The 11th Annual Scientific Meeting
Hong Kong Society of Biological Psychiatry

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

21-22 April 2018 (Saturday and Sunday)
Sheraton Hong Kong Hotel & Towers, 20 Nathan Road, Kowloon

Programme Book

www.hksbp.org

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Hong Kong Society of Biological Psychiatry

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Welcome Message

On behalf of the Organizing Committee, we take great pleasure in inviting you to participate in the 11th Annual Scientific Meeting (ASM) of the Hong Kong Society of Biological Psychiatry (HKSBP). The meeting will be organized on 21st (Saturday afternoon & evening) and 22nd (Sunday afternoon) in April 2018 at the Sheraton Hong Kong Hotel & Towers.

The theme of this year is Neural Circuitry: From Brain Development to Intervention—How Far Are We? We have invited 2 world-class experts to talk about neurotransmitters and neurocircuitry. Prof David J. NUTT is a British psychiatrist and neuropsychopharmacologist specializing in the research of drugs that affect the brain and conditions such as addiction, anxiety and sleep. Currently, he is the Edmond J Safra Chair in Neuropsychopharmacology at Imperial College London. While Prof Anthony A. GRACE is a Distinguished Professor of Neuroscience and a professor of psychiatry and psychology at the University of Pittsburgh, USA. His current work involves the role of dopamine in anhedonia and affective disorders, the mode of action of ketamine and novel antidepressant drugs, and novel treatments for schizophrenia and its prevention. Their lectures will cover new findings in the neurotransmitters and neurocircuitry in depression, schizophrenia, addiction and OCD in our meeting.

In addition, we have invited local speakers from the University of Hong Kong. Prof CHANG Chuen Chung, Raymond will talk about neurobiology of the dementing brain; while Prof LEE Mei Chun, Tatia is specialized in neuropsychology, she will talk about impulsive brain. Prof WING Yun Kwok from Chinese University of Hong Kong will give a lecture on neurobiology of sleep and young researchers from Japan to share their latest research papers. Our President, Dr WONG Ming Cheuk, Michael, will kick-off with a session on drug treatment and Prof. TANG Siu Wa and Dr. Sofia PAPPA will give lectures on the 2 lunch symposia respectively.

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

Yours sincerely,

Dr. CHUNG Kar Kin, Albert
Co-chairperson, Organizing Committee of 11th ASM
Hong Kong Society of Biological Psychiatry

Dr. CHEUNG Hon Kee, Henry
Co-chairperson, Organizing Committee of 11th ASM
Hong Kong Society of Biological Psychiatry

11th AGM Organizing Committee

Co-chairpersons:
Dr. CHUNG Kar Kin, Albert
Dr. CHEUNG Hon Kee, Henry

Scientific Committee Members:

Dr. LO Chun Wai
Professor TANG Siu Wa
Professor WING Yun Kwok
Dr. WONG Ming Cheuk, Michael

Members:
Dr. IU Pui Chuen
Dr. TAM Mo Shing, Paul
Dr. TSANG Suk Kwan, Jenny
Dr. WONG Chi Keung
Dr. WONG Chung Hin, Willy
Dr. YUEN Cheung Hang, Henry

HKSBP: Council Member 2017-2018

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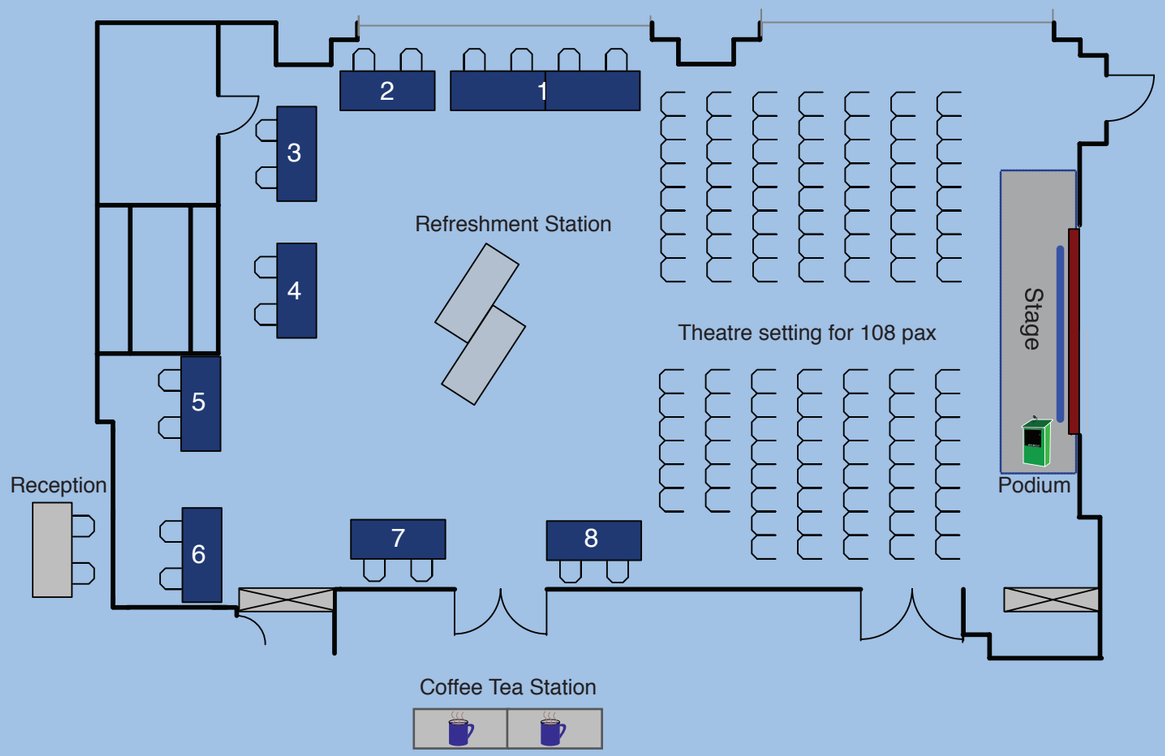
	Dr. TAM Mo Shing, Paul
	Professor WING Yun Kwok
	Dr. WONG Chi Keung
	Dr. WONG Chung Hin, Willy
	Dr. YUEN Cheung Hang, Henry

Floor Plan

Sung Room, 4/F, Sheraton Hong Kong Hotel & Towers, 20 Nathan Road, Kowloon
(The 2 lunch symposia are at Ming Room, 4/F)



1 janssen PHARMACEUTICAL COMPANIES OF Johnson & Johnson
2 Lundbeck
3 DKSH
4 gsk do more feel better live longer
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?

Time	Scientific Programme	Venue
	21 April 2018, Saturday	
11:30-12:00	Registration	Ming Room, 4/F
12:00	Lunch Starts	
12:45-13:45	**Lunch Symposium: Psychotropic Drugs: From Natural Compounds to Modern Drug Development** **Prof. TANG Siu Wa** Director, Institute of Brain Medicine (International) Emeritus Professor of Psychiatry, University of California, Irvine, USA Current Past and Founding President of HKSBP **Chairperson: Dr. LO Chun Wai, Council Member of HKSBP** *(Sponsored by Lundbeck Hong Kong)*	
14:00-14:40	**Welcome Remarks: Do We Know Our Treatment?** **Dr. WONG Ming Cheuk, Michael** President of HKSBP Consultant Psychiatrist, Queen Mary Hospital Honorary Associate Professor, Department of Psychiatry The University of Hong Kong **Chairperson: Dr. WONG Chi Keung, Council Member of HKSBP**	Sung Room, 4/F
14:40-15:40	**Plenary Lecture: Stress and Schizophrenia: Susceptibility in the Developing Brain** **Prof. Anthony A. GRACE** Distinguished Professor of Neuroscience Professor of Psychiatry and Psychology University of Pittsburgh, United Sates of America **Chairperson: Dr. WONG Chung Kwong, JP, Private Practice**	
15:40-16:00	Exhibition and Coffee Break	
16:00-16:45	**Neurobiology of the Dementing Brain** **Dr. CHANG Chuen Chung, Raymond** Lab Chief, Laboratory of Neurodegenerative Diseases, School of Biomedical Sciences LKS Faculty of Medicine, The University of Hong Kong **Chairperson: Dr. LI Seung Yau Derek, Private Practice**	
16:45-17:30	**Regulation of Human Behaviors** **Prof. LEE Mei Chun, Tatia** Chair Professor of Psychology Endowed Professor of Neuropsychology The University of Hong Kong **Chairperson: Dr. TSANG Suk Kwan, Jenny, Hon. Treasurer of HKSBP**	
17:30-18:30	**Free Papers from JSBP** 1. Fecal Microbiota Transplantation may has Positive Effects on Depressive Mood and Anxiety: A Pilot Open-Label Observational Study in Patients with Functional Gastrointestinal Disorders. **Dr. Shunya KUROKAWA**, Department of Neuropsychiatry, Keio University School of Medicine, Japan 2. Widespread White Matter Microstructural Abnormalities Related to Cognitive Impairment in Schizophrenia, Bipolar Disorder, and Major Depressive Disorder: A Tract-based Spatial Statistics Study **Dr. Shinichi YAMADA**, Department of Neuropsychiatry, Wakayama Medical University, Japan **Chairperson: Dr. TAM Mo Shing, Paul, Council Member of HKSBP**	
18:30-19:00	HKSBP's AGM (Members only)	Boardroom,2/F
	Cocktail Reception	Corridor, 4/F
19:00-20:00	**Plenary Lecture: Neural Circuits and Neurotransmitters in the Impulsive Brain** **Prof David J. NUTT** Edmund J. Safra Professor, Neuropsychopharmacology Head of Neuropsychopharmacology Unit and Molecular Imaging Imperial College London, United Kingdom **Chairperson: Prof. TANG Siu Wa, Director, Institute of Brain Medicine (International)**	Sung Room, 4/F
20:00-22:00	Conference Dinner	
	22 April 2018, Sunday	
11:30-12:00	Registration	Ming Room, 4/F
12:00	Lunch Starts	
12:45-13:45	**Lunch Symposium: Does Half-Life Matter After Antipsychotic Discontinuation?** **Dr. Sofia PAPPA** Consultant Psychiatrist, West London Mental Health Trust Honorary Senior Lecturer, Imperial College London, United Kingdom **Chairperson: Dr. WONG Ming Cheuk, Michael, President of HKSBP** *(Sponsored by Janssen Hong Kong)*	
14:00-15:00	**Plenary Lecture: The Circuitry of Depression: New Findings on Postpartum Depression** **Prof. Anthony A. GRACE** Distinguished Professor of Neuroscience Professor of Psychiatry and Psychology University of Pittsburgh, United Sates of America **Chairperson: FUNG Shun Sun, Desmond, Private Practice**	Sung Room, 4/F
15:00-15:15	Exhibition and Coffee Break	
15:15-16:15	**Plenary Lecture: Addiction: From Brain Mechanism to New Treatment** **Prof David J. NUTT** Edmund J. Safra Professor, Neuropsychopharmacology Head of Neuropsychopharmacology Unit and Molecular Imaging Imperial College London, United Kingdom **Chairperson: Dr. CHUNG Kar King, Albert, Co-chairperson of 11th ASM**	
16:15-17:00	**Neurobiology of Sleep** **Prof WING Yun Kwok** Chairman and Professor, Department of Psychiatry Associate Dean (Student Affairs), Faculty of Medicine, The Chinese University of Hong Kong Director of the Sleep Assessment Unit, Shatin Hospital **Chairperson: Dr. CHEUNG Hon Kee, Henry, Co-chairperson of 11th ASM**	
17:00	Closing Remarks by Dr. WONG Ming Cheuk, Michael, President of HKSBP	

* The organizing committee reserves the right to make changes to the programme without notice as and when deemed necessary.

Lunch Symposium: Psychotropic Drugs: From Natural Compounds to Modern Drug
12:45-13:45, 21 April 2018, Saturday

Prof. TANG Siu Wa

Director, Institute of Brain Medicine (International)
Emeritus Professor of Psychiatry, University of California, Irvine, USA
Current Past and Founding President of HKSBP

Professor Siu Wa Tang trained in Psychiatry,Neurochemistry and Biochemical Pharmacology at the University of Toronto, Canada. He was Head of Psychopharmacology at the Clarke Institute of Psychiatry, University of Toronto and former Chairman of the Department of Psychiatry, University of California, Irvine, USA. He founded the Pacific Rim Association of Pharmacogenetics and the Hong Kong Society of Biological Psychiatry. He co-founded the Institute of Brain Medicine to promote psychopharmacology in the Far East with Professor Brian Leonard and Professor Joseph Zohar in 2010 during the 2010 CINP meeting in Hong Kong.

Abstract

Natural compounds as medicine have served human for thousands of years and are widely popular as traditional medicine. As biological molecules co-evolved in humans and plants, the ability of natural compounds to work as medicine can be explained. In fact, modern psychotropic drugs have their origins in botanical molecules. This lecture examines a range of natural compounds from which modern psychotropics evolved, along with some of their unique multi-dimensional multi-target psychopharmacological aspects.

Welcome Remarks: Do We Know Our Treatment?
14:00-14:40, 21 April 2018, Saturday

Dr. WONG Ming Cheuk Michael

MB,BS(HK), MRCPsych(UK), FHKAM, FHKCPsych
Department of Psychiatry, Queen Mary Hospital, Hong Kong

Dr. Wong is working as a Consultant Psychiatrist in the Department of Psychiatry of Queen Mary Hospital in Hong Kong. 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 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 former Chairman of the Society for Advancement of Bipolar Affective Disorder, the current President of the Hong Kong Society of Biological Psychiatry, Chairman of the Hong Kong Association of Psychosocial Rehabilitation and one of the council members of the Asia Network of Bipolar Disorder. 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.

Abstract

As a clinician, we prescribe drugs almost every day. Quite often, we act like a reflex, prescribing a certain medicine for a certain condition. We may not give a thought to basic things about the illness and the drug. For example, we may not think of the pharmacokinetic of the drug, the advantage and disadvantage of a certain preparation of the drug, the mechanism of action of the drug and underlying neurobiology of the illness when we prescribe. These things are important as it guide us to find to best treatment for our patients. Besides, as we know more about the development, structure and organization of neuron network in the brain, we begin to understand more about the underlying neurobiology of various psychiatric conditions. We realize that the “effective” drugs which we are commonly using may not be treating the underlying cause of the illness. This presentation attempts to use a few everyday examples to illustrate the case and paves the way to the subsequent sessions on the neurobiology and neurocircuitry by various speakers.

Plenary Lecture: Stress and Schizophrenia: Susceptibility in the Developing Brain
14:40-15:40, 21 April 2018, Saturday

Prof. Anthony A. GRACE

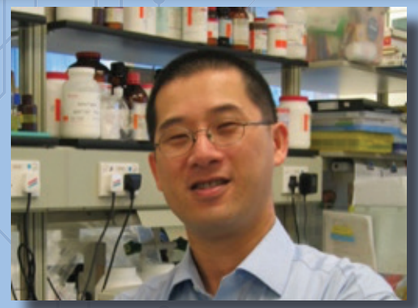
Distinguished Professor of Neuroscience
Professor of Psychiatry and Psychology
University of Pittsburgh, United Sates of America

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 as it relates to the pathophysiology of psychiatric disorders for over 40 years. His early work pioneered the mode of action of antipsychotic drugs and the identification and characterization of dopamine-containing neurons. Currently, Dr. Grace's work involves the role of stress in pathophysiology, 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 35,000 times (H index 96). 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, and the Outstanding Basic Research award from the SIRS. Dr. Grace is one of a handful of individuals that not only performs important basic research, but can to integrate this work into testable models relevant to the human condition.

Abstract

Substantial evidence demonstrates that schizophrenia involves a dysregulated dopamine system, potentially driven by overactivity in the hippocampus. Postmortem studies of schizophrenia brains show a substantial loss of parvalbumin GABAergic interneurons in the hippocampus; loss of this neuron likely drives hippocampal hyperactivity and dysrhythmic activity, leading to an over-responsive dopamine system. Our studies suggest that when the hippocampus is hyperactive and dysrhythmic, the dopamine system is hyper-responsive to stimuli, which can underlie the resultant hallucinations and delusions. 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 in the rat, we found that prepubertally these rats are more anxious, are hyper-responsive to stress, and show hyperactivity in the amygdala; furthermore relieving the stress early in life prevents the transition to "psychosis" in these rats. This suggests that 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 can lead to the schizophrenia phenotype. Moreover, elimination of the ability of the medial prefrontal cortex to regulate stress causes minor stressors to yield the schizophrenia phenotype. In contrast, multiple stressors given to adult rats result in a phenotype resembling models of depression. 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 leads to the intriguing possibility that genetic predisposition does not cause schizophrenia, but instead like the developmental disruption model 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.

Neurobiology of the Dementing Brain:
16:00-16:45, 21 April 2018, Saturday



Dr. CHANG Chuen Chung, Raymond

Lab Chief, Laboratory of Neurodegenerative Diseases, School of Biomedical Sciences, LKS Faculty of Medicine, The University of Hong Kong, Pokfulam, Hong Kong
Chief Editor, American Journal of Alzheimer's Disease and Other Dementias

Dr. Chang is the Lab Chief for the Laboratory of Neurodegenerative Diseases in the School of Biomedical Sciences, LKS Faculty of Medicine, member in The State Key Laboratory of Brain and Cognitive Sciences, The University of Hong Kong. Dr. Chang is the organizer and Secretary of HKU Alzheimer's Disease Research Network. He organizes International Alzheimer's Disease Conference every year since 2000. This conference is now co-organized by 8 universities and Hong Kong Science Park.
Dr. Chang's research interest is on four directions. (1) Pathophysiological changes of Alzheimer's disease (AD), (2) how different risk factors (post-operative cognitive dysfunctions, periodontitis, depression, cigarette smoking, air pollutants) stimulate systemic inflammation to affect neuroimmune responses leading to AD, (3) spreading of neurodegeneration in Parkinson's disease dementia, and (4) neurodegeneration of the retina and deterioration of visual functions in Alzheimer's disease. He has published over 132 peer-reviewed papers, 14 book chapters and edited 3 books in these areas. His h-index is 39 by Scopus.

Dr. Chang is the Chief Editor for "American Journal of Alzheimer's Disease and Other Dementias", Senior Editor for "Journal of Neuroimmune Pharmacology", Associate handling Editor for "Journal of Alzheimer's Disease", "Frontiers in Neurology", "Frontiers in Neurosciences" and "Frontiers in Psychiatry". He is in the Scientific Advisory Board of International AD/PD Symposium, and Scientific Review Committee in Alzheimer Association. He is the member of editorial board of more than 20 different journals, and grant reviewer for different grant agencies/Foundations.

Abstract

Raymond Chuen-Chung CHANG[1,2], PhD
[1]Laboratory of Neurodegenerative Diseases, School of Biomedical Sciences, LKS Faculty of Medicine, The University of Hong Kong[2] State Key Laboratory of Brain and Cognitive Sciences, The University of Hong Kong

One of the increasing challenges for psychiatrist is mental health associated with dementia in patients. This is because anatomical regions for cognitive functions are close to those regions with problems of mental health, including depression, anxiety, apathy, aggression/agitation, and psychosis. Therefore, it is important to revisit definition and current findings about dementia, and even our memory/cognitive functions.

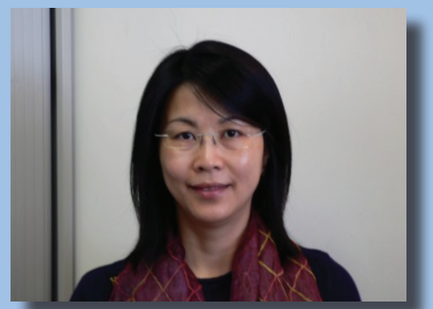
From neuroanatomical and neuropsychological points of view, we can make simple division of our cognitive functions into spatial/learning memory regulated by hippocampus/cingulate gyrus and executive functions regulated by pre-frontal cortex. In addition, we have striatum to help us for habitual and skill learning, cerebellum for sport/activity learning, nucleus accumbens for incentive rewarding motivation for learning, and amygdala for fear-associated learning. Degeneration of neurons in any part will affect our memory and cognitive functions.

For clinicians, you usually observe that patients with dementia is mixed dementia type. However, from scientific research, neuropathological changes start from even 30 years long before clinical symptoms. This is an important first take-home message. This is why we have to investigate different types of dementia. Since stroke and small blood vessels disease (SVD) receive much attention in Hong Kong, we are quite aware of vascular dementia. However, increasing lines of evidence have shown that many risk factors promote Alzheimer's type dementia (Alzheimer's disease, AD), which has definition of dementia with β-amyloid peptide and tau protein phosphorylation as initial pathogenesis. We have also Lewy body dementia (LBD), a second largest group of dementia which comprises of dementia with Lewy's body (DLB) and Parkinson's disease dementia (PDD). We also have frontal temporal lobe dementia (FTLD or FTD), multiple system atrophy (MSA), progressive supranuclear palsy (PSP), and corticobasal degeneration (CBD). With the increasing awareness of cognitive functions, research on cognitive functions has focused on cognitive functions in other neurodegenerative diseases and mental disorders such as multiple sclerosis (MS), amyotrophic lateral sclerosis (ALS), post-operative cognitive dysfunctions, and schizophrenia.

While we have different names of the neurodegenerative diseases depicting the etiology of the disease, the second take-home message is that we have increasing lines of evidence to demonstrate mixed pathology in the late state of AD, PD, LBD, and other neurodegenerative diseases. How does the pathology spread from one type to mixed types and how does the pathology spread from one region to other regions of the brain? All these questions will be the biggest challenges in scientific research. For clinician, detecting cognitive dysfunctions from one domain to another domain will give us more clinical data for neurodegeneration. This is the third take-home message for you.

Acknowledgement: The laboratory is partly supported by Innovative and Technology Fund (ITS/381/15) and Health and Medical Research Fund (02131496).

Regulation of Human Behaviors:
16:45-17:30, 21 April 2018, Saturday



Prof. LEE Mei Chun, Tatia

Chair Professor of Psychology
Endowed Professor of Neuropsychology
The University of Hong Kong

Professor Lee is the Chair Professor of Psychology and Endowed Professor in Neuropsychology at The University of Hong Kong. Professor Lee is a co-director of the State Key Laboratory of Brain and Cognitive Sciences of the University. During her tenure at the University, she has received numerous awards in recognition of her excellence in teaching and research.

Professor Lee endeavours to understand how the human brain functions. Her team employs both behavioral and neuroimaging research methodologies and work in collaboration with clinicians and neuroscientists to investigate the neural mechanisms underpinning those social cognitive and affective processes that define the human nature of an individual. She has published extensively, over 200 publications, and in high impact journals, e.g. Molecular Psychiatry and Cerebral Cortex.

Abstract

Human brain is known for its ability to regulate behaviors in accordance with the individual's goal and the situational demand. This ability enables us to select the most advantageous choice of behavior that enhances our survival. Previous findings of behavioral and functional imaging studies have indicated that efficient and effective regulation of behaviors depends on the coordinated effort of brain regions that are closely interacting with each other. Yet the quality of their coordinated output can be significantly modulated by emotions and neuropathologies. For example, individuals who are impulsive tend to present an attenuation of activity in the anterior cingulate region and may show an increased neural activity in the right inferior parietal region. In this presentation, behavioral and neuroimaging findings on the regulation of human behaviors will be reviewed, as well as the neural cognitive mechanisms underpinning decision making in people under the influence of depressed moods.

Free Papers from JSBP:
17:30-18:00, 21 April 2018, Saturday

Dr. Shunya KUROKAWA

Department of Neuropsychiatry,
Keio University School of Medicine, Japan

Shunya Kurokawa, M.D., is a psychiatrist in Tokyo. He graduated Yamagata University School of Medicine, Japan in 2012. After finishing his 2 year junior residency at Yokohama City University Hospital, he joined the Neuropsychiatry department of Keio University School of Medicine from 2014.

He is currently working as a psychiatrist at Keio University Hospital and a child psychiatrist at Shimada Rehabilitation Center for Disabled Children. His main clinical interests are on Neurodevelopmental disorders, psychosomatic diseases, and mood disorders.

He started his PhD course in 2017 with Dr. Kishimoto's "Integrated Innovation Lab for Psychiatry". His main research interests are on the microbiota-gut-brain axis in psychiatric disorders such as depression, anxiety and neurodevelopmental disorders. He has assessed the effect of Fecal Microbiota Transplant (FMT) to depression and anxiety in patients with IBS, Functional Diarrhea and Functional Constipation, and found that mood may be improved regardless of the gastrointestinal symptom change, and the increase of microbial diversity is related to the effect of FMT. This work is currently in preparation for publish. Also, he has the research license of ADOS-2 (Autism Diagnostic Observation Schedule - 2) and ADI-R (Autism Diagnostic Interview - Revised), planning to do future researches in Autism and ADHD.

Fecal Microbiota Transplantation may have positive effects on Depressive Mood and Anxiety : A Pilot Open-Label Observational Study in Patients with Functional Gastrointestinal Disorders.

Backgrounds: The "Microbiome-Gut-Brain Axis" is considered as a potential common underpinning pathophysiology of functional gastrointestinal disorders (FGIDs) and psychiatric disorders such as depression and anxiety. Fecal Microbiota Transplantation (FMT) has been reported to have therapeutic effects on diseases related to dysbiosis, but few studies have evaluated its effect on psychiatric symptoms such as depression and anxiety.

Methods: We followed 17 patients with either irritable bowel syndrome (IBS), Functional Diarrhea (FDr) or Functional Constipation (FC) who underwent FMT for the treatment of abdominal symptoms and observation of psychiatric symptoms. Changes in Hamilton Rating Scale for Depression (HAM-D) and subscale of sleep-related items, Hamilton Rating Scale for Anxiety (HAM-A) and Quick Inventory for Depressive Symptoms (QIDS) between baseline and 4 weeks after FMT, and intestinal microbiota using the 16SrRNA metagenome sequencing method were measured.

Results: We observed significant improvement in HAM-D total and sleep subscale score, as well as in HAM-A and QIDS scores ($p=0.007$, $p=0.007$, $p=0.01$, $p=0.007$, respectively). A subgroup of 8 patients who did not respond to FMT for the gastrointestinal symptoms also showed significant improvement in HAM-A total score ($p=0.024$) and trend-level improvement for the HAM-D total, sleep subscale score, and QIDS score ($p=0.062$, $p=0.062$, $p=0.066$, respectively). Baseline Shannon index indicated that microbiota showed lower diversity in patients with HAM-D$\geq$8 compared to those of healthy donors as well as patients with HAM-D<8.

There was a significant negative correlation between baseline Shannon index and HAM-D score, and a correlation between Shannon index change and HAM-D improvement after FMT.
Conclusions: Our results suggest that depression and anxiety symptoms may be improved by FMT regardless of gastrointestinal symptom change in patients with FGIDs, and that the increase of microbiota diversity may help improve patient's mood. Further study with larger sample size with a control group is needed in the future.

Free Papers from JSBP:
18:00-18:30, 21 April 2018, Saturday

Dr. Shinichi YAMADA

Department of Neuropsychiatry,
Wakayama Medical University, Japan

Shinichi Yamada is working as an assistant professor at Department of Neuropsychiatry, Wakayama Medical University, Japan. He received his Ph.D. from Wakayama Medical University in 2015. Main research theme of his postgraduate school was relationship between cognitive function and white matter microstructural abnormalities assessed by DTI in mood disorders (Yamada et al. Journal of Affective Disorders 2015). Recently, he involves in neuroimaging research of cognitive function in schizophrenia.

Widespread White Matter Microstructural Abnormalities Related to Cognitive Impairment in Schizophrenia, Bipolar Disorder, and Major Depressive Disorder: A Tract-based Spatial Statistics Study

Shinichi Yamada [a], Shun Takahashi[a], Takuya Ishida [a], Yuji Ohoshi [a], Tomikimi Tsuji [a], Masaki Terada [b], Satoshi Ukai [a]. Department of Neuropsychiatry, Wakayama Medical University, Wakayama, Japan[b]. Wakayama-Minami Radiology Clinic, Wakayama, Japan

Background: White matter (WM) microstructural abnormalities have been observed in patients with schizophrenia (SZ), bipolar disorder (BD), and major depressive disorder (MDD). Extensive evidence suggests that cognitive impairment in SZ, BD, and MDD, but much of its pathophysiology is unknown. The aim of this study was to examine the association between whole-brain WM microstructural abnormalities and their relationship with cognitive function in patients with SZ, BD, and MDD using tract-based spatial statistics (TBSS).

Methods: The subjects were 19 patients with SZ, 20 patients with BD, 18 patients with MDD, and 19 healthy controls. Using TBSS, we examined differences in fractional anisotropy (FA) of whole-brain WM among the 4 groups, and examined the correlations between FA and cognitive performance in the 4 groups.

Results: The FA of WM in widespread regions in the SZ, BD, and MDD groups was significantly reduced compared with those in the HC group. The mean FA of the WM skeleton with statistical differences correlated with performance on the Tower of London in the SZ group, correlated with performance on the list learning in the BD group, and correlated with performance on the digit sequencing task, token motor, and symbol coding in the MDD group.

Conclusions: Our results suggested widespread WM microstructural abnormalities, which were associated with cognitive impairment in patients with SZ, BD, and MDD. This study contributes to the understanding of WM pathophysiology in SZ, BD, and MDD.

Plenary Lecture: Neurotransmitters and Circuits in the Impulsive Brain
19:00-20:00, 21 April 2018, Saturday

Prof David J. NUTT

Edmund J. Safra Professor, Neuropsychopharmacology
Head of Neuropsychopharmacology Unit and
Molecular Imaging Imperial College London, United Kingdom

David Nutt is currently the Edmund J Safra Professor of Neuropsychopharmacology and Head of the Neuropsychopharmacology Unit in the Centre for Academic Psychiatry in the Division of Brain Sciences, Dept of Medicine, Hammersmith Hospital, Imperial College London. He is also visiting professor at the Open University in the UK and Maastricht University in the Netherlands.

He currently is the founder Chair of DrugScience.org.uk (formerly the Independent Scientific Committee on Drugs - ISCD) and has held many leadership positions in both the UK and European academic scientific and clinical organisations. These include presidencies of the European Brain Council, the British Neuroscience Association, the British Association of Psychopharmacology and the European College of Neuropsychopharmacology as well as Chair of the UK Advisory Council on the Misuse of Drugs. He is a Fellow of the Royal Colleges of Physicians, of Psychiatrists and of the Academy of Medical Sciences. He is also the UK Director of the European Certificate and Masters in Affective Disorders courses and a member of the International Centre for Science in Drug Policy.

In 2010 The Times Eureka science magazine voted him one of the 100 most important figures in British Science, and the only psychiatrist in the list. In 2013 he was awarded the Nature/Sense about Science John Maddox prize for Standing up for Science and in 2016 an Honorary Doctor of Laws from the University of Bath for contributions to science and policy.

Abstract

Impulsivity is a major problem in many psychiatric patients and especially in ADHD and addiction disorders. The talk will explore the role of pre-frontal cortex and sub-cortical processes in regulating impulsivity and explain how drugs such as stimulants and atomoxetine work to restrain this behaviour by redressing a relative dopamine and or noradrenaline deficiency in pre-frontal cortex. Thais relative deficiency means that the pre-frontal cortex then fails to adequately control sub-cortical activity especially in the basal ganglia so allowing impulsivity and hyperactivity to emerge. The new PET data supports the view that low dopamine receptor number in the striatum of pathological gamblers leads to their loss of control and impulsivity particularly in states of urgency.

The new PET and MRI imaging data also explain why there might be a dysregulation of dopamine systems in addiction because of alterations of endogenous GABA-A receptor function and of the endorphin systems in people addicted to either alcohol heroin tobacco or gambling

Heal DJ, Smith S, Gosden J, Nutt DJ (2013) Amfetamines, Past and Present – A Pharmacological and Clinical Perspective Journal of Psychopharmacology , 1-18

Clark L, Stokes PR, Wu K, Michalczuk R, Benecke A, Watson BJ, Egerton A, Piccini P, Nutt DJ, Bowden-Jones H, Lingford-Hughes AR (2012) Striatal dopamine D2/D3 receptor binding in pathological gambling is correlated with mood-related impulsivity NeuroImage 63: 40–46

Stokes PRA, Benecke A, Myers J, Watson B, Kalk N, Riano Barros D, Nutt DJ, Lingford-Hughes AR (2012) History of cigarette smoking is associated with increased limbic GABAA receptor availability Neuroimage DOI:10.1016/j.neuroimage.
2012.12.01

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Lunch Symposium: Does Half-Life Matter After Antipsychotic Discontinuation?
12:45-13:45, 22 April 2018, Sunday

Dr. Sofia PAPPA MD, PhD

Consultant Psychiatrist,
West London Mental Health Trust
Honorary Senior Lecturer, Imperial College London,
United Kingdom

Dr Sofia Pappa is a Consultant Psychiatrist at West London Mental Health Trust and an Honorary Senior Lecturer at Imperial College Medical School. Furthermore, she is the Clinical Research Lead and the Recovery Medical Lead for her Trust as well as the NIHR Specialty Lead in Mental Health for the North West London Clinical Research Network.

She has dual training in Neurology and Psychiatry and was awarded a European grant for the completion of her PhD. Subsequently, she obtained a scholarship for conducting postdoctoral research at the Institute of Psychiatry focusing on the neurological dysfunction in psychosis. Other interests include movement disorders and psychopharmacology.

Furthermore, she has significant experience in clinical management, having acted as the Clinical Lead for Adult Services in the past as well as extensive experience in teaching including in her roles as Imperial Medical School Tutor, Recovery College Tutor and main organiser of the annual educational meeting 'Psychiatry Masterclass' with the Institute of Psychiatry.

Abstract

Long-acting antipsychotic medication may significantly reduce relapse rates and enhance treatment continuation in patients with schizophrenia (Leucht 2012, 2017). Evidence has also shown that second generation antipsychotic medications are more acceptable to patients (Fleischacker 2009; Marinis 2007).

In my talk, I will present the findings of a large, independent evaluation on the use of second generation long acting antipsychotics (LAI) in my service between 2011-2017, reporting on retention & discontinuation rates as well as compliance rates, community initiation figures and hospital admission rates two years before and after the initiation of LAI. The results showed that introduction of LAI had a significant impact on the long term clinical outcomes in terms of reduced hospitalizations and high continuation and compliance rates in this naturalistic cohort.

Furthermore, I will advocate for the potential benefits of the earlier use of LAIs in the community; both an independent long-term comparative trial by Subotnik and a systematic review by Taylor have highlighted the value of earlier use of LAI preparations in psychosis. Of equal importance, a recent study demonstrated that functioning, including employment, was improved after short-term, once-monthly LAI, and was sustained to 18 months in Asia–Pacific patients with schizophrenia (Zhang, 2017)

Finally, I will provide an overview of the available evidence on the use of latest antipsychotic with an emphasis on the prophylactic value of long lasting formulation in relapse prevention after antipsychotic discontinuation whilst also discussing my own experience with this newly approved ultra-long-acting antipsychotic.

Plenary Lecture: The Circuitry of Depression: New Findings on Postpartum Depression
14:00-15:00, 22 April 2018, Sunday

Prof. Anthony A. GRACE

Distinguished Professor of Neuroscience
Professor of Psychiatry and Psychology
University of Pittsburgh, United Sates of America

(for bio details, please refer to p.8)

Abstract

The period after childbirth (i.e. postpartum period) is a time of elevated risk for the development of affective disorders. Indeed, the highest rates of anxiety and depression occur during the first few weeks, months or year postpartum compared with other times in a woman's life. In accordance, animal models of postpartum depression have also reported time-dependent effects on depressive-like behavior and anhedonia. In rodents, parity (i.e. the condition of having borne offspring) induces changes in DA-mediated behavioral responses, which may reflect some influence on DA neurotransmission. However, the neurobiological underpinnings of this increased female susceptibility to depression during the postpartum period remain poorly understood.

The dopamine (DA) system has traditionally been associated with anhedonia, the inability to derive pleasure from normally rewarding stimuli, and has been implicated repeatedly in the pathophysiology of depression. A causal link between a hypofunctioning DA system (i.e. decreased DA neuron activity) and stress-induced depression-related behaviors (i.e. anhedonia, despair, anxiety) has been demonstrated in animal models, with females showing greater responses. Surprisingly, little is known about DA system function in females following reproductive experience, including parity.

Compared to virgin females, early postpartum females exhibited higher levels of anxiety and reduced social motivation. Moreover, similar to rats exposed to three other models of depression (i.e., learned helplessness, amphetamine withdrawal, and chronic mild stress), postpartum females exhibited an attenuation of DA neuron population activity, as indexed by a reduction in the number of spontaneously firing neurons per electrode track in the VTA, compared with virgin rats. Collectively, our findings suggest that parity can drive changes in affective behavior (i.e. increases anxiety-like behavior) and reduces social motivation during the early postpartum period and that these behavioral changes are associated with an attenuation of DA activity within this period.

Plenary Lecture: Addiction From Brain Mechanisms to New Treatments
15:15-16:15, 22 April 2018, Sunday

Prof David J. NUTT

Edmund J. Safra Professor, Neuropsychopharmacology
Head of Neuropsychopharmacology Unit and
Molecular Imaging Imperial College London, United Kingdom
(for bio details, please refer to p.13)

Abstract

Addiction is a complex set of disorders in which both the entry factors and restraining factors are important. Entry factors include features suc has drug likeing and reward but also self-medication for stress-related disorders such as depression and PTSD. Some people just find that taking drugs makes then :whole" or gives meaning to their lives so find it hard to stop. The mechanisms underlying these processes are beginning to be understood and new treatments are being developed based on these. It turns out that dopamine has much less of a role to play in reward and the initiation of drug use than was previously thought and our own research suggests that the endorphin system may be crucially involved in some patients.

Addiction also is a form of (aberrant) learning and recent research suggests that alterations in the GABA-A system, a critical moderator of glutamate-induced learning, is abnormal in people addiction, particularly a newly discovered subtype the a5 GABA-A receptor which has a largely limbic distribution in humans. This discovery too offers a new potential approach to treatment development.

Finally Prof Nutt shall explore the greatest challenge in addiction treatment today – keeping people who have become abstinent drug free when they experience pressures to resume drug-taking. Currently for opioid addiction the only options are substitute medicines such as methadone and buprenorphine that reinstate addiction with a safer alternative drug or naltrexone which has low compliance. He will share data from our new large imaging study – ICCAM- that has used fMRI and drug challenge techniques to explore stress, reward and impulsivity systems in abstinent alcoholics and heroin and cocaine addicts. In this group we have tested a number of non-addictive drugs and shown some to moderate the fMRI brain responses in a manner that could indicate therapeutic potential.

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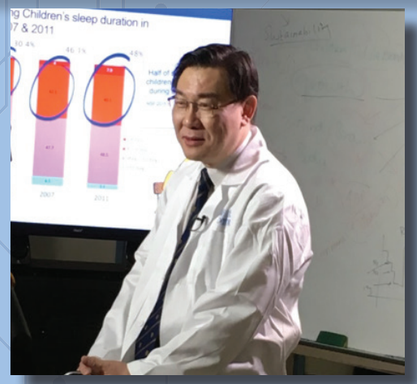
Neurobiology of Sleep:
16:15-17:00, 22 April 2018, Sunday



Prof WING Yun Kwok

Chairman and Professor, Department of Psychiatry
Associate Dean (Student Affairs), Faculty of Medicine,
The Chinese University of Hong Kong
Director of the Sleep Assessment Unit, Shatin Hospital

Professor Wing graduated from The Chinese University of Hong Kong, Hong Kong SAR, China. He is currently the Chairman and Professor in the Department of Psychiatry and Associate Dean (Student affairs) of the Faculty of Medicine of the Chinese University of Hong Kong. He is also the Director of the Sleep Assessment Unit of Shatin Hospital. He has been the Honorary Chief of Service in the Department of Psychiatry in both Shatin Hospital and Prince of Wales Hospital since 2003.

Professor Wing has diverse research interest in sleep and circadian medicine, psychiatric disorders, neuropsychiatry, and transcultural psychopharmacology with extensive publications in international journals. He is actively contribution to the scientific communities, including his leadership service role in the Hong Kong Society of Sleep medicine (ex-President, HKSSM) and Asian Sleep Society of Sleep medicine (ASSM, Vice-president (Research), Hong Kong Society of Biological Psychiatry (Committee member and Chairman of scientific committee). Furthermore, he also involved in the Collegium Internationale Neuropharmacologicum (Local organizing Committee of biennial CINP symposium, 2010), World Association of Sleep Medicine (Scientific Committee, 2011, 2013 and 2015) and World Sleep 2017 (Scientific Committee).

He has published more than 200 peer refereed publications and 6 book chapters. His current H-index is 48 (Google Scholar); 39 (Scopus) and 35 (ISI) (Aug 2017) respectively. He has given a number of plenary lectures in international and national sleep meetings including World Association of Sleep Meicine (WASM) 2015 (Korea) and Asean Sleep Congress 2015 (Singapore). He and his research group has established the first local epidemiological data of various sleep disorders including insomnia, narcolepsy, and parasomnia since 2 decades ago. Furthermore, he has constructed a RBD screening and severity scale – RBD-HK, which has been translated and used by a number of other research groups. In addition, he has recently conducted a large scale of cluster RCT study on sleep education in Hong Kong children and adolescents.

Professor Wing was awarded the distinguished national award for Sleep Medicine Scientific Technological Advance in China by the Chinese Medical Doctor Association at 2010 and distinguished contributions to the development of sleep medicine and sleep research by Chinese Sleep Research Society at 2016. He was also awarded the Teacher of the Year Awards, Faculty of Medicine, CUHK in 2012-13.

Abstract

Sleep is an essential part of human functioning. Recent advances in understanding sleep-related neurocircuitry and the discovery of its importance in metabolic clearance and memory consolidation have given new impetus of understanding its links with health and diseases. In this lecture, we will review some of the latest findings on the basic and clinical sciences of sleep and sleep disorders from neuroscience and psychiatric perspectives. We will discuss the associations of sleep disorders with neuropsychiatric disorders by using insomnia and REM sleep behavior disorder as examples. In addition, the roles of circadian rhythms on mood disorders will be also discussed.

Notes to Delegates

Meeting Organizer
Hong Kong Society of Biological Psychiatry

Meeting Secretariat
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Tel: +852 9658 9650
Fax: +852 3010 8969
E-mail: enquiry@hksbp.org

Meeting Date
21-22 April 2018, Saturday and Sunday

Meeting Venue
Sung Room, 4/F, Sheraton Hong Kong Hotel & Towers, 20 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 dinner conference, lunch symposia 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 21 April 2018 and 12:00–17:00 on 22 April 2018. The 2 lunch symposia are sponsored by Lundbeck and Janssen respectively on 21 and 22 April.

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

Insurance
The organizing committee of the 11th 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
Sheraton Hong Kong Hotel and Towers is a smoke-free premise. No indoor smoking is allowed.

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 11th ASM.

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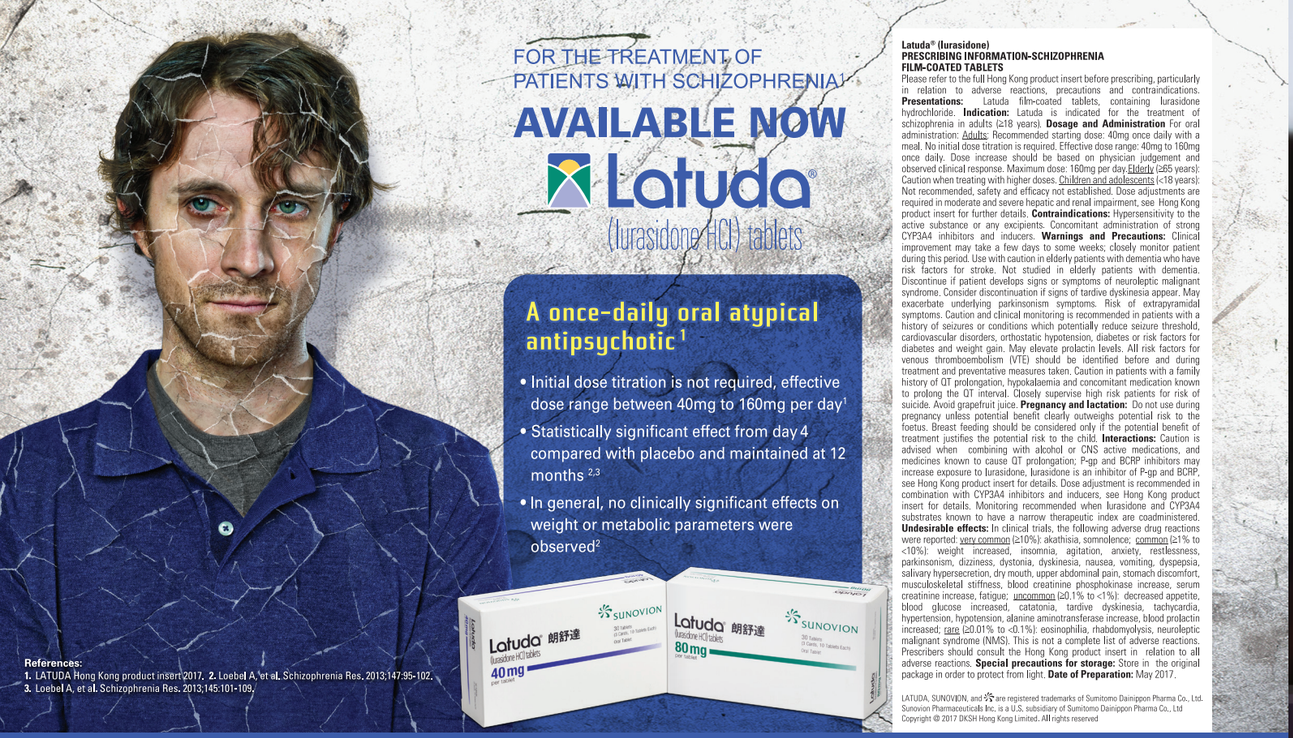

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Latuda® (lurasidone)
PRESCRIBING INFORMATION-SCHIZOPHRENIA
FILM-COATED TABLETS
Please refer to the full Hong Kong product insert before prescribing, particularly in relation to adverse reactions, precautions and contraindications. **Presentations:** Latuda film-coated tablets, containing lurasidone hydrochloride. **Indication:** Latuda is indicated for the treatment of schizophrenia in adults (≥18 years). **Dosage and Administration** For oral administration: Adults: Recommended starting dose: 40mg once daily with a meal. No initial dose titration is required. Effective dose range: 40mg to 160mg once daily. Dose increase should be based on physician judgement and observed clinical response. Maximum dose: 160mg per day. Elderly (≥65 years): Caution when treating with higher doses. Children and adolescents (<18 years): Not recommended, safety and efficacy not established. Dose adjustments are required in moderate and severe hepatic and renal impairment, see Hong Kong product insert for further details. **Contraindications:** Hypersensitivity to the active substance or any excipients. Concomitant administration of strong CYP3A4 inhibitors and inducers. **Warnings and Precautions:** Clinical improvement may take a few days to some weeks; closely monitor patient 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 patient develops signs or symptoms of neuroleptic malignant syndrome. Consider discontinuation if signs of tardive dyskinesia appear. May exacerbate underlying parkinsonism symptoms. Risk of extrapyramidal symptoms. Caution and clinical monitoring is recommended in patients with a history of seizures or conditions which potentially reduce seizure threshold, cardiovascular disorders, orthostatic hypotension, diabetes or risk factors for diabetes and weight gain. May elevate prolactin levels. All risk factors for venous thromboembolism (VTE) should be identified before and during treatment and preventative measures taken. Caution in patients with a family history of QT prolongation, hypokalaemia and concomitant medication known to prolong the QT interval. Closely supervise high risk patients for risk of suicide. Avoid grapefruit juice. **Pregnancy and lactation:** Do not use during pregnancy unless potential benefit clearly outweighs potential risk to the foetus. Breast feeding should be considered only if the potential benefit of treatment justifies the potential risk to the child. **Interactions:** Caution is advised when combining with alcohol or CNS active medications, and medicines known to cause QT prolongation; P-gp and BCRP inhibitors may increase exposure to lurasidone, lurasidone is an inhibitor of P-gp and BCRP, see Hong Kong product insert for details. Dose adjustment is recommended in combination with CYP3A4 inhibitors and inducers, see Hong Kong product insert for details. Monitoring recommended when lurasidone and CYP3A4 substrates known to have a narrow therapeutic index are coadministered. **Undesirable effects:** In clinical trials, the following adverse drug reactions were reported: 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 creatinine phosphokinase increase, serum creatinine increase, fatigue; uncommon (≥0.1% to <1%): decreased appetite, blood glucose increased, catatonia, tardive dyskinesia, tachycardia, hypertension, hypotension, alanine aminotransferase increase, blood prolactin increased; rare (≥0.01% to <0.1%): eosinophilia, rhabdomyolysis, neuroleptic malignant syndrome (NMS). This is not a complete list of adverse reactions. Prescribers should consult the Hong Kong product insert in relation to all adverse reactions. **Special precautions for storage:** Store in the original package in order to protect from light. **Date of Preparation:** May 2017.

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New formulation & NEW Hope for Depression with Insomnia

WELLBUTRIN XL™ ABBREVIATED PRESCRIBING INFORMATION INDICATIONS AND USAGE Treatment of major depressive disorder and prevention of seasonal major depressive episodes in patients with a diagnosis of seasonal affective disorder. **DOSAGE AND ADMINISTRATION** General Dosing Considerations: Insomnia may also be minimized by avoiding bedtime doses. WELLBUTRIN XL Tablets should be swallowed whole and not crushed, divided, or chewed. Major Depressive Disorder: The usual adult target is 300 mg/day, given once daily in the morning. Dosing should begin at 150 mg/day given as a single daily dose in the morning. If the 150-mg initial dose is adequately tolerated, an increase to the 300-mg/day target dose, given as once daily, may be made as early as day 4 of dosing. There should be an interval of at least 24 hours between successive doses. As with other antidepressants, full antidepressant effect of WELLBUTRIN XL may not be evident until 4 weeks of treatment or longer. An increase in dosage to the maximum of 450 mg/day, given as a single dose, may be considered for patients in whom no clinical improvement is noted after several weeks of treatment at 300 mg/day. Seasonal Affective Disorder: Dosing should begin at 150 mg/day given as a single daily dose in the morning. If the 150-mg initial dose is adequately tolerated, the dose of WELLBUTRIN XL should be increased to the 300-mg/day dose after 1 week. If the 300-mg dose is not adequately tolerated, the dose can be reduced to 150 mg/day. The usual adult target dose for WELLBUTRIN XL Tablets is 300 mg/day, given once daily in the morning. For patients taking 300 mg/day during the autumn-winter season, the dose should be tapered to 150 mg/day for 2 weeks prior to discontinuation. **CONTRAINDICATIONS** Patients with hypersensitivity to bupropion or any of the other components of the preparation; patients with a seizure disorder; patients undergoing abrupt discontinuation of alcohol or sedatives; patients currently being treated with any other preparation containing bupropion as the incidence of seizure is dose dependent; patients with a current or previous diagnosis of bulimia or anorexia nervosa. Concomitant use of WELLBUTRIN XL and monoamine oxidase inhibitors (MAOIs) is contraindicated. At least 14 days should elapse between discontinuation of MAOIs and initiation of treatment with WELLBUTRIN XL tablets. **WARNINGS AND PRECAUTIONS Clinical Worsening and Suicide Risk with Psychiatric Disorders:** Patients with major depressive disorder (MDD), both adult and pediatric, may experience worsening of their depression and/or the emergence of suicidal ideation and behavior (suicidality) or unusual changes in behavior, whether or not they are taking antidepressant medications, and this risk may persist until significant remission occurs. All patients being treated with antidepressants for any indication should be monitored appropriately and observed closely for clinical worsening, suicidality, and unusual changes in behavior, especially during the initial few months of a course of drug therapy, or at times of dose changes, either increases or decreases. Pooled analyses of short-term placebo-controlled trials of antidepressant drugs (SSRIs and others) showed that these drugs increase the risk of suicidal thinking and behavior (suicidality) in children, adolescents, and young adults (ages 18 to 24) with major depressive disorder (MDD) and other psychiatric disorders. Families and caregivers of patients being treated with antidepressants for major depressive disorder or other indications, both psychiatric and nonpsychiatric, should be alerted about the need to monitor patients for the emergence of agitation, irritability, unusual changes in behavior, as well as the emergence of suicidality, and to report such symptoms immediately to healthcare providers. Consideration should be given to changing the therapeutic regimen, including possibly discontinuing the medication, in patients whose depression is persistently worse, or who are experiencing emergent suicidality or symptoms that might be precursors to worsening depression or suicidality, especially if these symptoms are severe, abrupt in onset, or were not part of the patient's presenting symptoms. **Screening Patients for Bipolar Disorder:** Serious neuropsychiatric symptoms have been reported. A major depressive episode may be the initial presentation of bipolar disorder. It is generally believed that treating such an episode with an antidepressant alone may increase the likelihood of precipitation of a mixed/manic episode in patients at risk for bipolar disorder. Prior to initiating treatment with an antidepressant, patients with depressive symptoms should be adequately screened to determine if they are at risk for bipolar disorder; such screening should include a detailed psychiatric history, including a family history of suicide, bipolar disorder, and depression. **Seizures:** Bupropion is associated with a dose-related risk of seizures. The risk of seizures is also related to patient factors, clinical situations, and concomitant medications, which must be considered in selection. Should be discontinued and not restarted in patients who experience a seizure while on treatment. Patient factors: Predisposing factors that may increase the risk of seizure with bupropion use include history of head trauma or prior seizure, central nervous system (CNS) tumor, the presence of severe hepatic cirrhosis, and concomitant medications (e.g., antipsychotics, antidepressants, theophylline, systemic steroids) that lower seizure threshold. Clinical situations: Circumstances associated with an increased seizure risk include, among others, excessive use of alcohol or sedatives (including benzodiazepines); addiction to opiates, cocaine, or stimulants; use of over-the-counter stimulants and anorectics; and diabetes treated with oral hypoglycemics or insulin. Retrospective analysis of clinical experience gained during the development of bupropion suggests that the risk of seizure may be minimized if the total daily dose of WELLBUTRIN XL does not exceed 450 mg and the rate of incrementation of dose is gradual. **Impaired hepatic function:** WELLBUTRIN XL should be used with extreme caution in patients with severe hepatic cirrhosis. WELLBUTRIN XL should be used with caution in patients with hepatic impairment (including mild to moderate hepatic cirrhosis) and a reduced frequency and/or dose should be considered in patients with mild to moderate hepatic cirrhosis. All patients with hepatic impairment should be closely monitored for possible adverse effects that could indicate high drug and metabolite levels. **Impaired renal function:** Bupropion is extensively metabolized in the liver to active metabolites, which are further metabolized and excreted by the kidneys. WELLBUTRIN XL should be used with caution in patients with renal impairment and a reduced frequency and/or dose should be considered as bupropion and the metabolites of bupropion may accumulate in such patients to a greater extent than usual. The patient should be closely monitored for possible adverse effects that could indicate high drug or metabolite levels. **Paediatric Use:** Safety and effectiveness in the pediatric population have not been established. Anyone considering the use of WELLBUTRIN XL in a child or adolescent must balance the potential risks with the clinical need. **Geriatric use:** Reported clinical experience has not identified differences in responses between the elderly and younger patients, but greater sensitivity of some older individuals cannot be ruled out. **Cardiovascular Effects:** In clinical practice, hypertension, in some cases severe, requiring acute treatment, has been reported in patients receiving bupropion alone and in combination with nicotine replacement therapy. These events have been observed in both patients with and without evidence of preexisting hypertension. There is no clinical experience establishing the safety of WELLBUTRIN XL Tablets in patients with a recent history of myocardial infarction or unstable heart disease. Therefore, care should be exercised if it is used in these groups. **Pregnancy:** WELLBUTRIN XL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. **Nursing Mothers:** Bupropion and its metabolites are secreted in human milk. Decision should be made whether to discontinue nursing or to discontinue the drug. **INTERACTIONS** The potential exists for a drug interaction between WELLBUTRIN XL and drugs that are substrates of or inhibitors/inducers of the CYP2B6 isoenzyme (e.g., orphenadrine, thiotepa, cyclophosphamide, ticlopidine, and clopidogrel). Coadministration of bupropion with drugs that are metabolized by CYP2D6 isoenzyme including certain antidepressants (SSRIs and many tricyclics, e.g., nortriptyline, imipramine, desipramine, paroxetine, fluoxetine, sertraline), antipsychotics (e.g., haloperidol, risperidone, thioridazine), beta-blockers (e.g., metoprolol), and Type 1C antiarrhythmics (e.g., propafenone, flecainide), should be approached with caution and should be initiated at the lower end of the dose range of the concomitant medication. If bupropion is added to the treatment regimen of a patient already receiving a drug metabolized by CYP2D6, the need to decrease the dose of the original medication should be considered, particularly for those concomitant medications with a narrow therapeutic index. Drugs which require metabolic activation by CYP2D6 in order to be effective (e.g. tamoxifen) theoretically could have reduced efficacy when administered concomitantly with inhibitors of CYP2D6 such as bupropion. Although citalopram is not primarily metabolized by CYP2D6, in one study bupropion increased the Cmax and AUC of citalopram by 30% and 40%, respectively. In a series of studies in healthy volunteers, ritonavir (100 mg twice daily or 600 mg twice daily) or ritonavir 100 mg plus lopinavir 400 mg (Kaletra®) twice daily reduced the exposure of bupropion and its major metabolites in a dose dependent manner by approximately 20 to 80%. Similarly, efavirenz 600 mg once daily for two weeks reduced the exposure of bupropion by approximately 55%. This effect of ritonavir/Kaletra® and efavirenz is thought to be due to the induction of bupropion metabolism. Patients receiving any of these drugs with bupropion may need increased doses of bupropion but the maximum recommended dose of bupropion should not be exceeded. Multiple oral doses of bupropion had no statistically significant effects on the single dose pharmacokinetics of lamotrigine in 12 healthy volunteers. Administration of WELLBUTRIN XL to patients receiving either levodopa or amantadine concurrently should be undertaken with caution, There have been rare reports of adverse neuropsychiatric events or reduced alcohol tolerance in patients who were drinking alcohol during treatment with bupropion. The consumption of alcohol during treatment with WELLBUTRIN XL should be minimized or avoided. False-positive urine immunoassay screening tests for amphetamines have been reported in patients taking bupropion. Confirmatory tests, such as gas chromatography/mass spectrometry, will distinguish bupropion from amphetamines. Serotonergic psychiatric drugs should not be started in a patient receiving linezolid. Wait until 24 hours after the last dose of linezolid before starting the serotonergic psychiatric drugs. **EFFECTS ON ABILITY TO DRIVE AND USE MACHINE** Until they are reasonably certain that WELLBUTRIN XL do not adversely affect their performance, they should refrain from driving an automobile or operating complex, hazardous machinery. **ADVERSE REACTIONS** WELLBUTRIN XL has been demonstrated to have similar bioavailability both to the immediate-release formulation of bupropion and to the sustained-release formulation of bupropion. The information included under this subsection is based primarily on data from controlled clinical trials with WELLBUTRIN SR Tablets, the sustained-release formulation of bupropion. The following adverse events have been reported with a frequency of ≥1/100 to 1/10 (common) and ≥1/10 (very common). Immune system disorders Hypersensitivity reactions such as urticaria. Metabolic and Nutrition disorders anorexia. Psychiatric disorders Insomnia, agitation and anxiety. Nervous System disorder Headache, tremor, dizziness and taste disorders. Eye and labyrinth disorders Tinnitus. Ear and labyrinth disorders Tinnitus. Vascular disorders Increased blood pressure sometimes severe, flushing. Gastrointestinal disorders dry mouth, gastrointestinal disturbance including nausea and vomiting, abdominal pain, constipation. Skin and subcutaneous tissue disorders rash, pruritus, sweating. General disorders and administration site conditions fever, chest pain, asthenia **OVERDOSE** Seizures, hallucinations, loss of consciousness, sinus tachycardia, and ECG changes such as conduction disturbances (including QRS prolongation) or arrhythmias. Fever, muscle rigidity, rhabdomyolysis, hypotension, stupor, coma, and respiratory failure have been reported mainly when bupropion was part of multiple drug overdoses. Deaths associated with overdoses of bupropion alone have been reported in patients ingesting large doses of the drug. **Treatment:** Ensure an adequate airway, oxygenation, and ventilation. Monitor cardiac rhythm and vital signs. Activated charcoal should be administered. No specific antidotes for bupropion are known. Abbreviated Prescribing information based on version GDS22.

REFERENCE: 1. Wellbutrin™ Hong Kong prescribing information GDS22.

GlaxoSmithKline Limited
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HKRXBHC/0001/17 (02/2019)
Date of preparation: 27/02/2017

For PATIENTS WITH SCHIZOPHRENIA

Abilify Maintena®

(aripiprazole) for extended release injectable suspension

Help your patients stay relapse-free[2]

- Over 90% of patients remained relapse-free over 38 weeks[3]
- Significantly reduced hospitalization rates vs oral SOC antipsychotics in a naturalistic, open-label study[4]
- Significantly improved positive and negative symptoms as early as week 1 and at all time points, as measured by PANSS and CGI-S[5]

SOC = Standard of Care PANSS = Positive and Negative Syndrome Scale CGI-S = Clinical Global Impression – Severity Scale

References: 1. Abilify Maintena Package Insert. **2.** Kane JM et al. *J Clin Psychiatry.* 2012;73(5):617-624.
3. Fleischhacker WW et al. *Br J Psychiatry.* 2014;205(2):135-144. **4.** Kane JM et al. *J Med Econ.* 2013;16(7):917-925.
5. Kane JM et al. *J Clin Psychiatry.* 2014;75(11):1254-1260.

Otsuka Otsuka Pharmaceutical (H.K.) Ltd.
21/F, East Exchange Tower, 38 Leighton Road, Causeway Bay, Hong Kong. Tel: 2881 6299 Fax: 2577 5206

ONCE-MONTHLY 400mg
Abilify Maintena
(aripiprazole) extended release suspension for injection
Detailed information is available upon request

ABF201611 (approved in Sept 2016)

STILNOX® CR
ZOLPIDEM TARTRATE

combined for a
complete
sleep solution

Dual-layered to help patients fall asleep and maintain sleep[1]

First Layer
Immediate release for rapid onset of sleep[1]

Second Layer
Extended release of zolpidem for improved sleep maintenance[1]

for the elderly

for adults

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. Elderly: 6.25mg daily immediately before sleep. Hepatic impairment: 6.25mg daily. **Contraindications:** Hypersensitivity to zolpidem or excipients. Obstructive 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. Discontinue treatment once physical dependence develops. The attention of drivers of vehicles and machine operators should be drawn to the possible risk of drowsiness. 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 depression and psychosis. Discontinue treatment if psychiatric, paradoxical reactions or somnambulism occur. Limit repeat prescription without adequate medical supervision to avoid risk of abuse. **Interactions:** CNS depressants, alcohol, imipramine, chlorpromazine. 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. ***Full prescribing information is available upon request.***

API-HK-ZOL-13.11

sanofi-aventis Hong Kong Limited
Units 706-710, Level 7, Core C, Cyberport 3, 100 Cyberport Road, Hong Kong
Tel : (852) 2506 8333 Fax : (852) 2506 2537 Website: www.sanofi.hk

SA-HK.ZOL.17.02.0046

SANOFI

長效思緒康™
Once Daily
Seroquel XR™
quetiapine

ONE FOR ALL
MAJOR MENTAL DISORDERS

MDD = Major Depressive Disorder; GAD = Generalized Anxiety Disorder
* 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,
North Point, Hong Kong.
Tel: 2420 7388 Fax: 2422 6788

AstraZeneca
阿斯利康

Notes

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Lundbeck Hong Kong
Suite 4303, Central Plaza,
18 Harbour Road, Wanchai, Hong Kong
Tel: 2244 8888 Fax: 2827 2228
www.lundbeck.com

Brintellix® vortioxetine

Take care of ***more than mood***

BRINTELLIX (VORTIOXETINE) – PRESCRIBING INFORMATION
Presentation: Film-coated tables 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 an 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, trypthophan, 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: Decreased appetite, abnormal dreams, dizziness, diarrhoea, constipation, vomiting, itching. Uncommon: Grinding one's teeth, flushing, night sweats. Overdose: Symptomatic treatment. Marketing authorisation holder: Lundbeck HK Limited. Revision Date: 28 April 2015. Full prescribing information is available upon request

HK-BRN-2018.03-ADV01-MPL

An Unique Treatment Option to Achieve Recovery[1]

善妥達®
INVEGA TRINZA®
paliperidone palmitate extended-release injectable suspension
175 mg, 263 mg, 350 mg, 525 mg

Symptomatic Remission[1]
50.3%

Relapse Free[2]
91%

Functional Remission[1]
27.3%

≥4 MONTHS

Patients must be adequately treated with for

INVEGA® SUSTENNA® **AT LEAST 4 MONTHS**[3]

References: 1. Savitz A et al. Int Clin Psychopharmacology 2017, Vol32, No.6:329-336. **2.** Savitz A et al. Int J neuropsychopharmacol 2016:1-14. **3.** Invega Trinza USPI Mar2016;[Hong Kong approval date: 25Jan2017].

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. 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. **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/Pglycoprotein (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.**

Janssen
PHARMACEUTICAL COMPANIES OF Johnson & Johnson

Unit 1302-7, Tower 1, Grand Century Place, 193 Prince Edward Road West, Mongkok, Kowloon, Hong Kong.
Tel : 2736 1711 Fax : 2736 1926

STEP to Recovery
Sustenna Trinza Establishing Program

CP-56883