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ABSTRACTS

Brainstorming Session I-

SPREADING EVIDENCE-BASED INTERVENTIONS TO RESOURCE-CHALLENGED SETTINGS: BRAINSTORMING GLOBAL IMPLEMENTATION STRATEGIES

B-01 | Spreading evidence-based interventions to resource-challenged settings: brainstorming global implementation strategies

M.S. Bauer[1]; A.M. Kilbourne[2]

[1]*Harvard Medical School, Department of Veterans Affairs, Boston, USA,* [2]*Director,VA National Quality Enhancement Research Initiative (QUERI) & the University of Michigan, Ann Arbor, USA*

It is clear that building a strong evidence base does not guarantee the uptake of an intervention into clinical practice. In fact, evidence-based innovations take an average 17 years to be generally accepted, and over half of such interventions never make it into general usage. The situation is even more challenging in resource-constrained settings, since interventions developed in academic medical centers are not well adapted to spread outside of those relatively well resourced settings. This challenge is paramount for bipolar disorder, since many affected individuals are cared for in publicly funded clinics while, globally, care is often limited to non-mental health clinicians. This has been recognized recently, for instance, in the Global Alliance for Chronic Disease's commitment of tens of millions of dollars to fund trials to implement evidence-based mental health interventions in low and middle income countries. The field of implementation science focuses specifically on utilizing scientifically proven methods to move evidence-based practices into broad usage. Drs. Bauer and Kilbourne will briefly present the basics of implementation science, proposing that the challenge of implementation can be understood in terms of the characteristics of: the innovation itself, the recipient clinicians and treated individuals, the healthcare and policy context in which they work, and the implementation support needed. Drs. Bauer and Kilbourne will then lead a brainstorming session, engaging the session's global audience in discussion to identify factors that limit adoption of evidence-based practices for bipolar disorder in their specific contexts, and discussing implementation strategies that can address those limitations.

B-02 | Spreading evidence-based interventions to resource-challenged settings: brainstorming global implementation strategies

M.S. Bauer[1]; A.M. Kilbourne[2]

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Brainstorming Session II-

A PATIENT'S PERSPECTIVE: FROM TREATING AN ILLNESS TO IMPROVING HEALTH

B-03 | Overcoming bipolar disorder through self-management, technology, and behavioral health

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Can a patient functionally override and proactively mitigate their symptoms of bipolar disorder? Can technological and psychosocial processes of self-management focused on the improvement of health change the way patients and their families understand management of symptoms and recovery? Can the design of interfaces and our interactions with the internet be used to promote good mental health? Can we use machine learning, online platforms and behavioural datasets to engrain healthy social rhythms in a patient's lifestyle, and possibly alter the trajectory of an exacerbation? Bipolar Disorder has changed almost every aspect of my life since it's initial presentation. At the time of diagnosis, I was unable to keep up with my studies and left school. I am exploring these questions to help regain control of my thoughts, emotions, understandings and interactions with the world around me. By attempting to understand the illness and developing ways to self-manage behaviours, I am recovering, am employed as a software designer, and have returned to school. While I do not know if bipolar disorder can be truly cured, I believe that people with the bipolar disorder are capable of overcoming the symptoms and doing better than they were before the diagnosis.

B-04 | How do we know if treatment is optimal?

J. MacLean[1]

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What is bipolar disorder? At present there is no accepted definition that excludes symptomatology in the definition. There are many uncertainties including etiology of the disease process, and the area of injury. Present medical management of the bipolar disorders although helpful may not be optimal. Many authors propose an inflammatory component to the disease process. Patient behaviours which potentially decrease the inflammatory burden may contribute to the recovery process. An individual diagnosed with bipolar disorder should be be encouraged to continuously monitor and modify poor behaviour choices.

Brainstorming Session III-

INNOVATIVE MODELS TO EXPLORE SYSTEMIC ABNORMALITIES IN BIPOLAR DISORDER

B-05 | Targeting insulin pathways in mood and cognition in mood disorders

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[1]Mood Disorders Psychopharmacology Unit, Toronto Western Hospital, University Health Network, Toronto, Canada

High prevalence of metabolic syndrome has been consistently demonstrated in mood disorders. Insulin pathways dysfunction has been explored using new paradigms of research and a significant effect of this mechanism in mood and cognition has been explored. In this sense, a recent preliminary trial with liraglutide found an effect of this medication in cognition of individuals with mood disorders. In this presentation, the links between insulin and neurocircuits, molecular pathways and physiological mechanisms will be reviewed, exploring the hypothesis that pathological changes in insulin dysfunction are a significant contributor for clinical expression of mood disorders.

B-06 | The paradigm of premature aging in bipolar disorder

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Contemporary models for the pathophysiology of bipolar disorder (BD) posit that its causes and mechanisms go beyond the brain and consider that a set of peripheral metabolic, endocrine and immune

factors continuously influence neurotransmitters, brain circuits and structures, as well as determine the frequent presence of general medical comorbidities (GMC). Most contributors to GMC morbidity and mortality in BD are associated with metabolic abnormalities classically associated with the aging process, as age is one of the main isolated risk factor for their occurrence. Aging has been conceptualized as the accumulation of diverse changes occurring in cells and tissues, which are responsible for the increased risk of disease and death. The premature aging hypothesis is an innovative model to investigate pathophysiology of BD and to look for new promising targets for intervention. In this presentation, findings on premature aging of immune system, telomeres and epigenetic markers will be reviewed, offering new insights for clinical care and research.

Brainstorming Session IV-

CAN NONLINEARITY INFORM OUR CLINICAL PRACTICE?: TAKING A CLOSER LOOK AT MOOD VARIABILITY AND CLINICAL COURSE IN BIPOLAR DISORDER

B-07 | Mood variability: can it inform our clinical practice in bipolar disorder?

A. Ortiz[1]

[1]Department of Psychiatry, University of Ottawa, Ottawa, ON, Canada

Mood regulation is a complex and poorly understood process. It can be conceived as a system that allows flexible responses to changing conditions in the environment. When these processes are not properly working, the system tends to be more "rigid", with resulting depressive or manic episodes. In our current classification system, unfortunately, clinical outcome is often dichotomized, so that if a patient is not judged reaching criteria for an episode, the patient is considered to be "stable", in spite of considerable mood fluctuations, cognitive impairment or residual symptoms. Mood variability has too many nuances that are easily missed by dichotomizing mood into "stable" or "in an episode". In this talk, i would like to 1) Discuss the importance of understanding nonlinearity/complexity to inform our clinical practice; 2) describe our work on mood variability in healthy controls and bipolar disorders, as well as to 3) discuss new, unpublished data on mood regulation and variability in first-degree relatives of bipolar disorder patients. We consider that by studying mood variability from the nonlinear perspective in bipolar disorder, we can gather vast amounts of information on clinical course and treatment response. Moreover, nonlinear techniques can also be applied to inform our daily clinical practice, for instance, when it comes to complex cases distinguishing bipolar disorder from borderline personality disorder.

B-08 | Clinical course in bipolar disorder: an underrated key to pathogenesis

P. Grof[1]

[1]Psychiatry, University of Toronto, Toronto, ON, Canada

This talk will explore the roles of monotherapy and combinations in finding effective long-term treatment for individual patients with bipolar disorder. While current practice relies heavily on combinations, many bipolar patients can be successfully stabilized if the initial monotherapy is carefully selected according to the patient's clinical characteristics. The data show that: 1. unequivocal responders to long-term monotherapies such as lithium, lamotrigine or atypical neuroleptics each have a very different clinical profile including clinical presentation and course, comorbidity and, in particular, family history; 2. bipolar patients who respond very well to one long-term monotherapy have often completely failed on other monotherapies. In terms of clinical course, in particular, there are differences in the pre-treatment clinical course between responder groups. Lithium responders present with an episodic, fully remitting course and often have a predominance of depressive over manic episodes. On the other hand, lamotrigine and olanzapine responders tend to have mostly nonepisodic courses with residual symptoms and exacerbations. The differences in long-term clinical course are striking. For lithium responders in particular, the episodic fully remitting course is very characteristic. However, in current practice clinicians often treat before a pattern of recurrences is established, and frequently do not have enough time to evaluate the clinical course thoroughly enough to be able to use the findings in treatment decisions. We will share data spanning over 3 decades that show a clear pattern that can clearly inform our daily clinical practice.

Brainstorming Session V-

EPIDEMIOLOGICAL AND CLINICAL VARIABLES RELATED WITH THE PREDOMINANT POLARITY ON BIPOLAR DISORDER: CLINICAL IMPLICATIONS

B-09 | Predominant polarity on bipolar disorder: definition and prevalence

J. García-Jiménez[1]

[1]Psychiatry, Rehabilitation Center San Juan de Dios, Teruel, Spain

This session explain clearly the concept of polarity predominant (PP) in bipolar disorder patients. The speaker would explain how to define it and apply it to patients in clinical dairy practice. We give some data about studies that have calculated the prevalence of PP. We analysed the concept of polarity index in order to know how useful are the neuroleptics and mood stabilizers in function of the PP concept in BD.

B-10 | Clinical implications of predominant polarity on bipolar disorder

L. Gutiérrez-Rojas[1]

[1]Psychiatry, Campus de la Salud Hospital, Granada, Spain

In the second session we will explained with detail the clinical implications of PP. We would give to the audience some data about the relationship between PP and some variables as: sociodemographic (sex and age), age at onset of the illness, affective and psychotic symptoms, subtype of BD, comorbidity (especially suicide behaviour, anxiety and abuse disorders), clinical response to drugs, relapse and hospitalizations, personality features and functional outcome.

Brainstorming Session VI-

FAMILIAL TRANSMISSION OF MULTI-SYSTEM VULNERABILITIES IN BD

B-11 | Telomere shortening is associated with familial risk to bipolar disorder

S. Frangou[1]

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Variation in telomere length is heritable and is currently considered a promising biomarker of susceptibility for neuropsychiatric disorders, particularly because of its association with memory function and hippocampal morphology. In this study, we used quantitative polymerase chain reactions and a telomere-sequence to single-copy-gene-sequence ratio method to determine telomere length in genomic DNA extracted from buccal smears from 63 patients with bipolar disorder (BD), 74 first-degree relatives (49 relatives had no lifetime psychopathology and 25 had a non-BD mood disorder) and 80 unrelated healthy individuals. Participants also underwent magnetic resonance imaging to determine hippocampal volumes and cognitive assessment to evaluate episodic memory using the verbal paired associates test. Telomere length was shorter in psychiatrically-well relatives ($P = .007$) compared to unrelated healthy participants. Telomere length was also shorter in relatives (regardless of psychiatric status; $P < .01$) and patients with BD not on lithium ($P = .02$) compared to lithium-treated patients with BD. In the entire sample, telomere length was positively associated with left and right hippocampal volume and with delayed recall. This study provides evidence that shortened telomere length is associated with familial risk for BD. Lithium may have neuroprotective properties that require further investigation using prospective designs.

B-12 | Genetic liability of neurcognitive impairment in bipolar disorder: does the type of kinship matter?

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Introduction: Studies on heterogeneous groups (offspring, sibling, parent) of first degree relatives of patients reveal neurocognitive impairment as a genetic liability for bipolar disorder (BD). We aimed

to explore whether the pattern of neurocognitive impairment in offspring and siblings of BD differ.

Method: Demographic variables and neurocognitive performance of BD patients (n = 124), siblings (n = 419), offspring (n = 75), healthy controls (HC, n = 156) (mean age: 35.82 ± 10.761, 34.41 ± 9.55, 22.69 ± 5.083, 31.17 ± 11.032 years respectively) on a series of tests were compared using ANCOVA. Factor analysis was performed to understand the interaction between neurocognitive domains and kinship.

Results: Age and level of education differed significantly among the groups. Offspring were the youngest, siblings had the least years of education (11.32 ± 3.24). Patients performed significantly poorer than HC in all tests. The offspring and siblings were equally impaired in global cognition which explained 42.2% of the variance and correlated significantly to all tests. Factor analysis re-vealed six factors. Compared to HC, offspring performed significantly poorer in verbal memory, visual memory and verbal fluency whereas siblings were impaired in problem solving/reasoning even after controlling for age and education as covariates. Group differences disappeared for working memory and processing speed after controlling for age and education.

Conclusion: Our findings may indicate a cognitive impairment trajectory in genetically liable indi-viduals for BD supporting the developmental delay hypothesis. The memory and fluency impair-ment in younger age (offspring) may improve with ageing and executive function impairment may become evident at a later age as seen in siblings.