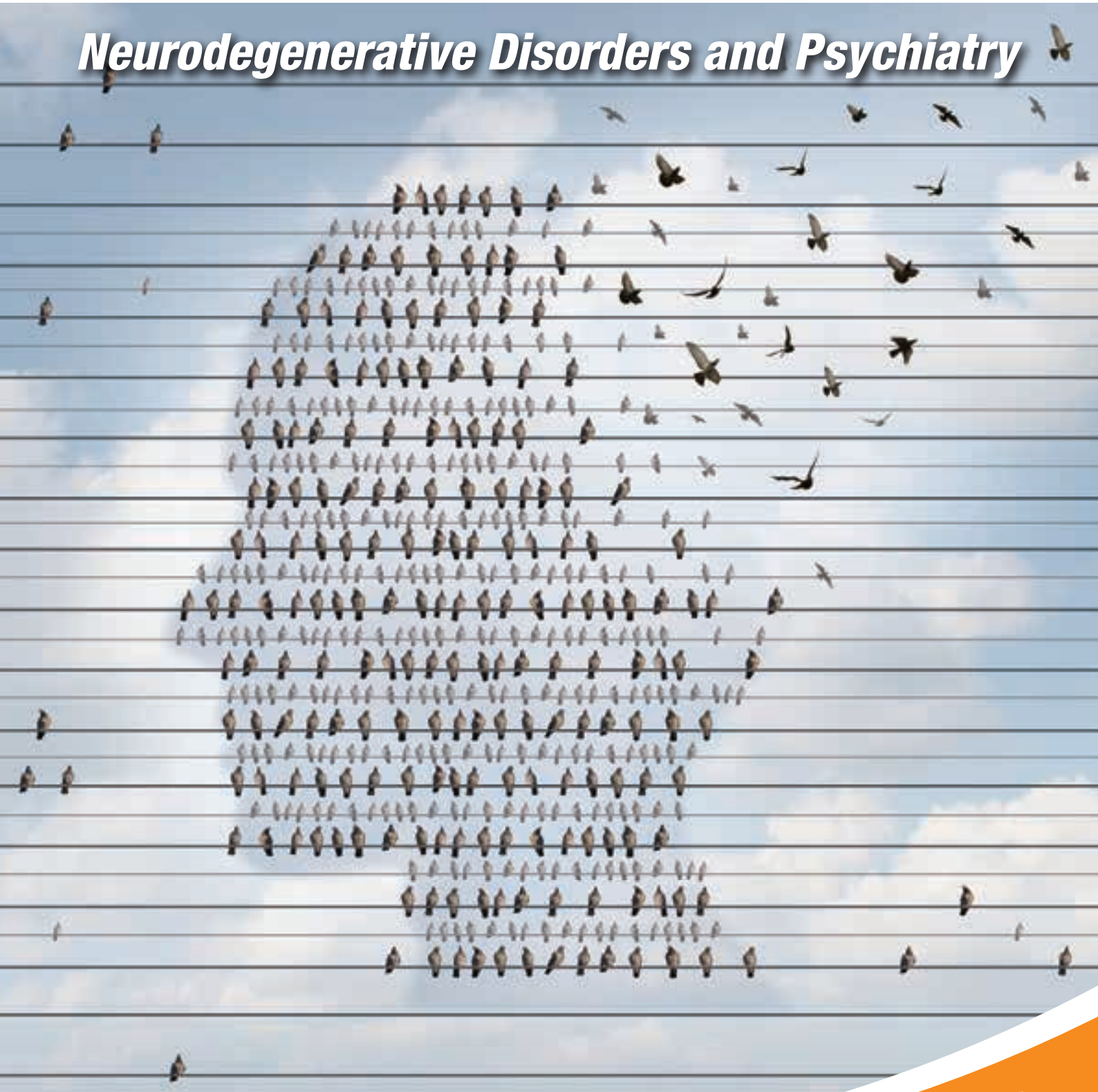
The Annual Scientific Meeting 2024 of
The Hong Kong Society of Biological Psychiatry

Neurodegenerative Disorders and Psychiatry



Programme Book

www.hksbp.org

March 16th – 17th 2024

The Cordis Hong Kong
Level 8, Cordis Hong Kong,
555 Shanghai Street, Kowloon

The Annual Scientific Meeting 2024 of
The Hong Kong Society of Biological Psychiatry

Neurodegenerative Disorders and Psychiatry

Table of Content

Neurodegenerative Disorders and Psychiatry

Welcome Message

On behalf of the Organizing Committee, I take great pleasure to welcome you to the Annual Scientific Meeting 2024 of the Hong Kong Society of Biological Psychiatry (HKSBP) on **16th (Saturday evening) and 17th (Sunday) in March 2024 at the Cordis Hotel Hong Kong, Mong Kok, Kowloon.**

The thematic for this year is Neurodegenerative Disorders and Psychiatry, and we have great honor to have renowned speakers sharing their works in the meeting. On Saturday evening, Dr. WONG Ming Cheuk Michael, President of HKSBP will talk about the multimodal antidepressant drug in the treatment of depression. On Sunday, Prof. So Kwok Fai, Director of Guangzhou-Hong Kong-Macau Institute of CNS Regeneration will share with us the anti-depression effect of goji glycopeptide. Prof. LEE Mei Chun Tatia from the University of Hong Kong will share her work on how loneliness can be a significant threat to the mental health of older adults. Dr. WONG Ming Cheuk Michael and Dr. TSANG Suk Kwan Jenny will discuss the role of NMDA receptors and its modulators in depression with case sharing. Other titles include “When ‘A’ partial agonist meets ‘R‘ partial agonist” by Dr. CHUNG Kar Kin Albert from the University of Hong Kong, and “Mechanistic investigations on how targeting GLP-1 signaling may help age-related neurodegeneration” by Dr. KO Ho Owen from the Chinese University of Hong Kong. Last but not least, Prof. WING Yun Kwok from the Chinese University of Hong Kong will share with us on the topic “Sleep and Psychiatry – the Windows for the Prevention of Neurodegeneration”.

We look forward to meeting you at this intellectually exciting educational event.

Yours sincerely,

Dr. CHUNG Kar Kin, Albert
Chairperson, Organizing Committee of the ASM 2024
Hong Kong Society of Biological Psychiatry

Annual Scientific Meeting 2024 Organizing Committee

Chairperson of Organizing Committee:	**Dr. CHUNG Kar Kin, Albert**
Chairperson of Scientific Committee:	**Prof. WING Yun Kwok**
Scientific Committee Members:	**Dr. WONG Ming Cheuk, Michael**
	Dr. CHEUNG Hon Kee, Henry
	Dr. TSANG Suk Kwan, Jenny
Other Members:	**Dr. IU Pui Chuen**
	Dr. LO Chun Wai
	Dr. TAM Mo Shing, Paul
	Prof. TANG Siu Wa
	Dr. WONG Chung Hin, Willy

HKSBP Council Member 2023-2024

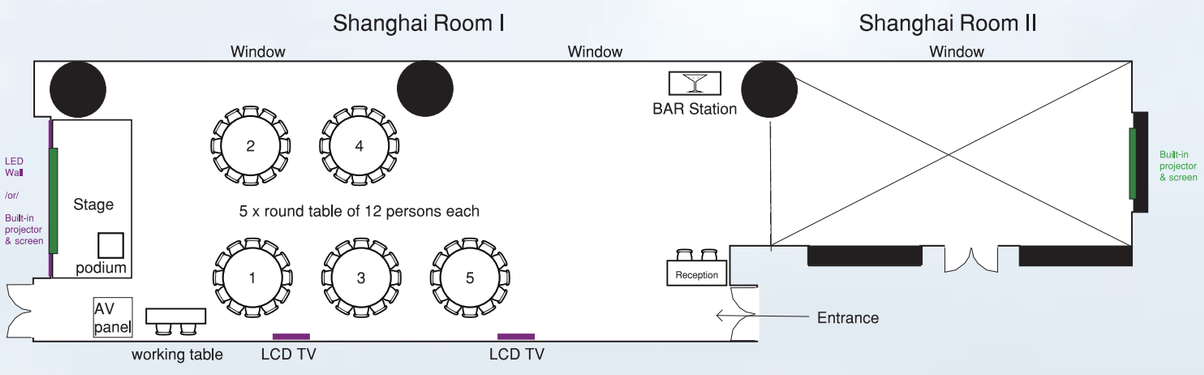
Current Past and Founding President:	**Prof. TANG Siu Wa**
President:	**Dr. WONG Ming Cheuk, Michael**
Vice President:	**Dr. CHUNG Kar Kin, Albert**
Honorary Secretary:	**Dr. CHEUNG Hon Kee, Henry**
Honorary Treasurer:	**Dr. TSANG Suk Kwan, Jenny**
Committee Members:	**Dr. IU Pui Chuen**
	Dr. LO Chun Wai
	Dr. TAM Mo Shing, Paul
	Prof. WING Yun Kwok
	Dr. WONG Chung Hin, Willy

The Annual Scientific Meeting 2024 of
The Hong Kong Society of Biological Psychiatry

Neurodegenerative Disorders and Psychiatry

Floor Plan for March 16th 2024, Saturday

The Cordis Hong Kong
Level 8, Cordis Hong Kong, 555 Shanghai Street, Kowloon

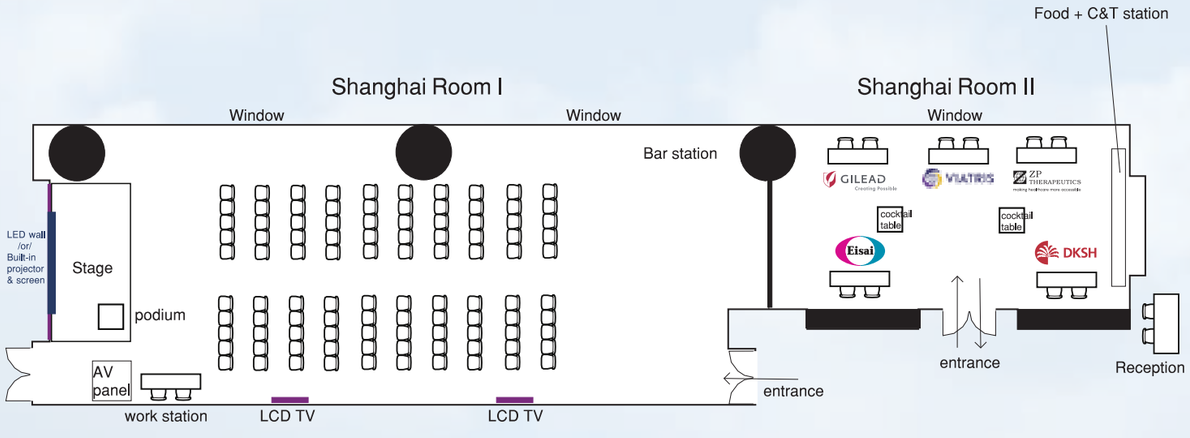


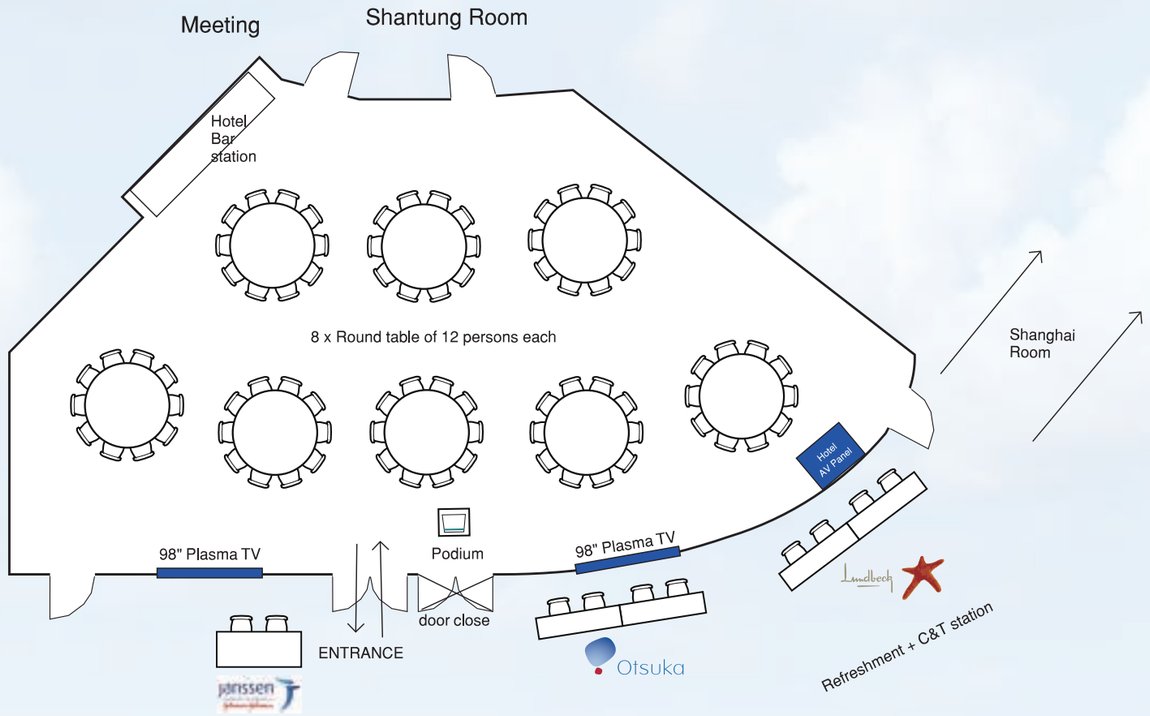
The Annual Scientific Meeting 2024 of
The Hong Kong Society of Biological Psychiatry

Neurodegenerative Disorders and Psychiatry

Floor Plan for March 17th 2024, Sunday

The Cordis Hong Kong
Level 8, Cordis Hong Kong, 555 Shanghai Street, Kowloon





Neurodegenerative Disorders and Psychiatry Scientific Programme

Time	March 16th 2024, Saturday	Venue
18:30	Registration	Foyer, Level 8
19:00-22:00 19:00-20:30 20:30-22:00	**Dinner Symposium** *(Sponsored by Lundbeck Hong Kong)* **Multimodal Antidepressant Drug – A Swiss Army Knife in the Treatment of Depression with Case Sharing** **Dr. WONG Ming Cheuk, Michael** Specialist in Psychiatry **Dr. CHEUNG Hon Kee, Henry** Specialist in Psychiatry **Chairperson: Dr. CHEUNG Hon Kee, Henry, Specialist in Psychiatry** Conference Dinner	Shanghai Room I, Level 8
Time	**March 17th 2024, Sunday**	**Venue**
09:00	Registration	Foyer, Level 8
09:00-9:30	Exhibition & Coffee	Shanghai Room II & Foyer, Level 8
09:30-10:30	**Lecture 1: The Anti-depression Effect of Goji Glycopeptide** **Prof. SO Kwok Fai** Director, GHM Institute of CNS Regeneration, Jinan University, Guangzhou, China **Chairperson: Dr. LO Ka Ying, Heidi, Clinical Assistant Professor, Department of Psychiatry, The University of Hong Kong**	Shanghai Room I, Level 8
10:30-11:30	**Lecture 2: Loneliness is a Significant Threat to the Mental Health of Older Adults** **Prof. LEE Mei Chun, Tatia** May Endowed Professor of Neuropsychology Chair Professor, Psychological Science and Clinical Psychology, The University of Hong Kong **Dr. Adrian WONG (Discussant)** Adjunct Associate Professor, Department of Medicine and Faculty of Medicine, The Chinese University of Hong Kong **Chairperson: Dr. CHANG Wing Chung, Head of Department of Psychiatry, LKS Faculty of Medicine, The University of Hong Kong**	
11:30-12:30	**Lecture 3: The Role of NMDA Receptors and its Modulators in Depression with Case Sharing** **Dr. WONG Ming Cheuk, Michael** Specialist in Psychiatry **Dr. TSANG Suk Kwan, Jenny** Specialist in Psychiatry **Chairperson: Dr. TSANG Suk Kwan, Jenny, Specialist in Psychiatry** *(Sponsored by Janssen Hong Kong)*	
12:30-14:00	**Lunch Symposium: When 'A' Partial Agonist Meet 'R' Partial Agonist** **Dr. CHUNG Kar Kin, Albert** Clinical Assistant Professor, Department of Psychiatry, University of Hong Kong **Chairperson: Dr. CHEUNG Yat Wo, Eric, Specialist in Psychiatry** *(Sponsored by Otsuka)*	Shangtung Room, Level 8
14:00-15:00	**Lecture 4: Mechanistic Investigations on How Targeting GLP-1 Signaling May Help Age-related Neurodegeneration** **Dr. KO Ho, Owen** Associate Professor, Department of Medicine and Therapeutics, Faculty of Medicine, The Chinese University of Hong Kong **Chairperson: Dr. HO Wing Lok, Philip, Assistant Professor, Faculty of Health and Social Sciences, The Hong Kong Polytechnic University**	Shanghai Room I, Level 8
15:00-16:00	**Lecture 5: Sleep and Psychiatry – the Windows for the Prevention of Neurodegeneration** **Prof. WING Yun Kwok** Chairman & Professor, Department of Psychiatry, Faculty of Medicine, The Chinese University of Hong Kong Choh-Ming Li Professor of Psychiatry, The Chinese University of Hong Kong **Chairperson: Dr. HUANG Bei, Joanne, Research Assistant Professor, Department of Psychiatry, The Chinese University of Hong Kong**	
16:00-16:30	Exhibition & Coffee	Shanghai Room II & Foyer, Level 8
16:30-17:00	**AGM** *(For HKSBP Members ONLY)*	Shangtung Room, Level 8

Dinner Symposium: Multimodal Antidepressant Drug – A Swiss Army Knife in the Treatment of Depression with Case Sharing

19:00-20:30, March 16th 2024, Saturday

Dr. WONG Ming Cheuk, Michael

Dr. WONG Ming Cheuk, Michael is a psychiatrist working in private practice. Michael was a Consultant Psychiatrist and former Chief of Service in the Department of Psychiatry of Queen Mary Hospital in Hong Kong before he left the public service. He was 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.

Michael actively participating in the regional activities of professional societies. He was 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 and Asian Association of Neuropsychopharmacology. Apart from these, he has organized a number of conferences for professional societies. For example, he was one of the members of the Local Organizing Committee of CINP World Congress Hong Kong 2010 and a member of the Scientific Committee of the Annual Conference of the International Society of Bipolar Disorder in the past. Besides, he is also a speaker in local, regional and international conferences, and one of the editors of the International Journal of Bipolar Disorder and reviewer in peer review journals.

He is also active in community services, particularly in the rehabilitation of mental patients and the promotion of mental health in the community.

Abstract

Major depressive disorder is a disorder which involves different symptom domains. Apart from emotional symptoms such as feeling of sadness, loss of enjoyment, anxiety and worries, the patient may have both cognitive and physical symptoms as well. Quite often, these symptoms of depression may not respond to the conventional antidepressant drugs to the same extent. Even when the emotional symptoms have improved with a course of the commonly used antidepressant drugs such as SSRI & SNRI, some patients may still have significant cognitive and physical symptoms such as poor concentration, lack of drive and sleep disturbance. This suggests that the conventional antidepressant drugs, which block the reuptake of monoamines in the synapses and thus increase monoamine activities, may not be able to address all the symptom of depression. Furthermore, too high dose of SSRI and SNRI may lead to the phenomenon of emotional blunting, which may be misinterpreted as the symptom of depression. Antidepressant drugs with addition mechanism of action will be needed to address the different symptom in different domains in the treatment of depression. This presentation will discuss about the mechanism of action of the newer generation multimodal antidepressant drugs and how these drugs improve the treatment outcome of depression.

Lecture 1: The Anti-depression Effect of Goji Glycopeptide

09:30-10:30, March 17th 2024, Sunday

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 China-SCINet (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). 2021 - 2023 is listed the world top 2% neuroscientists. "The World's Top Neuroscientists (2023)" - Top Ten in China, Cited more than 28,075 times , H-index 88 (GOOGLE SCHOLAR) He is the author and co-author of over 500+ publications (http://-scholar.google.com/citations?hl=en&user=SUPKYiQA-AAAJ&view_op=list_works)
; co-inventors of 46 patents.

Abstract

Subthreshold depression is a highly prevalent condition in adolescents who are at high risk for developing major depressive disorder. In preclinical models of neurological and psychiatric diseases, Lycium barbarum polysaccharide (LBP) extracted from Goji berries had anti- depressant effects including but not limited to anti-oxidative and anti-inflammatory properties. However, the effect of LBP on subthreshold depression is unclear. To investigate the clinical efficacy and safety of LBP for treating subthreshold depression in adolescents, we conducted a randomized, double-blind, placebo-controlled trial (RCT) with 29 adolescents with subthreshold depression recruited at The Fifth Affiliated Hospital of Guangzhou Medical University. The participants were randomly assigned to groups where they received either 300 mg LBP (LBP group, n = 15, 3 boys and 12 girls aged 15.13 2.17 years) or a placebo (placebo group, n = 14, 2 boys and 12 girls aged 15 1.71 years) for
6 successive weeks. Interim analyses revealed that the LBP group exhibited a greater change in Hamilton Depression Scale (HAMD-24) scores relative to the baseline and a higher remission rate (HAMD-24 total score 7) at 6 weeks compared with the placebo group. Scores on the Beck Depression Inventory-II (BDI-II), Pittsburgh Sleep Quality Index (PSQI), Kessler Psychological Distress Scale (Kessler), and Screen for Child Anxiety-Related Emotional Disorders (SCARED) were similar between the LBP and placebo groups. No side effects related to the intervention were reported in either group. These results indicate that LBP administration reduced depressive symptoms in adolescents with subthreshold depression. Furthermore, LBP was well tolerated with no treatment-limiting adverse events. Clinical trials involving a larger sample size are needed to further confirm the anti-depressive effects of LBP in adolescents with subthreshold depression. This study was approved by the Medical Ethics Committee of the Fifth Affiliated Hospital of Guangzhou Medical University (Guangzhou, China; approval No. L2019-08) on April 4, 2019 and was registered on ClinicalTrials.gov (identifier: NCT04032795) on July 25, 2019.

Lecture 2: Loneliness is a Significant Threat to the Mental Health of Older Adults

10:30 – 11:30, March 17th 2024, Sunday

Prof. LEE Mei Chun, Tatia

Tatia Lee is the Chair Professor of Psychological Science and Clinical Psychology and May Endowed Professor in Neuropsychology at The University of Hong Kong. Her research spans the frontiers of neuropsychology and human neuroscience, focusing on the neuroplastic basis of neurocognitive and affective processes underpinning normal and pathological psychological functions. Professor Lee has an impressive publication record of over 300 influential scientific articles. She has achieved significant international and national recognition for her outstanding contributions to the advancement of science. She was elected as a fellow of esteemed international societies, including the UK Academy of Social Sciences and The World Academy of Sciences. She is the Founding Chair of the Clinical Neuropsychological Society under the Chinese Cognitive Science Society of China and a Visiting Professor at King's College London.

Dr. Adrian WONG (Discussant)

Dr Adrian Wong is a registered Clinical Psychologist and an Associate Fellow of the Hong Kong Psychological Society. He graduated with distinction in professional clinical psychology training. Dr Wong is now a clinical psychologist in private practice and he serves as the Adjunct Associate Professor in the Faculty of Medicine at the Chinese University of Hong Kong. Dr Wong has more than 20 years of experience in the assessment and treatment of psychological and cognitive disorders. He has published over 120 articles in scientific journals and his research findings have translated into wide utilization in both public and private healthcare sectors in Hong Kong. As an advocate for psychological well-being in children, adults and elderly, Dr Wong has led over 100 training workshops for professionals and members of the public in Hong Kong, Greater China and around the globe. Dr Wong practices evidence-based assessment and treatment for developmental disorders and special education needs (e.g., Attention Deficit Hyperactivity Disorder, Autism, etc.) and for mood, anxiety and cognitive disorders in adults. Dr. Wong developed the Hong Kong Montreal Cognitive Assessment (HK-MoCA) which is now widely used as a standard cognitive screening tool in public and private healthcare sectors.

Abstract

Humans rely on safe and secure social environments to thrive as a social species. Research findings indicate that perceived loneliness (loneliness) is a risk factor for cognitive and mental health. Our previous work has also revealed that loneliness, when combined with a depressed mood, was negatively correlated to the general cognitive status of older adults.
In collaboration with scientists and clinical researchers from Taiwan, the US, and Hong Kong, we conducted a series of studies to understand the neurobiological basis of loneliness in late-life depression (LLD). Our findings indicate that grey matter volumes of the left putamen, caudate, and pallidum could differentiate between healthy controls and people suffering from LLD. Furthermore, a loneliness-related structural sub-network was found across the clinical participants with LLD. Loneliness was identified to have a unique role in the negative affective processing in LLD at functional brain connectional and network levels. Loneliness was also associated with altered neural regulatory functioning on self-referential processing and action control. The significant negative implication of loneliness on mental health calls for the employment of behavioural strategies for the intervention of the negative affective impacts of loneliness.

Lecture 3: The Role of NMDA Receptors and its Modulators in Depression with Case Sharing
11:30 – 12:30, March 17th 2024, Sunday

Dr. WONG Ming Cheuk, Michael

Dr. WONG Ming Cheuk, Michael is a psychiatrist working in private practice. Michael was a Consultant Psychiatrist and former Chief of Service in the Department of Psychiatry of Queen Mary Hospital in Hong Kong before he left the public service. He was 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.

Michael actively participating in the regional activities of professional societies. He was 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 and Asian Association of Neuropsychopharmacology. Apart from these, he has organized a number of conferences for professional societies. For example, he was one of the members of the Local Organizing Committee of CINP World Congress Hong Kong 2010 and a member of the Scientific Committee of the Annual Conference of the International Society of Bipolar Disorder in the past. Besides, he is also a speaker in local, regional and international conferences, and one of the editors of the International Journal of Bipolar Disorder and reviewer in peer review journals.

He is also active in community services, particularly in the rehabilitation of mental patients and the promotion of mental health in the community.

Abstract

Since the first tricyclic antidepressant drug was used in the treatment of depression, most of the subsequent antidepressant drugs such as other tricyclic antidepressant drugs, tetracyclic antidepressant drugs, SSRI, SNRI, NDRI, and the newer generation multimodal antidepressant drugs are modulators of monoamine activities in the brain. However, about 1/3 of patients with depression do not response to this class of drugs. On the other hand, drugs without direct effect on monoamine activities, e.g. Lithium, Valproate, Valproate, can have antidepressant effect or can augment the clinical effect of antidepressants. This leads us to think whether neurological systems other than the monoamine system are involved in the pathogenesis of depression. Among the various systems, the glutamatergic system is the most widely studied one. Abnormality in glutamatergic activities was found in areas of the brain which are involved in emotional regulation and depression. Clinical studies have found that some antagonists of NMDA receptor (one type of Glutamate receptors) have profound antidepressant effect. Among these NMDA receptor antagonists, Ketamine, an anaesthetic agent, is the most widely studied one. This presentation will look at the evidence of the involvement of the Glutamatergic systems in the pathology of depression and the role of some of the NMDA receptor antagonists in the treatment of depression.

Lecture 3: The Role of NMDA Receptors and its Modulators in Depression with Case Sharing
11:30 – 12:30, March 17th 2024, Sunday

Dr. TSANG Suk Kwan, Jenny

Dr. Jenny Tsang is a private psychiatrist who joined psychiatric service after she graduated from University of Hong Kong. Apart from general adult psychiatry, Dr. Tsang had also worked in other subspecialties including community psychiatry, psychogeriatric, child psychiatry, substance abuse and learning disability.

Dr. Tsang has received training in Psycho-oncology as visiting fellow at Oncology Centre of Princess Margaret Hospital of the University of Toronto and as Honorary Specialist Registrar at Psychiatric Department of Learning Disabilities of St. George's Hospital, London. After obtaining her specialist qualification in 2001, she worked in Liaison Psychiatry until she left the Hospital Authority in 2007. Dr Tsang has set up her private practice for more than 16 years. She remains deeply interest on psychopharmacology and Liaison Psychiatry.

Lunch Symposium: When ‘A’ Partial Agonist Meet ‘R‘ Partial Agonist

12:30 – 14:00, March 17th 2024, Sunday

Dr. CHUNG Kar Kin, Albert

Dr. Albert Chung is a Clinical Assistant Professor in the Department of Psychiatry, University of Hong Kong and is the psychiatrist in-charge of the Substance Abuse Clinic Services at Queen Mary Hospital, Hong Kong West Cluster. Passionate in teaching, he also serves as the Director of Education, Problem-based Learning Educator and Chief Examiner in the School of Clinical Medicine in the University of Hong Kong.

In addition, Dr. Chung is appointed as the Vice-President for the Hong Kong Society of Biological Psychiatry and serves as advisory group members in various government’s committees.

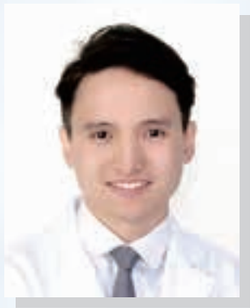
Dr. Chung was conferred fellowship with the Hong Kong Academy of Medicine and the Hong Kong College of Psychiatrists in 2010. He won the Distinguished Young Fellow award by the Hong Kong College of Psychiatrists in 2015. His research interests reside in clinical psychopharmacology, pharmacological and non-pharmacological interventions in substance misuse psychiatry (Substance misuse To Psychiatric disorders Program—“SToP Program").

Abstract

Schizophrenia and depression are both major global mental health concerns with significant impact on the patient’s functioning and subjective well-being. Enhancing early recovery should be the major collaborative goals between clinicians, patients and their families. With the development of drugs with partial D2 agonistic properties, life goes easier for all parties. The current talk will focus on how the newer D2 partial agonistic antipsychotic, Brexpiprazole, can shed light on treatment efficacy and better tolerability as compared to the other commonly used D2 drugs in managing schizophrenia and depression.

Lecture 4: Mechanistic Investigations on How Targeting GLP-1 Signaling May Help Age-related Neurodegeneration

14:00 – 15:00, March 17th 2024, Sunday



Dr. KO Ho, Owen

Ho Ko holds Bachelor of Medical Sciences (BMedSc), and Bachelor of Medicine and Bachelor of Surgery (MBChB) from the Chinese University of Hong Kong (CUHK). He pursued a PhD in neuroscience under the supervision of Thomas Mrsic-Flogel at University College London (UCL), where he worked closely with Sonja Hofer and Lee Cossell to reveal fundamental rules governing the connectivity between neurons with different functional roles in the brain. Based on his PhD works, he won a runner-up award of the 2014 Eppendorf & Science Prize for Neurobiology. Ho Ko is currently an associate professor at the Department of Medicine and Therapeutics, Faculty of Medicine, CUHK. He also serves the associate director of the Margaret K.L. Cheung Research Centre for Management of Parkinsonism and the Lau Tat-chuen Research Centre of Brain Degenerative Diseases in Chinese, and a principal investigator at the Li Ka Shing Institute of Health Sciences (LiHS) and the Gerald Choa Neuroscience Institute (GCNI) of CUHK. Leading a team with expertise in biology, chemistry and engineering, his current research works focus on three closely related themes: (i) Gliovascular dysfunction and therapeutic targeting in neurodegenerative disorders; (ii) Neural circuits mediating sensorimotor behaviors and their dysfunction in aging; (iii) Development of imaging tools and methods. Apart from a 2020 Croucher Innovation Award, his team’s research works have been recognized by a 2021 Excellent Young Scientists Fund from the National Natural Sciences Foundation of China, the 2022 Sir David Todd Lectureship from the Hong Kong College of Physicians, and an inaugural 2023 Asian Young Scientist Fellowship from the Future Science Awards Foundation.

Abstract

Identifying effective methods to counteract aging-associated changes is crucial for the development of therapeutics for many degenerative disorders. However, we still lack a readily implementable approach to alleviate aging phenotypes. Here, we conducted deep phenotyping in the aging mouse to demonstrate that glucagon-like peptide-1 receptor (GLP-1R) agonist (GLP-1RA) treatment effectively attenuates body-wide age-related changes. The effects are evident at multiple molecular levels, including the transcriptomes of various tissues, organs and circulating white blood cells, the plasma metabolome, and are accompanied by functional improvements in cognitive and physical performance. The molecular anti-aging effects are sensitive to hypothalamic GLP-1R knockdown, and correlate strongly with mTOR inhibition. In a cohort of human subjects treated with a GLP-1RA, we are employing longitudinal blood multi-omics to test potential generalization of the rejuvenation effects. Our findings have broad implications for the mechanistic understanding of the clinically observed pleiotropic effects of GLP-1RAs, the design of intervention trials for age-related diseases, and future developments of anti-aging-based therapeutics.

Lecture 5: Sleep and Psychiatry – the Windows for the Prevention of Neurodegeneration
15:00 – 16:00, March 17th 2024, Sunday

Prof. WING Yun Kwok

Professor Wing has made significant contributions to various areas of research including sleep and circadian medicine, psychiatric epidemiology across lifespan, neuropsychiatry, and latterly digital sleep and mental health. He has active contribution to the scientific communities, including his leadership role in the Hong Kong Society of Sleep Medicine (ex-President, HKSSM) and Asian Sleep Society of Sleep Medicine (ASSM, Vice-president). He was also involved in the World Association of Sleep Medicine (Scientific Committee, 2011, 2013 and 2015), World Sleep 2017, 2019 and 2022 (Scientific Committee) and program committee member in 2023. He has organised and chaired the Gordon Research conference on the "Cognitive Dysfunction in Brain diseases" in Hong Kong May 2019. He is currently serving at 4 major journals and has over 400 peer-reviewed publications in SCI ranked journals. He and his research group has established the first local epidemiological data of various sleep disorders including sleep deprivation, insomnia, narcolepsy, and parasomnia. In particular, the group has studied interaction of sleep and circadian dysfunction with psychiatric disorders. The group has also completed a novel prevention study of insomnia among at-risk adolescents. In addition, the group has extensive work on REM Sleep behavioral disorder, a sleep disorder with high specificity in predicting future alpha-synucelinopathy neurodegeneration and found a strong familial aggregation and staging pathology of alpha-synucleinopathy. The group is working on the gut-brain hypothesis at which the microbiota may have a significant role in predisposing to future synucleinopathy as well as the close relationship among psychiatric disorders, RBD and alpha-synucleinopathy.

Professor Wing has been 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 and mental health are not only pillar stones of a healthy life but also have a complex relationship with neurodegeneration. Sleep and mental health problems could contribute to pathoplastic process, act as both precursor and consequence of neurodegeneration. Notably, a variety of sleep and psychiatric disorders precede the onset of neurodegeneration by years, making them important target populations for the prevention of neurodegeneration. In particular, REM sleep behavior disorder (RBD), characterized by repetitive dream enactment behaviors, is considered to be the most specific precursor of alpha-synucleinopathies, as more than 90% of RBD eventually develop synucleinopathies within 15 years. In addition, depression has also been found to be associated with a higher risk of PD, dementia and RBD. In fact, RBD is not uncommon in depressed patients, and patients with depression co-morbid with RBD (MDD-RBD) have a later onset age of depression and higher risk of prodromal PD features than non-RBD psychiatric controls, suggesting that MDD-RBD is likely to be a subtype of depression representing an underlying synucleinopathy. Moreover, RBD is a familial aggregated disease, and the first-degree relatives of patients with RBD reported higher rates of RBD, PD, and dementia, and show increased neurodegenerative markers, suggesting that they may be at an even earlier stage of alpha-synucleinopathy than RBD patients. In addition, daytime sleepiness, anxiety, stress (stressful life events), etc., are also related to a higher risk of neurodegeneration including synucleinopathies and tauopathy, and could serve as potential targeted population for prevention. Similarly, both short and long sleep duration was suggested to be at risk of future dementia/cognitive decline. Early identification and intervention of the sleep and mental features, targeting populations at early "reversible" prodromal stages of disease and potentially modifiable factors may help to prevent future neurodegeneration.

Notes to Delegates

Meeting Organizer
Hong Kong Society of Biological Psychiatry

Meeting Secretariat
c/o Kays Asia (Hong Kong) Ltd.
Tel: +852 9420 5529
E-mail: conference.hksbp@gmail.com

Meeting Date
March 16th – 17th 2024, Saturday and Sunday

Meeting Venue
Level 8, Cordis Hong Kong, 555 Shanghai Street, Kowloon

On-site Registration
The registration counter is located at the entrance of Shanghai Room 1.
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 & dinner 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 Shanghai Room 1. Please wear your identification badge at all times during the event, as it serves as your admission to all scientific sessions, tea refreshments and lunch/ 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 9:00-17:00 on March 17th 2024. The dinner symposia on March 16th 2024, is sponsored by Lundbeck, while Lecture 3 on March 17th 2024, is sponsored by Janssen Hong Kong. Additionally, the lunch symposia on March 17th 2024, is sponsored by Otsuka.

Meal Arrangement
Tea break, lunch and dinner will be served in different venues of the same floor. Dinner will be served in Shanghai Room 1 on March 16th 2024. Tea and refreshment will be served in Shanghai Room 2 while lunch will be served in the Shantung Room on March 17th 2024.

Insurance
The organizing committee of the ASM 2024 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
The Cordis Hong Kong 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 ASM 2024.

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HAVE CONFIDENCE IN CONSISTENT CHC CURE WITH **EPCLUSA®** FOR TODAY'S PATIENT ENVIRONMENT[1]

Epclusa® is proved suitable for a minimal monitoring approach (MINMON):[2]

- No pre-treatment genotyping
- Full course of treatment dispensed at the first visit
- No laboratory monitoring
- Remote contacts **(Final assessment at week 24)**

CHC: Chronic Hepatitis C

Product photo shown is not actual size.

Reference:
1. Epclusa Prescribing Information. (Version: HK-APR22-EU-MAR21-CGPS-AUG20).
2. Solomon SS, Wagner-Cardoso S, Smeaton L, et al. A minimal monitoring approach for the treatment of hepatitis C virus infection (ACTG A5360 [MINMON]): a phase 4, open-label, single-arm trial. Lancet Gastroenterol Hepatol. 2022;7(4):307-317.

EPCLUSA® Abbreviated Prescribing Information (Version: HK-APR22-EU-MAR21-CGPS-AUG20) **Presentation:** Pink, diamond-shaped, film-coated tablet of dimensions 20 mm x 10 mm, debossed on one side with "GSI" and "7916" on the other side. **Indications:** Epclusa is indicated for the treatment of chronic hepatitis C virus (HCV) infection in patients aged 12 years and older and weighing at least 30 kg. **Dosage:** Adults: one tablet, taken orally, once daily with or without food for 12 weeks. Patients aged 12 to < 18 years and weighing at least 30 kg: one tablet, taken orally, once daily with or without food for 12 weeks. Adult patients who have previously failed therapy with an NS5A-containing regimen: Epclusa with ribavirin for 24 weeks may be considered. Elderly: No dose adjustment is warranted for elderly patients. Renal impairment: Epclusa can be used in patients with severe renal impairment (estimated glomerular filtration rate [eGFR] < 30 mL/min/1.73 m²) and end stage renal disease (ESRD) requiring haemodialysis with no dose adjustment. Hepatic impairment: No dose adjustment of Epclusa is required for patients with mild, moderate, or severe hepatic impairment (CPT Class A, B, or C). Safety and efficacy of Epclusa have been assessed in patients with CPT Class B cirrhosis, but not in patients with CPT Class C cirrhosis. Paediatric population: The safety and efficacy of Epclusa in children aged less than 12 years or weighing less than 30 kg have not yet been established. **Contraindications:** Hypersensitivity to the active substances or to any of the excipients. Medicinal products that are strong P-glycoprotein (P-gp) or strong cytochrome P450 (CYP) inducers (carbamazepine, phenobarbital, phenytoin, rifampicin, rifabutin and St. John's wort). **Warnings and Precautions:** Epclusa should not be administered concurrently with other medicinal products containing sofosbuvir. Severe bradycardia and heart block: Life-threatening cases of severe bradycardia and heart block have been observed when sofosbuvir-containing regimens are used in combination with amiodarone. Amiodarone should only be used in patients on Epclusa when other alternative anti-arrhythmic treatments are not tolerated or are contraindicated. Patients should undergo cardiac monitoring in an in-patient setting for the first 48 hours of coadministration, after which outpatient or self-monitoring of the heart rate should occur on a daily basis through at least the first 2 weeks of treatment. Monitoring should also be carried out for patients who have discontinued amiodarone within the past few months and are to be initiated on Epclusa. All patients with concurrent or recent use of amiodarone should be warned of the symptoms of bradycardia and heart block and should be advised to seek medical advice urgently should they experience them. HCV/HBV (hepatitis B virus) co-infection: Cases of HBV reactivation, some of them fatal, have been reported during or after treatment with direct-acting antiviral agents. HBV screening should be performed in all patients before initiation of treatment. HBV/HCV co-infected patients are at risk of HBV reactivation, and should therefore be monitored and managed according to current clinical guidelines. Patients who have previously failed therapy with an NS5A-containing regimen: There are no clinical data to support the efficacy of sofosbuvir/velpatasvir for the treatment of patients who have failed treatment with a regimen containing another NS5A inhibitor. Treatment with Epclusa + RBV for 24 weeks can be considered for patients who have failed therapy on an NS5A-containing regimen and who are deemed at high risk for clinical disease progression and who do not have alternative treatment options. Renal impairment: Safety data are limited in patients with severe renal impairment (eGFR < 30 mL/min/1.73 m²) and ESRD requiring haemodialysis. Epclusa can be used in these patients with no dose adjustment when no other relevant treatment options are available. Use with moderate P-gp inducers or moderate CYP inducers: Co-administration of such medicinal products that are moderate P-gp or moderate CYP inducers (e.g. efavirenz, modafinil, oxcarbazepine or rifapentine) with Epclusa is not recommended. Use with certain HIV antiretroviral regimens: Patients receiving Epclusa concomitantly with elbasvir/grazoprevir/cobicistat/emtricitabine/tenofovir disoproxil fumarate or with tenofovir disoproxil fumarate and a boosted HIV protease inhibitor should be monitored for tenofovir-associated adverse reactions. Use in diabetic patients: Diabetics may experience improved glucose control, potentially resulting in symptomatic hypoglycaemia, after initiating HCV direct-acting antiviral treatment. Glucose levels of diabetic patients initiating direct-acting antiviral therapy should be closely monitored, particularly within the first 3 months, and their diabetic medication modified when necessary. The physician in charge of the diabetic care of the patient should be informed when direct-acting antiviral therapy is initiated. CPT Class C cirrhosis: Safety and efficacy of Epclusa has not been assessed in patients with CPT Class C cirrhosis. Liver transplant patients: The safety and efficacy of Epclusa in the treatment of HCV infection in patients who are post-liver transplant have not been assessed. **Adverse reactions:** Common adverse drug reactions include rash. Cases of severe bradycardia and heart block have been observed when sofosbuvir-containing regimens are used in combination with amiodarone and/or other medicinal products that lower heart rate. Steven-Johnson syndrome with unknown frequency. **Drug interactions:** Patients treated with vitamin K antagonists: As liver function may change during treatment with Epclusa, a close monitoring of International Normalised Ratio (INR) values is recommended. Impact of DAA therapy on drugs metabolized by the liver: The pharmacokinetics of drugs that are metabolized by the liver (e.g. immunosuppressive agents such as calcineurin inhibitors) may be impacted by changes in liver function during DAA therapy, related to clearance of HCV. Interactions between Epclusa and other medicinal products: Acid reducing agents including antacids (aluminium, magnesium hydroxide, calcium carbonate), H2-receptor antagonists (famotidine, cimetidine, nizatidine, ranitidine), proton pump inhibitors (omeprazole, lansoprazole, rabeprazole, pantoprazole, esomeprazole); Antiarrhythmics such as amiodarone, digoxin; Anticoagulants such as dabigatran etexilate and Vitamin K antagonists; Anticonvulsants such as carbamazepine, phenytoin, phenobarbital and oxcarbazepine; Antimycobacterials such as rifampicin, rifabutin and rifapentine; HIV antiviral agents: reverse transcriptase inhibitors such as tenofovir disoproxil fumarate, efavirenz/ emtricitabine/ tenofovir disoproxil fumarate; Herbal supplements such as St. John's wort; HMG-CoA reductase inhibitors such as rosuvastatin, and other statins. Immunosuppressants such as ciclosporin and tacrolimus.
Before prescribing, please consult full prescribing information which is available upon request.
Epclusa is a registered trademark of Gilead Sciences, Inc., or its related companies. For medical enquiries, please send your request to asiamedinfo@gilead.com or call 800 908 348 (toll-free number).

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400 mg/100 mg tablets

HKG-HPC-00018_V1.1 01/12/2023

Seroquel XR®
quetiapine

ONE FOR ALL

Major Mental Disorders

APPROVED INDICATIONS

- Bipolar Depression - 50-600mg/day*
- Major Depressive Disorder (MDD) - 50-300mg/day*
- Schizophrenia - 400-800mg/day*
- Bipolar Mania - 400-800mg/day*
- Generalised Anxiety Disorder (GAD) - 50-150mg/day*

* 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 immediately 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 an effective 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 an effective 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, heart failure, 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 spiration 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.
Please read the complete prescribing information before prescribing.
This material is for Healthcare Professionals academic reference only, not for general public or for advertising purposes. Any decision on treatment made by Healthcare Professionals shall be based on the personal situation of patients and shall refer to the product information approved by local State Drug Administration.
27-11-2024-HK-SRQ-0013

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Spravato® (esketamine) nasal spray 速開朗®

A Breakthrough Therapy

for your patients with treatment-resistant depression (TRD), or who are experiencing a psychiatric emergency (PE) due to major depressive disorder (MDD)[1]

1st The first approved antidepressant in 30 years targeting the glutamate system[2,3]

Rapid efficacy: Offer rapid relief of depressive symptoms at 24 hours in TRD patients, and 4 hours in MDD-PE patients[4,5]

70% reduced risk of relapse in TRD patients who were stable responders at Week 16[6]

The chance to reclaim lives: Greater rates of remission at all time points in MDD-PE patients vs placebo[5]

Further information is available upon request.

MDD, Major Depressive Disorder; PE, Psychiatric Emergency; TRD, Treatment-Resistant Depression.

References: 1. SPRAVATO® Hong Kong Prescribing Information P02. 2. Hillhouse TM, et al. A brief history of the development of antidepressant drugs: From monoamines to glutamate. Exp Clin Psychopharmacol. 23(1): 1 -21. 2015. 3. Li YF. A hypothesis of monoamine (5-HT) - Glutamate/GABA long neural circuit: Aiming for fast-onset antidepressant discovery. Pharmacol Ther. 208:107494. 2020. 4. Popova V, et al. Efficacy and Safety of Flexibly Dosed Esketamine Nasal Spray Combined With a Newly Initiated Oral Antidepressant in Treatment-Resistant Depression: A Randomized Double-Blind Active-Controlled Study Am J Psychiatry. 2019;176(6):428-438. 5. Fu DJ, et al. Esketamine Nasal Spray for Rapid Reduction of Major Depressive Disorder Symptoms in Patients Who Have Active Suicidal Ideation With Intent: Double-Blind, Randomized Study (ASPIRE I). J Clin Psychiatry.12;81(3):19m13191. 2020 May. 6. Daly EJ, et al. Efficacy of Esketamine Nasal Spray Plus Oral Antidepressant Treatment for Relapse Prevention in Patients With Treatment-Resistant Depression. A Randomized Clinical Trial. JAMA Psychiatry. 1;76(9):893-903. 2019.

SPRAVATO®
ABBREVIATED PRESCRIBING INFORMATION
ACTIVE INGREDIENT(S): esketamine (as hydrochloride) INDICATION(S): Spravato, in combination with a SSRI or SNRI, is indicated for adults with treatment-resistant Major Depressive Disorder, who have not responded to at least two different treatments with antidepressants in the current moderate to severe depressive episode. Spravato, co-administered with oral antidepressant therapy, is indicated in adults with a moderate to severe episode of Major Depressive Disorder, as acute short-term treatment, for the rapid reduction of depressive symptoms, which according to clinical judgement constitute a psychiatric emergency. DOSAGE & ADMINISTRATION: The decision to prescribe Spravato should be determined by a psychiatrist. Spravato is intended to be self-administered by the patient under the direct supervision of a healthcare professional. Assessment before treatment - Prior to dosing with Spravato blood pressure should be assessed. Post-administration observation - After dosing with Spravato, blood pressure should be reassessed at approximately 40 minutes and subsequently as clinically warranted. Recommended dosing for Spravato in adults ≤65 years with treatment-resistant Major Depressive Disorder - Induction phase, Weeks 1-4: Starting day 1 dose: 56 mg; Subsequent doses: 56 mg or 84 mg twice a week); Maintenance phase, Weeks 5-8: 56 mg or 84 mg once weekly; From Week 9: 56 mg or 84 mg every 2 weeks or once weekly. Recommended dosing for Spravato in adults ≥65 years with treatment-resistant Major Depressive Disorder - Induction phase, Weeks 1-4: Starting day 1 dose: 28 mg; Subsequent doses: 28 mg, 56 mg or 84 mg twice a week* ; Maintenance phase, Weeks 5-8: 28 mg, 56 mg or 84 mg once weekly* ; From Week 9: 28 mg, 56 mg or 84 mg every 2 weeks or once weekly* (*All dose changes should be in 28 mg increments.) Evidence of therapeutic benefit should be evaluated at the end of induction phase to determine need for continued treatment. The need for continued treatment should be reexamined periodically. After depressive symptoms improve, treatment is recommended for at least 6 months. Acute short-term treatment of psychiatric emergency due to Major Depressive Disorder - Recommended dosage of Spravato for adult patients (<65 years) is 84 mg twice per week for 4 weeks. Dosage reduction to 56 mg should be made based on tolerability. After 4 weeks of treatment with Spravato, the oral antidepressant (AD) therapy should be continued, per clinical judgement. Patients should be advised not to eat for at least 2 hours before administration and not to drink liquids at least 30 minutes prior to administration. Patients who require a nasal corticosteroid or nasal decongestant on a dosing day should be advised not to administer these medicinal products within 1 hour before Spravato administration. Patients who have missed treatment session(s) during the first 4 weeks of treatment should continue with their current dosing schedule. For patients with treatment-resistant Major Depressive Disorder who miss treatment session(s) during maintenance phase and have worsening of depression symptoms, per clinical judgement, consider returning to the previous dosing schedule. Efficacy of Spravato in Japanese patients has been studied, but not established. Method of administration - For nasal use only. Do not prime before use. CONTRAINDICATIONS: Hypersensitivity to the active substance, ketamine, or to any of the excipients listed in the full prescribing information. Patients for whom an increase in blood pressure or intracranial pressure poses a serious risk: Patients with aneurysmal vascular disease (including intracranial, thoracic, or abdominal aorta, or peripheral arterial vessels); Patients with history of intracerebral haemorrhage; Recent (within 6 weeks) cardiovascular event, including myocardial infarction (MI). SPECIAL WARNINGS & PRECAUTIONS: Suicide/suicidal thoughts or clinical worsening - Effectiveness of Spravato in preventing suicide or in reducing suicidal ideation or behaviour has not been demonstrated. Use of Spravato does not preclude the need for hospitalisation if clinically warranted, even if patients experience improvement after an initial dose of Spravato. Close supervision of patients especially in early treatment and following dose changes. Patients and caregivers should be alerted to 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. Patients with a history of suicide-related events or those exhibiting a significant degree of suicidal ideation prior to commencement of treatment are known to be at greater risk of suicidal thoughts or suicide attempts and should receive careful monitoring during treatment. Neuropsychiatric and motor impairments - Spravato has been reported to cause somnalence, sedation, dissociative symptoms, perception disturbances, dizziness, vertigo and anxiety during the clinical trials. At each treatment session, patients should be monitored under the supervision of a healthcare professional to assess when the patient is considered stable based on clinical judgement. Respiratory depression - Respiratory depression may occur at high doses following rapid intravenous injection of esketamine or ketamine when used for anaesthesia. Close monitoring is required for sedation and respiratory depression. Effect on blood pressure - Spravato can cause transient increases in systolic and/or diastolic blood pressure which peak at approximately 40 minutes after administration of the medicinal product and last approximately 1-2 hours. A substantial increase in blood pressure could occur after any treatment session. Spravato is contraindicated in patients for whom an increase in blood pressure or intracranial pressure poses a serious risk. Before prescribing Spravato, patients with other cardiovascular and cerebrovascular conditions should be carefully assessed to determine whether the potential benefits of Spravato outweigh its risks. In patients whose blood pressure prior to dose administration is judged to be elevated, it is appropriate to adjust lifestyle and/or pharmacologic therapies to reduce blood pressure before starting treatment with Spravato. If blood pressure is elevated prior to Spravato administration a decision to delay Spravato therapy should take into account the balance of benefit and risk in individual patients. Blood pressure should be monitored after dose administration. Blood pressure should be measured around 40 minutes post-dose and subsequently as clinically warranted until values decline. If blood pressure remains elevated for a prolonged period of time, assistance should promptly be sought from practitioners experienced in blood pressure management. Patients who experience symptoms of a hypertensive crisis should be referred immediately for emergency care. Patients with clinically significant or unstable cardiovascular or respiratory conditions - Only initiate treatment with Spravato in patients with clinically significant or unstable cardiovascular or respiratory conditions if the benefit outweighs the risk. Spravato should be administered in a setting where appropriate resuscitation equipment and healthcare professionals with training in cardiopulmonary resuscitation are available. Refer to the full prescribing information for examples of conditions. Drug abuse, dependence, withdrawal - Individuals with a history of drug abuse or dependence may be at greater risk for abuse and misuse of Spravato. Prior to prescribing Spravato, each patient's risk for abuse or misuse should be assessed and patients receiving esketamine should be monitored for the development of behaviours or conditions of abuse or misuse, including drug seeking behaviour, while on therapy. Dependence and tolerance have been reported with prolonged use of ketamine. In individuals who were dependent on ketamine, withdrawal symptoms of cravings, anxiety, shaking, sweating and palpitations have been reported upon discontinuing ketamine. Ketamine, the racemic mixture of arketamine and esketamine, is a medicinal product that has been reported to be abused. The potential for abuse, misuse and diversion of Spravato is minimised due to the administration taking place under the direct supervision of a healthcare professional. Spravato contains esketamine and may be subject to abuse and diversion. Other populations at risk - Use with caution in patients with the following conditions. These patients should be carefully assessed before prescribing Spravato and treatment initiated only if the benefit outweighs the risk: (i) Presence or history of psychosis; (ii) Presence or history of manic or bipolar disorder; (iii) Hyperthyroidism that has not been sufficiently treated; (iv) History of brain injury, hypertensive encephalopathy, intrathecal therapy with ventricular shunts, or any other condition associated with increased intracranial pressure. Elderly (65 years of age and older) - May have a greater risk of falling once mobilised, therefore, these patients should be carefully monitored. Severe hepatic impairment - Due to expected increase in exposure and lack of clinical experience, Spravato is not recommended in patients with Child-Pugh class C (severe) hepatic impairment. Hepatotoxicity has been reported with chronic ketamine use, so the potential for such an effect due to long-term use of Spravato cannot be excluded. Urinary tract symptoms - Urinary tract and bladder symptoms have been reported with Spravato use. Recommended to monitor for urinary tract and bladder symptoms during the course of treatment and refer to an appropriate healthcare provider when symptoms persist. SIDE EFFECTS: The most commonly observed adverse reactions in treatment resistant depression patients treated with Spravato were dizziness, nausea, dissociation, headache, somnolence, vertigo, dysgeusia, hypoaesthesia, and vomiting. Refer to the full prescribing information for other side effects. PREGNANCY & LACTATION: Spravato is not recommended during pregnancy and in women of childbearing potential not using contraception. There are no or limited data on the use of esketamine in pregnant women. If a woman becomes pregnant while being treated with Spravato, treatment should be discontinued, and the patient should be counselled about the potential risk to the foetus and clinical/therapeutic options as soon as possible. It is unknown whether esketamine is excreted in human milk. A decision must be made whether to discontinue breast-feeding or to discontinue/abstain from Spravato therapy taking into account the benefit of breast feeding for the child and the benefit of therapy for the woman. INTERACTIONS: Concomitant use of Spravato with CNS depressants (e.g., benzodiazepines, opioids, alcohol) may increase sedation, which therefore should be closely monitored. Blood pressure should be closely monitored when Spravato is used concomitantly with psychostimulants (e.g., amphetamines, methylphenidate, modafanil, armodafinil) or other medicinal products that may increase blood pressure (e.g. xanthine derivatives, ergometrine, thyroid hormones, vasopressin, or MAOIs, such as, tranylcypromine, selegiline, phenelzine). PLEASE REFER TO FULL PRESCRIBING INFORMATION BEFORE PRESCRIBING. Spravato aPI ver.2.0.

Janssen, a division of Johnson & Johnson (HK) Ltd
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CP-334386 Feb 2023 4iC_2459

Janssen PHARMACEUTICAL COMPANIES OF Johnson&Johnson

REXULTI® brexpiprazole tablets

KEEP MOVING FORWARD

Re-engage Life for MDD Patients in 6 Weeks[1]

77% Response Rate[2*]

53% Remission Rate[2*]

REXULTI® improved MDD patients life engagement on below IDS-SR$_{10}$ subscale domain:[1^,3]

Physical (increase energy level)

Cognition (concentration/ decision making)

Emotion (capacity for pleasure)

Social (interpersonal sensitivity)

REXULTI® is indicated as an adjunctive therapy to ADT in adults with MDD.[4]

Start with REXULTI® 0.5 or 1 mg once daily and titrate up to the recommended target dosage of 2 mg once daily.[4]

Abbreviations: ADT: Antidepressant treatment; IDS-SR$_{10}$: Inventory of Depressive Symptomatology Self-Report (10 items); MDD: Major depressive disorder; MADRS: Montgomery-Asberg Depression Rating Scale.
References: 1. McIntyre RS, Therrien F, Ismail Z, et al. J Psychiatr Res. 2023 Jun;162:71-78. 2. Fava M, Okame T, Matsushima Y, et al. Int J Neuropsychopharmacol. 2017 Jan 1;20(1):22-30. 3. Thase ME, Ismail Z, Meehan SR, et al. J Psychiatr Res. 2023 May;161:132-139. 4. REXULTI® Hong Kong Package Insert. (Revised Sep 2021).
* Refers to 76.5% and 52.9% MADRS response and remission rates respectively. MADRS response is defined as a ≥ 50% reduction in MADRS total score from baseline. MADRS remission is defined as ≥ 50% reduction in MADRS total score from baseline and MADRS total score ≤ 10.[2]
^ This study investigated the effects of REXULTI® (2 - 3mg/day) adjunct to ADT on patient life engagement over the short and long term, using the 10-item Inventory of Depressive Symptomatology Self-Report (IDS-SR$_{10}$) Life Engagement subscale. Over 6 weeks, ADT + REXULTI® (n = 579) showed greater improvement in IDS-SR$_{10}$ Life Engagement subscale score than ADT + placebo (n = 583), with a least squares mean difference of -1.19 (95% confidence limits: -1.78, -0.59; p = 0.0001; Cohen's d effect size: 0.23). The 10 items are categorized into the following domains of IDS-SR$_{10}$ subscale; physical (feeling slowed down and energy level); cognitive (concentration/ decision making); emotion (view of my future, view of myself, response to the mood to good or desired events and capacity for pleasure or enjoyment); social (general interest, interpersonal sensitivity and interest in sex). These are some of the examples of each domain of IDS-SR$_{10}$ improvement in short-term efficacy and the improvement in particular domains (concentration/ decision making and feeling slowed down) is not statistically significant.[1,3]

Abbreviated Prescribing Information
REXULTI (Brexpiprazole) 0.25mg/0.5mg/1mg/2mg/3mg/4mg tablets. **INDICATION:** Schizophrenia in adults. Adjunctive Treatment of Major Depressive Disorder (MDD) in adults with an inadequate response to prior antidepressant treatments during the current episode. **DOSAGE:** Schizophrenia - starting dose for brexpiprazole is 1 mg once daily on days 1 to 4. Target dose range 2 mg to 4 mg once daily, Based on the patient's clinical response and tolerability, dose can be titrated to 2 mg once daily on day 5 through day 7 and then to 4 mg on day 8. Maximum daily dose 4 mg. MDD - starting dose for brexpiprazole is 0.5 or 1 mg once daily. Titrate to 1 mg once daily, then up to the recommended target dosage of 2 mg once daily. Dosage increases should occur at weekly intervals based on the patient's clinical response and tolerability. Maximum daily dose 2 mg. Refer to Package Insert for switching from and to brexpiprazole. For moderate to severe renal and hepatic impairment, and moderate, severe or end-stage renal impairment, maximum daily dose reduced to 3 mg for patients with schizophrenia and 1.25 mg for patients with MDD. Reduce dose in patients who are CYP2D6 poor metabolizers and for concomitant use with CYP2D6/CYP3A4 inhibitors. Adjust dose for concomitant use with CYP3A4 inducers. **CONTRAINDICATION:** Hypersensitivity to the drug and its excipients. Patients with dementia. **WARNINGS AND PRECAUTIONS:** Close supervision of high-risk patients for occurrence of suicidal behavior. Disruption of the body's ability to reduce core body temperature. Exercise complete fall risk assessments when initiating treatment, and recurrently for patients on long-term therapy. Orthostatic hypotension and syncope. QT prolongation. Dependence/Tolerance. Use with caution in patients with history of extrapyramidal symptoms. Dose reduction or cease treatment at signs and symptoms of tardive dyskinesia. Monitor hyperglycaemia, weight gain and dyslipidaemia. Seizures. Dysphagia. Higher risk of *impulse control disorders in patients with prior history.* Elevated prolactin levels. Rare cases of priapism. Leukopenia, neutropenia and agranulocytosis. Venous thromboembolism. Drug Reaction with Eosinophilia and Systemic Symptoms. Cease treatment upon signs and symptoms indicative of neuroleptic malignant syndrome or unexplained high fever. Not to be used in patients with hereditary galactose intolerance, total lactase deficiency or glucosegalactose malabsorption due to lactose contained. Dose reduction in moderate to severe hepatic impairment, moderate to end-stage renal impairment, CYP2D6 poor metabolizers. **Pregnancy and lactation:** not recommended in pregnancy. Nursing women should avoid breastfeeding. **ADVERSE REACTIONS:** Rash, weight increase, akathisia, dizziness, tremor, sedation, diarrhea, nausea, abdominal pain upper, back pain, pain in extremity, increased blood prolactin and creatine phosphokinase. **DRUG INTERACTIONS:** Predominantly metabolized by CYP3A4 and CYP2D6. Refer to Package Insert for dose adjustment for concomitant use with CYP inhibitors and inducers. **Please see full Prescribing information for details.** (Ref: HKPI Revised Sep 2021; Last Update: Oct 2022)

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