The 9th Annual Scientific Meeting
Hong Kong Society of Biological Psychiatry

Brain and the Environment

- 21-22 November 2015 (Saturday & Sunday)
- Cordis, Hong Kong at Langham Place
  555 Shanghai Street, Mongkok, Kowloon, Hong Kong

www.hksbp.org

Programme Book

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Welcome Message

On behalf of the Organizing Committee, I take great pleasure in inviting you to join the 9th Annual Scientific Meeting (ASM) of the Hong Kong Society of Biological Psychiatry (HKSBP). The meeting will be organized on **21st (Saturday afternoon)** and **22nd (Sunday afternoon) in November 2015** at the **Cordis, Hong Kong at Langham Place (formerly Langham Place, Mong Kok).**

The thematic of this year is **Brain and the Environment. Prof. James L Kennedy** will deliver a plenary lecture on epigenetics, covering how genes could be modified by environmental factors and a second talk on practical clinical implications in psychiatry. **Dr. Zhang Jihui** will lecture on sleep effect on gene expression. **Prof. Kelvin Chan** will enlighten us on natural products with effects on emotions and thought processes. There will 5 free papers on state of art in biological psychiatry presented by our distinguished researchers. **Dr. Lo Chun Wai** will share with us his views on changing psychiatric diagnosis in time. **Dr. Michael MC Wong** will talk on childhood adversity and subsequent mental and health problems.

Last but not the least, I would like to invite you to stay with us in the Lundbeck dinner symposium where **Mr. KK Cheung** and **Prof. Tang Siu Wa** will lecture on psychiatric drug development. I look forward to meeting you at the meeting and I am sure it will be an inspirational education event.

Dr. WONG Chi Keung
Chairperson, Organizing Committee
The 9th ASM, Hong Kong Society of Biological Psychiatry

9th ASM: Organizing Committee

Chairperson:
Dr. WONG Chi Keung

Members:
Dr. LO Chun Wai
Dr. TAM Mo Shing, Paul
Dr. TSANG Suk Kwan, Jenny
Dr. WONG Chung Hin, Willy
Dr. YUEN Cheung Hang, Henry

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Dr. CHUNG Kar Kin, Albert

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Dr. WONG Ming Cheuk, Michael

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Dr. YUEN Cheung Hang, Henry

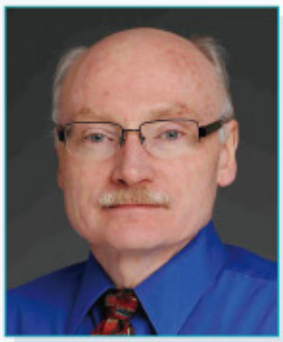
Floor Plan

Star Room, Level 42,
Cordis, Hong Kong at Langham Place,
555 Shanghai Street, Mongkok,
Kowloon, Hong Kong

1. Lundbeck
2. Eli Lilly
3. Janssen
4. Otuska
5. Sanofi-aventis
6. Servier

Scientific Programme

Saturday, 21 November 2015 |Star Room, Level 42, Cordis, Hong Kong (formerly Langham Place, Hong Kong)	
13:30 - 13:45	Registration
13:45 - 14:00	Welcome Remarks by ***Prof. TANG Siu Wa,*** President of HKSBP
14:00 - 15:00	**Epigenetics and DNA Sequence Combine to Mediate Environmental Stressors** ***Professor James L. KENNEDY,*** Professor and Co-Director of Division of Brain and Therapeutics, Department of Psychiatry, University of Toronto, Canada *Chairperson: Dr. WONG Ming Cheuk, Michael, Vice-President of HKSBP*
15:00 - 15:30	Exhibition and Coffee Break
15:30 - 16:30	**Genetics and Epigenetics of Insomnia** ***Dr. ZHANG Jihui,*** Assistant Professor, Department of Psychiatry, The Chinese University of Hong Kong *Chairperson: Dr. LO Chun Wai, Council Member of HKSBP*
16:30 - 17:30	**Natural Products with Effects on Mental and Physical Health** ***Professor Kelvin CHAN,*** Adjunct Professor in TCM Research, University of Western Sydney , University of Technology Sydney, Australia *Chairperson: Dr. TAM Mo Shing, Paul, Council Member of HKSBP*
17:30 - 18:30	**Free Paper Presentation** *Chairperson: Dr. WONG Ming Cheuk, Michael, Vice-President of HKSBP*
	Investigating Pathological Mechanisms of Depression as Risk Factors for Alzheimer's Disease ***Dr. CHANG Chuen Chung,*** Raymond, Associate Professor, School of Biomedical Sciences, The University of Hong Kong
	Exercise Improves Stress Coping Through a Dopamine D2 Receptor Pathway in the Medial Prefrontal Cortex ***Mr. CHEN Chong,*** PhD Candidate, Department of Psychiatry, Hokkaido University Graduate School of Medicine, Japan
18:30 - 19:00	Cocktail Reception / HKSBP's AGM (Members only)
19:00 - 22:00	**Dinner Symposium (Sponsored by Lundbeck Hong Kong)** *Chairperson: Dr. LI Seung Yau, Derek, Specialist in Psychiatry, Hong Kong*
19:00 - 19:30	**Drug Development: Some Interesting Statistical Data** ***Mr. CHEUNG Kin Kwong,*** Medical Director, Education & Training Manager, Lundbeck Hong Kong
19:30 - 20:15	**Revolution in Psychiatric Drug Development : Neuroscience Based and Target Specific Compounds** ***Prof. TANG Siu Wa,*** President of HKSBP
20:15 - 20:30	Q&A
20:30 - 22:00	Conference Dinner
Sunday, 22 November 2015 | Star Room, Level 42, Cordis, Hong Kong at Langham Place	
12:45 - 13:00	Registration
13:00 - 14:00	**Working Lunch: Free Paper Presentation** *Chairperson: Dr. CHUNG Kar Kin, Albert, Council Member of HKSBP*
	Pathogenic Role of Monoamine Oxidase-A in the Induction of depressive-like Behavior in Rats Exposed to Chronic Intermittent Hypoxia ***Mr. LAM Chun Sing,*** Thomas, PhD Candidate, School of Biomedical Sciences, The University of Hong Kong
	Neural Connectivity of Alexithymia: Specific Association with Major Depressive Disorder ***Ms. Nerissa HO,*** Research Associate, Department of Psychology, The University of Hong Kong
	A Revolutionary New Model to Study Psychiatric Disorders : Induced Pluripotent Stem Cells ***Dr. WONG Tai Wai,*** David, Executive Secretary, The Institute of Brain Medicine
14:00 - 15:00	**Pharmacogenetic Testing in Psychiatry: Science and Clinical Application** ***Professor James L. KENNEDY,*** Professor and Co-Director of Division of Brain and Therapeutics, Department of Psychiatry, University of Toronto, Canada *Chairperson: Dr. TSANG Suk Kwan, Jenny, Honorary Treasurer of HKSBP*
15:00 - 15:30	**Changing Diagnosis in Psychiatry, Is It Epigenetics?** ***Dr. LO Chun Wai,*** Specialist in Psychiatry, Hong Kong; Council Member of HKSBP *Chairperson: Prof. TANG Siu Wa, President of HKSBP*
15:30 - 15:55	**Childhood Adversity and Mental Health Problems: The Role of Epigenetics** ***Dr. WONG Ming Cheuk, Michael,*** Consultant and Chief of Service, Department of Psychiatry, Queen Mary Hospital, Hong Kong; Vice-President of HKSBP *Chairperson: Prof. TANG Siu Wa, President of HKSBP*
15:55 - 16:00	*Closing Remarks by **Prof. TANG Siu Wa**, President of HKSBP*



Professor James L. KENNEDY

Director, Molecular Brain Science, CAMH
Head of Neurogenetics Section, CAMH
Head, Tanenbaum Centre for Pharmacogenetics
Co-Director, Division of Brain and Therapeutics,
Dept. of Psychiatry, and
Professor, Department of Psychiatry and Institute of Medical Science, University of Toronto
Fellow, Royal Society of Canada

Dr. Kennedy has training in three major areas: neuroscience, clinical psychiatry, and molecular genetics. His main research interest over the past 20 years has been the identification of susceptibility genes for psychiatric disorders. His discoveries include: 1) the role of the DRD4 gene in ADHD, 2) the DRD3 gene predicting risk for Tardive Dyskinesia, 3) the 5HTTLPR genetic marker predicts risk for antidepressant induced mania, and several genes that predict antipsychotic response. Major current projects led by Dr. Kennedy include the IMPACT study examining a large sample (N=20,000) of patients to discover new pharmacogenetic markers, and determine the usefulness and cost-benefit of genetic testing applied to choice and dosage of psychiatric medications in clinical care. Furthermore, he is leading a large randomized controlled clinical trial of pharmacogenomic tests in 1,200 depression and schizophrenia patients, funded by Genome Canada. He has published pioneering findings relating gene variants in the dopamine, serotonin, and neurodevelopment systems to psychiatric disorders, neuroimaging (PET and MRI) and to treatment response. Dr. Kennedy has published more than 600 scientific articles with over 20,000 citations, and he is an active lecturer at numerous international conferences.

Lecture
21 November 2015

Brain Epigenetics and DNA Sequence Combine to Mediate Environmental Stressors

Professor James L. KENNEDY

Epigenetic modifications of DNA and chromatin structure are becoming progressively more important in our understanding of psychiatric diseases. There are two main epigenetic mechanisms, namely histone modification and DNA methylation. This lecture will focus on the latter mechanism, and will first examine the groundbreaking work of Michael Meaney's lab in showing that the cortisol receptor gene in rat pups has different DNA methylation patterns depending on the amount and quality of nursing and licking behaviour of the mother. Last year, Kaminsky et al (2014) reported that the spindle and kinetocore associated complex subunit 2 (SKA2) gene had a single nucleotide polymorphism (rs7208505) in the coding region for a C to T variation that exhibited variable degrees of methylation, and appeared to alter SKA2 function. SKA2 modulates the HPA axis by partnering with the cortisol receptor and assisting the receptor's transport and storage. Furthermore, this combination of DNA SNP and methylation predicted risk for suicidal behaviour in two human group samples, as well as predicting risk of suicide completion in the post mortem tissue of completers versus controls. My laboratory has examined this SKA2 site for the C/T sequence variant, and for the degree of methylation of the C nucleotide, in salivary lymphocytes from antidepressant responders vs non-responders. The results are negative for treatment response prediction thus far. We also examined the same DNA site and methylation variation in OCD patients, and found a significant correlation between methylation at the rs7208505 site and severity of YBOCS scores ($p < 0.001$). The methylation of the serotonin transporter gene will also be examined in terms of its role in anxiety disorders. In addition, we have performed genome-wide methylome analysis of 450,000 sites (Illumina 450k chip) in 600 patients at baseline who are followed for antidepressant treatment response. This large pilot study may yield epigenetic markers that predict treatment response. Overall, a number of promising findings are emerging in the field of epigenetics and psychiatry.

Dr. ZHANG Jihui

Assistant Professor, Department of Psychiatry,
The Chinese University of Hong Kong

Dr. Jihui Zhang is currently an assistant professor at the department of psychiatry, the Chinese university of Hong Kong. His research interests include pathogenesis of insomnia, the long-term outcomes of insomnia disorder, and REM sleep behavior disorder as a neurodegenerative disease. He received his bachelor degree in clinical medicine and master degree in psychiatry from Sun Yat-sen University in 2004 and 2007, respectively. In his PhD study between 2007 and 2010 at the Department of Psychiatry, the Chinese University of Hong Kong, he mainly focused on the clinical research in insomnia disorder. He was also trained in genetic epidemiology of mental disorders at the National Institute of Mental Health, National Institutes of Health. After training at the NIH, he moved back to Hong Kong for continuing his research journey. In the past 3 years, he received several major grants from Hong Kong and China funding agencies and also received a Young Investigator Award in the World Congress on Sleep Medicine in 2015.

Lecture

21 November 2015

Genetics and Epigenetics of Insomnia

Dr. ZHANG Jihui

Insomnia disorder is one of the most prevalent health problems in the general population. In line with other studies conducted in western countries, our previous prospective studies revealed that chronic insomnia, once established, runs a persistent course with several risk factors contributing to the maintenance of chronic insomnia. In past two decades, more and more studies have shown that insomnia disorder is not only contrubited by environmental factors, but also contributed by genetic factors. A number of twin studies and family studies have confirmed that the heritability of insomnia range from 30-60%. Our study has further shown that the measurement tools for insomnia significantly affect heritability estimation of insomnia phenotype, which suggests a necessity of refined measurement tool. Several candidate genes have been found to be associated with insomnia, such as CLOCK gene, 5-HTTLPR, and adenosine. Nonetheless, there are several significant limitations in these studies, including small sample size, poor phenotype documentation, and a lack of replication. Recently, some researchers also suggest that there are epigenetic mechanisms underlying the pathogenesis of insomnia. In this presentation, Dr. Jihui Zhang will discuss current evidence of genetics and epigenetics of insomnia and its limitations and challenges.

Professor Kelvin CHAN

Adjunct Professor, TCM Research, University of Western Sydney and University of Technology Sydney, Australia
Visiting Professor, Natural Products & TCM Research, Liverpool John Moores University, United Kingdom

Professor Kelvin Chan (DSc PhD FRPS FRSM), trained in Clinical Pharmacology in UK and formerly Joint Chair in Traditional Chinese Medicine (TCM) at The University of Sydney and Western Sydney University, currently holds the following academic positions: Adjunct Professor in TCM Research at Western Sydney University and University of Technology Sydney, Australia and Visiting Professor in Natural Products & TCM Research at Liverpool John Moores University, United Kingdom. He has taught Medical Pharmacology in orthodox medicine and TCM and related Pharmacy disciplines in the UK, UAE, Hong Kong and Australia. He has expertise and experience in R&D of GACP, GLP, GMP and GCTP in TCM with over 350 conference presentations, 256 CSI papers, 3 textbooks on Chinese medicine. Currently he focuses on quality standardisation and mechanism of Chinese materia medica for TCM practice and integration of TCM with biomedical monitoring and patients' reported outcomes in clinical studies.

Website: https://www.researchgate.net/profile/Kelvin_Chan4/?ev=hdr_xprf
Email: profkchan@gmail.com

Lecture
21 November 2015

Natural Products with Effects on Mental and Physical Health

Professor Kelvin CHAN

The human genome project, started 14years ago [1], has provided new knowledge of diseases and health and technology in cell-biology for screening bioactivities of natural products and Chinese materia medica (CMM) in drug discovery. It has changed the development of pharmacology (and psychopharmacology) and medicine. Pharmacology needs to consider tackling diseases not in the conventional 'drug-receptor' concept, but in a more 'holistic-response' approaches similar to Traditional Chinese medicine (TCM) practice. Drugs may not be designed to be as exact to a particular ligand or specific to a particular gene or protein subtype, several drugs may provide broad-acting regulatory mechanisms, which may regulate large groups of genes and proteins. Such approaches are also used to investigate TCM nowadays [2]. TCM products, usually in composite formulae (FuFang), and other natural products have been used as add-on medications in treatment of schizophrenia [3]. The survey of patients who were treated with neuroleptics in a West-Midland Health Trust (UK) prompted us to carry out a meta-analysis of add-on use of a popular herbal preparation, gingko extract in capsule {EGb} in schizophrenia [4]. EGb extract has statistically significant moderate therapeutic benefit as an add-on therapy for treatment of total and negative symptoms of chronic schizophrenia. However, precaution on the quality and safety [5] of natural products and TCM when co-administered [6] with pharmaceutical drugs is needed. This presentation summarizes our systematic approaches to investigate the chemical characteristics and cardiovascular/ neuro-protective effects of CMM, which may be useful for mental and physical health.

References: **1.**Nature (2001) 409: 860–921; **2.** Science (2015); 347 (6219 Suppl), S1—S25; **3.** Brit J Psychiat (2007), 190: 379-384; **4.** Intl J Neuropsychopharmacol (2010), 13, 257–271. **5.** J Ethnopharmacol (2005) 96, 1–18; **6.** Chin J Integr Med (2015): DOI: 10.1111/bcp.12598.

Free Paper

21 November 2015

Investigating Pathological Mechanisms of Depression as Risk Factors for Alzheimer's Disease

Dr. CHANG Chuen Chung, Raymond

Raymond Chuen-Chung Chang[1,2,3], Ginger Tsz-Hin Wong[1,4], Andrea Wing-Ting Tsang[1], Suthicha Wuwongse[1,4], Sally Shuk-Yee Cheng[1], Clara Hiu-Ling Hung[1,6], Yuen-Shan Ho[5], Andrew Chi-Kin Law[2,3,4]

[1]Laboratory of Neurodegenerative Diseases, Department of Anatomy; [2]Research Centre of Heart, Brain, Hormone and Healthy Aging, LKS Faculty of Medicine; [3]State Key Laboratory of Brain and Cognitive Sciences; [4]Neurodysfunction Laboratory, Department of Psychiatry, LKS Faculty of Medicine, The University of Hong Kong, [5]School of Nursing, Faculty of Health and Social Sciences, The Hong Kong Polytechnic University; [6]Institute of Chinese Medical Sciences, University of Macau

While therapeutic intervention of Alzheimer's disease (AD) is important, we should pay more attention to prevent elderly suffering from this memory-robbing disease. It has been predicted that an increase of AD pathological factors may start 30 years before clinical symptoms of cognitive impairment. Therefore, it is important to prevent dementia and AD. Epidemiological studies have revealed that depression is a risk factor leading to the development of AD. The aim of our study is to elucidate the biological mechanisms of how depression leads to cognitive impairment and even to AD.

We have used corticosterone as a model agent for depression and oligomeric β-amyloid (Aβ) peptide as a toxic agent for AD. Immunoreactivity for α-tubulin, tau and phospho-tau (pS396, pS404) was examined in primary cultures of hippocampal neurons prepared from embryonic day 18 rats. Phosphorylation of tau protein was examined by both Western-blot and immunofluorescent staining. Morphological changes of cytoskeleton were examined using immunocytochemical analysis or live cell imaging technique with confocal microscopy by transfecting a mCherry-actin/GFP-tubulin into neurons. Both corticosterone and Aβ peptide induce aggregation and subsequent loss of synaptic proteins, aggregation of tubulin and phosphorylation of tau protein. Altered post-translational modification and aggregation of microtubules were found in neurons after treatment with exposure to Aβ peptide or corticosterone. Aggregation of actin forming actin rods was also observed. All these events were reversed by taxol – a microtubule-stabilizing agent. Therefore, depression may prime neurons to be susceptible to Aβ toxicity. This explains why depression can be a risk factor for developing cognitive impairment in AD.

Our research has helped explain why some factors reported by epidemiological studies can be risk factors for AD. As AD-like pathology starts to develop 30 years before any cognitive impairment, advancement of our knowledge of the mechanisms of risk factors can help the scientific community develop ways to prevent progression to AD.

Acknowledgement:
The study is supported by Seed Funding for Basic Research (201411159021).

Exercise Improves Stress Coping Through a Dopamine D2 Receptor Pathway in the Medial Prefrontal Cortex

Mr. CHEN Chong

C Chen, S Nakagawa, Y Kitaichi, Y An, Y Omiya, N Song, M Koga, T Inoue, I Kusumi
Department of Psychiatry, Hokkaido University Graduate School of Medicine, Japan

Introduction:
Although exercise has been well-known for its beneficial effects on cognition and stress coping, the underlying neurobiological mechanism remains largely unknown. The purpose of the study is to investigate by what neurotransmitter(s) exercise improves stress coping within the medial prefrontal cortex (mPFC), the brain center for behavioral control.

Methods and Results:
Experiment 1: Exercise rats were raised with three weeks of free access to wheels while control rats were raised in same cages without wheels. The forced swim test (FST) was employed to assess stress coping. Exercise rats showed reduced immobility in the FST, suggesting stress coping effect.

Experiment 2: A second group of rats were raised in the same way and subjected to the FST under microdialysis. Several major neurotransmitters within the mPFC were sampled before and after FST. Exercise rats showed higher level of dopamine in the mPFC both at the basal level and after FST. There was no group difference regarding serotonin, noradrenaline, glutamate, glutamine, glycine, taurine, or alanine.

Experiment 3: A third group of rats were raised in the same way and underwent FST. 30 minutes before FST, exercise rats were locally injected dopamine D1 or D2 receptor antagonist (SCH-23390 and haloperidol, both at a concentration that does not affect performance in FST of normal rats) into mPFC, while control rats were locally injected only the vector. D2 but not D1 receptor antagonist completely abolished the stress coping effect of exercise.
Conclusion: Exercise improves stress coping by upregulating dopamine in the mPFC and in a D2 receptor-dependent way.

Acknowledgement:
This research is supported by Hokkaido University Clark Memorial Foundation.

Mr. CHEUNG Kin Kwong

Medical Director, Education and Training Manager,
Lundbeck Hong Kong

Mr. Cheung Kin Kwong completed his BSc Hons. degree and his MSc degree in the United Kingdom, and was awarded a scholarship by the UK's Atomic Energy Authority to pursue his master's degree there.

His professional titles include notably Chartered Chemist of Royal Society of Chemistry (UK), Chartered Scientist of the Science Council (UK) and Fellow of Institute of Management Specialists (UK).

He served in the HKSAR health service as a dispenser/pharmaceutical technician for 8 years, and was Head of Pharmacy for 12 years with Drs Anderson & Partners (a 20-clinic private chain). He also served as product manager and business unit manager for 11 years with Zuellig Pharma (HK). He is currently Medical Director as well as Education & Training Manager of Lundbeck Hong Kong. Lastly, he made inroads in academia too, undertaking the position of lecturer in Pharmacology in both Hong Kong and the UK for almost a decade.

Drug Development: Some Interesting Statistical Data

Mr. CHEUNG Kin Kwong

The process of developing a new drug is long, painstaking, very demanding, and requires a comprehensive collection of scientific resources. It is considered as a highly risky and incredibly expensive business venture which does not necessarily guarantee investment return.

Overall, the past decades has seen many pharmaceutical companies enjoying huge revenue from their drug blockbusters but also experiencing failure in launching many of their other pipeline drugs, some of which were withdrawn for both efficacy and safety reasons.

It is interesting to note that a few drugs only attracted limited medical attention regarding their initially approved indications, whereas their unexpected "side" effects led to clues that paved the way for new indications that ultimately produced blockbuster drugs.

Lately, many pharma companies are confronted with escalating R&D costs, increased drug development times attributable to both the change of regulatory standards and the fallen success rate in tackling complicated diseases related to Alzheimer's, arthritis and oncology, for example. Facing the challenges associated with fewer drug blockbusters emerging, the decline in profitability due to patent expiration, and also tough generic competition, many pharmaceutical companies have diversified their business strategies, aiming notably at biotechnological applications in personalized medicine, as well as therapeutics in specialty diseases.

A much expected transformation that is set to alter the pharmaceutical development propensity and interests irrevocably is the increased emphasis on preventive rather than curative medicine.

Professor TANG Siu Wa

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

Professor Tang is former Head of Psychopharmacology Department at the Clarke Institute, University of Toronto, former Professor and Chairman of Psychiatry, University of California, Irvine, USA and former Chair professor in Psychiatry, University of Hong Kong, China. He is now Emeritus Professor of Psychiatry, University of California, Irvine, USA and honorary professor of Anatomy, University of Hong Kong, China. He practices psychiatry and consults in psychopharmacology.

Professor Tang founded the Hong Kong Society of Biological Psychiatry (HKSBP), and with Dr. Brian Leonard and Professor Zohar founded the Institute of Brain Medicine (www.ibrainmedicine.org) for the promotion of education in brain related medicine and psychopharmacology.

Professor Tang's doctoral research in brain chemistry received both the Canadian Ontario provincial and Canadian national research prize in the late seventies. He is also the recipient of the 2010 Kraepelin-Alzheimer Medal (Munich, Germany) for his scientific and educational contribution to international psychopharmacology and the recipient of the 2011 World Federation of Societies of Biological Psychiatry Excellence in Education Award (Prague, Czech Republic).

Revolution in Psychiatric Drug Development: Neuroscience Based and Target Specific Compounds

Professor TANG Siu Wa

Discovery and development of drugs for the treatment of anxiety, mood disorders, psychosis and dementia have been mostly serendipitous in the last century. However, with the rapid advances in neuroscience research, we are coming to a new era where known pathways and targets in the brain could be manipulated with greater precision. This has resulted in a major revolution in psychiatric drug development, in that new target specific compounds are beginning to be added to the therapeutic repertoire for psychiatry. With this has come greater insight into human brain function in health and disease. We are now on the edge of a revolution in psychiatric drug design and development.

Free Paper

22 November 2015

Pathogenic Role of Monoamine Oxidase-A in the Induction of Depressive-like Behavior in Rats Exposed to Chronic Intermittent Hypoxia

Mr. LAM Chun Sing, Thomas

Chun-Sing Lam[1], George L. Tipoe[1,2], Man-Lung Fung[1,2]

[1]School of Biomedical Sciences; [3]Research Centre of Heart, Brain, Hormone and Healthy Aging, Li Ka Shing Faculty of Medicine, The University of Hong Kong

Increased risk of depression incidence has been reported in patients with severe obstructive sleep apnea (OSA) [1], but the direct link underlying the co-morbidity remains elusive. Chronic intermittent hypoxia (CIH), the hallmark features of OSA patients, causes hippocampal dysfunction by inducing oxidative stress, neuroinflammation and apoptosis [2]. Elevated monoamine oxidase-A(MAO-A) activities is observed in depressed patients resulting in the deficiency of monoamines including serotonin and overwhelming production of catalytic by-products hydrogen peroxides which could promote the progression of depression [3].M30 is a brain selective MAO inhibitor with anti-oxidative and iron-chelating properties conferring neuroprotective effect in different animal disease models [4-5].

We aim to investigate the role of MAO-A in CIH-triggered depressive-like behavior in rats. It is hypothesized that CIH induces depressive-like behavior by an increased MAO-A activity leading to monoamine deficiency, oxidative stress, neuroinflammation, apoptosis and synaptic degeneration in the hippocampus, which could be ameliorated by M30. CIH-treated rats displayed significant increased immobility time and reduced sucrose consumption in the forced-swimming test and sucrose-preference test respectively when compared with the normoxic control. Remarkable elevated levels of the protein expression and activity of MAO-A, and increased level of serotonin deaminated product 5-hydroxyindoleacetic acid were observed in the hippocampus of hypoxic rats. Increased lipid peroxidation extent accompanied by reduced protein expressions of antioxidant enzymes (SOD-2, GPx-1) were seen in the hypoxic group. Additionally, inflammatory cytokines (TNF-α, IL-1β and IL-6) and apoptotic markers (cleaved caspase-3, cleaved PARP-1) were significantly increased in the hypoxic group. Moreover, the anti-apoptotic Bcl-2 expression and synaptic vesicle proteins (synapsin-1 and synaptophysin) were significantly decreased in the hypoxic group. Furthermore, the protein expression of indoleamine 2,3, dioxygenase, a catabolic enzyme of tryptophan and serotonin, was significantly increased in the hypoxic group. Daily administration of M30 (5mg/kg i.p.) prior to the hypoxic treatment (AHI= 60) markedly normalized the depressive-like behavior and adverse changes in the hippocampus of the hypoxic group.

Monoamine oxidase-A is a pivotal candidate in the onset of depressive-like behavior induced by chronic intermittent hypoxia in rats, leading to serotonin depletion, oxidative stress, neuroinflammation, apoptosis and synaptic degeneration. Hence, MAO-A serves a potential target to prevent depression incidence in OSA patients.

Reference:

1. El-Sherbini et al., (2011) *Neuropsychiatric Disease Treatment7:* 715-721; **2.** Lam et al., (2015) *PLoS One*10(2): e0117990; **3.** Aluf et al., (2012) *Neuropharmacology*65:48-57; **4.** Youdim et al., (2013) *Experimental Neurobiololgy*(1): 1–10; **5.** Kupershmidtet al., (2012) *Antioxidants & Redox Signaling*17(6): 860-877

Neural Connectivity of Alexithymia: Specific Association with Major Depressive Disorder

Ms. Nerissa HO

Nerissa S.P. Ho[1,2], Michael M.C. Wong[3], Tatia M.C. Lee[1,2,4,5]

[1] Laboratory of Neuropsychology; [2] Laboratory of Cognitive Affective Neuroscience, The University of Hong Kong; [3] Department of Psychiatry, Queen Mary Hospital, Hong Kong; [4] Institute of Clinical Neuropsychology, The University of Hong Kong; [5] The State Key Laboratory of Brain and Cognitive Sciences, The University of Hong Kong

Alexithymia has been frequently associated with major depression disorders (MDD). Yet little is known about the exact relationship of Alexithymia and MDD. In order to explore this subject matter, the neural connectivity associated with alexithymia in people with MDD and matched nonclinical controls were compared.

Twenty-two females diagnosed with first-episode MDD and twenty-one matched nonclinical controls were MRI brain-scanned with diffusion-tensor-imaging and resting-state-functional-imaging methods, and self-reported the Chinese 20-item Toronto Alexithymia Scale.

Voxel-wise multiple regression analysis showed a group interaction effect regarding the correlation between white-matter-connectivity and alexithymia. Significant correlations were observed at the corpus-callosum in MDDs and at the right superior-longitudinal-fasciculus in the controls. These findings were then used to derive seeds for analyzing resting-state-functional-connectivity in each group separately. The results further revealed that alexithymia in MDDs were associated withreduced functional-connectivity in the right precentral-gyrus and several regions of the brain on the right, which are associated with cognitive regulation in the default-mode-network. In contrast, among the control subjects, it was correlated with increased functional-connectivity between the right inferior-frontal-gyrus-triangularis and the right superior-occipital-lobe, which is associated with emotional response to external stimuli.

These findings supported our a priori hypothesis that MDDs and controls have distinct white-matter correlates of alexithymia, and this corresponded to the neural correlates regarding cognitive and affective alexithymia respectively, as proposed previously. Extended impacts of these microstructural changes on remote functional networks might help explain the distinct behavioral characteristics of alexithymia for these groups, as well as implications for therapeutic intervention of MDD.

Free Paper

22 November 2015

A Revolutionary New Model to Study Psychiatric Disorders : Induced Pluripotent Stem Cells

Dr. WONG Tai Wai, David

The ability of embryonic stem cells to renew itself and proceed along an innate programme of differentiation to form tissues and organs had long fascinated scientists and the medical field. Ethical issues and inconvenience had limited its usage.

The study of Yamanaka to reverse the well differentiated state of somatic cells to primitive pluripotency sparked off great excitement, creativity and imagination. Since then many scientists had replicated the study and applied the technique to produce limitless iPSC and differentiated cells like neurones harbouring patient and disease specific genetic materials.

Studies at the cellular level can readily be carried out in genomic editing of the iPSC, genomic, biochemical and electrophysiological analysis of differentiated cells . It is hoped that mysteries underlying incurable diseases like schizophrenia, ALS, epilepsy etc can be deciphered.

Drug trials utilizing cellular responses and drugs specific to patients maybe designed.

It is hoped that, like spare parts of cars, human organs may in the not distant future, be produced and applied in repair and replacement.

Lecture
22 November 2015

Pharmacogenetic Testing in Psychiatry: Science and Clinical Application

Professor James L. KENNEDY

In genomics the amount of information available is increasing rapidly. At the same time our knowledge of inter-individual differences in terms of response and side effects to medications is at an early stage. For example, an important dilemma facing psychiatrists when they need to select an antipsychotic medication for their patient is the forced choice between risk for weight gain and diabetes with the newer generation drugs versus the risk of tardive dyskinesia and other motor side effects with first generation antipsychotics. We have developed a model of seven genes (melanocortin-4 receptor, serotonin 2C, neuropeptide Y, others) that predicts 67% of the variance in risk for this weight gain (Tiwari et al, 2015). In terms of antidepressant treatment there are now several replicated studies showing a significant benefit of gene guided medication selection over treatment as usual (Altar et al, 2015). Genes tested include CYP450, 5HT2A, and 5HTTLPR. In addition to significant clinical improvement and reduction of side effects there is also a documented reduction in health care costs when genetic guidance is used in antidepressant treatment. Regarding the concern that physicians will not be able to efficiently translate complex genetic information into clinical decision-making, we have surveyed over 200 psychiatrists and family practitioners in our Toronto-based pharmacogenetics study (www.IM-PACT.ca; n=4,200 patients tested) and found that the overwhelming majority of physicians found our user-friendly genetic report to be readily understandable, and over 90% believe that pharmacogenetics testing will become standard of care in the future.

Lecture
22 November 2015

Dr. LO Chun Wai

Specialist in Psychiatry, Hong Kong
Council Member, Hong Kong Society of Biological Psychiatry

Dr. Lo Chun Wai is a specialist in Psychiatry and has worked in the field of psychiatry for over forty years. He is now in private practice.

Changing Diagnosis in Psychiatry, Is it Epigenetics?

Clinical Diagnosis of Psychiatric Disorder can sometimes change over the years, as the disease progresses and the number of relapses increases. This Phenomenon is explored through the understanding of Neuroplasticity and Epigenetics.

Lecture
22 November 2015

WONG Ming Cheuk, Michael

Consultant and Chief of Service, Department of Psychiatry,
Queen Mary Hospital, Hong Kong
Vice-President, Hong Kong Society of Biological Psychiatry

Dr. Wong is the Consultant Psychiatrist and Chief of Service of 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 which has helped many patients to 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 is also actively participating in the regional activities of professional societies. He is the Chairman of the Society for Advancement of Bipolar Affective Disorder, Vice-President of the Hong Kong Society of Biological Psychiatry and member of the Task Force on Developing World of the World Federation of Societies of Biological Psychiatry.

Childhood Adversity and Mental Health Problems: The Role of Epigentics

Family is usually a place where people feel secure and get support. However at times it can be a stress and can have undesirable influence on the mental health of individuals. It is also an important factor in the development of mental illness. Genetic factor is an important aetiological factor in the development of mental illness, but the upbringing, parenting and family environment certainly play important role as well. Frustration, rejection, self-blaming, shameful feeling and over-protection are common among family members of mental patients. It is clear that there are periods during postnatal development in which the developing brain has heightened sensitivity to environmental influences. These sensitive periods represent a period of rapid brain development, where the early environment is able to shape neural circuits and thus determine structural and functional aspects of brain and behavior for the lifespan. This paper will try to hightlight the possible role of epigenetic in the aetiology of mental health problems in people who have experienced childhood adversity.

Notes to Delegates

Meeting Organizer
Hong Kong Society of Biological Psychiatry

Meeting Secretariat
c/o Kays Asia (Hong Kong) Ltd.
Tel: +852 5642 7378
Fax: +852 3010 8969
E-mail: enquiry@hksbp.org

Meeting Date
21-22 November 2015, Saturday and Sunday

Meeting Venue
Star Room, Level 42, Cordis, Hong Kong at Langham Place,
555 Shanghai Street, Mongkok, Kowloon, Hong Kong

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

Identification Badge
All participants will receive badges and programme book upon check-in at 13:30 on 21 November and 12:30 on 22 November. The registration counter is located at lift lobby of meeting venue. 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 entrance of Star Room, Level 42.

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 14:00 – 18:30 on 21 November 2015 and 13:00 – 16:00 on 22 November 2015. The dinner symposium on 21 November is exclusively sponsored by Lundbeck.

On-site Registration
The registration counter is located at the lift lobby of meeting venue, Star Room, Level 42. For on-site registration, payment must be made in cash in HK dollars.

Registration Fees

HKSBP Members	**Free of charge**
Non-HKSBP Members	**HKD450**
Students*	**HKD50**

**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.*

Meal Arrangement
Tea break, lunch and dinner will be served at Star Room, Level 42.

Insurance
The organizing committee of the 9th 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
Cordis, Hong Kong is a smoke-free premise. No indoor smoking is allowed.

Acknowledgements

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 9th ASM.

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1. Emsley R, Medori R, Koen L et al. Long-acting injectable Risperidone in the treatment of subjects with recent-onset psychosis 2. Invega® Sustenna® Package Insert (version Aug, 2012) 3. Risperdal® Consta® Package Insert (version Jan, 2012) 4. Wu DBC, Lee EHM, Chung WS et al. Cost analysis of risperidone long-acting injection in the treatment of schizophrenia and schizoaffective disorders in Hong Kong : An approach using generalized estimating equations 5. Gopal S, Berwaerts J, Nuamah I et al. Number needed to treat and number needed to harm with paliperidone palmitate relative to long-acting haloperidol, bromperidol, and fluphenazine decanoate for treatment of patients with schizophrenia

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Presentation: Milnacipran oral capsules. **Indications:** Treatment of major (i.e. characterised) depressive episodes in adults. **Dosage:** 50mg twice daily. Dosage should be reduced to 25mg twice daily in patients with creatinine clearance between 30ml/min and 60ml/min. Dosage should further be reduced to 25mg daily in patients with creatinine clearance below 30ml/min. **Contraindications:** Hypersensitivity to milnacipran or excipients. Concomitant use with non-selective MAO inhibitors, B-selective MAO inhibitors, digitalis, 5-HT1 receptor agonists (triptans), parenteral epinephrine or norephinephrine, clonidine, A-selective MAO inhibitors. Uncontrolled hypertension. Severe or unstable coronary heart disease. Lactation. **Precautions:** Patients with a history of suicide-related events. Children below 18 years of age. Renal failure. Patients with an obstruction to emptying the bladder. Hypertension or cardiac disease. Angle-closure glaucoma. Epilepsy. Patients taking diuretics or any other therapy known to cause hyponatraemia, and in cirrhosis or undernourished patients. Patients receiving concomitant treatment with oral anticoagulants, medicines acting on the platelet function such as NSAIDs and aspirin, or other medicinal products likely to increase the risk of bleeding. **Interactions:** Linezolid, alcohol, non-selective MAO inhibitors, MAO-A and MAO-B inhibitors, 5HT1D agonists, digitalis, adrenaline, noradrenaline, clonidine, lithium. **Undesirable effects:** Agitation, anxiety, depression, eating disorder, sleep disorder, suicidal behavior, headache, migraine, tremor, dizziness, dysaesthesia, somnolence, tachycardia, palpitations, hot flushes, hypertension, nausea, constipation, diarrhoea, abdominal pain, dyspepsia, vomiting, dry mouth, pruritis, rash, hyperhidrosis, musculoskeletal pain, dysuria, pollakiuria, ejaculation disorder, erectile dysfunction, testicular pain, fatigue. For uncommon, rare and very rare undesirable effects, please refer to the full prescribing information. **Preparation:** 25mg x 28's; 50mg x 28's. ***Full prescribing information is available upon request.***

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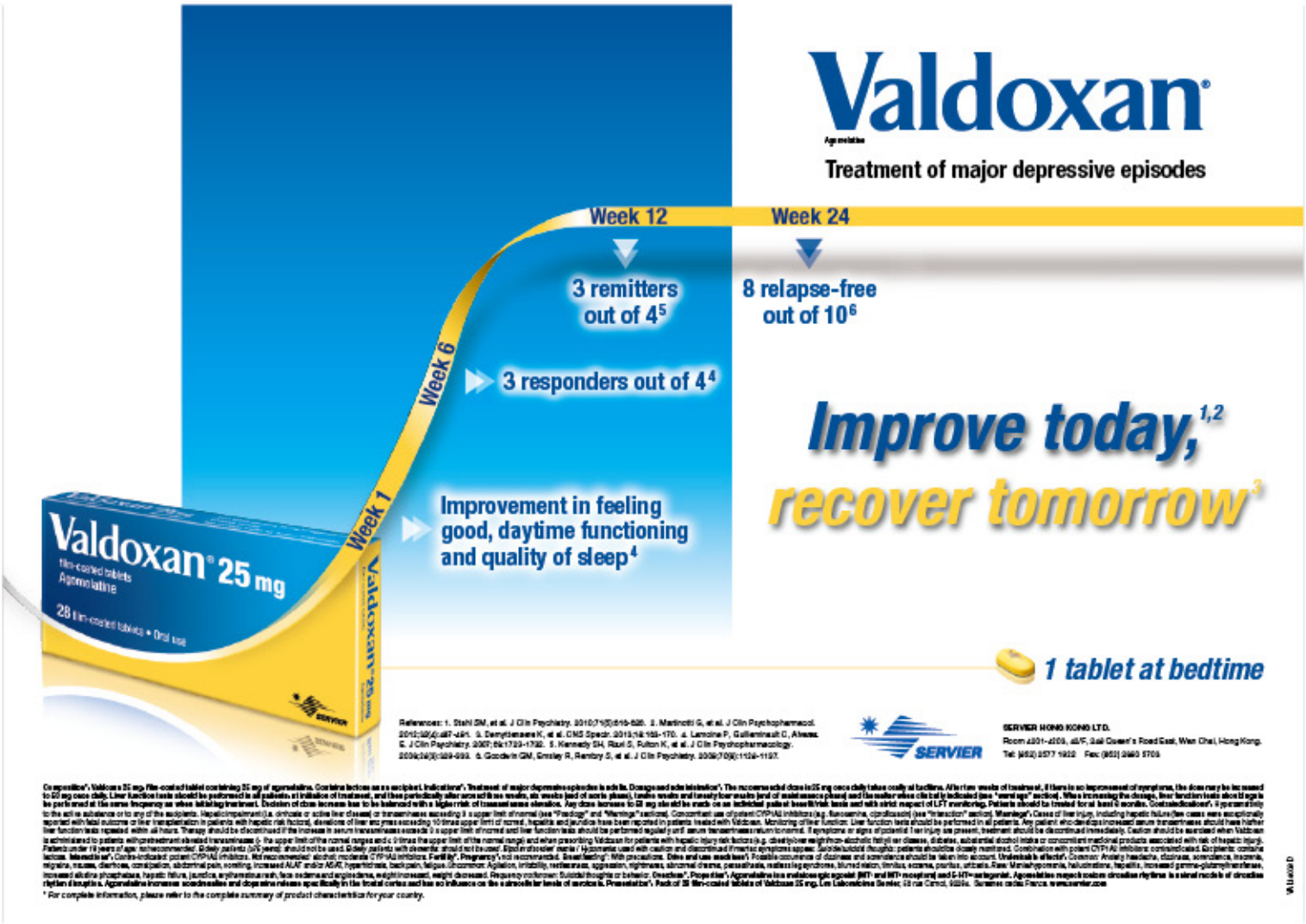

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Co-prescribe*: Valdoxan 25 mg film-coated tablet containing 25 mg of agomelatine. Contains lactose as an excipient. Indications*: Treatment of major depressive episodes in adults. Dosage and administration*: The recommended dose is 25 mg once daily taken orally at bedtime. After two weeks of treatment, if there is no improvement of symptoms, the dose may be increased to 50 mg once daily. Liver function tests should be performed in all patients at initiation of treatment, and then periodically after six and then weeks, six weeks (end of acute phase), twelve weeks and twenty four weeks (end of maintenance phase) and the earlier when clinically indicated (see "dose adjustment" section). When increasing the dosage, liver function tests should be performed at the same frequency as when initiating treatment. Decision of dose increase has to be balanced with a higher risk of treatment-emergent hepatic and with strict respect of LFT monitoring. Patients should be treated for at least 6 months. Contraindications*: Hypersensitivity to the active substance or to any of the excipients. Hepatic impairment (i.e. cirrhosis or active liver disease) or transaminases exceeding 3 x upper limit of normal (see "Posology" and "Warnings" sections). Concomitant use of potent CYP1A2 inhibitors (e.g. fluvoxamine, ciprofloxacin) (see "Interaction" section). Warnings*: Cases of liver injury, including hepatic failure (few cases were exceptionally reported with fatal outcome or liver transplantation in patients with hepatic risk factors), elevations of liver enzymes exceeding 10 times upper limit of normal, hepatitis and jaundice have been reported in patients treated with Valdoxan. Monitoring of liver function: Liver function tests should be performed in all patients. Any patient who develops increased serum transaminases should have further liver function tests repeated within 48 hours. Therapy should be discontinued if the increase in serum transaminases exceeds 3 x upper limit of normal and liver function tests should be performed regularly until serum transaminases return to normal. If symptoms or signs of potential liver injury are present, treatment should be discontinued immediately. Caution should be exercised when Valdoxan is administered to patients with pre-treatment elevated transaminases (> 1x the upper limit of the normal ranges and < 3 times the upper limit of the normal ranges) and when prescribing Valdoxan for patients with hepatic injury risk factors (e.g. obesity/overweight/non-alcoholic fatty liver disease, diabetes, substantial alcohol intake or concomitant medicinal products associated with risk of hepatic injury). Patient under 18 years of age: not recommended. Elderly patients (≥65 years): should not be used. Elderly patients with dementia: should not be used. Bipolar disorder/ mania / Hypomania: used with caution and discontinued if manic/ hypomanic symptoms appear. Suicide/suicidal thoughts: patients should be closely monitored. Combination with potent CYP1A2 inhibitors: contraindicated. Excipients: contains lactose. Interaction*: Contra-indicated: potent CYP1A2 inhibitors. Not recommended: alcohol, moderate CYP1A2 inhibitors. Fertility, Pregnancy and lactation*: Pregnancy: not recommended. Breast feeding*: With precautions. Driving and use machines*: Possible occurrence of dizziness and somnolence should be taken into account. Undesirable effects*: Common: Anxiety, headache, dizziness, somnolence, insomnia, migraine, nausea, diarrhoea, constipation, abdominal pain, vomiting, increased ALAT and/or ASAT, hyperhidrosis, back pain, fatigue. Uncommon: Agitation, irritability, restlessness, aggression, nightmares, abnormal dreams, paraesthesia, restless legs syndrome, blurred vision, tinnitus, eczema, pruritus, urticaria. Rare: Mental depersonalization, hallucinations, hepatitis, increased gamma-glutamyltransferase, increased alkaline phosphatase, hepatic failure, jaundice, erythema multiforme, face oedema and angioedema, weight increased, weight decreased. Frequency unknown: Suicidal thoughts or behavior. Overdose*: Properties*: Agomelatine is a melatonergic agonist (MT1 and MT2 receptors) and 5-HT2C antagonist. Agomelatine resynchronizes circadian rhythms in animal models of circadian rhythm disruption. Agomelatine increases noradrenaline and dopamine release specifically in the frontal cortex and has no influence on the extracellular levels of serotonin. Presentation*: Pack of 28 film-coated tablets of Valdoxan 25 mg. Les Laboratoires Servier, 50 rue Carnot, 92284 Suresnes cedex France. www.servier.com

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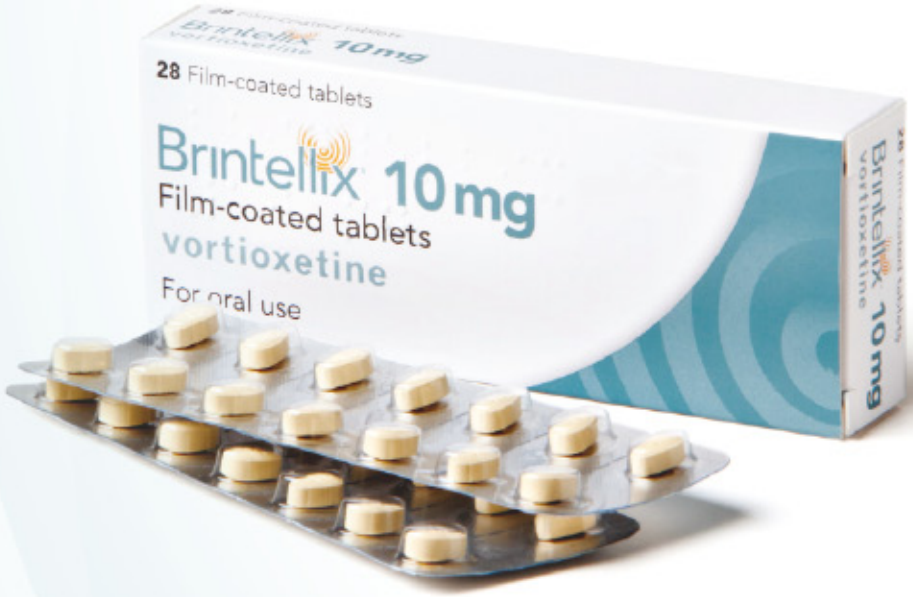

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