulation of mTORC1 activity may contribute to impaired autophagy, and neuronal injury (Lu et al., 2022; Wang et al., 2018). Our observation that OGD/R increased Rheb and mTOR expression is consistent with previous reports of alterations in mTORC1-related signaling following ischemic stress (Lu et al., 2022; Wang et al., 2018). The ability of fendosal to attenuate this upregulation suggests a potential mechanism by which the compound may influence mTORC1 pathway activity. However, whether these changes in protein expression translate to functional modulation of autophagic flux or alterations in the clearance of damaged cellular components remains to be determined through direct assessment of autophagy markers and functional assays.

The potential therapeutic implications of these findings are noteworthy. Traditional mTOR inhibitors such as rapamycin have shown neuroprotective effects but are limited by off-target effects and adverse consequences from non-selective inhibition of both mTORC1 and mTORC2 complexes (Harrison et al., 2009; Lamming et al., 2012). Compounds that modulate upstream regulators like Rheb may offer alternative approaches for targeting mTORC1 activity (Szwed et al., 2021). Furthermore, given the established bidirectional relationship between autophagy and inflammation (Netea-Maier et al., 2016; Qian et al., 2017), fendosal demonstrated anti-inflammatory properties (Lassman et al., 1978) combined with its observed effects on Rheb-mTORC1 expression suggest multiple potential mechanisms that could be relevant to stroke pathophysiology. However, the functional consequences of these molecular changes and their impact on neuronal outcomes require experimental validation.

However, several limits should be taken into account. Our study examined total protein expression of Rheb and mTOR but did not directly assess mTORC1 kinase activity through phosphorylation status or downstream effectors. Additionally, autophagic flux was not measured directly via LC3-II/LC3-I ratio or p62/SQSTM1 degradation, which are essential for confirming functional autophagy modulation. Future studies should evaluate mTOR phosphorylation at Ser2448, examine downstream targets such as p70S6K and 4E-BP1, assess autophagy markers including LC3 and p62, and validate these findings in in vivo stroke models. Despite these limitations, our results provide important preliminary evidence that fendosal modulates the Rheb-mTORC1 pathway under ischemic stress, representing a novel mechanism that warrants further investigation.

Acknowledgements

This research project is supported by Mahidol University (Fundamental Fund: fiscal year 2024 (FF-167/2567) by the National Science Research and Innovation Fund (NSRF)) to SM, and National Research Council of Thailand (NRCT) (N41A640224) to TP.

References

Ajoolabady, A., Wang, S., Kroemer, G., Penninger, J. M., Uversky, V. N., Pratico, D., Henninger, N., Reiter, R. J., Bruno, A., Joshipura, K., Aslkhodapasandhokmabad, H., Klionsky, D. J., & Ren, J. (2021). Targeting autophagy in ischemic stroke: From molecular mechanisms to clinical therapeutics. *Pharmacol Ther*, *225*, 107848. https://doi.org/10.1016/j.pharmthera.2021.107848

Chen, W., Yasen, M., Wang, H., Zhuang, C., Wang, Z., Lu, S., Jiang, L., & Lin, H. (2022). Celecoxib activates autophagy by inhibiting the mTOR signaling pathway and prevents apoptosis in nucleus pulposus cells. *BMC Pharmacol Toxicol*, *23*(1), 90. https://doi.org/10.1186/s40360-022-00633-y

Fu, X., Tan, T., & Liu, P. (2020). Regulation of Autophagy by Non-Steroidal Anti-Inflammatory Drugs in Cancer. *Cancer Manag Res*, *12*, 4595-4604. https://doi.org/10.2147/CMAR.S253345

Harrison, D. E., Strong, R., Sharp, Z. D., Nelson, J. F., Astle, C. M., Flurkey, K., Nadon, N. L., Wilkinson, J. E., Frenkel, K., Carter, C. S., Pahor, M., Javors, M. A., Fernandez, E., & Miller, R. A. (2009). Rapamycin fed late in life extends lifespan in genetically heterogeneous mice. *Nature*, *460*(7253), 392-395. https://doi.org/10.1038/nature08221

Lamming, D. W., Ye, L., Katajisto, P., Goncalves, M. D., Saitoh, M., Stevens, D. M., Davis, J. G., Salmon, A. B., Richardson, A., Ahima, R. S., Guertin, D. A., Sabatini, D. M., & Baur, J. A. (2012). Rapamycin-induced insulin resistance is mediated by mTORC2 loss and uncoupled from longevity. *Science*, *335*(6076), 1638-1643. https://doi.org/10.1126/science.1215135

Lassman, H. B., Wilker, J. C., Anderson, V. B., Agnew, M. N., Allen, R. C., & Novick, W. J., Jr. (1978). Fendosal (HP 129): a potent anti-inflammatory and analgesic compound. *Agents Actions*, *8*(3), 209-217. https://doi.org/10.1007/BF01966606

Li, Y., Yang, L., Dong, L., Yang, Z. W., Zhang, J., Zhang, S. L., Niu, M. J., Xia, J. W., Gong, Y., Zhu, N., Zhang, X. J., Zhang, Y. Y., Wei, X. M., Zhang, Y. Z., Zhang, P., & Li, S. Q. (2019). Crosstalk between the Akt/mTORC1 and NF-kappaB signaling pathways promotes hypoxia-induced pulmonary hypertension by increasing DPP4 expression in PASMCs. *Acta Pharmacol Sin*, *40*(10), 1322-1333. https://doi.org/10.1038/s41401-019-0272-2

Lu, G., Wang, Y., Shi, Y., Zhang, Z., Huang, C., He, W., Wang, C., & Shen, H. M. (2022). Autophagy in health and disease: From molecular mechanisms to therapeutic target. *MedComm (2020)*, *3*(3), e150. https://doi.org/10.1002/mco2.150

Mao, R., Zong, N., Hu, Y., Chen, Y., & Xu, Y. (2022). Neuronal Death Mechanisms and Therapeutic Strategy in Ischemic Stroke. *Neurosci Bull*, *38*(10), 1229-1247. https://doi.org/10.1007/s12264-022-00859-0

Mukda, S., Tsai, C. Y., Leu, S., Yang, J. L., & Chan, S. H. H. (2019). Pinin protects astrocytes from cell death after acute ischemic stroke via maintenance of mitochondrial anti-apoptotic and bioenergetics functions. *J Biomed Sci*, *26*(1), 43. https://doi.org/10.1186/s12929-019-0538-5

National Center for Biotechnology Information. (2025). *PubChem Compound Summary for CID 40821, Fendosal* https://pubchem.ncbi.nlm.nih.gov/compound/Fendosal

Netea-Maier, R. T., Plantinga, T. S., van de Veerdonk, F. L., Smit, J. W., & Netea, M. G. (2016). Modulation of inflammation by autophagy: Consequences for human disease. *Autophagy*, *12*(2), 245-260. https://doi.org/10.1080/15548627.2015.1071759

Panwar, V., Singh, A., Bhatt, M., Tonk, R. K., Azizov, S., Raza, A. S., Sengupta, S., Kumar, D., & Garg, M. (2023). Multifaceted role of mTOR (mammalian target of rapamycin) signaling pathway in human health and disease. *Signal Transduct Target Ther*, *8*(1), 375. https://doi.org/10.1038/s41392-023-01608-z

Qian, M., Fang, X., & Wang, X. (2017). Autophagy and inflammation. *Clin Transl Med*, *6*(1), 24. https://doi.org/10.1186/s40169-017-0154-5

Qin, C., Yang, S., Chu, Y. H., Zhang, H., Pang, X. W., Chen, L., Zhou, L. Q., Chen, M., Tian, D. S., & Wang, W. (2022). Signaling pathways involved in ischemic stroke: molecular mechanisms and therapeutic interventions. *Signal Transduct Target Ther*, *7*(1), 215. https://doi.org/10.1038/s41392-022-01064-1

Szwed, A., Kim, E., & Jacinto, E. (2021). Regulation and metabolic functions of mTORC1 and mTORC2. *Physiol Rev*, *101*(3), 1371-1426. https://doi.org/10.1152/physrev.00026.2020

Villa-Gonzalez, M., Martin-Lopez, G., & Perez-Alvarez, M. J. (2022). Dysregulation of mTOR Signaling after Brain Ischemia. *Int J Mol Sci*, *23*(5). https://doi.org/10.3390/ijms23052814

Wang, G., Chen, L., Qin, S., Zhang, T., Yao, J., Yi, Y., & Deng, L. (2022). Mechanistic Target of Rapamycin Complex 1: From a Nutrient Sensor to a Key Regulator of Metabolism and Health. *Adv Nutr*, *13*(5), 1882-1900. https://doi.org/10.1093/advances/nmac055

Wang, P., Shao, B. Z., Deng, Z., Chen, S., Yue, Z., & Miao, C. Y. (2018). Autophagy in ischemic stroke. *Prog Neurobiol*, *163-164*, 98-117. https://doi.org/10.1016/j.pneurobio.2018.01.001

Bio Design
A 3N Holding Company
@BioDesignTH
@BioDesign
@BioDesignTH
BioDesign CO., LTD.
info@biodesign.co.th
NOVEL SOLUTION FOR LIFE SCIENCE RESEARCH
Molecular Biology Reagents
QIAGEN
EZ2
End-to-end automation of nucleic acid extraction
QIAcuity
Digital PCRinstrument
QIAxcel
Advance automates DNA & RNA QC
Oxford Nanopore Technologies
MinION Mk1D
GridION
ElysION
PromethION™ 2 Solo
PromethION 2
PromethION 24
4bases
Sequencing kits
4eVAR Data Analysis
•BENKit panel
•BRaCA panel
•CFTR panel
•COVID panel
•FH panel
•MICROBIOME panel
•RIDS panel
•THALASSEMIA panel
Solarbio LIFE SCIENCES
Nucleic Acid Electrophoresis
Molecular Cloning -Related Enzymes
Nucleic Acid Extraction/Purification
PCR/qPCR
NEUATION
Vortex Mixer
Centrifuge
Digital 3D Shaker (Roker)
RotatorDisc Rotator
Stirrer
QPlast
Centrifuge Tube
Cryogenic Tube
Microcentrifuge Tube
Serological Pipettes
Cryogenic Rack & Box
ACCUMAX
Accumax FAB Pipette/ Low Force Pipette
Bottle Top Dispenser
Pipette Controller
BioDesign Co., Ltd. 5 Soi Prapasiri, Suthisarnvinitchai Road, Samsennok, HuayKwang, Bangkok 10310, Thailand
www.biodesign.co.th

THEERA TRADING. CO.,LTD.
BIO-RAD
• เครื่อง QX200 Droplet Digital™ PCR, Real-Time PCR และ PCR ประสิทธิภาพสูง
• ชุด Electrophoresis แนวนอน แนวตั้ง เครื่องจ่ายกระแสไฟฟ้า และชุดน้ำยาที่เกี่ยวข้อง
• เครื่องถ่ายภาพเจลและแผ่นเมมเบรน พร้อมโปรแกรมวิเคราะห์ภาพ
• ชุดน้ำยาวิเคราะห์และตรวจวัดโปรตีน
Cell Signaling TECHNOLOGY
แอนติบอดี (Antibody) ชนิด Primary, Conjugated Antibodies และ Secondary Antibodies สำหรับการศึกษาโปรตีนและการส่งสัญญาณต่างๆ ภายในเซลล์ด้วยเทคนิค
• Western Blotting
• Flow Cytometry
• Immunoprecipitation
• Immunohistochemistry
สาร Cytokines and growth factors ต่างๆ ทั้งแบบมี และไม่มี Carrier แอนติบอดี สำหรับงานตรวจวิเคราะห์โปรตีนทั้งภายในเซลล์และบนผิวเซลล์ ทั้งแบบมาตรฐานและติดฉลากเอนไซม์หรือสารเรืองแสง สารกระตุ้นเซลล์และสีย้อมเซลล์เรืองแสง
NEW ENGLAND Biolabs
เอนไซม์ น้ำยา สารมาตรฐาน และสารเคมีต่างๆ ในงานตัดต่อ ดัดแปลง สังเคราะห์ เพิ่มปริมาณ และตรวจวิเคราะห์ DNA RNA และ Protein
SOCOREX SWISS
Precision liquid handling
อุปกรณ์ดูดจ่ายสารละลายและสารเคมีแบบปรับปริมาตรได้
WLD-TEC
ตะเกียงแก๊สอัจฉริยะความปลอดภัยสูงทั้งแบบมาตรฐาน และแบบตั้งโปรแกรมได้ และเครื่องเผาลวดเขี่ยเชื้อแบบใช้ไฟฟ้า
บริษัท ธีระเทรดดิ้ง จำกัด
เลขที่ 27,29,31 ถนนเทอดไท แขวงบางยี่เรือ
เขตธนบุรี กรุงเทพมหานคร 10600
โทร. 02-466-4491-94
info@theetrad.com , sale@theetrad.com

TNS28 AWARDS

TNS28 Awards Announcement

The TNS28 Awards recognize outstanding research contributions presented at the conference. A total of 23,000 Thai Baht (THB) will be awarded across the following categories:

1. Best Abstract Award for Oral Presentations

- Four abstracts will be selected for oral presentation (15 minutes each).
- Each selected abstract will receive an award of 2,000 THB.
- Total prize amount: 8,000 THB (Supported by Professor Ingrid Liu).

2. Best Abstract Award for Flash Talks

- Three abstracts will be selected for flash talk presentations (5 minutes each).
- Each selected abstract will receive an award of 1,000 THB.
- Total prize amount: 3,000 THB (Supported by TNS).

3. Best Poster Presentation Awards

- Awards will be presented to outstanding poster presentations selected during the poster session as follows:
    - **Professor Prasop Ratanakorn Award** – 4,000 THB (for outstanding research achievements)
    - **Professor Roongtam Ladpli Award** – 2,000 THB
    - **Professor Ingrid Liu Award** – 2,000 THB
    - **Associate Professor Naipinich Kotchabhakdi Award** – 2,000 THB
    - **Mr. Tay Khoon Yang Award (Hi-Tech Imaging Co. Ltd.)** – 2,000 THB
- Total prize amount: 12,000 THB.

Overall Total

- The total prize value for all TNS28 Awards is 23,000 THB.
- These awards underscore the conference's commitment to recognizing excellence in research, innovation, and scientific communication.

ACKNOWLEDGEMENTS

Acknowledgements for TNS28

Special Thanks to our supports

AUTHOR INDEX

Adisorn Ratanayotha, 121
Alisa Damnernsawad, 177
Amarin Thongsuk, 142
Amit Bhatia, 90
Amit Jaisi, 86
Amornpun Sereemaspun, 75
Amrendra Jha, 120
Anantha Harijith, 197
Anchalee Vattarakorn, 219
Anda Cimpean, 78
Andrea Serio, 206
Anjana Bali, 94
Ankit Ganeshpurkar, 209
Anuj Babbar, 93
Anupong Thongklam Songsaad, 142
Anuradha Sharma, 93
Anusara Aranarochana, 159
Anyapat Atipimonpat, 221
Apinun Kanpiengjai, 153
Apinya Sayinta[1], 97
Apiwat Sirichoat[1], 159
Arbaz Sujat Shaikh, 138
Aree Wanasuntronwong, 222
Arnaud Monteil, 31, 36, 49
Ashata Dahal, 120
Bahrie Ramadan, 167
Banthit Chetsawang, 7, 8, 24, 25, 60, 184, 201, 204, 208, 211
Benjamard Thaweethee-Sukjai, 202
Benjamin Ongnok, 169, 171, 177, 181
Benjaporn Kiatpakdee, 116, 118, 125, 127
Bhornluck Paepetch Suato, 121
Boonrat Tassaneetrithep, 124
Brijesh Taksande, 140
Catleya Rojviriya, 59
Chainarong Tocharus, 144, 146, 153
Chairat Turbpaiboon, 8, 80, 82, 84, 121, 124
Chanisa Thonusin, 190
Charoensri Thonabulsombat, 142
Chatchawan Rattanabannakit, 129
Chattraporn Nantawanichakorn, 194, 221
Chinarat Changsangfa, 118, 127
Ching-Yi Tsai, 77
Chonnicha Subkod, 164, 211, 223
Daesoo Kim, 48
Dai Wenjing, 88
Dana Mohammad Khader AlNajmi, 201
Danail Stoychev, 191
Danuyada Wattanaumadechakul, 80
Darunee Rodma, 82, 84, 97
Dhanveer Ahamed Bathurutheen, 198
Dilip Thakur, 120
Divya Soni, 90
Dungdol Narasirilrk, 194
Ekkaphot Khongkla, 201
Fahsai Kaewpikul, 196
Fahsai Thaion, 211
Fareda Sapphot, 205
Farouk Raed, 201
Fikri Aminullah, 201
Francesca Robertson, 191
Gavin P. Reynolds, 202
Ghulam Hussain, 123
Guo Wan-Jun, 88
H.K.L Nguyen, 182
H.T Nguyen, 182
Ha Thi Thanh Huong, 55
Hanwen Mi, 171
Harapriya Baral, 138
Hathaitip Sritanaudomchai, 221
Hathaitip Sritanaudomchai, 194
Henry Houlden, 123
Hideki Yoshida, 31, 36, 64, 157
Hiranya Pintana, 67, 179
Honda Y, 99
Houzhi Cheng, 179
Ian Adams, 197
Ilan Davis, 191
Ingrid Y Liu, 42
Ingrid Y. Liu, 155, 158, 166
Ishwar Parhar, 72
Ismat Noor Paromi, 158
Jakkrit Nukitram, 114
James W. Fawcett, 78
Jamie A. O’Reilly, 100
Jana Dubisova, 78
Jariya U Welbat, 159
Jeffrey Y Lee, 191
Jessica C.F. Kwok, 78
Jiasui Yu, 200
Jim Vadolas, 118, 125, 127
Jim Valodas, 116
Jinnipa Matchwong, 194
Jintanaporn Wattanathorn, 114
Jirakhamon Sengking, 144, 146, 153
Jiraporn Panmanee, 130, 164, 184, 196, 211, 223
Jiraporn Tocharus, 144, 146, 153
Jittiporn Wongpun, 153
Juefang Gan, 153

Juntratip Jomrit, 86
Jureepon Roboon, 161, 173, 175, 202
K. Venkata Rao, 138
Kanlaya Prapainop Katewongsa, 204
Kanokporn Wantem, 205
Kanyaratana Bamrungsuk, 121
Kittikun Viwatpinyo, 125
Kornkanok Promthep, 130, 164, 184, 201, 211, 223
Kotchapit Maksri, 208
Kovit Pattanapanyasat, 194, 221
Kowit Hengphasatporn, 169
Krai Meemon, 82, 84, 97
Krisada Rungsang, 169
Kunlaya Somboonwiwat, 205
Kurisara Boonyakitmaitree, 193
Ladawan Khowawisetsut, 82, 84
Lalitta Suriya-Arunroj, 30, 35, 74
Lattanawan Inchaisri, 222
Li Tao, 88
Long-Jun Wu, 40
Mananya Potima, 192, 193
Manisha Suri, 94
Manoj P. Dandekar, 138
Maral Daneshpazhouh, 201
Mathurada Saephu, 144, 146
Monruedee Sukprasansap, 169, 171, 177
Morakot Sroyraya, 82, 84
Mubashir Raza, 166
Muhammad Jahangir, 88
Munnutchaya Tarmtiranont, 184
Nadiah Syafiqah Nor Azman, 86
Nakamura S, 99
Namfa Sermkaew, 86
Namphung Thongta, 219
Nanthanit Pholphana, 80
Naparat Promyoo, 144, 146
Napasakorn Verasaksuriya, 169
Napatsorn Jongapirattanakul, 121
Napol Kaewkascholkul, 205
Narawut Pakaprot, 7, 8, 50, 70, 219
Narinporn Tresteankij, 181
Narisorn Kitiyanant, 130, 164, 211, 223
Nataya Sritawan, 159
Nathorn Chaiyakunapruk, 202
Nattawadee Panyain, 169
Nhan Do Thi Thuy, 155
Nipon Chattipakorn, 179, 190
Nisarat Ruangsawasdi, 142
Nithi Asavapanumas, 8, 31, 35, 65
Nuchanart Rangkadilok, 80
Nuttanan Pholngam, 118, 125, 127
Nuttapon Songnaka, 86
Nutthida Phianchana, 101
Ogawa K, 99
Onrawee Khongsombat, 222
Pacharaporn Suklai, 206
Pakpoom Subsoontorn, 221
Panapat Uawithya, 80
Pannatorn Buntha, 86
Paranee Yatmark, 116
Parin Suwannapradpha, 116
Parinda Jamrus, 118, 125, 127
Patcharapha Poonthawatsanti, 193
Patcharapong Pantiya, 179
Pathidta Chodewaratham, 223
Patit Choksawangkarn, 177
Patlada Tangweerasing, 223
Pavla Jendelova[1], 78
Pawanrat Chalorak, 97
Paweena Kaewman, 161
Peeraporn Varinthra, 155, 158
Petr V. Gorelkin, 139
Phakkawat Thangwong, 146
Phetcharat Boonruamkaew, 86
Phossawee Kongkaew, 199
Piangkwan Sa-nguanmoo, 220
Pipob Suwanchaikasem, 201
Piyawat Katewongsa, 204
Plaiyfah Janthueng, 161
Ponthip Cheenkhwan, 175
Poramet Klangmongkon, 177
Pornkanok Nimnoi, 124
Pornprom Surakul, 221
Pornthip Chaichompoo, 116, 118, 125, 127
Prao Pongpipat, 193
Prapapan Temkitthawon, 161, 173
Prasert Sobhon, 82, 84
Prathima Basa, 197
Preeyanuch Manohong, 204
Puncharatsm Pannin, 159
Puneet Kumar, 79, 90
Q.C Pham, 182
Rabia Akram, 123
Rafi Aliefiyanto, 201
Raj Katariya, 140
Ranchana Yeewa, 7, 29, 33, 35, 54
Ratree Sawangjit, 202
Rujapope Sutiwisesak, 116
Rungrat Palakai, 204
Rungsarit Sunan, 82, 84, 97

Salinee Jantrapirom, 4, 7, 8, 30, 32, 35, 36, 69
Samuel T. Ward, 193
Samur Thanoi, 161, 173, 175, 202
Saovaros Svasti, 116, 118, 125, 127
Sawanya Charoenlappanit, 161, 173, 175
Shahid Mahmood Baig, 123
Shin-Meng Deng, 163
Shubham Upadhayay, 90
Simranjit Kaur, 138
Siraprapa Boobphahom, 208
Sirikran Juntapremjit, 92
Sirinapa Mysook, 204
Sirintip Kumfu, 220
Sirinun Boripantakul, 220
Siripaporn Kesyou, 167
Siriporn C Chattipakorn, 179, 190, 220
Siriporn Chamniansawat, 199
Siriporn Chattipakorn, 38, 44
Sittiruk Roytraku, 175
Sittiruk Roytrakul, 161, 173
Sivanan Sivasinprasasn, 153
Sompol Tapechum, 219
Somporn Sungkarat, 220
Sovaritthon Chansaengsee, 130
Stephanie Dumontoy, 167
Stephanie Efthymiou, 123
Steve Leu, 163
Suchiwa Pan-on, 175
Sudichhya Tamrakar, 130
Sujira Mukda, 130, 163, 184, 223
Sukonthar Ngampramuan, 118, 125, 127, 194, 196, 221
Sukrit Promtang, 97
Suk-yu Yau, 200
Sulaifan Waehama, 202
Suman Pokhrel, 120
Sunhapas Soodvilai, 169, 171, 177
Suparada Aiemongkot, 127
Supin Chompoopong, 2, 6, 7, 8, 24, 25, 82, 84, 121, 124
Suprakit Jiraratwattana, 129
Surapon Piboonpocanun, 130, 223
Sushil Kumar Singh, 209
Sutin Kingtong, 92
Sutisa Nudmamud-Thanoi, 57, 161, 167, 173, 175, 202
T.D.M Nguyen, 182
T.T.H Ha, 182
Tai Chaiamarit, 181, 191
Tanida Chokpanuwat, 181
Tanya Prasertporn, 223
Tara Sudhadevi, 197
Tarinee Chodchavanchai, 142
Tatcha Balit, 142
Tehreem Iman, 123
Tewin Tencomnao, 169, 171, 177
Theerawat Khunsanthia, 92
Thenmoly Damodaran, 198
Theptharin Charuraksa, 164, 223
Timothy W. Budd, 101
Titaree Yamsri, 157
Titikorn Chunchai, 179
Tiwari VK, 99
Toshihide Yamashita, 62
Tsutsui KI, 99
Utid Suriya, 171
Vangene Tay, 139
Vasilii S. Kolmogorov, 139
Vincent Van Waes, 167
Vishal Kumar, 79
Vorasith Siripornpanich, 31, 35, 53, 101, 192, 193, 196, 208
Vorasuk Shotelersuk, 46
Wachirapong Saleeon, 219
Wael Mohamed, 51
Wanfrutkon Waehama, 161, 173
Waracharee Srifa, 221
Waree Tiyaboonchai, 175
Wattana Watanapa, 219
Wongsakorn Siripan, 124
Wuttipong Marsraksa, 86
Yanmei Huang, 190
Yaowapa Trangan, 204, 208
Yasuteru Shigeta, 169
Yingrak boondam, 219
Yogesh Garg, 90
Yoottana Janthakhin, 92
Yosita Yongyuen, 86

INDEX

ทะเบียนเลขที่ จ.๓๖๕๔/๒๕๖๘ ส.ค.๕

ใบสำคัญแสดงการจดทะเบียนการแก้ไขเพิ่มเติมข้อบังคับ

ใบสำคัญฉบับนี้ออกให้เพื่อแสดงว่า สมาคมประสาทวิทยาศาสตร์ไทย สำนักงานใหญ่ตั้งอยู่ ณ ภาควิชากายวิภาคศาสตร์ คณะวิทยาศาสตร์ มหาวิทยาลัยมหิดล เลขที่ ๒๗๒ ถนนพระรามที่ ๖ เขตราชเทวี กรุงเทพมหานคร มีการจดทะเบียนการแก้ไขเพิ่มเติมข้อบังคับของสมาคม และนายทะเบียนสมาคมกรุงเทพมหานครได้รับจดทะเบียนการแก้ไขเพิ่มเติมข้อบังคับของสมาคม 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 แต่คณะกรรมการชุดใหม่ยังไม่ได้รับอนุญาตให้จดทะเบียนจากทางราชการ ก็ให้คณะกรรมการที่ครบกำหนดตามวาระรักษาการไปพลางก่อน จนกว่าคณะกรรมการชุดใหม่จะได้รับอนุญาตให้จดทะเบียนจากทางราชการ และเมื่อคณะกรรมการชุดใหม่ได้รับอนุญาตให้จดทะเบียนจากทางราชการเป็นที่เรียบร้อยแล้ว ก็ให้ทำการส่งและรับมอบงานกันระหว่างคณะกรรมการชุดเก่าและคณะกรรมการชุดใหม่ ให้เป็นที่เสร็จสิ้นภายใน ๓๐ วัน นับตั้งแต่วันที่คณะกรรมการชุดใหม่ได้รับอนุญาตให้จดทะเบียนจากทางราชการ

ให้ไว้ ณ วันที่ ๗ ตุลาคม พ.ศ. ๒๕๖๘

(นางสาวสิริญรัศมิ์ นุตตะรังค์)
นายอำเภอเวียงเก่า รักษาการในตำแหน่ง
ผู้อำนวยการส่วนทะเบียนมูลนิธิ และสมาคม ปฏิบัติราชการแทน
อธิบดีกรมการปกครอง
นายทะเบียนสมาคมกรุงเทพมหานคร

ทะเบียนเลขที่ จ.๓๖๕๔/๒๕๖๘ ส.ค.๖

ใบสำคัญแสดงการจดทะเบียนการแต่งตั้งกรรมการของสมาคมขึ้นใหม่ทั้งชุด
หรือการเปลี่ยนแปลงกรรมการของสมาคม

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

๑.	นางสุพิน	ชมภูพงษ์	นายกสมาคม
๒.	นายชัยเลิศ	พิชิตพรชัย	อุปนายก คนที่ ๑ และกรรมการกิจการพิเศษ
๓.	นางสุทิสา	ถาน้อย	อุปนายก คนที่ ๒
๔.	นายบัณฑิต	เจตน์สว่าง	กรรมการ
๕.	นางสมพร กันทรดุษฎี	เตรียมชัยศรี	กรรมการ
๖.	นายอัครเดช	ศิริพร	กรรมการและวิชาการ
๗.	นายชัยรัตน์	เติบไพบูลย์	กรรมการและผู้ช่วยวิชาการ
๘.	นายนราวุฒิ	ภาคาพรต	กรรมการและปฏิคม
๙.	นายอดิศร	รัตนโยธา	กรรมการและนายทะเบียน
๑๐.	นางมนัสพร	พัสระ	กรรมการและประชาสัมพันธ์
๑๑.	นายอมรพันธุ์	เสรีมาศพันธุ์	กรรมการและผู้ช่วยกิจการพิเศษ
๑๒.	นายประทีป	อมรรัตนพันธ์	กรรมการและผู้ช่วยกิจการพิเศษ
๑๓.	นางสาวกุลธิดา	ชัยธีระยานนท์	กรรมการและเหรัญญิก
๑๔.	นางสาวสุจิรา	มุกดา	กรรมการและเลขาธิการ
๑๕.	นางสาวอัจฉราภรณ์	คำศรีใจ	กรรมการและผู้ช่วยเลขาธิการ

ให้ไว้ ณ วันที่ ๗ ตุลาคม พ.ศ. ๒๕๖๘

(นางสาวสิริญรัศมิ์ นุตตะรังค์)
นายอำเภอเวียงเก่า รักษาการในตำแหน่ง
ผู้อำนวยการส่วนทะเบียนมูลนิธิ และสมาคม ปฏิบัติราชการแทน
อธิบดีกรมการปกครอง
นายทะเบียนสมาคมกรุงเทพมหานคร

คำสั่งมหาวิทยาลัยเชียงใหม่

ที่ ๓๕๔๓ /2568

เรื่อง แต่งตั้งคณะกรรมการจัดการประชุมวิชาการนานาชาติด้านประสาทวิทยาศาสตร์

The 28th Annual Thai Neuroscience Society Conference 2025 (TNS28)

หัวข้อ Neuroplasticity Across the Lifespan

....

ด้วยสมาคมประสาทวิทยาศาสตร์ไทย (Thai Neuroscience Society: TNS) ร่วมกับภาควิชาเภสัชวิทยา คณะแพทยศาสตร์ มหาวิทยาลัยเชียงใหม่ และศูนย์บูรณาการเทคโนโลยีการแพทย์ทันสมัย (Center of Multidisciplinary Technology for Advanced Medicine, CMUTEAM) ได้กำหนดจัดการประชุมวิชาการนานาชาติด้านประสาทวิทยาศาสตร์ครั้งที่ 28 (The 28th Annual Thai Neuroscience Society Conference 2025: TNS28) ภายใต้หัวข้อ "Neuroplasticity Across the Lifespan: Advancing Neuroplasticity Research Through Cutting-Edge Methodologies" โดยการประชุมวิชาการดังกล่าวนี้จะจัดร่วมกับ IBRO-APRC Supported Associate School ซึ่งได้รับการสนับสนุนหลักจากองค์กรระหว่างประเทศ (International Brain Research Organization: IBRO) โดยจะจัดขึ้นระหว่างวันที่ 27 – 31 ตุลาคม พ.ศ. 2568 ณ คณะแพทยศาสตร์ มหาวิทยาลัยเชียงใหม่ และโรงแรมแคนทารี ฮิลส์ เชียงใหม่ นอกจากนี้ยังมีกิจการพิเศษของสมาคมประสาทวิทยาศาสตร์ไทย คือ TNS Neuro Quiz 2025 (High School level) และ SKT Workshops (Pre-Post Conference workshops) โดยมีวัตถุประสงค์เพื่อติดตามวิทยาการความก้าวหน้า ในวิชาการจากวิทยากรรับเชิญและผู้ทรงคุณวุฒิ การแสดงผลงานทางวิชาการจากอาจารย์แพทย์ นักวิทยาศาสตร์ นักวิจัย นักศึกษา นักเรียนระดับมัธยมปลาย สมาชิกสมาคมฯ และบุคคลทั่วไป เพื่อเป็นการส่งเสริม และกระตุ้นให้ผู้เข้าร่วมประชุมฯ เข้าถึงความรู้ ความก้าวหน้าทางด้านประสาทวิทยาศาสตร์ที่ทันสมัย สามารถนำไปประยุกต์ใช้ทั้งในการดำเนินชีวิต และพัฒนาการจัดการเรียนการสอนในหลักสูตรทางการแพทย์และวิทยาศาสตร์สุขภาพ รวมทั้งได้เผยแพร่ผลงานวิจัย แลกเปลี่ยนความรู้ และวิทยาการต่าง ๆ ในสาขาวิชาประสาทวิทยาศาสตร์ และสาขาอื่น ๆ ที่เกี่ยวข้อง มีโอกาสสร้างความร่วมมือกันของนักวิชาการ นักวิจัยระหว่างประเทศ เพื่อสร้างความสัมพันธ์ ความเข้าใจ และความร่วมมือระหว่างผู้ทำงานในสาขาวิชาเดียวกันจากสถาบันทั้งในประเทศ และต่างประเทศ

เพื่อให้การดำเนินงานเป็นไปด้วยความเรียบร้อย คล่องตัว และมีประสิทธิภาพ อาศัยอำนาจตามความในมาตรา 35 และมาตรา 38 แห่งพระราชบัญญัติมหาวิทยาลัยเชียงใหม่ พ.ศ. 2551 จึงแต่งตั้งคณะกรรมการจัดการประชุมวิชาการนานาชาติด้านประสาทวิทยาศาสตร์ The 28th Annual Thai Neuroscience Society Conference 2025 (TNS28) หัวข้อ Neuroplasticity Across the Lifespan ดังมีรายนามต่อไปนี้

-2-

คณะกรรมการดำเนินการประชุมวิชาการ

ที่ปรึกษา

1. ศาสตราจารย์ ดร.นายแพทย์ศุภนิมิต ทีฆชุณหเถียร
2. ศาสตราจารย์ ดร.ภาวิช ทองโรจน์
3. ศาสตราจารย์ ดร.ปิยะรัตน์ โกวิทตรพงศ์
4. ศาสตราจารย์ ดร.สุขุมาล จงธรรมคุณ
5. รองศาสตราจารย์ ดร.เภสัชกรหญิงกนกวรรณ ติลกสกุลชัย

คณะกรรมการ

1.	ศาสตราจารย์ ดร.นายแพทย์ดำเนินสันต์ พฤกษากร	ประธานกรรมการ
2.	ศาสตราจารย์ ดร.พันโทหญิงสุพิน ชมภูพงษ์	ประธานกรรมการร่วม
3.	ดร.ลูก้า โล ปิคโคโล	รองประธานกรรมการ
4.	อาจารย์ ดร.เภสัชกรหญิงสาลินี จันทราภิรมย์	กรรมการและผู้ประสานงานหลัก
5.	ศาสตราจารย์ ดร.สุทิสา ถาน้อย	กรรมการ
6.	ศาสตราจารย์ ดร.สมพร กันทรดุษฎี เตรียมชัยศรี	กรรมการ
7.	ศาสตราจารย์ ดร.บัณฑิต เจตน์สว่าง	กรรมการ
8.	รองศาสตราจารย์ ดร.นายแพทย์ชัยเลิศ พิชิตพรชัย	กรรมการ
9.	รองศาสตราจารย์ ดร.นายแพทย์ชัยรัตน์ เติบไพบูลย์	กรรมการ
10.	รองศาสตราจารย์ ดร.นายแพทย์นราวุฒิ ภาคาพรต	กรรมการ
11.	รองศาสตราจารย์ แพทย์หญิงณัฐรุจี วิวรรธนดิฐกุล	กรรมการ
12.	รองศาสตราจารย์ ดร.กายภาพบำบัดอัครเดช ศิริพร	กรรมการ
13.	รองศาสตราจารย์ ดร.กุลธิดา ชัยธีระยานนท์	กรรมการ
14.	ผู้ช่วยศาสตราจารย์ ดร.นายแพทย์อดิศร รัตนโยธา	กรรมการ
15.	รองศาสตราจารย์ ดร.เภสัชกรหญิงรังษินี พงษ์ประดิษฐ์	กรรมการ
16.	ผู้ช่วยศาสตราจารย์ ดร.เภสัชกรหญิงจันทร์นภัสสร์ ธราวิจิตรกุล	กรรมการ
17.	ผู้ช่วยศาสตราจารย์ ดร.นายแพทย์อมรพันธุ์ เสรีมาศพันธุ์	กรรมการ
18.	ผู้ช่วยศาสตราจารย์ ดร.มนัสพร พัสระ	กรรมการ
19.	ผู้ช่วยศาสตราจารย์ ดร.ฤดีมาศ ยุบลพันธุ์	กรรมการ
20.	อาจารย์ ดร.ประทีป อมรรัตนพันธ์	กรรมการ
21.	ดร.วาสินี วงศ์คำมูล	กรรมการ
22.	ดร.สัตวแพทย์หญิงรัญชนา ยี่หวา	กรรมการและเลขานุการ 1
23.	รองศาสตราจารย์ ดร.สุจิรา มุกดา	กรรมการและเลขานุการ 2
24.	อาจารย์ ดร.อัจฉราภรณ์ คำศรีใจ	กรรมการและรองเลขานุการ
25.	นางสาวปัทชา ศึกษากิจ	กรรมการและผู้ช่วยเลขานุการ
26.	นางสาวยุพเรศ กองนาค	กรรมการและผู้ช่วยเลขานุการ

-3-

27. นางสาวธัญพิชชา มีสวัสดิ์ กรรมการและผู้ช่วยผู้ประสานงาน
28. นางสาวณัฐสินีย์ อยู่อ่อน กรรมการและผู้ช่วยผู้ประสานงาน
29. นางสาวฉันสุนีย์ ปานโต กรรมการและผู้ช่วยผู้ประสานงาน
30. นางสาวรัชนารี สืบสายแปง กรรมการและผู้ช่วยผู้ประสานงาน

โดยให้คณะกรรมการดังกล่าวมีหน้าที่

- วางแผน กำหนดเนื้อหาทางวิชาการของการประชุม เตรียมงาน ประสานงานกับฝ่ายต่าง ๆ ดำเนินการ จัดประชุมและสรุปติดตามการดำเนินงาน

คณะอนุกรรมการฝ่ายวิชาการ

1. ดร.ลูก้า โล ปิคโคโล ประธานอนุกรรมการ
2. รองศาสตราจารย์ ดร.กายภาพบำบัดอัครเดช ศิริพร ประธานอนุกรรมการร่วม
3. รองศาสตราจารย์ ดร.นายแพทย์ชัยรัตน์ เติบไพบูลย์ รองประธานอนุกรรมการ
4. ศาสตราจารย์ ดร.พันโทหญิงสุพิน ชมภูพงษ์ อนุกรรมการ
5. ศาสตราจารย์ ดร.สุขุมาล จงธรรมคุณ อนุกรรมการ
6. ศาสตราจารย์ ดร.สุทิสา ถาน้อย อนุกรรมการ
7. ศาสตราจารย์ ดร.บัณฑิต เจตน์สว่าง อนุกรรมการ
8. รองศาสตราจารย์ ดร.แพทย์หญิงศรัณยภิญ โพธิกานนท์ อนุกรรมการ
9. รองศาสตราจารย์ แพทย์หญิงณัฐรุจี วิวรรธนดิฐกุล อนุกรรมการ
10. รองศาสตราจารย์ ดร.เภสัชกรหญิงรังษินี พงษ์ประดิษฐ อนุกรรมการ
11. ผู้ช่วยศาสตราจารย์ ดร.เภสัชกรหญิงจันทร์นภัสสร ธราวิจิตรกุล อนุกรรมการ
12. ผู้ช่วยศาสตราจารย์ ดร.ฤดีมาศ ยุบลพันธุ์ อนุกรรมการ
13. อาจารย์ ดร.พัชริดา จันตารี อนุกรรมการ
14. ดร.สัตวแพทย์หญิงรัญชนา ยี่หวา อนุกรรมการ
15. ดร.วาสินี วงศ์คำมูล อนุกรรมการ
16. ดร.หิรัญญา ปินตานา อนุกรรมการ
17. ดร.ฐิติกร จันทร์ไชย อนุกรรมการ
18. อาจารย์ ดร.เภสัชกรหญิงสาลินี จันทราภิรมย์ อนุกรรมการและเลขานุการ

โดยให้คณะอนุกรรมการดังกล่าวมีหน้าที่

1. ติดต่อวิทยากรด้วยการออกจดหมายเชิญ เพื่อจัดเตรียมประวัติวิทยากร บทความหรือบทคัดย่อของวิทยากร
2. จัดลำดับการบรรยาย กำหนดประธาน รองประธานในแต่ละช่วงการประชุม
3. รับผลงานทางวิชาการที่ผู้เข้าร่วมการประชุมส่งผ่านช่องทางเว็บไซต์สมาคมฯ พิจารณาคัดเลือกผลงานสำหรับการเข้าร่วมประชุม พร้อมกำหนดลำดับหัวข้อ การนำเสนอผลงานทางวิชาการ
4. ดำเนินการเรื่องรางวัลการนำเสนอผลงาน กำหนดกรรมการ และพิจารณาตัดสินผลสำหรับผลงานทางวิชาการของผู้เข้าร่วมประชุม

5. จัดทำต้นฉบับหนังสือบทความหรือบทคัดย่อของการบรรยายของวิทยากร ผลงานทางวิชาการที่เข้าร่วมนำเสนอในการประชุมและเอกสารผู้สนับสนุนการประชุมเพื่อนำเสนอในรูปแบบอิเล็กทรอนิกส์ และ/หรือรูปเล่ม
6. ประสานงานกับกรรมการฝ่ายพิธีการ และประชาสัมพันธ์

คณะอนุกรรมการฝ่าย IBRO-APRC Supported Associate School

1. อาจารย์ ดร.เภสัชกรหญิงสาลินี จันทราภิรมย์ ประธานอนุกรรมการ
   Chair of the IBRO-APRC Supported Associate School 2025
2. ศาสตราจารย์ ดร.นายแพทย์ศุภนิมิต ทีฆชุณหเถียร อนุกรรมการ
3. รองศาสตราจารย์ แพทย์หญิงณัฐรุจี วิวรรธนดิฐกุล อนุกรรมการ
4. ผู้ช่วยศาสตราจารย์ ดร.เภสัชกรหญิงจันทร์นภัสสร์ ธราวิจิตรกุล อนุกรรมการ
5. รองศาสตราจารย์ ดร.เภสัชกรหญิงรังษินี พงษ์ประดิษฐ อนุกรรมการ
6. ผู้ช่วยศาสตราจารย์ ดร.ฤดีมาศ ยุบลพันธุ์ อนุกรรมการ
7. อาจารย์ ดร.พัชริดา จันตารี อนุกรรมการ
8. ดร.สัตวแพทย์หญิงรัญชนา ยี่หวา อนุกรรมการ
9. ดร.วาสินี วงศ์คำมูล อนุกรรมการ
10. ดร.ลูก้า โล ปิคโคโล อนุกรรมการและเลขานุการ
11. นางสาวยุพเรศ กองนาค อนุกรรมการและผู้ช่วยเลขานุการ
12. นางสาวธัญพิชชา มีสวัสดิ์ อนุกรรมการและผู้ช่วยเลขานุการ
13. นางสาวณัฐสินีย์ อยู่อ่อน อนุกรรมการและผู้ช่วยเลขานุการ
14. นางสาวฉันสุนีย์ ปานโต อนุกรรมการและผู้ช่วยผู้ประสานงาน
15. นางสาวรัชนารี สืบสายแปง อนุกรรมการและผู้ช่วยผู้ประสานงาน

โดยให้คณะอนุกรรมการดังกล่าวมีหน้าที่

1. นำเสนอโครงการเพื่อขอทุนในการจัด IBRO-APRC Supported Associate School: Winter School on Neuroplasticity Across the Lifespan ต่อ IBRO และประสานงานกับ Program committee ของ IBRO ในการประกาศรับสมัครนักเรียนที่ขอทุนสนับสนุนผ่านเว็บไซต์ของ IBRO
2. ทำการพิจารณาคัดเลือกใบสมัครที่เหมาะสมจะได้รับการสนับสนุนจาก IBRO-APRC Supported Associate School
3. ทำหนังสือขอความร่วมมือและอนุเคราะห์ในการใช้สถานที่เพื่อการบรรยาย ปฏิบัติการและการตั้งแสดง รวมทั้งเตรียมความพร้อมสื่อคอมพิวเตอร์และวีดิทัศน์
4. จัดทำตารางกิจกรรมการเรียนการสอนต่าง ๆ ดูแล และจัดเตรียมที่พัก อาหาร และการเดินทางของวิทยากร และ participants ของ IBRO-APRC Supported Associate School
5. ประสานงานด้านการเข้าร่วมประชุมเพื่อการร่วมบรรยายของ IBRO-APRC วิทยากร และการร่วมนำเสนอผลงานวิชาการโปสเตอร์ของ IBRO-APRC participants ใน TNS28

6. ทำรายงานสรุปพร้อมผลวิเคราะห์แบบประเมินการจัด IBRO-APRC Supported Associate School: Winter School on Neuroplasticity Across the Lifespan ต่อ IBRO Committee
7. ประสานงานกับกรรมการฝ่ายวิชาการและฝ่ายทะเบียน

คณะอนุกรรมการฝ่ายกิจการพิเศษ: TNS Neuro-Quiz, SKT workshop และ IL workshop

1.	รองศาสตราจารย์ ดร.นายแพทย์ชัยเลิศ พิชิตพรชัย	ประธานอนุกรรมการ
2.	ศาสตราจารย์ ดร.สมพร กันทรดุษฎี เตรียมชัยศรี	รองประธานอนุกรรมการ
3.	ศาสตราจารย์ ดร.พันโทหญิงสุพิน ชมภูพงษ์	อนุกรรมการ
4.	ศาสตราจารย์ ดร.บัณฑิต เจตน์สว่าง	อนุกรรมการ
5.	รองศาสตราจารย์ ดร.กายภาพบำบัดอัครเดช ศิริพร	อนุกรรมการ
6.	รองศาสตราจารย์ ดร.สุจิรา มุกดา	อนุกรรมการ
7.	ผู้ช่วยศาสตราจารย์ ดร.นายแพทย์อมรพันธุ์ เสรีมาศพันธุ์	อนุกรรมการ
8.	ผู้ช่วยศาสตราจารย์ ดร.ภิรมย์ เชนประโคน	อนุกรรมการ
9.	อาจารย์ ดร.กายภาพบำบัดสุกฤษฎิ์ พรมแตง	อนุกรรมการ
10.	อาจารย์ ดร.ประทีป อมรรัตนพันธุ์	อนุกรรมการ
11.	อาจารย์ ดร.สุพรรณ ยอดยิ่งยง	อนุกรรมการ
12.	อาจารย์ กายภาพบำบัดก่อลาภ กอบโกย	อนุกรรมการ
13.	นางสาวจิราภรณ์ การะเกตุ	อนุกรรมการ
14.	นางสาวอัจฉราพรรณ โพธิ์ทอง	อนุกรรมการ
15.	นายธนายุทธ อังกิตานนท์	อนุกรรมการ
16.	นายพงษ์ผไท กิจรุ่งโรจนาพร	อนุกรรมการ
17.	นายนำโชค ขุนหมื่นวงค์	อนุกรรมการ
18.	อาจารย์ ดร.อัจฉราภรณ์ คำศรีใจ	อนุกรรมการและเลขานุการ

โดยให้คณะอนุกรรมการดังกล่าวมีหน้าที่

1. จัดทำ announcement และจดหมายเพื่อประชาสัมพันธ์ TNS Neuro-Quiz, SKT workshop และ Innovative Learning Activity workshop
2. จัดทำการตอบคำถามรอบคัดเลือกแบบ Online และประสานงานด้านการเตรียมความพร้อมสื่อคอมพิวเตอร์ เวที และห้องประชุมเพื่อการตอบคำถามรอบชิงชนะเลิศแบบ On-site
3. ดำเนินการจัดกิจกรรม Innovative Learning Activity workshop หัวข้อ Utilizing Silica Aerogel Properties for Innovating New Product, หัวข้อ Exploring Science Concepts Through Board Games และ Robotic
4. ดำเนินการจัดกิจกรรม SKT Workshop: The Elderly memory and Health Healing
5. ประสานงานด้านการเตรียมความพร้อมสื่อคอมพิวเตอร์และสถานที่เพื่อจัดกิจกรรม TNS Neuro-Quiz, SKT workshop และ Innovative Learning Activity workshop

6. จัดหาโดยประสานกับผู้สนับสนุนหลัก เพื่อการมอบผลิตภัณฑ์ ของที่ระลึก โล่ รางวัล สำหรับ TNS Neuro-Quiz
7. จัดเตรียมใบประกาศเกียรติคุณการเข้าร่วมกิจกรรม สำหรับ TNS Neuro-Quiz และ SKT workshop
8. ประสานงานกับกรรมการฝ่ายประชาสัมพันธ์ ฝ่ายสถานที่และโสตทัศนูปกรณ์

คณะอนุกรรมการฝ่ายพิธีการ

1.	อาจารย์ ดร.เภสัชกรหญิงสาลินี จันทราภิรมย์	ประธานอนุกรรมการ
2.	ศาสตราจารย์ ดร.พันโทหญิงสุพิน ชมภูพงษ์	อนุกรรมการ
3.	รองศาสตราจารย์ ดร.นายแพทย์ชัยรัตน์ เติบไพบูลย์	อนุกรรมการ
4.	ผู้ช่วยศาสตราจารย์ ดร.นายแพทย์อดิศร รัตนโยธา	อนุกรรมการ
5.	ผู้ช่วยศาสตราจารย์ ดร.มนัสพร พัสระ	อนุกรรมการ
6.	อาจารย์ ดร.อัจฉราภรณ์ คำศรีใจ	อนุกรรมการ
7.	ผู้ช่วยศาสตราจารย์ ดร.ฤดีมาศ ยุบลพันธุ์	อนุกรรมการ
8.	ดร.สัตวแพทย์หญิงรัญชนา ยี่หวา	อนุกรรมการ
9.	ดร.วาสินี วงศ์คำมูล	อนุกรรมการ
10.	ดร.ลูก้า โล ปิคโคโล	อนุกรรมการเลขานุการ
11.	นางสาวณัฐสินีย์ อยู่อ่อน	อนุกรรมการและผู้ช่วยเลขานุการ

โดยให้คณะอนุกรรมการดังกล่าวมีหน้าที่

1. จัดเตรียมพิธีการ และพิธีกรตลอดการประชุม
2. ประสานงานด้านการนำเสนอในการบรรยายและโปสเตอร์ การเตรียมความพร้อม สื่อคอมพิวเตอร์และวิดีโอ
3. ดำเนินการประชุม จัดการเรื่องเวลาการนำเสนอ ป้ายชื่อวิทยากรและการมอบของที่ระลึก เกียรติบัตร รางวัล และโล่ประกาศเกียรติคุณ
4. ประสานงานกับกรรมการฝ่ายวิชาการ

คณะอนุกรรมการฝ่ายต้อนรับ

1.	ดร.ลูก้า โล ปิคโคโล	ประธานอนุกรรมการ
2.	รองศาสตราจารย์ ดร.นายแพทย์นราวุฒิ ภาคาพรต	รองประธานอนุกรรมการ
3.	ศาสตราจารย์ ดร.พันโทหญิงสุพิน ชมภูพงษ์	อนุกรรมการ
4.	ศาสตราจารย์ ดร.สุทิสา ถาน้อย	อนุกรรมการ
5.	ดร.สัตวแพทย์หญิงรัญชนา ยี่หวา	อนุกรรมการ
6.	อาจารย์ ดร.เภสัชกรหญิงสาลินี จันทราภิรมย์	อนุกรรมการเลขานุการ
7.	นางสาวณัฐสินีย์ อยู่อ่อน	อนุกรรมการและผู้ช่วยเลขานุการ

โดยให้คณะอนุกรรมการดังกล่าวมีหน้าที่

1. ต้อนรับวิทยากร แขกผู้มีเกียรติและผู้เข้าร่วมประชุม รวมถึงนักศึกษา IBRO-APRC Supported Associate School ทั้งในประเทศ และต่างประเทศ

-7-

2. กำกับดูแล การเดินทาง ที่พัก และให้คำแนะนำ ช่วยเหลือ ตอบข้อซักถาม วิทยากร แขกผู้มีเกียรติ และรวมถึงนักศึกษา IBRO-APRC Supported Associate School ทั้งในประเทศและต่างประเทศ
3. ประสานงานกับฝ่ายวิชาการและฝ่ายทะเบียน

คณะอนุกรรมการฝ่ายประชาสัมพันธ์

1. ดร.ลูก้า โล ปิคโคโล ประธานอนุกรรมการ
2. ผู้ช่วยศาสตราจารย์ ดร.มนัสพร พัสระ รองประธานอนุกรรมการ
3. ศาสตราจารย์ ดร.พันโทหญิงสุพิน ชมภูพงษ์ อนุกรรมการ
4. อาจารย์ ดร.เภสัชกรหญิงสาลินี จันทราภิรมย์ อนุกรรมการ
5. ดร.วาสินี วงศ์คำมูล อนุกรรมการ
6. ดร.สัตวแพทย์หญิงรัญชนา ยี่หวา อนุกรรมการและเลขานุการ
7. นางสาวณัฐสินีย์ อยู่อ่อน อนุกรรมการและผู้ช่วยเลขานุการ
8. นางสาวปัทชา ศึกษากิจ อนุกรรมการและผู้ช่วยเลขานุการ

โดยให้คณะอนุกรรมการดังกล่าวมีหน้าที่

- ประชาสัมพันธ์การประชุมวิชาการให้หน่วยงานทั้งภายใน และภายนอกมหาวิทยาลัยรับทราบ ผ่านทางรูปแบบต่าง ๆ เช่น ป้าย และเว็บไซต์ วางแผน จัดการและควบคุมเกี่ยวกับข้อมูล ที่ได้รับการลงทะเบียนผ่านทางเว็บไซต์ และจัดการด้านจดหมายอิเล็กทรอนิกส์

คณะอนุกรรมการฝ่ายลงทะเบียน

1. ผู้ช่วยศาสตราจารย์ ดร.ฤดีมาศ ยุบลพันธุ์ ประธานอนุกรรมการ
2. ผู้ช่วยศาสตราจารย์ ดร.นายแพทย์อดิศร รัตนโยธา รองประธานอนุกรรมการ
3. รองศาสตราจารย์ ดร.กุลธิดา ชัยธีระยานนท์ อนุกรรมการ
4. รองศาสตราจารย์ ดร.สุจิรา มุกดา อนุกรรมการ
5. ผู้ช่วยศาสตราจารย์ ดร.เภสัชกรหญิงจันทร์นภัสสร์ ธราวิจิตรกุล อนุกรรมการ
6. รองศาสตราจารย์ ดร.เภสัชกรหญิงรังษินี พงษ์ประดิษฐ อนุกรรมการ
7. อาจารย์ ดร.อัจฉราภรณ์ คำศรีใจ อนุกรรมการ
8. ดร.สัตวแพทย์หญิงรัญชนา ยี่หวา อนุกรรมการ
9. ดร.วาสินี วงศ์คำมูล อนุกรรมการ
10. อาจารย์ ดร.เภสัชกรหญิงสาลินี จันทราภิรมย์ อนุกรรมการและเลขานุการ
11. นางสาวณัฐสินีย์ อยู่อ่อน อนุกรรมการและผู้ช่วยเลขานุการ
12. นางสาวยุพเรศ กองนาค อนุกรรมการและผู้ช่วยเลขานุการ

โดยให้คณะอนุกรรมการดังกล่าวมีหน้าที่

1. ดำเนินการด้านการลงทะเบียนการประชุมผ่านทางเว็บไซต์
2. ประสานงานรายชื่อผู้เข้าร่วมสัมมนา จัดทำสรุปจำนวนรายชื่อผู้ร่วมการประชุม วิทยากร และจัดทำแฟ้มลงทะเบียนดำเนินการรับลงทะเบียนและแจกเอกสารผู้เข้าร่วมสัมมนาในวันสัมมนาวิชาการ

-8-

3. จัดเตรียมวัสดุ เอกสารลงทะเบียน ป้ายชื่อ และใบประกาศนียบัตรการเข้าร่วมการประชุม สำหรับผู้เข้าร่วมประชุมและวิทยากร
4. จัดทำรูปแบบประเมินโครงการ และสรุปการประเมินโครงการ
5. ประสานงานกับกรรมการฝ่ายวิชาการ

คณะอนุกรรมการฝ่ายสถานที่ และโสตทัศนูปกรณ์

1.	ดร.วาสินี วงศ์คำมูล	ประธานอนุกรรมการ
2.	ผู้ช่วยศาสตราจารย์ ดร.นายแพทย์อมรพันธุ์ เสรีมาศพันธุ์	รองประธานอนุกรรมการ
3.	รองศาสตราจารย์ ดร.นายแพทย์ชัยรัตน์ เติบไพบูลย์	อนุกรรมการ
4.	รองศาสตราจารย์ ดร.สุจิรา มุกดา	อนุกรรมการ
5.	ผู้ช่วยศาสตราจารย์ ดร.เภสัชกรหญิงจันทร์นภัสสร์ ธราวิจิตรกุล	อนุกรรมการ
6.	รองศาสตราจารย์ ดร.เภสัชกรหญิงรังษินี พงษ์ประดิษฐ	อนุกรรมการ
7.	อาจารย์ ดร.เภสัชกรหญิงสาลินี จันทราภิรมย์	อนุกรรมการ
8.	อาจารย์ ดร.ประทีป อมรรัตนพันธ์	อนุกรรมการ
9.	ผู้ช่วยศาสตราจารย์ ดร.ฤดีมาศ ยุบลพันธุ์	อนุกรรมการ
10.	ดร.สัตวแพทย์หญิงรัญชนา ยี่หวา	อนุกรรมการ
11.	ดร.ลูก้า โล ปิคโคโล	อนุกรรมการและเลขานุการ
12.	นางสาวปัทชา ศึกษากิจ	อนุกรรมการและผู้ช่วยเลขานุการ
13.	นางสาวฉันสุนีย์ ปานโต	อนุกรรมการและผู้ช่วยผู้ประสานงาน

โดยให้คณะอนุกรรมการดังกล่าวมีหน้าที่

1. จัดเตรียมสถานที่ประชุม และดูแลห้องประชุม
2. จัดเตรียม/ประสานงาน และดูแลโสตทัศนูปกรณ์ เช่น เครื่องเสียง เครื่องคอมพิวเตอร์ เครื่องโปรเจคเตอร์ และ Pointer
3. จัดเตรียมป้ายประชาสัมพันธ์สถานที่ประชุม
4. จัดหาอุปกรณ์ บอร์ด และควบคุมการติดตั้งสำหรับการนำเสนอผลงานในรูปแบบโปสเตอร์และบูธ
5. ประสานงานกับกรรมการฝ่ายวิชาการ และพิธีการ

คณะอนุกรรมการฝ่ายจัดเลี้ยงอาหาร

1.	ดร.สัตวแพทย์หญิงรัญชนา ยี่หวา	ประธานอนุกรรมการ
2.	ดร.วาสินี วงศ์คำมูล	อนุกรรมการ
3.	อาจารย์ ดร.เภสัชกรหญิงสาลินี จันทราภิรมย์	อนุกรรมการและเลขานุการ
4.	นางสาวณัฐสินีย์ อยู่อ่อน	อนุกรรมการและผู้ช่วยเลขานุการ
5.	นางสาวฉันสุนีย์ ปานโต	อนุกรรมการและผู้ช่วยผู้ประสานงาน

โดยให้คณะอนุกรรมการดังกล่าวมีหน้าที่

- ดูแลเรื่องการจัดเลี้ยงอาหารกลางวัน อาหารว่างและเครื่องดื่ม และการเลี้ยงต้อนรับ โดยประสานงานกับฝ่ายทะเบียน

-9-

คณะอนุกรรมการฝ่ายจัดหารายได้และการเงิน

1.	รองศาสตราจารย์ ดร.กุลธิดา ชัยธีระยานนท์	ประธานอนุกรรมการ
2.	ดร.สัตวแพทย์หญิงรัญชนา ยี่หวา	รองประธานอนุกรรมการ
3.	ศาสตราจารย์ ดร.พันโทหญิงสุพิน ชมภูพงษ์	อนุกรรมการ
4.	ศาสตราจารย์ ดร.สุขุมาล จงธรรมคุณ	อนุกรรมการ
5.	ผู้ช่วยศาสตราจารย์ ดร.ฤดีมาศ ยุบลพันธุ์	อนุกรรมการ
6.	ดร.วาสินี วงศ์คำมูล	อนุกรรมการ
7.	อาจารย์ ดร.เภสัชกรหญิงสาลินี จันทราภิรมย์	อนุกรรมการและเลขานุการ
8.	นางสาวปัทชา ศึกษากิจ	อนุกรรมการและผู้ช่วยเลขานุการ
9.	นางสาวรติกร แกมเงิน	อนุกรรมการและผู้ช่วยเลขานุการ
10.	นางสาวยุพเรศ กองนาค	อนุกรรมการและผู้ช่วยเลขานุการ

โดยให้คณะอนุกรรมการดังกล่าวมีหน้าที่

1. ดูแลงบประมาณ และการเบิกจ่ายให้กับคณะอนุกรรมการฝ่ายต่าง ๆ ทำบัญชีการเงินรายรับ - รายจ่ายของการประชุม รวมถึงการเก็บรวบรวม และตรวจสอบเอกสารประกอบการเบิกจ่าย
2. ทำเอกสารเพื่อหารายได้ และผู้สนับสนุนการจัดประชุม เพื่อการโฆษณาในหนังสือ และการออกร้านในงาน

ทั้งนี้ ตั้งแต่บัดนี้เป็นต้นไป

สั่ง ณ วันที่ 26 กันยายน พ.ศ. 2568

(ศาสตราจารย์ ดร.นายแพทย์พงษ์รักษ์ ศรีบัณฑิตมงคล)
อธิการบดีมหาวิทยาลัยเชียงใหม่

เอกสารฉบับนี้ใช้ลายมือชื่ออิเล็กทรอนิกส์ ตามพระราชบัญญัติ ว่าด้วยธุรกรรมทางอิเล็กทรอนิกส์ พ.ศ. 2544 และข้อบังคับมหาวิทยาลัยเชียงใหม่ ว่าด้วยการใช้ลายมือชื่ออิเล็กทรอนิกส์ พ.ศ. 2564" ส่งผ่านทางระบบ CMU e-Document ตามรหัสอ้างอิงเลขที่ B4607F-AE1-110

คำสั่งมหาวิทยาลัยเชียงใหม่

ที่ ๓๘๖๓ /2568

เรื่อง แต่งตั้งคณะกรรมการจัดการประชุมวิชาการนานาชาติด้านประสาทวิทยาศาสตร์
The 28th Annual Thai Neuroscience Society Conference 2025 (TNS28)
หัวข้อ Neuroplasticity Across the Lifespan เพิ่มเติม

....

ตามคำสั่งมหาวิทยาลัยเชียงใหม่ที่ 3547/2568 ลงวันที่ 28 กันยายน พ.ศ. 2568 ได้แต่งตั้งคณะกรรมการจัดการประชุมวิชาการนานาชาติด้านประสาทวิทยาศาสตร์ The 28th Annual Thai Neuroscience Society Conference 2025 (TNS28) หัวข้อ Neuroplasticity Across the Lifespan ไปแล้ว นั้น

เพื่อให้การดำเนินงานของคณะกรรมการจัดการประชุมวิชาการนานาชาติดังกล่าวเป็นไปด้วยความเรียบร้อย คล่องตัว และมีประสิทธิภาพ อาศัยอำนาจตามความในมาตรา 35 และมาตรา 38 แห่งพระราชบัญญัติมหาวิทยาลัยเชียงใหม่ พ.ศ. 2551 จึงแต่งตั้งคณะกรรมการจัดการประชุมวิชาการนานาชาติด้านประสาทวิทยาศาสตร์ The 28th Annual Thai Neuroscience Society Conference 2025 (TNS28) หัวข้อ Neuroplasticity Across the Lifespan เพิ่มเติม ดังมีรายนามต่อไปนี้

คณะกรรมการดำเนินการประชุมวิชาการ

1.	Professor Ingrid Liu	กรรมการ
2.	Professor Wael Mohamed	กรรมการ
3.	Dr. Arnaud Monteil	กรรมการ

ทั้งนี้ ให้คณะกรรมการดังกล่าวมีวาระการดำรงตำแหน่งและหน้าที่ตามคำสั่งมหาวิทยาลัยเชียงใหม่ที่ 3547/2568 ลงวันที่ 28 กันยายน พ.ศ. 2568 ตั้งแต่บัดนี้เป็นเป็นต้นไป

สั่ง ณ วันที่ 17 ตุลาคม พ.ศ. 2568

(ศาสตราจารย์ ดร.นายแพทย์พงษ์รักษ์ ศรีบัณฑิตมงคล)
อธิการบดีมหาวิทยาลัยเชียงใหม่

ที่ อว 8393(8).ศ.อม /178

ศูนย์บูรณาการเทคโนโลยีการแพทย์ทันสมัย
คณะแพทยศาสตร์ มหาวิทยาลัยเชียงใหม่
110 ถนนอินทวโรรส อำเภอเมืองเชียงใหม่
จังหวัดเชียงใหม่ 50200

18 มิถุนายน 2568

เรื่อง ขอเชิญเข้าร่วมงานประชุมวิชาการนานาชาติ **The 28th Annual Thai Neuroscience Society Conference 2025 (TNS28)**

เรียน อธิการบดี/คณบดี/หัวหน้าภาควิชา/ผู้อำนวยการ/ประธาน/เลขาธิการ/อาจารย์/นักวิจัย/นิสิต/นักศึกษา และสมาชิกสมาคมประสาทวิทยาศาสตร์ไทย

ด้วยศูนย์บูรณาการเทคโนโลยีการแพทย์ทันสมัย (CMUTEAM) คณะแพทยศาสตร์ มหาวิทยาลัยเชียงใหม่ คณะแพทยศาสตร์ มหาวิทยาลัยเชียงใหม่ ร่วมกับสมาคมประสาทวิทยาศาสตร์ไทย (Thai Neuro-science Society) ได้กำหนดการจัดงานประชุมวิชาการนานาชาติ **"The 28th Annual Thai Neuroscience Society Conference 2025 (TNS28)"** ภายใต้หัวข้อ "Neuroplasticity Across the Lifespan: Advancing Neuroplasticity Research Through Cutting- Edge Methodologies" **ระหว่างวันที่ 29 - 31 ตุลาคม พ.ศ. 2568 ณ โรงแรมแคนทารี ฮิลส์ เชียงใหม่** โดยมีวัตถุประสงค์เพื่อเป็นเวทีแลกเปลี่ยนองค์ความรู้สร้างเครือข่ายวิจัยจากผู้เชี่ยวชาญทั้ง ในประเทศไทยและในระดับนานาชาติจากหลากหลายองค์กรและเพื่อประโยชน์สำคัญในการพัฒนาความร่วมมือการรักษา โรคในระบบประสาทและสมองที่จะเกิดขึ้นในอนาคต โดยใช้เวทีการประชุมเป็นสื่อกลางในการเผยแพร่งานวิจัย และสร้าง เครือข่ายความร่วมมือเพื่อพัฒนางานวิจัยเชิงบูรณาการในประเทศไทยให้กว้างขวางยิ่งขึ้น นั้น

ในการจัดประชุมครั้งนี้ คณะผู้จัดการประชุมฯได้เชิญวิทยากรผู้มีชื่อเสียงมาเป็นผู้บรรยาย และเปิดโอกาสให้ ผู้เข้าร่วมประชุมสามารถนำเสนอผลงานวิจัย รวมถึงการเข้าร่วมกิจกรรม workshop จากผู้เชี่ยวชาญทางระบบประสาทและสมองโดยผู้ที่มีความสนใจสามารถดูรายละเอียดเพิ่มเติมที่เกี่ยวข้องพร้อมกำหนดการของงานประชุมวิชาการได้ทางเว็บไซต์ https://thaineuroscience.org/conference/tns28/ หากต้องการสอบถามข้อมูลเพิ่มเติม กรุณาติดต่อทางอีเมลที่ tns28.info@gmail.com ทั้งนี้ค่าลงทะเบียนและค่าใช้จ่ายต่าง ๆ สามารถเบิกจ่ายได้ตามระเบียบของทางกระทรวง การคลัง และผู้เข้าร่วมประชุมสามารถเข้าประชุมได้โดยถือไม่เป็นวันลาเมื่อได้รับการอนุมัติจาก ผู้บังคับบัญชา ผู้เข้าร่วมโครงการสามารถเข้าร่วมได้โดยถือไม่เป็นวันลา เมื่อได้รับการอนุมัติจากผู้บังคับบัญชา

ศูนย์บูรณาการเทคโนโลยีการแพทย์ทันสมัย (CMUTEAM) คณะแพทยศาสตร์ มหาวิทยาลัยเชียงใหม่
E-mail : cmuteam-med@cmu.ac.th โทรศัพท์ : 0 5393 4673

จึงเรียนมาเพื่อโปรดพิจารณาและหวังเป็นอย่างยิ่งว่าจะได้รับความอนุเคราะห์จากท่าน ขอขอบพระคุณเป็นอย่างสูงมา ณ โอกาสนี้

ขอแสดงความนับถือ

(ศ.ดร.นพ. ดำเนินสันต์ พฤกษากร)
ประธานกรรมการดำเนินการศูนย์ฯ

ศูนย์บูรณาการเทคโนโลยีการแพทย์ทันสมัย (CMUTEAM) คณะแพทยศาสตร์ มหาวิทยาลัยเชียงใหม่
E-mail : cmuteam-med@cmu.ac.th โทรศัพท์ : 0 5393 4673

No. MHESI 8393(8).C.OMIC/178

Center of Multidisciplinary
Technology for Advanced Medicine
(CMUTEAM), Faculty of Medicine,
Chiang Mai University, Chiang Mai,
Thailand

18th June 2025

Subject Invitation to attend the IBRO-APRC Supported Associate School

Dear Chanceller / Dean / Head of Department / Director / President / Secretary / Professor / Researcher / Student and Member of Thai Neuroscience Society

On behalf of the organizing committee, it is our great pleasure to invite you to "IBRO-APRC Supported Associate School" under the theme "Winter School on Neuroplasticity Across the Lifespan: Development, Aging, and Neurodegeneration", to be held at the 50th Anniversary Building, Faculty of Medicine, Chiang Mai University, Chiang Mai, Thailand, during 27th to 2nd November 2025. This academic activity will be conducted in a dual program format, combining both lecture and hands-on workshop training sessions, as well as the 28th Annual Thai Neuroscience Society Conference 2025. Renowned professors and neuroscience experts from Thailand and abroad will participate extensively in this program. The main objective is to provide Master's students, Ph.D. candidates, postdoctoral researchers, and interested medical professionals with opportunities to develop and enhance their research skills in neurobiology.

This academic service project presents a valuable opportunity for students, researchers, faculty members, and medical professionals to further their knowledge and research capabilities, while also fostering academic collaboration. Such engagement is expected to contribute significantly to the advancement of professional research and future education. For more details, interested individuals can visit the official website: https://ibro.org/training-opportunity/winter-school-on-neuroplasticity-across-the-lifespan/. For inquiries, please contact us via email at tns28.info@gmail.com. Participation in the event will not be considered as leave if prior approval is obtained from the supervisor.

We would like to extend our invitation to you and your colleagues to consider attending this academic event. Your participation would be greatly appreciated.

Kind regards,

(Prof. Dumnoensun Pruksakorn, M.D., PhD)
Chairman of the Center of Multidisciplinary
Technology for Advanced Medicine

Center of Multidisciplinary Technology
for Advanced Medicine (CMUTEAM)
E-mail: cmuteam-med@cmu.ac.th
Tel. (+66) 53 934 673

ที่ อว 8393(8).ศ.อม /178

ศูนย์บูรณาการเทคโนโลยีการแพทย์ทันสมัย
คณะแพทยศาสตร์ มหาวิทยาลัยเชียงใหม่
110 ถนนอินทวโรรส อำเภอเมืองเชียงใหม่
จังหวัดเชียงใหม่ 50200

18 มิถุนายน 2568

เรื่อง ขอเชิญเข้าร่วม “IBRO-APRC Supported Associate School”

เรียน อธิการบดี/คณบดี/หัวหน้าภาควิชา/ผู้อำนวยการ/ประธาน/เลขาธิการ/อาจารย์/นักวิจัย/นิสิต/นักศึกษา และสมาชิกสมาคมประสาทวิทยาศาสตร์ไทย

ด้วยศูนย์บูรณาการเทคโนโลยีการแพทย์ทันสมัย (CMUTEAM) คณะแพทยศาสตร์ มหาวิทยาลัยเชียงใหม่ คณะแพทยศาสตร์ มหาวิทยาลัยเชียงใหม่ ร่วมกับองค์กรระหว่างประเทศ International Brain Research Organization (IBRO) และ สมาคมประสาทวิทยาศาสตร์ไทย ได้กำหนดจัดกิจกรรมฝึกอบรมและงานวิชาการระดับนานาชาติ **“IBRO-APRC Supported Associate School”** ภายใต้หัวข้อเรื่อง “Winter School on Neuroplasticity Across the Lifespan: Development, Aging, and Neurodegeneration” **ระหว่างวันที่ 27 ตุลาคม - 2 พฤศจิกายน พ.ศ. 2568 ณ อาคารเรียนและปฏิบัติการ 50 ปี คณะแพทยศาสตร์ มหาวิทยาลัยเชียงใหม่** เพื่อจัดกิจกรรมวิชาการในรูปแบบ dual program ที่ผสมผสานระหว่างการจัดกิจกรรมฝึกอบรม (lecture and hands-on workshop) และงานประชุมวิชาการประจำปีของทางสมาคมประสาทวิทยาศาสตร์ไทย ครั้งที่ 28 (The 28th TNS Annual Thai Neuroscience Society Conference 2025) โดยมีคณาจารย์และผู้เชี่ยวชาญด้านระบบประสาทและสมองจากในและต่างประเทศเข้าร่วมอย่างมากมาย โดยโครงการนี้มุ่งเน้นให้นักศึกษาปริญญาโท นักศึกษาปริญญาเอก นักวิจัยหลังปริญญาเอก และแพทย์ผู้สนใจ ได้ฝึกฝนพัฒนาทักษะการวิจัยทางชีววิทยาของระบบประสาท

ดังนั้นกิจกรรมฝึกอบรมวิชาการนี้ จึงเป็นโอกาสสำคัญสำหรับนักศึกษา นักวิจัย อาจารย์ และแพทย์ผู้ที่สนใจ ในการพัฒนาความรู้ทักษะทางการวิจัย และการสร้างความร่วมมือทางการวิจัย เพื่อสร้างเสริมความก้าวหน้าในวิชาชีพวิจัย และการเรียนการสอนในอนาคต โดยผู้ที่มีความสนใจสามารถตรวจสอบรายละเอียดเพิ่มเติมได้ทางเว็บไซต์ https://ibro.org/training-opportunity/winter-school-on-neuroplasticity-across-the-lifespan/ หากต้องการสอบถามข้อมูลเพิ่มเติม กรุณาติดต่อทางอีเมลที่ tns28.info@gmail.com ทั้งนี้ในส่วนของค่าลงทะเบียนและค่าใช้จ่ายต่าง ๆ สามารถเบิกจ่ายได้ตามระเบียบกระทรวงการคลัง และผู้เข้าร่วมโครงการสามารถเข้าร่วมได้โดยถือไม่เป็นวันลา เมื่อได้รับการอนุมัติจากผู้บังคับบัญชา

ศูนย์บูรณาการเทคโนโลยีการแพทย์ทันสมัย (CMUTEAM) คณะแพทยศาสตร์ มหาวิทยาลัยเชียงใหม่
E-mail : cmuteam-med@cmu.ac.th โทรศัพท์ : 0 5393 4673

จึงเรียนมาเพื่อโปรดพิจารณาและหวังเป็นอย่างยิ่งว่าจะได้รับความอนุเคราะห์จากท่าน ขอขอบพระคุณเป็นอย่างสูงมา ณ โอกาสนี้

ขอแสดงความนับถือ

(ศ.ดร.นพ. ดำเนินสันต์ พฤกษากร)
ประธานกรรมการดำเนินการศูนย์ฯ

ศูนย์บูรณาการเทคโนโลยีการแพทย์ทันสมัย (CMUTEAM) คณะแพทยศาสตร์ มหาวิทยาลัยเชียงใหม่
E-mail : cmuteam-med@cmu.ac.th โทรศัพท์ : 0 5393 4673

Announcement of the TNS Neuro Quiz 2024 Winners

We are pleased to announce the winners of the **TNS Neuro Quiz 2024**, a competition that aims to inspire curiosity and deepen understanding in the field of neuroscience among students nationwide. The event brought together outstanding teams who demonstrated exceptional knowledge, teamwork, and critical thinking throughout the quiz.

The winners of this year's competition are as follows:

(1) **First Prize:** Suksanari School
Team members: Pattaranun Saepung, Banthita Mingein, Nartlada Vorrarat
Coach: Pantita Kannika

Receives the TNS Neuro-Quiz Award Trophy, Champion Award Certificate, and a cash prize of 30,000 Baht, plus an additional sponsorship prize of 10,000 Baht.

(2) **Second Prize:** Triam Udom Suksa Nomklao School
Team members: Nattapat Chomcheay, Krittapas Kittipittayakul, Thepasit Aimkoed
Coach: Nattharinee Wongkongkum

Receives an Award Certificate and an additional sponsorship prize of 5,000 Baht.

(3) **Third Prize:** Kasetsart University Laboratory School, Kamphaeng Saen Campus, Educational Researchand Development Center
Team members: Supakorn Chornjai, Thianrawich Thongthate, Pakonkiat Pongsroy
Coach: Jamari Lueangrungsap

Receives an Award Certificate and a cash prize of 3,000 Baht.

Congratulations to all the winners and participants for their remarkable performance and enthusiasm in advancing neuroscience education!

Announcement of the TNS27 Academic Achievement Awards

We are delighted to announce the winners of the **TNS27 Academic Achievement Awards** for **Outstanding Poster Presentations**. This year, a total of **8 awards** were presented to recognize excellence in research, creativity, and scientific communication. The awardees are as follows:

(1) **Professor Prasop Ratanakorn Award "Outstanding Research Achievement"**
Receives a **Certificate** and a **cash prize of 4,000 Baht**
Awardee: *Phongthon Kanjanasirirat*

P-45: GDNF and cAMP Enhance In Vitro Blood-Brain Barrier Integrity in a Humanized Tricellular Transwell Model. Phongthon Kanjanasirirat, Witchuda Saengsawang, Pimonrat Ketsawasomkron, Nithi Asavapanumas, Sitthivut Charoensutthivarakul, Suradej Hongeng

(2) **Professor Roongtam Ladpli Award**
Receives an **Award Certificate** and a **cash prize of 2,000 Baht**
Awardee: *Yuanyuan Zhao*

P-48: A Study of Lipid Regulation of Voltage-Gated K+ Channel Protein using the Caged Amino Acid System. Yuanyuan Zhao, Okamura Yasushi, Sompol Tapechum, Yoshioka Daisuke

(3) **Associate Professor Naipinich Kotchabhakdi Award**
Receives an **Award Certificate** and a **cash prize of 2,000 Baht**
Awardee: *Tanya Prasertporn*

P-04: Differential Proteomic Analysis of the Toll-Like Receptor 4 DepletedAstrocytes under Ischemic/Reperfusion Conditions. Tanya Prasertporn, Jiraporn Panmanee, Alisa Tubsuwan, Kornkanok Promthep, Sujira Mukda

(4) **TNS Best Poster Award** *(3 Awards)*
Each receives an **Award Certificate** and a **cash prize of 2,000 Baht**
Awardees:

(4.1) Richmond Arthur

P-03: Artemisinin Mitigates Huntington's Disease-Like Symptoms in Rats: Elucidating Its Neuroprotective Mechanism through Antioxidant and AntiInflammatory Pathways. Richmond Arthur, Uma Shanker Navik, Puneet Kumar

(4.2) Shubham Upadhayay

P-36: Raloxifene and Fulvestrant Provide Neuroprotection against Haloperidol-Induced Neurotoxicity In-Vitro and In-Vivo Model through Activation of Nrf2/HO-1 Pathway. Shubham Upadhayay, Puneet Kumar

(4.3) Kamonrapat Sompub

P-44: Analysis of the NF-țB Pathway Inactivation on Oligodendrocyte Differentiation in the Central Nervous System. Kamonrapat Sompub, Norihisa Bizen, Hirohide Takebayashi

(5) **Professor Ingrid Liu Award**
Receives an **Award Certificate** and a **cash prize of 2,000 Baht**
Awardee: *Wongsakorn Siripan*

P-11: Andrographis Paniculata Extract Affected Liver and Neuronal Developing Changes in Chick Embryo. Wongsakorn Siripan, Pornkanok Nimnoi, Chairat Turbpaiboon, Boonrat Tassaneetrithep, Benjaporn Homkajorn Songvasin, Tawit Suriyo, Supin Chompoopong

(6) **Best Imaging Technology Award**
Receives an **Award Certificate** and a **cash prize of 2,000 Baht**
Awardee: *Cheng-Wei Lim*

P-13: In Vitro Cytotoxicity Assessment of Ruxolitinib on Oligodendrocyte Precursor Cell (OPC) and Neural Stem/Progenitor Cell (NSPC). Cheng-Wei Lim, Gen Hamanaka, Anna C. Liang, Su Jing Chan, King-Hwa Ling, Eng H. Lo, Ken Arai, Pike-See Cheah

Congratulations to all award recipients for their outstanding contributions and dedication to advancing neuroscience research and innovation.

Announcement of the Thai Neuroscience Society (TNS)
Subject: Awardees of TNS Member Travel Award for FAONS Congress 2024

The Thai Neuroscience Society (TNS) is pleased to announce the selection of the Awardees for the TNS Member Travel Award for the upcoming FAONS Congress 2024. The Award Committee has carefully reviewed all submissions and finalized the list of Awardees:

Name of Applicant	TNS Membership	Title of Abstract
1. Rungsarit Sunan	240	**Alleviate Neuroinflammatory Cytokines after *Halymenia Durvillei* Extract Treatment of Vascular Cognitive Impairment in Mice**
2. Darunee Rodma	299	**Decreased in Alpha Synuclein after *Caulerpa Lentillifera* Treatment in MPP^+ Induced Parkinson's Like Model**
3. Danuyada Wattanaumadechakul	241	**Modulated Microglia Activation and Cholinergic Dysfunction after Color Rice Treatment in Mice with Mild Cerebral Hypoperfusion**

Congratulations to the selected Awardees!
To complete the scholarship award process, Awardees are required to submit an acceptance letter for presenting at the FAONS Congress 2024. Additionally, please ensure compliance with the Award Information, Requirements, and Notification outlined in the Announcement of the Thai Neuroscience Society (TNS) regarding the TNS Member Travel Award for FAONS Congress 2024, issued on January 5, 2024.

This announcement is made for public acknowledgement.

Announced on April 22, 2024

(Emeritus Prof. Dr. Sukumal Chongthammakun)
President of the Thai Neuroscience Society

For further queries please contact Assoc. Prof. Dr. Nopporn Jongkamonwiwat, TNS Secretary, Email: secretary.thaineurosci@gmail.com
President Office: Faculty of Science, Mahidol University, 272 Rama VI Road, Ratchathewi, Bangkok 10400
TNS Office: Department of Pharmacology and Physiology, Faculty of Pharmaceutical Sciences, Chulalongkorn University, Phayathai Road, Patumwan, Bangkok 10330 Tel.+662-218 8342 Fax. +662-218 8340, Website: www.thaineuroscience.org