Speaker 3: Jun Soo Kwon, Republic of Korea
Title: Compulsive-Impulsive Disorders as Reflected in the Structural and Functional Neuroimaging Studies

Abstract
In contrast to the traditional viewpoint of psychopathology, which compulsivity [a repetitive ritualistic behavior which persists albeit its inappropriateness to the given situation and often result in undesirable consequences] and impulsivity [which encompasses actions that are insufficiently conceived, prematurely expressed, excessively risky or inappropriate to the situation, and that often lead to undesirable outcomes] were positioned at opposite ends of a behavioral characteristics, recent neuroimaging studies raise the possibility of compulsivity and impulsivity being orthogonal factors that each contribute in varying degrees to various psychiatric conditions, including obsessive-compulsive disorder (OCD). In this presentation, I firstly will discuss two differential facets of compulsivity-impulsivity using the clinical (compulsion sub-score of Y-BOCS) and neurocognitive (stop signal task) measures and examined their neural underpinning as reflected in the small-world network of structural (DTI) and resting state functional connectivity network in OCD subjects. Later on, this presentation will also discuss the relationship between OCD and substance-related and addictive disorders (as defined in the DSM-5) from the spectrum-wise perspective of compulsivity and impulsivity, also using the frame of structural and functional connectome, to widen our understanding for clinical and neurocognitive dimensions of compulsivity-impulsivity phenomena.

Speaker 4: Jon Grant, USA
Title: Treatment of Compulsive and Impulsive Disorders

Abstract
Impulsivity and compulsivity have been considered opposite poles of a continuous spectrum, but their relationship appears to be more complex. Disorders characterized by impulsivity often have features of compulsivity and vice versa. The overlaps of the constructs of compulsivity and impulsivity, as well as their differences, suggest different pharmacological targets may be warranted. This presentation will discuss novel pharmacological interventions for compulsive and impulsive behaviors. Many of the pharmacological options that reduce impulsive or compulsive behaviors in certain disorders may be useful for these cognitive domains seen in other disorders. For the purposes of this talk, I will focus on several classes of pharmacological agents and discuss to what extent they may be effective in reducing impulsivity and compulsivity. Of course, there are multiple forms of impulsivity and compulsivity, and different forms of each can co-occur within the same person. In some cases, there are data regarding which type of impulsivity or compulsivity the pharmacological agent is targeting. In those cases, this talk will discuss the subtype in more detail. Additionally, the presentation will discuss the implications of basic neuroscience for developing novel neuropsychopharmacological interventions, and the relevant clinical evidence-base.

References
Grant JE, Chamberlain SR. Impulsive action and impulsive choice across substance and behavioral addictions: cause or consequence? Addict Behav. 2014 Nov;39(11):1632–9

S5: New Advances in Precision Psychiatry

Chair: Gwyneth Zai, Canada
Co-Chair: Kazutaka Shimoda, Japan

Speaker 1: Dan Rujescu, Germany
Title: Pharmacogenetics of Antipsychotic Response and/or Induced Side Effects

Abstract
Patients with Schizophrenia respond differently to antipsychotic drug treatment. The underlying mechanism for these individuals’ differences remains unknown. However, there is growing evidence indicating that genetic background contributes substantially to efficacy of antipsychotic drug. In order to test the genetic mechanism of treatment response of antipsychotic drugs, we performed exome sequencing in a group of patients with very good response and matched patients without response. And reduction rates of PANSS and sub-scales were used to evaluate treatment responses. We found that a number of genes (ZFNS804A, FIWL4, TG and KALRN) involved in treatment response of antipsychotic drug. Pathway analysis showed that pathways involved in glutamate, including the pathways involved in CNS and abnormal neurotransmitter level, and the N-methyl-D-aspartate (NMDA) and α-Amino-3-hydroxy-5-methyl-4-isoxazolopropionic acid (AMPA) mediated synaptic pathways, were detected to implicate in treatment response of antipsychotic drugs. Especially, the treatment response for negative symptoms was more significantly associated with these two pathways.

Speaker 2: Tao Li, China
Title: Genetics of Response to Antipsychotic Medication in Chinese Patients

Abstract
Patients with Schizophrenia respond differently to antipsychotic drug treatment. The underlying mechanism for these individuals’ differences remains unknown. However, there is growing evidence indicating that genetic background contributes substantially to efficacy of antipsychotic drug. In order to test the genetic mechanism of treatment response of antipsychotic drugs, we performed exome sequencing in a group of patients with very good response and matched patients without response. And reduction rates of PANSS and sub-scales were used to evaluate treatment responses. We found that a number of genes (ZFNS804A, FIWL4, TG and KALRN) involved in treatment response of antipsychotic drug. Pathway analysis showed that pathways involved in glutamate, including the pathways involved in CNS and abnormal neurotransmitter level, and the N-methyl-D-aspartate (NMDA) and α-Amino-3-hydroxy-5-methyl-4-isoxazolopropionic acid (AMPA) mediated synaptic pathways, were detected to implicate in treatment response of antipsychotic drugs. Especially, the treatment response for negative symptoms was more significantly associated with these two pathways.

Speaker 3: Gwyneth Zai, Canada
Title: Pharmacogenetics of Antidepressant Treatment Response in Obsessive-Compulsive Disorder

Abstract
Gwyneth Zai1,2,3,4 Carolina Cappi1, Vanessa Gaoncalves1, Roseli Shavitit1, Euripedes Constantino Miguel1, Margaret A. Richter1,2,4, James L. Kennedy1,2
1Neurogenetics Section, Centre for Addiction and Mental Health, Toronto, Canada 2Department of Psychiatry and Institute of Medical Science, University of Toronto, Toronto, Canada 3Behavioural and Clinical Neuroscience Institute and Department of Psychiatry, University of Cambridge, Cambridge, UK 4Frederick W. Thompson Anxiety Disorders Centre, Department of Psychiatry, Sunnybrook Health Sciences Centre, Toronto, Canada 5Department of Psychiatry, University of Sao Paulo School of Medicine, Sao Paulo, Brazil

Background: Precision medicine utilizing genetic testing has recently received much attention given that the variability of response and tolerability to psychotropic medications are partly due to an individual’s genetic variations. This has led to increasing research to investigate the role of specific genetic factors on psychotropic medication response and utility of testing in the clinical realm (Zai et al., 2014, Pharmacogenomics, 15, 1147–1157). Antidepressant medications are the first-line pharmacological treatment for mood, anxiety, and obsessive-compulsive and related disorders. However, 20–50% of patients show poor or minimal response to these medications.