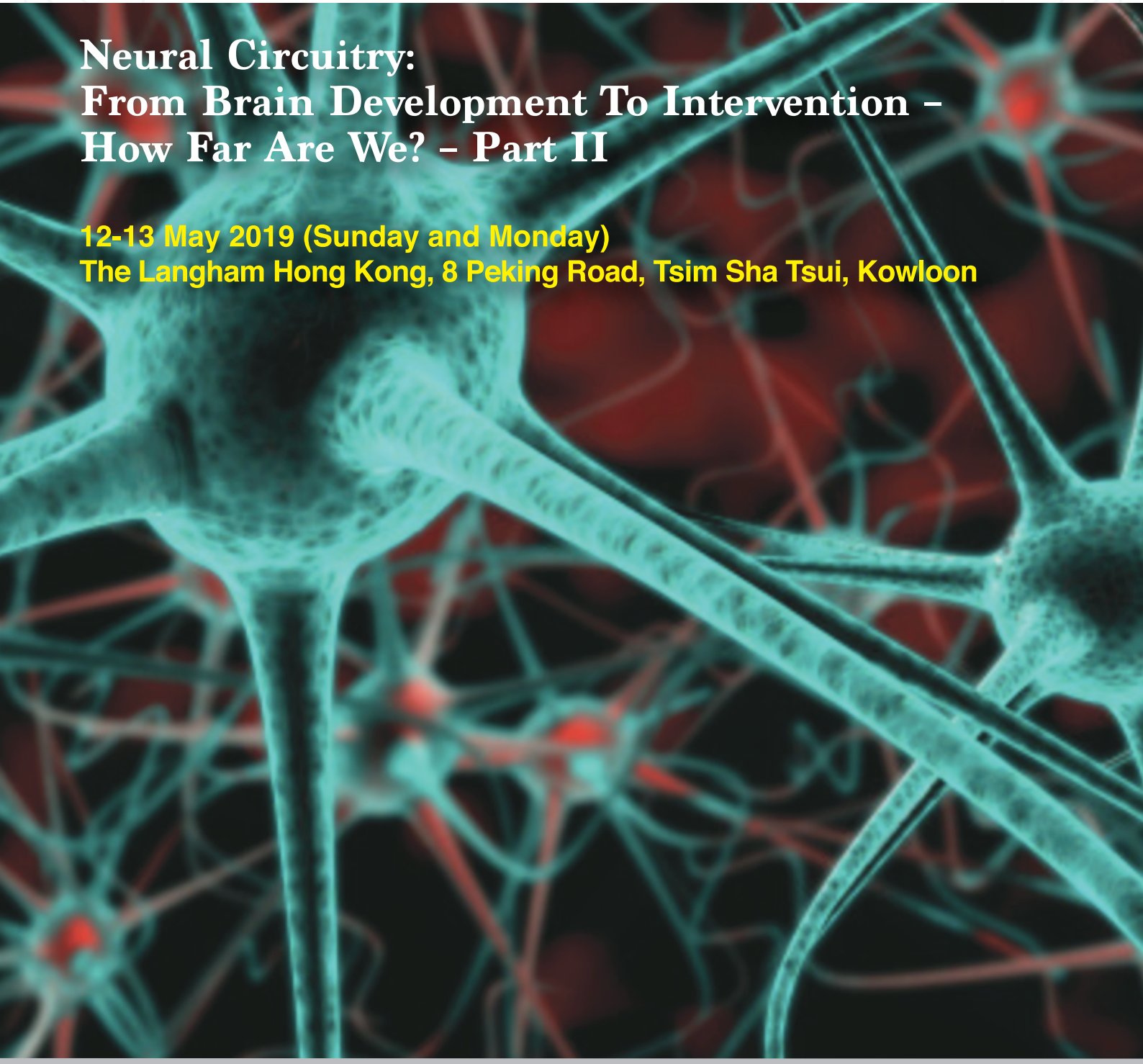
The 12th Annual Scientific Meeting
Hong Kong Society of Biological Psychiatry

Neural Circuitry: From Brain Development To Intervention – How Far Are We? – Part II

12-13 May 2019 (Sunday and Monday)
The Langham Hong Kong, 8 Peking Road, Tsim Sha Tsui, Kowloon



Programme Book

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Table of Content

Welcome Message

On behalf of the Organizing Committee, we take great pleasure in inviting you to participate in the 12th Annual Scientific Meeting (ASM) of the Hong Kong Society of Biological Psychiatry (HKSBP). The meeting will be organized on **12th (Sun) and 13th (Mon) in May 2019 at The Langham Hong Kong, Tsim Sha Tsui, Kowloon.**

The theme of this year is **Neural Circuitry: From Brain Development To Intervention – How Far Are We? – Part II**, which is a follow up meeting of the 11th ASM as we received lots of good feedbacks in the last meeting. **Prof. Anthony A. GRACE** is re-invited to give talks on his latest research updates on brain vulnerability to disorder. On Sunday, he will talk about **Adolescent Stress as a Risk Factor for the Development of Schizophrenia**; the topic of the following day is **Stress Susceptibility in Adulthood and the Development of Depression**. Currently, Prof Grace is a Distinguished Professor of Neuroscience and a Professor of Psychiatry and Psychology at the University of Pittsburgh, USA. In addition, **Prof. SO Kwok Fai** will give a plenary lecture on **The Retina-vLGN/IGL-habenula Pathway Underlies the Anti-depressive Effects of Light Therapy**, it is a new and interesting topic about brain circuitry on looming behavior. Prof So is currently the Director of Guangzhou-Hong Kong-Macau Institute of CNS Regeneration, Jinan University, Guangzhou, China. Further titles include **What is Consciousness? by Prof. LEE Mei Chu Tatia** from the University of Hong Kong; **Dendritic Spine Remodeling in Frontal Cortex Regulates Memory Functions: Neuropathology and Exercise Intervention by Dr. Zhang Li** from Jinan University, Guangzhou, China; **Procedures Targeting Memory Labile Stages to Erase Drug and Fear Memories by Prof. LU Lin** from Peking University and **Ketamine and ECT – An Added Benefit? by Dr. HE Hongbo** from Guangzhou Hui-Ai Hospital, China.

As usual, there is a free paper presentation session to encourage young researcher to participate in biological psychiatry research, this year we have a local researcher from The Hong Kong Polytechnic University. Last but not the least, lunch symposia will be provided on both days, we will have **Prof TANG Siu Wa** talks about **Natural to Designer Drugs for Brain Disorders** on Sunday and **Prof Robin EMSLEY** will give talk on **Changing the Course of Schizophrenia: Applying New Knowledge to Clinical Practice** on Monday.

We look forward to meeting at this educational, inspirational and intellectually exciting event.

Yours sincerely,

Dr. WONG Chi keung
Chairperson, Organizing Committee of the 12th ASM
Hong Kong Society of Biological Psychiatry

12th ASM Organizing Committee

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Scientific Committee Members:	**Dr. CHUNG Kar Kin, Albert** **Dr. LO Chun Wai** **Prof. TANG Siu Wa** **Prof. WING Yun Kwok** **Dr. WONG Ming Cheuk, Michael**
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12-13 May 2019 (Sunday and Monday)
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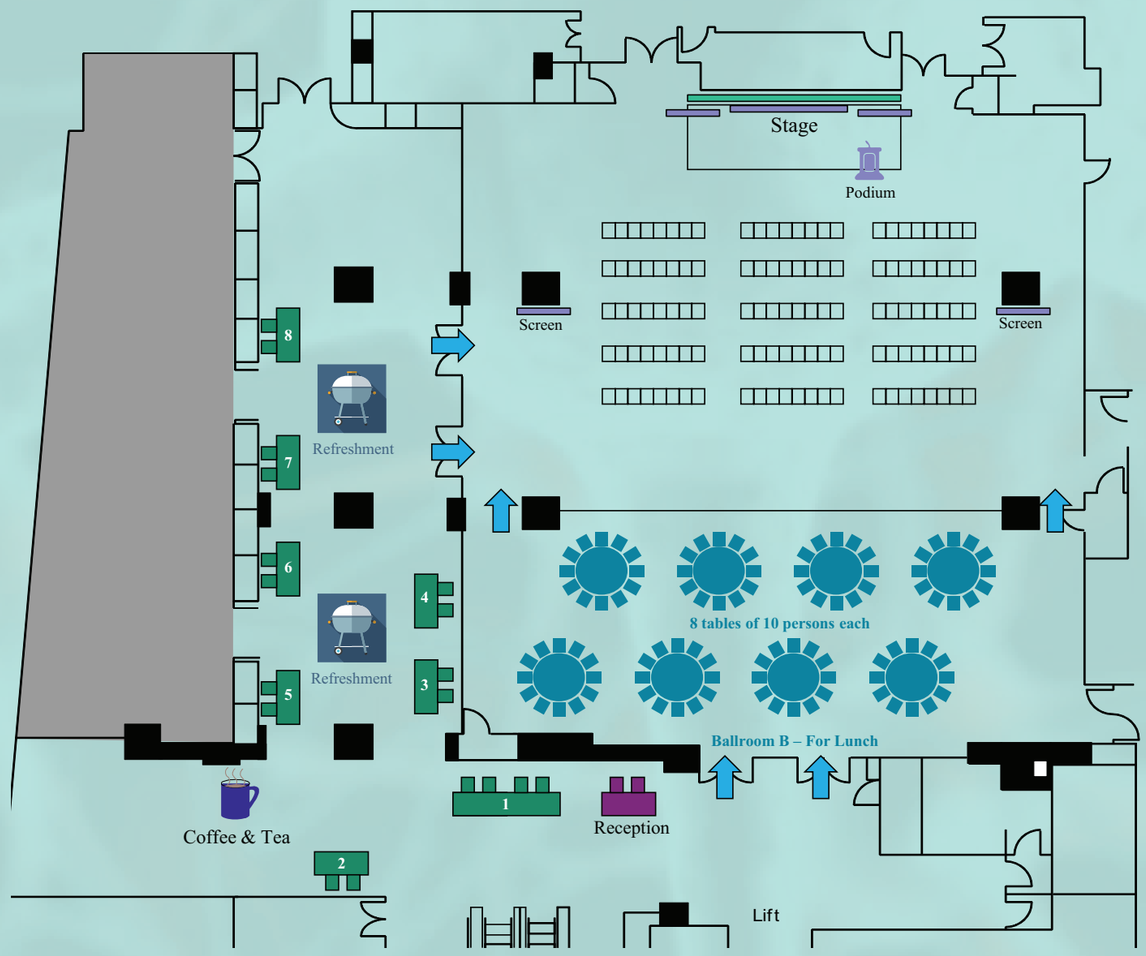
Floor Plan

Grand Ballroom, 2/F, The Langham Hong Kong, 8 Peking Road, Tsim Sha Tsui, Kowloon



1 Lundbeck
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4 gsk
5 LEE'S PHARM. 李氏大藥廠
6 Otsuka
7 SANOFI
8 ZUELLIG PHARMA

Neural Circuitry: From Brain Development To Intervention – How Far Are We? – Part II
Scientific Programme

Time	12 May 2019, Sun, Grand Ballroom, 2/F, The Langham Hong Kong
11:30-12:00	Registration
12:00	Lunch Starts
12:45-13:45	**Lunch symposium: Natural to Designer drugs for Brain Disorders** **Prof. TANG Siu Wa** Emeritus Professor of Psychiatry, University of California, Irvine, USA Current Past and Founding President of HKSBP **Chairperson: Dr. LEUNG Wai Ching, Private Psychiatrist** (*Sponsored by Lundbeck Hong Kong*)
14:00-14:30	**Free Paper: AdipoRon Ameliorates Streptozotocin-induced Impairment in Cognitive Impairment and Adult Hippocampal Neurogenesis** **Dr. YAU Suk Yu, Sonata** Department of Rehabilitation Sciences, The Hong Kong Polytechnic University **Chairperson: Dr. TAM Mo Shing, Paul, Council Member of HKSBP**
14:30-15:30	**Lecture 1: What is Consciousness?** **Prof. LEE Mei Chun, Tatia** May Endowed Chair Professor of Neuropsychology Director of the State Key Laboratory of Brain and Cognitive Sciences, The University of Hong Kong **Chairperson: Dr LO Chun Wai, Council Member of HKSBP**
15:30-16:00	Exhibition and Tea Break
16:00-17:00	**Plenary Lecture 1: Adolescent Stress as a Risk Factor for the Development of Schizophrenia** **Prof. Anthony A. GRACE** Distinguished Professor of Neuroscience Professor of Psychiatry and Psychology, University of Pittsburgh, United States of America **Chairperson: Prof. TANG Siu Wa, Current Past and Founding President of HKSBP**
Time	**13 May 2019, Mon, Grand Ballroom, 2/F, The Langham Hong Kong**
08:30-09:00	Registration
9:00-10:00	**Plenary Lecture 2: The Retina-vLGN/IGL-habenula Pathway Underlies the Anti-depressive Effects of Light Therapy** **Prof. SO Kwok Fai** Director, GHM Institute of CNS Regeneration, Jinan University, Guangzhou, China **Chairperson: Dr. WONG Chi Keung, Council Member of HKSBP**
10:00-10:45	**Lecture 2: Dendritic Spine Remodeling in Frontal Cortex Regulates Memory Functions: Neuropathology and Exercise Intervention** **Dr. ZHANG Li** Associate Professor, GHM Institute of CNS Regeneration, Jinan University, Guangzhou, China **Chairperson: Dr. CHEUNG Hon Kee, Henry, Hon. Secretary of HKSBP**
10:45-11:15	Exhibition and Tea Break
11:15-12:15	**Plenary Lecture 3: Stress Susceptibility in Adulthood and the Development of Depression** **Prof. Anthony A. GRACE** Distinguished Professor of Neuroscience Professor of Psychiatry and Psychology, University of Pittsburgh, United States of America **Chairperson: Dr. WONG Ming Cheuk, Michael, President of HKSBP**
12:15	Lunch Starts
13:00-14:00	**Lunch Symposium: Changing the Course of Schizophrenia: Applying New Knowledge to Clinical Practice** **Prof. Robin EMSLEY** Professor of Psychiatry, Faculty of Medicine and Health Sciences, University of Stellenbosch, Cape Town, South Africa **Co-chairpersons: Dr. TANG Man Ho, Private Psychiatrist; Dr. CHUI Mo Ching, Psychiatrist, Consultant of Queen Mary Hospital** (*Sponsored by Janssen Hong Kong*)
14:15-15:00	**Lecture 3: Procedures Targeting Memory Labile Stages to Erase Drug and Fear Memories** **Prof. LU Lin** Director & Professor, Institute of Mental Health/Peking University Sixth Hospital, Peking University, China Director & Professor, National Institute on Drug Dependence, Peking University, China **Chairperson: Dr. TSANG Suk Kwun, Jenny, Hon. Treasurer of HKSBP**
15:00-15:45	**Lecture 4: Ketamine and ECT - An Added Benefit?** **Dr. HE Hongbo** Associate Chief Psychiatrist, Head of Department of Research and Education, Head of Department of Psychosomatic Medicine of Guangzhou Hui-Ai Hospital, China **Chairperson: Dr. YEE Kay Cheuk, Kenneth & Dr. PAO Sze Yuan, Ronnie, Private Psychiatrists**
15:45-16:15	**AGM** (*For HKSBP Members ONLY*)

Lunch Symposium: Natural to Designer drugs for Brain Disorders
12:45-13:45, 12 May, 2019, Sunday

Prof. TANG Siu Wa

Professor Tang is a pharmacologist and a psychiatrist by training. He graduated from the University of Hong Kong medical school and obtained post graduate training in psychiatry, laboratory neurochemistry and pharmacology at the University of Toronto and University of California, obtaining his PhD, MBA, and specialist qualifications. He was Head of Psychopharmacology at the Clarke Institute of Psychiatry, University of Toronto in the 1980s and Chairman of Psychiatry, University of California, Irvine, USA in the 1990s and is now Emeritus professor of Psychiatry. He researched and published in the areas of clinical psychiatry basic and clinical psychopharmacology.

Abstract

There are many choices of remedies for brain disorders nowadays. They vary from natural to man-made products and from over-the-counter to prescription items. Both clinicians and patients are subjected to promotional, semi- or pseudo scientific and scientific information, all of which are sometimes difficult to verify without professional knowledge. This talk will present a systematic approach for evaluation of all remedies for brain disorders, and examine the science behind such an approach.

Free Paper: AdipoRon Ameliorates Streptozotocin-induced Impairment in Cognitive Impairment and Adult Hippocampal Neurogenesis
14:00 – 14:30, 12 May, 2019, Sunday

Dr. YAU Suk Yu, Sonata

Dr. Sonata Suk-yu Yau is currently an Assistant Professor in Department of Rehabilitation Sciences at Hong Kong Polytechnic University, Hong Kong. She obtained her Bachelor degree in Biochemistry from the Hong Kong University of Science and Technology in 2005, followed by a PhD degree in neuroscience in Department of Anatomy at The University of Hong Kong (HKU) in 2009. After her two years postdoctoral training at HKU, she moved to Division of Medical Sciences at University of Victoria, British Columbia, Canada with a post-doctoral fellowship awarded by Canadian Institute of Health Research and Fragile X Research Foundation of Canada. She is currently a short-term visiting Assistant Professor at Connecticut Mental Health Center at Yale University and an Honorary Assistant Professor at School of Biomedical Science, LKS Faculty of Medicine at HKU. She has been investigating the underlying mechanisms of physical exercise-promoted brain health in animal models including depression, diabetes. She also studies how hippocampal dysfunction can lead to cognitive impairment in neurodevelopmental disorders e.g. Fragile X Syndrome. She is interested in studying pharmacological and non-pharmacological interventions to promote brain functions using different diseased animal models. Her current research projects are centered on understanding the underlying mechanisms of physical exercise-induced brain health and examining novel therapeutic treatments for promoting brain health in animal models with neurological disorders. She has ample experience in animal behavioral, cellular and electrophysiological experiments by using animal models. So far, she has published 36 articles, 4 reviews and 3 book chapters, with H index 16. Within the first three year being an independent investigator at Hong Kong Polytechnic University, she has obtained three external research grants. Her funded projects involve active collaboration with internationally well-respected researchers from Hong Kong, mainland China, US, Canada and Australia.

Abstract

Diabetic patients have an increased risk for having cognitive impairment and developing depression. Physical exercise is an effective therapeutic for cognitive impairment such as depression and dementia. Cognitive impairment is often associated with neuronal loss and reduced synaptic plasticity in the brain. Our previous work has demonstrated that adiponectin is required for the antidepressant effects of exercise. Recently, AdipoRon, an adiponectin receptor agonist, is effective in treating diabetes in mouse model. Here we sought to examine whether AdipoRon acts as an exercise mimetic to restore impairment in learning and memory, and adult hippocampal neurogenesis associated with diabetes.

Methods

Six-week old diabetic and control C57BL6/J male mice received 20 mg/kg AdipoRon or voluntarily wheel running continuously for two weeks, followed by open field test, Y-maze test to anxiety and learning and memory performance. Immunohistochemical analysis with cell proliferation marker: Ki67 and immature neuronal marker: doublecortine was performed to examine changes of hippocampal adult neurogenesis. Electrophysiology was performed to measure changes in synaptic plasticity.

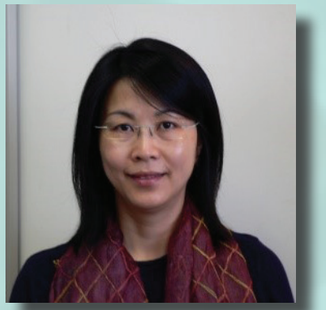
Results

Our behavioural test results demonstrated that both AdipoRon and exercise restored spatial recognition memory deficit in diabetic mice. These behavioural benefits were associated with enhancement in cell proliferation and long-tern potentation in the hippocampal dentate gyrus. However, AdipoRon did not mimic the effect of exercise on promoting survival and neuronal differentiation in diabetic mice.

Conclusion

Our data suggested that chronic administration with AdipoRon is effective in promoting learning and memory performance, which is likely linked to enhanced hippocampal cell proliferation and synaptic plasticity. However, AdipoRon can only partly mimic the effects of physical exercise on promoting hippocampal neurogenesis.

Lecture 1: What is Consciousness?
14:30-15:30, 12 May, 2019, Sunday



Prof LEE Mei Chun, Tatia

Tatia Lee is a May Endowed Chair Professor in Neuropsychology and Director of the State Key Laboratory of Brain and Cognitive Sciences at The University of Hong Kong. She is an elected Fellow of the American College of Professional Neuropsychology, American Psychological Association, and American Psychological Society.

Abstract

Consciousness has become a significant topic of interdisciplinary research. It is a construct that is hard to be fully defined. In medicine, consciousness may be reflected by patient's arousal and responsiveness. In neuropsychology, consciousness may be understood through phenomena such as anosognosia, blindsight, and altered states of consciousness induced by substances or interventions (e.g. hypnosis). Through reviewing how consciousness is being understood from both clinical and neuropsychological perspectives, there may be insight about the neural and psychological meanings and correlates of consciousness.

Plenary Lecture 1: Adolescent Stress as a Risk Factor for the Development of Schizophrenia
16:00-17:00, 12 May, 2019, Sunday

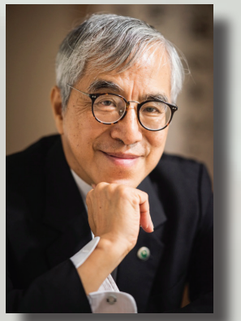
Prof. Anthony A. GRACE

Dr. Anthony A. Grace is a Distinguished Professor of Neuroscience and a Professor of Psychiatry and Psychology at the University of Pittsburgh. He has been involved in translational research related to the dopamine system for over 40 years. His early work pioneered the mode of action of antipsychotic drugs and the identification and characterization of dopamine-containing neurons. His current work involves novel treatments for schizophrenia and its prevention and the role of dopamine in affective disorders. He has published more than 300 articles and is cited more than 40,000 times. Dr. Grace has received several awards for his research, including the William K. Warren Award for Excellence in Schizophrenia Research, the Paul Janssen Schizophrenia Research Award and the Lilly Basic Scientist Award from the CINP, the Efron Award and the Axelrod Award from the ACNP, the Gold Medal award from the SOBP, the Outstanding Basic Research award from the SIRS.

Abstract

Substantial evidence demonstrates that schizophrenia involves a dysregulated dopamine system driven by overactivity in the hippocampus. Schizophrenia brains show a substantial loss of parvalbumin GABAergic interneurons in the hippocampus which likely drives the hyperactivity, leading to an over-responsive dopamine system. Our studies suggest that when the hippocampus is hyperactive the dopamine system is hyper-responsive to stimuli, which can underlie psychosis. A major question is why there is interneuron loss in the hippocampus. Parvalbumin interneurons early in life are susceptible to damage due to stress. In a developmental disruption model of schizophrenia, we found that prepubertally these rats are more anxious, hyper-responsive to stress, and show hyperactivity in the amygdala; furthermore relieving the stress early in life prevents the transition to "psychosis." Thus, schizophrenia susceptibility may be due to heightened sensitivity to the deleterious effects of stress. Indeed, multiple stressors given during this sensitive period to normal rats leads to the schizophrenia phenotype. Moreover, elimination of the ability of the medial prefrontal cortex to regulate stress enables minor stressors to yield the schizophrenia phenotype. In contrast, multiple stressors given to adult rats result in a depression-like phenotype. However, if the critical developmental period is first re-opened in the adult rat via histone decarboxylase inhibition, the same stressors now yield a schizophrenia phenotype. This suggests that genetic predisposition does not cause schizophrenia, but instead causes the individual to be hypersensitive to the deleterious effects of stress. Moreover, stress susceptibility may be a common link in familial risk for schizophrenia and depression. Therefore, controlling stress early in life in susceptible individuals may be an effective means to prevent transition to schizophrenia later in life.

Plenary Lecture 2: The Retina-vLGN/IGL-habenula Pathway Underlies the Anti-depressive Effects of Light Therapy
09:00-10:00, 13 May, 2019, Monday



Prof. SO Kwok Fai

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. 2017 elected a member of DABI (Dana Alliance for Brain Initiatives, www.dana.org). He is the author and co-author of over 430+ publications (http://scholar.google.com/citations?hl=en&user=SUPKYiQAAAAJ&view_op=list_works) ; co-inventors of 25 patents.

Abstract

Light plays a pivotal role in the regulation of affective behaviors. However, the precise circuits that mediate the impact of light on depressive- like behaviors are not well understood. Here, we show that light influences depressive-like behav-iors through a disynaptic circuit linking the retina and the lateral habenula (LHb). Specifically, M4- type melanopsin-expressing retinal ganglion cells (RGCs) innervate GABA neurons in the thalamic ventral lateral geniculate nucleus and intergen- iculate leaflet (vLGN/IGL), which in turn inhibit CaMKIIa neurons in the LHb. Specific activation of vLGN/IGL-projecting RGCs, activation of LHb- projecting vLGN/IGL neurons, or inhibition of post- synaptic LHb neurons is sufficient to decrease the depressive-like behaviors evoked by long- term exposure to aversive stimuli or chronic social defeat stress. Furthermore, we demonstrate that the antidepressive effects of light therapy require activation of the retina-vLGN/IGL-LHb pathway. These results reveal a dedicated retina-vLGN/ IGL-LHb circuit that regulates depressive-like be- haviors and provide a potential mechanistic expla- nation for light treatment of depression.

Lecture 2: Dendritic Spine Remodeling in Frontal Cortex Regulates Memory Functions: Neuropathology and Exercise Intervention
10:00-10:45, 13 May, 2019, Monday

Dr. ZHANG Li

Dr. Li Zhang is currently an Associate Professor in Guangdong-Hong Kong-Macau Institute of CNS Regeneration, Jinan University. After obtaining BSc degree in University of Hong Kong (1st class honor) and PhD degree in Biological Sciences from HKU, Dr. Li Zhang joined Jinan University as one principal investigator since 2014. His current research interest manly focuses on motor system and psychiatric disorders, including neuropathology of central motor disorder, comorbid of psychiatric diseases and motor dysfunction, and the intervention of mental illness using physical exercise. Dr. Li Zhang has published more than 10 papers since 2014, including those on Neuropsychopharmacology and Translational Psychiatry.

Abstract

Physical exercise training has well-known effects on the improvement of cognitive functions and mental status, the neurobiological mechanism, however, is still poorly understood. Current knowledge mainly focuses on the facilitation of hippocampal neurogenesis, or neuroprotection against neurotoxicity by exercise. On the other hands, we know little about the dynamic change of dendritic spines, which form the structural basis of neural plasticity and learning memory. Our group generated mouse chronic restraint stress models and found excess pruning of cortical spines by in vivo 2-photon transcranial imaging, in association with deficits of sensory dependent working memory. The adoption of treadmill exercise training effectively recued spine pruning and recovered memory deficits. Further molecular studies suggested elevation of brain derived neurotrophic factor (BDNF) in exercised brain. At the downstream of BDNF, treadmill training persistently activates mechanistic target of rapamycin (mTOR) signaling pathway, which helps to facilitate the expression of synaptic proteins. Moreover, exercise training increases spine formation rate in cortical regions and potentiated calcium spikes to improve synaptic plasticity, thus contributing to better acquisition of motor skill memory. Using pharmacological inhibition, we demonstrated that mTOR activation is necessary for exercise-improved neural plasticity. Those results enrich our understandings for environmental influences on neural plasticity, and further support the intervention of psychiatric disorders or cognitive dysfunctions using exercise paradigms.

Plenary Lecture 3: Stress Susceptibility in Adulthood and the Development of Depression
11:15-12:15, 13 May, 2019, Monday

Prof. Anthony A. GRACE

Dr. Anthony A. Grace is a Distinguished Professor of Neuroscience and a Professor of Psychiatry and Psychology at the University of Pittsburgh. He has been involved in translational research related to the dopamine system for over 40 years. His early work pioneered the mode of action of antipsychotic drugs and the identification and characterization of dopamine-containing neurons. His current work involves novel treatments for schizophrenia and its prevention and the role of dopamine in affective disorders. He has published more than 300 articles and is cited more than 40,000 times. Dr. Grace has received several awards for his research, including the William K. Warren Award for Excellence in Schizophrenia Research, the Paul Janssen Schizophrenia Research Award and the Lilly Basic Scientist Award from the CINP, the Efron Award and the Axelrod Award from the ACNP, the Gold Medal award from the SOBP, the Outstanding Basic Research award from the SIRS.

Abstract

Dysregulation of the mesolimbic dopamine (DA) system has garnered increasing attention in major depressive disorder (MDD). We have shown that rats exposed to either Chronic Mild Stress (CMS) or Learned Helplessness, two stress-induced animal models of depression, resulted in a reduction in ventral tegmental area (VTA) DA neuron population activity, i.e. the number of DA neurons active and available to respond to environmentally salient rewarding stimuli. This suggests that in MDD, the attenuated ability of the DA system to respond to rewarding stimuli could represent the neural substrate of clinical anhedonia. Drawing from human neuroimaging research, we found that overdrive of the infralimbic prefrontal cortex (ILPFC) in normal rats potently suppressed VTA DA neuron population activity, primarily in the medial, reward-related VTA DA neurons, via activation of the amygdala. In rats that underwent CMS, ILPFC inactivation restored VTA DA neuron population activity to normal levels. Furthermore, we found that the rapid acting antidepressant ketamine reversed the decrease in DA neuron activity in learned helplessness model of depression, primarily by restoring plasticity in the hippocampus-accumbens circuit. Thus, a single dose of ketamine restores hippocampal-accumbens drive, normalizes dopamine neuron firing, and reverses behavioral despair in the forced swim test.

Studies of patients at ultra high risk for schizophrenia show that those who do not convert to schizophrenia show increased susceptibility to affective disorders as adults. We now report that MAM models of schizophrenia in which diazepam is used prepubertally to circumvent pathology show increased susceptibility to depression as adults. Moreover, damage to the prelimbic PFC prepubertally will also increase susceptibility to depression. Therefore, hyper-responsivity to stress at early stages in life appear to make the individual more susceptible to depression as adults.

Lunch Symposium: Changing the Course of Schizophrenia: Applying New Knowledge to Clinical Practice
13:00-14:00, 13 May, 2019, Monday

Prof. Robin EMSLEY

Robin Emsley is Professor of Psychiatry at Faculty of Medicine and Health Sciences, University of Stellenbosch, Cape Town, South Africa. He holds the Sarah Turoff Endowed Chair in Schizophrenia Research and Education Committee Chair of Schizophrenia International Research Society. He is also a member of the Section of Schizophrenia and Section of Genetics in World Psychiatric Association.

Professor Emsley obtained his medical degree (MBChB) at the University of Cape Town in 1974 and his psychiatry degree (MMed) at the University of Stellenbosch in 1981. He received a Doctorate in Medicine in 1987 and a Doctor of Science degree in 2007 for studies in the psychopathology, neurobiology and psychopharmacology of schizophrenia.

Professor Emsley's main area of interest is in the neurobiology and psychopharmacology of schizophrenia. His group has published widely in this field, including studies in psychopharmacology, neuroimaging, pharmacogenomics, psychopathology and cognition. He is currently serving on the Editorial Board of several journals, including Schizophrenia Research, Psychiatry Research, Early Intervention in Psychiatry and npj Schizophrenia.

Abstract

Key features of our new understanding of schizophrenia:

- Recognising that the early years of illness are critical to the long-term outcome
- Targeting relapse prevention to avoid emergent refractoriness and illness progression
- Looking beyond just symptom reduction to promoting recovery in terms of:
- Psychopathology
- Functionality
- Quality of life

The course of schizophrenia: Early years are critical. This is when the illness is most aggressive – when relapses are most likely to occur and when illness progression is most likely to occur.

Dopamine hypothesis: The final common pathway. There is a direct relationship between D2 blockade and the expression of psychosis. Therefore, providing continuous, uninterrupted antipsychotic medication is the key

Despite the fact that antipsychotics are effective in relieving symptoms of psychosis in the short-term, the long-term outcome is typically poor.

Relapses are associated with brain volume reductions and may be the critical factor in the evolution of treatment refractoriness and illness progression.

Relapses are common and most often occur after patients discontinue treatment. A major factor here is insight impairment. Illness unawareness and symptom misattribution go hand in hand with failure to recognise the need for treatment. Insight impairment persists, even after favourable treatment response, suggesting that it is a trait rather than a state related phenomenon.

This has major implications for shared decision making in the management of patients with schizophrenia

A pragmatic approach to treating schizophrenia:

1. Recognizing the importance of effective early intervention
2. The burden of responsibility for adherence should not be left with the patient
3. Patient autonomy and independent living is not best addressed by leaving patients to make their own treatment decisions
4. Focusing psychosocial and pharmacological interventions on providing continuous treatment

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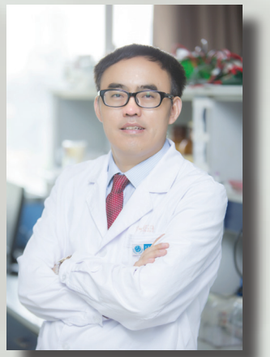
Lecture 3: Procedures Targeting Memory Labile Stages to Erase Drug and Fear memories
14:15-15:00, 13 May, 2019, Monday



Prof LU Lin

Prof. Lin Lu is a member of the Chinese Academy of Sciences. He received his MD/PhD degree at the West China University of Medical Sciences in 1999. He undertook post-doctoral research at the National Laboratory of Medical Neurobiology at the Shanghai Medical School of Fudan University and then at the National Institutes of Health in Maryland, USA, where he continued as a research scientist. He currently works as the director of Peking University Institute of Mental Health/ Peking University Sixth Hospital, National Clinical Research Center for Mental Disorders, National Center for Mental Health of Chinese Center for Disease Control and Prevention, and National Institute on Drug Dependence in China. He has published more than 200 peer-reviewed articles (with total citation over 10000) and his research focuses on the neurobiological mechanisms and clinical interventions of psychiatric disorders, including depression, sleep disorders and drug addiction.

Abstract

Strong emotional stimuli such as traumatic experiences in our daily life result in pathological alterations that usurps the normal neural systems underlying learning and memory. As a result, these maladaptive memories are always stronger and persist longer after learning than memories for neutral stimuli or events. Recent years a proliferation of research interest has been focusing on memory reconsolidation as a potential target for intervening maladaptive memories. Memory reconsolidation requires a series of molecular alterations to re-stabilize the memories and can be disrupted by behavioral interventions (e.g. extinction) or pharmacological interventions (e.g. propranolol). Our previous study showed that conditioned stimulus (CS) retrieval-extinction procedure could effectively decrease conditioned effects and drug seeking in rat models of relapse, as well as drug craving in abstinent heroin addicts. Recently we employed the exposure of an unconditioned stimulus (UCS) to trigger memory reconsolidation, followed by extinction or propranolol, to efficiently disrupt fear- and drug-related memories in both animal models and human studies. In comparison with CS-based reconsolidation interventions, the novel UCS-based reconsolidation interventions could effectively target multi-CS-associated memories and remote memories with stable and long-lasting inhibitory effect, offering us a more powerful non-invasive perspective and showing great translational potential in treating psychiatric disorders including post-traumatic stress disorder, phobia, and addiction.

Lecture 4: Ketamine and ECT – An Added Benefit?
15:00-15:45, 13 May, 2019, Monday

Dr HE Hongbo

Hongbo He, medical bachelor of clinical medicine from Tongji Medical University in Wuhan in 2000, PhD of neuroscience from Louisiana State University Health Science Center in New Orleans USA in 2011, currently is the associate chief psychiatrist, head of department of research and education and head of department of psychosomatic medicine of Guangzhou Hui-Ai Hospital (was known as Guangzhou Psychiatric Hospital), the standing committee member and secretory of psychiatry division of Guangdong Medical Association. Current research focus is to explore new methods of clinical interventions to improve the service outcome for depressive patients.

Abstract

Electroconvulsive therapy (ECT) is a rapid acting and effective treatment for both major depressive disorder (MDD) and bipolar disorder (BP). Both propofol and ketamine are commonly used anesthetic agents but recent clinical studies show that ketamine has rapid-acting antidepressant properties, itself, at sub-anesthetic doses. Meanwhile studies also showed that ketamine as ECT usually require less stimulus intensity to induce full seizure activities in the brain due to its pharmacological property of lack of GABAergic potentiation activity unlike other anesthesia, that was proposed to result in less cognitive impairment evidenced with some case reports. Thus using ketamine as ECT anesthesia was considering a potential more powerful treatment for depression with even less cognitive impairment, combining two effective treatments into one intervention–"one stone, two birds". Guangzhou Hui-Ai Hospital was the first psychiatric hospital in China currently with 1920 inpatients beds. Modified ECT has been applied clinically in our hospital since 1998, currently with more than 30 operations each work day. Around 20% inpatients last year has been received ECT treatments. Such large volume of ECT operations provide us unique opportunity to test the ideas of potential add on benefits of ketamine anesthesia of ECT. In this presentation, we would like to share our RCT results of using ketamine with propofol as ECT anesthesia and the possible neurobiological rationales behind.

Notes to Delegates

Meeting Organizer
Hong Kong Society of Biological Psychiatry

Meeting Secretariat
c/o Kays Asia (Hong Kong) Ltd.
Tel: +852 9658 9650
Fax: +852 3010 8969
E-mail: enquiry@hksbp.org

Meeting Date
12-13 May, 2019, Sunday & Monday

Meeting Venue
Ballroom, 2/F, The Langham Hong Kong, 8 Peking Road, Tsim Sha Tsui, Kowloon, Hong Kong

On-site Registration
The registration counter is located at the entrance of Ballroom. 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 lunch symposia and tea refreshments

Identification Badge
Each participant will receive a badge and a programme book upon check-in. The registration counter is located at entrance of Ballroom. Please wear your identification badge at all times during the event, as it serves as your admission to all scientific sessions, tea refreshments and lunches.

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 12:00–17:00 on 12 May, 2019 and 10:00–15:00 on 13 May, 2019. The 2 lunch symposia are sponsored by Lundbeck and Janssen respectively on12 and 13 May.

Meal Arrangement
Tea break and lunch will be served in the same meeting venue.

Insurance
The organizing committee of the 12th 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.

Photo Taking, Audio Recording and Video Shooting
No photo taking, audio recording and video shooting are allowed in the meeting rooms unless permission is granted.

Smoking Policy
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Acknowledgments

The Organizing Committee would like to extend their heartfelt thanks to the following sponsors for their generous support in making a great success of the 12th ASM.

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- For treatment of adult and adolescent patients age 15 – 17 with schizophrenia[1]
- No clinically significant effects on weight or metabolic parameters were observed[2]

References: 1. LATUDA Hong Kong product insert 2017. 2. Loebel A, et al. Schizophrenia Res. 2013;147:95–102.

Abbreviated prescribing information*
Presentation: Latuda film-coated tablets, containing lurasidone hydrochloride. **Indication:** For treatment of adult and adolescent patients age 15 to 17 years with schizophrenia. **Dosing and Administration:** Adult: Recommended starting dose is 40mg once daily with a meal; initial dose titration not required; maximum dose:160mg/ day; Adolescent: Recommended starting dose is 40mg once daily with a meal; initial dose titration not required; maximum dose: 80mg/ day. **Contraindication:** Hypersensitivity to the active substances or any excipients. Concomitant administration of strong CYP3A4 inhibitors and inducers. **Warning and Precaution:** Clinical improvement may take a few days to some weeks; closely monitor patients during this period. Use with caution in elderly patients with dementia who have risk factors for stroke. Not studied in elderly patients with dementia. Discontinue if patients develops signs or symptoms of neurologic malignant syndrome. **Undesirable effects:** In clinical trials, the following were reports: very common (>10%): akathisia, somnolence; common (≥ 1% to <10%): weight increased, insomnia, agitation, anxiety, restlessness, parkinsonism, dizziness, dystonia, dyskinesia, nausea, vomiting, dyspepsia, salivary hypersecretion, dry mouth, upper abdominal pain, stomach discomfort, musculoskeletal stiffness, blood creatine phosphokinase increase, serum creatinine increase, fatigue.

* This is not a complete list of information contained in Latuda product insert. Please refer to the full Hong Kong product insert before prescribing particularly in relation to dosing and administration, contraindication, warning and precautions, and undesirable effects.

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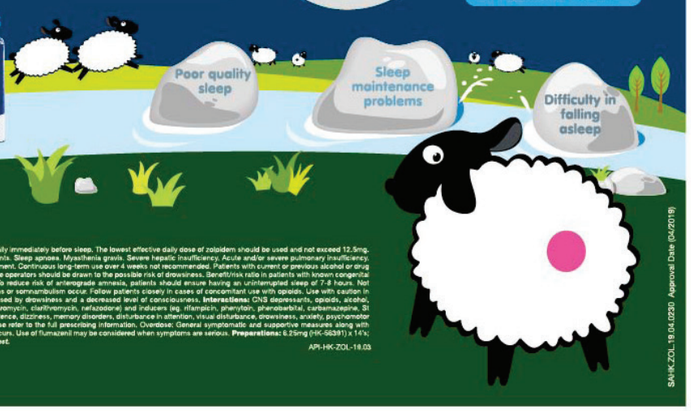

Reference:
1.Moen MD. et. al. Drugs Aging 2006; 23(10): 843-846.

Presentation: Zolpidem tartrate modified-release tablets. **Indications:** short-term treatment of insomnia in adults. **Dosage:** Adults: 12.5mg daily immediately before sleep. The lowest effective daily dose of zolpidem should be used and not exceed 12.5mg. Elderly: 6.25mg daily immediately before sleep. Hepatic impairment: 6.25mg daily. **Contraindications:** Hypersensitivity to zolpidem or excipients. Sleep apnoea. Myasthenia gravis. Severe hepatic insufficiency. Acute and/or severe pulmonary insufficiency. Prior or concomitant intake with alcohol. Children below 18 years of age. **Precautions:** Keep to the recommended dosage and duration of treatment. Continuous long-term use over 4 weeks not recommended. Patients with current or previous alcohol or drug abuse should be monitored carefully. Discontinue treatment once physical dependence develops. The attention of drivers of vehicles and machine operators should be drawn to the possible risk of drowsiness. Benzodiazepine relie in patients with known congenital long QT syndrome should be carefully considered. Patients with respiratory insufficiency or renal impairment should be monitored closely. To reduce risk of anterograde amnesia, patients should ensure having an uninterrupted sleep of 7-8 hours. Not recommended in patients with psychotic illness, including depression and psychosis. Discontinue treatment if psychiatric, paradoxical reactions or somnambulism occur. Follow patients closely in cases of concomitant use with opioids. Use with caution in patients with convulsive disorders. Limit repeat prescription without adequate medical supervision to avoid risk of abuse. Severe injuries caused by drowsiness and a decreased level of consciousness. **Interactions:** CNS depressants, opioids, alcohol, imipramine, chlorpromazine, fluvoxamine, ciprofloxacin. Caution advised for CYP3A4 inhibitors (eg. ketoconazole, itraconazole, ritonavir, telithromycin, clarithromycin, nefazodone) and inducers (eg. rifampicin, phenytoin, phenobarbital, carbamazepine, St John's wort). **Pregnancy & Lactation:** Not recommended. **Undesirable effects:** Nausea, constipation, diarrhoea, influenza, headache, somnolence, dizziness, memory disorders, disturbance in attention, visual disturbance, drowsiness, anxiety, psychomotor retardation, disorientation, fatigue, myalgia, muscle cramp, neck pain, back pain. For uncommon, rare and very rare undesirable effects, please refer to the full prescribing information. Overdose: General symptomatic and supportive measures along with immediate gastric lavage where appropriate. Intravenous fluids administered as needed. Sedative drugs should be withheld, even if excitation occurs. Use of flumazenil may be considered when symptoms are serious. **Preparations:** 6.25mg (HK-56391) x 14's; 12.5mg (HK-56392) x 14's. **Legal Classification:** Part 1, First & Third Schedules *Poison Full prescribing information is available upon request.*

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FDA approved once-monthly* Long Acting Injectable for the maintenance treatment of Bipolar I Disorder in adult[3]

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For the product's safety, contraindications and side effects or toxic hazards, please refer to the package insert.

References: 1. Calabrese JR, et al. *J Clin Psychiatry.* 2017;78(3):324-331.; **2.** Data on file. ABIMAI-178.; **3.** Otsuka Pharmaceutical Co., Ltd. (2017) *Abilify Maintena (aripiprazole) for Extended-Release Injectable Suspension Approved by U.S. FDA for Maintenance Monotherapy Treatment of Bipolar I Disorder* [Press release]. 29 July. Available at: https://www.otsuka.co.jp/en/company/newsreleases/2017/20170729_1.html (Accessed: 13 February 2019). **4.** Abilify Maintena package insert.

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* Dose range for acute treatment

Abbreviated Prescribing Information:
Presentation: Quetiapine fumarate extended-release tablet. **Indications: Bipolar Disorder:** Maintenance treatment of bipolar I disorder, as monotherapy or in combination with lithium or sodium valproate, for prevention of relapse/recurrence of manic, depressive or mixed episodes; Treatment of depressive episodes associated with bipolar disorder; Treatment of acute mania associated with bipolar I disorder as monotherapy or in combination with lithium or sodium valproate. **Schizophrenia:** Treatment of schizophrenia, prevention of relapse and maintenance of clinical improvement during continuation therapy. **Major Depressive Disorder (MDD):** Treatment of recurrent MDD in patients who are intolerant of, or who have an inadequate response to alternative therapies. **Generalised Anxiety Disorder (GAD):** Treatment of GAD. **Dosage:** Once daily, without food. **Bipolar Disorder: Maintenance treatment:** Use same dose as active treatment for prevention of manic, depressive or mixed episodes in bipolar disorder. Range 300-800 mg/day. **Bipolar Depression:** 50 mg (Day 1), 100 mg (Day 2), 200 mg (Day 3), 300 mg (Day 4). Can be titrated to 400 mg on Day 5 and up to 600 mg by Day 8. **Acute Mania:** 300 mg (Day 1), 600 mg (Day 2), up to 800 mg (after Day 2), alone or in combination with a mood stabilizer. Range 400-800 mg/day. **Schizophrenia:** 300 mg (Day 1), 600 mg (Day 2) and up to 800 mg after Day 2. Range 400-800 mg/day depending on response and tolerability. Same dosage for maintenance therapy. **Recurrent MDD:** Once daily in the evening. 50 mg (Day 1 & 2), increased to 150 mg on Day 3 & 4. Usual effective dosage: 150 mg. Range of 50-300 mg/day. Same dosage for maintenance therapy. **GAD:** 50 mg (Day 1 & 2), 150 mg (Day 3 & 4). Range 50-150 mg/day. **Switching from Seroquel immediate release:** Switch at equivalent total daily dose. Individual adjustments may be necessary. **Elderly:** 50 mg/day, increased in increments of 50 mg/day up to target dose depending on response and tolerability. Slower dose titration is recommended. **Elderly MDD:** 50 mg (Day 1-3), 100 mg (Day 4), 150 mg (Day 8), up to 300 mg depending on response and tolerability. **Elderly GAD:** 50 mg (Day 1-3), 100 mg (Day 4), 150 mg on Day 8. **Patients with renal impairment:** No dosage adjustment needed. **Patients with hepatic impairment:** 50 mg/day up to target dose. **Contraindications:** Hypersensitive to any components of this product. **Precautions:** Elderly patients with dementia-related psychosis or behavioural disorders; rare hereditary problems of galactose intolerance, lapp lactase deficiency or glucose-galactose malabsorption; concomitant use with ADHD medication; conditions predisposing to hypotension; family history of QT prolongation, congenital long QT syndrome, congestive heart failure, heart hypertrophy, hypokalaemia or hypomagnesaemia, concomitant medicines known to prolong QTc interval; history of seizures, conditions that potentially lower seizure threshold; elevation in core body temperature; risk for aspiration pneumonia. **Interactions:** Centrally acting drugs: thioridazine; lorazepam; levodopa and dopamine agonists. CYP3A4 inhibitors: azole antifungals; macrolide antibiotics; protease inhibitors; grapefruit juice. Hepatic enzyme inducers: carbamazepine; phenytoin. **Undesirable effects:** Sedation; somnolence; insomnia; dizziness; syncope; headache; increased appetite; weight gain; dysphagia; dry mouth; nausea & vomiting; constipation; dyspepsia; tachycardia; palpitations; orthostatic hypotension; rhinitis; dyspnoea; blurred vision; abnormal dreams & nightmares; asthenia; dysarthria; fatigue; myalgia; peripheral edema; irritability; pyrexia; lipid changes; worsening of metabolic factors; elevations in serum transaminases (ALT, AST), γ-GT & serum prolactin; increases eosinophils; decreases in total T4, free T4 & total T3, and increases in TSH; leucopenia and/or neutropenia; mild asthenia; withdrawal symptoms after abrupt cessation. **Full local prescribing information is available upon request. API.HK.SXR.0813 When treating patients with Seroquel IR/ Seroquel XR, it is advised to monitor metabolic parameters.**

Please contact (852) 2420-7388 or HKPatientSafety@astrazeneca.com for adverse drug reactions (ADR) reporting to AZHK.

Further information is available on request:
AstraZeneca Hong Kong Limited
Unit 1-3, 11/F, 18 King Wah Road,
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Tel: 2420 7388 Fax: 2422 6788

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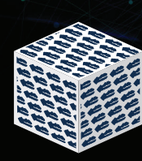

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- Increase employment by 50.6%[5]
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LAI=long-acting injection.

References:
1. Ravenstijn P, Remmerie B, Savitz A, Samtani MN, Nuamah I, Chang CT, et al. Pharmacokinetics, safety, and tolerability of paliperidone palmitate 3-month formulation in patients with schizophrenia: A phase-1, single-dose, randomized, open-label study. J Clin Pharmacol. 2016;56:330-9. 2. Morris MT, Tarpada SP. Long-Acting Injectable Paliperidone Palmitate: A Review of Efficacy and Safety. Psychopharmacol Bull. 2017;47:42-52. 3. Emond B, El khoury AC, Patel C, Pilon D, Morrison L, Zhdanava M, et al. Real-world outcomes post-transition to once-every-3-months paliperidone palmitate in patients with schizophrenia within US commercial plans. Current Medical Research and Opinion. 2019;35:407-16. 4. Berwaerts J, Liu Y, Gopal S, Nuamah I, Xu H, Savitz A, et al. Efficacy and Safety of the 3-Month Formulation of Paliperidone Palmitate vs Placebo for Relapse Prevention of Schizophrenia: A Randomized Clinical TrialPaliperidone Palmitate 3-Month Formulation for Schizophrenia. JAMA Psychiatry. 2015;72:830-9. 5. Zhang H, Turkoz I, Zhuo J, Mathews M, Tan W, Feng Y. Paliperidone Palmitate Improves and Maintains Functioning in Asia-Pacific Patients with Schizophrenia. Adv Ther. 2017;34:2503-17. 6. Gopal S, Xu H, McQuarrie K, Savitz A, Nuamah I, Woodruff K, et al. Caregiver burden in schizophrenia following paliperidone palmitate long acting injectables treatment: pooled analysis of two double-blind randomized phase three studies, NPJ Schizophr. 2017;3:23.

INVEGA SUSTENNA® PROLONGED RELEASE SUSPENSION FOR I.M. INJECTION 50, 75, 100, 150 MG
ABBREVIATED PRESCRIBING INFORMATION
ACTIVE INGREDIENT(S): Paliperidone palmitate. **INDICATION(S):** Schizophrenia in adults. Schizoaffective disorder in adults as monotherapy and as an adjunct to mood stabilizers or antidepressants. **DOSAGE & ADMINISTRATION:** I.M. use only. For patients naïve to oral paliperidone or oral or injectable risperidone, it is recommended to establish tolerability with oral paliperidone or oral risperidone prior to initiating treatment with INVEGA SUSTENNA. The recommended initiation dose is 150 mg on treatment day 1 and 100 mg one week later, both administered in the deltoid muscle. The recommended monthly maintenance dose is 75 mg; some patients may benefit from lower or higher maintenance doses within the recommended range of 25 to 150 mg based on individual patient tolerability and/or efficacy. Following the second dose, monthly maintenance doses can be administered in either the deltoid or gluteal muscle. Adjustment of the maintenance dose may be made monthly. **CONTRAINDICATIONS:** Known hypersensitivity to either paliperidone or risperidone, or to any of the excipients in the INVEGA SUSTENNA formulation. **SPECIAL WARNINGS & PRECAUTIONS: INCREASED MORTALITY IN ELDERLY PATIENTS WITH DEMENTIA-RELATED PSYCHOSIS: INVEGA SUSTENNA IS NOT APPROVED FOR THE TREATMENT OF PATIENTS WITH DEMENTIA-RELATED PSYCHOSIS.** Cerebrovascular Adverse Events, Including Stroke, in Elderly Patients with Dementia-Related Psychosis: Increased incidence of cerebrovascular adverse events (cerebrovascular accidents and transient ischemic attacks, including fatalities); Neuroleptic Malignant Syndrome: It has been reported in association with antipsychotic drugs, including paliperidone. Manage with immediate discontinuation of antipsychotic drug and other drugs not essential to concurrent therapy; intensive symptomatic treatment and medical monitoring; treatment of any concomitant serious medical problems for which specific treatments are available. QT Prolongation: Avoid use with drugs that prolong QTc interval. Also avoided in patients with congenital long QT syndrome and in patients with a history of cardiac arrhythmias. Tardive Dyskinesia: Discontinue drug if clinically appropriate. Metabolic Changes: Atypical antipsychotic drugs have been associated with metabolic changes that may increase cardiovascular/cerebrovascular risk. Hyperprolactinemia: Prolactin elevations occur and persist during chronic administration. Orthostatic Hypotension and Syncope: Use with caution in patients with known cardiovascular or cerebrovascular disease and patients predisposed to hypotension. Monitoring of orthostatic vital signs should be considered in patients who are vulnerable to hypotension. Falls: Somnolence, postural hypotension, motor and sensory instability have been reported with the use of antipsychotics which may lead to falls and consequently fractures or other fall-related injuries. Leukopenia, Neutropenia, and Agranulocytosis: Perform a complete blood count frequently during the first few months in patients with a history of a clinically significant low white blood cell count (WBC) or absolute neutrophil count (ANC) and history of drug-induced leukopenia/neutropenia. Consider discontinuation at the first sign of clinically significant decline in WBC in the absence of other causative factors. Patients with clinically significant neutropenia for fever or other symptoms or signs of infection should be monitored and treated promptly. Discontinue INVEGA SUSTENNA in patients with severe neutropenia. Potential for Cognitive and Motor Impairment: Use caution when performing activities requiring mental alertness. Seizures: Use cautiously in patients with a history of seizures or with conditions that lower the seizure threshold. Dysphagia: Use cautiously in patients at risk for aspiration pneumonia. Priapism: Drugs with alpha-adrenergic blocking effects have been reported to induce priapism. Severe priapism may require surgical intervention. Body Temperature Regulation: Appropriate care to patients experiencing conditions which may contribute to an elevation in core body temperature. **SIDE EFFECTS:** Injection site reactions, somnolence/sedation, dizziness, akathisia, and extrapyramidal disorder. Refer to the full prescribing information for other side effects. **PREGNANCY & LACTATION:** Pregnancy Category C. INVEGA SUSTENNA should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. Because of the potential for INVEGA SUSTENNA Version 4.0 Page 2 of 2 serious adverse reactions in nursing infants, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother. **INTERACTIONS:** Drugs that may cause orthostatic hypotension (e.g. nitrates, antihypertensive medicines). Strong inducers of both CYP3A4 and P-gp (e.g., carbamazepine, rifampin, or St John's wort). Dopamine agonists (Levodopa, bromocriptine, ropinirole and pramipexole). **PLEASE REFER TO FULL PRESCRIBING INFORMATION BEFORE PRESCRIBING.** API version to be quoted on promotional material: Invega Sustenna API ver.4.0

INVEGA TRINZA®
ABBREVIATED PRESCRIBING INFORMATION
ACTIVE INGREDIENT(S): Paliperidone palmitate. **INDICATION(S):** A 3-month injection which is indicated for the treatment of schizophrenia in patients after they have been adequately treated with INVEGA SUSTENNA (1-month paliperidone palmitate extended-release injectable suspension) for at least four months. **DOSAGE & ADMINISTRATION:** I.M. use only by a healthcare professional, administered once every 3 months. Care should be taken to avoid inadvertent injection into a blood vessel. To be used only after INVEGA SUSTENNA (1-month paliperidone palmitate extended-release injectable suspension) has been established as adequate treatment for at least four months. In order to establish a consistent maintenance dose, it is recommended that the last two doses of INVEGA SUSTENNA be the same dosage strength before starting INVEGA TRINZA. Initiate INVEGA TRINZA when the next 1-month paliperidone palmitate dose is scheduled with an INVEGA TRINZA dose based on the previous 1-month injection dose, using the equivalent 3.5-fold higher dose as shown in the full package insert. INVEGA TRINZA may be administered up to 7 days before or after the monthly time point of the next scheduled paliperidone palmitate 1-month dose. **CONTRAINDICATIONS:** Known hypersensitivity to either paliperidone or risperidone, or to any of the excipients in the INVEGA TRINZA formulation. **SPECIAL WARNINGS & PRECAUTIONS: INCREASED MORTALITY IN ELDERLY PATIENTS WITH DEMENTIA-RELATED PSYCHOSIS: INVEGA TRINZA is not approved for the treatment of patients with dementia-related psychosis.** Cerebrovascular Adverse Reactions: Increased incidence of cerebrovascular adverse reactions (e.g., stroke, transient ischemic attacks), including fatalities, were reported in placebo-controlled trials in elderly patients with dementia-related psychosis taking oral risperidone, aripiprazole, and olanzapine. Neuroleptic Malignant Syndrome: NMS has been reported with the use of antipsychotic medications, including paliperidone. Management should include immediate discontinuation of antipsychotic drugs and other drugs not essential to concurrent therapy, intensive symptomatic treatment and close medical monitoring, and treatment of any concomitant serious medical problems. QT prolongation: Avoid use with drugs that prolong QTc interval. Also avoided in patients with congenital long QT syndrome and in patients with a history of cardiac arrhythmias. Tardive Dyskinesia: Discontinue drug if clinically appropriate. Metabolic Changes: Atypical antipsychotic drugs have been associated with metabolic changes that may increase cardiovascular/cerebrovascular risk. Orthostatic Hypotension and Syncope: Use with caution in patients with known cardiovascular or cerebrovascular disease and patients predisposed to hypotension. Monitoring of orthostatic vital signs should be considered in patients who are vulnerable to hypotension. Falls: Somnolence, postural hypotension, motor and sensory instability have been reported with the use of antipsychotics which may lead to falls and consequently fractures or other fall-related injuries. Leukopenia, Neutropenia, and Agranulocytosis: Monitor patients with clinically significant neutropenia for fever or other symptoms or signs of infection and treated promptly if such symptoms or signs occur. Discontinue INVEGA TRINZA in patients with severe neutropenia (absolute neutrophil count <1000/mm³) follow their WBC until recovery. Hyperprolactinemia: Prolactin elevations occur and persist during chronic administration. Potential for Cognitive and Motor Impairment: Use caution when performing activities requiring mental alertness. Seizures: Use cautiously in patients with a history of seizures or with conditions that lower the seizure threshold. Dysphagia: Use cautiously in patients at risk for aspiration pneumonia. Priapism: priapism has been reported with oral paliperidone during postmarketing surveillance. Severe priapism may require surgical intervention. Body Temperature Regulation: Appropriate care to patients experiencing conditions which may contribute to an elevation in core body temperature. Intraoperative Floppy Iris Syndrome (IFIS): Current or past use of medicines with alpha1a-adrenergic antagonist effect should be made known to the ophthalmic surgeon in advance of surgery. **SIDE EFFECTS:** The most common adverse reactions (incidence ≥ 5% and occurring at least twice as often as placebo) were injection site reaction, weight increased, headache, upper respiratory tract infection, akathisia and parkinsonism. Refer to the full prescribing information for other side effects. **PREGNANCY & LACTATION:** Patients should be advised to notify their physician if they become pregnant/intend to become pregnant or intend to nurse during treatment with INVEGA TRINZA. **INTERACTIONS:** Drugs with Potential for Inducing Orthostatic Hypotension. Strong CYP3A4/P-glycoprotein (P-gp) inducers (e.g., carbamazepine, rifampin, or St. John's Wort). Levodopa and Other Dopamine Agonists. **PLEASE REFER TO FULL PRESCRIBING INFORMATION BEFORE PRESCRIBING.** Invega Trinza API ver.3.0

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BRINTELLIX® (VORTIOXETINE) – ABBREVIATED PRESCRIBING INFORMATION
Presentation: Film-coated tablets 5mg, 10mg and 20mg. **Indication:** Treatment of major depressive episodes in adults. **Dosage:** Adults: starting and recommended dose is 10mg, once-daily, taken with or without food. Elderly ≥65 years: Starting dose 5mg. Children and adolescents (<18 years): should not be used. **Discontinuation:** Patients can abruptly stop taking the medicinal product without the need for a gradual reduction in dose. **Contraindications:** Hypersensitivity to vortioxetine or to any of the excipients. Combination with MAO-inhibitors. Should not be used during pregnancy or lactation unless clearly needed and after careful consideration of the risk/benefit. **Special warnings and precautions:** Depression is associated with an increased risk of suicidal thoughts, self-harm and suicide. It is a general clinical experience that the risk of suicide may increase in the early stages of recovery. Close supervision of high-risk patients should accompany drug therapy. Patients (and caregivers) should be alerted about the need to monitor for any clinical worsening, suicidal behaviour or thoughts and unusual changes in behaviour and to seek medical advice immediately if these symptoms present. Should be introduced cautiously in patients who have a history of seizure or in patients with unstable epilepsy. Patients should be monitored for the emergence of signs and symptoms of Serotonin Syndrome or Neuroleptic Malignant Syndrome. Should be used with caution in patients with a history of mania/hypomania and should be discontinued in any patient entering a manic phase. There have been reports of cutaneous bleeding abnormalities with the use of SSRIs/SNRIs. Hyponatraemia has been reported rarely with the use of SSRIs/SNRIs Caution should be exercised for patients with renal or hepatic impairment. **Interactions:** Caution is advised when taken in combination with MAO-inhibitors, serotonergic medicinal products, products lowering the seizure threshold, lithium, tryptophan, St. John's Wort, oral anticoagulants or antiplatelet agents, and products predominantly metabolised by the enzymes CYP2D6, CYP3A4, CYP2C9 and Cytochrome P450. **Undesirable effects:** Adverse reactions are most frequent during the first or second week of treatment and usually decrease in intensity and frequency with continued treatment. Very common: Nausea. Common: abnormal dreams, dizziness, diarrhoea, constipation, vomiting, pruritus, including pruritus generalised. Uncommon: flushing, night sweats. Unknown: Serotonin Syndrome. **Overdose:** Symptomatic treatment. Marketing authorisation holder: Lundbeck HK Limited. Revision Date: May 2017. Full prescribing information is available upon request.

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