The 10th Annual Scientific Meeting
Hong Kong Society of Biological Psychiatry

Brain and the Environment II

11-12 March 2017 (Saturday and Sunday)

Eaton Hong Kong, 380 Nathan Road, Kowloon

Programme Book

www.hksbp.org

The 10th Annual Scientific Meeting
Hong Kong Society of Biological Psychiatry
Brain and the Environment II
11-12 March 2017 | Eaton Hong Kong

Hong Kong Society of Biological Psychiatry 香港生物精神醫學會

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Welcome Message

On behalf of the Organizing Committee, I take great pleasure in inviting you to join the 10th Annual Scientific Meeting (ASM) of the Hong Kong Society of Biological Psychiatry (HKSBP). The meeting will be organized at the **Eaton Hong Kong** on **11th (Saturday afternoon & evening)** and **12th (Sunday afternoon) in March 2017**.

The thematic of this year **Brain and the Environment II** is a follow up meeting of the 9th ASM. We have invited **Prof. Meyer-Lindenberg**, who is the Director of the Central Institute of Mental Health and Chairman of the Department of Psychiatry and Psychotherapy based in Mannheim in Germany and the Professor and Chairman of Psychiatry and Psychotherapy at the University of Heidelberg in Germany, to come along to deliver 2 plenary lectures. His first topic is urbanization and mental disorders and the biology behind these phenomena which is more academically oriented. His second lecture will be on the latest advances in psychiatry and more clinically oriented. **Prof. CHANG Chuen Chung, Raymond** will discuss how environmental pollutants affect the cognitive dysfunctions and Alzheimer's disease. **Dr. YAU Suk Yu, Sonata** will share her research on promotion of cognitive performance in an animal model of autism spectrum disorder. **Prof. SO Kwok Fai** will share research findings on how motor training reduces psychomotor retardation. There will 2 free papers on state of art in biological psychiatry as presented by our distinguished young researchers from Japan.

Last but not the least, I would like to invite you to join the 2 lunch symposia on both days and the dinner symposium on 11 March. The lunch symposium on 11 March will be delivered by **Prof. Dr. Ahmad Hatim SULAIMAN** on the care of schizophrenia patients while **Dr. WONG Ming Cheuk, Michael** will talk about the psychopharmacology of partial agonists in the lunch symposium on 12 March. On 11 March **Prof. TANG Siu Wa** will lecture on smart drugs and cognitive enhancers at the dinner symposium. I look forward to meeting you at all these occasions and I am sure this educational event will be inspirational and intellectually exciting.

Dr. WONG Chi-keung
Chairperson, Organizing Committee of the 10th ASM
Hong Kong Society of Biological Psychiatry

10th ASM Organizing Committee

Chairperson
Dr. WONG Chi Keung

Members
Dr. CHEUNG Hon Kee, Henry
Dr. CHUNG Kar Kin, Albert
Dr. TAM Mo Shing, Paul
Dr. TSANG Suk Kwan, Jenny
Dr. WONG Chung Hin, Willy
Dr. YUEN Cheung Hang, Henry

Scientific Committee

Chairperson
Dr. LO Chun Wai

Members
Professor TANG Siu Wa
Professor WING Yun Kwok
Dr. WONG Ming Cheuk, Michael

HKSBP: Council Member 2016-2017

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Vice President:	**Dr. WONG Ming Cheuk, Michael**
Honorary Secretary:	**Dr. WONG Chi Keung**
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Dr. CHEUNG Hon Kee, Henry
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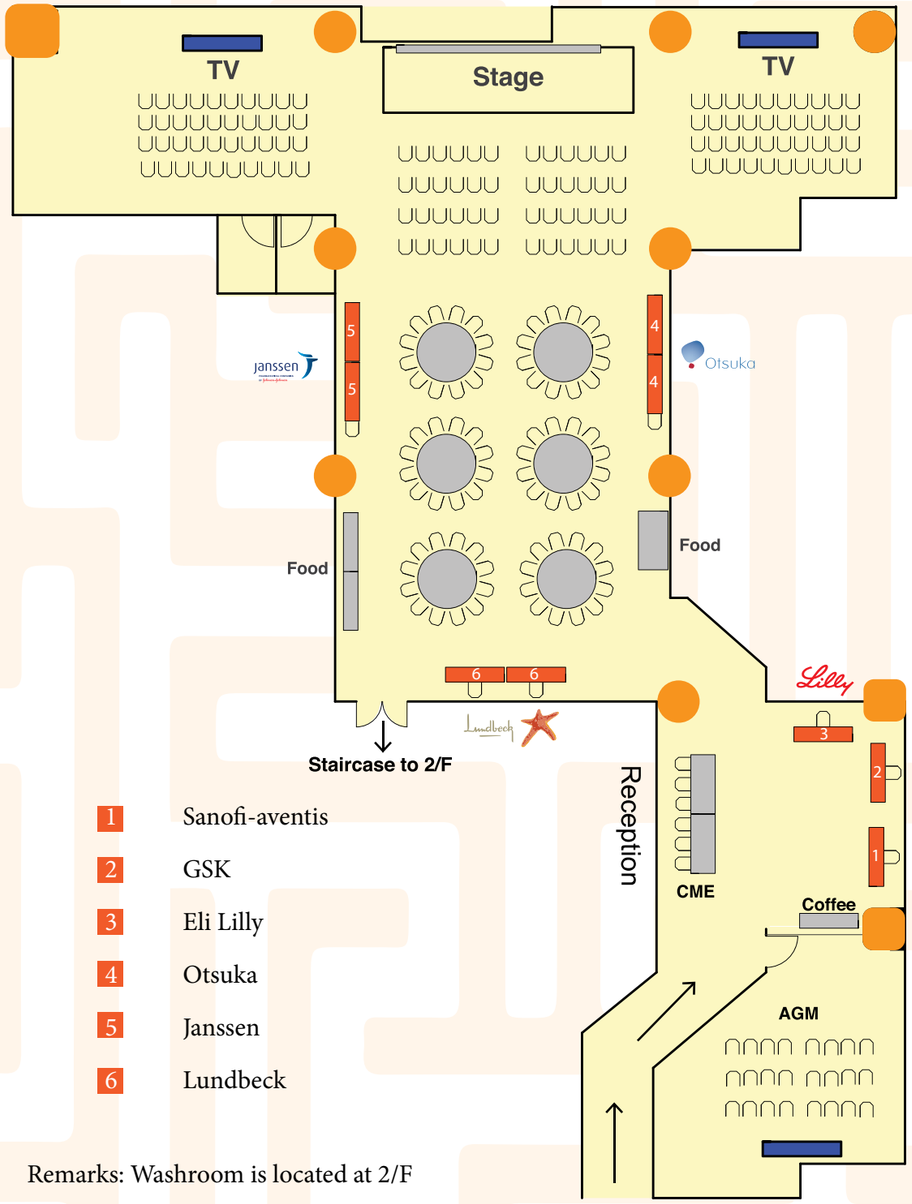
Dr. IU Pui Chuen
Dr. LO Chun Wai
Dr. TAM Mo Shing, Paul
Professor WING Yun Kwok
Dr. WONG Chung Hin, Willy
Dr. YUEN Cheung Hang, Henry

Floor Plan

Nathan Room, 1/F, Eaton Hong Kong, 380 Nathan Road, Kowloon



1 Sanofi-aventis
2 GSK
3 Eli Lilly
4 Otsuka
5 Janssen
6 Lundbeck

Remarks: Washroom is located at 2/F

Scientific Programme

Saturday, 11 March 2017

11:30-12:00	Registration
12:00 12:45-14:00	Lunch Starts **Lunch and Symposium** (Sponsored by Janssen Hong Kong) **Optimizing Care of Patients with Schizophrenia** ***Professor Dr. Ahmad Hatim SULAIMAN*** Professor, Department of Psychological Medicine, Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia Chairperson: Dr. WONG Ming Cheuk, Michael, Vice-President of HKSBP
14:00-15:15	Welcome Remarks by ***Professor TANG Siu Wa***, President of HKSBP **Plenary Lecture: Migration and Mental Disorders** ***Professor Andreas MEYER-LINDENBERG*** Director, Central Institute of Mental Health Medical Director of the Department of Psychiatry and Psychotherapy, Mannheim, Germany Professor and Chairman, Psychiatry and Psychotherapy at the University of Heidelberg, Germany Chairperson: Professor TANG Siu Wa, President of HKSBP
15:15-15:30	Exhibition and Coffee Break
15:30-16:30	**Environmental Pollutants as Risk Factors for Developing Cognitive Dysfunctions and Alzheimer's Disease** ***Professor CHANG Chuen Chung, Raymond*** Lab Chief, Laboratory of Neurodegenerative Disease, School of Biomedical Sciences, LKS Faculty of Medicine, HKU Chairperson: Dr. LO Chun Wai, Council Member of HKSBP
16:30-17:30	**Targeting Hippocampal Neuroplasticity for Promoting Cognitive Performance in an Animal Model of Autism Spectrum Disorder** ***Dr. YAU Suk Yu, Sonata*** Assistant Professor, Department of Rehabilitation Sciences, The Hong Kong Polytechnic University Chairperson: Dr. WONG Chi Keung, Honorary Secretary of HKSBP
17:30-18:00	**Free Paper: How Exercise Exerts Antidepressant-like Effect While Increasing Glucocorticoid Levels? The Pivotal Role of the Corticosterone-GR-dopamine-D2R Pathway in the Medial Prefrontal Cortex** ***Dr. CHEN Chong*** Research Scientist, RIKEN Brain Science Institute, Saitama, Japan Chairperson: Dr. CHEUNG Hon Kee, Henry, Council Member of HKSBP
18:00-18:30	**Free Paper: Hypothalamic-pituitary-adrenal Axis Function and Gene Expression Profiles as Useful Blood Biomarkers for Major Depressive Disorders** ***Dr. Hiroaki HORI*** Section Chief, Department of Adult Mental Health, National Institute of Mental Health, National Center of Neurology and Psychiatry (NCNP), Tokyo, Japan Chairperson: CHEUNG Hon Kee, Henry, Council Member of HKSBP
18:30-19:00	Cocktail Reception / HKSBP's AGM (Members only)
19:00-22:00	**Dinner and Symposium** (Sponsored by Lundbeck Hong Kong)
19:00-20:30	**Smart Drug and Cognitive Enhancers** ***Professor TANG Siu Wa***, President of HKSBP Chairperson: Dr. YUEN Cheung Hang, Henry, Council Member of HKSBP
20:30-22:00	**Conference Dinner**

Sunday, 12 March 2017

11:30-12:00	Registration
12:00 12:30-13:45	Lunch Starts **Lunch and Symposium** (Sponsored by Otsuka Pharmaceutical (HK)Ltd.) **The Psychopharmacology of Partial Agonists in Psychiatry** ***Dr. WONG Ming Cheuk, Michael***, Vice-President of HKSBP Consultant Psychiatrist and Chief of Service, Department of Psychiatry, Queen Mary Hospital Honorary Clinical Associate Professor, Department of Psychiatry, HKU Chairperson: Dr. TSANG Suk Kwan, Jenny, Council Member of HKSBP
13:45-15:00	**Plenary Lecture: Environmental Influences on Brain Circuitry : Implication for Treatment** ***Professor Andreas MEYER-LINDENBERG*** Director, Central Institute of Mental Health Medical Director of the Department of Psychiatry and Psychotherapy, Mannheim, Germany Professor and Chairman, Psychiatry and Psychotherapy at the University of Heidelberg, Germany Chairperson: Professor WING Yun Kwok, Council Member of HKSBP
15:00-16:00	**Motor Training Reduces Psychomotor Retardation via Gliogenesis in Rats with Depression-like Behaviour** ***Professor SO Kwok Fai*** Chair of Anatomy in the Department of Ophthalmology, and State Key Laboratory of Brain and Cognitive Sciences, HKU Director, Guandong-Hong Kong-Macau Institute of CNS Regeneration (GHMICR), Jinan University, Guangzhou, China Chairperson: Dr. TAM Mo Shing, Paul, Council Member of HKSBP
16:00	Closing Remarks by ***Professor TANG Siu Wa***, President of HKSBP

Lunch Symposium: 12:45-14:00, 11 March 2017, Saturday

Professor Dr. Ahmad Hatim SULAIMAN
MBBS, MPM, PhD
Professor, Department of Psychological Medicine,
Faculty of Medicine, University of Malaya,
Kuala Lumpur, Malaysia

Ahmad Hatim Sulaiman is a professor of psychiatry at the Department of Psychological Medicine, Faculty of Medicine, University of Malaya. He is also a consultant psychiatrist at the University of Malaya Medical Centre.

He is an editorial board member of the Malaysian Journal of Psychiatry and International Journal of Bipolar Disorders. He is a member of the International Early Psychosis Association, International Society of Bipolar Disorder and a life member of the Malaysian Psychiatric Association.

He has extensive experience in research and clinical trials, having been Principal Investigator for 70 pharmaceutically-sponsored international clinical trials and received study grants from numerous pharmaceutical companies.His clinical trials have been inspected by the US FDA in year 2009 and 2012. His publications include three books and 53 journal articles in ISI journals (with H-index of 8 and 203 citations).

Optimizing Care of Patients with Schizophrenia

Professor Dr. Ahmad Hatim SULAIMAN

Schizophrenia is a chronic disabling disease which is associated with functional impairment. Functional outcome is an essential concept to be kept in mind in the treatment and control of schizophrenic symptoms. Non-adherence is a major problem in the treatment of schizophrenia. In spite of recent progress in the treatment of schizophrenia during the last decades, non-adherence continues to be a frequent phenomenon, often associated to potentially severe clinical consequences and high costs.

With the aim of reducing the number of relapse and encouraging more patients to return back to society, this talk further emphasizes the role of Long Acting Injectable Antipsychotics (LAI) in helping patients recover functionally, and how it can reduce the number of hospitalization days in the hospital.

Plenary Lecture: 14:00-15:15, 11 March 2017, Saturday

Professor Andreas MEYER-LINDENBERG
Director, Central Institute of Mental Health
Medical Director of the Department of Psychiatry and Psychotherapy, Mannheim, Germany
Professor and Chairman, Psychiatry and Psychotherapy, University of Heidelberg, Germany

Prof. Meyer-Lindenberg is Director of the Central Institute of Mental Health, as well as the Medical Director of the Department of Psychiatry and Psychotherapy at the Institute, based in Mannheim, Germany, and Professor and Chairman of Psychiatry and Psychotherapy at the University of Heidelberg in Heidelberg, Germany. He is board certified in psychiatry, psychotherapy, and neurology. Before coming to Mannheim in 2007, he spent ten years as a scientist at the National Institutes of Mental Health, Bethesda, USA.

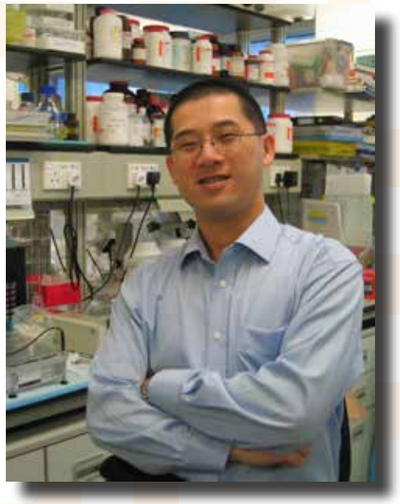
His research interests focus on the development of novel treatments for severe psychiatric disorders, especially schizophrenia, through an application of multimodal neuroimaging, genetics and enviromics to characterize brain circuits underlying the risk for mental illness and cognitive dysfunction.

Migration and Mental disorders

Professor Andreas MEYER-LINDENBERG

Global mobility, technological and economic opportunities, but also growing demographic disparities, political instability and environmental change drive an increasing flow of human migration. Over 200 million people worldwide live away from their native country, a number that is estimated to exceed 400 million by 2050. Migrants face a complex set of physical, psychological and social challenges and adjustments that can also affect their health. The health risk that has been most consistently identified concerns relative risk for schizophrenia, which is more than doubled in first- and second-generation migrants. Since increased risk has been consistently found across multiple countries and ethnicities, alternative explanations such as cultural diagnostic bias and selective migration (drift) have not been supported, and the risk increase is similar in the second generation (suggesting a ossible transgenerational epigenetic component), current explanatory models focus on the post-migratory milieu. Here, most researchers have proposed a causal role of social stress in migrants, including social evaluative threat and chronic social defeat (SD) as a consequence of social distance. Both from the host community and the migrant perspective, therefore, understanding the processes and consequences underlying social interactions between these groups is crucial and has implications from neuroscience over medicine and therapy far into the political realm. In this talk, we present translational work using validated fMRI stress induction paradigms, which provided initial evidence for specific neural alterations during social evaluative stress processing in a mixed sample of German second generation migrants. We found hyperactivity in a key regulatory system for negative emotion and stress centered on perigenual anterior cingulate cortex (pgACC) and downstream effector sites linked to schizophrenia risk such as frontoinsular cortex and VS. We also identified brain structural alterations in pgACC in migrant men. In recent work, we found that other social risk factors such as socioeconomic status and urban upbringing and living have convergent effects on social stress processing on pgACC. Taken together, these data suggest a stress-associated convergent “risk circuit” for psychosis.

Lecture: 15:30-16:30, 11 March 2017, Saturday



Professor CHANG Chuen Chung, Raymond
Lab Chief, Laboratory of Neurodegenerative Disease
School of Biomedical Sciences, LKS Faculty of Medicine
The University of Hong Kong

Dr. Chang is the Lab Chief for the Laboratory of Neurodegenerative Diseases in the School of Biomedical Sciences, member in The State Key Laboratory of Brain and Cognitive Sciences. Dr. Chang is also the Founder and Secretary of HKU Alzheimer's Disease Research Network. He organizes International Alzheimer's Disease Conference every year since 2000.

Dr. Chang's research interest is pathophysiological changes of Alzheimer's disease (AD) and the risk factors leading to AD. He has published over 120 peer-reviewed papers and 14 book chapters in these areas. His h-index is 34 by Scopus. Dr. Chang is in the Scientific Advisory Board of International AD/PD Symposium, Scientific Review Committee in Alzheimer Association, handling Associate Editor for Journal of Alzheimer's Disease, and Senior Editor for Journal of Neuroimmune Pharmacology, and Editor-in-Chief for 'American Journal of Alzheimer's Disease and Other Dementias. He is the member of editorial board of more than 20 different journals, and grant reviewer for different grant agencies/ Foundations.

Environmental Pollutants as Risk Factors for Developing Cognitive Dysfunctions and Alzheimer's Disease

Professor CHANG Chuen Chung, Raymond

Raymond Chuen-Chung Chang[1,2], Ran You[1], Clara Hiu-Ling Hung[1], Chunxia Hung[1], Janice Yuen-Shan Ho[3]

[1] Laboratory of Neurodegenerative Diseases, School of Biomedical Sciences, The University of Hong Kong, Hong Kong SAR, China
[2] State Key Laboratory of Brain and Cognitive Sciences, The University of Hong Kong, Hong Kong SAR, China,
[3] School of Nursing, Faculty of Health and Social Sciences, The Hong Kong Polytechnic University, Hung Hom, Kowloon, Hong Kong SAR, China

Increasing lines of evidence have shown that environmental pollutants have significant impact in cognitive functions for young children, elderly and patients with Alzheimer's disease (AD). While particular matters PM2.5 in the air pollutant have received much attention, silica nanoparticles are also important components that can damage neurons. Silica nanoparticles (SiO2-NPs) are typical and major components of mineral dust and many other airborne pollutants in the ambient air. In the current study, we employed fluorescein isothiocyanate-tagged SiO2-NPs (FITC-SiO2-NPs) to investigate the effects of SiO2-NPs on cognition and mood in animal model and primary culture of cortical neurons.

By using cell culture of cortical neurons, we are able to demonstrate that SiO2-NPs can markedly affect synaptic functions by inhibiting exocytosis. In laboratory animal, we performed intranasal instillation of SiO2-NPs because the major route of infiltration of silica nanoparticles is via inhalation. After two months of daily instillation, laboratory mice rendered clear impairment of short-term and spatial memory assessed by novel object recognition test and Morris water maze test. The locomotor and motor functions examined by open field test and rotarod test were preserved. Our animal experiment confirmed that the synaptic functions were impaired by SiO2-NPs. Furthermore, increased tau protein phosphorylation was observed.

Taken together, the results indicate that silica nanoparticles attribute to cognitive dysfunctions, which are most likely mediated via impairment of synaptic functions. Moreover, AD-like pathology such as tau protein phosphorylation is gradually developed. Therefore, environmental pollutants can be a risk factor for developing cognitive dysfunctions and even AD.

Acknowledgement

The study is partly supported by HKU Seed Funding for Basic Science Research (201311159171), and generous donation from Ms KW Chow.

Lecture: 16:30-17:30, 11 March 2017, Saturday

Dr. YAU Suk Yu, Sonata
Assistant Professor
Department of Rehabilitation Sciences
The Hong Kong Polytechnic University

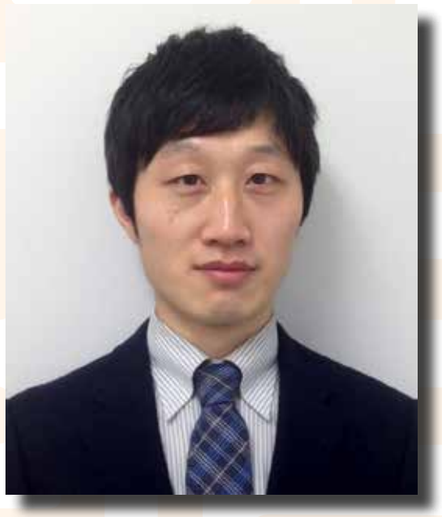
Dr. Yau obtained her Bachelor degree in Biochemistry from the Hong Kong University of Science and Technology in 2005, followed by a PhD in neuroscience in Department of Anatomy at The University of Hong Kong (HKU) in 2009. During her PhD and postdoctoral training at HKU, she investigated the underlying mechanisms of physical exercise-promoted brain health in animal models of depression. She had obtained several academic awards including postdoctoral fellowship, research fellowship, conference travel awards and outstanding presentation awards. Before joining the Department of Rehabilitation Sciences, Hong Kong Polytechnic University, she was awarded with a postdoctoral research fellowship supported by Canadian Institute of Health Research and Fragile X Research Foundation of Canada to investigate the underlying neural basis of learning and memory impairment in Fragile X Syndrome. She is interested in studying pharmacological and non-pharmacological interventions for promoting brain functions in disease animal models. Her current research projects are centered on understanding the underlying mechanisms of physical exercise-induced hippocampal neuroplasticity and examining novel therapeutic treatments for promoting cognitive performance in animal models of neurological disorders, such as autism and depression.

Targeting Hippocampal Neuroplasticity for Promoting Cognitive Performance in an Animal Model of Autism Spectrum Disorder

Dr. YAU Suk Yu, Sonata

Fragile-X syndrome (FXS) is the most common form of inherited intellectual disability and can be considered as the best characterized form of autism spectrum disorder. This disorder is caused by transcriptional repression of the gene Fmr1 which codes for the Fragile-X Mental Retardation Protein (FMRP). It is a severe disabling disease without effective treatments so far. FXS patients display varied behavioral deficits ranging from mild to severe cognitive impairment, hyperactivity, mood disorder to language problem. Transgenic mice without FMRP, Fmr1 knockout mice, show impairments in hippocampal-dependent learning and memory performance which is parallel to learning deficits observed in FXS patients. In this talk, I will present the latest findings on the neural basis underlying deficits in hippocampal neuroplasticity which has been implicated to learning and memory impairment associated with FXS. I will also highlight the potential therapeutic effects of minocycline and physical exercise training in the Fmr1 knockout mice. The findings will not only shed light on the neuropathology underlying cognitive impairment in FXS patients, but also introduce potential therapy for FXS and ASDs patients displaying cognitive impairment.

Free Paper: 17:30-18:00, 11 March 2017, Saturday



Dr. CHEN Chong, M.D., Ph.D.
Research Scientist
RIKEN Brain Science Institute
Saitama, Japan

Chong Chen, has received his medical training at the Xiangya Medical School, Central South University, Changsha, China and his Ph.D. from Department of Psychiatry, Hokkaido University Graduate School of Medicine, Sapporo, Japan. He is currently a research scientist at RIKEN Brain Science Institute, Wako, Japan.

His main research interest is the neurobiology of depression. He has been studying this from two perspectives: the Exercise-Glucocorticoid Paradox (i.e., a popular hypothesis is that depression is caused by over exposure to glucocorticoid, however, exercise increases glucocorticoid but exerts antidepressant-like effect) and computational psychiatry(i.e., building mathematical models and model-based fMRI to study the decision making deficits in depressed patients).

He has been awarded the Takakuwa Eimatsu Award for his pioneering research from Hokkaido University Graduate School of Medicine.

How Exercise Exerts Antidepressant-like Effect While Increasing Glucocorticoid Levels? The Pivotal Role of the Corticosterone-GR-dopamine-D2R Pathway in the Medial Prefrontal Cortex

Dr. CHEN Chong

1 Hokkaido University; 2 RIKEN Brain Science Institute

Introduction:

Despite the well-documented beneficial effects of exercise on depression, the precise underlying neurobiological mechanism remains unclear. Further, there even exists a paradox, which we called 'Exercise-glucocorticoid paradox'. Namely, exercise elevates the stress hormone glucocorticoid (CORT), which is believed to be a mediator of the detrimental effects of chronic stress. Therefore, there should be a mechanism by which exercise overrides the detrimental effects of CORT and achieves its beneficial effects.

Methods and results:

Experiment 1:

We replicated the 'Exercise-CORT paradox' and showed that voluntary wheel running exerts antidepressant-like effect, i.e., reduced immobility time of rats in the forced swim test (FST), while increasing basal CORT levels. Further, running upregulated overall dopamine in the medial prefrontal cortex (mPFC) without influencing other neurotransmitters such as noradrenaline, 5-HT, glutamate, etc.

Experiment 2:

The antidepressant-like effect of running in the FST was completely abolished by intra-mPFC pre-microinjection of a D2R but not D1R antagonist.

Experiment 3:

The antidepressant-like effect of running in the FST was abolished by intra-mPFC pre-microinjection of a GR antagonist. In the meantime, intra-mPFC pre-microinjection of the GR antagonist also downregulated dopamine in the mPFC of exercise rats, without any effect on 5-HT.

Discussion and conclusion:

These results, for the first time, suggest a causal pathway linking CORT-GR-dopamine-D2R, to the antidepressant-like effect of exercise. It is consistent with the observation that chronic stress reduces while antidepressants increase dopamine in the mPFC and that dopamine in the mPFC is associated with effortful behavior and motivation. We suggest that the functional rule of the elevated basal CORT is to increase medial prefrontal dopamine to exert control. Thus, the problem of chronic stress and depression may not be the increased CORT per se, but locate in the CORT-GR-dopamine-D2R pathway and the inability of increased CORT to elevate dopamine.

Acknowledgement:

This research was supported by Hokkaido University Clark Memorial Foundation.

Free Paper: 18:00-18:30, 11 March 2017, Saturday

Dr. Hiroaki HORI
Section Chief, Department of Adult Mental Health,
National Institute of Mental Health,
National Center of Neurology and Psychiatry (NCNP),
Tokyo, Japan

He is a section chief of Department of Adult Mental Health, National Institute of Mental Health, NCNP, Tokyo, Japan.

He graduated and received the M.D. degree from Kyoto University School of Medicine in 2002. He trained in psychiatry at Kyoto University Hospital and NCNP Hospital, and achieved Board Certification from the Japanese Society of Psychiatry and Neurology.

His research interest has been in psychoneuroendocrinology, neuropsychology, and genetics of major psychiatric disorders including schizophrenia, mood disorders and PTSD. After receiving the Ph.D. degree (Medicine) from Tokyo Medical and Dental University in 2009, he worked as a postdoctoral fellow at Department of Mental Disorder Research, National Institute of Neuroscience, NCNP, and then moved to the current position in 2014.

He is a member of several scientific societies including the Japanese Society of Biological Psychiatry (member and councilor). I have authored more than 100 scientific papers in peer-reviewed journals.

Hypothalamic-pituitary-adrenal Axis Function and Gene Expression Profiles as Useful Blood Biomarkers for Major Depressive Disorders

Dr. Hiroaki HORI

Introduction:
Major depressive disorder (MDD) is a common psychiatric condition and a leading cause of disability worldwide. However, the pathogenesis is yet to be fully understood, and there are no laboratory tests available to aid in its diagnosis or prognosis. Since MDD is shown to be associated with dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis and alterations in gene expression profiles, these abnormalities may serve as convenient blood biomarkers for MDD.

Methods and results:
To test this, we first conducted a series of studies on HPA axis function using the combined dexamethasone/CRH challenge test, which revealed that cortisol responses were markedly different depending on psychological characteristics of MDD patients. Specifically, patients with maladaptive personality profiles and avoidant coping style showed blunted cortisol reactivity as compared to patients without these characteristics. Similar associations between cortisol reactivity and psychological features were also observed in our healthy individuals. We then performed a microarray-based study of blood gene expression profiles in MDD patients relative to healthy controls and found a number of differentially expressed genes (DEGs) for MDD. These DEGs included several genes known to be involved in the regulation of HPA axis, such as CRHR2, FKBP4 and VAMP2. To examine the possible involvement of HPA axis in the mechanism of the transcriptome-wide identified 3 DEGs for depression, correlations between expression levels of these genes and cortisol reactivity were calculated in an expanded sample of depressed patients, which showed significant negative correlations. Furthermore, we investigated blood transcriptome signatures associated with HPA axis dysregulation using microarray, and found that pathways involved in the central nervous system, including neuroactive ligand-receptor interaction, immune system signaling, and tight junction pathways, were significantly enriched.

Conclusion:
These findings suggest an important role of HPA axis in the pathophysiology of depression and a potential usefulness of blood gene expression profiles as a biomarker for the disorder.

Dinner Symposium: 19:00-20:30, 11 March 2017, Saturday

Professor TANG Siu Wa

Director, Institute of Brain Medicine (International)
President, Hong Kong Society of Biological Psychiatry
Emeritus Professor of Psychiatry, University of California, Irvine, CA, USA
Honorary Professor of Anatomy, University of Hong Kong

Professor Tang is a psychiatrist and a pharmacologist. He graduated from the University of Hong Kong Medical School and obtained his PhD in Neurochemistry at the University of Toronto,Canada. He is Emeritus Professor of Psychiatry, University of California, Irvine, USA. His former students include chairmen and senior professors in universities in Canada, USA, Japan and China.

Professor Tang was the founder of the Hong Kong Society of Biological Psychiatry (HKSBP). Together with Professor Brian Leonard, then President of CINP and Professor S. Kasper, then President of WFSBP, Professor Tang organized a recurrent General Certificate Course in Psychopharmacology for the training of physicians in the proper use of psychotropic drugs. In 2010, Professor Tang, together with Dr Brian Leonard and Professor Joseph Zohar, President of ECNP, established the non-profit international educational organization, Institute of Brain Medicine.

Professor Tang's publications, research and teaching have been in the area of clinical and basic psychopharmacology. His research in brain noradrenaline metabolism was awarded both the Ontario provincial and Canadian national research competition prize in 1979. Professor Tang's scientific contribution to psychophrmacolgy was awarded the Kraepelin-Alzheimer Medal in 2010 and his teaching in psychopharmacology the WFSBP Excellence in Education award in 2011.

Smart Drugs and Cognitive Enhancers

Professor TANG Siu Wa

The new generation of patients may seek help from their clinicians for cognitive enhancement. Cognitive enhancers include everyday drinks like coffee and tea, food such as betel nuts, as well as medicine for the treatment of attention deficits and new compounds which were synthesized for increasing cognitive performance in examinations, shift works and other cognitive demanding tasks. Many clinicians are not familiar with this new field of medicine. This talk will discuss the neurobiology of these cognitive enhancing compounds and also their potential drug-drug interactions with other psychotropics.

Lunch Symposium: 12:30-13:45, 12 March 2017, Sunday

Dr. WONG Ming Cheuk, Michael
MB,BS(HK), MRCPsych(UK), FHKAM, FHKCPsych
Consultant Psychiatrist and Chief of Service,
Department of Psychiatry, Queen Mary Hospital
Honorary Clinical Associate Professor,
Department of Psychiatry
The University of Hong Kong
Vice-President of HKSBP

Dr. Wong is working in the Department of Psychiatry of Queen Mary Hospital in Hong Kong. He is currently the Consultant Psychiatrist and Chief of Service of the department. He is also an Honorary Clinical Associate Professor in the Department of Psychiatry of the University of Hong Kong. His main interests are in community psychiatry and rehabilitation, bipolar affective disorder and psychopharmacology. He has introduced the clubhouse model of psychiatric rehabilitation into Hong Kong and founded the Phoenix Clubhouse in the department which has helped many patients to re-integrate into the community and re-enter the job market.

Dr. Wong is also active in community services, particularly in the rehabilitation of mental patients and the promotion of mental health in the community. He serves as a co-chair of the District Task Group on Community Mental Health Support Services, Central Western, Southern and Islands District and as a member of the Rehabilitation District Co-ordinating Committee of the Central Western Southern & Island District Office, Social Welfare Department, HKSAR Government. He is also one of the council members of Fu Hong Society and a board member of the Chinese Rhenish Church Social Services Department.

He is also actively participating in the regional activities of professional societies. He is the Chairman of the Society for Advancement of Bipolar Affective Disorder, Chairman of the Hong Kong Association of Psychosocial Rehabilitation, Vice-President of the Hong Kong Society of Biological Psychiatry, and one of the council members of the Asia Network of Bipolar Disorder. He is active in the promotion of bipolar affective disorder and psychiatric rehabilitation to the professional and the public. Apart from these, Dr. Wong has organized a number of conferences for professional societies and he has been one of the members of the Local Organizing Committee of CINP World Congress Hong Kong 2010.

The Psychopharmacology of Partial Agonists in Psychiatry

Dr. WONG Ming Cheuk, Michael

With our present knowledge, we do not exactly know the aetiolgy of mental disorders. However as technology advances, we are able to find evidence of underlying abnormalities in the activities in the nerve networks in the brain in mental disorders. In some cases there are abnormally high activities while in some cases there are abnormally low or even lack of activities in certain nerve network and regions of the brain. This is supported by the fact that mental illnesses can be treated by drugs which either enhance or suppress brain activities caused by certain neurotransmitters, e.g. dopamine, serotonin. Many psychotropic drugs that we are using are either full agonists which fully activate the receptor of the naturally occurring neurotransmitters or full antagonists which block the receptors of the naturally occurring neurotransmitters so that the receptors are kept at the baseline constitutive activity. However the underlying pathology of mental disorder is not simply overactivity or underactivity of the neuronal network or neurotransmitter system. Furthermore, it is not always desirable to push the receptor response to the full extent or blocking all the activities. There is a class of drugs which is known as partial agonists which does not simply enhance or block receptor activities, but it also modifies the activity and stabilizes the system. This presentation will try to look at the role of partial agonists and their advantage over the full agonists/antagonists in the treatment of schizophrenia and mood disorder.

Plenary Lecture: 13:45-15:00, 12 March 2017, Sunday

Environmental Influences on Brain Circuitry : Implication for Treatment

Professor Andreas MEYER-LINDENBERG

(His biosketch can refer to p.10)

Mental health and the environment, especially regarding social life are intimately interrelated, as demonstrated by the frequent social deficits of psychiatric patients and the increased rate of psychiatric disorders in people exposed to social environmental adversity. In this lecture, we review emerging evidence that combines epidemiology, social psychology and neuroscience to bring neural mechanisms of social risk factors for mental illness into focus (Meyer-Lindenberg and Tost, Nat Neurosci 2011). In doing so, we discuss existing evidence on the effects of common genetic risk factors in social neural pathways and outline the need for integrative approaches to identify the converging mechanisms of social environmental and genetic risk in brain. Even for highly heritable disorders such as schizophrenia, environmental risk factors are relevant and often have higher associated risk than common genetic variants. Specifically, we discuss neuroimaging work that has begun to define neural mechanisms that might mediate environmental risk factors for schizophrenia, such as unstable social status (Zink et al., 2008), ethnic minority position, or urbanicity (Lederbogen et al., Nature 2011). Interestingly, the results of this work converge with imaging genetics studies that have characterized risk variants that by themselves show a degree of gene environment interaction or correlation, such as 5-HTTLPR (Pezawas et al., Nat Neurosci 2005) or MAO-A (Meyer-Lindenberg et al., PNAS 2006). This convergence on a systems-level suggests neural mechanisms by which environmental adversity might be reflected in an inability to process negative emotions in the context of the processing of the social environment. This systems-level definition also aids in constraining new approaches to ameliorate environmental risk, either through environmental interventions (Lederbogen et al., Nature 2011) or through molecular approaches such as prosocial neuropeptides (Meyer-Lindenberg et al., Nat Rev Neurosci 2011).

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Lecture: 15:00-16:00, 12 March 2017, Sunday

Professor SO Kwok Fai

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Director of GHM Institute of CNS Regeneration at Jinan University, Guangzhou, China; Chair of Anatomy in the Department of Ophthalmology and the State Key Laboratory of Brain and Cognitive Sciences, Jessie Ho Professor in Neuroscience, The University of Hong Kong; (http://www.eyeinst.hku.hk/Prof_So.htm), member of the Chinese Academy of Sciences, member of the Advisory Committee, Ministry of Education/ 2011 Program, member of Biolgical and Medicine Council/ Ministry of Education, member of Consultative Committee/ the national 973 Program/ major national research funding program in China (www.973.gov.cn/), Director of China Spinal Cord Injury Network (ChinaSCINet), Co-Chairman of the Board of Director of the ChinaSCINet (www.chinascinet.org), and Editor-in-Chief of Neural Regeneration Research (www.nrronline.org). Received PhD degree from MIT. He is one of the pioneers in the field of axonal regeneration in visual system. He was the first to show lengthy regeneration of retinal ganglion cells in adult mammals with peripheral nerve graft. He is currently using multiple approaches to promote axonal regeneration in the optic nerve and spinal cord. His team identifies neuroprotective and regenerative factors including: exercise, wolfberry, trophic factors, peptide nanofiber scaffold, and environmental manipulation. 1995 obtained the Natural Science Award of the National Natural Science Foundation of China. 1999 was elected Member of the Chinese Academy of Sciences. 2015 was elected US National Academy of Invention Fellow. He is the author and co-author of over 390+ publications (http://scholar.google.com/citations?hl=en&user=SUPKYiQAAAAJ&view_op=list_works); co-inventors of 25 patents.

Motor Training Reduces Psychomotor Retardation via Gliogenesis in Rats with Depression-like Behaviour

Professor SO Kwok Fai

Depressed patients with constrained gross and fine movements and latency in response have been regarded as having psychomotor retardation symptom. Alterations in the white matter integrity have been reported to be related to the symptom in patients and have further suggested the possible involvement of oligodendrocyte function and myelination in the symptom. However, the aforementioned phenomena have not been fully investigated. Additionally, motor training has previously been shown to improve motor performance in depressed patients with motor impairments, but the exact beneficial role of motor training on alleviating this symptom is still unclear.

Sprague-Dawley rats were treated with either vehicle or corticosterone (stress hormone in rodents), and were assigned into groups with or without rotarod training continuously for 14 days. Bromodeoxyuridine (BrdU) were injected during the last 3 days for tracing proliferating cells in the motor cortex. Our results showed that corticosterone induced both depression-like behaviors and motor deficits in animals, whereas these symptoms could be alleviated by rotarod training. In association with the behavioral changes, the BrdU positive cells were found to be altered obviously in the layer I of the motor cortex only under stress whereas this decrease could be reversed by rotarod training. These BrdU positive cells were mostly co-expressed with neuronal-glia antigen-2 (NG2), the percentage of co-expression is enhanced by rotarod training and altered by stress. We have further found that stress could reduce expression of myelin-related proteins in layer I which could be also increased by rotarod training.

Furthermore, we have found that these proliferating cells could possibly involved in the neural circuitry of motor activity, which these cells were activated during the rotarod training under both control and stress condition as they expressed c-fos and egr-1 (immediate-early gene markers) upon stimulation. However, the activation level was found to be lowered under stress.We have applied anti-mitotic drug for blocking these proliferating cells to determine their functional role which the rotarod effects were abolished and the motor performance was impaired. These findings may not only provide the importance of the glial cells in the layer I of the cerebral cortex in relation with psychiatric illness which has not been previously reported, but also provide a new insight into other illnesses which also share similar impairments in motor functions.

Notes to Delegates

Meeting Organizer
Hong Kong Society of Biological Psychiatry

Meeting Secretariat
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E-mail: enquiry@hksbp.org

Meeting Date
11-12 March 2017, Saturday and Sunday

Meeting Venue
Nathan Room, 1/F Eaton Hong Kong,
380 Nathan Road, Kowloon, Hong Kong

On-site Registration
The registration counter is located at the entrance of meeting room. For on-site registration, payment must be made in cash in HK dollars.

Registration Fees

HKSBP Members	Free of charge
Non-HKSBP Members	HKD 450
Students*	HKD 50

*It is limited to Undergraduates & Postgraduates of Neuro-science, Mental Health and Medicine related subjects. An official document from the appropriate department for verification is required.

Registration Entitlement
Fully registered participants are entitled to:
- Entry to all scientific sessions
- Visit the exhibition
- A full set of official publications
- A certificate of attendance
- Attend the symposium dinner, lunch and tea refreshments

Identification Badge
Each participant will receive a badge and a programme book upon check-in at 11:30 on both dates. The registration counter is located at entrance of meeting room. Please wear your identification badge at all times during the event, as it serves as your admission to all scientific sessions, tea refreshments, lunch and dinner.

Academic Accreditation
Continuing Medical Education (CME) credits have been applied from different medical colleges in Hong Kong. To obtain CME accreditation, please signify your attendance at the CME sign-in desk, which is located at the registration counter.

Official Language
The official language of this meeting is English. No simultaneous interpretation will be provided.

Exhibition
The exhibits are located at the same floor as meeting venue. The opening hours of the exhibition runs from 12:00 – 18:30 on 11 March 2017 and 12:00 – 16:00 on 12 March 2017. The dinner symposium on 11 March is exclusively sponsored by Lundbeck. And the 2 lunch symposia are sponsored by Janssen and Otuska respectively on 11 and 12 March.

Meal Arrangement
Tea break, lunch and dinner will be served in Nathan Room, 1/F.

Insurance
The organizing committee of the 10th ASM does not responsible for personal accident and/or damage to the property of participants. Participants should make their own arrangement for personal insurance.

Lost and Found
Please take good care of your personal belongings. Do not leave them unattended. Neither the Meeting Organizer nor the Meeting Secretariat will be responsible for any loss or damage of your personal properties. Should you require any assistance, please contact our staff at the registration counter.

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No photo taking, audio recording and video shooting are allowed in the meeting rooms unless permission is granted.

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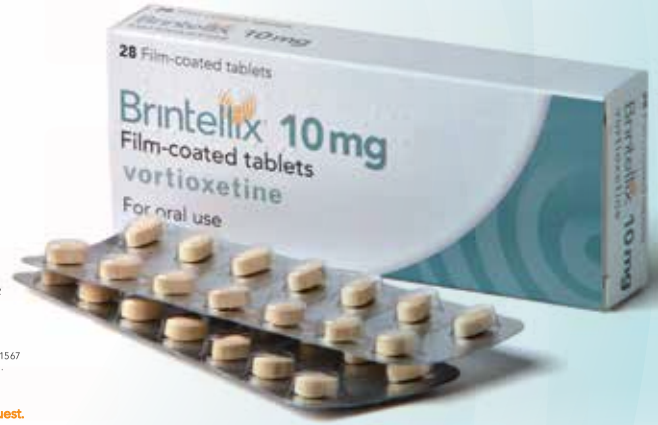

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