

International Drug Addiction and Neurodegeneration Symposium-I

主題：成癮和神經退化性疾病

時間：2016年2月24日(三)9時至16時

地點：國衛院竹南院區行政大樓B1第3會議室(苗栗縣竹南鎮科研路35號)

Keynote Speaker



Roy Wise
Senior scientist
National Institute on Drug Abuse,
NIH, U.S.A

Topic:
The Self-Addicted Brain

其他講員

Invited Speakers



Barry Hoffer
Scientist Emeritus
National Institute on
Drug Abuse, NIH, U.S.A



Chaim G Pick
Professor and Chairman
Tel-Aviv University, Israel



Mikko Airavaara
Principal Investigator
University of Helsinki,
Finland

王昀 國家衛生研究院 · 王聲昌 國家衛生研究院 · 柯瓊媛 臺北醫學大學
陳雲翔 輔仁大學 · 陳慧誠 國家衛生研究院 · 劉玉麗 國家衛生研究院
劉誠珍 國家衛生研究院 · 謝奉勳 國家衛生研究院

教育學分
台灣成癮科學學會5學分
台灣精神醫學會5學分



相關訊息：<http://tamtp.nhri.org.tw/>

主辦單位：國家衛生研究院神經精神中心、臺北醫學大學神經損傷及再生研究中心與
神經再生醫學博士學位學程、台灣成癮科學學會

National Health Research Institutes

International Drug Addiction and Neurodegeneration Symposium- I

Organizers: National Health Research Institutes

Graduate Institute of Neural Regenerative Medicine, Taipei Medical University

Taiwanese Society of Addiction Science

Date: Wednesday, February 24, 2016		
Venue: 3 rd Conference Room, B1, Administion Building, NHRI, Zhunan, Taiwan		
09:00-09:20	Registration	
09:20-09:30	Opening Remarks	
09:30-10:30	Keynote Speech Dr. Roy Wise Senior Scientist National Institute on Drug Abuse, NIH, U.S.A. Topic: The Self-Addicted Brain.	Moderator: Jih-Heng Li (李志恒) Professor College of Pharmacy, Kaohsiung Medical University, Taiwan. President Pharmaceutical Society of Taiwan
10:30-10:45	Dr. Yu-Li Liu (劉玉麗) Principal Investigator National Health Research Institutes. Topic: APBB2 is associated with multiple substance use in a methadone maintenance population.	San-Yuan Huang (黃三原) Professor National Defense Medical Center Taiwan.
10:45-11:00	Dr. Sheng-Chang Wang (王聲昌) Principal Investigator National Health Research Institutes. Topic: Characteristics of ketamine users in Taiwan.	President, Taiwanese Society of Addiction Science.
11:00-11:30	Dr. Barry Hoffer Scientist Emeritus National Institute on Drug Abuse, NIH, U.S.A. Topic: Neurotrophic factors; past present and future.	
11:30-11:45	Dr. Yun-Hsiang Chen (陳雲翔) Assistant Professor Fu-Jen Catholic University, Taiwan. Topic: Should Methadone Addicts Worry About Flu.	
11:45-12:00	Dr. Hwei-Hsien Chen (陳慧誠) Principal Investigator National Health Research Institutes. Topic: Reversal effect of sodium benzoate on	

	delayed extinction and stronger drug-primed reinstatement of methamphetamine seeking in rats prenatally exposed to morphine.	
12:00-13:00	Lunch	
13:00-13:30	Dr. Chaim G Pick Professor and Chairman Tel-Aviv University, Israel. Topic: Hyperbaric Oxygen therapy and Caloric Restriction as a treatment for traumatic brain injury in mice.	Moderator: Jih-Heng Li (李志恒) Professor College of Pharmacy, Kaohsiung Medical University, Taiwan. President Pharmaceutical Society of Taiwan Pao-Luh Tao (陶寶綠) Professor National Health Research Institutes.
13:30-13:45	Dr. Chiung-Yuan Ko (柯瓊媛) Assistant Professor Taipei Medical University, Taiwan. Topic: Neuroinflammation in Alzheimer's disease: function of astrocytic CCAAT/enhancer binding protein delta.	
13:45-14:00	Dr. Feng Shiun Shie (謝奉勳) Principal Investigator National Health Research Institutes. Topic: Metabolic stress in the pathogenesis of Alzheimer's disease.	
14:00-14:15	Break	
14:15-14:30	Dr. Mikko Airavaara Principal Investigator Institute of Biotechnology, University of Helsinki, Finland. Topic: Secondary damage and time-course of microglial activation after cortical stroke in rats.	
14:30-14:45	Dr. Seong Jin Yu (劉誠珍) Principal Investigator National Health Research Institutes. Topic: Neuroprotective action of posiphen in stroke.	
14:45-15:00	Dr. Yun Wang (王昀) Principal Investigator National Health Research Institutes. Topic: Neuroprotective action of dopamine neuron stimulating peptide.	
15:00-15:30	Closing Remarks Dr. Barry Hoffer	

Keynote Speech:

ROY A. WISE

Senior Scientist, National Institute on Drug Abuse,
NIH, U.S.A.



TOPIC: The Self-Addicted Brain

Education

B.A., MA.	California State University	1963, 1965
Ph.D.	McGill University	1968

Experience

Assistant Professor	California State University	1968-1969
Assistant Professor	Concordia University	1969-1973
Associate Professor	Concordia University	1973-1977
Professor	Concordia University	1977-1999
Visiting Scientist	Laboratory of Neuropharmacology, NIMH, Washington, D.C.	1975-1976
Chief, Behavioral Neuroscience Branch, Intramural Research Program National Institute on Drug Abuse		1997-date
Deputy Director, Intramural Research Program, National Institute on Drug Abuse		2000-2010

University Administration

Graduate Program Director, Department of Psychology	1972-1975, 1979-1984
Co-director, Center for Research on Drug Dependence	1972-1983
Director, Center for Studies in Behavioral Neurobiology	1983-1991

Committee Appointments

National Institute on Drug Abuse (U.S.)	
Grant review study section	1976-1980
Board of Scientific Counselors	1982-1985
Working Group on Affect and Motivation NSF, (US)	1985
Society for Neuroscience Program Committee	1986-1989
Organizing Committee, Winter Conference on Brain Research	1993-1996
Advisory Council, NeuroScience Network, Networks of Centers of Excellence (Canada)	1990-1997

Awards and Honors

Merit Award, National Institute on Drug Abuse (1989-1997)
Fellow, Royal Society of Canada, Academy of Science (1997)
Laurea ad honorem in Medicine, University of Cagliari, (Mar 2, 1999)

Special lecture and closing remark:

Barry J Hoffer

Scientist Emeritus, National Institute on Drug Abuse, NIH, U.S.A.

Adjunct Professor, Depts. of Neurosurgery and Genetics,
Case Western Reserve University School of Medicine



TOPIC: Neurotrophic factors; past present and future.

Education

University of Rochester, NY	Ph.D.	1965	Physiology
University of Rochester, NY	M.D.	1967	Medicine

Experience

1968-1970	Research Associate, SMR, IRP, NIMH, Laboratory of Neuropharmacology, St. Elizabeths Hospital, Washington, D.C.
1970-1973	Medical Officer, SMR, IRP, NIMH, Laboratory of Neuropharmacology, St. Elizabeths Hospital, Washington, D.C.
1973-1976	Chief, Section on Developmental Neurobiology, SMR, IRP, NIMH, Laboratory of Neuropharmacology, St. Elizabeths Hospital, Washington, D.C.
1976-1996	Professor, Pharmacology, University of Colorado Medical School, CO
1985-1989	Director, Interdisciplinary Neuroscience Program, University of Colorado Medical School, CO
1989-1996	Professor, Psychiatry, University of Colorado Medical School, CO
1996-2010	Scientific Director, National Institute on Drug Abuse, NIH
2010-present	Scientist Emeritus, National Institute on Drug Abuse, NIH
2010-Present	Adjunct Professor, Depts. of Neurosurgery and Genetics, CWRU School of Medicine

Special lecture

Chaim G. Pick

Current position:

Professor and Chairman
Dept. of Anatomy and Anthropology
Sackler School of Medicine
Tel-Aviv University, ISRAEL



Topic: Hyperbaric Oxygen therapy and Caloric Restriction as a treatment for traumatic brain injury in mice.

Education:

1976-1980 - Bar-Ilan University, Ramat-Gan B.A. Psychology

1982-1985 - The Hebrew University, Jerusalem M.Sc. Anatomy

1986-1990 - The Hebrew University, Jerusalem Ph.D. Anatomy

Special lecture

Mikko Airavaara

Current position:

Principal Investigator

Team Leader, AoF Research Fellow

Institute of Biotechnology, University of Helsinki, Finland.



TOPIC: Secondary damage and time-course of microglial activation after cortical stroke in rats.

Academic credential

2012 Docent, University of Helsinki. Field: Pharmacology and drug discovery

2006 Ph.D. University of Helsinki, Faculty of Pharmacy, Finland

RESEARCH INTERESTS

Drug therapies for the diseased brain are still based on alleviating symptoms. The major challenges we face are to understand the changes of the brain functions during neurodegeneration, in order to find new treatments for devastating diseases like Parkinson's disease and stroke. By using modern biotechnological tools new effective and safe drugs can be developed. My research interest is in neurotrophic factors, how the normal brain works and how brain functions are altered in the diseased brain. Our main focus is in ischemic brain injury, Parkinson's disease and the mechanisms of neurodegeneration. We have established novel rodent models where ischemic injury is restricted to cortex enabling studies of functional recovery, brain adaptations, neuronal rearrangements, plasticity and transdifferentiation of reactive astrocytes. Stroke is the major health care problem and academic research groups have an important endeavor in finding novel drug targets that would lead to treatments to hasten the recovery after stroke. Pharmaceutical industry is showing no interest in developing new drugs for stroke and in the future the cost for society will increase. We are focused on novel neurotrophic factors cerebral dopamine neurotrophic factor (CDNF) as well as mesencephalic astrocyte-derived neurotrophic factor (MANF) as well as primate-specific Glial cell line-Derived Neurotrophic Factor Opposite Strand gene (GDNFOS) and their signaling in health and disease. Our main focus is in the mechanism of endoplasmic reticulum stress, inflammation and protein aggregations. In our research, we utilize transgenic animals, adeno-associated viral vectors, primary cultures from embryo and adult.

Invited speaker

Yu -Li Liu (劉玉麗) Ph. D.

Principal Investigator
Center for neuropsychiatric Research,
NHRI



Topic: APBB2 is associated with multiple substance use in a methadone maintenance population.

Objectives: *APBB2* (amyloid beta (A4) precursor protein-binding, family B, member 2) has been reported as a candidate gene for opioid dependence. In this study, we analyzed the genetic variants on *APBB2* in association with the methadone maintenance treatment (MMT) responses.

Methods: 344 heroin-dependent patients undergoing MMT were recruited and assessed for urine amphetamine and morphine tests, withdrawal severity, and treatment-induced side effects. The DNAs were genomewide genotyped for all patients. The single nucleotide polymorphisms (SNPs) on *APBB2* genetic region were selected for association analyses with methadone treatment responses. The *APBB2* genetic expression levels were measured by real-time polymerase chain reaction (PCR) from EBV-transformed patients' lymphoblastoids. The plasma amyloid beta-40, and 42 were measured by enzyme linked immunosorbent assays. The plasma morphine concentration was assayed by high performance liquid chromatography.

Results: Patients with urine amphetamine test positive had significantly higher percentage of positive in urine morphine tests ($P=0.005$) and insomnia side effect symptom score ($P=0.018$) than amphetamine test negative patients. In single locus association analyses, both genotypes and allele types of SNPs rs3935357, rs13126487 and rs4861075 at *APBB2* intron 6 were significantly associated with urine amphetamine test MMT patients (general linear model (GLM), $P=0.0003$, 0.001 , and 0.0002 for genotype, and 0.0003 , 0.002 , 0.002 for allele type, respectively). The promoter SNP rs78704328 is associated with plasma morphine concentration ($P=0.0003$), plasma amyloid beta-42 ($P=0.0000004$), and ratio of 42/40 ($P=0.0006$).

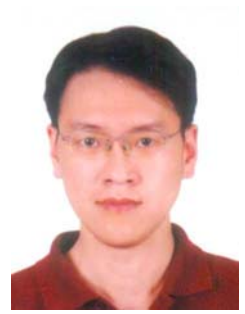
Conclusion: MMT patients who had combined use of amphetamine had higher percentage with urine morphine test positive also. The genetic variants on intron 6 of *APBB2* could be indicators for MMT patients combined use of amphetamine. The promoter rs78704328 could be indicator for combine heroin use and neurodegenerative amyloid beta-42 plasma concentration.

Invited speaker

Sheng Chang Wang (王聲昌) M.D.

Principal Investigator
Center for Neuropsychiatric Research,

Topic: Characteristics of ketamine users in Taiwan.



Invited speaker

Yun-Hsiang Chen (陳雲翔) Ph. D.

Assistant Professor
Fu-Jen Catholic University, Taiwan.



Topic: Should Methadone Addicts Worry About Flu.

Growing evidence has indicated that opioids enhance replication of human immunodeficiency virus and hepatitis C virus in cells. However, it is unknown whether opioids can increase replication of human influenza A virus (H1N1). We found that methadone but not morphine or buprenorphine could improve H1N1 replication in human lung epithelial cells. Also, treatment with methadone significantly increased H1N1 replication in mouse lungs. Our data might raise concerns regarding the possible consequence of an increased risk of serious influenza A virus infection in people who receive treatment in methadone maintenance programs.

Invited speaker

Hwei-Hsien Chen (陳慧誠) Research Associate

Principal Investigator
Center for Neuropsychiatric Research,
NHRI



Topic: Reversal effect of sodium benzoate on delayed extinction and stronger drug-primed reinstatement of methamphetamine seeking in rats prenatally exposed to morphine.

Ying-Ling Shen¹, Tzu-Yi Chan¹, Tsai-Wei Hung¹, Pao-Luh Tao¹, Ruey-Ming Liao^{2,3,4}, Ming-Huan Chan^{2,4},
and **Hwei-Hsien Chen**^{1,2}

¹Center for Neuropsychiatric Research, National Health Research Institutes, 35 Keyan Road, Zhunan, Miaoli County 35053, Taiwan

²Institute of Neuroscience, ³Department of Psychology, ⁴Research Center for Mind, Brain and Learning, National Cheng-Chi University, 64, Sec.2, ZhiNan Road, Wenshan District, Taipei City, 11605, Taiwan

Prenatal morphine (PM) affects the development of brain reward system and cognitive function. Our previous study demonstrated that PM exposure did not affect the association memory formation during acquisition of methamphetamine (MA) conditioned place preference and intravenous self administration (SA) but impaired extinction learning and increased MA-primed reinstatement in both tasks. Enhancing NMDA receptor -mediated neurotransmission has been considered a novel approach to facilitate extinction learning. Sodium benzoate, a potent, competitive inhibitor of d-amino acid oxidase, can raise the levels of d-amino acids to enhance the NMDA receptor function. The present study examined whether sodium benzoate is capable of improving the delayed extinction and stronger drug-primed reinstatement of MA SA in rats prenatally exposed to morphine. Pregnant Sprague-Dawley rats were administered with morphine or saline during embryonic days 3-20. The male adult offspring were trained intravenous SA of MA under the fixed ratio 5 schedule, thereafter placed on an extinction schedule prior to receiving cue and priming injections of 1 mg/kg (i.p.) MA. Sodium benzoate (100 mg/kg, i.p.) or saline was given after the completion of each extinction session before cue and MA priming induced reinstatement. Sodium benzoate significantly reduced the days to reach extinction criteria before cue-induced reinstatement, but did not affect the extinction learning before MA priming-induced reinstatement in the prenatal morphine- and saline-exposed rats. In addition, sodium benzoate treatment reduced the MA priming-induced reinstatement. These findings revealed that sodium benzoate treatment facilitated the consolidation of extinction learning and reduced the drug-primed reinstatement, suggesting the sodium benzoate facilitates extinction learning and has beneficial effect on extinction learning impairment and MA drug seeking after PM exposure.

Invited speaker

Chiung Yuan Ko(柯瓊媛) Ph.D.

Assistant Professor

Program for Neural Regenerative Medicine, College of Medical Science and Technology, Taipei Medical University, Taiwan



Topic:Neuroinflammation in Alzheimer's disease: function of astrocytic CCAAT/enhancer binding protein delta.

Previous studies have shown that astrocyte activation can induce reactive oxygen species production and the secretion of pro-inflammatory cytokines in an inflammatory environment, eventually causing activation and migration of microglia and macrophages, neuronal loss and enhancement of the anti-apoptotic ability of the astrocyte. Recently, immunohistochemistry demonstrated the accumulation of CCAAT/enhancer binding protein delta (CEBPD) in reactive astrocytes surrounding A β peptide deposits in AD. One CEBPD target, pentraxin-3 participates in the attenuation of macrophage-mediated phagocytosis of damaged neurons. Moreover, once astrocytic CEBPD is induced by IL-1 β , the recovered GSK3 β activity phosphorylates CEBPD at serine 167, which then promotes the migration and activation of microglia/macrophages through MCP-1 and MMP-1, respectively. Furthermore, CEBPD plays a functional role in contributing to the anti-apoptotic ability of astrocytes. The Zinc Finger Protein 179 gene is activated by CEBPD and mediates CEBPD-induced anti-apoptosis in astrocytes by collaborating with the transcription repressor PLZF to inactivate the expression of the proapoptotic genes insulin-like growth factor-binding protein 3 and BCL2-interacting killer. Finally, miR135a was responsive to the induction of CEBPD and further negatively regulated thrombospondin 1 (THBS1/TSP1) transcription by directly targeting its 3'-untranslated region in astrocytes. This discovery suggested that the CEBPD/miR135a/THBS1 axis could be a therapeutic target of AD.

Invited speaker

Feng Shie Shiun(謝奉勳) Ph.D.

Principal Investigator
Center for Neuropsychiatric Research,
NHRI



Topic: Metabolic stress in the pathogenesis of Alzheimer's disease.

The majority of Alzheimer's disease (AD) cases are sporadic, meaning that non-genetic factors largely contribute to the pathogenesis of the disease. Manifestations of insulin resistance in AD patients have led to much attention suggesting that diabetes mellitus (DM)-associated metabolic stress can be an important non-genetic risk factor in AD. However, the role of metabolic stress in the early development of AD pathology remains unclear. Recently, metabolic stress was induced by a high-fat diet and low-dose injection of streptozotocin (HFSTZ) before the appearance of β -amyloid ($A\beta$) senile plaques in APP/PS1 transgenic mice. We found that, HFSTZ treatment exacerbated $A\beta$ burden and astrocyte activation in the vicinity of $A\beta$ plaques. Moreover, we observed an upregulation of astrocytic S100B expression in the brain parenchyma of HFSTZ-treated APP/PS1 transgenic mice concurrent with increased interleukin-6 expression in cerebral microvascular cells, elevated serum $A\beta$ levels, and apparent hepatic steatosis. A significant interaction between HFSTZ and genetic background of AD was found, indicating that APP/PS1 transgenic mice are more vulnerable to HFSTZ treatment. Body weight gain, high hepatic triglyceride, and hyperglycemia were positively associated with serum $A\beta$. Importantly, HFSTZ treatment impaired nest construction and cerebral glucose metabolism in several brain regions of APP/PS1 transgenic mice during the early stage of AD. These results suggest that HFSTZ-induced peripheral metabolic stress may contribute to vascular inflammation and astrocyte reactivity in the parenchyma and may impair activity of daily living skill and cerebral glucose metabolism in APP/PS1 transgenic mice. Our data also suggests that the interplay between genetic background of AD and HFSTZ-induced metabolic stresses contributes to the development of obesity and hepatic steatosis. Alleviating metabolic stresses could be critical to prevent $A\beta$ accumulation at the early stage of AD.

Invited speaker

Yu Seong Jin(劉誠珍) Ph.D.

Principal Investigator
Center for Neuropsychiatric Research,
NHRI



Topic:Neuroprotective action of posiphen in stroke.

Phenserine, an acetylcholinesterase (AChE) inhibitor, has been used to improve cognitive function in patients with Alzheimer's disease (AD). Posiphen, a stereoisomer of phenserine, has been shown to possess neuroprotective effects in animal models of AD. Its mechanism of action is still not clear as posiphen has no anti- AChE activity. The purpose of this study is to examine the neuroprotective effect of posiphen against stroke. Primary cortical neurons (PCNs) were prepared from rat embryonic cortical tissues. Posiphen significantly attenuated glutamate – mediated loss of MAP2 immunoreactivity. Pretreatment of posiphen antagonized NMDA-induced increase in intracellular Ca^{++} . These data suggest that posiphen has a neuroprotective action against excitatory amino acid-induced neurotoxicity in culture. The protective effect of posiphen was further examined in an animal model of stroke. Adult male rats were subjected to transient (60 min) middle cerebral artery occlusion (MCAo). Posiphen or vehicle was given systemically after MCAo. Stroke animals receiving posiphen showed a significant reduction in neurological scores and infarct volume. Post-treatment with posiphen also reduced IBA1 immunoreactivity in the perilesioned area, suggesting posiphen attenuated microglia activation in stroke brain. Taken together, our data support a novel pharmacological therapy for stroke. Early post-treatment with posiphen reduced behavioral deficits, inflammation, and brain infarction in stroke animals.

Invited speaker

Wang Yun(王昀) M.D. Ph.D.

Principal Investigator

Center for Neuropsychiatric Research,
NHRI



Topic: Neuroprotective action of dopamine neuron stimulating peptide.

Dopamine neuron stimulating peptide (DNSP) is a peptide in the proregion of pro-glial cell line derived neurotrophic factor- α (pro-GDNF α). Limited reports have indicated that DNSP has a trophic effect, different from GDNF, in nigral dopaminergic neurons. The physiological function of DNSP in other brain regions or disease models has not been reported. In this study, we first characterized the distribution of DNSP. The cellular distribution of DNSP was studied in primary neuronal culture obtained from the fetal ventromesencephalic (VM) and cortical tissues. In the VM culture, all tyrosine hydroxylase (+) cells expressed DNSP. DNSP immunoreactivity was also found in other nondopaminergic neurons, such as GAD neurons. Similarly, in cortical cultures, DNSP was co-expressed in NeuN (+) or GAD (+) neurons. In both VM and cortical culture, minimal DNSP immunoreactivity was found in GFAP cells, suggesting DNSP is mainly present in neuronal cells. The modulation of DNSP mRNA expression was next examined in an animal model of stroke. Adult male rats were subjected to transient distal middle cerebral artery occlusion (MCAo) to generate ischemia in the right cerebral cortex. A time-dependent upregulation of DNSP in the lesioned side cortex peaked on days 1-2 and returned to the basal level by day 10. These data suggest that DNSP is regulated by ischemia in stroke brain. The physiological function of DNSP in the injured cerebral cortex was examined both in vivo and in vitro. In primary cortical neuronal culture, treatment with DNSP protected against the glutamate-mediated loss of MAP2 and MTT activity. Adult male rats were used for in vivo study; DNSP was given intra-cerebroventricularly at 5 min prior to distal MCAo. Treatment with DNSP significantly reduced brain infarction. Animals receiving DNSP demonstrated a significant reduction in Bederson's neurological score and body asymmetry. Since DNSP is a small peptide with 11 amino acids, it can pass through the blood-brain barrier when given intranasally. In another set of animals, DNSP was given intranasally after MCAo. Similar to the i.c.v. pretreatment, post- intranasal treatment with DNSP also significantly reduced the volume of infarction. These data suggest that DNSP is protective against ischemic brain injury. We and others previously demonstrated that ischemic brain injury induces glutamate overflow. Primary cortical neuronal cells were overexpressed gCaMP5 (a Ca^{++} probe) through AAV1 infection. Perfusion with glutamate-induced a rapid increase in $[Ca^{++}]_i$, which was significantly attenuated by the pretreatment with DNSP. In conclusion, we have demonstrated that DNSP is expressed in non-dopaminergic neurons and can be upregulated after ischemic brain injury. Pre or early post-treatment with DNSP reduced ischemic brain injury, possibly through the reduction of glutamate-mediated Ca^{++} influx.

Moderator:**Jih-Heng Li (李志恒)**

Professor

School of Pharmacy

Kaohsiung Medical University

President

Pharmaceutical Society of Taiwan

**Education:**

1973-1977, B.Pharm. School of Pharmacy, Kaohsiung Medical College, Taiwan.

1985-1987, M.Phil. Department of Environmental Medicine, New York University, U.S.A.

1987-1989, Ph.D. Department of Environmental Medicine, New York University, U.S.A.

1989-90, Postdoc., Department of Radiation Oncology, University of Pennsylvania, U.S.A.

Certification:

1977, Qualified pharmacist in Taiwan.

1981, Qualified pharmacist of public health (for civil service) in Taiwan.

Employment and Position:

Jan. 2016- President, Pharmaceutical Society of Taiwan

Aug. 2015- Professor, School of Pharmacy, Kaohsiung Medical University, Taiwan.

Aug.2009-July 2015 Professor and Dean, College of Pharmacy, Kaohsiung Medical University, Taiwan.

Jan. 2013-Dec.2014 Member, Committee of Medical Education, Ministry of Education, Taiwan

Feb.2008-July 2009 Professor and Director of Research and Development, Kainan University, Taiwan.

Sep.2005-Jan.2008 Specialist General, Department of Health (DOH) and Director of the Office for Avian Flu Control, Department of Health, Taiwan.

Jul.1999-Sep.2005 Director-General, National Bureau of Controlled Drugs(管制藥品管理局), DOH, Taiwan.

Mar.1994-Jun.1999 Director-General, National Narcotics Bureau(麻醉藥品經理處), DOH, Taiwan.

Moderator:

Pao-Luh Tao (陶寶綠) Ph.D.

Professor
Center for Neuropsychiatric Research,
NHRI



Education

- | | |
|-----------|--|
| 1968-1972 | B.S., Department of Pharmacy, College of Medicine, National Taiwan University, Taiwan, ROC |
| 1972-1974 | M.S., Graduate Institute of Biophysics, National Defense Medical Center, Taiwan, ROC |
| 1988-1991 | Ph.D., Department of Pharmacology, University of Minnesota, U.S.A. |

Research and Professional Positions Held in Chronological Sequence

- | | |
|--------------|--|
| 1974-1979 | Associate Investigator, Medical Research Department, Tri-service General Hospital, Taiwan, ROC |
| 1979-1985 | Instructor, Department of Pharmacology, National Defense Medical Center, Taiwan, ROC |
| 1985-1991 | Associate Professor, Department of Pharmacology, National Defense Medical Center, Taiwan, ROC |
| 1991-2010 | Professor, Department of Pharmacology, National Defense Medical Center, Taiwan, ROC |
| 1999-2005 | Chairman, Department of Pharmacology, National Defense Medical Center, Taiwan, ROC |
| 2010-2012 | Investigator; Division of Mental Health and Addiction Medicine, Institute of Population Health Sciences, National Health Research Institutes, Taiwan, ROC. |
| 2012-2015 | Investigator; Center for Neuropsychiatric Research, National Health Research Institutes, Taiwan, ROC. |
| 2015-present | Guest Investigator; Center for Neuropsychiatric Research, National Health Research Institutes, Taiwan, ROC. |

Research Interests

- Mechanisms of morphine tolerance and dependence
- How to prevent tolerance, dependence and chronic toxicities of morphine
- How to improve the analgesic effect of morphine
- The mechanisms of drug addiction and how to prevent or treat drug addiction

Moderator:

San-Yuan Huang (黃三原) Ph.D.

Professor
National Defense Medical Center



Graduated in medicine at the National Defense Medical Center, Taipei, Taiwan, ROC. He completed his psychiatry residency at Tri-Service General Hospital, Taipei, Taiwan. After psychiatry resident training, Dr. Huang received his Ph.D. degree also from the National Defense Medical Center. His academic research focuses on gene-gene, and gene-behavior-environment interaction in substance use disorders such as alcohol dependence, depressive alcoholism and other illegal drug abuse. After Ph.D. degree, he main academic interests/ research are in gene, brain image with SPECT and PET, psycho-immunology in addiction and mental related disorder.

Currently Dr. Huang is the president of Taiwanese society of addiction science (TSAS). He is also a professor in Psychiatry at National Defense Medical Center, and the Director of the Department of Psychiatry at the Tri-Service General Hospital. He is active in addiction services, particularly in the rehabilitation of addictive patients and promotion of mental health in the addiction.