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10th Mind-Body Interface International Symposium

PNIRASia-Pacific Symposium | ISNPR Regional Meeting

第十屆身心介面國際研討會暨中國醫藥大學附設醫院40週年院慶國際學術研討會

心智迷宮：早期生命經驗對成年身心健康的影響



The Mind Maze

Early Life Experiences That Shape Adult Health of Mind and Body

Free Web Conference 免費線上會議

Oct. 30 – Nov. 1, 2020

Taichung & Tainan, TAIWAN



CONTENT

PROGRAMME OVERVIEW	2
PROGRAMME DETAILS	3
MODERATOR & SPEAKER	7
OPENING REMARKS.....	7
PK1. PLENARY KEYNOTE SPEECH.....	8
S11. PNIRASIA-PACIFIC SYMPOSIA (I).....	9
PK2. PLENARY KEYNOTE SPEECH.....	12
PK3. PLENARY KEYNOTE SPEECH.....	13
S22. PNIRASIA-PACIFIC SYMPOSIA (II).....	14
S22. ISNPR SESSION	17
S23. POSTER BLITZ	19
AWARD CEREMONY.....	19
PK4. PLENARY KEYNOTE SPEECH.....	20
S31. MIND-BODY SESSION	21
COLSING REMARKS.....	24
VIRTUAL POSTER PRESENTATION	25

PROGRAMME OVERVIEW

	(FRI) Oct. 30	(SAT) Oct. 31	(SUN) Nov. 1
	CMUH@Taichung (on-site)	CMUH@Taichung (web) NCKU@Tainan (on-site)	CMUH@Taichung (web) NCKU@Tainan (on-site)
0830-0900		REGISTRATION	REGISTRATION
0900-0950		PK3. Plenary Keynote Michael Irwin	PK4. Plenary Keynote Angelos Halaris
0950-1200		S21. PNIRASia-Pacific (II) Keith Kelley Teresa M. Reyes Sarah J. Spencer Elisa Hill-Yardin Michael Dragunow	S31. Mind-Body Session Long-Jun Wu Che-Sheng Chu Tien-Yu Chen Cheng-Hao Tu
			CLOSING
1200-1300	REGISTRATION	LUNCH	
1300-1330	OPENING CEREMONY & GROUP PHOTO		
1330-1420	PK1. Plenary Keynote Jane Pei-Chen Chang	S22. ISNPR Session Wolfgang Marx Scott Teasdale Joseph Firth Philip B. Ward	
1420-1550	S11. PNIRASia-Pacific (I) Annabelle Réaux-Le Goazigo Jan Pieter Konsman Sanae Hasegawa-Ishii		
1550-1610	BREAK	BREAK	S23. Poster Blitz
1610-1700	PK2. Plenary Keynote Hsing-Chang Ni	AWARD CEREMONY	

1. PK: Plenary Keynote; S: Session

2. PNIRS: Psychoneuroimmunology Research Society

3. ISNPR: International Society for Nutritional Psychiatry Research

4. CMUH: Lecture Room, 1st Floor, Cancer Center of China Medical University Hospital, Taichung 中醫大附設醫院癌症中心一樓階梯教室

5. NCKU= 4th Floor of College of Medicine, National Cheng Kung University, Tainan 成功大學醫學院四樓

PROGRAMME DETAILS

(Fri) Oct. 30, 2020

CMUH@Taichung (on-site)

1200-1300	REGISTRATION
1300-1330	OPENING CEREMONY & GROUP PHOTO <i>CHAIR: Mien-Chie Hung & Kuan Pin Su</i>
1330-1420	PK1. Child Mental Health in the 21st Century: Nurture and Nutrition in Developmental Neurosciences <i>SPEAKER: Jane Pei-Chen Chang, TAIWAN</i> <i>MODERATOR: Kuan-Pin Su</i>
	S11. PNIRASia-Pacific Session (I) Psychoneuroimmunology Approaches That Shape Health <i>MODERATOR: Jane Pei-Chen Chang</i>
1420-1445	How does chronic dry eye shape peripheral and central nervous systems? <i>Annabelle Réaux-Le Goazigo, FRANCE</i>
1445-1510	Cognitive deficits and changes in cytokine expression in a mouse model of childhood leukemia <i>Jan Pieter Konsman, FRANCE</i>
1510-1535	Changes in the brain morphology and microenvironment caused by nasal inflammation <i>Sanae Hasegawa-Ishii, JAPAN</i>
1535-1550	Discussion
1550-1610	BREAK
1610-1700	PK 2. Application of Repetitive Transcranial Magnetic Stimulation and Theta Burst Stimulation in Autism Spectrum Disorder <i>SPEAKER: Hsing-Chang Ni, TAIWAN</i> <i>MODERATOR: Jane Pei-Chen Chang</i>

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0830-0900 **REGISTRATION**

0900-0950 **PK3. Tai Chi and co-regulation of sleep, antiviral Immunity, and inflammation in older adults**
SPEAKER: Michael Irwin, USA
MODERATOR: Kuan-Pin Su

S21. PNIRASia-Pacific Session (II)
Psychoneuroimmunology Approaches That Shape Health
MODERATOR: Keith Kelley

0950-1000 Psychoneuroimmunology approaches that shape health
Keith Kelley, USA

1000-1025 Early life diet can shape the development of executive function deficits in mice: examination of prefrontal cortical involvement
Teresa M. Reyes, USA

1025-1050 Sex differences in early life dietary programming of obesity and stress
Sarah J. Spencer, AUSTRALIA

1050-1115 The gut-brain interface in neurodevelopmental disorders
Elisa Hill-Yardin, AUSTRALIA

1115-1140 Targeting Brain Barrier Cells for Regulating Brain Inflammation
Michael Dragunow, NEW ZEALAND

1140-1200 Discussion

1200-1330 **LUNCH**

S22. ISNPR Session
CHAIR & MODERATOR: Phillip B. Ward & Jane Pei-Chen Chang

1330-1355 Diet and depression: exploring the biological mechanisms of action
Wolfgang Marx, AUSTRALIA

1355-1420 Nutritional psychiatry in psychotic disorders: current hypotheses and research challenges
Scott Teasdale, AUSTRALIA

1420-1445 Protecting physical health in people with mental illness: findings from the Lancet Psychiatry Commission
Joseph Firth, UK

1445-1510 Taking it to the street: real-world implementation of lifestyle interventions for people living with mental illness
Philip B. Ward, AUSTRALIA

1510-1530 Discussion

1530-1600 **BREAK**

S23. Poster Blitz
MODERATOR: Jane Pei-Chen Chang

- 1600-1605 The serotonin transporter is associated with increased risk of suicide attempts in young adults with a family history of domestic violence
Shui Jiang, CANADA
- 1606-1611 Genetic variations of ionotropic glutamate receptor pathways on interferon- α -induced depression in patients with hepatitis C viral infection
Szu-Wei Cheng, TAIWAN
- 1612-1617 Plasma D-glutamate levels for detecting mild cognitive impairment and Alzheimer's disease: Machine learning approaches
Chun-Hung Chang, TAIWAN
- 1618-1623 Genome-wide DNA methylation signatures of resilience in young adults
Andrew Ke-Ming Lu, TAIWAN
- 1624-1629 Antidepressants treatment regulates the neurotransmitter levels and attenuate apoptosis induced by arecoline oxidative metabolite
Srinivasan Nithiyantham, TAIWAN

1630-1700 **AWARD CEREMONY**
CHAIR: Jane Pei-Chen Chang

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0830-0900	REGISTRATION
0900-0950	PK4. Heart Rate Variability: A Window into the Mind-Body Connection <i>SPEAKER: Angelos Halaris, USA</i> <i>MODERATOR: Kuan-Pin Su</i>
	S31. Mind-Body Session <i>MODERATOR: Kuan-Pin Su & Cheng-Hao Tu</i>
0950-1015	Microglia-astrocyte interaction in a mouse model of neuromyelitis optica <i>Long-Jun Wu, USA</i>
1015-1040	Non-invasive brain stimulation for mild cognitive impairment and Alzheimer's disease: from investigation to therapeutic application <i>Che-Sheng Chu, TAIWAN</i>
1040-1105	Clinical application of cranial electrotherapy stimulation for the treatment of generalized anxiety disorder in Taiwan <i>Tien-Yu Chen, TAIWAN</i>
1105-1130	Inverse correlation between omega-3 concentration and emotional brain response on major depression <i>Cheng-Hao Tu, TAIWAN</i>
1130-1150	Discussion
1150-1200	CLOSING <i>CHAIR: Kuan Pin Su</i>

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MODERATOR & SPEAKER

OPENING REMARKS

TIME 13:00-13:30, Fri 30 Oct. 2020

VENUE CMUH@Taichung (on-site)

CHAIRS:

Mien-Chie Hung, PhD

President, China Medical University and Academician, Academia Sinica, Taiwan

Kuan Pin Su, MD, PhD

Professor College of Medicine, China Medical University (CMU), Taiwan

Vice President, Tainan Municipal An-Nan Hospital-affiliated with China Medical University, Taiwan

Director, Mind-Body Interface Research Centre, China Medical University Hospital, Taiwan

Honorary Professor of Institute of Psychiatry-King's College London, UK

PK1. PLENARY KEYNOTE SPEECH

TIME 13:30-14:20, Fri, Oct. 30 2020

VENUE CMUH@Taichung (on-site)

Moderator: Kuan-Pin Su, MD, PhD

Professor College of Medicine, China Medical University (CMU), Taiwan

Vice President, Tainan Municipal An-Nan Hospital-affiliated with China Medical University, Taiwan

Director, Mind-Body Interface Research Centre, China Medical University Hospital, Taiwan

Honorary Professor of Institute of Psychiatry-King's College London, UK

Child Mental Health in the 21st Century: Nurture and Nutrition in Developmental Neurosciences

Jane Pei-Chen Chang, MD, PhD

Chief, Child Psychiatry Division, Department of Psychiatry, China Medical University Hospital, Taiwan

Visiting Researcher, Department of Psychological Medicine, Institute of Psychiatry, Psychology and Neuroscience (IoPPN), King's College London, UK

Due to the advances in technology, child mental health in the 21st Century is full of challenges and opportunities. No one in the 1980s would have ever imagined that in 2020, child obesity, gaming disorder, loneliness and depression would be the keywords when it comes to child mental health. Even as we are tackling these problems, 2020 was a hard hit by Covid-19, where social distancing, working from home, wearing personal protective gear became the norm. However, it is because of these challenges, we were able to find new solutions and strategies to overcome the obstacles. Nurture has long been agreed by most as one of the crucial building blocks in the shaping of childhood experiences and even the outcome of both mental and physical health in adults. However, nutrition, equally as important, has not been given enough attention in the shaping of the child's mental health until recent times. In this lecture, I will provide a review of the recent cutting-edge nutritional studies in child mental health, with a special focus on personalised medicine.

S11. PNIRSASIA-PACIFIC SYMPOSIA (I)

TIME 14:20-15:50, Fri, Oct. 30 2020

VENUE CMUH@Taichung (on-site)

Psychoneuroimmunology Approaches That Shape Health (I)

Chair: Keith Kelley, PhD

*Professor Emeritus of Immunophysiology University of Illinois
Editor-in-Chief Emeritus Brain, Behavior, and Immunity*

Moderator: Jane Pei-Chen Chang, MD, PhD

*Chief, Child Psychiatry Division, Department of Psychiatry, China Medical University Hospital, Taiwan
Visiting Researcher, Department of Psychological Medicine, Institute of Psychiatry, Psychology and Neuroscience (IoPPN), King's College London, UK*

The idea that there are reciprocal systems of communication between the immune and central nervous systems began to gain traction in the 1980s. The first issue of *Brain, Behavior, and Immunity* appeared on March 1, 1987 and on January 1, 2000, it became the official journal of the Psychoneuroimmunology Research Society (PNIRS). These early developments made it clear that the brain and immune system can no longer be considered to be captains of their own ship. Today the focus is on mediators and mechanisms of this cross-talk and their potential importance for animal and human health. This PNIRASIA-Pacific symposium features seven experts from five countries who will highlight their most recent advances in psychoneuroimmunology research. Of growing prominence is the influence of gender differences on health, particularly during developmental and early life periods. Diet has emerged as an important component. New results will be reported that have identified differential epigenetic regulation of higher order executive functions in male and female mice following maternal malnutrition (Reyes). Overfeeding during the neonatal period also affects a number of hypothalamic neuropeptides that are differentially regulated by sex (Spencer). Similar sex differences are observed in mice being treated for cancer. Consistent with these results, new data show that a standard chemotherapy regimen causes working memory deficits in male but not female mice (Konsman). One of the major emerging research areas is the role of the gut microbiome in health. Although gut-associated lymphoid tissue communicates with the gut intrinsic nervous system, little is known about its potential role in development and early life. Mice expressing the autism-associated Neuroligin-3 R451C mutation at the synapse were used to explore this neural-immune gut communication system in neurodevelopmental disorders (Hill-Yardin). Novel data will then be presented on the role of inflammation in two major sensory systems. Chronic dry eye disease is linked to neuronal, glial and immune cell activation in the trigeminal ganglion. This leads to central nociceptive reorganization, dry eye pain and induction of anxiety-like behaviors (Reaux-Le Goazigo). Another major sensory system, the olfactory mucosa, has now been shown to also be sensitive to nasal inflammation. Activation of TLR-4 receptors in the nasal cavity of mice leads to activation of microglia in the olfactory bulb, secretion of pro- and anti-inflammatory cytokines and the subsequent reversible atrophy of olfactory sensory neurons (Hasegawa-Ishii). Although microglia have predominantly been studied in neuroinflammation, new data will be presented showing there are other important actors. Endothelial cells, pericytes, the meninges and choroid plexus secrete both chemokines and intercellular adhesion molecules. These cells have been isolated from humans and cultured with inflammatory stimuli in vitro. New experiments have now established that drugs that are already approved by the FDA regulate the synthesis and secretion of MCP-1, ICAM-1 and VCAM (Dragunow). These new data not only reinforce the concept of reciprocal communication pathways between the immune system and brain but further establish the multi-faceted roles of inflammation in shaping animal and human health from conception to adulthood.

TIME: 14:20-14:45 Session Speaker

How Does Chronic Dry Eye Shape Peripheral and Central Nervous Systems?

Annabelle Réaux-Le Goazigo, PhD

INSERM Senior Researcher (Group Leader) at the Vision Institute, Sorbonne University, 17, rue Moreau, F-75012 Paris, France

Dry eye disease (DED) is a major public health problem in ophthalmology. DED is a multifactorial disease in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities play etiological roles. In the last decade, the relationship between DED and psychiatric disorders has been also gaining attention. Although DED is a common disorder that affects the quality of life of millions people worldwide, the cellular and molecular mechanisms of the disease are not fully understood. A better understanding of these mechanisms participating in the transition from acute to chronic dry eye pain are crucial issues for developing the effective management and a therapeutic strategy to alleviate this debilitating condition.

This presentation provides novel information about the main (cellular and molecular) actors that shape the peripheral and central nervous systems in a novel preclinical model of persistent dry eye that mimics the human disease. We recently reported pro inflammatory gene expression, activation of neuronal, glial and immune cells in the trigeminal ganglion confirming neuroimmune and neuronal–glial interactions during dry eye pain. Bidirectional neuron–glia interactions also affect corneal processing and transmission of ocular pain leading to a reorganization of the peripheral and central nociceptive systems. Indeed, we have found that corneal inflammation and nerve damage modify the characteristics of trigeminal primary sensory neurons, resulting in their hyperexcitability. This sustained ongoing activity of corneal neurons then triggers neuroinflammatory responses and presynaptic rearrangements in the central trigeminal brainstem. In addition, still recent evidence from our group demonstrates that persistent dry eye induced anxiety-like behaviours and neuronal changes in central structures linked to the emotional affective aspects of pain. Further fundamental and clinical functional and morphological imaging studies will help to depict how chronic dry eye pain does shape the brain. Altogether, a better understanding of the sequence and nature of the neuro immune events that drive these neurobiological mechanisms will offer significant promise for the discovery of new approaches and targets for the management of dry eye pain.

TIME: 14:45-15:10 Session Speaker

Cognitive Deficits and Changes in Cytokine Expression in A Mouse Model of Childhood Leukemia

Jan Pieter Konsman, PhD

Senior scientist at National Center for Scientific Research and University of Bordeaux, France

Survivors of childhood acute lymphoblastic leukemia (ALL) are at an increased risk for cognitive deficits, as chemotherapy has adverse effects on the brain. Children are particularly vulnerable because the prefrontal cortex (PFC), largely responsible for higher cognitive function, is still developing. To investigate potential mechanisms leading to cognitive deficits, we have developed a model of juvenile leukemia survival. Male and female mice were injected with L1210 leukemic cells at P19. Starting at P21, mice received a standard chemotherapy regimen consisting of 4 cycles of methotrexate, vincristine, and leucovorin. Cognitive deficits were assessed using social recognition, novel object, and operant behavioral tests. Using RT-qPCR, PFC was analyzed for changes in immune and plasticity factors, while intestinal tissue was analyzed for markers of inflammation and gut integrity. Male, but not female, survivors showed working memory deficits, and an increase in CCL2 and IL-1beta mRNA (acutely, 1-5 days post chemo) in PFC that normalized by adulthood. Inflammatory genes were not altered in female PFC, however they showed a persistent upregulation of PSD-95 and synaptophysin. In the small intestine, male survivors showed an elevation in IL-1β and

TNF-alpha acutely, and this persisted into adulthood, a response that was absent in female survivors. The current studies establish a foundation for testing potential therapeutic approaches to protect the juvenile brain during chemotherapy.

TIME: 15:10-15:35 Session Speaker

Changes in the Brain Morphology and Microenvironment Caused by Nasal Inflammation

Sanae Hasegawa-Ishii, PhD

Associate professor, Kyorin University, Japan

Background: The olfactory mucosa (OM) is exposed to a variety of environmental toxins such as bacteria, viruses, molds, pollen, dust, and environmental chemicals. These toxins cause nasal inflammation in the OM, which may lead to rhinitis and rhinosinusitis when it persists. Increasing number of epidemiological studies indicate that chronic nasal inflammation is associated with various neurological and psychiatric disorders, such as Alzheimer's disease, Parkinson's disease, depression and anxiety. In addition to these data, the anatomical connections of the OM to the brain via olfactory sensory neurons (OSNs) and to the subarachnoid space via the epineurium of olfactory nerves raise the possibility that nasal inflammation may perturb the homeostasis of the brain and leptomeninges, leading to neuropsychiatric disorders.

Objective: We addressed the question how the nasal inflammation induces changes in the brain morphology and microenvironment in adult and neonatal mice.

Methods: C57BL/6 male mice were used. To induce chronic nasal inflammation, we repeatedly administered lipopolysaccharide (LPS) or saline as a control into a unilateral nostril 3 times per week for 1, 2, 3, 10, 18 and 24 weeks. To analyze the recovery, we repeatedly administered LPS intranasally for 10 weeks and kept the mice without any additional treatment for another 10 weeks. To induce acute nasal inflammation, mice received a single administration of LPS or saline into a unilateral nostril and were sacrificed at several (4, 12, 48) hours after the administration. The effects of intranasal LPS administration on the brain were examined histologically and biochemically.

Results: After nasal administration of LPS to adult male mice, macrophages and neutrophils infiltrated the olfactory mucosa, some of these inflammatory infiltrates released IL-1beta, and the OSNs were lost particularly in the lateral side of the OM. In the olfactory bulb (OB), the first relay station of the olfactory information in the brain, microglia were activated and astrocytes were hypertrophied within 1 day, the activity of interneurons decreased, and the synaptic markers were reduced within 3 weeks. In addition, the OBs were atrophied particularly in the specific layers that were associated with tufted cells within 10 weeks. At this time point, the gene expression for both pro-inflammatory cytokines such as IL-1beta and TNF-alpha and anti-inflammatory cytokines such as IL-10 increased in the OBs of LPS-treated mice. Furthermore, the OBs recovered from the atrophy when the LPS-induced nasal inflammation subsided by the cessation of LPS administration. Studies on the effects of intranasal LPS administration on the neonatal brain and the comparison of the inflammatory responses between adults and neonates are ongoing.

Conclusion: In adult male mice, chronic nasal inflammation induced morphological changes in the specific OB neurocircuit related to tufted cells and altered the cytokine microenvironment in the OB. Furthermore, the tufted cells retain a high degree of plasticity even in adult mice that enables a recovery from morphological damages after inflammation subsides.

TIME: 15:35-15:50

Discussion

PK2. PLENARY KEYNOTE SPEECH

TIME 16:10-17:00, Fri, Oct. 30 2020

VENUE CMUH@Taichung (on-site)

Moderator: Jane Pei-Chen Chang, MD, PhD

*Chief, Child Psychiatry Division, Department of Psychiatry, China Medical University Hospital, Taiwan
Visiting Researcher, Department of Psychological Medicine, Institute of Psychiatry, Psychology and Neuroscience (IoPPN), King's College London, UK*

Application of Repetitive Transcranial Magnetic Stimulation and Theta Burst Stimulation in Autism Spectrum disorder

Hsing-Chang Ni, MD, PhD

*Associate Professor, Department of Psychiatry, Chang Gung Memorial Hospital at Linkou, Taiwan
Director of Department of Child and Adolescent Psychiatry, Chang Gung Memorial Hospital at Linkou, Taiwan*

Autism spectrum disorder (ASD) is a neurodevelopmental disorder with lifelong deficits in social communication and restricted repetitive behaviours. Despite there have been no effective pharmacological or psychological treatments for ASD, repetitive transcranial magnetic stimulation (rTMS) and theta burst stimulation (TBS) are two potential non-invasive neuromodulation techniques in treating ASD.

This presentation includes two parts. First, I will mention the development, progress and current consensus regarding the applications of rTMS and TBS in ASD. Second, I will briefly introduce the findings of our work regarding the application of TBS over the posterior superior temporal sulcus in ASD.

PK3. PLENARY KEYNOTE SPEECH

TIME 09:00-09:50, Sat, Oct. 31 2020

VENUE NCKU@Tainan (on-site), CMUH@Taichung (web)

Moderator: Kuan-Pin Su, MD, PhD

Professor College of Medicine, China Medical University (CMU), Taiwan

Vice President, Tainan Municipal An-Nan Hospital-affiliated with China Medical University, Taiwan

Director, Mind-Body Interface Research Centre, China Medical University Hospital, Taiwan

Honorary Professor of Institute of Psychiatry-King's College London, UK

Tai Chi and Co-Regulation of Sleep, Antiviral Immunity, and Inflammation in Older Adults

Michael Irwin, MD

Distinguished Professor, Vice Chair of Research, Department of Psychiatry and Biobehavioral Sciences,

David Geffen School of Medicine, UCLA; Director, Cousins Center for Psychoneuroimmunology, Semel

Institute for Neuroscience and Human Behavior at UCLA; Director, Mindful Awareness Research Center,

Semel Institute for Neuroscience and Human Behavior at UCLA, USA

Background: Insomnia is prevalent in older adults and is a risk factor for morbidity and mortality. Sleep has a homeostatic role in the regulation of the immune system. In turn, insomnia contributes to declines in anti-viral immunity and increases in inflammation, which may lead to increased risk for infectious disease and inflammatory-related disorders in older adults.

Methods: Randomized controlled trial methods are used to evaluate the effects of Tai Chi on insomnia, antiviral immunity, and inflammatory outcomes.

Results: Tai Chi, as compared to an active comparator condition sleep education, improves insomnia and depressive symptoms in older adults, and is non-inferior to the gold standard of treatment, cognitive behavior therapy of insomnia (CBT-I) in older breast cancer survivors with effects durably maintained over one year follow-up. Tai Chi, as compared to health education, increases viral specific cellular immunity to varicella zoster virus, and augments response to herpes zoster vaccine in older adults. Further, Tai Chi as well as other mind-body interventions such as mindfulness meditation and yoga, reverse inflammation as indexed by systemic markers, cellular production of inflammatory, and inflammatory transcriptional profiles in older adults, with similar robust effects in adults and cancer survivors. and inflammation.

Conclusions: By reversing age-related declines in antiviral immunity and inflammation, the treatment of insomnia with Tai Chi has the potential to mitigate adverse risks for chronic diseases of aging and to promote health span in diverse populations.

S22. PNIRSASIA-PACIFIC SYMPOSIA (II)

TIME	09:50-12:00, Sat, Oct. 31 2020
VENUE	NCKU@Tainan (on-site), CMUH@Taichung (web)

Psychoneuroimmunology Approaches That Shape Health (II)

Chair & Moderator: Keith Kelley, PhD

Professor Emeritus of Immunophysiology University of Illinois

Editor-in-Chief Emeritus Brain, Behavior, and Immunity

The idea that there are reciprocal systems of communication between the immune and central nervous systems began to gain traction in the 1980s. The first issue of *Brain, Behavior, and Immunity* appeared on March 1, 1987 and on January 1, 2000, it became the official journal of the Psychoneuroimmunology Research Society (PNIRS). These early developments made it clear that the brain and immune system can no longer be considered to be captains of their own ship. Today the focus is on mediators and mechanisms of this cross-talk and their potential importance for animal and human health. This PNIRASia-Pacific symposium features seven experts from five countries who will highlight their most recent advances in psychoneuroimmunology research. Of growing prominence is the influence of gender differences on health, particularly during developmental and early life periods. Diet has emerged as an important component. New results will be reported that have identified differential epigenetic regulation of higher order executive functions in male and female mice following maternal malnutrition (Reyes). Overfeeding during the neonatal period also affects a number of hypothalamic neuropeptides that are differentially regulated by sex (Spencer). Similar sex differences are observed in mice being treated for cancer. Consistent with these results, new data show that a standard chemotherapy regimen causes working memory deficits in male but not female mice (Konsman). One of the major emerging research areas is the role of the gut microbiome in health. Although gut-associated lymphoid tissue communicates with the gut intrinsic nervous system, little is known about its potential role in development and early life. Mice expressing the autism-associated *Neurologin-3* R451C mutation at the synapse were used to explore this neural-immune gut communication system in neurodevelopmental disorders (Hill-Yardin). Novel data will then be presented on the role of inflammation in two major sensory systems. Chronic dry eye disease is linked to neuronal, glial and immune cell activation in the trigeminal ganglion. This leads to central nociceptive reorganization, dry eye pain and induction of anxiety-like behaviors (Reaux-Le Goazigo). Another major sensory system, the olfactory mucosa, has now been shown to also be sensitive to nasal inflammation. Activation of TLR-4 receptors in the nasal cavity of mice leads to activation of microglia in the olfactory bulb, secretion of pro- and anti-inflammatory cytokines and the subsequent reversible atrophy of olfactory sensory neurons (Hasegawa-Ishi). Although microglia have predominantly been studied in neuroinflammation, new data will be presented showing there are other important actors. Endothelial cells, pericytes, the meninges and choroid plexus secrete both chemokines and intercellular adhesion molecules. These cells have been isolated from humans and cultured with inflammatory stimuli *in vitro*. New experiments have now established that drugs that are already approved by the FDA regulate the synthesis and secretion of MCP-1, ICAM-1 and VCAM (Dragunow). These new data not only reinforce the concept of reciprocal communication pathways between the immune system and brain but further establish the multi-faceted roles of inflammation in shaping animal and human health from conception to adulthood.

TIME: 09:50-10:00 Session Chair

Keith Kelley, PhD

*Professor Emeritus of Immunophysiology University of Illinois
Editor-in-Chief Emeritus Brain, Behavior, and Immunity*

TIME: 10:00-10:25 Session Speaker

Early Life Diet Can Shape the Development of Executive Function Deficits in Mice: Examination of Prefrontal Cortical Involvement

Teresa M. Reyes, PhD

Associate Professor, Pharmacology and Systems Physiology, University of Cincinnati, USA

TIME: 10:25-10:50 Session Speaker

Sex Differences in Early Life Dietary Programming of Obesity and Stress

Sarah J. Spencer; PhD

Associate Professor/ Principal Research Fellow, RMIT University, Australia

Background: Overfeeding during the first weeks of life in male rats causes a disruption in the peripheral and central leptin systems that likely contribute to obesity. These differences persist into adulthood. Thus, neonatally overfed male rats have elevated circulating leptin in the first two weeks of life, and disrupted connectivity of neuropeptide Y (NPY), agouti-related peptide (AgRP), and pro-opiomelanocortin (POMC) neurons within the regions of the hypothalamus responsible for control of energy balance and food intake. They also have excessive weight gain and adult obesity. Female rats that are neonatally overfed experience similar changes in circulating leptin levels as well as in their long-term body weight. However, there may be differences in how these changes contribute to brain development long-term.

Method: We manipulated the litter sizes that Wistar rat pups were suckled in, reducing the litters to only four pups (in comparison with those controls suckled in litters of 12). The pups suckled in small litters therefore had greater access to the dam's milk and became significantly heavier by as early as postnatal day 7. At weaning the pups were given free access to normal rat chow and in adulthood we examined metabolic and stress-related outcomes.

Results: Our data suggest that that the peripheral metabolic changes that occur in both male and female rats after neonatal overfeeding are not mirrored centrally in females by changes in hypothalamic NPY, AGRP, and POMC fiber density. In accordance, the adult obesity is less pronounced in neonatally overfed females than in males. On the other hand, neonatal overfeeding leads to long-term changes in ghrelin signaling pathways in females, but not males, that leaves females vulnerable to hyper-active hypothalamic-pituitary-adrenal (HPA) axis responses to stress.

Conclusion: These findings are suggestive of sex differences in the effects of neonatal overfeeding and of differences in the ability of the female and male central systems to respond to changes in the early life nutritional environment, with resilience in some systems and vulnerability in others.

TIME: 10:50-11:15 **Session Speaker**

The Gut-Brain Interface in Neurodevelopmental Disorders

Elisa Hill-Yardin, PhD, Samiha S Sharna, Chalystha YQ Lee, Madushani Herath, Suzie Hosie, Gayathri K Balasuriya, Ashley E Franks

Associate Professor, Head of Gut-Brain Axis Laboratory, ARC Future Fellow and Senior Vice Chancellor's Research Fellow, RMIT University, Australia

Background: Many gene mutations altering the central nervous system are associated with neurodevelopmental disorders, however effects on the immune system are less well characterised. The intrinsic nervous system of the gut bidirectionally interacts with the gut-associated lymphoid tissue (GALT). We therefore investigated for changes in the nervous system and gut-associated lymphoid tissue in the caecum in a mouse model of autism. The autism-associated R451C missense mutation in the neuroligin-3 gene encodes a synaptic adhesion protein and results in impaired neurotransmission in the brain. The caecum houses a large supply of microbes and is involved in generating immune responses via the caecal patch that regulates microbial content and immune responses. Within the gut, the mucus lining the gastrointestinal tract forms an important barrier between the host and the microbial content. However, whether the autism-associated R451C mutation alters the caecal enteric nervous system, mucus properties and immune function is unknown.

Method: We assessed for gross anatomical changes in the caecum and quantified the proportions of caecal submucosal and myenteric neurons in wild-type and NL3R451C mice using immunofluorescence. In the caecal patch, we assessed total cellular density as well as the density and morphology of Iba-1 labeled macrophages to identify whether the R451C mutation affects neuro-immune interactions. We further stained caecal tissue sections with Alcian Blue to determine if mucus thickness is altered in NL3R451C mice.

Results: We identified that mice expressing the R451C mutation have significantly reduced caecal weight compared to wild-type mice. In NL3R451C mice, caecal ganglia contain more neurons and increased numbers of Nitric Oxide (NO) producing neurons (labeled by Nitric Oxide Synthase; NOS) per ganglion in both the submucosal and myenteric plexus. Although overall caecal patch cell density was unchanged, NL3R451C mice have an increased density and altered morphology of Ionized calcium binding adaptor molecule 1 (Iba-1) labeled enteric macrophages. Specifically, macrophages in NL3R451C mice were smaller and more spherical in morphology.

Conclusion: We have identified changes in both the nervous system and immune system caused by an autism-associated mutation in Nlgn3 encoding the postsynaptic cell adhesion protein, Neuroligin-3. These findings provide further insights into the potential modulation of neural and immune pathways in the context of neurodevelopmental disorders.

TIME: 11:15-11:40 **Session Speaker**

Targeting Brain Barrier Cells for Regulating Brain Inflammation

Michael Draganow, PhD

Professor, University of Auckland, New Zealand

TIME: 11:40-12:00

Discussion

S22. ISNPR SESSION

TIME 13:30-15:30, Sat, Oct. 31 2020

VENUE NCKU@Tainan (on-site), CMUH@Taichung (web)

Nutritional Psychaitry Research

Session Chair: Philip B Ward, PhD

*Chief, Child Psychiatry Division, Department of Psychiatry, China Medical University Hospital, Taiwan
Visiting Researcher, Department of Psychological Medicine, Institute of Psychiatry, Psychology and*

Moderator: Jane Pei-Chen Chang, MD, PhD

*Chief, Child Psychiatry Division, Department of Psychiatry, China Medical University Hospital, Taiwan
Visiting Researcher, Department of Psychological Medicine, Institute of Psychiatry, Psychology and
Neuroscience (IoPPN), King's College London, UK*

TIME: 13:30-13:55 Session Speaker

Diet and Depression: Exploring the Biological Mechanisms of Action

Wolfgang Marx, PhD

Alfred Deakin Postdoctoral Research Fellow, Food & Mood Centre, Deakin University, Victoria, Australia

The field of Nutritional Psychiatry has generated observational and efficacy data supporting a role for healthy dietary patterns in depression onset and symptom management. To guide future clinical trials and targeted dietary therapies, this presentation will provide an overview of what is currently known regarding underlying mechanisms of action by which diet may influence mental and brain health. The mechanisms of action associating diet with health outcomes are complex, multifaceted, interacting, and not restricted to any one biological pathway. Numerous pathways were identified through which diet could plausibly affect mental health. These include modulation of pathways involved in inflammation, oxidative stress, epigenetics, mitochondrial dysfunction, the gut microbiota, tryptophan-kynurenine metabolism, the HPA axis, neurogenesis and BDNF, epigenetics, and obesity. Furthermore, existing limitations and recommendations for future research studies within this area will be discussed.

TIME: 13:55-14:20 Session Speaker

Nutritional Psychiatry in Psychotic Disorders: Current Hypotheses and Research Challenges

Scott Teasdale PhD, Sabrina Mörkl, Annabel Sandra Müller-Stierlin PhD

Postdoctoral Research Fellow, School of Psychiatry, UNSW Sydney, Australia

The role of nutrition in mental illness has received considerable attention over recent years. There is considerable evidence for improving the nutritional intake of people with schizophrenia-spectrum disorders to improve physical health status, however evidence for nutritional interventions in the prevention and treatment schizophrenia-spectrum disorders remains limited in quantity and quality. Pathways currently in focus include: i) nutritional deficits and impairments in glucose metabolism, ii) inflammation, and iii) altered gut microbiota. All of which appear to be interconnected. Key limiting factors for advancing research in this field are research challenges associated with assessing and interpreting inflammatory profiles, microbiota and subjective nutritional assessments, which is further complicated by illness characteristics. This presentation describes the state of evidence for key hypotheses, including underlying mechanisms, implicated in schizophrenia-spectrum disorders, the challenges in nutritional psychiatry research and the current state of nutrition interventions in mental healthcare.

TIME: 14:20-14:45 Session Speaker

Protecting Physical Health in People with Mental Illness: Findings from the Lancet Psychiatry Commission

Firth Joseph, PhD

Research Fellow, University of Manchester

The poor physical health, and associated premature mortality, of people with mental illness is increasingly recognised as a human rights issue. The 2019 Lancet Psychiatry Commission brought together a team of clinicians, researchers, and stakeholders to summarise the latest evidence for understanding and addressing these physical health disparities. The Commission involved 5 different 'Parts':

In Part 1, a systematic meta-review was undertaken, and identified cardiometabolic diseases as highly-common and early-arising in people treated for mental illness; impeding physical health, psychological well-being and functional recovery. Part 2 determined key targets and priorities for tackling physical health risks associated with mental illness. Part 3 discussed the effects of psychotropic medications on physical health, and outlined pharmacological strategies for attenuating this. Parts 4 and 5 presented recommendations for the use physical health interventions to improve outcomes in mental health populations; recommending these be implemented immediately in young people treated for mental illness (even prior to physical comorbidities arising).

The evidence behind these recommendations, along with strategies for implementing evidence-based physical health interventions, will be presented.

TIME: 14:45-15:10 Session Speaker

Taking It to the Street: Real-World Implementation of Lifestyle Interventions for People Living with Mental Illness

Philip B Ward, PhD

Professor, School of Psychiatry, UNSW Sydney, Australia

TIME: 15:10-15:30

Discussion

S23. POSTER BLITZ

TIME 16:00-16:30, Sat, Oct. 31 2020

VENUE NCKU@Tainan (on-site), CMUH@Taichung (web)

Moderator: Jane Pei-Chen Chang, MD, PhD

*Chief, Child Psychiatry Division, Department of Psychiatry, China Medical University Hospital, Taiwan
Visiting Researcher, Department of Psychological Medicine, Institute of Psychiatry, Psychology and Neuroscience (IoPPN), King's College London, UK*

TIME: 16:00-16:05

PP015. The Serotonin Transporter Associated with Increased Risk of Suicide Attempts in Young Adults with a Family History of Domestic Violence
Shui Jiang (Canada)

TIME: 16:06-16:11r

PP023. Genetic Variations of Ionotropic Glutamate Receptor Pathways on Interferon- α -induced Depression in Patients with Hepatitis C Viral Infection
Szu-Wei Cheng (Taiwan)

TIME: 16:12-16:17

PP033. Plasma D-glutamate Levels for Detecting Mild Cognitive Impairment and Alzheimer's Disease: Machine Learning Approaches
Chun-Hung Chang, PhD (Taiwan)

TIME: 16:18-16:23

PP011. Genome-wide DNA Methylation Signatures of Resilience in Young Adults
Andrew Ke-Ming Lu (Taiwan)

TIME: 16:24-16:29

PP024. Antidepressants Treatment Regulates the Neurotransmitter Levels and Attenuate Apoptosis Induced by Arecoline Oxidative Metabolite
Srinivasan Nithiyantham (Taiwan)

AWARD CEREMONY

TIME 16:30-17:00, Sat, Oct. 31 2020

VENUE NCKU@Tainan (on-site), CMUH@Taichung (web)

CHAIR: Jane Pei-Chen Chang, MD, PhD

*Chief, Child Psychiatry Division, Department of Psychiatry, China Medical University Hospital, Taiwan
Visiting Researcher, Department of Psychological Medicine, Institute of Psychiatry, Psychology and Neuroscience (IoPPN), King's College London, UK*

PK4. PLENARY KEYNOTE SPEECH

TIME 09:00-09:50, Sun, Nov. 1 2020

VENUE NCKU@Tainan (on-site), CMUH@Taichung (web)

Moderator: Kuan-Pin Su, MD, PhD

Professor College of Medicine, China Medical University (CMU), Taiwan

Vice President, Tainan Municipal An-Nan Hospital-affiliated with China Medical University, Taiwan

Director, Mind-Body Interface Research Centre, China Medical University Hospital, Taiwan

Honorary Professor of Institute of Psychiatry-King's College London, UK

Heart Rate Variability: A Window into the Mind-Body Connection

Angelos Halaris, MD, Camilla Guccione, PhD, Keri Heilman, PhD, Stephen W. Porges, PhD

Professor of Psychiatry Loyola University Chicago School of Medicine; Medical Director Department of

Psychiatry Loyola University Medical Center, USA

Background: Stress is associated with variations in autonomic activity that disrupt homeostatic processes. The Autonomic Nervous System (ANS) responds to the needs of the internal viscera and external stimuli. Homeostasis is associated with internal viscera regulation, whereas the stress response prioritizes external stimuli over internal needs. Thus, stress occurs when an organism's physiological demands are no longer adequately fulfilled by the Parasympathetic Nervous System (PNS) functioning. Consequently, measurement of parasympathetic tone may be viewed as an index of stress and stress vulnerability. A widely used measure of Sympathetic Nervous System (SNS) and PNS activity is Heart Rate Variability (HRV). To date, HRV is widely used in cardiology, neuropsychology, and more recently in psychiatry. This study detects autonomic regulation in depressive disorders to obtain objective markers of potential utility in the diagnostic differentiation.

Methods: We investigated Respiratory Sinus Arrhythmia (RSA), Low-Frequency (LF) of Heart Rate Variability, and Systolic Blood Pressure (SBP) in patients with Bipolar Disorder Depressed (BDD=31), patients with Major Depressive Disorder (MDD=32), and in healthy controls (HC=32). Since BDDs were maintained on specific medications to manage manic/hypomanic symptoms, we explored whether atypicals, anticonvulsants, or their combination could independently affect the above physiological parameters.

Results: Correlational results showed that the Cardiac Vagal Tone (CVT=LF+RSA) was highly correlated with RSA and LF, suggesting that this combined measure might provide a more robust estimate of vagal activity. When the autonomic measures were analyzed as a combined variable, it discriminated BDDs and MDDs from HCs, although the SBP of the BDDs may have been affected by mood stabilizers. However, since RSA, LF, and CVT are highly intercorrelated, pairwise comparisons showed that only RSA was significantly lower in the BDD group compared to HCs, and MDDs.

Conclusion: RSA and SBP can be assessed in outpatient settings, facilitating the differential diagnosis in major affective disorders. However, concomitant administration or maintenance on psychotropic medications can potentially increase SBP.

S31. MIND-BODY SESSION

TIME 09:50-11:50, Sun, Nov. 1 2020

VENUE NCKU@Tainan (on-site), CMUH@Taichung (web)

Moderator: Kuan-Pin Su, MD, PhD

Professor College of Medicine, China Medical University (CMU), Taiwan

Vice President, Tainan Municipal An-Nan Hospital-affiliated with China Medical University, Taiwan

Director, Mind-Body Interface Research Centre, China Medical University Hospital, Taiwan

Honorary Professor of Institute of Psychiatry-King's College London, UK

TIME: 09:50-10:15 Session Speaker

Microglia-astrocyte Interaction in A Mouse Model of Neuromyelitis Optica

Long-Jun Wu, PhD

Professor, Departments of Neurology and Neuroscience; Consultant, Departments of Neurology and

Immunology; Director, Neuroimmune Interaction Laboratory; Associate Director, Neuroscience PhD Program

of Mayo Clinic College of Medicine, USA

Neuromyelitis optica (NMO) is an autoantibody triggered neuroinflammatory disease mostly attacks spinal cord and optic nerve. Most NMO patients are seropositive for autoantibodies against water channel protein aquaporin-4 (AQP4-IgG) in astrocytic end-feet. Histopathology studies to date have highlighted prominent activation of microglia, the sentinels in central nervous system (CNS), in early NMO lesions. However, the role of microglia in the evolution of NMO is unknown. We developed an informative murine model in which NMO patient-derived IgGs are infused continuously into the spinal subarachnoid space. The outcome was that NMO-IgG induced motor impairment and NMO-compatible pathology in wild-type mice but not in AQP4-null mice. In vivo spinal cord imaging revealed that NMO-IgG induced microglia-astrocyte physical interaction. In mice depleted of microglia, both motor impairment and NMO characteristic immunohistopathology were significantly reduced. We further discovered that early astrocyte-microglia interaction requires the C3a fragment of complement derived from astrocytic C3. Mice genetically lacking C3 or C3a receptor lacked motor deficits and NMO pathology after NMO-IgG infusion. Our study revealed that early-activated CNS-intrinsic complement components mediate astrocyte-microglia signaling that drives NMO lesion evolution, and identifies microglia as a potential target for NMO therapeutic interception.

TIME: 10:15-10:40 Session Speaker

Non-invasive Brain Stimulation for Mild Cognitive Impairment and Alzheimer`s Disease: from Investigation to Therapeutic Application

Che-Sheng Chu, MD

Attending Physician, Department of Psychiatry & Center for Geriatric and Gerontology, Kaohsiung Veterans General Hospital, Kaohsiung, Taiwan

Background: No systematic review or meta-analysis has assessed the efficacy of omega-3 polyunsaturated fatty acids (PUFAs) for anxiety. We evaluated the anxiolytic effects of omega-3 PUFA treatment compared with controls in varied populations.

Methods: Clinical trials assessing the anxiolytic effect of omega-3 PUFAs in humans, either in placebo-controlled or non-placebo-controlled designs were searched up to March 4, 2018. Out of 104 selected articles, 19 entered the final data extraction stage. Two authors independently extracted the data according to a pre-determined list of interests. We performed a random-effects model meta-analysis and conducted this study based upon PRISMA guidelines. The main outcomes were set as the changes in the severity of anxiety symptoms after omega-3 PUFA treatment.

Results: In total, 1203 participants with omega-3 PUFA treatment and 1037 participants without omega-3 PUFA treatment revealed that omega-3 PUFA improved clinical anxiety symptoms compared with control arms (Hedges' $g = 0.374$, 95% confidence interval = 0.081 to 0.666, $p = 0.012$). Subgroup analysis showed that the treatment effects were significantly better in subgroups with specific clinical diagnoses than in subgroups without clinical conditions. The anxiolytic effect of omega-3 PUFAs was significantly better than that of controls only in subgroups with a higher dosage (at least 2 g/day) and not in subgroups with a lower dosage (less than 2 g/day).

Conclusion: Our review indicates that omega-3 PUFAs might help to reduce the symptoms of clinical anxiety. Further well-designed studies are needed in populations where anxiety is the main symptom.

TIME: 10:40-11:05 Session Speaker

Clinical Application of Cranial Electrotherapy Stimulation for the Treatment of Generalized Anxiety Disorder in Taiwan

Tien-Yu Chen, MD and Hsin-An Chang, MD

Attending Physician and Assistant Professor, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan

Background: Patients with generalized anxiety disorder (GAD) can be treated by cognitive-behavioral therapy (CBT) and anxiolytic-hypnotic medications. However, the resources for CBT are limited in Taiwan and most of the patients with GAD were treated by medications. Nevertheless, the long-term use of anxiolytic-hypnotic medications may cause a change in sleep structure and increase the risk of anxiolytic abuse. Therefore, a novel treatment for GAD is warranted in Taiwan. Alpha-Stim Cranial electrotherapy stimulation (CES) is a U.S. Food and Drug Administration (FDA)-approved treatment for insomnia, depression, and anxiety consisting of pulsed, low-intensity current applied to the earlobes or scalp. In the early of 2020, U.S. FDA downgraded the CES devices designed to treat anxiety and insomnia from Class III to Class II, leaving CES devices for depression in the most highly regulated Class III category. Although CES is widely used in many countries, doctors in Taiwan have only limited experience in this device.

Question/Hypothesis: We hypothesize that adjunctive use of Alpha-Stim CES for 6 weeks in patients with GAD may improve their anxiety symptoms and heart rate variability (HRV).

Method: In this pilot study, we enrolled subjects with the first-time diagnosis of GAD who refuse to take antidepressant or regular anxiolytic medications for their GAD. We only prescribe alprazolam for emergent use (Pro re nata, PRN used). They received 60 min per day Alpha-Stim CES device with electrical stimulation by generating bipolar, asymmetric, rectangular waves with a frequency of 0.5 Hz and a current intensity with the lowest therapeutic dose at 100 μ A via electrodes placed on the ear lobes for 6 weeks. GAD-7 and Resting HRV was examined before and after the CES adjunctive treatment.

Result: In this preliminary study, we enrolled 28 newly diagnosed GAD participants (Male: 5, Female: 23, mean age: 46.8 years old) who only used PRN anxiolytics. CES was used as an adjunctive treatment for 6 weeks. After a 6-week treatment, the GAD-7 scores were significantly decreased from 17.5 ± 2.96 to 9.92 ± 3.23 ; $P < 0.001$. The heart rate was significantly decreased after CES treatment at both first time use of CES (84.7 ± 11.4 to 76.77 ± 12.96 ; $P = 0.007$) and the end of the study (81.12 ± 12.05 to 76.27 ± 13.65 ; $P = 0.046$). In addition, the high frequency (HF) power was significantly increased when we performed resting HRV for the participants immediately after first time 60-min CES treatment (3.27 ± 1.50 to 4.28 ± 1.81 ; $P = 0.035$) and a trend of increase at the end of the study (4.02 ± 1.66 to 4.22 ± 1.83 ; $P = 0.675$).

Conclusion: Adjunctive CES treatment for patients with GAD in Taiwan could decrease their severity of anxiety and might enhance the parasympathetic activity. A further double-blind, sham-controlled design to test the efficacy of CES to GAD patients in Taiwan is warranted.

TIME: 11:05-11:30 Session Speaker

Inverse Correlation between Omega-3 Concentration and Emotional Brain Response on Major Depression

Cheng-Hao Tu, PhD; Chun-Ming Chen, PhD; Chuan-Chih Yang, PhD; Kuan-Pin Su, MD, PhD

Assistant Professor, Graduate Institute of Acupuncture Science, China Medical University, Taichung, Taiwan

Depression is an increasing mental health problem around whole world. Patients with major depression (MDD) have decreased responsiveness to the positive emotion stimuli and/or increased responsiveness to negative emotion stimuli. Recent clinical trials indicated that polyunsaturated fatty acid (PUFA) supplements can improve the symptoms of MDD, with the higher treatment efficacy of eicosapentaenoic acid (EPA) than of docosahexaenoic acid (DHA). However, the possible central mechanisms of EPA and DHA treatment remained unexplored. Twenty-four MDD patients have been participated in this double-blind, randomized-controlled study. All patients have been randomly allocated into EPA or DHA group for a 12-week treatment session. The 21-item Hamilton depression rating scale has been used to assess the depressive symptoms as clinical outcomes. Blood samples from vein have been taken to measure the membrane PUFA level on erythrocyte by gas chromatography of methyl esters. The brain responses to emotional picture stimuli have been measured by a 3T MRI scanner. The results showed that EPA may have more clinical efficacy than DHA on MDD patients. The EPA level was significantly increased after the treatment of EPA but not DHA, while the DHA level was both significantly increased after the treatment of EPA and DHA. The improvement of clinical symptoms has a trend to negative correlated with EPA level within EPA group, while neither significant correlation between DHA level within EPA group nor both EPA and DHA within DHA treatment group has been found. Imaging results further revealed that change of EPA level was negatively correlated with the changes of activity of right ventrolateral prefrontal cortex and anterior insula cortex when processing positive emotion stimuli with EPA treatment, whereas the DHA level was also negatively correlated with the activity of left anterior insula and right dorsolateral prefrontal cortex when processing negative emotion stimuli with DHA treatment. These results raised a possible role of docosapentaenoic acid, an intermediary metabolite between EPA and DHA, on the PUFA treatment of MDD.

TIME: 11:30-11:50

Discussion

COLSING REMARKS

TIME 11:50-12:00, Sun, Nov. 1 2020

VENUE NCKU@Tainan (on-site), CMUH@Taichung (web)

CHAIR: Kuan-Pin Su, MD, PhD

Professor College of Medicine, China Medical University (CMU), Taiwan

Vice President, Tainan Municipal An-Nan Hospital-affiliated with China Medical University, Taiwan

Director, Mind-Body Interface Research Centre, China Medical University Hospital, Taiwan

Honorary Professor of Institute of Psychiatry-King's College London, UK

VIRTUAL POSTER PRESENTATION

PP001	Maternal dietary intake of fish and polyunsaturated fatty acids and child neurodevelopment at 6 months and 1 year of age: a nationwide birth cohort—the Japan environment and children’s study Kei Hamazaki, Japan
PP003	Cognitive inflexibility of Parkinson’s disease dementia -- using 6-OHDA injection mice model Sheng Ta Tsai, Taiwan
PP007	The antidepressant-like effects of liquid dietary supplement of Cordyceps militaris and Armillaria mellea formula in unpredictable chronic mild stress animal model Huai-Syuan Huang, Taiwan
PP008	Will melatonin be beneficial to prevent episodic migraine? From the view of network meta-analysis Ping-Tao Tsenga, Taiwan
PP009	Restless legs syndrome in children and adolescents with attention-deficit/hyperactivity disorder: prevalence, mimic conditions, risk factors, and association with functional impairment Maytinee Srifuengfung, Thailand
PP011 ★	Genome-wide DNA methylation signatures of resilience in young adults Andrew Ke-Ming Lu, Taiwan
PP012	Evidence in the activation by oxaliplatin, a platinum-based chemotherapeutic agent, of hyperpolarization-activated cation and electroporation-induced currents Sih-Wei Lee, Taiwan
PP013	Folate supplementation in a schizophrenia patient with genetic variations in MTRR A66G and C524T Lin Ching Jung, Taiwan
PP014	Education program involved “diet, exercise, and mind-body balance” aspects to improve prognosis for cancer survivors Chia-Ching Lin, Taiwan
PP015 ★	The serotonin transporter is associated with increased risk of suicide attempts in young adults with a family history of domestic violence Shui Jiang, Canada
PP016	Nature-relatedness, physical activities and psychological distress among youth in Indonesia Alifa Syamantha Putri, Indonesia
PP017	Oxidative DNA damage in mental illness (Schizophrenia, Bipolar disorder, Depression): a meta-analysis Goh Xue Xin, Malaysia
PP018	History of child maltreatment when assessing adults with criterion a for PTSD and the usefulness of measuring heart rate Randdy Ferreira, Portugal
PP019	Effect of 8 week mindfulness yin yoga on stressed nurses Anhui Michelle Chu, Taiwan
PP020	Statins and the risks of decompensated liver cirrhosis and hepatocellular carcinoma Wei Che Chiu, Taiwan
PP022	Therapeutic effect of yoga in depression! Study to assess the role of yogic exercises and its effect on salivary biomarkers Deepika Singh, India
PP023 ★	Genetic Variations of Ionotropic Glutamate Receptor Pathways on Interferon- α -induced Depression in Patients with Hepatitis C Viral Infection Szu-Wei Cheng, Taiwan

PP024 ★	Antidepressants treatment regulates the neurotransmitter levels and attenuate apoptosis induced by arecoline oxidative metabolite Srinivasan Nithiyantham, Taiwan
PP025	Traditional Chinese medicine clinical research of bipolar disorder Shu-Ping Chen, Taiwan
PP026	Remyelination of nervonic acid on APP/PSEN1 mice, animal model of Alzheimer's disease Rabia Parveen, Pakistan
PP027	Mediational role of psychological flexibility between flourishng and subjective well being among elderly Ridhima Shukla, India
PP028	Neurocorrelates of free will revisited Paola Di Maio, Taiwan
PP029	Evidence based insights into digital behavior change: an exploratory study Sneha Saha, India
PP030	Neurophysiological basis of chronic pre-natal and adult stress-induced visceral pain in females Jinghong Chen, China
PP031	How coping strategies could predict trauma in Portuguese First Responders Ana Oliveira, Portugal
PP032	The interaction between depression and lung cancer: explore cellular and neuroimmunological mechanisms Bai-Ping Liu, China
PP034	Mindfulness intervention improves inflammatory biomarkers in older adults with mild cognitive impairment: a randomized controlled trial Ted Kheng Siang Ng, Malaysia
PP033 ★	Plasma D-glutamate levels for detecting mild cognitive impairment and Alzheimer's disease: Machine learning approaches Chun-Hung Chang, Taiwan
PP035	Horticultural therapy ameliorates biomarkers of immunosenescence and inflammaging in community-dwelling older adults: a randomized controlled trial Ted Kheng Siang Ng, Malaysia
PP036	Glutamatergic medications as adjunctive therapy for obsessive-compulsive disorder in adults: a systematic review Mohammadreza Shalbfan, Iran

★5-minute Poster Blitz Presentation

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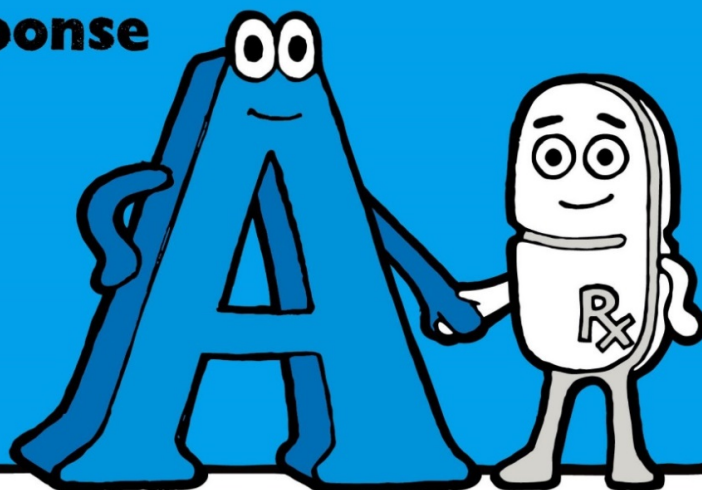
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成人	重鬱症之輔助治療	2-5 mg/day	2-15 mg/day	15 mg/day	
	思覺失調症	10 or 15 mg/day	10 or 15 mg/day	30 mg/day	
	雙極性疾患之躁症發作及混合型發作，單獨使用	15 mg/day	15 mg/day	30 mg/day	
	雙極性疾患之躁症發作及混合型發作， 鋁鹽或Valproate輔助治療 第一型雙極性疾患維持治療，鋁鹽或Valproate輔助治療	10-15 mg/day	15 mg/day	30 mg/day	
青少年（13-17歲）	思覺失調症	2 mg/day	10 mg/day	30 mg/day	
兒童（10-17歲）	雙極性疾患之躁症發作及混合型發作， 單獨使用及鋁鹽或Valproate輔助治療 第一型雙極性疾患維持治療，鋁鹽或Valproate輔助治療	2 mg/day	10 mg/day	30 mg/day	
	兒童（6-17歲）	自閉性疾患伴隨之急躁易怒	2 mg/day	5-15 mg/day	15 mg/day
兒童（6-18歲）	妥瑞氏症	< 50 公斤	2 mg/day	5 mg/day	10 mg/day
		≥ 50 公斤	2 mg/day	10 mg/day	20 mg/day

● **藥理作用：**作用機制仍然未知。有假設指出，aripiprazole可能經由對多巴胺D₂和血清素5-HT_{1A}接受體的部分促進作用，以及對血清素5-HT_{2A}接受體的拮抗作用而產生療效。

● **注意事項：**可與食物併服，不須依患者之肝腎功能調整劑量。

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● **不良反應：**頭痛、噁心、嘔吐、暈眩等。

● **疾病分類代碼：**

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雙極性情感異常，躁型 296.4
雙極性情感異常，混合型 296.6
重鬱症，單純發作 296.2
重鬱症，復發 296.3
自閉症 299
妥瑞氏症 307.23

ICD 9 **ICD 10**
F20
F31.1
F31.6
F32
F33
F84
F95.2

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入檢測，免除技術人
員的人力。

專屬個人健康管理平
台，手機簡易操作，
遠端填寫問卷綜合評
估，Line上的健管家綁
定註冊互動。

一系列 有效的改善方案

Effective ways to improve



- 神經改善營養組合：維衡靈、關欣寧
- 去除疲勞活力補給品：攝補挺
- 腸胃道健康益生菌：微益多、微益寧

HEALTHY, NUTRITION AND BALANCE | ABOUT STAYING HEALTHY



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2020

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