The International Stress and Behavior Society (ISBS)

Program and Abstracts

3rd Caribbean Biomedical Research Days CBRD-2016

Rodney Bay, St. Lucia
January 16-18, 2016

CONFERENCE PROGRAM:

DAY 1, Sat, January 16, 2016

Dolphins Conference Center, Bay Gardens Beach Resort & Spa, Rodney Bay, St. Lucia

09.00 – 10.00	**Registration**
10.00 – 10.20	**CONFERENCE OPENING REMARKS** Prof. AV Kalueff, ISBS President **BRIEF INTRODUCTION: HISTORY OF ISBS AND CBRD** **WELCOMING ADDRESSES**
10.20 – 11.20	**Opening Plenary Lecture: MODELING GENE-ENVIRONMENT INTERACTION: A DIMENSION PERSPECTIVE.** M Pletnikov, ISBS Fellow, Department of Psychiatry and Behavioral Sciences, Johns Hopkins University School of Medicine, Baltimore, MD, USA
11.20 – 12.35	**ISBS Special Lecture: NURTURING ETHICAL RESPONSIBLE CITIZENS IN THE AGE OF INDIVIDUALISM.** U Seraphin, ISBS Fellow Inductee, Saint Lucia Allied Health Practitioners Association (AHPA), St. Lucia, WI
12.35 - 01.30	**ISBS Presidential Lecture: TRANSLATIONAL RESEARCH OF NEURODEVELOPMENTAL DISORDERS - THE ISBS TASK FORCE REPORT.** AV Kalueff, ISBS Fellow, St. Petersburg State University, St. Petersburg, Ural Federal University, Ekaterinburg, Russia; ZENEREI Research Center, Slidell, LA, USA; Guangdong Ocean University, Institute of Marine Drugs and Nutrients, Zhanjiang, China
01.30 – 02.30	***Lunch Break (free time)***

02.30-02.55	**ESTIMATING WHOLE BRAIN CONNECTIVITY DYNAMICS FOR RESTING STATE fMRI DATA.** I Cribben, Y Yu, Department of Finance and Statistical Analysis, Alberta School of Business, Alberta, Canada; Statistical Laboratory, University of Cambridge, Cambridge, UK
02.55-03.20	**NEUROLOGICAL AND NEUROPSYCHOLOGICAL OUTCOMES AFTER SURGERY FOR MOYAMOYA DISEASE.** SK Gupta, Department of Neurosurgery, PGIMER, Chandigarh, India
03.20-03.55	**FLATTENED SHEET-LIKE FORNIX FORMING A 'COBRA HOOD' DEFORMITY: A PREVIOUSLY UNREPORTED VARIANT OF FORNIX ANATOMY AND ITS IMPLICATIONS FOR SURGICAL APPROACHES TO THE THIRD VENTRICLE.** T Gupta, Department of Anatomy, PGIMER, Chandigarh, India
03.55-04.20	**GENERAL DISCUSSION AND CONCLUDING REMARKS**
04.20-04.40	**BENEFITING FROM CANNABIS.** G St. Rose, ISBS Fellow, Eden Herbs, Creative Health Center, St. Lucia, WI
04.40 – 05.10	***Coffee Break***

DAY 2, Sun, January 17, 2016

Dolphins Conference Center, Bay Gardens Beach Resort & Spa, Rodney Bay, St. Lucia

9.30 – 10.00	**Registration**
10.00 – 10.50	**ISBS Plenary Lecture: NEUROIMMUNE FUNCTION IN DEPRESSION AND SUICIDE.** GN Pandey, Department of Psychiatry, University of Illinois at Chicago, Chicago, IL, USA
10.50 - 11.10	**GENETIC RISK FACTORS AND ADOLESCENT CANNABIS ABUSE: THE ROLE OF ASTROCYTES.** M Pletnikov, ISBS Fellow, Department of Psychiatry and Behavioral Sciences, Johns Hopkins University School of Medicine, Baltimore, MD, USA
11.10-12.10	**ISBS Plenary Lecture: CURRENT CONTROVERSIES IN PAIN RESEARCH**. WC Parris, Department of Anesthesiology, Duke University School of Medicine, Durham, NC, USA
12.10-12.30	**DISCUSSION**
12.30 – 01.40	***Lunch Break (free time)***

01.40 - 02.00	**UPDATE ON CANNABIS NEUROBIOLOGY.** M Fraites, St. Lucia, WI
02.00 - 03.00	**ISBS Special lecture: HOW HETERO-NORMATIVITY AND HOMO-NEGATIVITY IMPEDES LBGTQI PEOPLE FROM ACCESSING HARM REDUCTION SERVICES.** M Day, Caribbean Drug and Alcohol Research Institute (CDARI), St. Lucia, WI
03.00-03.20	**DISCUSSION**
03.20 – 03.40	***Coffee Break***
03.40 - 04.00	**DEPRESSIVE DISORDERS: NEW CLASSIFICATION AND MANAGEMENT.** G Swamy, St. Lucia, WI
04.00 - 04.15	**BOOK PRESENTATION. THE RIGHTS AND WRONGS OF ZEBRAFISH: PRINCIPLES OF BEHAVIORAL PHENOTYPING (2016)**
04.15 – 05.00	**ROUNDTABLE I: PERSPECTIVES AND CHALLENGES OF BIOMEDICINE AND BIOMEDICAL EDUCATION IN THE CARIBBEAN.** Moderators: AV Kalueff, USA, L Newman and M Day, St. Lucia

DAY 3, Mon, January 18, 2016

Dolphins Conference Center, Bay Gardens Beach Resort & Spa, Rodney Bay, St. Lucia

10.00 - 11.30 **INTERACTIVE GUIDED POSTER MINI-SYMPOSIUM**

IMPAIRED EFFICIENCY OF BASE EXCISION REPAIR SYSTEM IN NUCLEAR AND MITOCHONDRIAL EXTRACTS FROM PERIPHERAL BLOOD LYMPHOCYTES OF PATIENTS WITH ALZHEIMER'S DISEASE. T Sliwinski, D Kwiatkowski, P Czarny, M Toma, P Galecki, A Bachurska, J Szemraj, University of Lodz Department of Molecular Genetics, Medical University of Lodz, Department of Adult Psychiatry, Department of Medical Biochemistry, Lodz, Poland

ANTIDEPRESSANT-MEDIATED INCREASE IN THE EXPRESSION AND RELEASE OF GLIAL-DERIVED NEUROTROPHIC FACTOR LEADS TO INCREASED NEUROGENESIS. S Avissar, D Fux, M Golan, G Schreiber, Ben Gurion University, Beer Sheva, Barzilai Medical Centre, Ashkelon, Israel

IMMUNOGENETICS IN DEPRESSED PATIENTS WITH MAJOR DEPRESSIVE AND BIPOLAR DISORDERS. O Mikova, P Marinov, A Angelova, A Mihaylova, M Ivanova, Ts Lukanov, M Demirev, Foundation Biological Psychiatry, Sofia, Bulgaria; Alexandrovska University Hospital, Sofia, Bulgaria

PESPECTIVES ON ZEBRAFISH BEHAVIORAL PHENOMICS. AV Kalueff, ISBS Fellow, St. Petersburg State University, St. Petersburg, Ural Federal University, Ekaterinburg, Russia; ZENEREI Research Center, Slidell, LA, USA; Guangdong Ocean University, Institute of Marine Drugs and Nutrients, Zhanjiang, China

A NOVEL UNDERSTANDING OF MAJOR DEPRESSIVE DISORDER: THE ROLE OF INFLAMMATION AND IMPLICATION FOR IMMUNE MODULATORS - SYSTEMATIC REVIEW. N Amaladoss, E Sheppy, Michael G. DeGroote School of Medicine, McMaster University, Department of Psychiatry and Behavioral Neurosciences, McMaster University, Burlington, Ontario, Canada

SERUM KIBRA mRNA AND PROTEIN EXPRESSION AND COGNITIVE FUNCTIONS IN DEPRESSION. M Talarowska, M Kowalczyk, P Gałecki, J Szemraj, Department of Adult Psychiatry, Department of Medical Biochemistry, Medical University of Lodz, Lodz, Poland

11.30 – 12.00 Coffee Break

12.00 – 12.45	**ROUNDTABLE II: ETHICS IN BIOLOGY AND MEDICINE.**
	Moderators: AV Kalueff and M Pletnikov, USA
12.45 – 01.30	**ROUNDTABLE III: ANIMAL MODELS IN BIOMEDICAL RESEARCH: FROM LOGIC TO BIOETHICS.**
	Moderators: AV Kalueff and M Pletnikov, USA
01.30 - 01.45	**OFFICIAL CLOSING OF THE CONFERENCE.**
	ANNOUNCING FUTURE ISBS AND CBRD CONFERENCES

CONFERENCE ABSTRACTS

DAY 1, Sat, January 16, 2016

Opening Plenary Lecture:

MODELING GENE-ENVIRONMENT INTERACTION: A DIMENSION PERSPECTIVE. M Pletnikov, ISBS Fellow, Department of Psychiatry and Behavioral Sciences, Johns Hopkins University School of Medicine, Baltimore, MD, USA

Schizophrenia has long been considered as a disorder with multifactorial origins. Recent studies have advanced our knowledge of the genetic architecture of the disease. However, heritability estimates suggest a contribution of non-genetic factors. Various environmental risk factors have been associated with the increased risk for schizophrenia. These include season of birth, maternal infections, obstetric complications, stressful experiences during childhood, and drug abuse during adolescence. Despite the progress in identification of genetic and environmental risk factors, we have a poor understanding of the mechanisms of gene-environment interaction (GxE) in schizophrenia and psychoses at large. In this lecture I will review the current animal models of GxE relevant to psychotic disorders and propose that dimensional perspective will facilitate mechanistic studies of these disorders.

ISBS Special Lecture:

NURTURING ETHICAL RESPONSIBLE CITIZENS IN THE AGE OF INDIVIDUALISM. U Seraphin, ISBS Fellow Inductee, Saint Lucia Allied Health Practitioners Association (AHPA), St. Lucia, WI

Ethical responsibilities, in contrast to legal responsibilities, are moral rules that represent generally accepted good practices. Ethical responsibilities are critical for a well-functioning society because they include respect and honest dealing between individuals. As behavioral scientists, we tend to look at ethical problems from a psychological, rather than business, religious or legal, points of view. Thus, if we understand why people are motivated to behave ethically or unethically, we will know better how to prevent or minimize unethical behavior. In light of the expanding convolution of contemporary values, it is especially important to attentively nurture the inherent desire in each developing human person to seek good and avoid evil, especially during the critical years, such as adolescence. Individualism in decision-making entails making a choice that best serves one's long-term self-interest. In theory, if everyone makes decisions based on self-interest, everyone will benefit, which is why some businesses ethically favor this approach. Individualism considers personal benefit to be the most important factor when making a decision. Essentially, the company/society uses the selfish inclinations of individual workers as a motivational tool to improve overall performance. Long-term benefits are more desirable than short-term benefits, the theory argues, so long-term prospects should improve for everyone. The special goal of this talk is to present a comprehensive framework for developmentally appropriate activities, programming, and spiritual formation that support and nurture the development of ethical values. This holistic approach also integrates the resources of the community in a common effort for inter-generational mentoring of the young in becoming good citizen. We attempt to engage participants into critical thought and active self-discovery, and to examine how we see our moral value system to ensure commitment to personal integrity and moral social action within the fabric of the society. This will help develop moral reasoning to face the ethical realities and personal choices in nurturing ethically responsible citizens in the era of individualism, and be empowered to embrace the worthy adventure of living a moral, ethical life.

ISBS Presidential Lecture:

TRANSLATIONAL RESEARCH OF NEURODEVELOPMENTAL DISORDERS - THE ISBS TASK FORCE REPORT. AV Kalueff, ISBS Fellow, and the Task Force on Neurodevelopmental Disorders, The International Stress and Behavior Society (ISBS), New Orleans, LA, USA; St. Petersburg State University, St. Petersburg, Ural Federal University, Ekaterinburg, Russia; ZENEREI Research Center, Slidell, LA, USA; Guangdong Ocean University, Institute of Marine Drugs and Nutrients, Zhanjiang, China

Neurodevelopmental disorders (NDDs) are prevalent and debilitating brain illnesses due to aberrant brain development and cognitive, motor, language and affective disabilities. Common NDDs include autism spectrum disorder (ASD), intellectual disability, communication/speech disorders, motor/tic disorders and attention deficit hyperactivity disorder (ADHD), which remain an unmet biomedical concern. Genetic, epigenetic, and environmental factors play a key role in NDD pathogenesis. Multiple therapeutic approaches to NDDs include pharmacotherapy, behavioral therapy and rehabilitation, such as physical or speech/language therapy. However, specific and effective treatments for NDDs are lacking, as we do not know the biological targets and the exact symptoms, which are also often detected at clinically advanced stages, past the best therapeutic intervention period. Collectively, this necessitates further translational research in this field and the development of valid preclinical models, novel biomarkers and therapies. Animal models are also becoming an indispensable tool to study NDDs. To address these challenges, the International Stress and Behavior Society (ISBS) has established the Strategic Task Force on NDDs - a team of international experts representing different clinical and preclinical fields (Homberg et al., 2015). Here, we discuss the neurobiological mechanisms of NDDs and the existing preclinical tests and models which target key (social, cognitive, motor) neurobehavioral domains. Paralleling clinical findings across various selected common NDDs, we comprehensively evaluate various tests and models of animal neurobehavioral development, social behaviors, restricted interests and behavioral perseverations. Covering both traditional (rodent) and alternative NDD models and outlining the emerging areas of research, we emphasize how preclinical models play a key role in gaining translational and mechanistic insights into NDDs and their therapy.

ESTIMATING WHOLE BRAIN CONNECTIVITY DYNAMICS FOR RESTING STATE fMRI DATA. I Cribben, Y Yu, Department of Finance and Statistical Analysis, Alberta School of Business, Alberta, Canada; Statistical Laboratory, University of Cambridge, Cambridge, UK

INTRODUCTION: Recently, in functional magnetic resonance imaging (fMRI), there has been an increased interest in quantifying changes in connectivity between brain regions over an experimental time course. The application of graphical modeling has been instrumental in these analyses and has enabled the examination of the brain as an integrated system. In this work, we propose a new statistical method, called Network Change Points Detection (NCPD), which provides important insights into the time varying nature of the connectivity of brain regions. NCPD is applied to various simulated data sets as well as to a resting state functional Magnetic Resonance Imaging (fMRI) data set. The results illustrate the ability of NCPD to observe how the network structure changes over the time course. NCPD also allows us to identify common functional states across subjects. The temporal features of this novel connectivity method provides a more accurate understanding of the large-scale characterizations of brain disorders such as Alzheimer's disease and may lead to better diagnostic and prognostic indicators. **METHODS:** In this work, the objective is to understand whole brain dynamics or the situations where the number of brain regions exceeds the number of time points in the experiment. NCPD uses spectral clustering to study the network structure between brain regions and uses a non-parametric metric to detect the change in the structures across the time course. The method is very flexible as there is no *a priori* assumption on where the changes occur. We apply the new method to a resting state fMRI data set (Habeck et al., 2012). **RESULTS AND DISCUSSION:** There is evidence of state changes in the resting-state networks for all subjects. The results have led to the robust identification of cognitive states at rest. Several of the states are common across subjects. In particular, NCPD allows us to find common cognitive states that recur in time, across subjects, and across groups in a study. **RESEARCH SUPPORT:** This research was supported by a grant from the Alberta Health Services and from the Pearson Faculty Fellowship at the Alberta School of Business.

NEUROLOGICAL AND NEUROPSYCHOLOGICAL OUTCOMES AFTER SURGERY FOR MOYAMOYA DISEASE. SK Gupta, Department of Neurosurgery, PGIMER, Chandigarh, India

INTRODUCTION: Moya-Moya disease (MMD) is a progressive occlusive disease of the cerebral vasculature with particular involvement of the circle of Willis. The purpose of this study was to study the clinical profile and effectiveness of surgery in MMD patients. **METHODS:** The demographic profile, the clinical symptomatology was recorded. Diagnosis was confirmed by CT/CTAngiography, MRI/ MR angiography and/or DSA. SPECT was done to assess brain perfusion. The effectiveness of revascularization procedures was studied in terms of improvement in clinical profile, angiographic changes, and neuropsychological assessment. **RESULTS AND DISCUSSION:** 28 patients (32 sides) with MMD have been operated. The mean age was 13.7 years. There were 3 adult patients whose presentation was with ICH. The rest patients were all children with ischemia. Seizures were present in 12 patients (57.1%), focal deficit in 18 (85.7%), aphasia in 2 (9.5%) and features of raised intra cranial pressure in 1 patient (4.7%). Patients were investigated by DSA or MRA. SPECT studies were performed to detect areas of hypo-perfusion. STA-MCA anastomosis was done/attempted in all patients. The clinical progression of the disease was halted in almost all patients. One patient developed an additional ischemic event during surgery. Improvement in neuropsychological status was also observed especially in terms of increase in attention span and improved scholastic performance in school. Radiology showed regression in Moya Moya vessels in majority of the patients. Surgical revascularization procedures were effective in halting the neurological progression of the disease and also resulted in neuropsychological and radiological improvement.

FLATTENED SHEET-LIKE FORNIX FORMING A 'COBRA HOOD' DEFORMITY: A PREVIOUSLY UNREPORTED VARIANT OF FORNIX ANATOMY AND ITS IMPLICATIONS FOR SURGICAL APPROACHES TO THE THIRD VENTRICLE. T Gupta, Department of Anatomy, PGIMER, Chandigarh, India

INTRODUCTION: The fornix is main efferent tract from the hippocampus and is an important component of memory pathways. Variations of fornix anatomy are not commonly encountered. **METHODS:** Fornix anatomy was studied in 30 cadavers of normal adult healthy males who had died in road accidents. **RESULTS AND DISCUSSION:** In 10 of the 30 brains, the crura and the body of fornix were bilaterally broad and flat like a sheet, rather than the usual compact bundle, forming a cobra-like hood over the roof of the third ventricle. The maximum width was approximately 16 mm on the right side (mean: 11.7 mm) and 11 mm on the left (mean: 8.5 mm). Knowledge of this variation will be useful during the inter forniceal or subchoroidal approach to third ventricle tumors, especially in the latter, as an unexpected lateral span of the fornix in the surgical corridor can result in inadvertent injury to it, leading to memory defects.

BENEFITING FROM CANNABIS. G St. Rose, ISBS Fellow, Eden Herbs, Creative Heath Center, St. Lucia, WI

The current discussion and exchange of views with regard to the use of marijuana/cannabis/Hemp needs to be carried out with a comprehensive knowledge of their nature and potential. The differences in industrial hemp and medicinal or recreational marijuana will be discussed. The particular cannabinoids in medicinal marijuana and their specialized use is related to the ratios of cannabidiol and tetrahydrocannabinol. In summary, a plant/herb which was cultivated and used for multiple purposes without any destructive societal consequences around the world prior to 1937, is now underutilized and criminalized, whereas persons are deprived of its great economical and medicinal activities.

DAY 2, Sun, January 17, 2016

ISBS Plenary Lecture:

NEUROIMMUNE FUNCTION IN DEPRESSION AND SUICIDE. GN Pandey, Department of Psychiatry, University of Illinois at Chicago, Chicago, IL, USA

INTRODUCTION: Abnormalities of immune functions have been implicated in the neurobiology of depression and suicide. This is based on the observed abnormalities of cytokines in the serum of depressed and suicidal patients. Although serum levels of cytokines have been studied in depression, it is not clear if similar abnormalities exist in the brain. Also, the levels of membrane-bound cytokine receptors have not been studied in these disorders. Therefore, we have studied cytokines and their membrane-bound receptors in the plasma and postmortem brain of depressed and suicidal subjects. **METHODS:** We determined the protein and mRNA expression of proinflammatory cytokines and TLRs in the postmortem brain (prefrontal cortex [PFC], Brodmann area 9 [BA9]) of 24 depressed suicide victims and 24 normal control (NC) subjects and mRNA of proinflammatory cytokines and their membrane-bound receptors in lymphocytes of 30 depressed patients and 30 control subjects. Protein expression was determined using either ELISA or Western blot, and mRNA expression using the qPCR technique. Patients were diagnosed according to DSM-IV SCID. **RESULTS AND DISCUSSION:** We found that the gene expression of proinflammatory cytokines IL-1β, IL-6 and TNF-α were significantly increased in the lymphocytes of depressed patients compared with NC subjects. We also found that the mRNA levels of IL-1β, IL-6, TNF-α and their receptors IL-1R1, TNFR1, TNFR2 and IL-1RA were increased with no changes in IL-1R2 and IL-6R in the lymphocytes of depressed patients compared to controls. We also found that the gene and protein expressions of proinflammatory cytokines IL-1β, IL-6 and TNF-α, TLR-3 and TLR-4 were significantly increased, whereas the levels of IL-10 were significantly decreased in the PFC of depressed suicide subjects. These studies show abnormalities of both adaptive and innate immunity in depression and suicide, and also that the receptors for the cytokines and the TLRs may be an appropriate target for developing new therapeutic agents for the treatment of depression and suicidal behavior. **RESEARCH SUPPORT:** This work was supported by the US National Institutes of Health, NIMH RO1 MH098554.

GENETIC RISK FACTORS AND ADOLESCENT CANNABIS ABUSE: THE ROLE OF ASTROCYTES. M Pletnikov, ISBS Fellow, Department of Psychiatry and Behavioral Sciences, Johns Hopkins University School of Medicine, Baltimore, MD, USA

The role of psychiatric genetic risk factors in astrocytes is poorly studied. Astrocytes express cannabinoid receptors 1 (CB1) that mediate adverse cognitive effects of chronic adolescent exposure to tetrahydrocannabinol (THC). We evaluated how astrocytic expression of a genetic risk factor, mutant Disrupted-In-Schizophrenia-1 (DISC1), modulates cognitive dysfunction produced by adolescent cannabis. Control and mutant DISC1 mice were injected daily with 8 mg/kg, sc of Δ9-THC for 21 days beginning at postnatal day (P) 30 or P90. A separate cohort of mice was identically treated with THC and was given doxycycline (DOX) food to shut down expression of mutant DISC1 during THC injections. 3 weeks after treatment, the different cohorts of mice were evaluated in cognitive tests. Mice were sacrificed, and parvalbumin+(PV) neurons number estimation and RNAseq of hippocampal tissue samples were performed. Compared to control groups, THC-treated DISC1 mice exhibited deficits in recognition memory, and these deficits were not present in THC-DISC1 mice given DOX, indicating the critical role of adolescent expression of mutant DISC1. THC exposure produced a synergistic decrease in the number of PV+ neurons and significant changes in cAMP and adenosine signaling in the hippocampus. Our findings suggest a new role for astrocytes in mediating adverse effects of adolescent exposure to cannabis.

ISBS Plenary Lecture:

CURRENT CONTROVERSIES IN PAIN RESEARCH. WC Parris, Department of Anesthesiology, Duke University School of Medicine, Durham, NC, USA

Chronic Pain Medicine is a little over three decades old, and during that time great progress has been made in successfully addressing the management of both acute and chronic pain syndromes. Yet for too long, patients with chronic pain were just allowed to suffer needlessly, and this situation not only was ethically unsatisfactory, but resulted in significant morbidity and occasional mortality. Fortunately, that epoch is already behind us, and in most cities throughout the world, some kind of Pain Management is offered to patients with chronic pain. Despite this great progress, there are still areas where progress is lagging, and several pain mechanisms are either poorly understood or not understood at all. In this presentation, several areas where progress has been made will be reviewed, and highlights and “lowlights” will be discussed. The topics to be explored include: A new paradigm for Pain Medicine; Epidural steroids; Cervical epidural steroid injections; The blessings and curses of narcotic administration; Fidapin: a new topical analgesic; Substance P and its relevance; Animal models of Cancer Pain; Neurolytic procedures; Percutaneous neuroplasty for failed back surgery and Spinal Stenosis; Ethics in Pain Medicine. These topics are not all controversial, but are examples of notable progress made in the scientific arena of Pain Medicine, and have the potential to effect drastic changes on the way chronic pain patients will be managed in the future. One major source of encouragement is the large number of research projects that are currently ongoing, and the growing literature that is being published daily on Pain-related topics. All this scholarly activity bodes well for the future, and the hope is that better, safer, more effective and longer-lasting drugs and procedures will be offered to more comprehensively manage chronic pain patients.

UPDATE ON CANNABIS NEUROBIOLOGY. M Fraites, St. Lucia, WI

I will outline the earliest known history of cannabis and some of the most significant studies and discoveries made in this field, showing that humans have long been inextricably linked with cannabis. The first isolation of the THC molecule from cannabis was a breakthrough that led to the discovery and understanding of the endocannabinoid system. However, we have insight, but not a full understanding, of how the endocannabinoid system can be used to prevent, diagnose and treat a disease, defect or symptom of illness. The endocannabinoid system is comprised of receptors, their endogenous ligands, and the proteins synthesized to degrade them. Research has identified various cannabinoid receptors in the brain and immune cells which respond to agonists or inverse agonists which may be endogenous, synthetic or phyto-derived. The cannabinoid receptors are abundant in the mammalian brain. Appropriate levels of cannabinoids appear to be required to support pregnancy, and breast milk contains cannabinoids for the development and growth of the newborn. With a firm understanding of the endocannabinoid system and the 60+ cannabinoids, we can prevent or eliminate the few negative possible outcomes of Cannabis use and maintain optimum health.

DEPRESSIVE DISORDERS: NEW CLASSIFICATION AND MANAGEMENT. G Swamy, St. Lucia, WI

Diagnostic criteria for mood disorders will be discussed here based on the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) developed by the American Psychiatric Association, and on the International Classification of Disorders (ICD) developed by the Word Health Organization (WHO).

BOOK PRESENTATION

THE RIGHTS AND WRONGS OF ZEBRAFISH: PRINCIPLES OF BEHAVIORAL PHENOTYPING. Editors: AV Kalueff and AM Stewart (Springer, 2016). Zebrafish *(Danio rerio)* is a small aquatic vertebrate fish species which has travelled all the way from India to rapidly emerge as a promising model organism in developmental biology and genetics. Recently, this fish has entered the waters of neuroscience and biological psychiatry, again quickly becoming an indispensable model species in this field. With a high genetic homology to humans (70-75%), it is perhaps unsurprising that humans and fish are very similar physiologically – perhaps even more than we can admit. Therefore, it should not come as a surprise that zebrafish can be considered as an excellent model of human neuropsychiatric disorders. While some classical psychiatrists may not too easily be persuaded by this generalization, the current book explains, in a domain-by-domain manner, how exactly we can use zebrafish models to best study a wide range of human brain disorders and disordered phenotypes. Written by top experts in the field, this book is also well illustrated, collectively making it a useful and balanced up-today reading.

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ISBS Special lecture:

HOW HETERO-NORMATIVITY AND HOMO-NEGATIVITY IMPEDES LBGTQI PEOPLE FROM ACCESSING HARM REDUCTION SERVICES. M Day, Caribbean Drug and Alcohol Research Institute (CDARI), St. Lucia, WI

INTRODUCTION: As a public health professional working with a highly diverse population persons with HIV, I have observed how diversity is often indicated by visible attributes, such as race or sex. However, diversity encompasses many more variables than these observable factors and includes sexual orientation. In the Caribbean, much effort has been expended to educate medical professionals working with PWHIV about issues related to sexual diversity and sexual orientation as an effort to remove or lessen the barriers that keep lesbian, gay, bisexual, transsexual, queer and intersex (LGBTQI) people from accessing or utilizing health care services. **OBJECTIVE:** To introduce clinicians and other health care professionals working with PWHIV to the concept of heteronormativity, its effect on society, and its influences on the manner in which they deliver health care services to their patients. Heteronormativity is defined as a cultural understanding in which heterosexuality is the norm and the resulting social institutions are based on the assumption that men are sexually and romantically attracted to women and women are attracted likewise to men; the possibility of same-sex attraction is neither acknowledged by the public at large nor recognized by its social institutions. Homo-negativism, on the other hand, is an inclusive term that describes purposeful, negative attitudes and behaviors toward non-heterosexuals. Homo-negativism is learned and incorporates the social context in which negative, prejudicial, or discriminatory attitudes and/or behaviors toward non-heterosexuals are developed and maintained. Widespread heteronormativity empowers homonegativity to reinforce rigid gender stereotypes that lock men and women into their respective role. **CONCLUSIONS:** A concerted effort must be made by service providers to create an environment of acceptance of the diversity of gender expression, gender identity and sexual orientation among our clients and patients.

DAY 3, Monday, January 18, 2016

INTERACTIVE GUIDED POSTER MINI-SYMPOSIUM

IMPAIRED EFFICIENCY OF BASE EXCISION REPAIR SYSTEM IN NUCLEAR AND MITOCHONDRIAL EXTRACTS FROM PERIPHERAL BLOOD LYMPHOCYTES OF PATIENTS WITH ALZHEIMER'S DISEASE.

T Sliwinski, D Kwiatkowski, P Czarny, M Toma, P Galecki, A Bachurska, J Szemraj, University of Lodz Department of Molecular Genetics, Medical University of Lodz, Department of Adult Psychiatry, Department of Medical Biochemistry, Lodz, Poland

INTRODUCTION: Oxidative damage and reduced DNA repair efficiency may be the reason that causes the neurodegenerative disease such as Alzheimer's disease (AD). These oxidative modifications of DNA bases are removed via the base-excision repair (BER) pathway, which substantially contributes to the stability and integrity of the genome. Oxidative DNA damage in AD may occur both in the central nervous system and in peripheral blood lymphocytes. Additionally, previous reports have also associated mitochondrial dysfunction with AD. Loss of mitochondrial genomic integrity is also implicated in AD pathology. DNA repair processes in mitochondria are not as comprehensive as they are in the nucleus but BER is well conserved in this compartment. Taken together, this data suggest that the altered levels of BER in nucleus as well as in mitochondria may be linked with Alzheimer's disease occurrence. **OBJECTIVES:** The aim of presented studies was to determine potential relationship between the efficacy level of base excision repair system in nuclear and mitochondrial extracts from peripheral blood lymphocytes and risk of Alzheimer's disease in Polish population. **METHODS:** In the present work we determined the efficacy level of BER system in nuclear and mitochondrial extracts from peripheral blood lymphocytes of 40 AD diagnosed patients and 50 healthy individuals. Efficacy level of base excision repair was evaluated by a functional assay in vitro using 5′ radiolabeled ds-oligodeoxynucleotides containing AP-site. **RESULTS AND DISCUSSION:** We observed an association between AD occurrence and the decreased level of BER system in nuclear extracts from peripheral blood lymphocytes of patients with AD. Additionally, significantly decreased level of this repair system was also noticed in mitochondria of peripheral blood lymphocytes of AD patients when compared with healthy controls. CONCLUSIONS: Our results suggest that the impaired efficacy of BER system may be linked with AD risk by the modulation of the cellular response to oxidative stress and might be considered as diagnostic biomarker of AD in peripheral blood lymphocytes. **RESEARCH SUPPORT:** This study was supported by the Polish National Science Centre (grant DEC-2012/05/B/NZ7/03032).

ANTIDEPRESSANT-MEDIATED INCREASE IN THE EXPRESSION AND RELEASE OF GLIAL-DERIVED NEUROTROPHIC FACTOR LEADS TO INCREASED NEURO-GENESIS. S Avissar, D Fux, M Golan, G Schreiber, Ben Gurion University, Beer Sheva, Barzilai Medical Centre, Ashkelon, Israel

INTRODUCTION: Neurotrophic factors were found to contribute to neuronal plasticity in relation to the pathophysiology of major depression and to the mechanism of action of antidepressants. Recent postmortem studies demonstrate that mood disorders are characterized by specific histopathological changes in both neurons and glial cells. Cell loss or atrophy may also be related to developmental factors resulting in diminished amounts of neurotrophic factors release that might be responsible for several of the pathological changes in major depression. Glial cell-derived neurotrophic factor (GDNF), essential for glial survival, plasticity and development, has been implicated in the mechanism of action of antidepressant drugs (ADs). Beta-arrestin1, a member of the arrestin protein family, was found to play a role in AD mechanism of action. The present study aimed at evaluating whether ADs therapeutic effects are achieved through β-arrestins-mediated regulation of GDNF neuroprotective action by promoting cellular survival and plasticity of neurons compromised in major depression. **METHODS:** C6 glioma cells were treated for 3 days with 10 □M imipramine. After 72 h, medium was collected (conditioned medium) and used as a supplement to the SH-SY5Y regular growth medium. Forty eight hours after SH-SY5Y seeding, growth medium was replaced with starvation medium (1% FCS) supplemented with 20%, 30% and 40% C6 conditioned medium for 48 h. The cytosol of SH-SY5Y cells was obtained by fractionation and analyzed by Western blot for effects on β-arrestin 1, neurogenesis markers: NSE, NeuN, GAP43, MAP2, and brain-derived neurotrophic factor (BDNF). **RESULTS AND DISCUSSION:** Imipramine significantly increased both GDNF expression and release from C6 glioma cells, but was unable to induce such effects in beta-arrestin1 knock-down cells, thus supporting the involvement of beta-arrestin1 in antidepressant regulation of neurotrophic factors. Exposure of SH-SY5Y neuroblastoma cells to GDNF-conditioned medium collected from imipramine-treated C6 glioma cells (CM) resulted in a significant increase in both β-arrestin 1 protein levels and in neurogenesis markers (e.g. NSE, NeuN, GAP43, MAP2) as well as in BDNF in these neuronal cells. Our findings suggest that exposure of SH-SY5Y neuronal cells to GDNF secreted by glial cells treated with the antidepressant drug imipramine results in the activation of neurogenesis and differentiation processes in these neuronal cells. This is reflected by increased levels of brain-derived neurotrophic factor and several protein markers of neurogenesis, and thus provides further support to the neurotrophic hypothesis of depression. **RESEARCH SUPPORT:** SA is incumbent of Eugene Hecht Chair in Clinical Pharmacology.

IMMUNOGENETICS IN DEPRESSED PATIENTS WITH MAJOR DEPRESSIVE AND BIPOLAR DISORDERS. O Mikova, P Marinov, A Angelova, A Mihaylova, M Ivanova, Ts Lukanov, M Demirev, Foundation Biological Psychiatry, Sofia, Bulgaria; Alexandrovska University Hospital, Sofia, Bulgaria

INTRODUCTION: Depressive disorders are the most common psychiatric disorders in Europe, with an approximately 13% lifetime incidence with 4% being diagnosed with major depression in the previous 12 months. The depressive disorders contribute 11% of the global years live with disability. Differentiating between patients with Bipolar Disorder (BD) in the depressed state of their disease and patients with Major Depressive Disorder (MDD) in a depressed episode has always been a challenging task. The importance of early life experience in developing chronic diseases in adulthood has been stressed in a number of previous studies. We studied the relationship of childhood trauma and psychiatric illness in adulthood and immunogenetic factors in order to try to create an approach to better differentiate these depressive states. **METHODS:** We measured several psychometric scales, including the Childhood Trauma Questionnaire (CTQ), and performed a number of biological tests in patients with MDD and BD, as well as in healthy controls. DNA extraction was performed according to the validated, (using iPrepTM PureLinkTM gDNA Blood Kit and iPrepTM Purification Instrument), laboratory protocol. For HLA-DR, DQ genotyping two molecular techniques were chosen – PCR-SBT and PCR-SSP. For MBL2 genotyping, including 6 functionally relevant SNPs, a Luminex-based assay was applied. Statistical analysis: Allele frequencies were estimated by maximum-likelihood analysis using the Arlequin program v1.1 (Ivanova et al., 2008). Standard deviations were calculated from 100 bootstrap iterations. Hardy–Weinberg equilibrium was tested by a hidden Markov chain with 100,000 steps, implemented in the Arlequin program. Arlequin software was also used to estimate maximum-likelihood three-locus haplotype frequencies from genotypic data through an expectation-maximization (EM) algorithm (Slatkin et al., 1996). **RESULTS AND DISCUSSION:** We measured CTQ and performed immunogenetic analysis for 63 patients with MDD, 47 patents with BD and 42 healthy sex- and age-matched controls. Results showed significant differences between the two patient groups for both the psychological questionnaire for childhood trauma and immunogenetic analysis. These include with higher frequency of B*08, and DQB1*05:03 in BPD than MDD. Haplotypes A*02 B*35 DRB1*1101 DQB1*0301 and A*24 B*35 DRB1*1101 DQB1*0301 were observed with higher frequency only in MDD, while A*24 B*35 DRB1*1401 DQB1*0503 and A*02 B*35 DRB1*1104 DQB1*0301 haplotypes were found with higher frequency in BPD. The significance of these results is to be discussed further. They surely show us that these two depressive populations differ greatly in psychological and biological terms and that a better diagnosis using an optimal diagnostic biomarker/psychological marker panel for depression can be achieved. **RESEARCH SUPPORT:** This research was supported by EU grants: Moodinflame, and Psychaid.

PESPECTIVES ON ZEBRAFISH BEHAVIORAL PHENOMICS. AV Kalueff, ISBS Fellow, St. Petersburg State University, St. Petersburg, Ural Federal University, Ekaterinburg, Russia; ZENEREI Research Center, Slidell, LA, USA; Guangdong Ocean University, Institute of Marine Drugs and Nutrition, Zhanjiang, China

INTRODUCTION: The zebrafish (*Danio rerio*) is a promising model organism for *neurophenomics*, which represents a new field of neuroscience that links neural phenotypes to various genetic and environmental determinants. Experimental designs in this field often include testing same animals in multiple tests – i.e., using test batteries. However, the effects of prior experimental manipulations on zebrafish performance in various behavioral paradigms remain unclear. **METHODS:** Here, we examine the influence of stressful procedures and test batteries on adult zebrafish anxiety-like behaviors in two commonly used models - the novel tank (NTT) and the light-dark box (LDB) tests. **RESULTS AND DISCUSSION:** Overall, we found no overt behavioral strain differences between short-fin wild-type (WT) and mutant pink glowfish in both tests under baseline (control) conditions. In contrast, an acute severe stressor (a 30-min car transportation) revealed the strain differences, including significantly lower WT anxiety-like behavior in both NTT and LDB compared to mutant glowfish groups. However, WT zebrafish showed no overt NTT or LDB responses following a mild stressor (5-min 40-Wt light) exposure. Likewise, no differences were observed between control WT zebrafish and fish tested in batteries when NTT and LDB were either run immediately one after another, or with a 1-day interval. Collectively, these findings suggest that zebrafish may be less sensitive (e.g., than other popular species, such as rodents) to the test battery effect, and show that stronger stressors may be needed (to complement low-to-moderate stress aquatic screens) to reveal phenotypical variance. Strengthening the value of zebrafish models in neurophenotyping research, this indicates the potential of using test batteries and a wider spectrum of pre-test stressors in zebrafish *in-vivo* behavioral assays. **RESEARCH SUPPORT:** This work is supported by the International platform grant, Department of Education of Guangdong Province, China (Guangdong Ocean University, GDOU) and GDOU Research Institute of Marine Drugs and Nutrition (RIMDN).

A NOVEL UNDERSTANDING OF MAJOR DEPRESSIVE DISORDER: THE ROLE OF INFLAMMATION AND IMPLICATION FOR IMMUNE MODULATORS - SYSTEMATIC REVIEW. N Amaladoss, E Sheppy, Michael G DeGroote School of Medicine, McMaster University, Department of Psychiatry and Behavioral Neurosciences, McMaster University, Burlington, Ontario, Canada

Depression is classically attributed to hypoactivity of certain monoamine neurotransmitters, and most anti-depressants were developed based upon this theory. Unfortunately, up to 1/3 of depressed patients derive little benefit from conventional pharmacotherapy, prompting the search for new frameworks on the pathogenesis of depression. The correlation of depression with chronic inflammatory conditions suggests that immune responses, particularly inflammation, may contribute to depression manifestation. Here, we systematically review current evidence correlating depression severity with inflammatory biomarkers [1], as well as evaluate the efficacy of immunomodulatory therapy [2] as a treatment modality for depression. **REFERENCES:** 1) Dowlati Y et al. 2010. A meta-analysis of cytokines in major depression. Biol Psychiatry. 67(5): 446-57. 2) Raison CL et al. 2013. A randomized controlled trial of the tumor necrosis factor antagonist infliximab for treatment-resistant depression: the role of baseline inflammatory biomarkers. JAMA Psychiatry 70(1): 31-41.

SERUM KIBRA mRNA AND PROTEIN EXPRESSION AND COGNITIVE FUNCTIONS IN DEPRESSION. M Talarowska, M Kowalczyk, P Gałecki, J Szemraj, Department of Adult Psychiatry, Department of Medical Biochemistry, Medical University of Lodz, Lodz, Poland

INTRODUCTION: Genes participating in synaptic signalling or plasticity in brain regions such as the prefrontal cortex (PFC) and the hippocampus have been implicated in cognition. Recently, a new gene (KIBRA, WWC1) has been added to this group due to its impact on memory performance. Recurrent depressive disorder (rDD) is a multi-factorial disease, that one of the typical features is cognitive impairment. The main objective of this study was to perform an analysis of the KIBRA gene on both mRNA and protein levels in patients suffering from rDD and to investigate the relationship between KIBRA expression and cognitive performance. **METHODS:** The study comprised 236 subjects: patients with rDD (n=131) and healthy subjects (n=105, HS). Cognitive function assessment was based on: Trail Making Test, The Stroop Test, Verbal Fluency Test and Auditory Verbal Learning Test. **RESULTS AND DISCUSSION:** Both mRNA and protein expression levels of KIBRA gene were significantly higher in healthy subjects when compared to rDD. The presented relationship is clear even after taking age, education and sex of the examined subjects into consideration. No statistically significant relationship was found in the experiments between any of the conducted tests and KIBRA gene expression on mRNA level for both the rDD and HS groups. The presented study has limitations related to the fact that patients were being treated with antidepressant. This is relevant due to the fact that some antidepressants may affect mRNA expression. Number of patients and healthy subjects may result in the lack of statistical significance in some cases. **CONCLUSIONS:** The results of our study show decreased expression of the KIBRA gene on both mRNA and protein levels in depression, whereas we did not find any significant relationship between KIBRA. **RESEARCH SUPPORT:** Research was supported by Medical University grant no 502-64-065.

For notes

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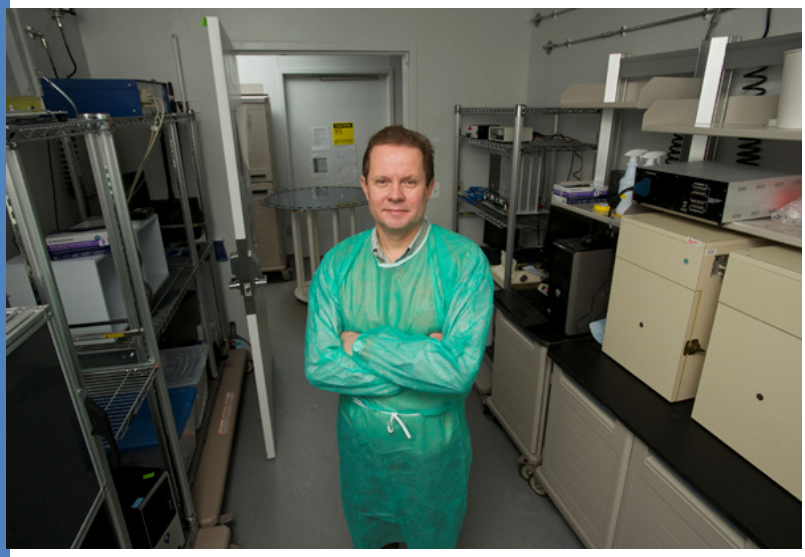

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